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March 6, 2001

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Re: Request For Immediate Governmental Action/Regulation Relating To DuPont's C-8 Releases In Wood County, West Virginia And Notice Of Intent To Sue Under The Federal Clean Water Act, Toxic Substances Control Act, And Resource Conservation And Recovery Act - NOTE: For Inclusion In USEPA Docket No. OPPTS-50639A

Ladies and Gentlemen:

Our law firm represents Wilbur Earl Tennant and Sandra K. Tennant (Route 3, Box 17, Washington, WV 26181, (304) 863-8787), James David Tennant and Della Marie Tennant (Route 3, Box 372, Parkersburg, WV 26101, (304) 863-5428), and Erwin Jackson Tennant (Route 3, Box 17A, Washington, WV 26181, (304) 863-6977) (collectively, the "Tennants") in connection with a lawsuit that is currently pending against E.I. duPont de Nemours & Co., Inc. ("DuPont") in Federal Court in Parkersburg, West Virginia, styled *Tennant v. E.I. duPont de Nemours & Co., Inc.*, Civil Action No. 6:99-0488 (S.D. W.Va.). The Tennants have sued DuPont in connection with the release of various pollutants and contaminants from DuPont's Dry Run Landfill in Wood County, West Virginia. (See Exhibit 133.) The Tennants believe that

such releases have resulted in and continue to result in personal injury and property damage to the Tennants, including the death of several hundred head of the Tennants' cattle and serious health problems for the Tennants.

During the course of the litigation, we have confirmed that the chemicals and pollutants released into the environment by DuPont at its Dry Run Landfill and other nearby DuPont-owned facilities may pose an imminent and substantial threat to health or the environment. More specifically, information currently available to the Tennants confirms that DuPont has been releasing and continues to release into the air, land, and water, including human drinking water supplies, an essentially unregulated, confirmed animal carcinogen known as ammonium perfluorooctanoate (a/k/a C-8/FC-143/APFO/PFOA) (CAS No. 3825-26-1) (hereinafter "C-8").[1] Hundreds of head of cattle, along with numerous deer, fish, frogs, and other animals, have died in the area affected by the C-8 releases, and area residents exposed to the C-8 releases have been suffering ill health effects that are believed to be associated with C-8 exposure. For example, one of our clients, Wilbur Earl Tennant, has been in and out of the hospital repeatedly over the last few years suffering from respiratory problems, chemical burns, and other health problems after exposure to materials from the Dry Run Landfill.

For the reasons discussed in more detail below, the Tennants hereby request that each of your agencies intervene in the Tennants' pending lawsuit and order the immediate investigation, assessment, containment, removal, and remediation of DuPont's C-8 releases into the environment from the Dry Run Landfill, including an order that DuPont immediately cease and desist all C-8 releases and that appropriate medical care/testing/evaluation be provided to the Tennants. The Tennants also request that DuPont's permit to operate the Dry Run Landfill be immediately revoked and that all operations at that landfill be suspended until adequate scientific demonstrations are made to prove that the C-8 releases have been abated and will not recur.

In addition, the Tennants specifically request that USEPA exercise its authority under TSCA to order DuPont to immediately cease all manufacturing activities involving C-8 until DuPont can prove through appropriate scientific testing and research that its usage of C-8 does not pose an unreasonable risk of injury to health or the environment. In the meantime, the Tennants request that your agencies take those steps necessary to begin regulating C-8 releases into the environment. In that regard, the Tennants request that, at a minimum, USEPA include C-8 among the chemicals that it proposed in October of 2000 to regulate under TSCA on the grounds that the chemicals "may be hazardous to human health and the environment." (See Exhibit 123.) The Tennants believe that the information recently obtained from DuPont regarding C-8's potential threat to human health, (see e.g., Exhibits 71, 125, and 126), warrants regulation of C-8 at least as aggressively as the related perflourinated chemicals manufactured by 3M.

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[1] Currently available information also indicates unusual levels of iodide/iodine, along with Triton in Dry Run Creek. (See Exhibit 91.)

This letter also constitutes notice on behalf of the Tennants and a class of other individuals similarly situated of their intent to bring citizen suit claims against DuPont in connection with DuPont's C-8 releases into air, land, and water from DuPont's Washington Works facility in Wood County, West Virginia under the Federal Clean Water Act ("CWA"), Toxic Substances Control Act ("TSCA"), and Resource Conservation and Recovery Act ("RCRA").[2] The factual and legal basis of such citizen suit claims is explained in detail below.

Additional documentation in support of the basic facts summarized below is available at our offices in Cincinnati, including a chronologically-organized database of the over 110,000 pages of documents produced to date by DuPont on this topic.

## I. DuPont Has Used C-8 Primarily At Its Washington Works Plant In Wood County, West Virginia.

C-8 is a perfluorinated detergent/surfactant manufactured in the United States by 3M Company that DuPont uses in connection with its manufacture of Teflon®-related products. (See Exhibits 1 and 118.)[3] DuPont has used C-8 as a reaction aid in its production of polytetrafluoroethylene (PTFE) and tetrafluoroethylene (TFE) co-polymers at its Washington Works facility outside Parkersburg, West Virginia since the early 1950s. (See Exhibit 118.) Wastes from the Washington Works' C-8 processes are either vented to the air following incineration, dumped into the Ohio River, sent to DuPont's Chambers Works facility in Deepwater, New Jersey for treatment and discharge, or disposed of at landfills. (See id.) The polymer product manufactured at the Washington Works is either sold directly to DuPont's customers (in the United States and abroad) or transferred to DuPont's Spruance Plant in Richmond, Virginia for use in the production of Teflon® and PTFE-coated fibers or transferred to DuPont's Parlin Plant in Parlin, New Jersey for use in the production of Teflon® finishes, some of which is then used in consumer cookware. (See id.) C-8 may remain in some of the products sold from DuPont's Washington Works, Spruance Plant, and Parlin Plant. (See id.) Some of DuPont's Teflon® materials have been used in medical implants that are inserted directly into the human body. (See Exhibit 132.)

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[2] Please note that, although the Tennants already have filed claims against DuPont under the CWA and RCRA, these pending claims relate only to releases from DuPont's Dry Run Landfill. This letter provides notice of the Tennants' intention to also bring separate claims against DuPont under the CWA, TSCA, and RCRA with respect to releases from DuPont's nearby Washington Works plant in Wood County, West Virginia, on behalf of themselves and a class of others similarly situated.

[3] ®DuPont's registered trademark.

## II. DuPont Has Known That Excessive Exposure To C-8 Causes Adverse Effects.

DuPont has worked closely with 3M since at least the 1970s to investigate the toxic and carcinogenic effects of C-8 on animal and human health. (See id. and Exhibits 2, 24, and 49.) Through such company-sponsored studies, DuPont acquired knowledge by at least the early 1980s that C-8 was toxic and carcinogenic to animals, whether through inhalation, direct skin contact, or ingestion. (See Exhibits 12, 49, and 71.) Around the same time, DuPont also became aware that C-8 is biopersistent/bioaccumulative in animals and humans. (See Exhibits 30, 49 and 71.)[4]

In response to the mounting toxicity data on C-8, and because C-8 was essentially an unregulated chemical that, according to USEPA, had simply "sail[ed] under the agency regulatory radar screen" for decades, (see Exhibit 114), DuPont established in the 1980s its own internal standards for what it considered to be acceptable C-8 exposure levels for humans. For exposure to C-8 via air emissions/inhalation routes, DuPont determined that an "acceptable exposure limit" (AEL) for humans is 0.01 $mg/m^3$ (skin), with an acceptable "community exposure guideline" (CEG) for airborne emissions of 0.0003 $mg/m^3$. (See Exhibits 2-4, and 9.) For human exposure to C-8 through contaminated water, DuPont established a CEG of 1 ppb. (See id.) DuPont also began routine monitoring of the levels of C-8 in the blood of its own employees, including employees at Washington Works, as early as 1981, (see Exhibit 118), and began looking for alternatives to C-8. By 1993, DuPont believed it may have found a viable, less toxic alternative to C-8, (see Exhibit 42), but decided to keep using C-8 anyway.

Later in 1993, a study conducted by the University of Minnesota linked C-8 exposure with increased prostate cancer among human males. (See Exhibits 47 and 51.) By 1996, DuPont also had been informed that new tests were linking C-8 to DNA damage. (See Exhibit 60.) In response, DuPont, 3M, and others commissioned studies to further assess the potential effects of C-8 on humans through tests on monkeys. (See Exhibits 77, 84, 93, and 105.) By November of 1998, DuPont knew that one of the monkeys in the study receiving a 30 mg/kg dose of C-8 was suffering severe health effects. (See Exhibit 90.) By February of 1999, DuPont knew that one of the monkeys involved in the C-8 testing receiving the lowest dose of C-8 (3 mg/kg) had suffered such severe health effects that it had to be sacrificed. (See Exhibit 94.) By May of 1999, DuPont knew that a second monkey in the study had also suffered such severe health effects that it had to be sacrificed. (See Exhibits 103, 105, 107, 108 and 125.) The preliminary monkey study results also confirmed adverse liver effects among all of the monkeys in the study, regardless of exposure levels. (See id. and Exhibits 125 and 126.) Thus, because even exposure to the lowest

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[4] DuPont also became aware of evidence as early as 1981 that at least two children born to its Washington Works employees who worked with C-8 while pregnant appeared to have been born with birth defects similar to those observed among rats exposed to high levels of C-8. (See Exhibit 13.)

dose of C-8 during the studies (3 mg/kg) produced adverse observable effects, a "no observable effects level" (NOEL) could not be found for C-8 in primates. (See Exhibits 105, 126.)

3M eventually notified USEPA of the preliminary results of the monkey study in a filing under TSCA, Section 8(e) during November of 1999. (See Exhibit 111.) Within only a few months, USEPA notified 3M that it intended to pursue more rigorous regulation of the perfluorinated chemicals manufactured by 3M. (See Exhibits 113 and 120.) Soon thereafter, 3M publicly announced that it would "voluntarily" withdraw from the market all of its perfluorinated chemical products, including the C-8 that it sells to DuPont for use in DuPont's Teflon® products, and the chemicals 3M uses to make its Scotchguard® products. (See Exhibits 113 and 114.)[5]

After learning that DuPont was one of the principal users of 3M's C-8 product, USEPA's TSCA Division requested in April of 2000 that DuPont supply information regarding DuPont's usage and release of C-8 within the United States. (See Exhibit 112.) DuPont produced some C-8 research data to USEPA on May 25, 2000, (see Exhibit 115), followed by preliminary usage and release information in a letter dated June 23, 2000. (See Exhibit 118.) In its C-8 disclosure letter to USEPA, DuPont confirmed that it has used C-8 primarily at its Washington Works site and that it had released C-8 into the air, water, and land at the Washington Works, into water at its Parlin Plant, Spruance Plant, and Chambers Works, into soils at the Chambers Works, and into soil and water at the "Local," Letart, and Dry Run Landfills owned and operated by DuPont near the Washington Works in West Virginia. (See id.) DuPont did not, however, reference any of the results of the C-8 monkey studies. (See id.) On October 18, 2000, USEPA proposed to begin regulating most of 3M's perfluorinated chemicals under TSCA on the grounds that the chemicals "may be hazardous to human health and the environment." (See Exhibit 123 (65 Fed. Reg. 62319-33 (Oct. 18, 2000)).) USEPA deferred, however, regulation of C-8, pending further review of the information being obtained from 3M and DuPont. After receiving a draft of this letter in November of 2000, DuPont sent revised C-8 usage and release information to USEPA in a letter dated January 25, 2001. (See Exhibit 136.) As of today's date, however, the Tennants are not aware of the results of the C-8 monkey studies having been "finalized" or published.

## III. DuPont Promised Not To Dispose Of Toxins Like C-8 In Its Dry Run Landfill.

In the early 1980s, DuPont approached the Tennants seeking to buy several hundred acres of the Tennants' property for the purposes of constructing a landfill near the base of Dry Run Creek in Wood County, West Virginia. (See Exhibit 14.) In response to initial resistance from the Tennants to the idea of selling any portion of their land for a landfill, DuPont promised the Tennants that no hazardous materials would ever be disposed of in the landfill. (See Exhibit 14.) After receiving DuPont's verbal and written assurances that no harmful chemicals would ever be disposed of in the proposed landfill and that the Tennants would be permitted to graze their

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[5] ®3M's registered trademark.

cattle along the adjacent Dry Run Creek,[6] the Tennants eventually agreed to sell a portion of their property to DuPont for construction of the "non-hazardous" landfill. DuPont received a permit to operate the Dry Run Landfill as an unlined, non-hazardous, solid waste landfill in 1982, and began actual landfilling operations at the Landfill in 1984. (See Exhibit 5.)

## IV. DuPont Has Dumped Thousands Of Tons Of C-8 Wastes Into The Dry Run Landfill.

Soon after DuPont began operating the Dry Run Landfill in 1984, DuPont received the results of internal sampling confirming that C-8 was leaching into groundwater beneath three old, unlined anaerobic digestion ponds at the Washington Works that DuPont previously had used for the disposal of thousands of tons of C-8-soaked sludges. (See Exhibits 9, 17, 20, and 31.) DuPont's internal sampling indicated that, not only was C-8 getting into the groundwater that DuPont used for the Washington Works' drinking water, but C-8 also was migrating through the groundwater under the Washington Works and into the Lubeck Public Service District's ("Lubeck PSD's") immediately-adjacent public drinking water wells. (See Exhibits 17, 18, 20, and 31.) Internal DuPont sampling confirmed C-8 in the Lubeck PSD community drinking water supply as high as 1.5 ppb in 1984, (see Exhibits 17, 18, and 20), increasing to as high as 1.9 ppb in 1987, (see Exhibits 19 and 20), and further increasing to as high as 2.2 ppb in 1988 (see Exhibits 27 and 28. See also Exhibit 33.) All of these levels exceed DuPont's own 1 ppb CEG for community drinking water. (See Exhibits 2-4, and 9.)

Upon receipt of those results, DuPont decided to try to remove the source of the C-8 in the public and company drinking water supplies by digging up and removing the sludges from Washington Works' three anaerobic digestion ponds and dumping the tons of C-8-contaminated sludge[7] into the Dry Run Landfill. (See Exhibits 20, 21, 22, 23, and 26.) After DuPont submitted data to the West Virginia Division for Environmental Protection ("WVDEP") asserting that the sludges were "non-hazardous" under RCRA, WVDEP granted DuPont permission to dispose of approximately 7,100 tons of the sludge in the unlined Dry Run Landfill. (See Exhibits 21, 23, and 25.) DuPont completed the sludge disposal in 1988. (See Exhibit 6.)

Rather than abate the presence of DuPont's C-8 in the public drinking water supply, DuPont simply purchased the Lubeck PSD well property and the wells were moved approximately two miles further down-gradient from the Washington Works. (See Exhibits 9, 30, 31, and 97.) DuPont then notified its employees to immediately cease all sampling of the

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[6] DuPont even agreed to lease back to the Tennants for cattle pasture significant portions of the landfill property along the Dry Run Creek. Those leases remained in effect until the Tennants began complaining about the Dry Run Landfill to USEPA. (See Exhibit 5.)

[7] DuPont confirmed C-8 levels as high as 610 ppm in the sludge taken from the three ponds. (See Exhibit 9.)

former Lubeck PSD wells and to destroy all previously-drawn, unanalyzed Lubeck PSD well samples. (See Exhibit 29.)

Also in 1989, WVDEP informed DuPont that new landfill regulations had gone into effect in the State of West Virginia requiring existing, unlined landfills to be upgraded with more rigorous waste containment mechanisms, including liners and more extensive groundwater monitoring well systems. (See Exhibit 32.) In response, DuPont installed a series of new groundwater monitoring wells at its Dry Run Landfill and at its nearby, unlined Letart Landfill in Mason County, West Virginia where DuPont had been disposing of most of its Teflon® and other C-8 wastes from the Washington Works as non-hazardous solid waste since the 1960s. (See Exhibit 121.) After DuPont's initial groundwater sampling at the Letart Landfill confirmed the presence of C-8 at 0.7 ppm, (see Exhibit 9), DuPont began investigating whether any C-8 also was leaching out of the waste at the Dry Run Landfill. (See Exhibit 6.) By April of 1990, DuPont had confirmed that C-8 was, in fact, leaching from the Dry Run Landfill and discharging directly into the Dry Run Creek at levels as high as 1.6 ppm – more than 100 times DuPont's own internal standard for drinking water of 1 ppb. (See Exhibits 9, 35, 37, 41, and 136.) Soon thereafter, DuPont abandoned its efforts to seek a new permit for the Letart Landfill, and notified WVDEP that it had decided, instead, to simply close that landfill "for economic reasons." (See Exhibits 74 and 121.)[8] DuPont proceeded, however, with its efforts to get a revised permit for the Dry Run Landfill that would allow DuPont to continue to operate the landfill without having to install a liner. (See Exhibit 50.)

After confirming elevated C-8 levels in the water at Dry Run, DuPont began investigating how to get rid of the approximately 7,100 tons of C-8-contaminated sludge that it dumped into the landfill in 1988, which DuPont assumed was a source of the C-8 being detected in Dry Run Creek. (See Exhibits 7, 8 and 38.) Although DuPont initially notified WVDEP that it would remove the C-8-contaminated sludges from the Dry Run Landfill and dispose of the material at its Letart Landfill, (see Exhibits 36 and 39), DuPont simply moved the sludges to another location within the Dry Run Landfill in 1991. (See Exhibits 5 and 6.)

By the summer of 1993, WVDEP inspectors noticed increasingly excessive amounts of sediment and discoloration building up in the leachate collection ponds at the Dry Run Landfill. (See Exhibit 44.) In response, DuPont, despite knowledge that the leachate contained high levels of C-8 and despite knowledge that the Tennants' cattle were drinking the water in Dry Run Creek, ordered the drains on its leachate collection ponds opened for more than two weeks (after monthly sampling had been completed (see Exhibit 45)), so that the leachate could flow out of

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[8] After DuPont finally shut down its unlined, "non-hazardous" Letart Landfill in 1996, it began paying to dispose of its C-8-contaminated wastes at a RCRA hazardous waste facility in Alabama. (See Exhibit 121.)

the ponds and directly into the Dry Run Creek. (See Exhibits 46 and 86.)[9] Although WVDEP requested that DuPont submit acute toxicity sampling results for the leachate being discharged out of the sedimentation ponds, (see Exhibit 44), DuPont successfully avoided taking any such samples until four months after the original leachate had drained into the creek. (See Exhibit 48.) The acute toxicity results that DuPont did eventually submit to WVDEP confirmed a 15% mortality, even among neonates exposed to the water four months later. (See id.) In the meantime, dozens of the Tennants' cattle were dying along the Dry Run Creek bed and the Tennants and their family and friends were exposed to C-8.

By the fall of 1994, DuPont had adopted a corporate plan to start routinely dumping C-8 wastes into the Dry Run Landfill, in anticipation of the upcoming closure of its Letart Landfill. (See Exhibit 130.) Thus, in furtherance of this corporate plan, but without any authorization or approval of any kind from WVDEP, DuPont began dumping its C-8-contaminated biocake wastes into the Dry Run Landfill that Fall. (See Exhibits 5 and 86.) According to DuPont's own analyses, the biocake contained 930 ppb of C-8. (See Exhibits 6, 58, 85, and 87.) By the spring of 1995, discolored, foul-smelling water was observed being discharged out of the Dry Run Landfill sedimentation ponds into Dry Run Creek, with almost knee-high suds and foam present along the Dry Run Creek bed, which DuPont assumed contained C-8. (See Exhibits 5, 53, 54, 56, 88 and 91.) At the same time, even more of the Tennants' cattle were dying.

In response to repeated pleas from the Tennants that WVDEP force DuPont to take action to address the black odorous water and foam being discharged into the Dry Run Creek where their cattle were drinking and dying, WVDEP notified DuPont that it would need to start taking steps to address its improper discharges into Dry Run Creek and to upgrade the Dry Run Landfill. (See Exhibits 5 and 57.) After it became evident that little progress was being made by DuPont in response to WVDEP's requests,[10] the Tennants notified USEPA of the problem and provided copies of videotapes showing the discolored foaming water and dead animals along the Dry Run Creek bed. (See Exhibit 61.) Around the same time, the West Virginia Department of Natural Resources contacted DuPont in response to recent reports of numerous deer killed or dying in the area of the Dry Run Creek. (See Exhibit 59.) Despite such complaints, DuPont did nothing to disclose to the Tennants that C-8 was in the Dry Run Creek, nor did DuPont suggest in any way to the Tennants that their cattle should not be drinking the water in the Creek. (See Exhibit 74.) Instead, DuPont kept silent on the C-8 issue and took the position with the public and the regulatory agencies that all of the problems with the creek were simply the result of some high

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[9] DuPont also ordered the landfill drain opened in 1989 and again in 1995 so that the contents of the sedimentation pond could flow directly into Dry Run Creek, without any apparent notice to or permission from WVDEP. (See Exhibits 34 and 55.)

[10] Discolored, foaming water continued in Dry Run Creek throughout the remainder of 1995, 1996, 1997, 1998, and into 1999.) (See Exhibits 62, 63, 89, and 92.)

iron sulfide levels that had been fully addressed and completely resolved. (See Exhibits 5, 74, and 78.)[11]

In October of 1996, USEPA contacted DuPont and informed the company that it would be initiating an inspection of the Dry Run Landfill in response to the recent reports of hundreds of dead cattle and deer in the area of the Dry Run Creek. (See Exhibits 5, 64, and 68.) On the exact same day that DuPont learned of USEPA's pending inspection, Eli McCoy (with WVDEP's Water Division) forwarded to DuPont a draft complaint to aid DuPont in diffusing any potential enforcement action by USEPA relating to the discharge problems at the Dry Run Landfill. (See Exhibits 5 and 65.) Within a matter of weeks, DuPont completed its negotiations with the State and entered a consent decree to bar further governmental enforcement action in exchange for DuPont's payment to WVDEP of a $200,000 penalty. (See Exhibits 5, 67, and 69.) Soon thereafter Mr. McCoy left WVDEP and began working for the same DuPont consultant that would assist DuPont in complying with the consent decree - Potesta & Associates. (See Exhibit 73.)

As part of the December 1996 settlement with WVDEP, DuPont finally agreed to begin implementing upgrades to the Dry Run Landfill, such as installation of the type of liner that was required under the State's landfill regulations since 1988, and construction of a leachate collection system. (See Exhibits 66 and 69.) DuPont also finally agreed to cease the disposal of its biocake wastes at the Dry Run Landfill. (See id.) Thus, by the time USEPA actually commenced its ecological risk assessment activities in the Dry Run Landfill area in 1997, DuPont allegedly had stopped disposing of its C-8-contaminated biocake sludge at the Dry Run Landfill and had allegedly begun collecting C-8-contaminated leachate from the Landfill for transport to the Washington Works for treatment and discharge directly into the Ohio River. (See Exhibits 5, 70, and 72.)

By the end of 1997, USEPA released to DuPont a draft of its Ecological Risk Assessment Report for the Dry Run Landfill. (See Exhibit 75.) USEPA's report indicated that, although adverse impacts were clearly evident among numerous animals, plants, and other wildlife in the area of the Dry Run Creek, USEPA had not been able to identify any particular known, regulated chemical as the clear cause of the observed problems. (See id. at 52) USEPA, therefore, recommended further assessment and identification of numerous "tentatively identified compounds" that had been detected in various environmental media in the area of Dry Run Creek that might be contributing to the problems. (See id.) In response to the suggestion of further governmental investigation, DuPont immediately requested and USEPA agreed to discuss a "collaborative" effort to further investigate conditions in the area of Dry Run Creek. (See

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[11] DuPont's practices with respect to making public the company's knowledge of the toxicity of its products was addressed in detail in In re E.I. duPont de Nemours & Co., 918 F. Supp. 1524 (M.D. Ga. 1995) (court imposed over $100 million in sanctions against DuPont).

Exhibits 79 and 83.) Part of that collaborative effort included DuPont's agreement that it would disclose more fully the precise identities of each of the various types of chemicals it had dumped into the Dry Run Landfill that DuPont had not previously identified for USEPA. (See Exhibit 83.) Although DuPont had been monitoring C-8 levels in Dry Run Creek for years and had confirmed C-8 in the water each time, DuPont eventually identified C-8 as being only "possibly" present in the Dry Run Landfill in a list of dozens of chemicals that it sent to USEPA in late 1998 - almost a year after the USEPA had completed its draft Risk Assessment Report. (See Exhibit 83.)[12]

Because of USEPA's persistent concerns that something in the Dry Run Creek was killing hundreds of head of the Tennants' cattle, (see Exhibit 78),[13] DuPont also agreed to jointly fund an investigation into the health of the Tennants' cattle. Specifically, DuPont agreed in the Spring of 1999 to create a "Cattle Team" to "independently" investigate such issues. By that time, however, less than a few dozen of the Tennants' cattle were even still alive. The Cattle Team was comprised of three veterinarians selected by DuPont, including Greg Sykes, a DuPont employee who had been involved in DuPont's internal investigations into the effects of C-8 on animals for many years, (see Exhibit 24), and three veterinarians selected by USEPA. (See Exhibit 95.) Despite DuPont's knowledge that C-8 was a toxic animal carcinogen (as reenforced to DuPont by the recent C-8 monkey study results (see, e.g., Exhibits 87 and 166)), that the Tennants' cows were drinking out of Dry Run Creek, the information currently available to the Tennants does not indicate that anyone from DuPont ever disclosed such facts to the other members of the Cattle Team during the course of the Cattle Team's investigation. (See Exhibit 93.) Consequently, there is no evidence that the Cattle Team even considered the potential impact of C-8 on the Tennants' cattle, despite the release of the C-8 monkey study results to DuPont well before the final Cattle Team Report was released in December of 1999. (See Exhibit 109.) Again, DuPont kept completely silent on the C-8 issue and sat back and let the Cattle Team "independently" investigate the health of the Tennants' cattle, even though the USEPA-appointed Cattle Team members would never have any reason even to think to look at C-8.

Over the last several years, while DuPont was working with USEPA on their "collaborative" effort to address environmental problems in the area of Dry Run Creek, several of the Tennants have been in and out of the hospital suffering from respiratory problems, chemical

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12 At around the same time, DuPont, again, ordered the Dry Run Landfill sedimentation pond drain opened, so that the foul-smelling contents could discharge directly into the Dry Run Creek where the few remaining head of the Tennants' "[c]attle were wallowing in the stream just beyond the fence." (See Exhibits 81 and 82.)

13 At least two other local residents, including at least one current DuPont employee, also have complained that their cattle appear to have been harmed by something in Dry Run Creek. (See Exhibits 54 and 117.)

burns, and other health problems after having been exposed to fugitive air emissions and liquid discharge from DuPont's Dry Run Landfill. Moreover, despite installation several years ago of a leachate collection system that was supposed to prevent contaminants from the Dry Run Landfill from getting into the Dry Run Creek, DuPont's own monitoring reports confirm that C-8 is still getting into the Dry Run Creek with results as high as 87 ppb in the creek, as recently as the Summer of 1999, and as high as 27.6 ppb during the Fall of 2000 – readings more than twenty times DuPont's CEG for C-8 in water. (See Exhibit 134.) Thus, DuPont's own monitoring reports confirm that, despite installation of a purported leachate collection system, there is a continuing, ongoing discharge of high levels of C-8 from the Dry Run Landfill into Dry Run Creek.

## V. DuPont Has Known That Its C-8 Wastes Have Leached Into Drinking Water.

In addition to DuPont's failure to disclose to the Tennants or the USEPA-appointed Cattle Team members the full extent of its knowledge regarding the nature, extent, and likely effects upon wildlife of the C-8 it has been releasing and continues to release into Dry Run Creek, the information currently available to the Tennants indicates that DuPont also has not fully disclosed to USEPA, WVDEP, local governmental entities, its neighbors, or the public its knowledge of the full extent of the impact of its C-8 wastes on local drinking water.

As part of its efforts to complete its RCRA Facility Investigation Report ("RFI Report") for the Washington Works, DuPont was required to investigate whether any of its former solid waste management units, including the three anaerobic digestion ponds that were closed in 1988, are contributing to any release of wastes onto neighboring properties and whether any wastes are exposing any persons to unreasonable health risks. (See Exhibits 98 and 99.) In connection with its RFI efforts, DuPont took more samples of the groundwater under the Washington Works site that it uses for drinking water at the Plant. (See Exhibits 10, 11, 76, and 99.) DuPont also arranged for the sampling of groundwater under the neighboring GE Plastics Plant that GE uses for its own plant drinking water. (See Exhibits 10 and 11.) Sampling confirmed C-8 in the Washington Works' drinking water as high as 3.3 ppb[14] and as high as 0.71 ppb in the neighboring GE Plastics drinking water supply. (See Exhibits 10, 11, 43, 76, 96, 99, 102, 104,

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[14] It is noted that, although DuPont had been sampling three drinking water wells at the Washington Works (wells 331, 332, and 336), when it came time to actually report the results to USEPA in its RFI Report, Dupont was careful to sample only the drinking water well that had previously yielded C-8 results less than 1 ppb (well 336), and conveniently did not even sample the wells that traditionally had yielded the higher C-8 results, nor did DuPont report these higher results in its RFI Report. (See Exhibits 76, 96, 99). Yet, when even the well with the C-8 readings traditionally below 1 ppb yielded a result of 1.9 ppb, DuPont fabricated a new 3.0 ppb "screening level" for C-8 to avoid having to reference any drinking water results exceeding DuPont's own 1 ppb CEG in its own plant drinking water. (See Exhibit 99).

106, 110 and 129.) DuPont even found C-8 as high as 0.8 ppb in the new Lubeck PSD drinking water wells, which are now located approximately two miles farther away from the Washington Works site. (See Exhibits 10-11, 40, and 41.)[15] Recent sampling of the private drinking water wells on the Tennants' property down-gradient from the Dry Run Landfill also has now confirmed C-8 in those drinking water wells. (See Exhibit 131.) DuPont has even investigated what C-8 levels might be present at various cities along the Ohio River, based upon DuPont's on-going releases of C-8 into the River from the Washington Works facility. (See Exhibits 40, 100, and 118.)[16] Approximately 24,000 pounds of C-8 also is discharged directly into the air every year from the Washington Works Site, although it is not clear that C-8 is actually permitted for such air discharge by DuPont. (See Exhibits 101 and 118.)

Thus, it is evident that the residents living in at least the area near DuPont's Washington Works facility, Letart Landfill, and Dry Run Landfill (the "DuPont Sites") may have been and may continue to be exposed to DuPont's C-8 through DuPont's on-going and continuous releases of C-8 into the air, land, and water at and/or around those Sites, (see Exhibit 80), including direct ingestion of C-8 in the C-8-contaminated drinking water extracted from wells at the Washington Works Plant, the neighboring GE Plastics Plant, the Lubeck PSD well fields, and private residential and agricultural properties near DuPont's Sites.[17] Local wildlife and the environment may be similarly exposed. Despite DuPont's knowledge for years of the nature, extent, and effect of these C-8 releases on human health and the environment, including the

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15 Sampling results from 1991 confirmed C-8 at 2.4 ppb in the new Lubeck wells with C-8 levels as high as 3.9 ppb in the tap water of several local, Lubeck-area homes. (See Exhibit 128.) Sampling in August of 2000 confirmed C-8 still present in the new Lubeck PSD wells at levels as high as 0.59 ppb. (See Exhibit 119.)

16 DuPont has been evaluating the levels of C-8 in the Ohio River, which is a source of drinking water for numerous communities, since at least 1982. (See Exhibit 15.)

17 In August of 2000, after the Tennants had made it known to DuPont that they had become aware of the C-8 in the Lubeck PSD wells, DuPont drafted a letter for the Lubeck PSD to send to its water customers to "disclose" the existence of the C-8. (See Exhibit 124.) In that letter, however, DuPont was very careful to refer only to the current C-8 levels in the current Lubeck PSD wells, and avoided any mention whatsoever of the earlier C-8 readings that were substantially above DuPont's 1 ppb CEG. (See id.) DuPont again was careful to avoid any public disclosure of its knowledge of earlier C-8 drinking water results that were well-above DuPont's 1 ppb CEG in recent statements provided to local Parkersburg newspapers, even though DuPont had received in November a draft of this letter referencing the higher C-8 levels. (See Exhibit 135.)

bioaccumulative/biopersistent nature of the material,[18] it appears that DuPont has allowed and continues to allow these releases to occur unabated for fear of not being able to continue to make its Teflon® products, if it cannot use C-8. This situation is particularly disturbing, given that DuPont apparently has known of ways to remediate C-8-laden soils since the early 1990s but because of the expense, chose to do nothing "pending further actions that may be dictated by the EPA for remediation of the Washington Works site." (See Exhibit 122.) Even more disturbing is the fact that DuPont has known for years that C-8 levels in the Washington Works and old Lubeck PSD drinking water wells far exceeded its own 1 ppb CEG but has done absolutely nothing in response. DuPont has chosen, instead, to focus either on current, somewhat lower C-8 levels, or to simply fabricate a totally new drinking water "screening level" of 3 ppb for the Washington Works Plant when faced with having to disclose to USEPA in its RFI report for the Washington Works the existence of C-8 in the Plant's drinking water at levels well above 1 ppb. (See Exhibits 99 and 124.)

## VI. DuPont Should Be Ordered To Remediate Its C-8 Releases And To Immediately Shut Down Its Manufacturing Processes Involving C-8 Until Adequate Demonstrations Are Made That There Is No Unreasonable Risk To Health Or The Environment.

Over the years, DuPont has successfully avoided fully disclosing the nature and extent of the C-8 problem at its Dry Run Landfill by characterizing C-8 as an unregulated "non-hazardous" waste and/or substance under applicable law. Consequently, when the Federal and State agencies have asked questions about the nature and quantity of toxic wastes handled by DuPont at the Dry Run Landfill, DuPont has omitted any comprehensive discussion of C-8 on the grounds that it is not a "hazardous waste," "hazardous substance," or otherwise listed or regulated waste under current laws. DuPont shrewdly avoided any permit limits on its C-8 emissions and/or dumping at its Washington Works facility and Dry Run Landfill through similar corporate strategies. Thus, although DuPont has known for years that C-8 is an animal carcinogen and bioaccumulative/biopersistent substance, it has continued to knowingly dump thousands of tons of the waste into the environment at unlined, uncontrolled landfills and has allowed the waste to be disposed directly into the air, Ohio River, and local drinking water supplies, arguing that there has not been any improper disposal and/or release of any regulated material.

In addition, DuPont has been careful to refer to the chemical in conflicting, inconsistent ways in its filings with regulatory agencies - sometimes calling it "C-8," sometimes calling it "FC-143," sometimes calling it "PFOA," sometimes calling it "APFO," and sometimes calling it by its full chemical name - "ammonium perfluorooctanoate" - thereby making it difficult for the agencies to understand how all the information interrelates. As confirmed by USEPA's recent

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[18] DuPont's own employees even raised concerns about Teflon® customer exposure to C-8 as early as 1983. (See Exhibits 16 and 52.)

proposal to begin regulating 3M's previously-unregulated perfluorinated chemicals, DuPont's past corporate strategy for diverting regulatory attention away from C-8 should stop now.

Based upon the foregoing facts, the Tennants hereby respectfully request that your agencies intervene in the Tennants' pending Federal Court litigation and order the immediate investigation, assessment, containment, removal, and remediation of DuPont's on-going C-8 releases into the environment by virtue of the authority granted to your agencies under at least the following laws and their implementing regulations:

- The Toxic Substances Control Act, as amended, 15 U.S.C. §§ 2601-2692;
- The Federal Clean Water Act, as amended, 33 U.S.C. §§ 1251-1387;
- The Safe Drinking Water Act, as amended, 42 U.S.C. §§ 300f-300j-26;
- The Federal Clean Air Act, as amended, 42 U.S.C. §§ 7401-7671q;
- The Resource Conservation and Recovery Act, as amended, 42 U.S.C. §§ 6901-6992k;
- The Comprehensive Environmental Response, Compensation and Liability Act, as amended, 42 U.S.C. §§ 9601-9675;
- The West Virginia Air Pollution Control Act, W.Va. Code §§ 22-5-1 through 22-5-18;.
- The West Virginia Water Pollution Control Act, W.Va. Code §§ 22-11-1 through 22-11-28;
- The West Virginia Groundwater Protection Act, W.Va. Code §§ 22-12-1 through 22-12-14;
- The West Virginia Natural Streams Preservation Act, W.Va. Code §§ 22-13-1 through 22-13-15;
- The West Virginia Solid Waste Management Act, W.Va. Code §§ 22-15-1 through 22-15-21;
- The West Virginia Hazardous Waste Management Act, W.Va. Code §§ 22-18-1 through 22-18-25; and

- The West Virginia Hazardous Waste Emergency Response Fund Laws, W.Va. Code §§ 22-19-1 through 22-19-6.

The Tennants also request that your agencies exercise their respective authority under the referenced laws to order DuPont to **immediately** cease and desist its C-8 releases into the environment, as addressed in this letter and to provide for immediate, appropriate medical care/testing/evaluation of the Tennants. The Tennants further request that DuPont's permit to operate the Dry Run Landfill be **immediately** revoked until adequate scientific demonstrations are made to prove that the C-8 releases have been abated, will not recur, and pose no unreasonable risk to human or animal health or the environment.

With respect to minimizing harm to the public health and the environment from future C-8 releases, the Tennants hereby specifically request that USEPA exercise its authority under the Toxic Substances Control Act to order DuPont to immediately cease all manufacturing activities using C-8, including DuPont's Teflon® manufacturing operations, until DuPont either confirms that it has stopped its usage of C-8 entirely or has made adequate scientific demonstrations to prove that its continued usage of C-8 (whether from 3M or any other source) does not pose an unreasonable risk of injury to health or the environment. In the meantime, the Tennants request that your agencies take these steps necessary to regulate C-8 emissions/releases to the environment. As mentioned above, the Tennants believe that such steps should include, at a minimum, including C-8 among the list of perfluorinated chemicals that USEPA proposed in October of this year to begin regulating under TSCA on the basis that the chemicals "may be hazardous to human health and the environment." (See Exhibit 123.)

## VII. The Tennants Intend To Bring Citizen Suit Claims Against DuPont Under The CWA, TSCA, And RCRA If Appropriate Action Is Not Taken Immediately To Abate And Remediate DuPont's C-8 Releases From Its Washington Works Facility.

As explained above, DuPont has been and continues to discharge C-8 from its Washington Works Facility in Wood County, West Virginia into the air, groundwater, and Ohio River. Moreover, the C-8 discharged by DuPont has been contaminating and continues to contaminate the land, air, and human and animal drinking water supplies.

### A. DuPont Is Violating The CWA.

Section 505(a)(1) of the Clean Water Act ("CWA") permits citizens to commence a civil action against "any person ... who is alleged to be in violation of (A) an effluent standard or limitation under this chapter." 33 U.S.C. §1365(a)(1). "Effluent standard or limitation" is defined under the CWA to include, among other things, "a permit or condition thereof issued under Section 1342 of this title," such as state-issued but federally-enforceable NPDES discharge permits. Id. at §1365(F). Based upon information currently-available to the Tennants, DuPont's NPDES permit for its Washington Works facility specifies that DuPont shall not discharge any

effluent in violation of applicable Water Quality Standards. (See, e.g., WV/NPDES Permit No. WV0001279, Conditions A.1 - A.10, C.12, and H.2). The West Virginia Water Quality Standards prohibit DuPont from discharging into surface or groundwaters any "materials in concentrations which are harmful, hazardous, or toxic to man, animal, or aquatic life." W. Va. Code St. R. tit. 46, §46-1-3.2 (2000). Based upon currently-available information, as described above, DuPont has been discharging and continues to discharge C-8 into surface and groundwaters in concentrations exceeding DuPont's own CEG for human drinking water and at concentrations that are otherwise harmful, hazardous, or toxic to man, animal, or aquatic life, constituting a continuing violation of the West Virginia Water Quality Standards, and thereby constituting a continuing violation of DuPont's NPDES permit terms and the CWA. See, e.g., 33 U.S.C. §§1311(a), 1342. Notice is, therefore, hereby provided that the Tennants, on behalf of themselves and a class of others similarly situated, intend to file suit against DuPont, pursuant to Section 505(a)(1) of the CWA, within sixty (60) days of this notice to obtain appropriate relief for the violations of the CWA referenced herein.

**B. DuPont Is Violating TSCA.**

Section 20(a)(1) of the Toxic Substances Control Act ("TSCA") permits citizens to commence a civil action against "any person . . . who is alleged to be in violation of [TSCA] or any rule promulgated under Sections 2603, 2604, or 2605 of [TSCA], or Subchapters II or IV of [TSCA]." 15 U.S.C. § 2619(a)(1). TSCA requires any "person who manufactures, processes, or distributes in commerce a chemical substance or mixture and who obtains information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment" to "immediately" inform USEPA of "such information, unless such person has actual knowledge that" USEPA has been adequately informed of such information. Id. at § 2607(e). TSCA also requires each person who manufactures or processes a chemical substance to comply with the regulations adopted by USEPA under TSCA governing the reporting to USEPA of certain research and adverse health effects information relating to such chemical substances. See id. at § 2607(a), (c), (d); 40 C.F.R. Parts 716 and 717. Failure to comply with such TSCA requirements constitutes a violation of TSCA. See 15 U.S.C. § 2614. As indicated above, the information currently available to the Tennants indicates that DuPont has not reported to USEPA all information within DuPont's possession regarding C-8 that is required to be reported to USEPA under Section 8(a), (c), (d), and (e) of TSCA, 15 U.S.C. § 2607 (a), (c), (d), and (e), such as the results of the C-8 monkey studies and the Tennants' allegations of adverse health effects among themselves, their cattle, and area wildlife arising from exposure to DuPont's C-8. Notice is, therefore, hereby provided that the Tennants, on behalf of themselves and a class of others similarly situated, intend to file suit against DuPont, pursuant to Section 20(a)(1) of TSCA, within sixty (60) days of this notice to obtain appropriate relief for the violations of TSCA referenced herein.

**C. DuPont's C-8 Releases From Its Washington Works Facility May Present An Imminent And Substantial Endangerment To Health Or The Environment Under RCRA.**

Section 7002(a)(1)(B) of the Resource Conservation and Recovery Act ("RCRA") permits citizens to commence a civil action against:

> [a]ny person ..., including any past or present generator, past or present transporter, or past or present owner or operator of a treatment, storage, or disposal facility, who has contributed or who is contributing to the past or present handling, storage, treatment, transportation, or disposal of any solid or hazardous waste which may present an imminent and substantial endangerment to health or the environment.

42 U.S.C. § 6972(a)(1)(B). As discussed above, DuPont's past and on-going disposal of C-8 into soil, water, and air from DuPont's Washington Works Facility has resulted in C-8 in soil, water, and air at and/or around the Washington Works Facility in amounts, levels, and/or concentrations which, based upon the currently-available information, may present an imminent and substantial endangerment to health or the environment. Notice is, therefore, hereby provided that the Tennants , on behalf of themselves and a class of others similarly situated, intend to file suit against DuPont, pursuant to Section 7002(a)(1)(B) or RCRA, within ninety (90) days of this notice to obtain appropriate relief for the imminent and substantial endangerment referenced herein.

Please confirm as soon as possible how your respective agencies plan to address our request for your involvement in this important public health and environmental matter. In that regard, please let us know if you will intervene in the Tennants' Federal Court proceedings or if

you would like to review any of the additional backup documentation maintained here at our Cincinnati offices. We would be happy to meet with you at your offices to discuss this matter in more detail. Thank you.

On behalf of the Tennants,

Robert A. Bilott

RAB/mdm
Enclosures
cc: Larry A. Winter, Esq. (West Virginia Counsel for the Tennants) (w/o encls.)
Paula Durst Gillis, Esq. (Counsel for DuPont) (w/ encls.)
(by **CERTIFIED MAIL NO: 70000600002406963531, RETURN RECEIPT REQUESTED & REGISTERED MAIL NO: R410009299, RETURN RECEIPT REQUESTED**)
Registered Agent for E.I. duPont de Nemours & Co., Inc. (w/o encls.)
(CT Corporation System, 707 Virginia Street, East, Charleston, WV 25301
by **CERTIFIED MAIL NO: 70000600002406963500**)

H:\TENNANT\RequestLtr.wpd

05573515 C8 Solution Revised 6-MAY-1999 Printed 6-MAY-1999

## CHEMICAL PRODUCT/COMPANY IDENTIFICATION

**Tradenames and Synonyms**

20% Ammonium Perfluorooctanoate Solution

**Company Identification**

MANUFACTURER/DISTRIBUTOR
DuPont
1007 Market Street
Wilmington, DE 19898

PHONE NUMBERS
Product Information : 1-800-441-7515
Transport Emergency : CHEMTREC 1-800-424-9300
Medical Emergency : 1-800-441-3637

## COMPOSITION/INFORMATION ON INGREDIENTS

**Components**

| Material | CAS Number | % |
|---|---|---|
| Ammonium Perfluorooctanoate | 3825-26-1 | 20 |
| Water | 7732-18-5 | 80 |

## HAZARDS IDENTIFICATION

**Potential Health Effects**

Skin contact may cause skin irritation with discomfort or rash. Evidence suggests that skin permeation can occur in amounts capable of producing the effects of systemic toxicity.

Eye contact may cause eye irritation with discomfort, tearing, or blurring of vision.

Inhalation may cause irritation of the upper respiratory passages, with coughing and discomfort.

Ingestion may cause gastrointestinal tract irritation; abnormal liver function as detected by laboratory tests; or abnormal blood forming system function with anemia.

This compound is absorbed by the body and may be detected in the blood stream following ingestion, inhalation or skin contact. Animal and human experience indicate that this compound has a long half-life in the blood, and may be

OTG000352

(HAZARDS IDENTIFICATION - Continued)

detected years after exposure.

Individuals with preexisting diseases of the liver, or bone marrow may have increased susceptibility to the toxicity of excessive exposures.

**Carcinogenicity Information**

None of the components present in this material at concentrations equal to or greater than 0.1% are listed by IARC, NTP, OSHA or ACGIH as a carcinogen.

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**FIRST AID MEASURES**

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**First Aid**

INHALATION

If inhaled, remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Call a physician.

SKIN CONTACT

In case of contact, immediately flush skin with plenty of water for at least 15 minutes while removing contaminated clothing and shoes. Call a physician. Wash contaminated clothing before reuse.

EYE CONTACT

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes. Call a physician.

INGESTION

If swallowed, immediately give 2 glasses of water and induce vomiting. Never give anything by mouth to an unconscious person. Call a physician.

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**FIRE FIGHTING MEASURES**

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**Flammable Properties**

Non-flammable.

Hazardous decomposition products including carbon dioxide, carbon monoxide, hydrogen fluoride, toxic gases or particles may be formed during combustion. These products may cause severe eye, nose, and throat irritation or toxic effects.

(FIRE FIGHTING MEASURES - Continued)

**Extinguishing Media**

Water Spray, Foam, Dry Chemical, CO2.

**Fire Fighting Instructions**

Wear self-contained breathing apparatus (SCBA) and full protective equipment.

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**ACCIDENTAL RELEASE MEASURES**

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**Safeguards (Personnel)**

NOTE: Review FIRE FIGHTING MEASURES and HANDLING (PERSONNEL) sections before proceeding with clean-up. Use appropriate PERSONAL PROTECTIVE EQUIPMENT during clean-up.

Evacuate personnel, thoroughly ventilate area, use self-contained breathing apparatus.

**Initial Containment**

Dike spill.

**Spill Clean Up**

Soak up with sawdust, sand, oil dry or other absorbent material.

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**HANDLING AND STORAGE**

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**Handling (Personnel)**

Avoid inhalation. Avoid contact with eyes, skin or clothing. Wash thoroughly after handling. Wash clothing after use. Avoid circumstances that produce respirable particles unless suitable ventilation and respirator are used.

**Handling (Physical Aspects)**

Keep container tightly closed.

**Storage**

Store in a well ventilated place.

## EXPOSURE CONTROLS/PERSONAL PROTECTION

### Engineering Controls

Use only with adequate ventilation.
Vent heated extruder or dryer fumes outside work area. Do not aerosolize. In spray applications, use airless type pressure spray equipment at less than 60 psi, and exhaust ducts, drip pans, or other design features to minimize worker exposure to mists and overspray.

### Personal Protective Equipment

EYE/FACE PROTECTION

Wear safety glasses or coverall chemical splash goggles.

RESPIRATORS

Wear NIOSH approved respiratory protection, as appropriate.

PROTECTIVE CLOTHING

Where there is potential for skin contact have available and wear as appropriate impervious gloves, apron, pants, and jacket.

### Exposure Guidelines

**Applicable Exposure Limits**
Ammonium Perfluorooctanoate

| | | |
|---|---|---|
| PEL | (OSHA) | : None Established |
| TLV | (ACGIH) | : 0.01 mg/m3, 8 Hr. TWA, Skin, A3 |
| AEL * | (DuPont) | : 0.01 mg/m3, 8 Hr. TWA, Skin |

* AEL is DuPont's Acceptable Exposure Limit. Where governmentally imposed occupational exposure limits which are lower than the AEL are in effect, such limits shall take precedence.

## PHYSICAL AND CHEMICAL PROPERTIES

### Physical Data

| | |
|---|---|
| Boiling Point | : 100 C (212 F) |
| Freezing Point | : 0 C (32 F) |
| Form | : Liquid. |

OTG000355

## STABILITY AND REACTIVITY

**Chemical Stability**

Stable at normal temperatures and storage conditions.

**Incompatibility with Other Materials**

None reasonably foreseeable.

**Decomposition**

Hazardous decomposition products including carbon dioxide, carbon monoxide, hydrogen fluoride, toxic gases or particles may be formed during combustion. These products may cause severe eye, nose, and throat irritation or toxic effects.

**Polymerization**

Polymerization will not occur.

## TOXICOLOGICAL INFORMATION

**Animal Data**

Ammonium Perfluorooctanoate:

| | |
|---|---|
| Inhalation 4 hour LC50: | 980 mg/m3 in rats |
| Skin absorption LD50: | 4278 mg/kg in rabbits |
| Oral LD50: | 470 mg/kg in rats |

> for 100%

Ammonium Perfluorooctanoate is a skin and eye irritant, but is untested for animal sensitization. Effects of a single inhalation exposure include eye and nasal irritation, respiratory difficulty and liver enlargement. A single dermal exposure produced nonspecific effects such as weight loss and irritation, diarrhea, lethargy, labored breathing and cyanosis. A single ingestion exposure resulted in weight loss, gastrointestinal irritation and enlarged liver. Repeated exposures produced liver, kidney, pancreas and testes changes; anemia and cyanosis. Tests in male rats demonstrate tumorigenic activity based on an increased incidence of benign testicular, pancreatic, and liver tumors. Tests in animals demonstrate no developmental toxicity. Tests in animals for reproductive effects have not been performed. This compound does not produce genetic damage in bacterial cell cultures but has not been tested in animals.

OTG000356

## ECOLOGICAL INFORMATION

**Ecotoxicological Information**

Ammonium Perfluorooctanoate:
48 hour LC50, fathead minnows: 1,500 mg/L.

## DISPOSAL CONSIDERATIONS

**Waste Disposal**

Treatment, storage, transportation, and disposal must be in accordance with applicable Federal, State/Provincial, and Local regulations.

## REGULATORY INFORMATION

**U.S. Federal Regulations**

TSCA Inventory Status : Listed.

TITLE III HAZARD CLASSIFICATIONS SECTIONS 311, 312

Acute : Yes
Chronic : Yes
Fire : No
Reactivity : No
Pressure : No

## OTHER INFORMATION

**NFPA, NPCA-HMIS**

NPCA-HMIS Rating
Health : 3
Flammability : 0
Reactivity : 0

Personal Protection rating to be supplied by user depending on use conditions.

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The data in this Material Safety Data Sheet relates only to the specific material designated herein and does not relate to use in combination with any other material or in any process.

Responsibility for MSDS : J. S. Coates
DuPont Specialty Chemicals
Address : Chambers Works
Telephone : 609-540-3251

OTG000357

(Continued)

End of MSDS

OTG000358

EID087269

ATTACHMENT 2

is TSCA CBI.
DCN 6301000014
No sanitized version
was provided.

**Anthony J Playtis**
08/04/2000 09:04 AM

To: H David Ramsey/AE/DuPont@DuPont
cc:
Subject: C-8 Exposure Limits

Here's the information on C-8, aka ammonium perfluorooctanoate.

Du Pont Acceptable Exposure Limit (AEL)

0.01 mg/m3, 8-hr TWA (skin); "Not likely to be a human carcinogen or developmental toxin"

Du Pont Community Exposure Guidelines (CEG)

Airborne 0.0003 mg/m3
Drinking Water 1 ug/L

ACGIH Threshold Limit Value (TLV)

0.01 mg/m3 TWA (skin, A3); The A3 notation means the following:

"*Confirmed Animal Carcinogen with Unknown Relevance to Humans:* The agent is carcinogenic in animals at a relatively high dose, by route(s) of administration, at site(s), of histologic type(s), or by mechanism(s) that may not be relevant to worker exposure. Available epidemiologic studies do not confirm an increased risk of cancer in exposed humans. Available evidence does not suggest that the agent is likely to cause cancer in humans except under uncommon or unlikely routes or levels of exposure."

OSHA Permissible Exposure Limit (PEL)

None established.

EID078904

# C-8

## Control Levels

| | | |
|---|---|---|
| TLV | - | 0.01 MG/M$^3$, Liver Effects |
| AEL | - | 0.01 MG/M$^3$,<br>Liver Effects<br>Cancer Study Results<br>Biopersistence |
| $CEG_A$ | - | 0.0003 MG/M$^3$ |
| $CEG_W$ | - | 1 PPB |

0006832

EID078905

A Community Exposure Guideline ("CEG") is an exposure guideline established by Haskell Laboratory. The CEG assumes a 24-hour lifetime exposure by all, including the most sensitive individuals, in an exposed community population. Exposure above the CEG will not necessarily result in any adverse effects. Where data indicates that the CEG may be approached or exceeded, Haskell, the appropriate Business and Legal will evaluate, what action, if any should be taken. It is the Company's intent to maintain exposure below the CEG.

5-May-97
RJZ:ssb
13

# C-8 OVERVIEW - MAN

EID078906

- Major Biological Concern:

  Long-Term Effects in Man Related to

  Persistence in Human Blood

- 1/2 Life > 1 Year (3-4 Best Estimate)

006624

# C-8 OVERVIEW - MAN

- No Liver Function Changes in Workers

  3M

  DuPont

- Alleged Finding Relating Prostate Cancer in 3M Workplace with C-8

  - Tenuous, 3M Working on Details

EID078907

009635

# C-8 TOXICOLOGY REVIEW

EID078908

000035

## ACUTE

- Moderate Acute Toxicology -

LD50, Rat 540 mg/kg Oral

7,000 mg/kg Dermal

Rabbit 4,300 mg/kg Dermal

ALC Rat 4 hr 800 mg/m3

- Moderate Eye Irritant
- Non-Irritating to Skin

EID078909

# C-8 TOXICOLOGY OVERVIEW

## REPEATED

- Rodent, Oral — Striking Liver Enlargement

  30 ppm - Little Effect

  300 ppm - Pronounced Effect

- Monkey, Oral — 10 mg/kg - No Effects

  30 mg/kg - Not Tolerated

# C-8 TOXICOLOGY OVERVIEW

EID078910

009035

## REPEATED

- Rat, Dermal — 20 to 2,000 mg/kg

  Liver Damage

  Elevated Blood Levels

  Detected in Blood 47 Days Post-Exposure

EID078911

# TOXICOLOGY OVERVIEW

Inhalation - 1 mg/m3 - NOAEL

7 + 84 mg/m3 - Liver Damage (Reversible)

- Increased Blood Level

- Male vs Female Rat Clearance Differs

EID078912

# TOXICOLOGY OVERVIEW

**Lifetime Feeding - 30/300 ppm - Liver Effects**

**Leydig Cell Tumors at 300 ppm**

**Mechanism of Tumor Response Involves Chronic, Low-level Hormonal Change**

000619

REASONING BEHIND AEL

- INHALATION NOAEL 1 MG/M$^3$

  EFFECTS AT 8 MG/M$^3$ MILD, REVERSIBLE

- LOW LEVEL (?) ACTIVITY IN TESTES (TUMORS)

  30 PPM IN DIET = 1.5 MG/KG/DAY

  100% ABSORPTION OF INHALED DOSE, 70 KG WORKER = 10.5 MG/M$^3$

  THIS IS 1) EFFECT LEVEL
  2) HUMAN CLEARANCE FROM BODY SLOW

  SAFETY (UNCERTAINTY) FACTOR SHOULD BE LARGE (1000)

- RECOMMEND 0.01 MG/M$^3$ (10.5 ÷ 1000)



EID078913

RJZ007321

## REASONING BEHIND CEG (COMMUNITY EXPOSURE GUIDELINE)

AS FOR THE AEL (0.01 MG/M$^3$)

- ADDITIONAL FACTORS
  1) 24 HR EXPOSURE IN COMMUNITY VS 8 HR AT WORK
  2) DIVERSE POPULATION IN COMMUNITY
  3) BIO-PERSISTENCE IN MAN

- RECOMMEND 0.0003 MG/M$^3$ (33 FOLD REDUCTION)



RJZ007322

# HUMAN EXPOSURE

| Source | Preventative Method |
| --- | --- |
| Air | • Reduce Air Levels<br>• Breathing Protection |
| Skin Adsorption | • C-8 in Solution<br>• Chemical Gloves<br>• TYVEK® Clothing |
| Ingestion | • Reduce Concentration in Drinking Water |

EID078915

RJZ007323

DUPONT WASHINGTON WORKS
DRY RUN LANDFILL CHRONOLOGY -- 1995-1998

| | |
|---|---|
| August 1994 | Filter cake begins to be landfilled at Dry Run |
| January 1995 | TSS exceedence; start of high BOD/COD results; first notice of "black water" |
| February/early March 1995 | Pond Improvement Team organized |
| | Addition of antifoam |
| March/April 1995 | Water drainage improvements: diversion ditches and french drains installed |
| | Chemical treatment of fill area, pond, outfall begun (sulf control, hydrogen peroxide, antifoam) |
| March 23, 1995 | Cindy Musser inspection and recommendations |
| May 1995 | Beginning of enhanced monitoring and data collection on weekly basis |
| | Second sedimentation pond installed |
| May 3 | Cindy Musser inspection; progress noted |
| June 1995 | Lab testing confirmed filter cake contribution to high TOC problems |
| | 24-hour aeration begun in lower pond |
| | Algae control: copper sulfate treatment |
| | Lower pond visually improved |
| October 1995 | WTAP-TV news story |
| November 1995 | Musser/Britvec inspection (at our invitation); favorably impressed with progress |
| February 1996 | Zeto, Hill, Musser inspection to acquaint Zeto and Hill of Landfill operation |
| October 15, 1996 | DEP issued letter of intent to file civil action due to Washington Works' failure to meet new solid waste management standards |
| November 1996 | Final filtercake disposal at Landfill |
| December 31, 1996 | DEP issued Order and Washington Works agreed to settlement of all issues, including payment of $250,000 |

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| | |
|---|---|
| December 31, 1996, continued | DuPont and the West Virginia Division of Environmental Protection (WVDEP) entered a settlement agreement pertaining to the operation and maintenance of the Dry Run Landfill. As part of the settlement agreement, DuPont agreed to perform additional maintenance on existing water diversion ditches, transport landfill leachate to Washington Works for treatment until design improvements planned for the landfill are completed, and stop disposal of plant filtercake in the landfill until design improvements planned for the landfill are completed. |
| February 1997 | Autopsy done in Belpre, OH (6-month-old bull calf) refers to "emaciation, poor nutritional state; intestinal parasites." |
| March 1997 | Autopsies (2 older cows) done for EPA:<br><br>1. "The only significant toxicologic finding in these two animals was low copper concentration. Since this may indicate a dietary problem, analysis of feed is recommended."<br>2. "No significant pathologic findings."<br>3. "No toxic compounds were detected by GC/MS* in the liver, kidney, urine or fat." |
| June 1997 | EPA ecosystem study |
| July 1997 | Public hearing; included testimony of local veterinarian interpreting autopsy data and concluding no Landfill effect on the autopsied animals, based on info in the autopsy reports |
| December 1997 | Draft report of EPA ecosystem study |

Doc 60531

# DRY RUN LANDFILL EVENTS TIMELINE

| | |
|---|---|
| * ACQUIRED DRY RUN PROPERTY | 1982-1983 |
| * NUMEROUS LEASES<br>TENNANT<br>COLLINS<br>KAUFMAN | 1983-1984 |
| * BEGAN LANDFILL OPERATION | 1984 |
| * DISPOSAL OF TFE POND SEDIMENT | 1989 |
| * RELOCATION OF TFE POND MATERIAL (SUPERNATE) TO LINED CELL | 10/25/91 |
| * DISPOSAL OF FLUDGE OF DRY RUN STARTED (SUMMER) | 93 OR 94 |
| * STATE INSPECTION | 05/16/94 |
| * FILTER PRESS INSTALLED AT WASTE WATER TREATMENT PLANT | 08/94 |
| * BEGAN DISPOSAL OF FILTER CAKE IN DRY RUN | 09/94 |
| * BLACK WATER APPEARED | 01/95 |
| * INSPECTION BY STATE DEPT ENV PROTN | 03/23/95 |
| * TENNANT MADE STREAM VIDEOS | AP-MY95 |
| * INSPECTION BY STATE | 05/03/95 |
| * ROUTINE INSPECTION OF DOWNSTREAM BEGAN | 05/95 |
| * BLACK WATER PROBLEM SOLVED | 05/95 |
| * INSPECTION BY STATE ENFORCEMENT & PERMIT WRITER | 10/95 |
| * TENNANT CONTACTED DNR (ELKINS OFFICE) ABOUT "STREAM POISONING DEER" & WV DEPT OF AG | 12/95 |
| * TENNANT BEGAN TALKING IN COMMUNITY ABOUT CATTLE LOSSES | 95PM-96AM |
| * DISCUSSION WITH WV DEPT OF AG VET, WV EXT SERV AGENT, & LOCAL LARGE ANIMAL VET | SPRNG 96 |
| * WASHINGTON WORKS COMMUNITY RESPONSIBLE CARE TEAM STARTED | 01/22/96 |
| * EPA SOIL & WATER SAMPLING | 10/96 |
| * DRAFT COMPLAINT FROM WV DEP | 10/96 |
| * FREEDOM OF INFORMATION ACT REQUEST BY DuPONT TO EPA | 11/06/96 |
| * TERRADON/POTESTA | 11/96 |
| * BEGAN DISPOSAL OF FILTER CAKE AT NORTHWEST LANDFILL | 12/96 |
| * ADMIN SETTLEMENT REACHED WITH WV DEP CONCERNING ALLEGED VIOLATION | 12/96 |
| * BEGAN TRUCKING LEACHATE BACK TO PLANT | 01/97 |
| * DISSECTION OF CATTLE VIDEO | 02/97 |
| * NOTIFICATION BY EPA OF CONCERNS QUESTION | 02/97 |
| * RESPONSE TO EPA | 03/20/97 |
| * MEETING WITH COMMUNITY LEADERS<br>(LEGISLATORS - COMMUNITY AWARE? - ECC) | 05/97 |
| * "OPEN HOUSE" FOR COMMUNITY AT DRY RUN | 06/97 |
| * EPA ENVIRONMENTAL RISK ASSESSMENT SAMPLING | 06/97 |
| * FLUORIDE SAMPLES TAKEN BY DuPONT | 06/23/97 |
| * LETTER TO SITE EMPLOYEES BY PLANT MANAGEMENT | 07/27/97 |
| * PERMIT APPLICATION PUBLIC HEARING | 07/97 |
| * CEASED DISPOSAL OF ASBESTOS AT DRY RUN | 01/01/98 |
| * SPELTER BECAME ISSUE | 03/98 |
| * TENNANT LEASE CANCELLED | 04/98 |
| * BEGAN SECURITY PATROLS ON WEEKENDS (CONTRACT AGENCY) | 06/98 |
| * INSPECTION BY STATE | 07/07/98 |
| * MEETING WITH WV DEP OFFICE OF WATER QUALITY TO ENGAGE THEM IN EPA ACTIVITY | 07/98 |

LKI000768

## DRY RUN LANDFILL: C-8 HISTORY SUMMARY

- SUPERNATE POND SOIL DISPOSAL:

  - 7100 TONS OF SOIL CONTAINING ~ 85 PPM C-8 DISPOSED OF NEAR THE BOTTOM OF THE LANDFILL 11/88.

  - THIS SOIL WAS MOVED TO THE TOP OF THE LANDFILL AND BURIED IN A CLAY-LINED SEPARATE CELL 10/91.

- C-8 GROUNDWATER AND SURFACE WATER MONITORING:

  - BEGAN MONITORING ANNUALLY FOR C-8 IN 1991.

  - GROUNDWATER CONCENTRATIONS RANGE <1 - 15 PPB.

  - LEACHATE CONC. AT O.F. 001 RANGES 30 - 200 PPB.

    * PROPERTY BOUNDARY STREAM CONCENTRATION RANGES 2 - 25 PPB.

  - DATA REPORTED ANNUALLY TO WVDEP BEGINNING WITH 1991 ANNUAL REPORT.

    * 1998 RESULTS:

| | | |
|---|---|---|
| LEACHATE | = 56 | PPB |
| OUTLET 001 | = 17 | PPB |
| STREAM #1 | = 1 | PPB |
| STREAM #2 | = 4.6 | PPB |
| PROP. BND. | = 0.88 | PPB |

- ~ 7 POUND/YR C-8 DISPOSED OF WITH BIOCAKE (930 PPB).

- 600M POUNDS/YR OF FLUOROPOLYMER WASTE DISPOSAL BEGAN 1/96; DOES NOT CONTAIN C-8.



EID078867

RJZ006998

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# C-8 HISTORICAL PROSPECTIVE

- Known Contaminant Sources
  - Supernate Ponds (Closed 1988)
    (Excavated Soil removed to Dry Run Landfill)
    Excavated Soil Levels - 234 ppm Max
    Residual Level in Remaining Riverbank Soil-173 ppm Max
  - Lubeck Public Service Division
    Test Well 27 - 1-2 ppb
    Employee Water Taps - 1-2 ppb
  - Letart Landfill
    Landfill Leachate - 3 ppm
    Groundwater Wells - 1.6 ppm (Poss. Construction Cont.)
  - Dry Run Landfill
    Landfill Leachate - 0.7 ppm
    Leachate Stream at Property Line - 0.1 ppm

- Regulatory Knowledge
  - EPA SWMU Survey - 6/5/85
    C-8 in trace levels in Supernate waste
    C-8 found in ppb levels in aquifer
  - NPDES Permit Application - 9/2/86
    Surfactants in 002/005 Discharge (Not specific mention)
  - Supernate Sludge Disposal Request - 7/30/87, 8/23/90
    Supernate Sludge contained 85 ppm C-8
  - Lubeck Public Service Division - 6/13/89
    C-8 has been found in aquifer in ppb levels
    Stated no health hazard
  - Letart Landfill Permit Application - 9/21/89
    C-8 in stream and wells reported in required analysis

## AIM

The aim of this meeting is to discuss what should be considered an acceptable level of C-8 in both surface water and ground water and to develop a plant strategy for dealing with the C-8 contamination at the Dry Run landfill.

## AGENDA

Review:

- C-8 - What is it?
- C-8 - Overview of limits and guides
- Source of C-8 contamination at Dry Run - Supernate pond sludge - 14,000,000 pounds - C-8 concentration - 275 ppm
- Location and amount of current known contamination

Discuss:

- What is an acceptable level of C-8 in surface water?
- What is an acceptable level of C-8 in ground water?
- Based on these levels, what is the appropriate action to be taken at Dry Run at this time

Some Options:

- No Action, monitor for further contamination
- Cap material in place, monitor for further contamination
- Move material to Letart landfill
- Incinerate
- New technology
  - Calcining
  - RO
  - Ion Exchange

ATTACHMENT 9

is TSCA CBI.

DCN 6301 000004

No sanitized version

was provided.

PFOA Concentrations

| | | |
|---|---|---|
| Washington Works drinking water | 1999 | 0.2 – 0.5 ppb (building samples) |
| | 1999 | 0.082 ppb (drinking water wells) |
| | 1998 | 1.9, 1.0, and 0.4 ppb (drinking water wells) |
| Outfall 005 | 1990's | 50-60 ppb |
| GE Plastics drinking water | 1999 | 0.5 ppb (drinking water well) |
| Lubeck Public Service District | 2000 | 1st Q: 0.8, 0.44, and 0.313 ppb<br>2nd Q: 0.315, 0.516, and 0.21 ppb |
| | 1992 | 0.2, 0.4, and 0.09 ppb |
| Old LPSD wells + WW well 27 | 1984-88 | 9 samples ranging from 1.0 – 2.2 ppb |
| Dry Run Landfill | 1999 | |
| Leachate | | 34 ppb |
| Outlet 001 | | 66 ppb |
| Stream Sampling Station No. 1 | | 0.54 ppb |
| Stream Sampling Station No. 2 | | 87 ppb |
| Property Line | | 39 ppb |
| Letart Landfill | 2000 | |

Monitoring wells are identified by Zones, with A nearest the surface and F the deepest.

| | |
|---|---|
| Zone A | |
| MW-1 | 17,400 ppb |
| MW-7 | 219 ppb |
| MW-8 | 2,100 ppb |
| Zone D/E | |
| MW-4 | 172 ppb |
| Zone F | |
| MW-2A | 453 ppb |
| MW-5B | 1,030 ppb |
| MW-6 | 9.4 ppb |

EID089310

Blood Monitoring Data 1995

None higher than 5 ppm. Previously, levels had been detected up to 33 ppm.

000057

PFOA Concentrations

| | | |
|---|---|---|
| Washington Works drinking water | 1999 | 0.2 – 0.5 ppb (building samples) |
| | 1999 | 0.082 ppb (drinking water wells) |
| | 1998 | 1.9 ppb (drinking water wells) |
| Outfall 001 | 1999 | 66 ppb |
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| MW-6 | 9.4 ppb |

| | |
|---|---|
| Blood Monitoring Data | 1995 |

None higher than 5 ppm. Previously, levels had been detected up to 33 ppm.

EID089516

CC: R. J. Burger
C. R. Campbell
J. R. Broadway
J. L. Granquist
R. J. Zipfel
P. Thistleton
G. H. Stoltz
C. A. Robinson
K. G. Kronberg
J. F. Doughty

# C-8 COMMUNICATIONS MEETING

## OUTLINE, TALK & CHARTS

C. E. STEINER
7/31/80

**PERSONAL & CONFIDENTIAL**

EID079399

INTRODUCTION

- C-8's desirable process qualities
- Short C-8 history in TFE & FEP Manufacture

TOXICITY

- Oral toxicity - slightly toxic
  - Compare to other compounds
- Skin contact - slightly to moderately toxic
- Inhalation toxicity - highly toxic
  - Compare to other compounds
  - Concentrations found in area are lower

INITIAL BLOOD TESTS

- 3M Data
- Our Results

RECOGNIZING EXPECTED OPERATOR QUESTIONS - A transition

- Some disbelieve based on past experience
- Short history of chemicals in industry showing why we are careful

MEDICAL RECORD STUDIES

- No evidence of health problem
- Studies thorough

PROVISIONAL AEL

- AEL committee has set provisional AEL of 0.55 mpb
- Not yet firm AEL
- This very low number is to protect people who work with C-8 every day
- The low provisional AEL and goal to reduce blood fluorine is the reason we are making changes in equipment and procedures.

EQUIPMENT IMPROVEMENTS

- Goal to reduce exposure to solid C-8, airborn C-8 and C-8 solutio
- Ingredients addition hood and stack
- Eliminate Weighing Citric Acid in C-8 hood
- Raising Dryer Air supply Inlets
- Seal Dryer Leaks
- Additional Dryer Windows
- Increase Ventillation During Outages
- Removing C-8 from Dryer Exhausts

## PROTECTIVE EQUIPMENT

### Clothing and Gloves

- Needs to be disposable to prevent secondary contamination.
- An EOD is being prepared to evaluate clothing.
- Different protection levels for 3 exposure classes

### Breathing

- Equipment improvements will reduce airborne C-8 but high C-8 concentrations will still remain in some areas.
- Breathing air will be installed - ultimate solution.
- Comfo II air respirator with GMAH cartridge acceptable.

## TESTING

### Personal Air Samples

- Will Resample.

### Blood Samples

- Blood sampling will be resumed.
- Frequent sampling is not necessary.

### Area Air Samples

- Will continue to define progress.
- Often exceed provisional AEL before improvements.

## SUMMARY

- C-8 is toxic but can be handled safely.
- People working with C-8 generally accumulate organic fluorine in the blood, and levels generally correlate with job exposure potential.
- Although this has caused no health effects continued exposure is not tolerable.
- Our basic goals are to reduce exposures to below the provisional AEL, and to reduce organic fluorine levels in blood of exposed workers and prevent accumulation in new workers.
- This will require equipment changes that are being done.
- It will also require use of disposable protective clothing and us of breathing air or respirators for certain jobs.

One other ingredientient is needed -- your cooperation in controlling this hazard.

CES
6/3/80

EID079401

C-8 COMMUNICATION MEETING

The purpose of this meeting is to bring everyone up to date on our findings regarding C-8, our immediate program , and our long term plans.

Most of you know that C-8 is a fluorochemical surfactant that is used for producing fine powder, dispersion, granular and FEP. It has unique properties that allow it to wet Teflon's surface, shorten reaction cycle time, stabilize dispersions and provide sites for reactions. It has been used for Teflon® manufactur for over 25 years. Other chemicals have been tested but none match C-8's properties. Four years ago it was introduced in FEP manufacture where it was a manufacturing improvement.

Let's look over the highlights of the Technical history of C-8. In 1965 tests showed that C-8 was slightly toxic when swallowed. This was not surprising. There is a dose level where almost every chemical becomes poisonous, even water. (Chart 1). This chart shows the oral toxicity of C-8 relative to some common chemicals. These tests were done on animals, and represent what dose would kill 50% of the animals tested. I've scaled up the dose from test data to animal weights comparable to an operator's weight. You can see that C-8 is not as toxic as acetone. It has a lower toxicity like table salt.

C-8, like table salt, can also be absorbed through the skin where it is about as toxic as it is orally. But, based on this low toxicity, no change in our safety program was necessary.

In 1969 it was found that C-8 was more toxic by inhalation, Chart 2. This second chart shows the approximate concentration that will kill test animals in a 4 hour period. This approximate lethal concentration for rats exceeds anything we have measured in the plant. The highest level ever measured in the plant is about 1/4 of that level -- and that a 1.1mpm leak at the feed end of No. 3 dryer which has been repaired. The other C-8 concentrations are generally about 1,000 to 10,000 times lower than this so people working in the area see no immediate effect. (.004-mpm)

However, since 3M informed us in 1978 of organic fluorine being detected in the blood of their employees who worked with C-8, we have been reviewing and expanding our C-8 program. We have concluded that personnel routinely exposed to C-8 will absorb it in their body. Tests at Washington Works show that blood fluorine levels which indicate C-8 levels generally correlate with potential job exposure.

Repeated exposures can result in accumulation of C-8 in the blood. One of the things that we are studying with the blood samples is the rate that C-8 is eliminated from the body.

Some of the old timers remember when C-8 was treated with less respect and they wonder "Why is it suddenly harmful now?"

Throughout the chemical industries over the last 50 years this story has been repeated with the same disbelief but often with more drastic consequences.

For example, carbon tetrachloride was used to clean auto parts and as a fire extinguisher for years, and now it is known to cause damage in some people and is used with care. The same story has been repeated several times for things like chloroform (which was used in cough suryp), methyl alcohol and other chemicals.

The difference between the ending of the C-8 story and the others is that Du Pont is reacting while C-8 levels in the blood are low and before any damage is done in the body.

The medical data show that no one has been injured by C-8 (Chart 4). The Medical Division after a thorough study has concluded that ". . .there is no conclusive evidence of an occupationally related health problem among workers exposed to C-8." All that was noted was a small increase in two liver enzyme levels. After 25 years of handling C-8 we see no damage among the workers. However, the potential is there -- C-8 has accumulated in the blood.

Because of this accumulation we have decided to undertake programs to minimize accumulation of C-8 in the blood of new workers.

The AEL Committee of Haskell Laboratories has set a provisional Allowable Exposure Limit or "AEL" at 0.55 mpb of C-8 in air. This very low proposal is based on a safety factor of 800 below the level where reversible liver effects were observed. An AEL is the same thing as a TLV or EGL -- it is a safe concentration in the air of a working environment.

In order to meet the expected low AEL, equipment changes are necessary to protect from solid, liquid and airborn C-8.

The next transparencies show the changes that have been made recently to protect against C-8 exposure. To date we have:

- Modified the Fine Powder/Dispersion ingredients addition hood to reduce C-8 emissions and bring the mixing operations into the hood. C-8 tools will also be stored in the hood where possible.
- Improved the C-8 addition hood exhaust stack. The hood exhaust stack was close to an H & V inlet on the roof.
- Removed operations that don't have to be done in the C-8 hood -- like citric acid weighing. This has reduced exposure of concentration to the operators

The dryers have been improved also:

- Air supply inlets have been raised to remove C-8 rich air from the ceiling.

- Seals of No. 3 dryer doors and seams have been improved.
- Inspection windows have been added to reduce need to open dryer doors.

We have also put guards inside the dryer that will permit using the exhaust fans to remove C-8 when dryers are being cleaned. This has reduced some C-8 concentrations, but more work is to be done; for example, we plan to cover injection pump tanks, seal openings in floor and vent oscillating feeder compartments, sealing No. 3 dryer fans.

The next chart shows the three different protection levels required for three exposure classes: Low dry exposure, high dry exposure and wet exposure. A disposable garments of the appropriate design, gloves and air protection are recommended for each of these exposure classes. Sample garments have been selected and an EOD will be run to evaluate this clothing. Tyvek® was selected over cloth or paper garments because it is light fairly resistant to tearing, a good filter and disposable. Disposability is required to prevent secondary contamination when laundering. During this EOD, sample garments will be tried and evaluated by operators and mechanics.

C-8 will permeate all glove materials over a period of time. New flock lined latex gloves will be used in jobs where C-8 exposure is likely. Even these gloves will be permeated by C-8 over a period of time, so these gloves will be disposed of after each shift.

Breathing protection is very important to reducing C-8 exposures. Equipment improvements will reduce airborn C-8 in most areas but there will still be areas where exposure is possible. A COMFO II air respirator with a special GMAH cartridge is required as a minimum. Breathing air is better and will be available soon. The yellow 3M masks are not acceptable.

I've had some questions on future C-8 air samples and blood samples. We now have our baseline data and have mapped out the problem areas. The procedures are modified and equipment improved so C-8 exposures will be reduced.

Blood sampling will probably be done on an annual basis in the future to define the real improvements in C-8 control.

Let me summarize the items covered:

- C-8 is toxic, but it can be used and controlled below the proposed toxic limit.
- In the past, people working with C-8 have accumulated organic fluorine in the blood and levels generally correlate with job exposure potential.
- Although this has caused no health effects, continued exposure should be minimized with controls.
- Our objective is to reduce exposures to below the provisional AEL, and to reduce organic fluorine levels in blood of exposed workers and to limit accumulation in new workers.

- This will require equipment changes that are partially comp
- It will also require use of disposable protective clothing and use of breathing air or respirators for certain jobs.
- One other ingredient is needed -- Total Division cooperation in controlling this material.

AJP00761



EID079408

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Mark type only employee number not name attached

~~PERSONAL & CONFIDENTIAL~~

~~C-8~~ BLOOD SAMPLING RESULTS

## Births and Pregnancies

| ~~Current~~ (1v) PPM C-8 in Blood ~~April 1981~~ | Status |
|---|---|
| 0.45 | Normal child - born June 1980. Transferred out of Fluorocarbons 4/79. |
| 0.28 | Normal child - born April 1981. |
| 0.078 | Normal child - born April 1981. Umbilical cord blood 0.055 ppm. |
| 1.5 | ~~Five months pregnant~~. On pregnancy leave |
| 0.013 | ~~Five months pregnant~~. Normal child - born August 19 |
| 2.5* | Child - 2 plus years. Unconfirmed eye and tear duct defect. |
| 0.048 | Child - 4 months. One nostril and eye defect. Babies blood 0.012ppm |
| 2.007 | Normal child - born July 1981 |

*Current blood level - in fluorocarbons area only one month before pregnancy.

~~RDI.ldr~~



EID079575

000072

~~R. E. Hansel - Bldg. 1~~
~~C. W. Crowe - Bldg. 211~~
H. C. Wright - Bldg. 1

CC: W. A. Bower
J. F. Thomas
A. C. Huston
~~W. A. Barlow~~
File: 37.00.00

September 21, 1981

TO: J. J. DI NICOLA

FROM: D. H. SNYDER

WASHINGTON WORKS
LANDFILL FACT SHEET

Ref: (1) Letter D. H. Snyder to C. W. Crowe 9/1/81, same title.
(2) Letter, C. W. Crowe to E. R. Kimmel, A. L. Skinner and J. L. Stowell, 9/11/81, same title.

Attached is a copy of the Landfill Fact Sheet which we reviewed by phone on 9/16/81. It will be used by J. R. Draper of the Real Estate Division of General Services Department in his contacts with landowners. It is in response to requests by some owners.

C. W. Crowe of the Departmental Engineers Office reviewed this with both Legal and Public Affairs. They have approved its use.

/jas(1669A)
Attachment

EXHIBIT
5

CF001293



EID022224

## LANDFILL INFORMATION

The landfill will contain nonhazardous wastes only, including ash from plant boilers, waste plastics, glass, scrap metal, paper, and trash, all transported by covered truck or closed containers.

It will be designed and operated with full consideration for neighbors. The ash will be wetted, and the road will be paved to control dust and mud. The fill will be covered with dirt at the end of each day.

Daytime only operation is planned. The site could receive 10 to 14 truckloads per day.

The probable start-up date is late 1983.

CF001294

EID022225

000074

DUPONT

*The miracles of science*™

Study Title

# MODELING RELEASES OF AMMONIUM PERFLUOROOCTANOATE INTO THE OHIO RIVER


Author

William R. Berti



Draft Report Completed on

May 22, 2000



Performing Laboratory

E. I. du Pont de Nemours and Company
Environmental and Microbiological Sciences & Engineering
Corporate Center for Engineering Research
Central Research & Development
Glasgow, Building 300, P.O. Box 6101
Newark, DE 19714-6101



Report No. EMSE-054-00

CCER
CORPORATE CENTER FOR ENGINEERING RESEARCH


RP00196




EID108608

**CONTAINS CONFIDENTIAL BUSINESS INFORMATION**


000075

GENERAL INFORMATION

Chemical Studied: Ammonium perfluorooctanoate

Synonyms/Codes: C-8, FC-143

DuPont Notebook No.: Not Applicable

Sponsor; Robert Pinchot
E. I. du Pont de Nemours and Company
BARLML CRP-711/2210-B

Study Initiated/Completed: September 1999 to May 2000

RP001968



CONTAINS CONFIDENTIAL BUSINESS INFORMATION

# MODELING RELEASES OF AMMONIUM PERFLUOROOCTANOATE INTO THE OHIO RIVER

## SUMMARY

PDM indicated that C-8 concentrations of 1.0 g C-8/L would be exceeded about 50% of the time during the year. C-8 concentrations of in the river would exceed 0.1 g C-8/L 90% of the time during the year and 10 g C-8/L about 2.2% of the time during the year.

Average annual C-8 concentrations in the Ohio River calculated by using a Microsoft® Excel spreadsheet was 0.423 g C-8/L. Modeled C-8 concentrations in the river ranged from a low of 0.199 g C-8/L in March to a high of 0.965 g C-8/L in September, which correspond to high and low river flows, respectively. Average Ohio River flows and volume data calculated from the US Geological Survey was collected at the Belleville Dam and used in the spreadsheet model. The Belleville Dam is on the Ohio River 13 miles downstream of the Washington Works Plant. This river flow data is the closest location downstream from the plant where this type of information is available.

Study Conducted and
Report Prepared by: _____

William R. Berti (date)
Senior Research Biologist

RP001969



EID108610

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## INTRODUCTION/PURPOSE

C-8 (ammonium perfluoroocatanoate) is a fluorinated surfactant used in the manufacturing of Teflon® at Washington Works. A portion the C-8 (18,416 kg in 1996) was released to the Ohio River as part of an NPDES permitted release for the facility (WV0001279). The molecular weight of C-8 is 431.098 g/mole. Other properties of C-8 are listed below.

- Made by 3M Company
- Health rating of 2
- Exposure limits 0.01 $mg/m^3$ skin 8 hr
- $LD_{50}$ acute oral rat = 680 mg/kg
- $BOD_{20}$ = nil
- Biodegradation = nil
- BCF = 1.8
- Molecular formula $CF_3(CF_2)_6COO^-NH_4^+$
- pH ~ 5 (0.5% aqueous)
- pKa = 2.8 (-COOH)
- Melting point = 56-58°C (-COOH)
- COD = 700 mg/kg
- Koc = 25
- Water solubility > 1000 mg C-8/L
- Vapor pressure (at 22C) = $7.1 * 10^{-05}$ mm Hg

Our objective was to calculate the concentrations of C-8 that could reasonably be expected to occur in the Ohio River downstream of the NPDES permitted outfall at the DuPont Washington Works.

## MATERIALS/METHOD

Releases of ammonium perfluorooctanoate (C-8) to the Ohio River from the DuPont Washington Works Plant were modeled using the Probabilistic Dilution Model (PDM Beta Version 4.0 Beta June 11, 1999, US EPA Office of Pollution Prevention and Toxics) and a constructed Microsoft® Excel spreadsheet model. C-8 release data for 1996 were used in both modeling exercises.

PDM inputs included the NPDES number of WV0001279. This corresponds to a reach number of 05030202039, a mean streamflow of 4.75E+04 million liters per day (MLD), a low streamflow of 9980.47 MLD and an effluent flow of 216.75 MLD. We assumed that C-8 was released 335 days/year, the loading was 55.138 kg/day, and the wastewater treatment efficiency was 0%. We then varied the C-8 concentration as the Concentration of Concern (COC) from 0.1 to 50 g C-8/L to determine the COC percent of year exceedence.

Microsoft® Excel spreadsheet used river flow data from the US Geological Survey (USGS: <http://waterdata.usgs.gov/nwis-w/WV/data.components/hist.cgi?statnum=03159530>). This is the mean daily river flow in cubic feet/sec measured at the Belleville Dam from 10/1/1974 to 9/30/1985. The Belleville Dam is on the Ohio River 13 miles downstream of the Washington Works Plant. This river flow data is the closest location downstream from the plant where this type of information is available. This river flow data compares well with Ohio River monthly average flows at Pittsburgh, PA, Huntington, WV, and Cincinnati, OH from the US Army Corps of Engineers (ftp://www.lrd-wc.usace.army.mil/Monthly_htm/). The average daily river flow for the month was calculated from available data between those two dates. This was then converted to the average measured flow per month. We assumed the C-8 was discharged to the river at a constant rate except for the month of October when production at Washington

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Works is usually suspended for annual maintenance. We assumed that the total discharge of was 18,416 kg C-8 (40,600 lbs C-8) divided by 11 months. Table shows the results of this spreadsheet model.

## RESULTS/DISCUSSION

PDM indicated that C-8 concentrations of 1.0 g C-8/L would be exceeded about 50% of the time during the year. C-8 concentrations of in the river would exceed 0.1 g C-8/L 90% of the time during the year and 10 g C-8/L about 2.2% of the time during the year (Table 1). Figure 1 shows model input/output.

Average annual C-8 concentrations in the Ohio River calculated by constructing and using a Microsoft® Excel spreadsheet model was 0.423 g C-8/L (Table 2). Modeled C-8 concentrations in the river ranged from a low of 0.199 g C-8/L in March to a high of 0.965 g C-8/L in September, which correspond to high and low river flows, respectively. Average Ohio River flows and volume data calculated from the US Geological Survey was collected at the Belleville Dam and used in the spreadsheet model. The Belleville Dam on the Ohio River 13 miles downstream of the Washington Works Plant. This river flow data is the closest location downstream from the plant where this type of information is available.

RP001971



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TABLE 1: Probabilistic Dilution Model (PDM) results of C-8 concentrations and percent of year that the concentration of C-8 will be exceeded in the Ohio River.

| Concentration of C-8 | Percent of year concentration exceeded |
|---|---|
| *g/L* | % |
| | |
| 0.1 | 90.9 |
| 0.5 | 71.6 |
| 1 | 51.0 |
| 3 | 17.8 |
| 6 | 6.1 |
| 10 | 2.2 |
| 25 | 0.2 |
| 50 | 0.0 |

RP001972



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Table 2. Microsoft Excel spreadsheet calculations of modeled C-8 concentrations in the Ohio River.

| Month | Measured flow | Measured flow | C8 discharge to Ohio River in 1996 | Estimated C8 concentration in Ohio River |
|---|---|---|---|---|
| | *$ft^3/sec$* | *L/mo* | *kg* | *g/L* |
| | | | | |
| January | 64,001 | 4.69E+12 | 1,674 | 0.357 |
| February | 88,909 | 6.74E+12 | 1,674 | 0.248 |
| March | 111,236 | 8.43E+12 | 1,674 | 0.199 |
| April | 94,017 | 6.90E+12 | 1,674 | 0.243 |
| May | 67,107 | 5.09E+12 | 1,674 | 0.329 |
| June | 47,379 | 3.48E+12 | 1,674 | 0.482 |
| July | 30,989 | 2.35E+12 | 1,674 | 0.713 |
| August | 30,502 | 2.31E+12 | 1,674 | 0.724 |
| September | 25,339 | 1.73E+12 | 1,674 | 0.965 |
| October | 33,030 | 2.50E+12 | 0 | 0.000 |
| November | 42,330 | 3.11E+12 | 1,674 | 0.539 |
| December | 78,986 | 5.99E+12 | 1,674 | 0.280 |
| | | | | |
| **Average** | | **4.44E+12** | | **0.423** |
| **Total** | **713,824** | **5.33E+13** | **18,416** | **0.345** |

RP001973

EID108614



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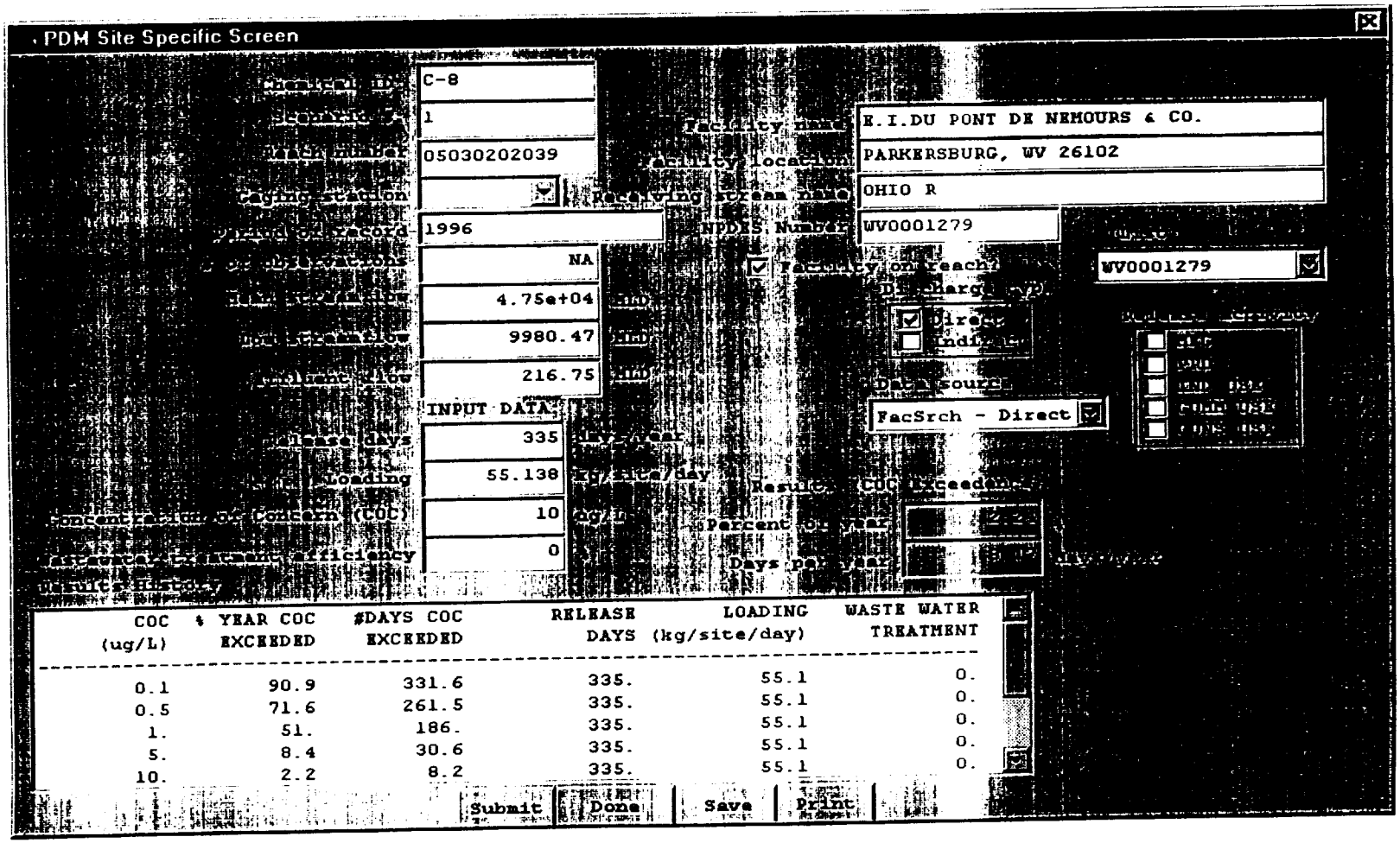
ADSORPTION-DESORPTION STUDIES OF AMMONIUM PERFLUOROOCTANOATE

FIGURE 1: Probabilistic Dilution Model (PDM) Beta Version 4.0 input and output screen.




EID108615



RP001974

CONTAINS CONFIDENTIAL BUSINESS INFORMATION

T. A. Foster
R. N. Taylor
P. Thistleton

October 19, 1982

PERSONAL AND CONFIDENTIAL

TO: R. J. ZIPFEL

FROM: J. G. LOSCHIAVO J.G.L.

ESTIMATE OF HUMAN C-8 EXPOSURE RESULTING FROM DRINKING OHIO RIVER WATER

As you requested, I estimated human exposure to C-8 as a result of drinking Ohio River water. As a result, I've found human exposure to C-8 to be very low. Using 1982 manufacturing conditions (and no scrubbing of Fine Powder Dryer exhaust), persons would be exposed to a C-8 dose roughly equivalent to 0.08% of the acceptable exposure limit (AEL). Using projected manufacturing conditions for 1987 (with scrubbing of Fine Powder Dryer exhaust), persons would be exposed to a C-8 dose roughly equivalent to 0.35% of the AEL. It should also be mentioned that C-8 is more toxic via the inhalation route than the oral route. Therefore, these percentages would in actuality be even lower.

Human C-8 exposure calculations were made using three separate sets of conditions:

- Case 1 - Reflects 1982 manufacturing conditions with no scrubbing of the Fine Powder Dryer exhaust.
- Case 2 - Reflects 1982 manufacturing conditions with scrubbing of the Fine Powder Dryer exhaust.
- Case 3 - Reflects projected 1987 manufacturing conditions with scrubbing of the Fine Powder Dryer exhaust.

The results of my calculations are contained in the attached Table. My calculations are also attached.

JGL:sdc
1799B-1

EID079287

000081

TABLE 1. Estimated Ohio River C-8 Concentrations and Human C-8 Exposure Reflecting 1982 and 1987 Manufacturing Conditions

| Case No. | C-8 Concentration in River Water (ppm by wt.) | Average Daily Amount of C-8 Ingested (μg) | % of Equivalent ADI Dose |
|---|---|---|---|
| 1 | $0.8 \times 10^{-4}$ | 0.08 | 0.08 |
| 2 | $1.1 \times 10^{-4}$ | 0.11 | 0.11 |
| 3 | $3.5 \times 10^{-4}$ | 0.35 | 0.35 |



EID079288

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## CALCULATIONS

General Assumptions:

- An average person drinks 1 liter of water per day.
- An average person inhales $10m^3$ of air over an 8-hour shift.
- Average Ohio River flow rate is 13,880,000,000 lb. water per hour.
- The scrubber on the Fine Powder Dryer stacks would be 90% efficient.
- When the Fine Powder Dryer exhaust is not scrubbed, 3% of the total C-8 added to Fine Powder/Dispersion goes to the River.
- When the Fine Powder Dryer exhaust is scrubbed, 31% of the total C-8 added to Fine Powder/Dispersion goes to the River.
- Approximately 81% of the total C-8 added to FEP goes to the River.

Specific Case Assumptions:

### Case 1

- Fine Powder/Dispersion will manufacture 6.9 MMAP during 1982.
- An average of 0.0022 lb. of C-8 is added to make each pound of Fine Powder/Dispersion (35% solids).
- FEP will manufacture 6.7 MMAP during 1982
- An average 0.0016 lb. of C-8 is added to make each pound of FEP (low C-8 recipe).

### Case 2

- Same as above except the Fine Powder Dryer exhaust is scrubbed throughout 1982.

### Case 3

- Fine Powder/Dispersion will manufacture 16 MMAP during 1987.
- An average 0.0033 lb. of C-8 will be added to make each pound of Fine Powder/Dispersion (45% solids).
- FEP will manufacture 11 MMAP during 1987.
- An average 0.0030 lb. of C-8 will be added to make each pound of FEP (high C-8 recipe).

## Case 1 Calculations

Fine Powder/Dispersion:

$$\frac{\left(6.9 \times 10^6 \frac{\text{lb. TEFLON®}}{\text{yr.}}\right)\left(0.0022 \frac{\text{lb. C-8}}{\text{lb. TEFLON®}}\right)(.03)}{365 \frac{\text{days}}{\text{yr.}} \times 24 \frac{\text{hr.}}{\text{day}}} = 0.05 \frac{\text{lb. C-8}}{\text{hr.}} \text{ goes to River}$$

FEP:

$$\frac{\left(6.7 \times 10^6 \frac{\text{lb. TEFLON®}}{\text{yr.}}\right)\left(0.0016 \frac{\text{lb. C-8}}{\text{lb. TEFLON®}}\right)(0.81)}{365 \frac{\text{days}}{\text{yr.}} \times 24 \frac{\text{hr.}}{\text{day}}} = 1.00 \frac{\text{lb. C-8}}{\text{hr.}} \text{ goes to River}$$

Total C-8 going to the River per hour = $1.00 + 0.05 = 1.05 \frac{\text{lb. C-8}}{\text{hr.}}$

C-8 Concentration in River (wt./wt.) $= \left(\frac{1.05 \text{ lb. C-8/hr.}}{13{,}880{,}000{,}000 \text{ lb. water/hr.}}\right) \times 10^6 = 8 \times 10^{-5}$ ppm

Amount of C-8 Ingested with Drinking Water Each Day $= \left(1 \text{ liter of water}\right)\left(1000 \frac{\text{gm water}}{\text{liter water}}\right)\left(8 \times 10^{-5} \text{ppm}\right)\left(10^{-6}\right)$

$= 8 \times 10^{-8}$ gm. or 0.08 µg of C-8

Amount of C-8 Inhaled at the AEL Concentration over an 8-hr. Shift $= \left(10 \frac{\mu\text{g of C-8}}{\text{m}^3 \text{ of air}}\right)\left(10\text{m}^3 \text{ of air}\right) = 100$ µg of C-8

Ingesting 0.08 µg of C-8 is equivalent to:

$$\frac{0.08 \ \mu\text{g of C-8}}{100 \ \mu\text{g of C-8}} \times 100 = 0.08\% \text{ of the AEL}$$



EID079290

## Case 2 Calculations

Fine Powder/Dispersion:

$$\frac{\left(6.9 \times 10^6 \frac{\text{lb. TEFLON®}}{\text{yr.}}\right)\left(0.0022 \frac{\text{lb. C-8}}{\text{lb. TEFLON®}}\right)(0.31)}{365 \frac{\text{days}}{\text{yr.}} \times 24 \frac{\text{hr.}}{\text{day}}} = 0.54 \frac{\text{lb. C-8}}{\text{hr.}} \text{ going to River}$$

FEP:

Same as shown in Case 1.

**Total C-8 going to the River per hour = 1.00 + 0.54 = 1.54 $\frac{\text{lb. C-8}}{\text{hr.}}$**

C-8 Concentration in River (wt./wt.) $= \left(\frac{1.54 \text{ lb. C-8/hr.}}{13{,}880{,}000{,}000 \text{ lb. water/hr.}}\right) \times 10^6 = 1 \times 10^{-4}$ ppm

Amount of C-8 Ingested with Drinking Water Each Day $= (1 \text{ liter of water})\left(1000 \frac{\text{gm water}}{\text{liter water}}\right)(1.1 \times 10^{-4} \text{ ppm})(10^{-6}$

$= 1.1 \times 10^{-7}$ gm. or 0.11 $\mu$g of C-8

Amount of C-8 Inhaled at the AEL Concentration over an 8-hour Shift = 100 $\mu$g of C-8

Ingesting 0.11 $\mu$g of C-8 is equivalent to:

$$\frac{0.11 \mu\text{g of C-8}}{100 \mu\text{g of C-8}} \times 100 = 0.11\% \text{ of the AEL}$$







DEF 002716

## Case 3 Calculations

Fine Powder/Dispersion:

$$\frac{\left(16 \times 10^6 \frac{\text{lb. TEFLON®}}{\text{yr.}}\right)\left(0.0033 \frac{\text{lb. C-8}}{\text{lb. TEFLON®}}\right)\left(0.31\right)}{365 \frac{\text{days}}{\text{yr.}} \times 24 \frac{\text{hr.}}{\text{day}}} = 1.85 \frac{\text{lb. C-8}}{\text{hr.}} \text{ going to River}$$

FEP:

$$\frac{\left(11 \times 10^6 \frac{\text{lb. TEFLON®}}{\text{yr.}}\right)\left(0.0030 \frac{\text{lb. C-8}}{\text{lb. TEFLON®}}\right)\left(0.81\right)}{365 \frac{\text{days}}{\text{yr.}} \times 24 \frac{\text{hr.}}{\text{day}}} = 3.06 \frac{\text{lb. C-8}}{\text{hr.}} \text{ going to River}$$

Total C-8 going to the River per hour = 1.85 + 3.06 = 4.91 $\frac{\text{lb. C-8}}{\text{hr.}}$

C-8 Concentration in River (wt./wt.) $= \left(\frac{4.91 \text{ lb. C-8/hr.}}{13{,}880{,}000{,}000 \text{ lb. water/hr.}}\right) \times 10^6 = 3.5 \times 10^{-4}$ ppm

Amount of C-8 Ingested with Drinking Water Each Day $= \left(1 \text{ liter of water}\right)\left(1000 \frac{\text{gm water}}{\text{liter water}}\right)\left(3.5 \times 10^{-4} \text{ ppm}\right)\left(10^{-}\right.$

$= 3.5 \times 10^{-7}$ gm. or 0.35 $\mu$g of C-8

Amount of C-8 Inhaled at the AEL Concentration over an 8-hour Shift = 100 $\mu$g of C-8

Ingesting 0.11 $\mu$g of C-8 is equivalent to:

$$\frac{0.35 \ \mu\text{g of C-8}}{100 \ \mu\text{g of C-8}} \times 100 = 0.35\% \text{ of the AEL}$$

W.H. Bower
H.T. Begg

November 28, 1993

To: T. M. Kemp

From: J. F. Doughty

C-8 Environmental Status

EID079735

Reference: some title letter J. W. Raines to J. C. Bespenba
10/21/93.

I have recently reviewed the above letter and am concerned that the information presented at the meet does not adequately reflect the real situation.

(1) The information on the front page of the attach letter from Joe Lochiavo was given to Raine by phone prior to the meeting. Based upon the limited data available, Joe and I have concluded that a small amount of C-8 is found in the blood of those not working in Teflon,

but it seems to come to equilibrium in less than a year. The attached chart "New Hires - Teflon®" gives the impression that C-8 continues to build up the longer people spend on the plant.

(2) C-8 in blood data has been obtained from Del Port employees at the Spruance plant under the assumption that they would have levels similar to those of our dispersion customers. The average concentration for 27 people was 0.02[illegible] The average concentration for non-Teflon® employees at Washington Works was 0.026 ppm. EID079736

Conclusion: If we have concern about exposures off-plant, we have an equal or greater problem with our customers. I made this point to Rick prior to the meeting and see no record of discussion of this information in the meeting.

6/13/84

PERSONAL & CONFIDENTIAL

TO: H. V. BRADLEY
D. G. WIKA
T. M. KEMP
T. L. SCHRENK

FROM: J. A. SCHMID

C-8 IN WATER
**********

BELOW IS A COMPARISON BETWEEN THE 3/15/84 SAMPLES, AND THOSE TAKEN ON 6/4/84.

| | C-8 IN PPB | |
|---|---|---|
| LOCATION | 3/15/84 | 6/4/84 |
| ******** | ********* | ******** |
| LITTLE HOCKING | 0.8 | NOT DETECTED |
| LUBECK WATER | | |
| * POWELLS STORE | 1.2 | 1.0 |
| * LUBECK HILL | NOT SAMPLED | 1.5 |

I WOULD HAVE TO CONCLUDE THAT IN THE CASE OF LITTLE HOCKING, THE C-8 DROPPED BELOW THE DETECTION LIMIT OF 0.6 PPB. ALSO, RECOGNIZING THAT WE ARE OPERATING AT THE EXTREME OF THE TEST'S SENSITIVITY, THE BEST WE CAN SAY ABOUT THE LUBECK WATER IS THAT IT IS PRESENT IN THE 1.0 PPB RANGE.

REV.6/14/84 - 6/13/84 DATA WAS REPORTED AS FLUORINE NOT C-8.



EID079102

PERSONAL AND CONFIDENTIAL

CC: T. A. Foster

June 14,1984

TO: J. A. Schmid

FROM: J. F. Doughty

UPDATE on C-8 IN WATER SAMPLES

The attached table shows the C-8 in water data including the most recent data. I conclude the new data confirm the original data.

1. The Du Pont data shows that the test does not see C-8 up river and the sampling system does not contaminate the sample.

2. The second Washington sample had essentially the same C-8 content as the first.

3. The new Lubeck sample shows essentially the same concentration as the Washington sample. Thus the Washington sample is from the Lubeck Water System as I suspect or at least the Lubeck system has the same concentration.

4. The original Little Hocking sample was very close to the detection limit for the test. The concentration now appears to be below the detection limit.

I do not plan to do additional sampling unless further information is needed. The concentrations are very low and in my judgement are not cause for concern.



EID079271

| LOCATION | DISTANCE (MILES) | SIDE | ppb C-8*** |
|---|---|---|---|
| PARKERSBURG | 7.5 up stream | WV | ND |
| DU PONT | 0.5 up stream | WV | |
| (3/15/84) | | | ND |
| (6/4/84) | | | ND |
| DISTRIBUTION CENTER OF PARKERSBURG | 0.25 down stream* | WV | ND |
| WASHINGTON | 0.25 down stream | WV | |
| (3/15/84) | | | 1.2 |
| (6/4/84) | | | 1.0 |
| LUBECK | 0.25 down stream | WV | |
| (6/4/84) | | | 1.5 |
| LITTLE HOCKING | 3 down stream | OHIO | |
| (3/15/84) | | | 0.8 |
| (6/4/84) | | | ND |
| BELLEVILLE | 12 down stream | WV | ND |
| REEDSVILLE | 14 down stream | OHIO | ND |
| RAVENSWOOD | 29 down stream | WV | ND |
| RACINE | 50 down stream | OHIO | ND |
| POINT PLEASANT | 74 down stream | WV | ND |
| ** GALLIPOLIS | 79 down stream | OHIO | ND |

*well is back from the river

**first community to take water directly from the river

***values obtained from Experimental Station multiplied by 1.5 to convert to C-8 vs F content originally reported

ND = below the detection limit of 0.6 as C-8 (0.4 as F)

000095

DuPont

# INTEROFFICE MEMORANDUM

Date: 12-May-1987 03:06pm EST
From: TONY PLAYTIS
PLAYTIS
Dept: TEFTECH
Tel No: 2775

TO: ROGER ZIPFEL ( ZIPFEL )

CC: JOHN CRUM ( CRUM )

Subject: C8 In Water

Attached is a copy of the analytical report for our five water samples, which are identified as follows.

#1 - Washington Works drinking fountain, B3.

#2 - Powell's General Store, Washington WV

#3 - Lubeck Pennzoil, Lubeck WV

#4 - Mason's Village Market, Little Hocking OH

#5 - 812 20th Street, Vienna WV

C. L. Hill obtained samples 1-4 by driving to each location and asking to have a plastic bottle filled with drinking water. Sample 5 was taken by D. K. Moore at his home. All samples were taken on 3/13/87.

Note that the results are expressed as ppb F. When converted to ppb C8, the result of 1.3 ppb becomes 1.9 ppb. This result is higher than those from 1984, but considering how close we are to the detection limit of the test, the difference is probably not significant.

EID079091

Polymer Products Department
Research & Development Division
Experimental Station

cc: M. A. Kaiser - 256
S. R. Laas - 256
M. Lombarski - 269
B. S. Shepard - 323
G. J. Sloan - 323
PRAL File - 256
I.C. - 323

ANALYTICAL REPORT

May 7, 1987

To: A. J. Playtis - PPD, Washington Works

From: M. J. Vilone and R. M. Vasta - PPD, ESL 269

mgv QMV

PERFLUOROOCTANOATE (C8) IN WATER
(Job No. 870-441; PRAL Nos. 87-2933 - 2937, Notebook No. E44875)

Five samples of water have been analyzed for perfluorooctanoate (C8) by electron capture gas chromatography. Method ES-567 was used with the following modifications: sample size was 10 g; lyophilization was ~18-20 hours; concentration of perfluorodecanoate internal standard was decreased 10 fold. Spiked standards at concentrations of 0.4, 0.5, 0.8, 1.0 and 1.9 ppb were examined. A reproducible detectable peak was observed for 0.4 ppb and we have used this as our detection limit. No C8 peak was detected in the spiked standards <.4 ppb. For the quantitation we had linear calibration curves over the range of 0.4 to 1.9 ppb. The samples were freeze dried, derivitized, and analyzed in duplicate. The results are expressed as ppb fluoride where ppb F = 0.688 x ppb perfluorooctanoate.

The results are given in the attached table. If you have any questions, don't hesitate to call.

gmn
Attachment

Keywords:
GC
Perfluorooctanoate
Water

EID079092

000189

Perfluorooctanoate in Water

| PRAL | Designation | ngF/g. $H_2O$ (ppb) [a] |
|---|---|---|
| 87-2933 | #1 | n.d. |
| 87-2934 | #2 | ~~1.3~~ 1.9 as C-8 |
| 87-2935 | #3 | 1.3 1.9 as C-8. |
| 87-2936 | #4 | n.d. |
| 87-2937 | #5 | n.d. |

[a] n.d. = none detected; detection limit = 0.4 ppb
.58 as C-8



EID079093

DuPont

INTEROFFICE MEMORANDUM

Date: 4-May-1987 02:09pm EST
From: TONY PLAYTIS
PLAYTIS
Dept: TEFTECH
Tel No: 2775

TO: ROGER ZIPPEL ( ZIPPEL )

Subject: C8 in Water

The following results have been received by phone; a letter will follow by the end of the week. The detection limit of the test is 0.4 ppb.

| Sample | ppb C8 |
|---|---|
| Washington Works drinking fountain, B3 | <0.4 |
| Powell's General Store, Washington WV | 1.3 |
| Lubeck Pennzoil, Lubeck WV | 1.3 |
| Mason's Village Market, Little Hocking OH | <0.4 |
| 812 20th Street, Vienna WV | <0.4 |

RJZ009211

000102

EID079094

000103

# C-8 REDUCTION & CONTROL PROGRAMS

## STATUS

R. J. ZIPFEL

6-11-87

003546-19 - 06/09/87 - RJZ:kst

EID079685

# WASHINGTON WORKS

## OFF PLANT EMISSIONS

| | POUNDS C-8 | |
|---|---|---|
| | 1983 | 1986 |
| WATER | 21,700 | 19,100 |
| AIR | 12,700 | 16,200 |
| PRODUCT | 8,000 | 8,300 |
| OFF-PLANT DISPOSAL | 7,700 | 8,300 |
| | 50,100 | 51,900 |

003546-10611 - 06/03/87 - RJZ:kst

EID079694

# C-8 LEVELS IN DRINKING WATER

| | DISTANCE FROM NO. 5 OUTFALL (miles) | | C-8 LEVEL – PPB | |
|---|---|---|---|---|
| | | | 1984 | 1987 |
| PARKERSBURG | 7.50 | UPSTREAM | ND | ND |
| WASH. WORKS | 0.50 | UPSTREAM | ND | ND |
| LUBECK WATER DISTRICT | 0.25 | DOWNSTREAM | 1.1 | 1.9 |
| | 0.25 | DOWNSTREAM | 1.5 | 1.9 |
| LITTLE HOCKING | 3.00 | DOWNSTREAM | 0.6 | ND |
| RAVENSWOOD | 29.0 | DOWNSTREAM | ND | -- |

ND = NOT DETECTABLE

003546-7 - 06/10/87 - RJZ:kst

EID079695

009106

# SUPERNATE POND ELIMINATION

BENEFIT -- ELIMINATE C-8 CONTAMINATION OF LUBECK WATER SYSTEM AQUIFER FROM THESE PONDS

STATUS -- PROJECT APPROVED ($320M) 11/86

-- PERMIT APPROVAL 3Q87

-- START-UP 11/87

003546-33 - 06/10/87 - RJZ:kst



EID079701

E. I. du Pont de Nemours & Company
P.O. Box 1217
Parkersburg, W. Va. 26102

Polymer Products Department

CC: R. V. Mihailovich, 3WM53
U.S. EPA Region III
841 Chestnut Bldg.
Philadelphia, PA 19107

Greg Henger, Acting Supvr.
WV Div. of Water Resources
6321 Emerson Avenue
Parkersburg, WV 26101

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

June 15, 1987

Mr. David W. Robinson, Chief
WV Division of Water Resources
1201 Greenbrier Street
Charleston, West Virginia 25311

Attention: Industrial Waste Section

Dear Mr. Robinson:

This letter is to comply with Section D.1, Page 21 of NPDES Permit WV0001279, and Section D.1, Page 17 of WV Permit IW-6238-82, informing you of planned physical changes at our Plant and a minor change in discharge from outlets 002 and 005. The changes planned were reviewed in a telephone conversation with Mr. Randy Sovic of your staff and are as follows:

1) Relocate the No. 006 outlet sample location,

2) Add Calgon H-900G microbiocide to our Process Water wells on Blennerhassett Island to control iron bacteria, and

3) Close three anaerobic digestion ponds.

The details of the planned changes are shown in Attachments 1, 2 & 3.

Please call me on 863-4271 if you have any questions.

Very truly yours,

A. C. Huston
Environmental Control Consultant
Washington Works

ACH:hcw
Attachments

1573H



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ATTACHMENT 3

Page 1 of 3

# CLOSE ANAEROBIC DIGESTION PONDS

## Background

Three anaerobic digestion ponds located on the Ohio River banks are used for containment and treatment of some non-hazardous aqueous waste from our fluoropolymer production facilities. The waste includes water, inert suspended solids and a non-ionic detergent, and is held in the ponds to anaerobically digest the detergent.

The three ponds have no outlet and are operated so they do not overflow. This requires shipment of digested waste off-site to another Du Pont location for further treatment and ultimate disposal. Since the cost of disposal of the waste stream has been increasing rapidly and about 50% of the material shipped is rainwater, a storage tank is being constructed for the waste to significantly reduce the volume. It is anticipated that two ponds will be closed immediately, with the third being closed by the end of the year when tank construction is complete.

## Description

1) The ponds are of earthen construction, approximately 6 feet deep with sloped earthen banks about 22 feet wide at the base. All dike walls were reconstructed in 1975 or 1976 with red clay. Bentonite was mixed in with the clay before replacing the walls. Bentonite was also used to seal leaks in the bottom of the upriver pond.

2) Page 3 of this attachment (WWSK-13675) shows the location, size and elevation of the three impoundment units.

3) The combined volume of the ponds is estimated to be about 3 million gallons.

## Closure Plan

1) The contents of the digestion ponds will be pumped into rail cars and shipped off-site for disposal.

2) Visible lumps of polytetrafluoroethylene (inert solids) are to be hand shoveled into 55-gallon steel drums and landfilled at Letart.

3) The bottom of the ponds will then be removed, including sludge and discolored soil, to a depth of 3 to 4 feet below the bottom of the ponds.

1573H-6

ATTACHMENT 3



## CLOSE ANAEROBIC DIGESTION PONDS (Cont'd.)

4) The sludge and earth removed from the ponds will be disposed of in our Dry Run landfill (Permit IWL-6282-82).

5) It is then planned to push the earthen walls inward, fill with clean soil, level, and plant grass on the leveled area.

### Sludge Test Results

Analysis of a composite sample obtained from twelve locations within the eastern most (up river) pond showed the following results:

COMPOSITE SAMPLE COMPOSITION (DRY WEIGHT BASIS)

| | |
|---|---|
| Fluoropolymers | 13.6% |
| Triton® | 9.7% |
| Ammonium perfluorooctanoate (FC-143) | 85 ppm |
| Zinc* | 47 ppm |
| Inorganic fluoride | 26 ppm |
| Chloride | 20 ppm |

*insoluble, at about background level

The sludge tested non-hazardous by the RCRA EP toxicity test as shown below:

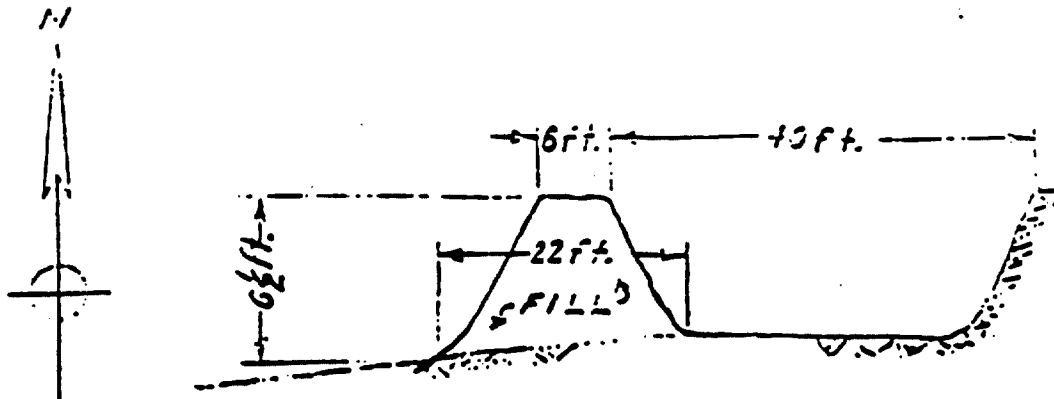
EP TOXICITY TEST

| Metal | Concentration (ppm) | RCRA Limits (ppm) |
|---|---|---|
| Arsenic | 0.006 | 5.0 |
| Barium | <1 | 100.0 |
| Cadmium | <0.1 | 1.0 |
| Chromium | <0.5 | 5.0 |
| Lead | <0.5 | 5.0 |
| Mercury | <0.005 | 0.2 |
| Selenium | <0.05 | 1.0 |
| Silver | <0.1 | 5.0 |

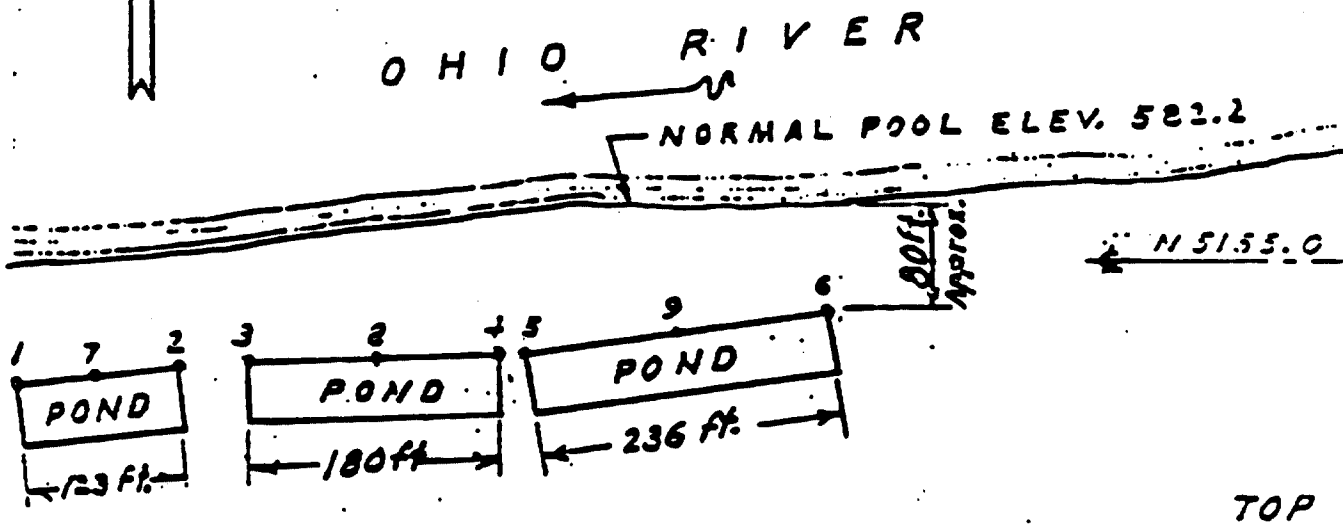
1573H-7

ATTACHMENT 3
Page 3 of 3

THIS DRAWING HAS BEEN FURNISHED BY E. I. Du. ANT DE NEMOURS AND CO. THE INFORMATION AND KNOW-HOW THEREON MAY NOT BE USED NOR THE DRAWING REPRODUCED WITHOUT THE WRITTEN PERMISSION OF DUPONT. ALL REPRODUCTIONS IN WHOLE OR IN PART, INCLUDING VENDORS SHOP DRAWINGS, SHALL BEAR OR REFER TO THIS STAMP.




TYPICAL PROFILE




| STA. NO. | LONGITUDE | LATITUDE | STA. NO. | TOP ELEV. L.P. |
|---|---|---|---|---|
| 1 — | 81° 40' 19.3" | 39° 16' 18.4" | 7 — | 601.91 |
| 2 — | 81° 40' 18.1" | 39° 16' 18.5" | 8 — | 600.25 |
| 3 — | 81° 40' 17.6" | 39° 16' 18.5" | 9 — | 593.4+ |
| 4 — | 81° 40' 15.8" | 39° 16' 18.7" | | |
| 5 — | 81° 40' 15.7" | 39° 16' 18.7" | Elev. Ref. 533.1 | |
| 6 — | 81° 40' 13.3" | 39° 16' 18.8" | of top of screen | |
| | | | wall WW- 45230. | |

E. I. DUPONT DE NEMOURS & CO. — WASHINGTON WORKS

DWN. BY J. C...

DATE 2/...

WWSK-...

# DIKE ELEVATIONS & COORDINATES ANAEROBIC DIGESTION PONDS

P-400

cc: T. M. Kemp
G. R. Alms/S. V. Gangal
J. G. Loschiavo - D12022-3
J. E. Crum
D. H. Flensborg
G. L. Herridge
J. F. Kline
R. D. Lanyon
W. A. Ott
J. L. Post
D. A. Schneider
W. M. Stewart

July 7, 1987

TO: C. A. DYKES
J. A. SCHMID

FROM: R. J. ZIPFEL

C-8 CONTROL PROGRAM

My review on this issue with Dr. Bruce Karrh on June 11, 1987, went very well with little change required to our present control program. Dr. Karrh was most interested in the presence of C-8 outside the plant boundaries. He stated that we need to place the highest priority on these environmental issues. Dr. Karrh also accepted the position that our employees will continue to have C-8 in their blood. My charts and the specific comments made during the review are attached.

The following is a restatement of the specific program items in our C-8 control plan with the necessary action steps indicated.

I. ADMINISTRATIVE CONTROLS

A. EQUIPMENT DESIGN

1. IMPLEMENT AN IMPROVED FINE POWDER DRYER GASKET DESIGN (RESP - KLINE)

B. PROTECTIVE EQUIPMENT

1. AUDIT WASHINGTON WORKS C-8 IN BLOOD AND C-8 IN AIR DATA TO DETERMINE IF CURRENT PROTECTIVE EQUIPMENT (AND OPERATING PROCEDURES) ARE THE OPTIMUM AVAILABLE (RESP - LOSCHIAVO)

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RJZ026552

C. C-8 MONITORING

1. REDUCE EMPLOYEE BLOOD TESTING TO ONLY THE FOLLOWING JOBS:

   - RAW DISPERSION AUTOCLAVE OPERATOR
   - DISPERSION OPERATOR
   - FINE POWDER DRYER OPERATOR
   - ENVIRONMENTAL OPERATOR (SUMP)
   - DISPERSION PACKOUT OPERATOR
   - GRANULAR POLYKETTLE OPERATOR
   - FINE POWDER PACKOUT OPERATOR
   - FEP POLYKETTLE OPERATOR
   - FEP WET FINISHING OPERATOR
   - FEP DISPERSION OPERATOR
   - FIRST LINE SUPERVISION OF ABOVE PERSONNEL

   (RESP - LANYON)

   NOTE: BEFORE WE CHANGE OUR BLOOD SAMPLING PROGRAM, WE WILL WANT TO HAVE AN INTERNAL BTO COMMUNICATION (RESP - ZIPFEL)

2. PERSONAL C-8 IN AIR SAMPLING - MONTHLY SAMPLES SHOULD BE REQUIRED OF THE FOLLOWING JOBS:

   - RAW DISPERSION AUTOCLAVE OPERATOR
   - GRANULAR POLYKETTLE OPERATOR
   - FINE POWDER DRYER OPERATOR
   - DISPERSION OPERATOR
   - FEP POLYKETTLE OPERATOR
   - FEP WET FINISHING OPERATOR
   - FEP DISPERSION OPERATOR

   (RESP - POLYMERS - OTT
   COPOLYMERS - POST)

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RJZ026553

3. AREA C-8 IN AIR MONITORING - AS NEEDED BY AREA AROUND CRITICAL EQUIPMENT (e.g., FINE POWDER DRYERS) AND TO RESPOND TO HIGH LEVELS NOTED WITH PERSONAL SAMPLES (RESP - POLYMERS - OTT
COPOLYMERS - POST)

NOTE: IN GENERAL, WE ARE TAKING FAR TOO MANY AREA SAMPLES AND NOT ENOUGH PERSONAL SAMPLES

4. DOCUMENTATION - EXPLANATIONS OF REASONS AND ACTION RESPONSE FOR AIR LEVELS (PERSONAL SAMPLES ONLY) ABOVE 50% OF THE AEL (0.56 MPB) REQUIRE IMPROVEMENT IN THE DIVISION'S ENVIRONMENTAL SAMPLING BOOKS (RESP - POLYMERS - OTT
COPOLYMERS - POST)

D. C-8 CONTROL LEVELS

1. ESTABLISH MAXIMUM SAFE C-8 IN BLOOD AND C-8 IN DRINKING WATER LEVELS (RESP - G. L. KENNEDY - HASKELL)

**NOTE: ONCE A SAFE LEVEL IS ESTABLISHED, THOSE PERSONNEL EXCEEDING 50% OF THIS LEVEL WILL BE REQUIRED TO BE REMOVED FROM THE EXPOSURE AREA**

II. PROCESS CHANGES

A. IMPLEMENT USE OF PURCHASED LIQUID C-8 AS IS BEING DONE AT DORDRECHT AND CHAMBERS WORKS (RESP - POLYMERS - CRUM
COPOLYMERS - HERRIDGE)

B. NEW SURFACTANTS

1. TBSA IN FEP - REDUCE PRIORITY ON THIS PROGRAM TO THE POINT WHERE BUSINESS NEEDS JUSTIFY FURTHER USE (QUALITY AND COST) (RESP - HERRIDGE)

2. C-9, C-10, C-12 - NO ENVIRONMENTAL LIMITS ARE PLACED ON THE USE OF NEW SURFACTANTS

C. C-8 RECOVERY

1. FINE POWDER DRYER VENT - DEVELOP BASIC DATA TO IDENTIFY RECOVERY PROCESS BY 4Q87 (RESP - SCHNEIDER)

2. FEP AQUEOUS STREAMS - APPLY ABOVE TECHNOLOGY IF ECONOMICAL (RESP - SCHNEIDER)

D. LUBECK WATER DISTRICT CONTAMINATION

1. ELIMINATE SUPERNATE PONDS - GOAL IS NOVEMBER 1987 (RESP - FLENSBORG)

2. DETERMINE MODE OF CONTAMINATION (RESP - STEWART)

RJZ026554

Attachments
003855-3 - RJZ:kst

EID091377

E. I. DU PONT DE NEMOURS & COMPANY
INCORPORATED
P.O. Box 1217
PARKERSBURG, W. VA. 26102

POLYMER PRODUCTS DEPARTMENT

CC: R. V. Mihailovich, 3WM53
U.S. EPA Region III
841 Chestnut Bldg.
Philadelphia, PA 19107

Greg Henger, Supervisor
WV Div. of Water Resources
6321 Emerson Avenue
Parkersburg, WV 26101

July 30, 1987

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Mr. David W. Robinson, Chief
WV Division of Water Resources
1201 Greenbrier Street
Charleston, West Virginia 25311

Attention: Industrial Waste Section

Dear Mr. Robinson:

Ref: Consolidated Permit WV/NPDES
Permit No. WV0001279
Permit Modification No. 5,
dated 7/7/87

This letter is to comply with Item 3 of the reference Permit Modification and Section D.1 of WV Permit No. IWL-6282-82, informing you of a planned physical change at our Plant which will result in a minor change in the waste material disposed of in Dry Run Landfill.

The material to be disposed of will be approximately 7100 tons of non-hazardous sludge and dirt from closing three anaerobic digestion ponds. The sludge test results are shown in Table 1 attached. Transportation of the sludge and dirt will be by road in open-top trailers.

Please call me on 863-4271 if you have any questions.

Very truly yours,

A. C. Huston
Environmental Control Consultant
Washington Works

/hcw
Attachment

1660H-1

JMM003564



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TABLE 1

## SLUDGE TEST RESULTS

Analysis of a composite sample obtained from twelve locations within the eastern most (up river) pond showed the following results:

### COMPOSITE SAMPLE COMPOSITION (DRY WEIGHT BASIS)

| | |
|---|---|
| Fluoropolymers | 13.6% |
| Triton® | 9.7% |
| Ammonium perfluorooctanoate (FC-143) | 85 ppm |
| Zinc* | 47 ppm |
| Inorganic fluoride | 26 ppm |
| Chloride | 20 ppm |

*insoluble, at about background level

The sludge tested non-hazardous by the RCRA EP toxicity test as shown below:

### EP TOXICITY TEST

| Metal | Concentration (ppm) | RCRA Limits (ppm) |
|---|---|---|
| Arsenic | 0.006 | 5.0 |
| Barium | <1 | 100.0 |
| Cadmium | <0.1 | 1.0 |
| Chromium | <0.5 | 5.0 |
| Lead | <0.5 | 5.0 |
| Mercury | <0.005 | 0.2 |
| Selenium | <0.05 | 1.0 |
| Silver | <0.1 | 5.0 |

1660H-3



EID007535

Medicine
Health Physics
Industrial Hygiene
Toxicology
Medical Department/3M

3M Center
St. Paul, Minnesota 55144
612/733 1110

3M

FAX (612)733-1773

FACSIMILE TRANSMISSION INFORMATION SHEET

Date NOV 30, 1987 Number of pages 4

Attention Gerry Kennedy or R.W. Rickard, Ph.D.

Location HASKELL LAB

Fax No. 302-366-5207

Special Instructions or Information:

ORIGINATOR'S NAME Peggy S. Perkins

Telephone number to verify complete transmission, or to relate problems: 612 733-3222



EID071428

000001

3M Medical Department
Medicine
Health Physics
Industrial Hygiene
Toxicology

Building 220-2E 02 3M Center
St Paul Minnesota 55144-1000
612 733 1110

3M

November 30, 1987

Robert G. Geil, D.V.M.
3030 South 9th Street
Kalamazoo, MI 49009

Dear Dr. Geil:

As a follow-up to our recent telephone conversation regarding the "Two Year Oral (Diet) Toxicity/Carcinogenicity Study of Fluorochemical FC-143 in Rats," we are writing to you to grant permission to discuss the report with Greg Sykes, D.V.M. of Haskell Laboratory for Toxicology and Industrial Medicine.

The enclosed materials include Dr. Sykes' October 29, 1987 memo to Charles Reinhardt, M.D. regarding the study. A copy of the 3M report was provided to G. L. Kennedy, Jr. at Haskell. Sykes' memo should serve as a guide to the matters he wishes to discuss with you. I should point out in passing that Dr. Sykes apparently did not realize that the primary interpretation of the data in the report was made by you, C. D. King, D.V.M., Ph.D. and J. L. Allen, Ph.D. I am not certain whether the people at Haskell are aware that you and Dr. King were external consultants retained by 3M Toxicology Services/Medical Department and 3M Riker Laboratories, respectively, to prepare the report.

Additionally, enclosed are the first 31 pages of the report that was issued in August 1987. The postmortem findings begin on page 14. The findings regarding Leydig cell adenomas are on page 18. The discussion section begins on page 19 and the Leydig cell adenomas are discussed on page 22. If you would like a copy of the entire four volume report, please let me know and we will send it.

To save you searching through your files, selected pages of the "final draft report," mailed to you with a May 12, 1986, cover letter from C. Chesney, D.V.M., Ph.D., are also enclosed. This enclosure includes pages 15, 16, 22 and 23.

We discussed the statistical analysis of the incidence of Leydig cell adenomas in comparison to the incidence in 954 control rats at Haskell, 880 control rats in Charles River CD® Rat, 24 month published data and the incidence noted by you for 610 control male rats at IRDC. I think the data of these three control groups are similar and seem to support the fact that it is quite

unusual to find no Leydig cell adenomas in male rats of 24 months age as was the case in the FC-143 study. The Haskell data is attached for your information. I believe you have the Charles River report since you brought it to our attention.

Enclosed also is an extract from a trip report prepared by S. V. Elrod, Ph.D., 3M Riker Laboratories. The information presented at the meeting is certainly of interest as regards the Leydig cell adenomas noted in the FC-143 high dose animals.

As discussed with you by telephone, we feel that any further review of slides from this study and any additional sectioning of tissues should be done under your guidance. We thank you for assistance in this matter.

We trust you will submit your statement for fees for services in connection with this additional consultation in the same manner as before.

In summary, please feel free to discuss this report with Dr. Sykes. If you need any further documents from our files, please let me know. Thank you again for your assistance.

Sincerely,

Roger G. Perkins

Roger G. Perkins, Ph.D.
Senior Toxicology Specialist
Diplomate of the American Board of Toxicology

RGP:bh (TS105 2.8)

Enclosures:
1) Memo Greg Sykes, D.V.M. to Charles Reinhardt, M.D. October 29, 1987
2) Two Year Oral (Diet) Toxicity/Carcinogenicity ... first 31 pages of Volume 1 of 4
3) Extracted pages from May 12, 1986 draft
4) Letter Gerry Kennedy to Roger Perkins October 16, 1987
5) Extract of Memo S. V. Elrod to E. H. Erickson November 17, 1987

dc: G. L. Kennedy, Jr.
Haskell Laboratory
Elkton Road, P.O. Box 50
Newark, DE 19714

C. D. King, D.V.M., Ph.D.
Xenos
P.O. Box 5008
North Branch, NJ 08876

EID071430

STATE OF WEST VIRGINIA
DEPARTMENT OF NATURAL RESOURCES
DIVISION OF WATER RESOURCES
1201 Greenbrier Street
Charleston, West Virginia 25311

ARCH A. MOORE, JR.
Governor

RONALD R. POTESTA
Director

ROBERT K. PARSONS
Deputy Director

December 17, 1987

Mr. A. C. Huston
E. I. du Pont de Nemours & Company
Incorporated
P.O. Box 1217
Parkersburg, WV 26102

PLANT FILE COPY
DO NOT REMOVE

RE: Water Pollution Control Permit
No. IWL-6282-82
Dry Run Landfill (Wood Co.) WV
Modification No. 1

Dear Mr. Huston:

This serves as Modification No. 1 for the above referenced facility. This modification is granted pursuant to your letters of July 30, 1987 and September 15, 1987 requesting permission to dispose of approximately 7,100 tons of non-hazardous sludge and dirt, which will result from the closure of three (3) anaerobic digestion ponds at the Washington Works facility.

Should you have any questions regarding the above information, please do not hesitate to contact John Britvec, Geologist at (304) 366-5880.

Very truly yours,

D. W. Robinson

D. W. Robinson, Chief

DWR/jb/me

cc: Greg Henger, Supv.
Cindy Musser, Insp.

CF001520

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000135

Z-49 REV. 3/81

E. I. DU PONT DE NEMOURS & COMPANY
INCORPORATED
WILMINGTON, DELAWARE 19898

POLYMER PRODUCTS DEPARTMENT

CC: D. G. WIKA, PPD
W. H. MARTIN, FIBR
R. W. MICHAUD, FIBR
K. D. DASTUR, C&P
R. Z. FORTNEY, INTL
D. P. GLEASON, PPD

April 27, 1988

TO: O. L. DARBY, DU PONT TOKYO
S. HAYASHI, SHIMIZU WORKS, JAPAN
G. L. KENNEDY, HASKELL LAB
R. D. LANYON, WASHINGTON WORKS
R. J. ZIPFEL, WASHINGTON WORKS
A. E. MORRIS, CHAMBERS WORKS
K. J. HUISMAN, DORDRECHT
J. P. BOLLMEIER, EXPERIMENTAL STA.
T. W. OLCOTT, FAYETTEVILLE

FROM: H. A. SMITH H.A. Smith

AMMONIUM PERFLUOROOCTANOATE (C-8) FOLLOW-UP MEETING

Ref: H. A. Smith to R. D. Lanyon, et al, 2/29/88

A meeting to review our progress and path forward will be held:

Date: Wednesday, June 29, 1988

Time: 8:00 a.m. - 4:30 p.m.
Lunch will be provided

Place: Kent Room
Hotel du Pont

We will be discussing:

- Strategy for managing C-8 in the workplace.
- Strategy for managing C-8 in the community.
- Future program regarding C-8 in blood - monitoring and goals.

Please come prepared to discuss and share information on the following:

1. Your personnel air monitoring results grouped by jobs.
2. Practices implemented to minimize skin contact.

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EID079641

3. A summary of your C-8 in blood data if you have any. Summarize as follows:

   o %+ Number of employees at a level of 0.5 ppm or less.

   o %+ Number of employees at a level of 1 ppm or less.

   o %+ Number of employees at a level of 5 ppm or less.

   o %+ Number of employees at a level of 10 ppm or less.

   o %+ Number of employees at a level of above 10 ppm.

   Do not overwork your blood data. If it is of questionable value or too sparce to be of real value, do nothing with it. We do not need to over focus on this subject at this time.

4. The CEG (Community Exposure Guideline) for C-8 is 0.3 ug/m$^3$. Does your site meet this guideline?

5. Any information available on C-8 in groundwater, public waters, etc.

A copy of 3M's latest MSDS and Toxicity Summary Sheet dated February 1988 reflecting the results of their long-term (two-year) rat feeding study is attached.

HAS/is
Attachment

AJP002268



EID079642

INTEROFFICE MEMORANDUM

Date: 29-Aug-1988 03:43pm
From: ANTHONY J. [TONY] PLAYTIS
PLAYTIS
Dept: PPD-SPD
Tel No: 304-863-2775

TO: Roger J. Zipfel ( ZIPFEL )

CC: JOHN E. CRUM ( CRUM )
CC: WALTER M. STEWART ( STEWART )

Subject: Test Results - C8 In Groundwater

Mary Jane Vilone at ESL telephoned the results listed below for the water samples we submitted in May as part of our monitoring program. She did this before writing the usual report because she was surprised by the very high levels seen in the Letart samples.

| Sample Description | C8 Level |
|---|---|
| B212 Drinking Fountain 5/12/88, 14:00 | none detected |
| Lubeck Water - Playtis Home 5/12/88, 17:00 | 2.2 ppb |
| Little Hocking Water - Ritenour Home, 5/12/88 | none detected |
| Test Well #27 5/11/88, 13:00 | 1.5 ppb |
| Letart Upper Pond 5/10/88, 11:00 | 1.6 ppm |
| Letart Lower Pond 5/10/88, 11:00 | 2.3 ppm |

RJZ009207

000130

EID079090

INTEROFFICE MEMORANDUM

Date: 30-Jan-1989 01:18pm
From: ANTHONY J. [TONY] PLAYTIS
PLAYTIS
Dept: PPD-SPD
Tel No: 304-863-2775

TO: Roger J. Zipfel ( ZIPFEL )

CC: JOHN E. CRUM ( CRUM )
CC: WALTER M. STEWART ( STEWART )

Subject: Test Results - C8 In Water

The following results have been received from ESL for the November samples for monitoring C8 in local water sources. They had a terrible time getting their analysis to give the necessary level of sensitivity, but they are confident that they now know what caused their difficulties.

| Sample Description | ppb C8 |
|---|---|
| B212 Drinking Fountain 11/2/88, 15:45 | none detected |
| Test Well #27 11/4/88, 08:30 | 1.3 |
| Lubeck Water - Playtis Home 11/2/88, 17:00 | 1.4 |
| Little Hocking Water - Ritenour Home, 11/7/88, 06:00 | contaminated sample, analysis not possible |

EID079089

000132

INTEROFFICE MEMORANDUM

Date: 31-May-1989 10:54am
From: WILLIAM E CRAWLEY
CRAWLEWE
Dept: PPD-"TEFLON"TECH
Tel No: 304-863-2742

TO: ANTHONY J. [TONY] PLAYTIS ( PLAYTIS )

Subject: C-8 Sampling

We have decided that it is not necessary to continue sampling the Lubeck system for C-8 to see the impact of the Supernate Pond closure. Effective immediately, we will not need to take any more sampling from the Lubeck system. Any Lubeck samples that are being held for analysis should be discarded to save the sampling cost.

We do want to continue to sample Water Well No. 27 on a quarterly basis to monitor the impact of the Supernate Pond closure. We should also continue to take water samples around ? Dupont, Letart Landfill on a monthly basis.

Please call me if you have any questions. Thanks, Bill.

RJZ009200



EID079087

H. V. BRADLEY
WORKS MANAGER
E. I. DU PONT DE NEMOURS & CO., INC.
WASHINGTON WORKS PLANT
P. O. BOX 1217
PARKERSBURG, WV 26102

June 12, 1989

STANDBY PRESS RELEASE

Du Pont's purchase of the Lubeck Public Service District (LPSD) facility on Rector Lane is progressing satisfactorily.

The LPSD initiated the transaction in late 1986, offering sale to Du Pont to facilitate relocation of the LPSD's well field to a larger location to meet their forecast increased demands. After careful study, Du Pont agreed to purchase the wells to obtain additional process water and real estate to facilitate current and future Plant operations.

The Plant has provided the LPSD with information to include in its response to a request from the West Virginia Public Service Commission for groundwater analytical data. Information has been provided from testing that has taken place in compliance with requirements of the permitting process of the Resource Conservation and Recovery Act (RCRA). This includes a test well that is the Plant's well closest to the LPSD wells. Additionally, since the mid-1970s, samples have been taken to ensure that Plant operations are not adversely affecting water quality. We believe that the data indicate that the water is safe and reliable.

-more-



EID079154

Both the LPSD and Washington Works agree that Du Pont's purchase of the LPSD wells will benefit both LPSD customers and Du Pont. The sale of the property will enable the LPSD to make a transition to its new facility more quickly and to reduce the cost increase to its customers. Benefits to the Plant include additional real estate adjoining the warehouse where products are stored, improving traffic flow and alleviating congestion around the warehouse. The water wells located on the property will also help fill the Plant's increasing needs for process water. Additionally, the purchase will allow the Plant to delay expansion of its well system on Blennerhassett Island.

EDITORIAL CONTACT: H. D. RAMSEY
PLANT SUPERINTENDENT
WASHINGTON WORKS PLANT
304-863-2739

EID079155

Questions and Answers
(Background Information for Plant Media Contacts)

. Q: What is FC-143?

A: It's an ammonium salt of a fluorinated carboxylic acid. It is a surfactant--a detergent-like material.

2. Q: What does Washington Works use FC-143 for?

A: It is used in the manufacture of Teflon*. End uses of Teflon* include applications in the fields of medicine, space, the semiconductor industry, and non-stick cookware.

3. Q: Is FC-143 harmful?

A: The issue is concentration--how much and when. Animal studies with rats have demonstrated that it is slightly to moderately toxic. In these animal studies, there was an indication of liver toxicity. The Material Safety Data Sheet (MSDS) notes human skin irritations, tearing, and respiratory discomfort with over-exposures. However, there has been no adverse effect on employee health associated with FC-143 exposure.

4. Q: How long has Washington Works been using FC-143?

A: Since August of 1951, nearly 38 years.

5. Q: Where does FC-143 come from?

A: We purchase it from 3M Corporation, and there is a Material Safety Data Sheet (MSDS) available to you that gives a good summary of its properties.

6. Q: Why were you sampling for FC-143?

A: We began testing employees for FC-143 in 1979 when we were informed by 3M that it had been found to accumulate in human blood. We also initiated a program in 1983 to track FC-143 outside the areas where it was used.

7. Q: At the 1-2 ppb level, will it accumulate in the blood of those who drink it?

A: We do not have the information to confirm it, but it is our technical judgment that it would not accumulate at these low levels.



EID079156

8. Q: Why does it accumulate in the blood of humans?

A: We don't precisely know the mechanism. We do know that it does not readily decompose, react, or break down in the biological processes of the human body. It is expelled from the body slowly.

9. Q: What is the allowable safe limit in drinking water?

A: Du Pont has calculated a safe level of 5 ppb for drinking water. This was calculated by converting the daily dose based on Du Pont's AEL (acceptable exposure limit) for our plant workers, to an equivalent dose if exposure were to occur via the drinking of 2 liters of water per day. Appropriate reductions were then incorporated as safety factors.

10. Q: To what level has it been measured to accumulate in the blood of those who are exposed to it at the Plant?

A: We have detected levels up to 33 ppm, with a significant percentage at 5 ppm or lower. There has been no adverse effect on employee health at these levels.

11. Q: How often is the blood of employees monitored for FC-143?

A: Every other year for Washington Works employees who work with FC-143. Consistent with our occupational health standards, we also continue to monitor to affirm the adequacy of our personal protection practices and procedures.

12. Q: How did FC-143 get into the LPSD water system?

A: We believe it could have come from three on-site collection ponds. Those ponds were closed in 1988 and replaced with metal collection tanks. The waste is shipped to another Du Pont site for disposal. This change was a result of Du Pont's Corporate Environmental Policy.

13. Q: What are you doing to reduce FC-143 emissions from your plant?

A: We are currently in compliance with all regulatory requirements, but to comply with Corporate environmental direction, we have additional plans. We have a program to reduce air emissions going into operation during the last quarter of 1989 (at a cost of $3,800,000), and we are also studying means to remove FC-143 from wastewater.

14. Q: If the stuff is not harmful, why are you spending any money to reduce the air and water emissions?

A: It continues to be Du Pont's policy to minimize aqueous and air emissions by reduction of waste. Even though the material has no known ill effects, it is our intent to minimize exposure which could cause concern associated with accumulation in the blood.

15. Q: Is the presence of FC-143 in the LPSD water system the reason for Du Pont's purchase of LPSD wells and property adjacent to the Plant?

A: We would have made the purchase whether or not the FC-143 was present. We need an additional well water supply, and we will make good use of the additional real estate. Additionally, the purchase allows us to delay the expansion of our well system on Blennerhassett Island.

16. Q: What was detected in the water system?

A: A full scale of normal water tests--such as for hardness and chlorides—was conducted. Additionally, the water was analyzed for more than 240 compounds, using U.S. EPA monitoring requirements for groundwater as a reference, under the Resource Conservation and Recovery Act (RCRA). From the more than 240 materials that were analyzed, only trace quantities of 17 materials were detected. These results do not represent a concern.

7. Q: How long have you known FC-143 was present in the Lubeck Water ystem?

A: Since 1983.

18. Q: Why have you not made this information public knowledge until now?

A: At that time, we were not sure of the findings, since the level found was so low--close to the detection limit. We did not regard the level to be a health problem.

19. Q: Isn't this a female reproductive concern?

A: No. Initial tests by 3M with rats indicated a cause for concern, but more extensive testing demonstrated that the problem was not with FC-143, but with the original testing procedure.

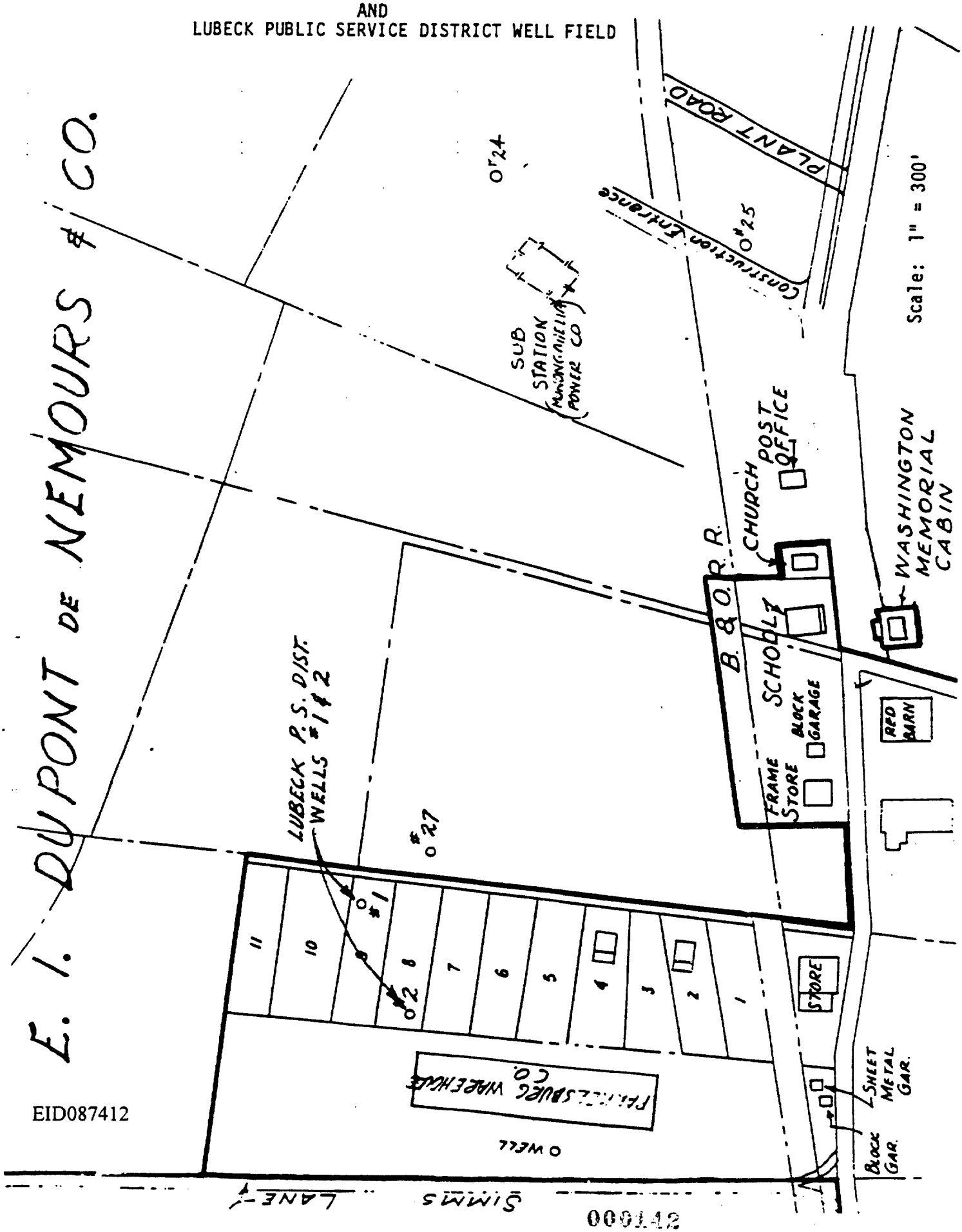
20. Q: What is Du Pont paying for the wells?

A: $1.8 million.

RJZ009762

LOCATION OF DU PONT WELL 27
AND
LUBECK PUBLIC SERVICE DISTRICT WELL FIELD

ATTACHMENT 1




EID087412

000142

Received 6-26-89

STATE OF WEST VIRGINIA
DEPARTMENT OF NATURAL RESOURCES
DIVISION OF WATER RESOURCES
1201 Greenbrier Street
Charleston, West Virginia 25311

GASTON CAPERTON
Governor

J. EDWARD HAMRICK III
Director

LARRY W. GEORGE
Deputy Director

June 21, 1989

PLANT FILE COPY
DO NOT [illegible]
File 2.303

Mr. A. C. Huston
E. I. DuPont de Nemours & Co., Inc.
P. O. Box 1217
Parkersburg, WV 26102

CERTIFIED RETURN RECEIPT REQUESTED

RE: West Virginia Solid Waste Management Regulations
Permit Numbers:
IWL-6282-82 and 3494

Dear Mr. Huston:

As you should be aware, the West Virginia Department of Natural Resources (DNR) filed Title 47, Series 38, "Solid Waste Management Regulations", which became effective on November 4, 1988 (on an emergency basis). This letter will serve as notification that your facility will now be governed by these regulations and to inform you of what you will be required to do to comply with these regulations.

The regulations specify that all solid waste disposal facilities must have a liner under the site and that the liner system must include a subbase, leachate detection zone, composite liner (compacted clay topped with a synthetic liner), and leachate collection with a protective cover zone. However, the regulations also acknowledge the fact that existing landfills may not have the specified liner system in place.

To allow for the continued operation of existing facilities, a provision has been included in the regulations whereby a permittee may petition the director to allow use of an alternate system. The petition must include a demonstration that the currently employed system will provide protection of groundwater resources.

The division wishes to begin reviewing these proposals as soon as possible. With this in mind, you are required to submit proposals for the groundwater protection demonstrations to the Division of Water Resources by July 31, 1989 using the Emergency Solid Waste Management Regulations. The results of your demonstration must be submitted with your application for permit renewal or Permit Application Addendum unless your permit expires within six (6) months of the date of this letter in which

instance this agency will entertain a request for a permit extension to allow a reasonable time period to complete the necessary demonstration. If your permit has already expired and all allowances exhausted for extension as provided by statute, you are advised to expeditiously submit your demonstrations. This agency will execute enforcement discretions in these instances.

The purpose of soliciting proposals now is to assure both you and the division that the demonstration will be acceptable prior to its initiation. The proposal should contain a compliance schedule for completion of the demonstration.

Should you require further information or assistance please contact Mr. John G. Britvec of my staff at (304) 366-5880.

Very truly yours,

Laidley Eli McCoy, Ph. D.
Chief

LEM:jbw



EID007494

INTEROFFICE MEMORANDUM

Date: 30-Jun-1989 03:51pm
From: ANTHONY J. [TONY] PLAYTIS
PLAYTIS
Dept: PPD-SPD
Tel No: 304-863-2775

TO: WILLIAM E CRAWLEY ( CRAWLEWE )

CC: JOHN E. CRUM ( CRUM )
CC: WALTER M. STEWART ( STEWART )
CC: Bill Tice ( TICE )
CC: Roger J. Zipfel ( ZIPFEL )

Subject: C8 In Water Test Results From ESL

| SAMPLE | PPB C8 |
|---|---|
| test well 27, 5/4/89 | 0.44 |
| P-12 drinking fountain, 5/8/89 | none detected |
| Lubeck Water (Playtis), 5/7/89 | 0.73 |
| Little Hocking Water (Ritenour) 5/4/89 | contaminated sample, analysis not possible |

| SAMPLE | PPM C8 |
|---|---|
| Letart upper pond, 11/23/88 | 4.2 |
| " lower " , " | 2.3 |
| " upper " , 12/30/88 | 0.96 |
| " lower " , " | 1.5 |
| " upper " , 1/24/89 | 3.2 |
| " lower " , " | 3.1 |
| " upper " , 2/28/89 | 1.2 |
| " lower " , " | 1.9 |
| " upper " , 3/17/89 | 1.1 |
| " lower " , " | 1.1 |
| " upper " , 4/25/89 | 0.49 |
| " lower " , " | 1.5 |

000117

EID079124

000113

INTEROFFICE MEMORANDUM

Date: 3-Aug-1989 08:31am
From: HAROLD BUMGARNER
BUMGARHE
Dept: Power & Services
Tel No: (304) 863-4796

TO: SEE BELOW

Subject: LOWER POND LEAKAGE DRY RUN

FOLLOWING OUR INSTRUCTIONS, BOSO DRAINED THE LOWER POND, THEN DUG INTO THE BANK TO LOCATE AND REPAIR THE LEAK. THIS METHOD OF REPAIR, USING BENTONITE CLAY WAS RECOMMENDED BY THE W. VA. SOIL CONSERVATION SERVICE. MELVIN DID THE WORK ON 8/2. THE PATH OF WATER THROUGH THE GROUND WAS VERY EVIDENT AND HE WAS ABLE TO DIG THROUGH IT AND PACK THE HOLE AND ADJACENT AREAS WITH THE CLAY (HE USED A TON OF BENTONITE). WE ARE CONFIDENT THE LEAK IS REPAIRED BUT PROOF WILL REQUIRE PERMITTING THE POND TO REFILL WITH RAIN WATER. THE AMOUNT OF ALGAE PRESENT, BASED ON APPEARANCE, IS REDUCED OVER WHAT WE HAD SIX WEEKS AGO. BUT WE DO SEE ALGAE SO BOSO WILL PUT A SMALL AMOUNT OF THE COPPER SULFATE ALGAECIDE IN THE POND AS IT FILLS.

WE HAVE ASKED BOSO TO REMOVE THE SETTLED SOLIDS FROM THE UPPER POND TO INCREASE ITS VOLUME. HE INTENDS TO DO THIS WORK OVER THE NEXT FEW DAYS.

YOAK TELLS ME THAT HIS SAMPLE REPORT FOR JULY WILL SHOW NO FLOW FROM THIS POND. THE AUGUST SAMPLE WILL INDICATE THE SUCCESS OF THESE MODIFICATIONS.

Distribution:

TO: FELIX DAVIS, JR. ( DAVISF )
TO: R J YOAK ( YOAKRJ )
TO: Billy D. Garner ( GARNER )
TO: ROBERT L. CARLSEN ( CARLSERL )
TO: ROGER C GAUL ( GAULRC )

CC: PENNY C MAHONEY ( MAHONEPC )
CC: WALTER M. STEWART ( STEWART )
CC: Arthur C. [Art] Huston, Jr. ( HUSTON )

EID014083

INTEROFFICE MEMORANDUM

Date: 12-Apr-1990 02:45pm
From: ANTHONY J. [TONY] PLAYTIS
PLAYTIS
Dept: PPD-ER
Tel No: 304-863-2228

TO: Charles T. Alt ( ALT )
TO: PENNY C MAHONEY ( MAHONEPC )
TO: WALTER M. STEWART ( STEWART )

Subject: C8 At Dry Run

Attached are results for various water samples taken at the Dry Run Landfill. The numbers refer to various stream sampling locations noted on my hand-drawn map. I asked Jim Yoak to take these samples because I was concerned about C8 leaching out of the material taken out of the Teflon* supernate pond sites. Since some of these results are nonzero, we may want to do some additional testing.



EID080167

INTEROFFICE MEMORANDUM

Date: 9-Apr-1990 08:50am
From: Peter D. Spohn
SPOHNPD
Dept: PPD-SPD
Tel No: 863-4732

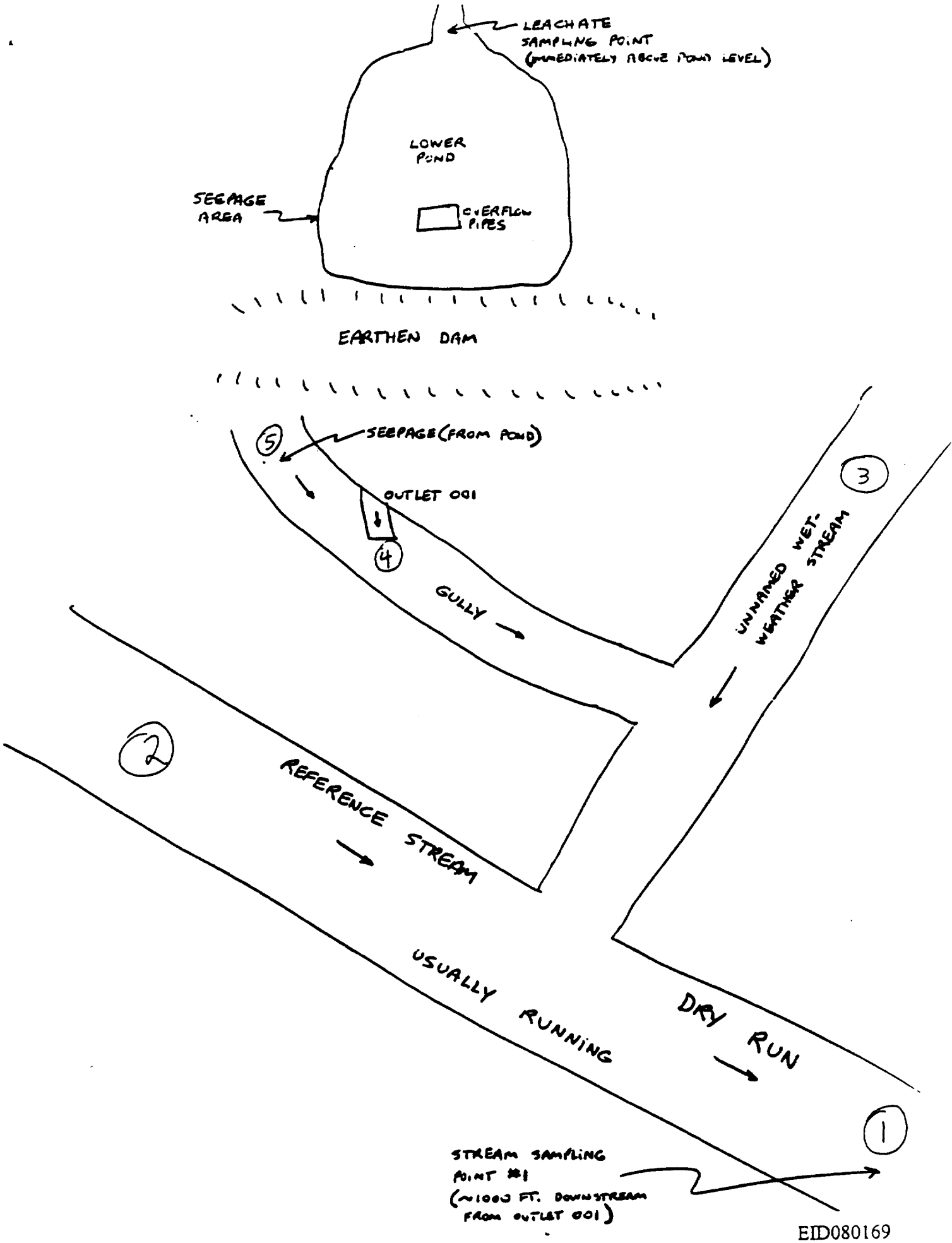
TO: ANTHONY J. [TONY] PLAYTIS ( PLAYTIS )

Subject: Dry Run C8 Analyses

Here are the results:

| Sample | ppm C8 |
|---|---|
| D.R.#1 | 1.6 |
| D.R.#2 | 0.0 |
| D.R.#3 | 0.0 |
| D.R.#4 | 0.6 |
| D.R.#5 | 0.4 |

These values were calculated from the area of the peaks.


LEACHATE SAMPLING POINT (IMMEDIATELY ABOVE POND LEVEL)
LOWER POND
SEEPAGE AREA
OVERFLOW PIPES
EARTHEN DAM
5
SEEPAGE (FROM POND)
OUTLET 001
4
GULLY
3
UNNAMED WET-WEATHER STREAM
2
REFERENCE STREAM
USUALLY RUNNING
DRY RUN
1
STREAM SAMPLING POINT #1 (~1000 FT. DOWNSTREAM FROM OUTLET 001)

EID080169

SUPERNATE SLUDGE TO LETART LANDFILL

(Letter, Stewart to Robertson, August 23, 1990)

BCC: J. B. Allen, Legal
C. F. Muska, Wilmington
C. C. Chien, Louviers
Route to:
M. S. Eaton
H. D. Ramsey
C. R. Campbell
F. Davis
L. W. Goin
L. K. Ireland
A. C. Sobrero
W. E. Crawley
T. L. Byrd
B. D. Garner
R. C. Gaul
Route to:
~~C. T. Ait~~
~~F. C. Rauch~~
P. C. Mahoney

- Do we have any TCLP tests and would they show any different?

We have sampled. Results should be back today or tomorrow

EID051924

E. I. DU PONT DE NEMOURS & COMPANY
INCORPORATED
P.O. Box 1217
PARKERSBURG, W. VA. 26102

POLYMER PRODUCTS DEPARTMENT

August 23, 1990

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Mr. Max Robertson, Acting Chief
Waste Management Section
WV Division of Natural Resources
1260 Greenbrier Street
Charleston, WV 25311

Re: Letter, Huston to Robinson, 7/30/87
Letter, Huston to Robinson, 9/15/87
Letter, Robinson to Huston, 12/17/87
NPDES Permit: WV0076244
NPDES Permit Application: WV0076066
Permit Number: 3495

Dear Mr. Robertson:

This letter is to inform you of our intent to relocate fluoropolymer contaminated sludge from our Dry Run landfill to the Letart landfill.

It is our site policy to segregate fluoropolymer waste from other plant wastes due to fire potential. In an effort to consolidate all of the fluoropolymer waste, we will be relocating approximately 7,000 tons of non-hazardous sludge and dirt from its current location at the Dry Run Landfill to the Letart Landfill in Mason County. This sludge is the only fluoropolymer waste known to have been disposed of in Dry Run. All other fluoropolymer waste has been disposed of in the Letart landfill. The sludge was generated during the closure of three fluoropolymer anaerobic digestion ponds during 1988. The movement of this soil to the Dry Run landfill was discussed with and approved by the DNR in the referenced letters (attached). We believe that the waste material to be relocated is covered under our existing Letart Landfill permit.

We plan on beginning this movement early in September, 1990. If you have any questions or comments, please, feel free to contact me on 863-4271.

Very truly yours,

W. M. Stewart

W. M. Stewart
Environmental Control Consultant
Washington Works

Attachments

EID051925



000156

INTEROFFICE MEMORANDUM
WASHINGTON WORKS
POLYMERS

March 27, 1991

TO:
C. R. Campbell
C. F. Muska
L. W. Goin
T. L. Byrd
A. J. Platiss
R. J. Yoak
H. D. Ramsey
W. M. Stewart
L. K. Ireland
M. G. McClusky
L. Williams

From: P. C. Mahoney PCM

QUARTERLY C-8 RESULTS AT DRY RUN LANDFILL

Attached is an updated figure showing the C-8 results for the fourth quarter, 1990. Due to a miscommunication, samples were not taken during the third quarter, 1990. Samples were taken during the first quarter, 1991 but the results have not been received.

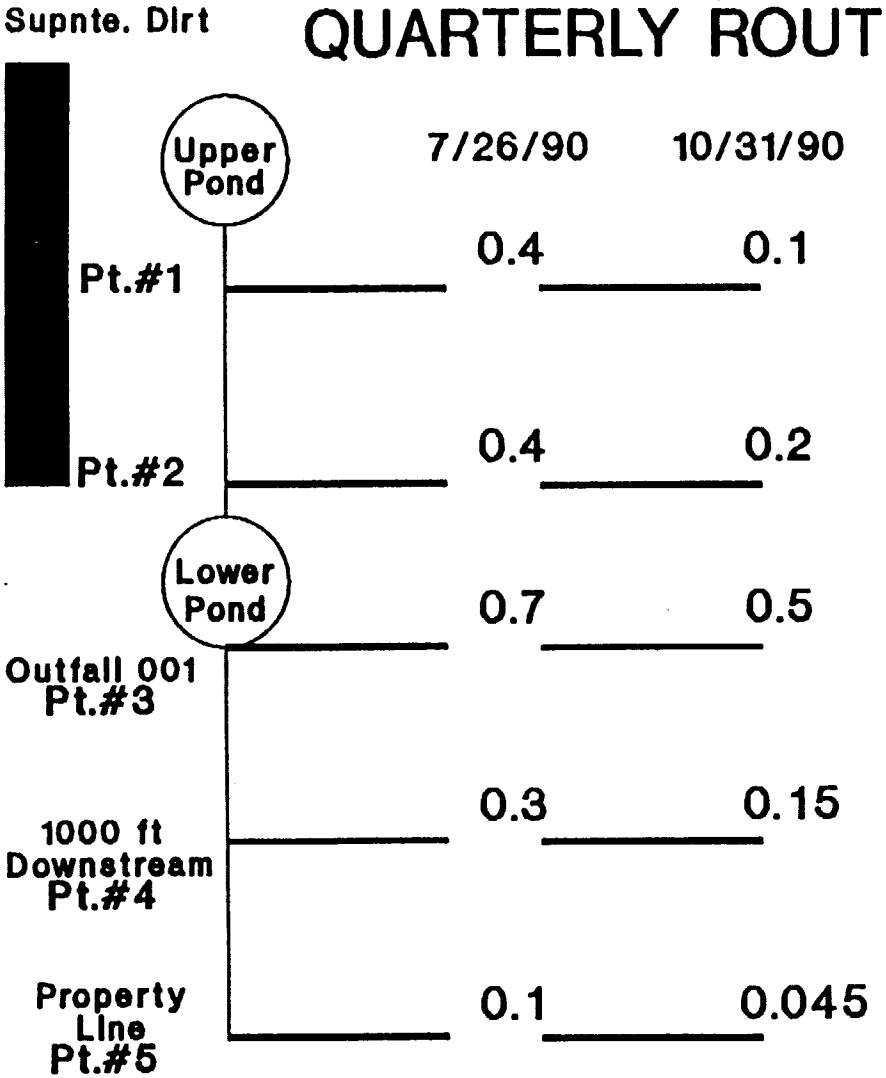
The levels of C-8 appear to be decreasing, but with just two data points it would be premature to assume this will be a continuing trend. I will issue the first quarter results as soon as they are available.

AJP001651



EID080172

# C-8 IN PPM - DRY RUN LANDFILL
# QUARTERLY ROUTINE SAMPLES





000159

EID080173

AJP001652

000150

W 7 5

## AIM

TO VIEW C-8 ISSUES AT WASHINGTON WORKS IN A WAY THAT ALL CONCERNS ARE ADDRESSED SO THAT C-8 CAN BE EFFECTIVELY MANAGED AT THE SITE.

## AGENDA

| ITEM | RESPONSIBILITY |
|---|---|
| INTRODUCTION | |
| • REVIEW OF AREAS AFFECTED | STEWART |
| • C-8 BALANCE FOR 1990 | MC CLUSKY |
| • AIR DISPERSION MODEL RESULTS | |
| CEG STATUS, C-8 IN WATER | STEWART |
| C-8 TEST STATUS | MC CLUSKY |
| RECOMMENDATIONS FOR DETERMINING SIZE OF PLUME FROM SUPERNATE PONDS | CHIEN<br>VANDELL |
| PLANS FOR TIEING IN LPSD WATER WELLS TO PLANT PROCESS WATER SYSTEM | IRELAND<br>GARNER |
| OTHER ISSUES | |
| • RELOCATION OF SUPERNATE SLUDGE FROM DRY RUN | MAHONEY |
| • LETART PERMIT | STEWART |

727

BACKGROUND
C-8

WHAT IS C-8?

- AMMONIUM PERFLUOROOCTANOATE
- FC-143
- FC-118 (20% SOLUTION)
- CAS NO. - 3825-26-1

WHAT IS IT USED FOR?

- DISPERSING AGENT - POLYMERIZATION (USED SINCE LATE 1950's OR EARLY 1960's)

WHERE IS IT USED?

- TEFLON) POLYMERS - ALL POLYMERIZATION BATCHES
- TEFLON) COPOLYMERS - FEP POLYMERIZATION

HOW IS IT RELEASED TO ENVIRONMENT?

- AIR - FINE POWDER AND FEP DRYERS
- WATER - DISPERSION & FEP SUPERNATE STREAMS
  - FLOAT TANKS
  - FEP COAGULATORS
  - POLYMERIZATION UNIT STREAMS
- LAND - SUPERNATE PONDS (NOW CLOSED)
  - SOLIDS (SOME WET) SENT TO LANDFILLS

HOW TOXIC IS IT?

- ACUTE - MODERATE
- CHRONIC - TREATED AS VERY TOXIC BECAUSE C-8 BIO-ACCUMULATES IN BLOOD
- AEL - 10 $\mu$G/M$^3$
  CEG - 0.3 $\mu$G/M$^3$

/hcw (4/15/91)
830



EID080798

## C-8 DETECTED

### WASHINGTON WORKS SITE

| | PPM |
|---|---|
| • SUPERNATE POND AREA | |
| - CONTAMINATED SOIL | 100 |
| - GROUNDWATER (-20 FT) | 100 |

| | PPB |
|---|---|
| • WEST END OF SITE | |
| - GROUNDWATER (-75 FT) | 2-3 |

### LETART LANDFILL

| | PPM |
|---|---|
| • SURFACE WATER | |
| - LEACHATE POND OVERFLOW | 2-3 |
| • GROUNDWATER | |
| - OLD WELL SAMPLE | 2-3 |

### DRY RUN LANDFILL

| | PPM |
|---|---|
| • SUPERNATE SLUDGE | 75-100 |
| • SURFACE WATER | |
| - UPPER POND | 2-3 |
| - SEDIMENTATION BASIN | 2-3 |
| - DISCHARGE STREAM AT PROPERTY LINE | .04-.2 |
| • GROUNDWATER | |
| - MONITOR WELLS | ND |

# C-8 IN DRY RUN
# MANAGEMENT PLAN

PROPOSAL NO. 1 - LEAVE SUPERNATE SLUDGE IN PLACE, STABILIZE THE C-8 TO RETARD MIGRATION BY COVERING WITH 2 FT NATIVE CLAY

PROS

- EASY TO DO - LESS EXPENSIVE
- NO TRUCKS ON HIGHWAYS
- NOT A COMMUNITY ISSUE AT LETART
- REDUCES SURFACE WATER CONTAMINATION LEVEL
- PERMIT MODIFICATION NOT AN ISSUE

CONS

- MAY NOT PROTECT GROUNDWATER
- SURFACE WATER CONTAMINATION MAY BE ABOVE CEG

PROPOSAL NO. 2 - REMOVE SUPERNATE SLUDGE FROM DRY RUN AND RELOCATE THE MATERIAL TO LETART

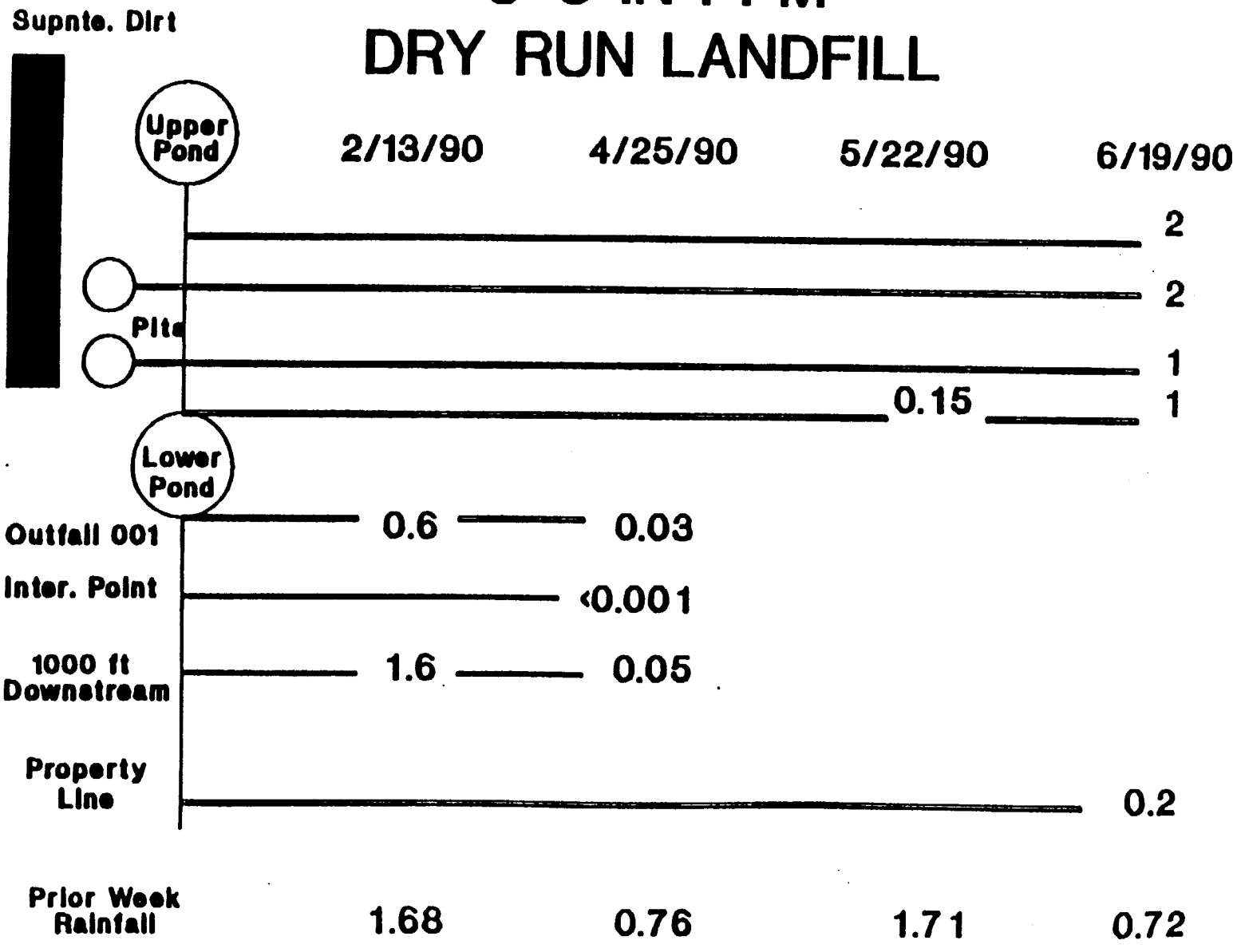
PROS

- REMOVE SOURCE FROM THE LANDFILL
- REDUCES SURFACE WATER CONTAMINATION LEVEL
- GROUNDWATER CONTAMINATION POTENTIAL IS REDUCED

CONS

- TRUCKS ON HIGHWAY
- COMMUNITY ISSUE AT LETART
- NEED PERMIT FROM WV-DNR
- SURFACE WATER MAY STILL BE ABOVE CEG
- GROUNDWATER MAY STILL BE CONTAMINATED
- COST EXPECTED TO BE 75-100M $
- OTHER PERMIT ISSUES AT LETART

# C-8 IN PPM
# DRY RUN LANDFILL




0001385

EID080801

GW006840

000166

# TEMPORARY OPERATING INSTRUCTIONS

TOI NO. 91-6

TITLE: Relocation of supernate sludge within Dry Run Landfill

AREA: Power and Services  Dry Run Landfill

Extent of work: 09/23/91 to 10/31/91

Objective: The objective of this work is to relocate the supernate sludge that was originally landfilled at Dry Run Landfill in 1988. The sludge will be moved from its current location to another location within the landfill. This new location will reduce the potential of leaching from the supernate sludge. All reasonable efforts will be made to completely remove the sludge from the existing location.

SAFETY: All safety rules now in effect at the Dry Run Landfill will remain in effect during execution of this T.O.I.

MSDS for FC-143 FLUORAD (C-8) is attached. (Attachment A-1)
MSDS for Triton X-100 is attached. (Attachment A-2)

PPE required during handling of the supernate material:

- Safety glasses with side shields
- Long sleeved shirts
- Rubber gloves

No smoking materials will be permitted in the vicinity of the supernate material.

Avoid breathing dust contaminated with the supernate material. If dust is generated during the excavation or movement of the sludge, the sludge should be sprayed with water to reduce the dust problem.

If the dusting problem persists, the Site Occupational Health Coordinator (A. J. Playtis) will be consulted.

RAK002923

EID024306

000137

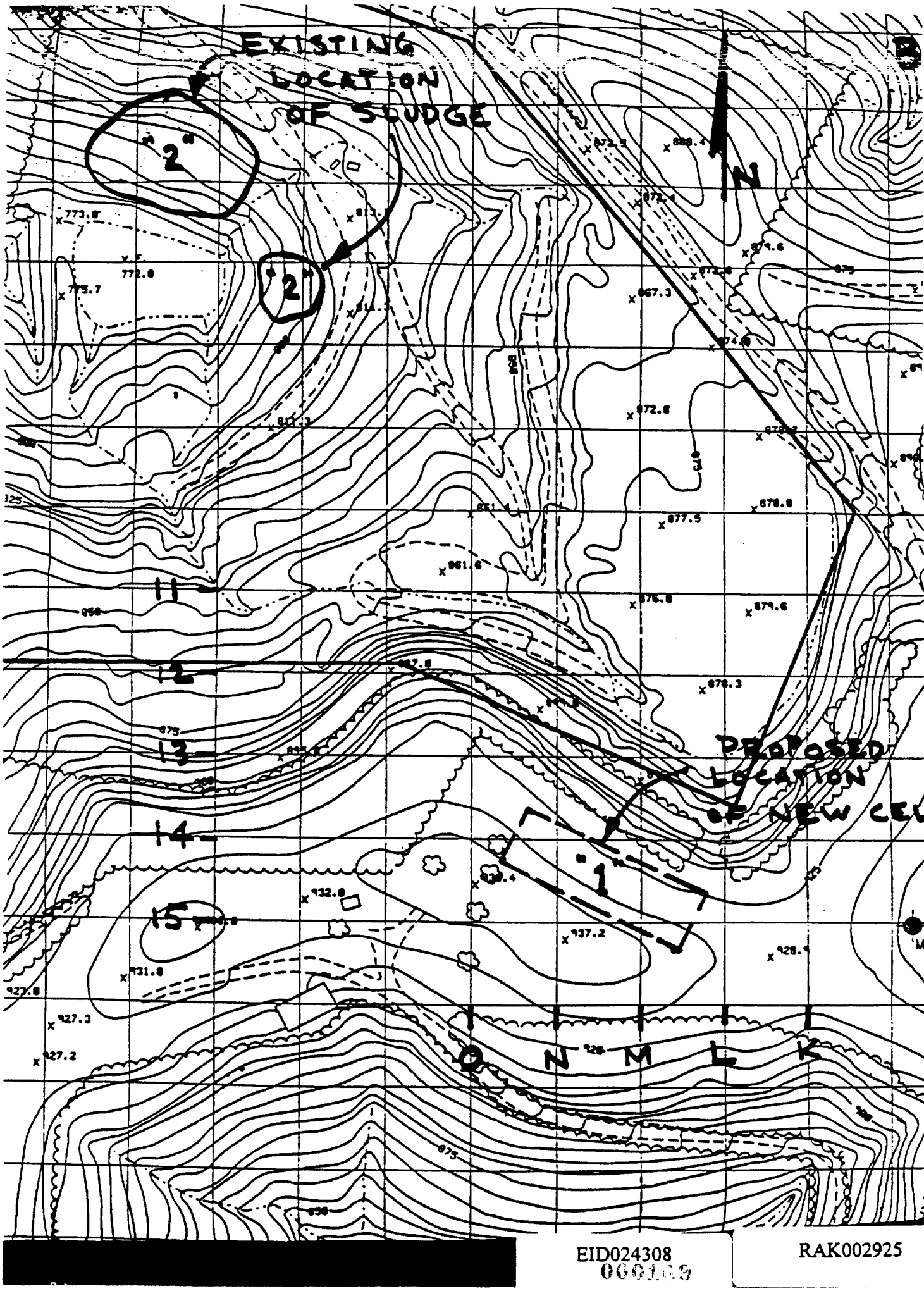
WWM516
JOEGASTON
SCALE: 1" - 50'

RAK002924



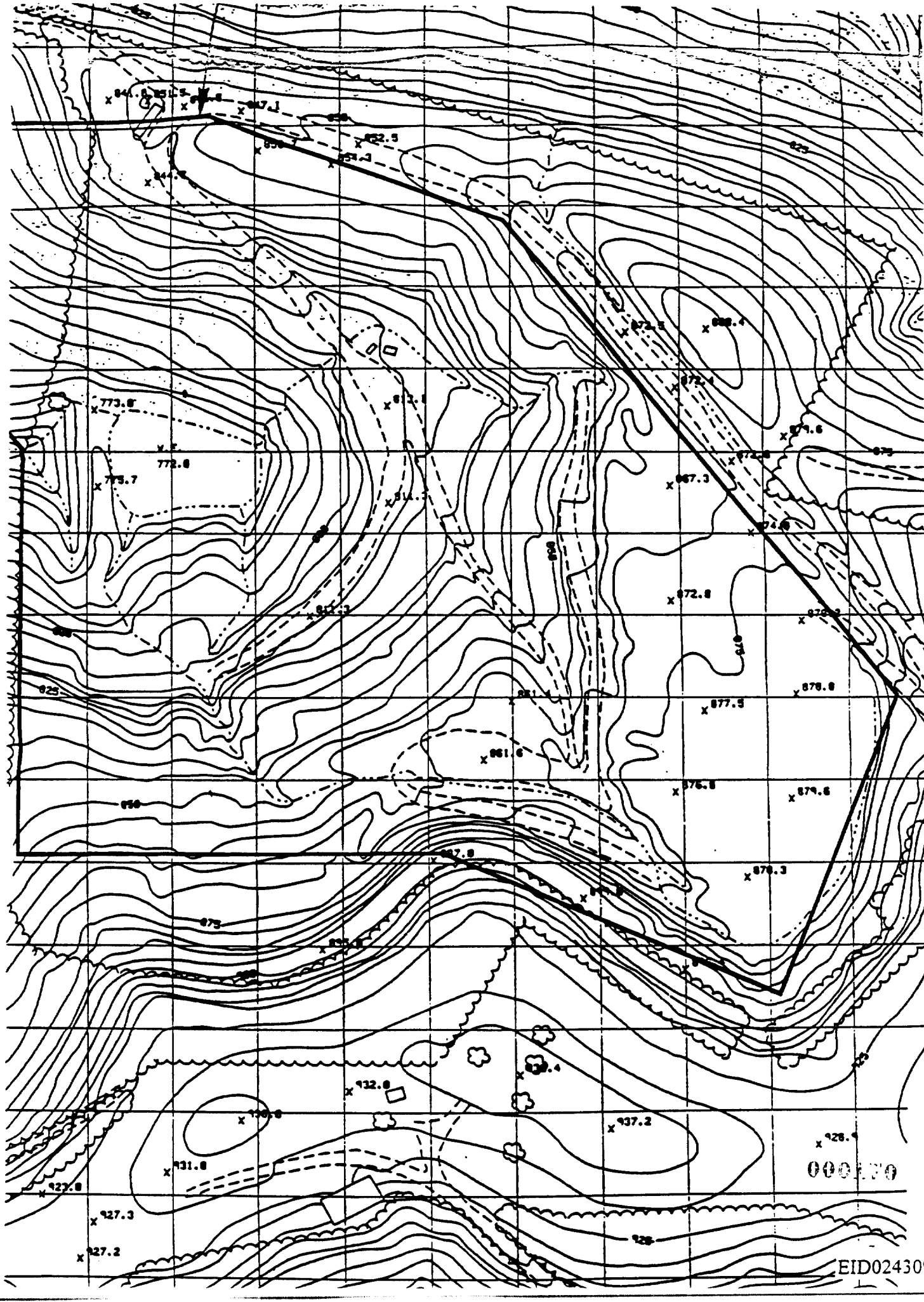

EID024307

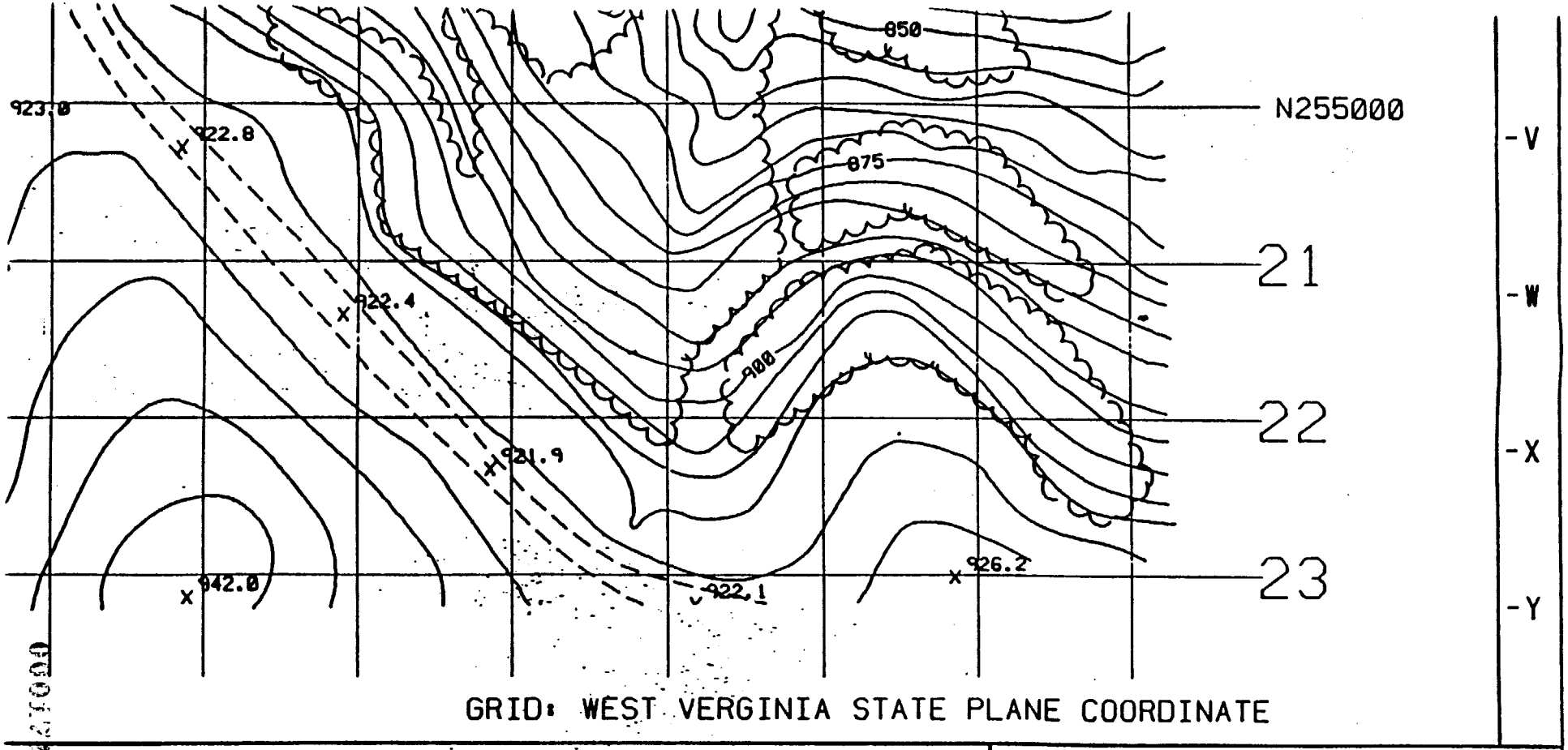
991600




EID024308

RAK002925


EID02430




# E.I. DUPONT DE NEMOURS & CO. INC.
# NON HAZARDOUS LANDFILL
# DRY RUN, WOOD COUNTY, W.VA.
# TOPOGRAPHIC MAP 1989

THIS DRAWING HAS BEEN FURNISHED BY E.I.DUPONT DE NEMOURS & CO. THE INFORMATION AND KNOW-HOW THEREON MAY NOT BE USED NOR THE DRAWING REPRODUCED WITHOUT THE WRITTEN PERMISSION OF DUPONT. ALL REPRODUCTIONS IN WHOLE OR IN PART, INCLUDING VENDOR'S SHOP DRAWINGS, SHALL BEAR OR REFER TO THIS STAMP.

| | | |
|---|---|---|
| SCALE | 1" = 100' | DATE |
| DRAWN BY | J. I. GASTON | 1-30-91 |
| UPDATED BY | J. I. GASTON | 5-1-91 |
| CHECKED BY | | |
| APPROVED BY | | |

| ELEC. CODE CLASS | WWJ NO. | FAA NUMBER | PROJ. NO. | WASHINGTON WORKS | DF |
|---|---|---|---|---|---|
| | | | | WW M516 | AR |

EID0C

RAK00927/

Downloaded by gastonji at ws_26 on Thu May 02,1991

Z-47 REV. 10-90

DUPONT

WASHINGTON WORKS
P.O. Box 1217
PARKERSBURG, WV 26102-1217

DU PONT POLYMERS

ATTORNEY - CLIENT
PRIVILEGED INFORMATION

cc: T. L. Eichstadt
L. W. Goin
M. M. McClusky
P. C. Mahoney
H. D. Ramsey
W. M. Stewart

October 3, 1991

CONFIDENTIAL

TO: J. B. ALLEN - D7156
W. P. ANDERSON - D12026
T. R. BARRY - N9442-1
C. C. CHIEN - L3359
W. D. COBB - D11090
M. S. DEAK - D12016
M. S. EATON - WW
J. C. LEITINGER - N7410
J. S. LINDELL - D11056
T. D. VANDELL - L33E63
D. M. VON SCHRILTZ - D11090

FROM: R. J. ZIPFEL - WW

C-8 IN WATER SAMPLE RESULTS
OHIO RIVER, SITE TEST WELLS, AND NEW LUBECK WATER SYSTEM

The site conducted an extensive sampling program of the Ohio River, plant site test wells and the new Lubeck Public Water System wells for the presence of C-8 in September, 1991. This program was to further analyze the new Lubeck Public Water System wells, to gain further information on the C-8 levels in the Ohio River and in the aquifer beneath the site, and to begin to qualify an outside Du Pont analytical laboratory for C-8 in water data (CH2M HILL).

The results of the sampling program showed the following:

1. Non-detectible levels of C-8 in the Ohio River upstream of the site.

2. C-8 levels at expected values in the Ohio River downstream of the site and in the aquifer beneath the site (old Lubeck Water Systems wells).

3. Seven (7) of eight (8) results from the new Lubeck Water Systems wells showed non-detectible levels of C-8. The eighth sample gave a C-8 result near the lower analytical detection limit.

The complete data package and path forward from this segment of work are attached. Any questions on these results or other items in this communication package should be directed to Penny Mahoney, Dave Ramsey or the writer.

RJZ:sal
Attachment

EID079110

PRIVILEGED & CONFIDENTIAL

## SAMPLING PROGRAM SUMMARY

- OVERVIEW OF SAMPLING RESULTS
- SUMMARY
- PATH FORWARD

950-1

## C-8 SAMPLING RESULTS

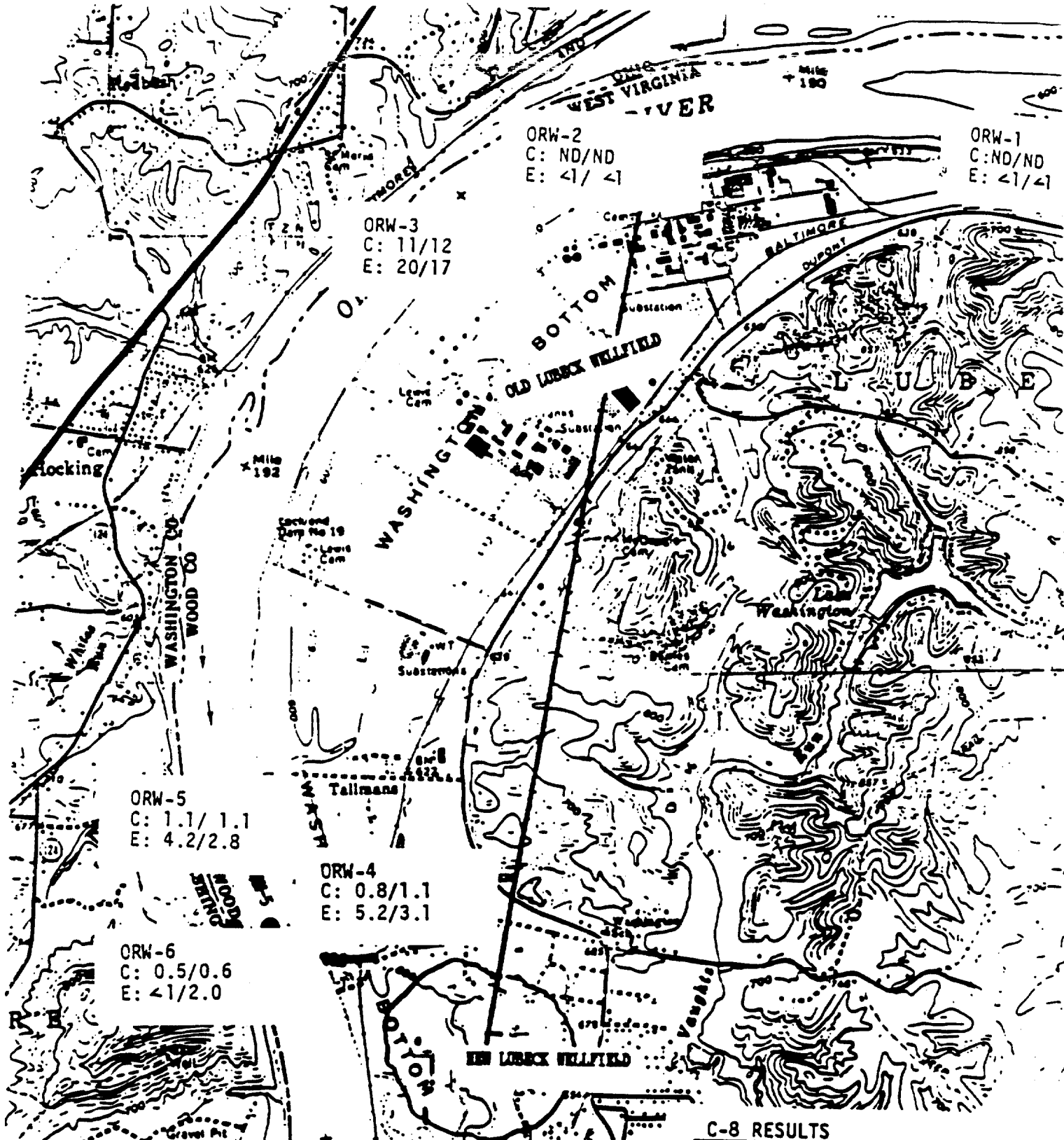
| | Experimental Station (PPB C-8) | $CH_2M$ Hill* (PPB C-8) |
|---|---|---|
| Test Well #27 | 7.1 | 1.2 2.5/3.2 |
| Well #331 | < 1 | 0.1 .6/.6 |
| Well MW-4 | 2.7 | 0.2 .7/.6 |
| Ohio River -1 | < 1 | Not Detected |
| Ohio River -1 Duplicate | < 1 | Not Detected |
| Ohio River -2 | < 1 | Not Detected |
| Ohio River -2 Duplicate | 3.9 | Not Detected |
| Ohio River -3 (Near Outfall) | 19.6 | 11.0 |
| Ohio River -3 Duplicate | 16.8 | 12.0 |
| Ohio River -4 | 5.2 | 0.8 |
| Ohio River -4 Duplicate | 3.1 | 1.1 |
| Ohio River -5 | 4.2 | 1.1 |
| Ohio River -5 Duplicate | 2.8 | 1.1 |
| Ohio River -6 | < 1 | 0.5 |
| Ohio River -6 Duplicate | 2.0 | 0.6 |
| New Lubeck Well -F | < 1 | Not Detected |
| New Lubeck Well -F Dupl | < 1 | Not Detected |
| New Lubeck Well -B | < 1 | 0.3 |
| New Lubeck Well -B Dupl | < 1 | Not Detected |
| Old Lubeck Well -1 | 8.8 | Destroyed |
| Old Lubeck Well -1 Dupl | 4.1 | 1.1 |
| Old Lubeck Well -4 | 4.2 | 0.9 |
| Old Lubeck Well -4 Dupl | 2.9 | 1.7 |
| TW 33 | | 75 46 |

Parcel
New LL Samples

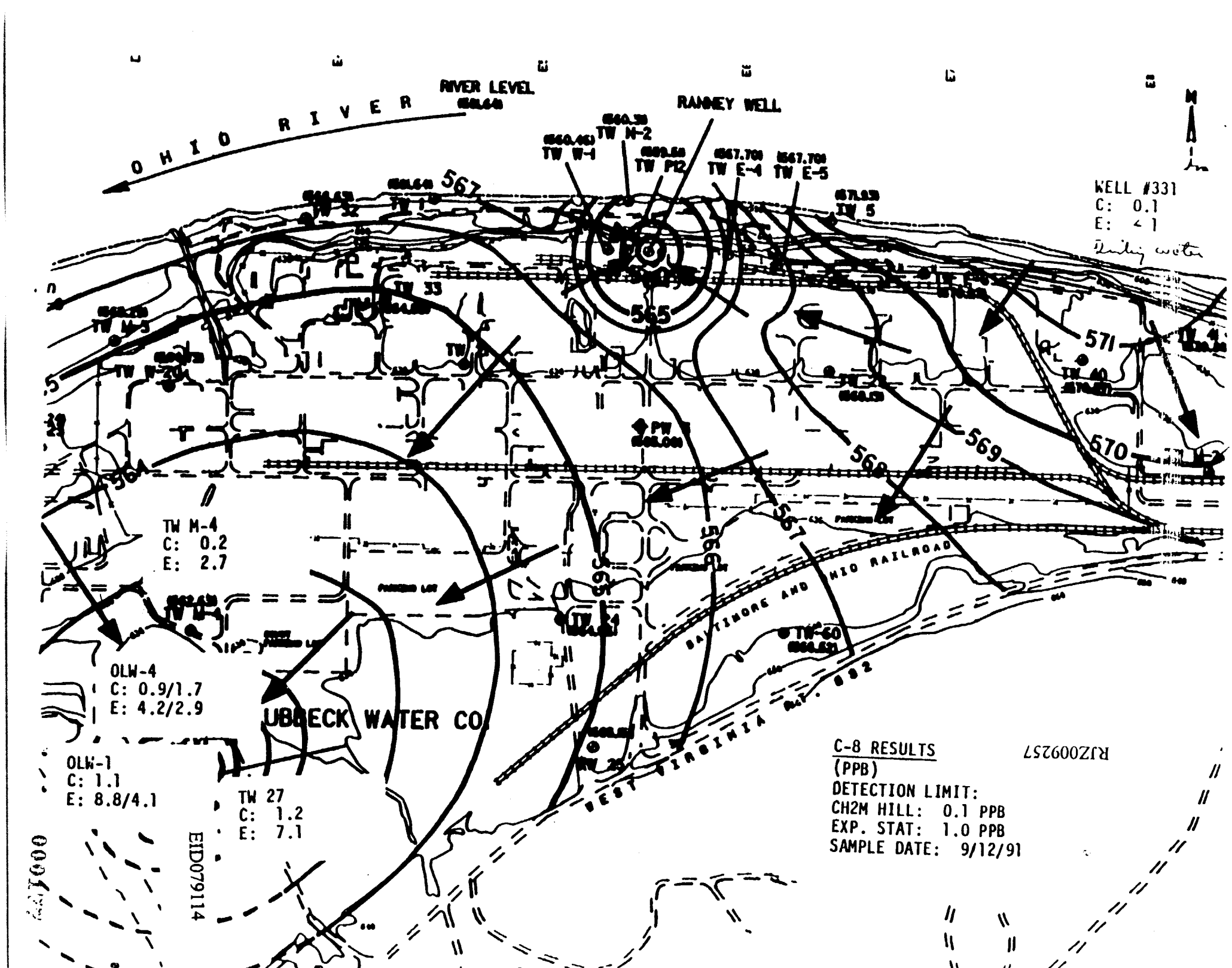
* $CH_2M$ Hill experienced analytical difficulties. Absolute numbers may be low.

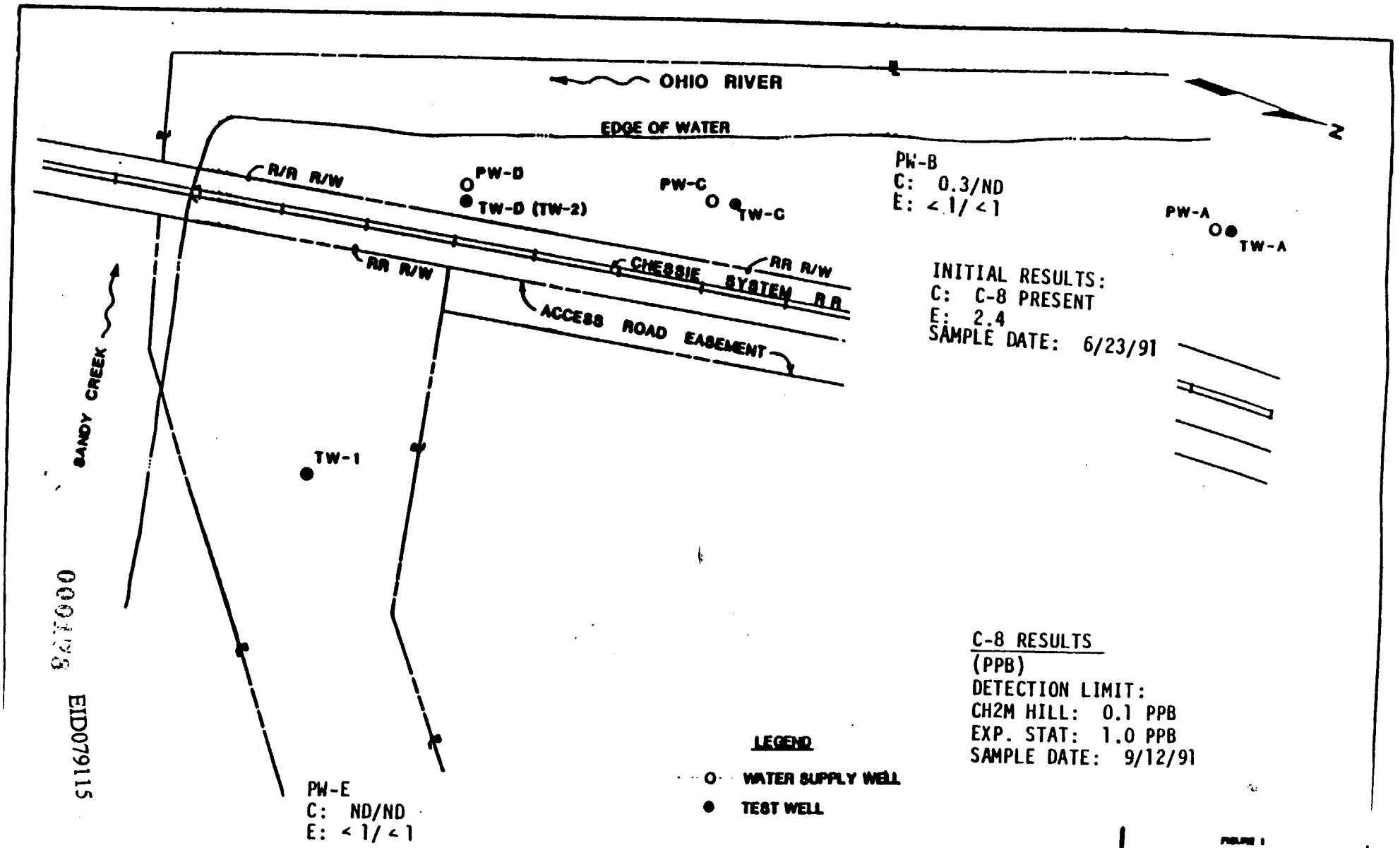
10/2/91
951

EID079112


ORW-2
C: ND/ND
E: <1/ <1
ORW-1
C:ND/ND
E: <1/<1
ORW-3
C: 11/12
E: 20/17
ORW-5
C: 1.1/ 1.1
E: 4.2/2.8
ORW-4
C: 0.8/1.1
E: 5.2/3.1
ORW-6
C: 0.5/0.6
E: <1/2.0
WEST VIRGINIA
OHIO RIVER
OLD LUBECK WELLFIELD
NEW LUBECK WELLFIELD
WASHINGTON CO
WOOD CO
Tallmans
Sandy
C-8 RESULTS
(PPB)
DETECTION LIMIT:
CH2M HILL: 0.1 PPB
EXP. STAT: 1.0 PPB
SAMPLE DATE: 9/11/91

EID079113


RIVER LEVEL
OHIO RIVER
RANNEY WELL
TW N-2
TW W-1
TW P12
TW E-4
TW E-5
TW 5
WELL #331
C: 0.1
E: < 1
TW 33
TW M-3
TW M-4
C: 0.2
E: 2.7
OLW-4
C: 0.9/1.7
E: 4.2/2.9
OLW-1
C: 1.1
E: 8.8/4.1
TW 27
C: 1.2
E: 7.1
UBBECK WATER CO.
TW-60
BALTIMORE AND OHIO RAILROAD
WEST VIRGINIA
565
567
568
569
570
571
C-8 RESULTS
(PPB)
DETECTION LIMIT:
CH2M HILL: 0.1 PPB
EXP. STAT: 1.0 PPB
SAMPLE DATE: 9/12/91
RJZ009257
EID079114


OHIO RIVER
EDGE OF WATER
N
R/R R/W
PW-D
TW-D (TW-2)
PW-C
TW-C
PW-B
C: 0.3/ND
E: < 1/ < 1
PW-A
TW-A
RR R/W
RR R/W
CHESSIE SYSTEM RR
ACCESS ROAD EASEMENT
INITIAL RESULTS:
C: C-8 PRESENT
E: 2.4
SAMPLE DATE: 6/23/91
SANDY CREEK
TW-1
PW-E
C: ND/ND
E: < 1/ < 1
C-8 RESULTS
(PPB)
DETECTION LIMIT:
CH2M HILL: 0.1 PPB
EXP. STAT: 1.0 PPB
SAMPLE DATE: 9/12/91
LEGEND
WATER SUPPLY WELL
TEST WELL
NOTE: WELL DESIGNATION IN PARENTHESIS IS CROSS REFERENCED TO WELL AS IDENTIFIED WHEN DRILLED IN 1985.
FIGURE 1
SITE MAP
BURGESS & NIPLE, LTD
ENGINEERS & ARCHITECTS
MAY 1988

EID079115

## TOXICOLOGY

- LIMITS

| | ug/m$^3$ | ppb |
|---|---|---|
| - TLV | 100 | 5.6 |
| - AEL | 10 | 0.56 |
| - $CEG_A$ | 0.3 | 0.0015 |
| - $CEG_W$ | - | 1.0 |

- CALCULATED DOSE

| | |
|---|---|
| - AEL | 100 ug/day |
| - CEG | 6 ug/day |

# C-8 DETECTED

| WASHINGTON WORKS SITE | PPM |
|---|---|
| • SUPERNATE POND AREA | |
| - CONTAMINATED SOIL | 100 |
| - GROUNDWATER (-20 FT.) | 100 |
| • WEST END OF SITE - WELLS 27 & 4 | PPB |
| - GROUNDWATER (-75 FT.) | 2-4 |
| • NO. 5 OUTFALL | |
| - DISCHARGE TO OHIO RIVER | 40 |

| WASHINGTON WORKS EMPLOYEES | PPM |
|---|---|
| • 1991 (VOLUNTARY TESTING) | UP TO 5 |

| DRINKING WATER (LPSD) | PPB |
|---|---|
| • 1984 - 1989 | 0.7 - 2.2 |
| • 1991 | 3.8* |

| LETART LANDFILL | PPM |
|---|---|
| • SURFACE WATER | |
| - LEACHATE POND OVERFLOW | 2-3 |
| • GROUNDWATER | |
| - WELL SAMPLES | 0.05 - 0.7 |

| DRY RUN LANDFILL | PPM |
|---|---|
| • SUPERNATE SLUDGE | 75-100 |
| • SURFACE WATER | |
| - UPPER POND | 2-3 |
| - SEDIMENTATION BASIN | 2-3 |
| - DISCHARGE STREAM AT PROPERTY LINE | 0.04 - .2 |
| • GROUNDWATER | PPB |
| - MONITOR WELLS | 2-5 |

*Revised analytical method

EID079259

## GROUNDWATER ISSUES

1) PLANT SITE

- ROUTINE SAMPLE RESULTS
- VERIFICATION INVESTIGATION

2) LETART LANDFILL

- LINER DEMONSTRATION
- SITE OVERVIEW
- PROPOSED STRATEGY

3) DRY RUN LANDFILL

- DETECTED IN DOWNGRADIENT WELLS

@C Please for C-8 Alternate @ L

INTEROFFICE MEMORANDUM

Date: 25-Jan-1993 08:13am
From: TONY L EICHSTADT
EICHSTTL
Dept: POLY
Tel No: 304-863-2531

TO: MAYNARD S EATON, JR ( EATONMS )
TO: H. David Ramsey,, Jr. ( RAMSEY )

Subject: C-8 ALTERNATE

EID087668

000184

HDR001553

INTEROFFICE MEMORANDUM

Date: 22-Jan-1993 12:23pm
From: JAMES R LAWSON
LAWSONJR
Dept: PPD
Tel No: 304-863-4618

TO: Distribution List

Subject: Great News on Surfactant

Bruce, this is terrific news. The favorable reports on both acute toxicity and bioaccumulation combined with prior work showing this surfactant should be suitable for FEP means that, for the first time, we have a viable candidate to replace C8 in much of "Teflon". Even better, the candidate would be produced internally, improving our overall cost position.

We have been waiting on this toxicity assessment before commencing additional polymerization testing. I think the focus of work should now switch to our organization. Obviously, we will need pure material for testing. Can you provide us with pound quantities?

The focal point for our internal efforts should be Roger Zipfel. Please contact him to develop our path forward.

...Jim

INTEROFFICE MEMORANDUM

Date: 21-Jan-1993 09:55pm
From: Bruce E. Baker
BAKERBE AT A1 AT JLCL01
Dept: DuPont Chemicals
Tel No: 540-3490

TO: Hsu-Nan Huang ( HUANGH AT A1 AT JLCL01 )
TO: K. Spencer Prowse ( PROWSEKS AT A1 AT JLCL01 )

CC: A. Charles Sobrero ( SOBRERAC AT A1 AT WWPS )
CC: James R. Lawson ( LAWSONJR AT A1 AT WWPS )

Subject: C6-TBS: the rest of the story

Folks:

Gerry Kennedy has finished his bioaccumulation testing on C6-TBS, and I have some good news, and I have some even better news.

If you'll recall, the good news (I've attached the previous memo, which explains it in detail) was that C6-TBS was between 32 and 53 times less bioaccumulative in the liver of male mice than was 3M's C8 product, in side by side comparison testing. As I said then, that was only half the picture; it said that C6-TBS was not as readily eliminated from the bloodstream into the liver, but could not say whether it was simply remaining in the bloodstream (and hence, still bioaccunulating).

The better news is that the blood work on the mice is finished. A detailed report on the overall picture is forthcoming, but I wanted you all to be aware of the information Gerry shared with me today.

Essentially, what was done was to feed 3 groups of mice meals spiked with varying levels of C6-TBS and C8 (the third group was a "control" group). We were then to allow up to 28 days of recovery, during which every 7 days blood samples were taken and ppm Fluorine in the blood was determined. As you will see, results were so good we halted the testing after the first 7 day sample was analyzed, to save unnecessary effort and expense.

The results (in ppm Fluorine in the bloodstream, the average of five animals for each data point) are as follows:

EID087670

| | Day "Zero" of Recovery | Day 7 of Recovery |
|---|---|---|
| Control | 0.30 | 0.56 |

000186

HDR001555

| | | |
|---|---|---|
| 30 ppm diet C6-TBS | 5.30 | 0.35 |
| 30 ppm diet 3M's C8 | 33.23 | 19.26 |
| 300 ppm diet C6-TBS | 8.19 | 0.45 |
| 300 ppm diet 3M's C8 | 71.52 | 22.20 |

Taken in combination, these results say that when equal doses are ingested by male mice, six to nine times more C8 is found in the bloodstream than C6-TBS. Furthermore, the half-life of C8 in the bloodstream appears to be around seven days, whereas the even at the highest concentration tested C6-TBS blood fluorine levels had returned to the control baseline in that same timeframe (Gerry's comment was that t-1/2 could have been as short as one day; it's tough to tell from the data because we had anticipated greater values and so did not take any samples between day zero and day seven). And finally, the liver data says that when the material does leave the bloodstream, somewhere between 30 and 50 times more of the C8 winds up in the liver than does C6-TBS.

Taken as a whole, Gerry's conclusion was that C6-TBS is indeed measurably less bioaccumulative than C8.

As I promised above, I'll be writing up a complete report very soon (with Gerry's assistance), and will have copies of this report sent to each of you when it is finished.

Bruce

Jim/Charlie-- I've left it to you to forward this note to other members of your organization, as you know much better than I who specifically is interested on your end.

Hsu-Nan/Spencer:

Some good news just in on the C6 homologue of Zonyl TBS, and since I'll be out of the office this week, I thought I'd share it with you "before I go."

Gerry Kennedy just sent me a plot of percent increase in liver weight (vs. control) as a function of ppm fluorosurfactant in the diet. He tested Zonyl TBS, the C6-TBS, and perfluorooctanoic acid (3M's "C8"). The results are encouraging.

At the higher end of the scale, both TBS and the C6 version look good relative to C8. Specifically, at 100% increase in liver weight gain, the values look like this:

C8 -- 13.1ppm

TBS -- 378ppm

C6-TBS -- 705ppm

The key data are at 50% increase in liver weight, which shows the C6-TBS to be clearly better than the other two:

C8 -- 4.4ppm

TBS -- 3ppm

C6-TBS -- 142ppm

Even at 25% increase in liver weight, the comparison between C8 and the C6-TBS is quite encouraging:

C8 -- ca 2ppm

C6-TBS -- ca 30ppm

Gerry's cover note says: "C8 = 30-50X more potent than C6-TBS."

Please note that this is only half the story; the percent fluorine in the bloodstream work is still being done. At this point, we can say it's clearly not accumulating in the the liver to the extent that C8 does. What we don't know yet is whether that's because it's staying in the bloodstream to a greater extent than C8, or whether in fact it's being eliminated more efficiently.

EID087672

The blood testing should be finished soon; if it turns out to be as good as this result, we may in fact have found ourselves a less bioretentive fluorosurfactant. Keep those fingers crossed.



HDR001557

Bruce

EID087673

HDR001558

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000190

C-8 IN WASHINGTON WORKS DRINKING WATER

Recent sampling of the four Washington Works drinking water wells have shown C-8 levels of 0.1, 0.5, 2.9 & 3.3 parts per billion. These values are surprisingly high but do not pose a health threat to our employees.

The allowable exposure level of C-8 for our employees is set by our corporate AEL. This level is 0.01 mg/m3 or 0.56 ppb. This is an airborne control level. A control level for employee drinking water has not been set because such a control level was not deemed necessary.

Exposure to C-8 is best understood by examining the AEL on a daily dose level basis. At the AEL an employee can be safety exposed to a 12 hour dose of 0.10 mg of C-8. If an employee were to drink water with a C-8 level of 3.3 ppb he would only be exposed to a daily dose of 0.006 mg., or 1/20th of the AEL (note: C-8 is essentially fully absorbed by the human body if breathed or consumed).

At this time all operating areas on site are in compliance to the AEL for airborne C-8 exposure. The slight additional exposure imposed by drinking water with C-8 levels of 3-4 ppb should not affect our compliance to the daily dose of C-8 allowed by the AEL.

We should not get confused by the current CEG (Community Exposure Guideline) for C-8 in water of 1.0 ppb. This guide level is for exposure to the general public and not for the healthy workforce here on site.

However, this situation should not be taken lightly. The site enviromental group and the C-8 team will endeavor to fully understand these recent sample results and provide you with future programs.

Attached is the technical basis for the allowable dose exposure levels from Haskell.

000132

DEPARTMENT OF COMMERCE, LABOR & ENVIRONMENTAL RESOURCES
DIVISION OF ENVIRONMENTAL PROTECTION
1201 Greenbrier Street
Charleston, WV 25311-1088

Gaston Caperton
Governor
John M. Ranson
Cabinet Secretary

David C. Callaghan
Director
Ann A. Spaner
Deputy Director

July 8, 1993

Mr. W.M. Stewart
Sr. Environmental Control Consultant
E.I. DuPont de Nemours and Company
P.O. Box 1217
Parkersburg, WV 26102-1217

March 1 88 Request to landfill Big Sandy bio sludge

Permit 1/12/89

Re: Solid Waste/NPDES Water Pollution Control Permit No. WV0076244, Dry Run Landfill

Dear Mr. Stewart:

Landfill bio sludge

This agency's June 28, 1993 field review of the above facility revealed non-compliance with Conditions D.1. and G.6. of the above permit as excessive sediment has accumulated in the lower sedimentation pond. Also, the upper sedimentation pond is not being operated in compliance with Condition D.1. due to excessive sediment accumulation. Furthermore, the discharge from the lower pond, Outlet No. 001, is causing a discoloration of the receiving stream, apparently due to an accumulation of algae within the pond. Therefore, the sedimentation ponds must be cleaned out and the cause of algal growth must be determined. To assist in determining the cause of algal growth, this agency requests that the Outlet 001 be analyzed for Nitrite, Nitrate, Ammonia Nitrogen, Total Phosphorus, and BOD.

Cindy's letter Mr. Crosier

To facilitate the cleanout of the lower sedimentation pond, this agency suggests that the upper sedimentation pond be cleaned out and subsequently placed into service during the cleanout of the lower pond.

This agency also requests that the discharge from Outlet No. 001 be analyzed for acute toxicity as determined by percentage mortality (fourty-eight hour screening bioassay) to fathead minnows and Daphnia. Dissolved Oxygen concentration shall be determined prior to running the bioassay.

Within thirty days of the date of this letter, your company shall submit 1) acute toxicity testing results, 2) the above referenced laboratory analyses, and 3) a schedule for cleaning out the upper and lower sedimentation ponds.

Stated everyone used having problems with algal growth.

Naresh.

EID012563

000133

Should you have any questions concerning the above, please contact me at 367-2720.

Sincerely,

OFFICE OF WATER RESOURCES

John G. Britvec

John G. Britvec, Geologist
Industrial Branch

JGB/jb

cc: Cindy Musser, Insp., Environmental Enforcement, Dist. VI.
Supervisor, Environmental Enforcement, Dist. VI.

PROCEDURE FOR CLEANING OUT LOWER SEDIMENT AT DRY RUN LANDFILL

1. In early August place bails of hay at the narrowest point of the hollow between the lower sediment pond and the active fill area to minimize sedimen going to the pond and out the outfall if it does rain.

2. After the August monthly samples have been taken remove the drain plug from the lower pond overflow line and drain the lower pond.

3. Allow the lower pond to dry out for approximately two weeks.

4. Remove the sediment from the lower pond using a backhoe and a bulldozer. Landfill the sediment in the active landfill area.

5. Measure the dimensions of the lower pond and note them on the a sketch of the pond.

6. Replace the drain plug in the overflow line to place the lower sediment pond back in service.

000197

INTEROFFICE MEMORANDUM

Date: 05-Aug-1993 01:31pm
From: RICHARD A KIRSCHNER, JR
KIRSCHRA
Dept: POWER & SERVICES
Tel No: (304) 863-2992

TO: Thomas R. Waldron ( WALDROTR )

Subject: Dry Run Pond,

Tom,

I'm sure you've seen Walt's and Ed's memos. When you talk to Boso please be sure he knows what's going on and what to expect. Also, if Melvin doesn't think it will do any harm, have him open the valve all the way. The faster this stuff leaves, the better. That silt fence is looking better and better all the time. Is he eventually going to use one?

Thanks,

The "worst" golfer around...

EID017129

000139

681000

F1-C?

GW0000077

# Mortality Among Employees of a Perfluorooctanoic Acid Production Plant

**Frank D. Gilliland, MD, PhD**
**Jack S. Mandel, PhD, MPH**

*Perfluorooctanoic acid (PFOA) has been found at low levels (10 to 100 parts per billion) in sera of the general population and at higher levels in occupationally exposed workers. Although PFOA has been reported to be a promoter of rodent hepatocarcinogenesis and to alter reproductive hormones in humans and rodents, there is little information on human health effects associated with PFOA exposure. The present study examined the relationship between PFOA and mortality using a retrospective cohort mortality design. The cohort consisted of 2788 male and 749 female workers employed between 1947 and 1983 at a plant that produced PFOA. The all-causes standardized mortality ratio was .75 (95% confidence interval [CI], .56 to .99) for women and .77 (95% CI, .69 to .86) for men. Among men the cardiovascular standardized mortality rate was .68 (95% CI, .58 to .80) and the all-gastrointestinal diseases was .57 (95% CI, .29 to .99). There was no significantly increased cause-specific standardized mortality ratio for either men or women. Ten years of employment in exposed jobs was associated with a 3.3-fold increase (95% CI, 1.02 to 10.6) in prostate cancer mortality compared to no employment in PFOA production. There were only six prostate cancer deaths overall and four among the exposed workers; thus, the results must be interpreted cautiously. If prostate cancer mortality is related to PFOA, PFOA may increase prostate cancer mortality by altering reproductive hormones in male workers.*

Perfluorooctanoic acid (PFOA) and its salt, ammonium perfluorooctanoate, are perfluorinated surfactants. Because of their unique surface active properties they are used in a large number of industrial applications and consumer products including plasticizers, lubricants, wetting agents, and emulsifiers.[1-3] Despite their widespread use, little is known about potential adverse health effects.

PFOA induced marked hepatomegaly and peroxisome proliferation in rodent livers.[3-8] The chemically diverse group of xenobiotics that induce peroxisomes is of concern because of its association with nongenotoxic hepatocarcinogenesis.[3-10] PFOA did not produce an increased number of hepatocellular carcinomas in a 2-year rat feeding study.[8] However, biphasic (initiation and promotion) and triphasic (initiation, selection, and promotion) hepatic carcinogenesis studies in rodents have shown significantly increased numbers of carcinomas in the PFOA-treated rats.[11,12] It has been suggested that the marked rodent hepatomegaly produced by PFOA is a marker for carcinogenic potential.[13] The observations of increased Leydig cell tumors in a 2-year rat PFOA feeding study and of disruption of the hypothalamic-pituitary-gonad axis in PFOA-treated rats[8] are consistent with the hypothesis that PFOA-associated tumors are mediated by a hormonal nongenotoxic mechanism.

PFOA has a long half-life in humans. A study of occupationally exposed workers showed that the half-life in men is greater than 1.5 years.[14] Hence, accumulation of PFOA may occur from small, frequent PFOA doses. PFOA in the serum of the gen-

From the Division of Environmental and Occupational Health, School of Public Health, University of Minnesota, Minneapolis, Minnesota (Dr Gilliland, Dr Mandel); and Department of Internal Medicine, Occupational and Environmental Medicine Section, St Paul Ramsey Medical Center, St Paul, Minnesota (Dr Gilliland).

Address correspondence to: Frank D. Gilliland, MD, University of New Mexico School of Medicine, New Mexico Tumor Registry, 900 Camino de Salud NE, Albuquerque, NM 87131.

0096-1736/93/3509-0950$03.00/0

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EID088658

000200

eral populations of industrialized countries[15–19] is likely to be the result of an accumulation of small PFOA doses.

No health problems related to PFOA exposure were observed in a cross-sectional study among workers employed at the PFOA production plant.[14] Cross-sectional studies of PFOA-exposed workers at this plant have shown that PFOA was associated with decreased free testosterone and increased estradiol.[20]

To determine whether mortality from any cause was associated with occupational exposure to PFOA, a retrospective cohort mortality study was conducted at a plant that has produced PFOA since 1947.

## Methods

The plant consists of several divisions, with PFOA production restricted to the Chemical Division. A number of other specialty chemicals have been produced in this division. The study cohort consisted of workers who were employed at the plant for at least 6 months between Jan 1, 1947, and Dec 31, 1983. Data were abstracted from plant personnel records, which were maintained on all workers ever employed at the plant. Vital status was ascertained from the Social Security Administration for the period 1947 to 1982 and from the National Death Index for the period 1979 to 1989. All workers with unknown vital status were traced using a variety of tracing strategies such as directory assistance, Metronet and TRW searches, reverse directories, motor vehicle registration lists, contacting neighbors and relatives, and the post offices. Death certificates were obtained from the appropriate state health departments for those identified as, or presumed to be, deceased. Information concerning the data and cause of two deaths which occurred outside the United States was obtained from family members. A nosologist coded the death certificates for underlying cause of death according to the International Classification of Diseases, 8th revision. The reliability of the coding was evaluated by resubmitting a random sample of death certificates for coding by the same nosologist. In the 25 death certificates from 1970 to 1989 resubmitted to the nosologist for ICD coding, there were no changes in the major categories of cause of death.

Workers were categorized as exposed or unexposed to PFOA based on their job histories. Exposed workers were defined as all workers employed for 1 month or more in the Chemical Division. Unexposed workers were employees who either never worked in the Chemical Division or worked in the Chemical Division for less than 1 month. Cumulative exposure to PFOA was estimated using the surrogate measure of months of Chemical Division employment.

The observed numbers of cause-specific deaths were compared to the expected numbers of deaths obtained by applying sex- and race-specific quinquennial age, calendar period, and cause-specific mortality rates for the United States and Minnesota populations to the distribution of observed person-time.[21,22] Because less than 1% of plant employees were nonwhite, white male and white female rates were used for comparison. For women, only United States rates were used because cause- and calendar period-specific Minnesota rates for women were not available. The effects of latency, duration of employment, and work in the Chemical Division were examined using stratified standardized mortality ratio (SMR) analyses. Cause-specific mortality rates were compared between exposed and unexposed workers using stratified SMRs.[23] SMRs were calculated for men based on US and Minnesota white male mortality rates for three latency intervals (10, 15, and 20 years) and three categories of duration of employment (5, 10, and 20 years). The SMRs were calculated using the program developed by Monson.[22]

The relative risk (RR) and 95% confidence interval (CI) for deaths from all causes, cancer, cardiovascular diseases, and other selected causes were estimated using proportional hazard models.[24,25] The time to event or censoring was defined as time from first employment to event or to December 31, 1989. In models for specific causes of death, deaths from other causes were censored at the time of death. Age at first employment, year of first employment, and duration of employment were included as covariates in the model. The analyses were stratified by gender. The appropriateness of the proportional hazard assumptions was tested using stratified models with graphical analysis of log (−log[survival]) versus follow-up time relationships and models that tested the significance of a product term between exposure and log follow-up time.[25,26] Proportional hazard calculations were conducted using SAS.[25]

## Results

A total of 3537 workers employed at the plant between Jan 1, 1947 and Dec 31, 1983 were identified from company records. Six workers who had incomplete employment records were excluded from the study. The cohort consisted of 2788 (79%) men and 749 (21%) women (Table 1). Men

**TABLE 1**
Characteristics of Female and Male Employees, 1947–1989

| | Chemical Division | | Non-Chemical Division | | Total | |
|---|---|---|---|---|---|---|
| | Female | Male | Female | Male | Female | Male |
| Number of workers | 245 | 1339 | 504 | 1449 | 749 | 2788 |
| Person-years of observation | 6029.0 | 33385.3 | 13280.4 | 37732.4 | 19309.4 | 71117.7 |
| Mean follow-up (y) | 24.6 | 24.8 | 26.4 | 26.0 | 25.8 | 25.5 |
| Mean age at employment (y) | 28.8 | 25.6 | 26.9 | 28.9 | 27.6 | 27.3 |
| Mean year of death | 1965.0 | 1963.8 | 1962.8 | 1962.3 | 1963.5 | 1963.0 |
| Mean year of death | 1981.3 | 1978.3 | 1979.2 | 1978.1 | 1979.6 | 1978.2 |
| Mean age at death (y) | 58.7 | 54.2 | 54.4 | 58.1 | 55.4 | 56.4 |

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contributed 71,117.7 person-years of observation, which were equally divided between the Chemical Division and non-Chemical Division. Women contributed 19,309.4 person-years, two-thirds of which were in the non-Chemical Division.

Vital status was obtained for 100% of the cohort (Table 2). There were 50 deaths among the women (11 in the Chemical Division cohort and 39 in the non-Chemical Division cohort) and 348 deaths among the men (148 deaths in the Chemical Division group and 200 in the non-Chemical Division group). Death certificates were obtained for 99.5% of deaths.

For women, the SMR for all causes of death (SMR = .75; 95% CI, .56 to .99) was significantly lower than expected (Table 3). There was no association with duration of employment or latency for deaths from all causes, cancer, and cardiovascular diseases (data not shown). Mortality among Chemical Division women was less than expected. In Chemical Division women, the all-causes SMR was .46 (95% CI, .23 to .86) and the cancer SMR was .36 (95% CI, .07 to 1.05). The all-causes SMR for the non-Chemical Division women was .91 (95% CI, .64 to 1.24) and the cancer SMR was .91 (95% CI, .49 to 1.52) (data not shown).

Using Minnesota rates for comparison, the SMR for men for all causes, for cardiovascular diseases, and for all gastrointestinal diseases was significantly less than 1 (Table 4). None of the cause-specific SMRs was large nor was any significantly different from 1. The results were similar when the expected numbers of male deaths were based on US mortality rates. For the three latency intervals, the SMRs for deaths from all causes ranged from .75 to .77. For all cancers, the SMRs ranged from 1.06 to 1.12 and were nonsignificant.

Among men, there was no association between any cause of death and duration of plant employment. The all-causes SMRs were .86 (95% CI, .72 to 1.01) for the Chemical Division group and .69 (95% CI, .59 to .79) for the non-Chemical Division group (data not shown). The SMRs for prostate cancer were 2.03 (95% CI, .55 to 4.59) in the Chemical Division group and .58 (95% CI, .07 to 2.09) in the non-Chemical Division cohort. In the Chemical Division group, there were 4 observed and 2 expected deaths from prostate cancer. There was no significant association between any cause of death and latency in either exposure group. For the Chemical Division cohort, the prostate cancer SMR was 1.61 (95% CI, .32 to 4.70) in the greater than 15-year latency group.

Table 5 presents the final proportional hazard model for all-causes, all-cancer, and prostate-cancer mortality among the 2788 male workers employed for more than 6 months. The estimated relative risk for all-cause mortality for a 1-year increase in age at first employment was 1.08 (95% CI, 1.07 to 1.09). Year of first employment and duration of employment were negatively associated with deaths from all causes. The risk associated with months employed in the Chemical Division was small and nonsignificant.

In the final prostate cancer mortality model, length of employment in the Chemical Division was positively and significantly associated with prostate cancer risk. The relative risk for a 1-year increase in Chemical Division employment time was 1.13 (95% CI, 1.01 to 1.27) For 10 years' employment in the Chemical Division, the relative risk was estimated to be 3.3 (95% CI, 1.02 to 10.6) compared with workers never employed in the Chemical Division. Age at first employment was positively associated with prostate cancer mortality. Length of time employed in the Chemical Division was not significantly related to mortality from lung cancer, gastrointestinal cancer, pancreatic cancer, or diabetes mellitus.

**TABLE 2**
Vital Status and Cause of Death Ascertainment among Female and Male Employees, 1947–1989

| Vital Status | Chemical Division | | | | Non-Chemical Division | | | | Total | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Female | | Male | | Female | | Male | | Female | | Male | |
| | No. | % | No. | % | No. | % | No. | % | No. | % | No. | % |
| Alive | 234 | 95.3 | 1191 | 88.9 | 465 | 91.6 | 1249 | 86.2 | 699 | 93.3 | 2440 | 87.5 |
| Dead | 11 | 4.7 | 148 | 11.1 | 39 | 8.4 | 200 | 13.8 | 50 | 6.7 | 348 | 12.5 |
| Total | 245 | 100.0 | 1339 | 100.0 | 504 | 100.0 | 1449 | 100.0 | 749 | 100.0 | 2788 | 100.0 |

**TABLE 3**
Observed (Obs) and Expected (Exp) Deaths, Standardized Mortality Ratios (SMR) and 95% Confidence Intervals (CI) for 749 Female Employees

| Cause of Death | Obs | Exp | SMR | 95% CI |
|---|---|---|---|---|
| All causes | 50 | 66.74 | 0.75 | 0.56–.99 |
| Cancer | 17 | 23.04 | 0.71 | 0.42–1.14 |
| Gastrointestinal | 2 | 4.54 | 0.44 | 0.05–1.59 |
| Respiratory | 4 | 4.72 | 0.95 | 0.26–2.43 |
| Breast | 3 | 5.87 | 0.51 | 0.10–1.49 |
| Genital | 2 | 3.37 | 0.59 | 0.07–2.14 |
| Lymphopoietic | 3 | 2.04 | 1.47 | 0.30–4.29 |
| Cardiovascular | 10 | 12.39 | 0.81 | 0.49–1.29 |
| Cerebrovascular | 3 | 3.51 | 0.86 | 0.01–4.80 |
| Gastrointestinal | 3 | 3.41 | 0.88 | 0.18–2.57 |
| Injuries | 4 | 6.23 | 0.64 | 0.17–1.64 |
| Suicide | 1 | 1.78 | 0.56 | 0.01–3.13 |

## Discussion

This was the first retrospective cohort mortality study of workers employed in a PFOA production plant. Mortality from all causes in both men and women was significantly less than expected. Because of the healthy worker effect, internal comparisons

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TABLE 4
Deaths and Standardized Mortality Ratios (SMR) Based on Minnesota White Male Rates, Among 2788 Male Employees, 1947–1989, and 1339 Men Ever Employed in the Chemical Division, 1947–1989

| Causes of Death | All Male Employees | | | | Men Employed in Chemical Division | | | |
|---|---|---|---|---|---|---|---|---|
| | Obs | Exp | SMR | 95% CI | Obs | Exp | SMR | 95% CI |
| All causes | 347 | 450.79 | 0.77 | 0.69–0.86 | 148 | 172.96 | 0.86 | 0.72–1.01 |
| Cancer | 103 | 97.29 | 1.05 | 0.86–1.27 | 40 | 36.31 | 1.10 | 0.79–1.50 |
| Gastrointestinal | 24 | 26.78 | 0.90 | 0.57–1.33 | 9 | 9.77 | 0.92 | 0.42–1.75 |
| Colon | 9 | 9.42 | 0.96 | 0.44–1.81 | 4 | 3.46 | 1.15 | 0.31–4.01 |
| Pancreas | 8 | 5.58 | 1.43 | 0.62–2.83 | 4 | 2.04 | 1.96 | 0.53–5.01 |
| Respiratory | 31 | 30.42 | 1.02 | 0.69–1.45 | 12 | 11.26 | 1.07 | 0.55–1.86 |
| Lung | 29 | 28.94 | 1.00 | 0.67–1.44 | 11 | 10.70 | 1.03 | 0.51–1.84 |
| Prostate | 6 | 6.07 | 0.99 | 0.36–2.15 | 4 | 1.97 | 2.03 | 0.55–4.59 |
| Testis | 1 | 0.92 | 1.09 | 0.01–6.05 | 1 | 0.44 | 2.28 | 0.03–12.66 |
| Bladder | 3 | 2.18 | 1.37 | 0.28–4.01 | 1 | 0.75 | 1.33 | 0.02–7.40 |
| Lymphopoietic | 13 | 12.07 | 1.09 | 0.57–1.84 | 5 | 4.76 | 1.05 | 0.34–2.45 |
| Cardiovascular | 145 | 212.19 | 0.68 | 0.58–0.80 | 54 | 76.65 | 0.70 | 0.53–0.92 |
| CHD* | 110 | 159.09 | 0.69 | 0.57–0.83 | 43 | 57.74 | 0.74 | 0.54–1.00 |
| Cerebrovascular | 10 | 24.66 | 0.60 | 0.32–1.02 | 4 | 8.53 | 0.47 | 0.13–1.20 |
| All gastrointestinal | 12 | 21.13 | 0.57 | 0.29–0.99 | 8 | 8.27 | 0.97 | 0.42–1.91 |
| All respiratory | 13 | 21.75 | 0.60 | 0.32–1.06 | 7 | 7.77 | 0.91 | 0.36–1.87 |
| Diabetes | 8 | 6.52 | 1.23 | 0.53–2.42 | 3 | 2.55 | 1.18 | 0.24–3.44 |
| Injuries | 38 | 47.74 | 0.80 | 0.56–1.08 | 31 | 31.72 | 0.98 | 0.66–1.39 |
| Suicide | 12 | 15.09 | 0.79 | 0.41–1.39 | 10 | 6.99 | 1.43 | 0.68–2.63 |

*CHD, coronary and atherosclerotic heart disease.

were made between Chemical Division and non-Chemical Division employees. There were no significantly elevated SMRs in Chemical Division or non-Chemical Division employees. However, prostate cancer mortality was associated with length of employment in the Chemical Division in proportional hazard analysis. Ten years of employment in the Chemical Division was associated with an estimated 3.3-fold increase (95% CI, 1.02 to 10.60) in prostate cancer mortality.

The use of prostate cancer mortality to assess the association between PFOA and prostate cancer occurrence is problematic. Age-adjusted prostate cancer mortality rates from 1983 to 1989 (949 per 100,000) were only 25% of the incidence rates (99.4).[27] This low proportion of deaths among cases attributed to prostate cancer reflects the high risk of death for competing causes for this disease of elderly men. Given the small number of observed deaths from prostate cancer in the study, and the observed difference in incidence and mortality rates, the suggested association between PFOA exposure and prostate cancer must be viewed as hypothesis generating and should not be overinterpreted. The association may be real, may have been a chance finding, or may be the result of an unrecognized environmental factor. However, the biologic plausibility for any association between PFOA employment and prostate cancer is provided by animal toxicologic and human epidemiologic data that show an association between PFOA and reproductive hormone changes.[20]

The all-causes, all-cancer, and all-cardiovascular mortality among women was less than expected in the overall cohort. The low SMRs are most likely to be a result of the healthy worker effect. Latency and duration of plant employment did not have a strong relationship with the healthy worker effect.

The interpretation of this study requires consideration of methodological issues. SMRs for the subgroups of workers are not strictly comparable. We attempted to calculate standardized rate ratios; however, the rates were based on small numbers and produced unstable ratios. Estimates of PFOA exposure were based on job history, and categorization of workers into ever versus never employed in the Chemical Division may not reflect the biologic effective dose of PFOA. PFOA exposure was apparently widespread among employees not directly exposed to PFOA,[14] and the exposure categorization may misclassify workers as unexposed when they were ex-

TABLE 5
Proportional Hazard Regression Model of Factors Predicting Mortality among All Male Employees*

| Variable | All Causes of Death | | | | Cancer Deaths | | | | Prostate Cancer Deaths | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | β | SE(β) | P | RR† | β | SE(β) | P | RR† | β | SE(β) | P | RR† |
| Year of first employment | −0.55 | 0.009 | 0.0001 | 0.946 | −0.031 | 0.019 | 0.11 | 0.969 | 0.010 | 0.081 | 0.9 | 1.011 |
| Age at first employment (y) | 0.079 | 0.006 | 0.0001 | 1.08 | 0.078 | 0.011 | 0.0001 | 1.081 | 0.082 | 0.045 | 0.06 | 1.085 |
| Duration of employment (y) | −0.34 | 0.001 | 0.0001 | 0.967 | −0.028 | 0.009 | 0.002 | 0.972 | −0.07 | 0.052 | 0.18 | 0.932 |
| Months in chemical division | 0.001 | 0.001 | 0.24 | 1.001 | 0.002 | 0.001 | 0.2 | 1.002 | 0.01 | 0.005 | 0.03 | 1.01 |

* Abbreviations used are: β, regression parameter; SE(β), standard error of the slope parameter; RR, relative risk.
† Relative risk for one unit change in independent variable.

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posed. Such misclassification would be expected to bias the effect estimates toward the null if increased exposure increases death rates. Months employed in the Chemical Division may better reflect the biologic effective dose because cumulative exposure reflects the bioaccumulation of PFOA. Workers were exposed to many other xenobiotics, such as benzene and asbestos, during their employment at the plant. However, none of these materials has been associated with prostate cancer.

Although the mean age at first employment and mean year of first employment are similar in the Chemical Division and non-Chemical Division cohorts of men and women, the comparisons of the rates of disease are confounded by differences in the distribution of age at risk. The use of an internal comparison group may reduce, but not eliminate, confounding if the internal comparison groups have different distributions of these time factors. Because the disease occurrence relationship is defined in terms of cumulative exposure, the true effect of PFOA exposure may have been biased toward or away from the null by uncontrolled confounding by time factors.[28,29]

Further research is needed to evaluate and confirm the association between PFOA and prostate cancer. The findings in this study are based on a small number of cases and could have resulted from chance or unrecognized confounding from exposure to other factors. Studies of prostate cancer incidence in this and other PFOA-exposed work forces may clarify the suggested increase in prostate cancer risk.

## Acknowledgments

This work was supported in part by National Institute for Occupational Safety and Health Grant T150H07098-16, and the 3M Corporation.

## References

1. Guenthner R, Vietor M. Surface active materials from perfluorocarboxylic and perfluorosulfonic acids. *I&EC Product Research and Development* 1962;1: 165–169.
2. Griffith F, Long J. Animal toxicity studies with ammonium perfluorooctanoate. *Am Ind Hyg Assoc J.* 1980;41:576–583.
3. Olson C, Andersen M. The acute toxicity of perfluorooctanoic and perfluorodecanoic acids in male rats and their effects on fatty tissue. *Toxicol Appl Pharmacol.* 1983;70:362–372.
4. Pastoor T, Lee K, Perri M. Gillies, P. Biochemical and morphological studies of ammonium perfluorooctanoate-induced hepatomegaly and peroxisome proliferation. *Exp Mol Pathol.* 1987;47: 98–109.
5. Just W, Gorgas K, Hartl F, Heinemann P, Salazar M, Schimassek H. Biochemical effects and zonal heterogeneity of peroxisome proliferation induced by perfluorocarboxylic acids in rat liver. *Hepatology.* 1989;9:570–581.
6. Kennedy G. Dermal toxicity of ammonium perfluorooctanoate. *Toxicol Appl Pharmacol.* 1985;81:348–355.
7. Kennedy G, Hall G, Brittelli J, Chen H. Inhalation toxicity of ammonium perfluorooctanoate. *Food Chem Toxicol.* 1986;24:1325–1329.
8. Cook J, Murray S, Frame S, Hurtt M. Induction of Leydig cell adenomas by ammonium perfluorooctanate: A possible endocrine related mechanism. *Toxicol Appl Pharmacol.* 1991;113:209–213.
9. Reddy J, Azarnoff D, Hignite C. Hypolipidemic hepatic peroxisom proliferators form a novel class of chemical carcinogens. *Nature.* 1980;283:397–398.
10. Reddy J, Lalwani N. Carcinogenesis by hepatic peroxisome proliferators: evaluation of the risk of hypolipidemic drugs and industrial plasticizers to humans. *Crit Rev Toxicol.* 1983;12:1–85.
11. Nilsson R, Beije B, Preat V, Erixon K, Ramel C. On the mechanism of the hepatocarcinogenicity of peroxisome proliferators. *Chem Biol Interact.* 1991; 78:235–250.
12. Abdellatif A, Preat V, Vamecq J, Nilsson R, Roberfroid M. Peroxisome proliferation and modulation of rat liver carcinogenesis by 2,3-dichlorophenoxyacetic acid, 2,4,5-trichlorophenoxyacetic acid, perfluorooctanoic acid and nafenopin. *Carcinogenesis.* 1990;11:1899–1902.
13. Takagi A, Sai K, Umemura T, Hasegawa R, Kurokawa Y. Short-term exposure to the peroxisome proliferators, perfluorooctanoic acid and perfluorodecanoic acid, causes significant increases of 8-hydroxydeoxyguanosine in liver DNA of rats. *Cancer Lett.* 1991;57:55–60.
14. Ubel F, Sorenson S, Roach D. Health status of plant workers exposed to fluorochemicals: a preliminary report. *Am Ind Hyg Assoc J.* 1980;41:584–589.
15. Guy W. Fluorocompounds of human plasma: analysis, prevalence, purification, and characterization, PhD thesis. Rochester, NY: University of Rochester; 1972.
16. Guy W, Taves D, Brey W. Organic fluorocompounds in human plasma: prevalence and characterization. In: Filler R, ed. *Biochemistry involving carbon-fluorine bonds. ACS Symposium Series.* New York: American Chemical Society; 1976:117–134.
17. Guy W. Ionic and organic fluorine in blood. In: Johansen E, Taves D, Olsen T, ed. *Continuing evaluation of the use of fluoride.* Boulder, CO: Westview Press; 1979.
18. Taves D. Evidence that there are two forms of fluoride in human serum. *Nature.* 1968;217:1050–1051.
19. Taves D. Comparison of "organic" fluoride in human and nonhuman serums. *J Dent Res.* 1971;50:783.
20. Gilliland F. Fluorocarbons and human health: studies in an occupational cohort, PhD thesis. Minneapolis, MN: University of Minnesota; 1992.
21. Berry G. The analysis of mortality by the subject-years method. *Biometrics.* 1983; 39:173–184.
22. Monson R. Analysis of relative survival and proportional mortality. *Comput Biomed Res.* 1974;7:325–332.
23. Miettinen O. Standardization of risk ratios. *Am J Epidemiol.* 1972;96: 383–388.
24. Cox D. Regression models and life tables. *J R Stat Soc (B).* 1972;34: 187–220.
25. SAS Institute. SAS User's Guide: Statistics. 6th ed. Cary, NC: SAS Institute Inc; 1990.
26. Kalbfleisch J, Prentice R. *The statistical analysis of failure time data.* New York: John Wiley & Sons; 1980.
27. Miller R. Cancer statistics review 1973–1989. Bethesda, Maryland. US Department of Health and Human Services, National Institutes of Health, National Cancer Institute. NIH Pub. No. 92-2789, 1992.
28. Checkoway H, Pierce N, Crawford-Brown D. Research methods in occupational epidemiology. New York: Oxford University Press; 1989.
29. Robins J. A new approach to causal inference in mortality studies with a sustained exposure period. *Math Modeling.* 1986;7:1393–1512.

EID088662

AH&M ENVIRONMENTAL, INC.
6404 MacCorkle Avenue, SW
St. Albans, WV 25177

# BIOASSAY REPORT

FOR

**E. I. DUPONT**
**WASHINGTON WORKS**
**P. O. BOX 1217**
**PARKERSBURG, WV 26102**

**AHM NO. 93-114**

*DAPHNIA PULEX*

DECEMBER 8 - 10, 1993, 48 HOUR STATIC ACUTE DEFINITIVE

## INTRODUCTION

AH&M Environmental, Inc. conducted a 48 hour static acute bioassay to determine the toxicity of a landfill leachate collected by E. I. DuPont, Washington Works. A grab sample was collected on December 7, 1993. The sample was tested for toxicity using the freshwater organism, *Daphnia pulex*. E. I. DuPont, Washington Works is located in Parkersburg, West Virginia 26102. The test was conducted on December 8 - 10, 1993.

## DILUTION WATER

The dilution water used to perform the definitive toxicity test was moderately hard synthetic freshwater that is favorable to the aquatic organisms used in the test. The moderately hard synthetic fresh water is prepared according to EPA protocol " Methods for Measuring the Acute Toxicity of Effluent and Receiving Water to Freshwater and Marine Organisms, EPA/600/4-90/027." The water is allowed to acrate for 24-48 hours prior to use in a test. Water quality analysis is conducted on the dilution water and if at any time water quality is not adequate, an additional batch will be prepared or an alternate source will be chosen.

## LEACHATE SAMPLE:

The sample collected was a grab sample collected on December 7, 1993. The sample was collected by DuPont Washington Works personnel, refrigerated, and picked-up by AH&M Environmental, Inc. on December 8, 1993 at approximately 1100 hours. The effluent was translucent and had an orange color. An odor was observed and a small amount of sediment was present. The receiving temperature of the sample was 12.4C. Samples were stored at 4C and were used in the test prior to the 36 hour holding time.

## TEST METHODS:

A definitive toxicity test was performed for E. I. DuPont, Washington Works according to EPA protocol "Methods for Measuring the Acute Toxicity of Effluent and Receiving Waters to Freshwater and Marine Organisms, EPA/600/4-90/027". Using a 0.5 dilution series, a concentration range of 100%, 50.0%, 25.0%, 12.5%, 6.25% and 0% (control) was used to test the toxicity of the sample.

The test vessels were 30 ml plastic disposable test vessels. The test volume was 25 ml. Upon receipt, samples are logged in, assigned an AHM test number and initial parameters are recorded. The test concentrations are then mixed and decanted to the test vessels. Each test vessel is randomly distributed and at least 20 organisms of a given species are exposed to each effluent concentration, five (5) per replicate.

The test data measured in the tests are biological, chemical and physical data. Death/mortality is the effect measured in the acute test. Mortality of the test chambers are monitored for early mortality during the first few hours, and only every 24 hours thereafter. Chemical and physical data (ie., pH, conductivity, dissolved oxygen and temperature) are measured at a minimum in all test concentrations every 24 hours. Total alkalinity and total hardness are measured in the controls and in the highest concentration at the beginning of the test.

The test temperature range is 25+/-1C. Dissolved oxygen is not allowed to exceed below 40% saturation. If however, the sample has a high oxygen demand and the test concentrations exhibit dissolved oxygen levels less than 40% saturation, aeration of all test chambers will occur prior to introducing organisms to the test chambers.

The test organisms used in the test were 1-24 hour *Daphnia pulex* which were cultured at AH&M Environmental, Inc. *Daphnia pulex* adults were isolated on 12/07/93 and received on 12/08/93. The Daphnia neonates produced are believed to be disease free and in the best of health. AH&M Environmental, Inc. performs reference toxicant testing and maintains a control chart for the Daphnia neonates cultured in house to assess the health of our cultured organisms and the techniques applied by the technicians.

## RESULTS:

The adverse effects measured in the 48 hour definitive test for E. I. DuPont, Washington Works was death/mortality. At 48 hours 15% mortality occurred in the 100%, 5% mortality occurred in the 50%, and 0% mortality occurred in the 25%, 12.5%, 6.25% and 0% (controls). The LC50 for Washington Works sample was >100%. The Acute Toxic Unit was not measurable. See Section V, of the Test Summary for Daily Percent Mortality. All Chemical analysis of effluent and dilution water are in appendix A. A copy of the laboratory bench sheet is in appendix B.

AH&M ENVIRONMENTAL, INC.
6404 MacCorkle Avenue, SW
St. Albans, West Virginia 25177

# TEST SUMMARY SHEET

FACILITY: E. I. DuPont Washington Works
ADDRESS: Parkersburg
West Virginia 26102
TELEPHONE NO: (304) 863-4633
TEST DATE: 12/08-10/93

CONTACT: Jim Yoak
OUTFALL NO:
NPDES NO: WV
SAMPLE ID: Landfill Leachate
REPORT DATE: 12/20/93

## I. SAMPLE INFORMATION

A. Method of Sample Shipment: AH&M Environmental, Inc. courier

B. Condition Upon Arrival At Labortory:

1. Temperature 12.4 C
2. pH 8.25 Std Units
3. Chlorine 0 mg/l
4. Conductivity 500 umhos
5. DO 10.1 mg/l
6. Appearance Translucent, orange colored liquid, odor present, and a small amount of sediment

C. Collection Dates: 12/07/93

D. Collection Location: Prior to discharge

## II. DILUENT INFORMATION

A. Method of Diluent Shipment: Not applicable prepared inhouse
B. Treatment of Diluent: Moderately hard synthetic freshwater prepared to EPA Protocol 600/4-90/027.

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TEST SUMMARY SHEET

C. Condition of dilution water

1. Temperature 25.4 C
2. pH 8.07 Std Units
3. Chlorine 0 mg/l
4. Conductivity 300 umhos
5. DO 8.0 mg/l
6. Appearance Translucent, colorless liquid, with no sediment and/or odor

D. Collection/Preparation Dates: 12/01/93

E. Collection/Preparation Location: AH&M Environmental, Inc.

III. TEST START

A. Test Organism: *Daphnia pulex*

B. Age: <24 hrs Isolated 12/07/93

C. Test Vessel Size: 30 ml

D. Test Volume: 25 ml

E. No. of Replicates: 4

F. No. of Organisms per Replicates: 20

G. Test Dates/Times: Beginning Date: 12/08/93 Time: 1210
Ending Date: 12/10/93 Time: 1210

H. 100% Effluent at 0 Hours

1. Chlorine Initial 0 mg/l Adjusted Not Applicable
2. Salinity Initial 0 ppt Adjusted Not Applicable
3. pH Initial 8.19 std. units Adjusted Not Applicable

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TEST SUMMARY SHEET

4. Alkalinity 268 mg/l
5. Hardness 200 mg/l
6. Conductivity 600 umhos
7. DO Initial 8.1 mg/l Adjusted Not Applicable
   Aeration Period (if necessary) Not Applicable

I. DILUENT at 0 Hours

1. pH Initial 8.05 std. units Adjusted Not Applicable
2. Salinity Initial 0 ppt Adjusted Not Applicable
3. DO Initial 8.0 mg/l Adjusted Not Applicable
   Aeration Period (if necessary) Not Applicable
4. Alkalinity 62 mg/l
5. Hardness 94 mg/l
6. Conductivity 310 umhos

## IV. TEST RESULTS

A. Test Acceptability

1. Control Survival 100%
2. Average Weight per Control Organism -

B. Statistical Analysis

1. Method of Statistical Analysis Not Applicable
2. 48 Hour LC50 >100%
3. 95% Lower and Upper Confidence Intervals Not Applicable

C. $TU_a$

1. $TU_a = 100/LC50 = 100/>100\% =$ AA (Below detectable)



WVDEP 029

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St. Albans, West Virginia 25177

TEST SUMMARY SHEET

## V. TABLE 1 DAILY PERCENT MORTALITY TEST CONCENTRATION

| HOUR | CONTROL | 6.25% | 12.5% | 25.0% | 50.0% | 100.0% |
|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 24 | 0 | 0 | 0 | 0 | 5 | 15 |
| 48 | 0 | 0 | 0 | 0 | 5 | 15 |

## VI. COMMENTS



WVDEP 029

CC0212

DUPONT

HASKELL LABORATORY

FOR DUPONT USE ONLY

LIMITED DISTRIBUTION

This review reflects the available toxicity literature, both published and unpublished. Studies have not been evaluated for scientific merit. Contact Haskell Laboratory if you have questions.

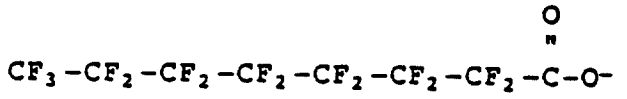
Common Name: Ammonium Perfluorooctanoate (APFO)

Chemical Name: Octanoic acid, pentadecafluoro-, ammonium salt

Synonyms: C-8
FC-143

CAS Registry No.: 3825-26-1

Chemical Structure:



$$CF_3-CF_2-CF_2-CF_2-CF_2-CF_2-CF_2-C(=O)-O^- \ NH_4^+$$

Physical Properties (5):

| | |
|---|---|
| Description: | Light-colored powder, slight odor |
| Molecular Weight: | 431 |
| Boiling Point: | Sublimes @ 130°C |
| Melting Point: | -- |
| Bulk Density: | 0.6-0.7 g/mL |
| Vapor Pressure: | $7 \times 10^{-5}$ mm Hg @ 20°C |
| Flash Point/Flammability: | -- |
| Explosive Limits: | -- |
| Solubility: | Soluble in water, ethanol and acetone |
| Conversion Factors: | 1 mg/L = 57 ppm<br>1 ppm = 17.6 mg/m³ |

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This literature search contains 23 pages of text and 62 references. An * in the left margin indicates new data added at this update.

This search was prepared by Richard C. Graham. See the last page of this document for its updating history.

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Exposure Standards:

DuPont AEL = 0.01 mg/m³ (8-hour TWA), skin

* DuPont Community Exposure Guideline (air) ($CEG_a$) = 0.3 ug/m³

* DuPont Community Exposure Guideline (water) ($CEG_w$) = 1 ug/L

* ACGIH TLV = 0.1 mg/m³, skin

DOT Classification:

None

EPA RCRA Status:

None

FDA Status:

None

TSCA Inventory:

Yes

TOXICITY

Summary:

C-8 has moderate acute oral toxicity with an LD50 in rats of 470 mg/kg. An aqueous paste of C-8 produced mild to moderate irritation on the skin of rabbits. Clinical signs of toxicity were seen at doses as low as 1500 mg/kg. Instillation of the solid material into the rabbit eye produced moderate corneal opacity, iritis and conjunctivitis. The ocular effects gradually receded. Prompt washing of the eye reduced the effects and these eyes recovered more quickly. By the acute inhalation route, C-8 is highly toxic with a four-hour ALC in the rat of 0.8 mg/L. In a two-week subacute inhalation study performed at 11 and 83 mg/m³, liver degeneration, enlarged livers and increases in liver enzymes were observed at both concentrations. A second study was run at 1, 7.6 and 83.9 mg/m³. No compound-related effects were seen in the rats exposed at 1 mg/m³. Exposure-related mortality was seen at the high level of exposure. Additionally, macroscopic and microscopic liver pathology was observed in the animals exposed at 7.6 and 83.9 mg/m³. These liver effects appeared to be reversible. Subacute studies by the oral and skin absorption routes of administration confirm the effects of C-8 on the liver. In two chronic feeding studies in rats, C-8 produced an increase in the incidence of tumors in the liver, pancreas, and testis. C-8 was not teratogenic in tests by inhalation and gavage and was not mutagenic in the Ames test.



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A. Acute

1. Oral

- ALD (rat) = 480 mg/kg (3.18).
- ALD (rat) = 670 mg/kg (3.1,3.5).
- * LD50 (rats) = 390 mg/kg (11.10).
- LD50 (male rats) = 470 mg/kg (3.15).
- LD50 (female rats) = 482 mg/kg (3.15).
- LD50 (rats) = 540 mg/kg (5,12).
- LD50 (mice) = 457 mg/kg (3.16).
- LD50 (male guinea pigs) = 178 mg/kg (3.14).
- LD50 (female guinea pigs) = 217 mg/kg (3.14).
- C-8 was slightly toxic when administered in single oral doses to the following groups of rats: weanlings (21 days old): males, LD50 = 573 mg/kg and females, LD50 = 580 mg/kg; young adults (approximately eight to ten weeks old): males, LD50 = 470 mg/kg and females, LD50 = 453 mg/kg; older rats: males, LD50 = 336 mg/kg and females, LD50 = 343 mg/kg; newborn (< two days old): males, LD50 = 243 mg/kg and females, LD50 = 258 mg/kg. Toxicity in regards to the sex of the rats showed no significant difference. However, newborn (<two days old) and older rats had lower LD50 values than weanlings or young adult rats (3.32).
- C-8 is moderately toxic when administered orally to young adult male and female rats that were untreated or surgically-modified (ovarectomized or castrated). Its LD50 is between 400 and 491 mg/kg of body weight. No changes in liver weight to body weight ratios were seen in female rats given single doses of up to 200 mg/kg (ovarectomized or normal). A single oral dose of either 100 or 200 mg/kg produced an increase in liver weight of male rats. Castration reduced the magnitude of the liver weight increase but rats castrated and given 200 mg/kg had heavier livers than did the controls. Clinical signs seen in C-8 treated rats included stained face and perineal area and sporadic weight loss (3.21).

- Two male beagles given a single dose of 450 mg/kg APFO died within 48 hours. Plasma enzymes were markedly elevated 24-48 hours after ingestion. Dogs given 200 mg/kg had elevated GPT and GOT in 24 hours which normalized after one week. This increase in plasma enzyme levels is indicative of cellular damage (3.3).

- The oral LD50 of C-8 was determined in rats pretreated with phenobarbital sodium or proadifen hydrochloride. There were no significant differences in the LD50 value of C-8 (478 mg/kg) following pre-treatment with either phenobarbital sodium or proadifen hydrochloride (3.19).

- APFO was administered to rats by stomach tube in single doses ranging from 1.5-2250 mg/kg. Clinical signs of toxicity were inactivity, a red discharge around the nose, perineal discoloration and weight loss. Animals receiving doses $\geq$ 200 mg/kg showed enlarged livers (3.1).

- Rats receiving intragastric doses as low as 60-90 mg/kg had enlarged livers (3.5). Gastrointestinal irritation was also observed in rats ingesting APFO (3.5,3.6).

- APFO, given in single 12 mg/kg doses to three rats, produced no clinical signs of toxicity (3.2).

- Pre-dosing or post-dosing with Dowex® 1-X2-Cl Ion Exchange Resin at 1000 mg/kg changes the toxic effects of C-8 in rats. All rats dosed with the Dowex®, either before or after C-8 had reduced mortalities compared to the rats dosed with C-8 alone (3.23).

* - Groups of young adult rats were administered a single dose of ethanol at 6000 mg/kg or a 15% solution in their drinking water for 14 days and/or a single dose of C-8 at dose levels of 200, 480, or 670 mg/kg. In the rats administered a single dose of ethanol followed 24 hours later by C-8, all the rats administered 670 mg/kg of C-8 died within 8 days after dosing. In the rats administered ethanol for 14 days before C-8 dosing, all the rats administered 480 and 670 mg/kg of C-8 died within 6 days after dosing. Liver weight/body weight ratios showed a sharp increase in all the rats that received C-8 when compared to controls and to the rats that received only ethanol. There were no significant differences between the untreated controls and the rats that received only ethanol or between the ethanol pre-treated rats administered C-8 and the rats that received only C-8 (3.34).



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* • See Related Reference 13 for information on the toxicity and kinetics of perfluorooctanoic acid.

2. Skin

• LD50 (rabbits) = 4278 mg/kg (3.10,6).

Rabbits were dosed at 7500, 5000, 3000 and 1500 mg/kg. Skin irritation occurred at all levels. Clinical signs included weight loss and labored breathing at all levels, wetness underneath the body, cyanosis, diarrhea, and lethargy appearing at most levels (3.10).

• LD50 (male rats) = 6959 mg/kg (3.10,6).

Male rats dosed at 3000, 5000 and 7500 mg/kg showed initial weight loss. Rats dosed at 5000 or 7500 mg/kg were lethargic on the day of treatment and the majority had wet and/or stained perineal areas. Chromodacryorrhea was seen in rats dosed at 7500 mg/kg (3.10,6).

• Female rats dosed at 5000 or 7500 mg/kg showed mild skin irritation and initial weight loss. At 7500 mg/kg a stained face and a wet and stained perineal area were also noted (3.12,6).

* • Ammonium perfluorooctanoate did not produce irritation when applied to the skin of rabbits (5).

• APFO (500 mg) was applied as an aqueous paste to the trunk and lateral areas of six male rabbits. After 24 hours semi-occluded contact, APFO caused mild to moderate skin irritation. Observation at 48 hours indicated slight to moderate irritation (3.9,6).

• Permeation of five types of gloves by C-8 was evaluated. Three of the gloves (Neoprene®, neosynthetic latex and natural latex) had a measurable breakthrough time and permeation rate after eight hours of continuous exposure to a 30% C-8 solution (3.22).

• See Related Reference 14 for additional information.

3. Eyes

• APFO caused generalized moderate corneal opacity with a small area of severe opacity, intermittent moderate iritis and moderate conjunctivitis when 38.3 mg of solid test material was placed into the right conjunctival sac of a rabbit's eye. The ocular effects gradually receded, however, the small



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area of corneal opacity persisted and at 21-28 days was mild with vascularization. An eye dosed with the test material and promptly washed had a small area of slight to moderate corneal opacity and moderate to slight conjunctivitis with no iritic effect. The eye was normal within seven days except for mild conjunctival redness, which was normal within 14 days (3.8).

- APFO (100 mg) was moderately irritating to the eyes of rabbits (mean score = 14.0; highest possible score = 110.0). Iridial and conjunctival effects were evident in unwashed eyes, but only conjunctival effects appeared when eyes were washed after exposure (5).

- Rats exposed to APFO during a four-hour inhalation period exhibited corneal opacity and ulceration which were still microscopically evident after 42 days (3.6).

4. Inhalation

- Four-hour ALC (rats) = 0.8 mg/L (3.6,7).

  Groups of rats were exposed for four hours to concentrations of APFO ranging from 0.38 mg/L to 5.7 mg/L. At a concentration of 2.2 mg/L or higher, all rats died within 48 hours after exposure. Liver enlargement reached a maximum seven to 14 days after exposure returning to normal within 42 days. APFO was highly toxic when inhaled but no changes in cell morphology were observed (3.6,7).

* - 4-hour LC50 (rats) = 980 mg/m$^3$ (11.12).

- No deaths resulted from a one hour exposure to a nominal concentration of APFO (18.6 mg/L). Clinical signs during exposure were red nasal discharge, yellow staining of the ano-genital fur, dry rales, red material around the eyes, excessive salivation, and lacrimation. Similar signs appeared post-exposure in a 14-day observation period. Bilateral mottling of the lungs was observed in eight of ten autopsies (5).

5. Intraperitoneal Injection

- LD50 (mice) = 192 mg/kg (11.3).

* - See Related References 15 and 16 for information on the toxicity of perfluorooctanoic acid.



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B. Extended Studies

1. Oral

* • Biochemical and morphological changes associated with APFO-induced hepatomegaly were studied in rats. Groups of male rats were administered 50 mg/kg/day of APFO for 1, 3, or 7 days. Some rats received $C^{14}$-labeled sodium acetate 24 hours after the last dose. The rats were killed after the last dose or 3 hours after the administration of radiolabeled sodium acetate, and the livers were removed for examination. Liver weights were significantly increased in all APFO-treated rats. The relative amount of DNA/g of liver decreased; however, the total amount of hepatic DNA was not significantly affected. Cytochrome P-450 and benzphetamine-N-demethylase activity were significantly increased by APFO administration. Carnitine-acetyltransferase and carnitine-palmitoyl-transferase activities were also significantly increased. Ethoxyresorufin-O-demethylase activity was decreased after one or three doses. Glutathione-S-transferase and uridine-diphosphoglucuronyl-transferase were not significantly affected by APFO administration. Incorporation of radiolabeled sodium acetate into hepatic lipids except for diacylglycerols, squalene, and sterols containing 27 carbon atoms was significantly increased by APFO administration. Proliferation of the endoplasmic reticulum, mitochondria, and peroxisomes was observed in treated rats. The hepatomegaly induced by APFO is due to hypertrophy rather than hyperplasia. Peroxisome proliferation does not necessarily result in hypolipidemia, as evidenced by the observed increase in lipogenesis in the treated rats (9).

• Administration of ten successive doses of 6.7 mg/kg to six rats caused moderate enlargement of the liver and slight enlargement of kidneys and testes. Simultaneously there was a slight depression of pancreatic weight and the lungs of rats showed slightly enhanced pneumonitis (3.2).

• In a two-week feeding study, 25% Teflon® with APFO dispersing agent was administered in the diet of a group of six rats. After a two-week recovery period the treated animals had slightly heavier livers than the controls or animals receiving Teflon® without the APFO (3.2).

* • C-8 produced an increased incidence of Leydig cell adenomas in rats fed 300 ppm of C-8 for 2 years. A hormonal (non-genotoxic) mechanism was examined since C-8 was negative in short-terms tests for genotoxicity.

Groups of adult male rats were administered C-8 by gavage at doses of 1, 10, 25, or 50 mg/kg/day for 14 days. Also included, in addition to an ad libitum control group, was a pair-fed control group to the 50 mg/kg/day C-8 group. A dose-dependent decrease in body weight and relative accessory sex organ (ASO) weights was seen, with the relative ASO weights of the 50 mg/kg/day group significantly less than those of the pair-fed controls. Serum estradiol levels were elevated in the 10, 25, and 50 mg/kg/day groups. Estradiol levels in the 50 mg/kg/day group were 2.7-fold greater than those of the pair-fed controls. The increase in serum estradiol levels occurred at the same dose levels as the increase in hepatic beta-oxidation activity. A significant downward trend with dose was seen in serum testosterone levels when compared with the ad libitum controls. However, when the 50 mg/kg/day group was compared with their pair-fed controls, no significant differences were seen. Challenge experiments, which can identify the presence and location of a lesion in an endocrine axis, were undertaken to clarify the significance of this downward trend in serum testosterone following the administration of C-8. In this experiment, a group of rats was administered 50 mg/kg/day of C-8 for 14 days. One hour before termination, rats received either a human chorionic gonadotropin (hCG), gonadotropin-releasing hormone (GnRH), or naloxone challenge. Following hCG challenge, serum testosterone levels in the 50 mg/kg/day group were significantly decreased by 50% from those of the ad libitum controls. Similar decreases, although not significant, were seen in serum testosterone following GnRH and naloxone administration to the 50 mg/kg/day C-8 group.

These studies suggest that the decrease in serum testosterone following C-8 administration was due to a lesion at the level of the testis. Androstenedione levels in the C-8 treated rats were decreased 60% from those in the ad libitum controls, suggesting the decrease in serum testosterone following hCG challenge may be due to a decrease in the conversion of 17-alpha-hydroxyprogesterone to androstenedione. The conclusion was that the elevated estradiol levels in the C-8 treated rats may be responsible for the decreased relative



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accessory sex organ weight and serum testosterone levels seen in this study as well as the increased incidence of Leydig cell adenomas observed in a chronic, 2-year feeding study in rats administered C-8 (2).

* • A group of male rats was administered by gavage 25 mg/kg/day of C-8 for 14 days. In addition to an ad libitum control group, a second control was pair-fed to the 25 mg/kg C-8 group. A decrease in body and accessory organ unit weights and an increase in liver weights was seen in the C-8 group when compared with the ad libitum control. When the C-8 group was compared to the pair-fed control group only the liver weight was increased. Although the serum testosterone concentration remained unchanged, the serum estradiol concentration was significantly higher in the C-8 group than in the pair-fed control group (1).

• An increased liver weight/body weight ratio was observed in mice fed 30 ppm of C-8 in their diet for 14 days. When C-8 was combined with an equal amount of nonadecafluorodecanoic acid, a similar effect was produced (3.31).

• Groups of ten mice were fed a diet containing 30, 300 or 3000 ppm of C-8 for 14 days. All the mice fed 3000 ppm of C-8 died. Deaths also occurred at 300 ppm. Body weight loss was indicated at 300 and 3000 ppm. Liver weight/body weight ratios showed a sharp increase over controls in the mice fed the 30 ppm C-8 diet (3.26,8).

• Groups of 32 mice were fed a diet containing 10, 30, 100, 300, 1000, 3000 or 10,000 ppm of C-8 for 14 days. Deaths occurred in the 1000, 3000 and 10,000 ppm groups. Body weight loss was indicated at the end of each week at 300 ppm and more. Liver weight/body weight ratios showed a sharp increase at 10 ppm and were dose-dependent. Clinical signs of toxicity included weakness, tremors, piloerection, pallor, stained perineal area and unkempt appearance (3.17).

• C-8 was administered three times a week for three weeks to rats and mice at three dose levels, 0.1, 1.0 or 10 mg/kg. Weakness followed by death occurred in two female mice dosed at 10 mg/kg. Sporadic weight loss was seen in the female rats and both sexes of mice. A significant liver weight increase was noted in both male and female mice administered 1 and 10 mg/kg. Significantly increased liver weights were also noted in male rats administered 10 mg/kg (3.33).



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- Groups of mice were fed a diet containing 0.01, 0.03, 0.1, 0.3, 1.0, 3, 10 or 30 ppm of C-8 for 21 days. Livers were significantly heavier at 30 ppm, slightly heavier at 3 ppm and appeared normal at 10 ppm. No changes in liver weight were seen in the groups fed 1 ppm or less of C-8 (3.29,8).

- Rats were fed 0-30,000 ppm APFO daily for 28 days. At 10,000 and 30,000 ppm all animals died within the first week. In general, animals showed decreased body weight and an increased liver weight (at >30 ppm, males and >300 ppm, females). Treatment-related histopathologic changes appeared in all test groups. In a similar test using mice all animals dosed at $\geq$ 1000 ppm died within the first two weeks. At doses of $\geq$ 3000 ppm a rough hair coat and muscular weakness were evident in mice. Slight cyanosis, decreased body weight gain and increased average liver weight were also noted (5).

- Groups of ten rats were fed diets containing 10, 30, 100, 300 or 1000 ppm of C-8 for 90 days. No changes considered to be directly related to C-8 were seen in general behavior, appearance or survival. A slight decrease in body weight gain and food consumption was seen for male rats in the 300 and 1000 ppm groups. Hematologic, biochemical and urinalysis values for the female rats showed no changes considered to be related to C-8. A few values obtained for the males showed a slight deviation from the control values (i.e. slightly lower erythrocyte count, and elevated blood urea nitrogen and alkaline phosphatase values). Compound-related gross observations such as enlargement and varying degrees of discoloration on the surface of the liver were observed among male rats in the 1000 ppm group. There were no such observations among female rats from the 1000 ppm group or in males or females from lower dietary levels. Statistically significant variations in sex-group mean organ weights, which were considered compound related, occurred in liver of rats in the 300- and 1000 ppm groups. All other variations were unaccompanied by any morphologic alterations. Microscopically, compound-related lesions were confined to the liver. The lesions consisted of focal to multifocal, very slight to slight, cytoplasmic enlargement of hepatocytes located in centrilobular-midzonal regions of the affected liver lobules, accompanied in some instances by increased amount of yellowish-brown pigments resembling lipofuscin in cytoplasm of hepatocytes and occasionally in sinusoidal lining cells. The incidence and relative severity of the above lesions



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were predominantly among males and more pronounced among rats at 1000 ppm. The other changes recorded in the liver and other tissues were lesions of naturally occurring diseases and they were present in most instances among the control and test rats (5,11.1).

* • Groups of rats were fed diets containing 1, 10, 30, or 100 ppm of C-8 for 13 weeks. Body weight and cumulative body weight gain for the 100 ppm rats were significantly lower than those of the control group. Hepatic palmitoyl CoA oxidase activity was significantly increased in rats administered 30 and 100 ppm of C-8, and rats administered 10 ppm had transiently higher levels. Enzyme levels after 8 weeks of recovery following the 13 weeks of dietary administration were not significantly different from those of the controls. Administration of C-8 caused increased absolute and relative liver weights and hepatocellular hypertrophy in the livers of rats administered 10, 30, or 100 ppm of C-8 after 4, 7, and 13 weeks of treatment. The progression of hepatocellular hypertrophy did not appear to be affected by the length of treatment. The changes observed are suggestive of a compound-related effect on intracellular metabolism and may be associated with peroxisome proliferation. At the end of recovery, these changes were not observed, indicating that the increase in liver weights was reversible. There was no consistent pattern for the hormone determinations which could be attributed to C-8 administration. None of the hormone levels were significantly different from controls although there appeared to be some indication of elevated estradiol for the 100 ppm group at the week 5 determination. The NOAEL was 1 ppm (11.13).

• Groups of four monkeys were administered 3, 10, 30 or 100 mg/kg of C-8 per day for 90 days. The monkeys treated with the higher dose, (100 mg/kg/day) all died during weeks two through five of the study. At the 30 mg/kg/day dosage level, three monkeys died during weeks seven through 12. They all showed signs of toxicity in the gastrointestinal tract (anorexia, emesis, sometimes brown in color, black stools), pale face and gums, swollen face and eyes, slight to severe decreased activity and prostration. The monkeys of the 30 and 100 mg/kg/day dosage levels showed body weight losses from the first week of the study. Because of the early deaths of the monkeys at the 100 mg/kg/day dosage level, the clinical laboratory tests were not conducted. The monkeys at the 30 mg/kg/day dosage level showed, in the first month of the study,



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slight increase in prothrombin time and in activated partial thromboplastin time (A.P.T.T.) values, as well as decreased alkaline phosphatase activity in the serum (statistically significant). Only one monkey from this dosage level in this period showed a low albumin value. At the end of the study, the only remaining monkey from the 30 mg/kg/day dosage level showed apparent anemia, low blood glucose, alkaline phosphatase, total protein and albumin values. There was no mortality at the 10 mg/kg/day dosage level. One monkey had black stool on several days in week 12 and occasionally anorexia and one monkey exhibited pale face and gums. At this dosage level there was a very slight increase in the A.P.T.T. values in the female monkeys during the first month of the study (not statistically significant). There were no changes in the other indices and no changes in the body weight. In single monkeys from the 3 and 10 mg/kg/day dosage levels, there were trends toward decreased alkaline phosphatase in the serum. In the control and the 3 mg/kg/day dosage level there was no mortality, no changes in the body weights and no signs of toxicity. Soft stool, diarrhea or emesis were observed occasionally. The mortality and the above mentioned signs of toxicity in the 30 and 100 mg/kg/day dosage levels were compound-related. There was a trend toward the same signs of toxicity in single monkeys at the 10 mg/kg/day dosage level. The 3 mg/kg/day dosage level seems to be free of signs of toxicity. There is an evident relationship between the administered doses and the degree of the toxicity. No gross or microscopic lesions which were considered compound-related were seen in tissues other than the adrenals, bone marrow, spleen and lymph nodes for male and female monkeys at the 30 and 100 mg/kg/day dosage levels. Microscopically, the adrenals from male and female monkeys at the 30 and 100 mg/kg/day dosage levels have compound-related marked diffuse lipid depletion; the bone marrow from male and female monkeys at the 30 and 100 mg/kg/day dosage levels had compound-related slight to moderate hypocellularity; the spleen and lymph nodes from male and female monkeys at the 30 and 100 mg/kg/day dosage levels had compound related moderate atrophy of lymphoid follicles. Statistically significant variations in sex group mean weights of a few organs occurred between the control and experimental groups. These variations were of unknown biological significance and were not accompanied by morphological alterations (5,11.2).



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* • Groups of 120 male and 120 female rats were fed diets containing either 30 or 300 ppm of C-8 for up to two years, while a control group received only untreated feed. The major in-life findings associated with C-8 administration consisted of a dose-dependent decrease in mean body weight gain and a treatment-related increase in food consumption in males; a slight treatment-related increase in the incidence of ataxia was observed in the females. There was no increase in mortality observed in either treatment group when compared to the control population.

C-8 related hematologic changes seen in the treated rats consisted of decreased red blood cell counts, hemoglobin, and hematocrit values seen at various times throughout the two-year test period. While the decreases in erythrocyte counts were observed very early in the study, this condition did not progress into a generalized anemia by the end of the two-year study.

Histopathologically, C-8 associated toxic changes were found in the liver. These changes were characterized by increased liver weights, increased size of the liver cells with vacuolation of the cytoplasm, and some evidence of hepatocellular degeneration with occasional signs of necrosis. As with the red blood cell findings, these liver changes were noted early in the study and showed very little evidence of progression over the remainder of the two-year study.

The incidence of tumors found in this study was relatively low and the types of neoplasms found were not different from the tumor profiles commonly found in geriatric rats. Hepatocellular tumors were very slightly increased in the high-dose male rats; however, not to the extent that would be expected considering the morphological evidence of chronic hepatocellular stimulation first seen at the one-year necropsy (11.11).

All of the liver tumors found in the above study were carcinomas and the incidence in the control, 30 ppm, and 300 ppm groups (males: 3/50, 1/50, 5/50; females: 0/50, 0/50, 1/50) did not appear to be dose related. The incidence of nodular hyperplasia in the liver (males: 1/50, 0/50, 2/50; females: 0/50, 0/50, 3/50) was also slightly increased, though not statistically significantly (3.35).



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All of the other remarkable tumor incidence values produced in this study were associated with endocrine or endocrine-sensitive organs (3.35):

The incidence of mammary gland fibroadenomas (10/50 (20%), 19/50 (38%), 21/50 (42%)) suggested a compound-related effect. However, when compared to Haskell's historical control incidence for this strain of rat (37%), there does not appear to be any compound-related effect.

The incidence of testicular Leydig cell adenomas (0/50 (0%), 3/50 (6%), 7/50 (14%)) was also suggestive of a compound-related effect. When compared to Haskell's control incidence for this strain of rat (6.1%, range 1-12%), the incidence in the 300 ppm group shows a statistically significant increase.

* • C-8 was included in a mechanistic bioassay investigating extrahepatic tumor induction by compounds which induce peroxisome proliferation. In this study, 300 ppm of C-8 was fed to rats for two years. Increased incidences of combined (single, multiple) hepatic adenomas (10/76 versus 2/80 in ad libitum controls and 3/79 in pair-fed controls), Leydig cell adenomas 8/76 versus 0/79 and 2/78 in control groups), and combined pancreatic acinar cell adenomas (7/76 versus 0/80 and 1/79 in control groups) were noted. The tumor incidences were outside the historical control incidence range for Haskell Laboratory, and in addition, age-adjustment statistics also supported the conclusion that the tumor incidences were elevated for the liver, pancreas, and testis (3.36,11.14).

2. Skin

• Three groups of 15 male rats were exposed dermally for six hours/day, five days/week for two weeks to 20 mg/kg, 200 mg/kg or 2000 mg/kg APFO. At 200 or 2000 mg/kg rats lost weight during the exposure period and showed slight reddening of the skin. Salivation was noted at 2000 mg/kg. Dose-related increases were noted in enzymes monitoring liver function. Liver damage was found in all groups following the tenth dose. During treatment there was an increase in liver weight. Coagulative necrosis of the epidermis at the dose site was noted in two rats dosed at 2000 mg/kg after ten days. Blood organofluoride levels were elevated on the tenth day of exposure. These decreased during a 14-42 day recovery period (3.11,6).



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3. Inhalation

- Groups of 20 rats were exposed, head only, to particular atmospheres of 11 or 83 mg/m³ of C-8, six hours a day, five days a week for two weeks. The exposed rats showed a dose-related suppression of body weight. Liver injury or dysfunction was suggested by dose-related elevations of plasma enzymes for up to 28 days after the last exposure. Liver degeneration was detected histologically at the end of the exposure period. These liver effects were not observed after 14, 32 or 42 days of recovery. Eye examinations revealed no compound-related effects (3.7).

- Groups of rats were exposed six hours a day, five days a week for two weeks to 1.0, 7.6 or 83.9 mg/m³ of C-8. Observations of clinical signs during exposure showed only slight nasal and ocular discharge. However, after three to four days on test at 83.9 mg/m³ one rat died during exposure and another was sacrificed in extremis. Both of these rats had lost a considerable amount of weight. Body weight of the 1.0 mg/m³ groups were similar to those of the controls while the body weights of the 7.6 mg/m³ group were significantly higher. Organ to body weight ratios demonstrated a dose-related significant increase in lung, liver and testes weight immediately after exposure ended. The liver/body weight ratios were significantly higher in the 83.9 mg/m³ group through a 28-day recovery period. Clinical laboratory measurements demonstrated an increase in serum alkaline phosphatase in all exposure groups after ten exposures, which persisted in the 83.9 mg/m³ group through 14 days of recovery. Pathological evaluation revealed heavy livers and microscopic lesions in the 7.6 and 83.9 mg/m³ groups. These changes were reversible following a 28-day recovery period. Blood fluoride analysis clearly demonstrated a dose-related presence of C-8 in all groups including the controls. The amount of C-8 in blood decreased with time, but was detected after 84 days recovery in both controls and the 83.9 mg/m³ group (3.13,7).

* • Repeated exposure of rats to C-8 produced liver changes and elevated organofluoride levels (11.12).

C. Carcinogenic Potential

* • In chronic, 2-year feeding studies in rats, C-8 produced an increased incidence of tumors in the liver, pancreas, and testis (3.35,3.36,11.11,11.14).



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* • See Related References 17-23 for information on the hepatocarcinogenicity of perfluorooctanoic acid.

D. Mutagenic Potential

- APFO was tested in microbial assays using Salmonella typhimurium strains TA 98, TA 100, TA 1535, TA 1537 and TA 1538 and Saccharomyces cerevisiae strain D-4. All tests were negative with and without activation (5,12).

* • C-8 had a low order of cytotoxicity in C3H 10T-1/2 cells and no evidence of transformation was observed (11.8).

* • See Related Reference 24 for information on the mutagenicity of perfluorooctanoic acid.

E. Developmental/Reproductive Toxicity

- The oral administration of APFO at 25, 50, 75, 100 or 150 mg/kg/day to rats during the period of organogenesis (days six through 15 of gestation) did not result in any deaths. A toxic effect of reduced body weight gain occurred between days six and nine of gestation in the 150 mg/kg/day dose group. The two nonpregnant 150 mg/kg/day rats had a more severe effect on body weight on day nine of the study than the pregnant high dose dams. They lost a considerable amount of weight and one was observed to have urinary incontinence on days 11, 12, and 13. The pregnant dams of the 25, 50, 75, and 100 mg/kg/day dose groups did not have abnormal clinical signs and gained weight at comparable levels to the control group. Four fetuses were examined from each of four dams in the 25 and 150 mg/kg/day dose groups for eye changes. All of the readable fetuses sectioned had eye changes consisting of one or more of the following: large lens cleft, dark streak running one-half to three-quarters of the way through the lens or disorganized lens fibers. The lens abnormalities occurred in the same location as those observed in the two previous teratology studies on chemically related compounds. The abnormalities in this study appeared more pronounced than in the previous studies. In the previous studies, the teratogenic effect was a developmental eye abnormality which appeared to be an arrest in development of the primary lens fibers forming the embryonal lens nucleus, followed by secondary aberrations of the secondary lens fiber of the fetal nucleus. The same general morphological changes occurred in this study (11.6).

- Groups of pregnant rats were administered 0.05, 1.5, 50 or 100 mg/kg of C-8 on days 6-15 of gestation. Three rats in the high dose group died. C-8 caused low mean body weights in this group. No teratogenic or embryotoxic effects were found (11.7).

- Pregnant rats were administered 100 mg/kg/day by gavage on days 6-15 of gestation. Maternal deaths occurred and during the dosing period, the surviving dams gained about one-third less body weight than the control dams. External, visceral, and skeletal alterations were sought, and the eyes of several fetuses of each litter in both groups were examined stereoscopically and histologically for alterations. The only finding noted that could possibly be C-8 related was an increased incidence of fetuses with ossification sites on the first lumbar vertebrae versus the incidence in the control group. This difference in incidence was statistically significant only if analyzed by a one-tailed test. Its presence was probably a response to generalized stress evoked by the toxic state of the dams. The postpartum viability, growth rate, and development of the offspring from additional dams given C-8 were not demonstrated to be adversely affected by the C-8 administered. Criteria of development included examination of the pups for external alterations and ophthalmoscopic examination of the eyes (3.25,10).

- Groups of pregnant rabbits were administered 1.5, 5 or 50 mg/kg of C-8 in distilled water on days 6-18 of gestation. At 50 mg/kg the dams lost more weight than the untreated controls. No teratogenic effects were observed (11.9).

- Groups of pregnant rats were exposed for six hours a day on days 6-15 of gestation to 0.14, 1.2, 9.9 or 21 mg/m$^3$ of C-8. Maternal deaths occurred at the 21 mg/m$^3$ concentration and overt toxicity was evident among the surviving dams and among those of the 9.9 mg/m$^3$ group. No teratogenic response was observed in any of the exposed groups. Embryofetal toxicity was observed only in the 21 mg/m$^3$ group. Other than a temporary reduction in the body weight of the pups from the 21 mg/m$^3$ group, no adverse effects were seen in the offspring (3.24,10).

## F. Metabolism

### Animal Studies

- Sex and species differences in the excretion and disposition of radiolabeled C-8 were observed in a study in rats, mice, hamsters, and rabbits. The



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female rat and the male hamster excreted over 99% of the administered dose by 120 hours after dosing. Conversely, the male rat and the female hamster excreted 39 and 60% of the administered dose by 120 hours post-dosing. Both sexes of rabbits excreted the C-8 as rapidly and completely as the female rat and male hamster. The male and female mice excreted only 21% of the administered dose in 120 hours (3.28).

* • Male and female rats were administered radiolabeled C-8 by intravenous injection. Females had excreted essentially 100% of the administered dose within 24 hours while the males excreted only 20% of the administered dose. Radioactive tissue residues were not detectable after 17 days in the females, while at 36 days, male rats had 2.8% of the carbon-14 in the liver, 1.1% in plasma, and lower but still detectable amounts in other organs (11.5).

• The uptake and clearance of C-8 from the blood of female rats following a single oral dose was rapid with the peak reached one-two hours post-treatment and with virtual total clearance by 24 hours. A dose-response was demonstrated with no apparent changes in blood C-8 levels following multiple oral dosing. The slower clearance rate in male rats was demonstrated following a single oral dose. The same general statements apply following inhalation exposure. A single six-hour inhalation exposure resulted in peak blood levels within one hour after cessation of exposure, the material rapidly cleared from the blood, the number of exposures did not affect blood levels, and male rats cleared the compound much more slowly. Pregnant and non-pregnant rats showed similar C-8 blood levels following either oral or inhalation exposures. Specific data follows (3.20):

<u>Oral administration, female rats, C-8 levels as a function of time post-dosing.</u>

C-8 levels of 14 ppm were seen 15 minutes following the dose (25 mg/kg). These levels rose to a peak of approximately 30 ppm at one to two hours, dropped to 26 ppm by eight hours and to 0.7 and 0.045 ppm at 24 and 168 hours, respectively.

Conclude: C-8 is absorbed and rapidly cleared from the blood of female rats given a single oral dose.

Oral administration, female rats, C-8 levels as a function of dose.

C-8 levels at 1/2 hour following treatment ranged from 3 to 162 ppm, doses administered 2.5 to 150 mg/kg. The same dose response was seen at 24 hours with blood values ranging from 0.12 (2.5 mg/kg) to 18 (150 mg/kg) ppm. The response was linear.

Conclude: C-8 in blood is directly related to the amount of C-8 administered orally.

Oral administration, male and female rats following a single oral dose of 25 mg/kg.

C-8 levels in female rats were 16 ppm (1/2 hour), 26 ppm (eight hours), 0.7 ppm (24 hours) and 0.045 ppm (168 hours). The corresponding values for male rats were 23, 63, 50 and 23 ppm.

Conclude: C-8 is retained in the blood of male rats to a greater extent than female rats following oral treatment.

Oral administration, female rats, C-8 levels as a function of number of doses.

Blood levels in female rats given one versus 11 oral doses of C-8 were not considerably different. Concentrations at 1/4 hours post-treatment were 14 and 17 ppm, one and 11 doses, respectively.

At 1/2 hour C-8 concentrations were 16 and 25 ppm; at eight hours, 26 and 13 ppm; at 24 hours, 0.7 and 0.8 ppm; and at 168 hours, 0.045 and 0.10 ppm, one and 11 doses, respectively.

Conclude: C-8 does not appear to accumulate in the blood of female rats following repeated oral treatment. The number of treatments does not seem to influence the C-8 blood level with dose remaining constant.

Inhalation exposures, female rats, C-8 levels as a function of time post-exposure.

C-8 levels of 96 ppm were seen 15 minutes following a single six-hour inhalation exposure to 10 $mg/m^3$. This level stayed around 100-110 ppm through one hour, fell to approximately 70 ppm at eight hours, further decreased to 52 ppm at 24 hours and dropped to 0.39 ppm 168 hours later. This same general pattern was seen in rats exposed to either 0.1 or 1 $mg/m^3$. The lag phase seen in oral exposures is not





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seen here due to blood sampling following a six-hour inhalation exposure (rather than a single oral dose at a given, finite time).

Conclude: C-8 is absorbed and rapidly cleared from the blood of female rats given a single six-hour inhalation exposure.

Inhalation exposures, female rats, C-8 levels as a function of dose.

C-8 levels at 1/2 hour following a single six-hour inhalation exposure ranged from 2 to 100 ppm, airborne concentrations of 0.1 to 10 mg/m$^3$. The same dose response was seen at two hours (2, 17, 69 ppm), eight hours (0.85, 4, 71 ppm) and 24 hours (0.14, 0.56, 52 ppm) - concentrations corresponding to 0.1, 1 and 10 mg/m$^3$. The response is linear at 1/2 hour (correlation coefficient of 0.999).

Conclude: C-8 in blood is directly related to the amount of C-8 inhaled. At the highest level used, the clearance rate is somewhat slower than seen at the lower levels. This suggests massive overloads in the clearance system.

Inhalation exposures, male and female rats following a single six-hour inhalation of 10 mg/m$^3$.

C-8 levels in female rats were 109 ppm (1/2 hour), 69 ppm (two hours), 71 ppm (eight hours), and 52 ppm (24 hours). The corresponding values for male rats were 137, 157, 182 and 147 ppm.

Conclude: C-8 is retained in the blood of male rats to a greater extent than female rats following inhalation exposure.

Oral administration to pregnant and non-pregnant female rats, C-8 levels following a single oral dose.

C-8 levels in both pregnant and non-pregnant rats following a single 25 mg/kg oral dose were essentially the same 16 and 10 ppm at 1/2 hour, 33 and 39 ppm at two hours, 26 and 31 ppm at eight hours, non-pregnant and pregnant rats, respectively.

Conclude: C-8 clearance following oral dosing is not altered in pregnant vs. non-pregnant rats.



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Inhalation and oral treatment, pregnant rats, C-8 levels as a function of number of doses.

Blood levels in pregnant rats given either one, six or ten doses of C-8 were essentially the same 1/2 and 24 hours following the last oral treatment. At 1/2 hour the levels were 18, 12 and 12 ppm, one, six and ten treatments, respectively. When comparing the levels seen at two and eight hours following a series of ten consecutive doses, there appears to be a lowering of C-8 blood concentrations in the rats given ten doses (15 compared to 25 ppm at two hours, 11 compared to 31 at eight hours). This may indicate an enhanced clearance of C-8 with multiple dosing but would require confirmation prior to acceptance as fact. Blood levels following one or ten consecutive six hours/day inhalation exposures were not different.

Conclude: C-8 does not appear to accumulate in the blood of pregnant rats following repeated oral or inhalation exposures.

- Radiolabeled C-8 was transferred from maternal blood to the fetuses of pregnant rats. A single 10 mg/kg dose was administered orally on day 19 of gestation. Maternal blood and placental levels of C-8 increased between two and four hours after dosing then decreased between four and eight hours after dosing (3.27).

- In a previous study, Dowex® Ion Exchange Resin was able to reduce the acute lethal effects of C-8. In this study, rats and mice were given Dowex® resin 24 hours after dosing with C-8. No signs of enhanced elimination of C-8 via the feces, urine or exhaled air was seen (3.30).

* - See Related References 26-39 for information on the metabolism of perfluorooctanoic acid. Also see Related References 61 and 62 for analytical methods for the determination of C-8 and perfluorooctanoic acid in biological samples.

Human Studies

* - Perfluorooctanoic acid has a long half-life in humans. A study of occupationally exposed workers at a plant which produces C-8 showed organic fluorine levels in the blood ranging from 1 to 71 ppm. One worker with a level of 70 ppm was removed from the fluorochemical production area and his blood analyzed for organic fluorine over several months. After 18 months, his organic fluorine level had decreased only to 39 ppm (45% reduction) (12).



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G. Biochemical Studies

* • In ex vivo studies, Leydig cells were isolated from rats following 14 days of dosing. The hCG-stimulated Leydig cells from the C-8-treated rats had statistically greater increases in testosterone production than in controls. In contrast, cultured Leydig cells treated in vitro with C-8 produced a dose-dependent decrease in testosterone and at 500 uM produced an increase in estradiol (1).

* • See Related References 40-60 for information on peroxisome proliferation and oxidative phosphorylation of perfluorooctanoic acid.

H. Clinical Reports of Human Exposure

* • Health screening examinations were offered to the employees of a 3M plant that produced C-8 as well as other fluorochemicals. No health problems related to exposure to fluorochemicals were encountered among those examined. Additionally, no relationship was observed between deviations from normal between laboratory test results and blood levels of organic fluorine (the liver enzyme SGGT was the most frequently encountered test result exceeding the normal range). C-8 exposure levels ranged from 0.03 to 7.6 $mg/m^3$ (12).

* • A study was made of Washington Works employees potentially exposed to C-8. Results of blood chemistry testing (SGOT, LDH, AP, and bilirubin) indicated no conclusive evidence of an occupationally-related health problem among workers exposed to C-8 (3.38).

I. Epidemiology

• A retrospective cohort mortality study was made of employees at a 3M plant where C-8, along with other fluorocompounds, is manufactured. Records on 4218 employees were reviewed. Only those who worked for six months or more (3688 workers) were included in the mortality follow-up. Of the 180 known deaths, 177 death certificates were obtained. Overall the number of deaths was significantly less than expected. The observed-to-expected ratio for cancer deaths was 1.0 (11.4,12).

* • In a retrospective cohort mortality study, the relationship between mortality and employment at a perfluorooctanoic acid (PFOA) production plant were investigated. The cohort consisted of 2788 male and



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749 female workers employed between 1947 and 1983. The all-causes standardized mortality rate (SMR) was 0.75 for men and 0.77 for women. Among men, the cardiovascular SMR was 0.68 and the all-gastro-intestinal diseases SMR was 0.57. There was no significantly increased cause-specific SMR for either men or women. The SMRs for prostate cancer were 2.03 in the Chemical Division group (exposed) and 0.58 in the non-Chemical Division group (not exposed to chemicals). In the Chemical Division group there were 4 observed and 2 expected deaths from prostate cancer. There was no significant association between any cause of death and latency in either group. In the Chemical Division group, the SMR for prostate cancer was 1.61 in the greater than 15-year latency group. For all men employed at this plant, 6 deaths were recorded for prostate cancer versus 6 expected. Further research is needed to evaluate and confirm the association between PFOA and prostate cancer (4).

J. Aquatic/Environmental Studies

* • 96-hour LC50 (bluegill sunfish) = 569 mg/L (11.12).

* • 96-hour LC50 (bluegill sunfish) = 634 mg/L (3.37).

* • 96-hour LC50 (fathead minnows) = 766 mg/L (11.12).

• 48-hour LC50 (bluegill sunfish) = 1500 mg/L (3.4).

• 48-hour LC50 (snails) = 820 mg/L (3.4).

* • 48-hour EC50 (Daphnia magna) = 632 mg/L (11.12).

• 50% growth reduction (diatoms) = 2400 mg/L; safe level (seven days) = 720 mg/L (3.4).

• 14-day EC50 (green alga, Selenastrum capricornutum) = 73 mg/L (11.12).

• COD = Nil; 20-day BOD = Nil (11.12).

• The Soil Absorption Coefficient (Koc) is 17; this indicates very high mobility in a sandy loam soil (11.12).



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REFERENCES


1. Biegel, L. B. et al., The Toxicologist, 13(1):399 (Abstract 1563) (1993).

2. Cook, J. C. et al., Toxicol. Appl. Pharmacol., 113(2):209-217 (1992).

3. DuPont Co., Haskell Laboratory Data:

   1. MR-604-7, HL-55-61
   2. MR-604-7, HL-56-61
   3. MR-639-1, HL-123-65
   4. Acad. Nat. Sci. Philadelphia Data (1968) (C-1782)
   5. MR-1070-1, HL-128-68
   6. MR-1198-1, HL-160-69
   7. MR-2753-1, HL-253-79
   8. MR-3567-1, HL-635-79
   9. MR-3567-1, HL-636-79
   10. MR-3567-1, HL-659-79
   11. MR-3567-1, HL-589-80
   12. MR-5342-1, HL-682-80
   13. MR-3692-1, HL-205-81
   14. MR-5342-1, HL-291-81
   15. MR-5342-1, HL-295-81
   16. MR-5342-1, HL-329-81
   17. MR-5418-1, HL-560-81
   18. MR-5342-1, HL-565-81
   19. MR-5342-1, HL-567-81
   20. MR-4140-1, MR-4147-1, and MR-4152-1, HL-593-81
   21. MR-5342-1, HL-600-81
   22. MR-4198-1, HL-612-81
   23. MR-5343-1, HL-828-81
   24. MR-4129-1, HL-881-81
   25. MR-4130-1, HL-1-82
   26. MR-5418-1, HL-12-82
   27. MR-4275-1, HL-61-82
   28. MR-4153-1, HL-62-82
   29. MR-5418-1, HL-323-82
   30. MR-4370-1, HL-405-82
   31. MR-5343-1, HL-537-82
   32. MR-5454-1, HL-788-82
   33. MR-5454-1, HL-138-83
   34. MR-5343-1, HL-79-84
   35. Letter, G. P. Sykes to C. F. Reinhardt, dated 10-29-87 (AEL File)
   36. MR-5686-1
   37. MR-9309-1
   38. Report dated 1-15-81, W. E. Fayerweather, Epidemiology Division of Corporate Medical (C-4124)

4. Gilliland, F. D. and J. S. Mandel, J. Occup. Med., 35(9):950-954 (1993).





EID087816

HDR001744

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REFERENCES (CONT'D)


5. Griffith, F. D. and J. E. Long, Am. Ind. Hyg. Assoc. J., 41(8):576-583 (1980).

6. Kennedy, G. L., Jr., Toxicol. Appl. Pharmacol., 81(2):348-355 (1985).

7. Kennedy, G. L., Jr. et al., Food Chem. Toxicol., 24(12):1325-1329 (1986).

8. Kennedy, G. L., Jr., Toxicol. Lett., 39(2-3):295-300 (1987).

9. Pastoor, T. P. et al., Exp. Mol. Pathol., 47(1):98-109 (1987).

10. Staples, R. E. et al., Fundam. Appl. Toxicol., 4(3,Part 1):429-440 (1984).

11. 3M Co. Data.

    1. IRDC Report No. 137-089 (1978) (J-3546)
    2. IRDC Report No. 137-090 (1978) (J-3545)
    3. Personal communication, J. Long to P. J. Gort (1/8/79) (C-4124)
    4. Schuman, L. M. and J. S. Mandel, Epidemiology Associates Report (1980) (Submitted to EPA on 3-22-82, See Letter W. H. Pearlson to T. O'Bryan) (C-4124)
    5. Riker Laboratories, Drug Metabolism Report 1-20 (1980) (Cited in Reference 11.11)
    6. Report M-601 (1981)
    7. Report No. 0681TR0110 (1981) (J-5918)
    8. Environmental Pathology Laboratory, Dept. Lab. Med. Path., Stone Research Laboratories (1981) (J-4124)
    9. Gortner, E. G. et al., Riker Laboratories Data (1982) (C-4124)
    10. Hazleton Laboratories America, Inc., Report Dated 2-6-87 (Submitted to EPA, TSCA8eCAP, Fiche 536984)
    11. Riker Laboratories, Inc., Report 0281CR0012 (1987) (J-7446)
    12. MSDS (1993) (Cited in DuPont MSDS No. 25617211)
    13. Hazleton Wisconsin, Inc., Report HWI-6329-100 (1993) (J-9878)
    14. EPA Submission (8e Notification Letter dated 10-11-93 (AEL File)

12. Ubel, F. A. et al., Am. Ind. Hyg. Assoc. J., 41(8):584-589 (1980).





EID087817

EID087817

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RELATED REFERENCES

Oral

13. Kojo, A. et al., Arch. Toxicol., Suppl. 9:465-468 (1986).

"Toxicity and kinetics of perfluorooctanoic acid in the Wistar rat."

Skin

14. DuPont Co., Haskell Laboratory Data, MR-4234-1, HL-736-81.

"FC-143 occluded skin test on rabbits."

Injection Studies

15. Olson, C. T. and M. E. Andersen, Toxicol. Appl. Pharmacol., 70(3):362-372 (1983).

"The acute toxicity of perfluorooctanoic acid and perfluorodecanoic acid in male rats and effects on tissue fatty acids."

16. Smith, F. A. et al., Toxicol. Appl. Pharmacol., 2:54-58 (1960).

"Screening of fluorine-containing compounds for acute toxicity."

Carcinogenic Potential

17. Abdellatif, A. G. et al., Arch. Int. Physiol. Biochim., 98(5):113 (1990) (BIOSIS/91/14278).

"The promoting activity of perfluorooctanoic acid. A novel promoter of rat liver carcinogenesis."

18. Abdellatif, A. G. et al., Carcinogenesis, 11(11):1899-1902 (1990).

"Peroxisome proliferation and modulation of rat liver carcinogenesis by 2,4-dichlorophenoxyacetic acid, 2,4,5-trichlorophenoxyacetic acid, perfluorooctanoic acid, and nafenopin."

19. Abdellatif, A. G. et al., Toxicol. Appl. Pharmacol., 111(3):530-537 (1991).

"The modulation of rat liver carcinogenesis by perfluorooctanoic acid, a peroxisome proliferator."



EID087818

HDR001746

000235

RELATED REFERENCES (CONT'D)

Carcinogenic Potential (Cont'd)

20. Nilsson, R. et al., Chem.-Biol. Interact., 78(2):235-250 (1991).

"On the mechanism of the hepatocarcinogenicity of peroxisome proliferators."

21. Revici, E., U.S. Patent No. 4,624,851 (1986) (CA 106:169039n).

"Treatment of symptoms of neoplastic diseases."

22. Takagi, A. et al., Cancer Lett., 57(1):55-60 (1991).

"Short-term exposure to the peroxisome proliferators, perfluorooctanoic acid and perfluorodecanoic acid, causes significant increase of 8-hydroxydeoxyguanosine in liver DNA of rats."

23. Takagi, A. et al., J. Environ. Pathol. Toxicol. Oncol., 11(3):145-149 (1992).

"Hepatomegaly is an early biomarker for hepatocarcinogenesis induced by peroxisome proliferators."

Mutagenic Potential

24. Kubinski, H. et al., Mutat. Res., 89:95-136 (1981).

"DNA-cell binding (DCB) assay for suspected carcinogens and mutagens."

Developmental Toxicity

25. Gombar, V. K. et al., Quant. Struct.-Act. Relat., 10(4):306-332 (1991) (CA 117:24988).

"A QSAR model of teratogenesis."

Metabolism

26. Goecke, C. M. et al., Chem. Res. Toxicol., 5(4):512-519 (1992).

"A comparative toxicological investigation of perfluorocarboxylic acids in rats by fluorine-19 NMR spectroscopy."



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HDK001747

000033

RELATED REFERENCES (CONT'D)

Metabolism (Cont'd)


27. Hanhijarvi, H. et al., Proc. Soc. Exp. Biol. Med., 171:50-55 (1982).

"The sex-linked difference in perfluorooctanoate excretion in the rat."

28. Hanhijarvi, H. et al., Pharmacol. Toxicol., 61(1):66-68 (1987).

"Elimination and toxicity of perfluorooctanoic acid during subchronic administration in the Wistar rat."

29. Hanhijarvi, H. et al., New Developments in Biosciences: Their Implications for Laboratory Animal Science; The Third Symposium of the Federation of European Laboratory Animal Science Associations, Amsterdam, pp. 409-412 (1988) (BIOSIS/88/23936).

"A proposed species difference in the renal excretion of perfluorooctanoic acid in the beagle dog and rat."

30. Permadi, H. et al., Biochem. Pharmacol., 44(6):1183-1191 (1992).

"Effects of perfluoro fatty acids on xenobiotic-metabolizing enzymes, enzymes which detoxify reactive forms of oxygen and lipid peroxidation in mouse liver."

31. Rozner, M. A. and D. R. Taves, Fed. Proc., 40(3, Part 1):675 (1981).

"A mechanism for lipid peroxidation by perfluorooctanoic acid."

32. Singer, L. and R. Ophaug, Crit. Rev. Clin. Lab. Sci., 18(2):111-140 (1982).

"Ionic and nonionic fluoride in plasma (or serum)."

33. Vanden Heuvel, J. P. et al., Lipids, 24(6):526-531 (1989) (TOXBIB/89/364052).

"Isolation and purification of perfluorodecanoic and perfluorooctanoic acids from rat tissues."

34. Vanden Heuvel, J. P. et al., J. Biochem. Toxicol., 6(2):83-92 (1991) (TOXBIB/92/045858).

"Tissue distribution, metabolism, and elimination of perfluorooctanoic acid in male and female rats."

RELATED REFERENCES (CONT'D)

Metabolism (Cont'd)

35. Vanden Heuvel, J. P. et al., J. Biochem. Toxicol., 7(1):31-36 (1992) (TOXBIB/92/269270).

"Renal excretion of perfluorooctanoic acid in male rats: Inhibitory effect of testosterone."

36. Venkateswarlu, P. et al., J. Assoc. Off. Anal. Chem. Int., 75(4):672-677 (1992) (BIOSIS/93/04091).

"Direct determination of individual organic fluorine compounds by aluminum monofluoride molecular absorption spectrometry."

37. Ylinen, M. et al., Arch. Environ. Contam. Toxicol., 14(6):713-718 (1985).

"Quantitative gas chromatographic determination of perfluorooctanoic acid as the benzyl ester in plasma and urine."

38. Ylinen, M. et al., Pharmacol. Toxicol., 65(4):274-277 (1989).

"Stimulation by oestradiol of the urinary excretion of perfluorooctanoic acid in the male rat."

39. Ylinen, M. et al., Bull. Environ. Contam. Toxicol., 44(1):46-53 (1990).

"Disposition of perfluorooctanoic acid in the rat after single and subchronic administration."

Biochemical Studies

40. Ellenbogen, E. and P. H. Maurer, Congr. Intern. Biochem., Resumes Commons., 3rd Congr., Brussels, p. 12 (1955) (CA 50:16895e).

"Heat denaturation of serum albumin in presence of perfluorooctanoic acid."

41. Handler, J. A. et al., Toxicol. Lett., 60(1):61-68 (1992).

"Induction of peroxisomes by treatment with perfluorooctanoate does not increase rates of hydrogen peroxide production in intact liver."



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000244

RELATED REFERENCES (CONT'D)

Biochemical Studies (Cont'd)


42. Hasegawa, R. et al., Free Radical Biol. Med., 9(Suppl. 1):50 (1990) (BIOSIS/91/07820).

"Oxidative DNA damage in rat liver and kidney induced by peroxisome proliferators or ferric nitrilotriacetate."

43. Intrasuksri, U. and D. Feller, Fed. Am. Soc. Exp. Biol., 5(6):A1571 (1991).

"Characteristics of peroxisome proliferation by pefluorinated octanoic acid in cultured rat hepatocytes."

44. Kawashima, Y. et al., Biochem. Pharmacol., 42(10):1921-1926 (1991).

"Induction by perfluorooctanoic acid of microsomal 1-acylglycerophosphocholine acyltransferase in rat kidney: Sex-related difference."

45. Kawashima, Y. and H. Kozuka, Toxicology, 71(1-2):151-160 (1992).

"Cytosolic long-chain acyl-CoA hydrolase, a suitable parameter to measure hepatic response to peroxisome proliferators."

46. Keller, B. J. et al., Biochim. Biophys. Acta, 1102(2):237-244 (1992) (BIOSIS/93/00333).

"Several nongenotoxic carcinogens uncouple mitochondrial oxidative phosphorylation."

47. Keller, B. J. et al., Toxicology, 71(1/2):49-61 (1992).

"Inhibition of mitochondrial respiration and oxygen-dependent hepatotoxicity by six structurally dissimilar peroxisomal proliferating agents."

48. Klevens, H. B. and E. Ellenbogen, Discussions Faraday Soc., 18:277-288 (1954) (CA 49:14430h).

"Protein-fluoro acid interaction-bovine serum albumin-perfluorooctanoic acid."

49. Levitt, D. and A. Liss, J. Toxicol. Environ. Health, 20(3):303-316 (1987).

"Perfluorinated fatty acids alter merocyanine 540 dye binding to plasma membranes."

RELATED REFERENCES (CONT'D)

Biochemical Studies (Cont'd)


50. Levitt, D. and A. Liss, Toxicol. Appl. Pharmacol., 86(1):1-11 (1986).

"Toxicity of perfluorinated fatty acids for human and murine B cell lines."

51. Sohlenius, A.-K. et al., Biochem. J., 285(3):779-783 (1992) (BIOSIS/92/30087).

"The effects of perfluorooctanoic acid on hepatic peroxisome proliferation and related parameters show no sex-related difference in mice."

52. Sohlenius, A.-K. et al., J. Biochem. Toxicol., 7(4):205-212 (1992) (BIOSIS/93/13220).

"Perfluorooctanoic acid has persistent effects on peroxisome proliferation and related parameters in mouse liver."

53. Sohlenius, A.-K. et al., Pharmacol. Toxicol., 72(2):90-93 (1993).

"Perfluorooctane sulfonic acid is a potent inducer of peroxisomal fatty acid beta-oxidation and other activities known to be affected by peroxisome proliferators in mouse liver."

54. Thottassery, J., Fed. Am. Soc. Exp. Biol., 4(3):A571 (1990).

"Do hormones play a role in peroxisomal enzyme induction - A study on the effects of perfluorooctanoic acid in adrenalectomized rats."

55. Thottassery, J. et al., Hepatology, 15(2):316-322 (1992) (TOXBIB/92/137911).

"Regulation of perfluorooctanoic acid--induced peroxisomal enzyme activities and hepatocellular growth by adrenal hormones."

56. Uy-Yu, N. et al., J. Pharmacobio.-Dyn., 13(9):581-590 (1990) (BIOSIS/91/03735).

"Effects of chronic administration of perfluorooctanoic acid on fatty acid metabolism in rat liver: Relationship among stearoyl-coenzyme A desaturase, 1-acylglycero-phosphocholine acyltransferase, and acyl composition of microsomal phosphatidylcholine."





EID087823

000213

EID00173

RELATED REFERENCES (CONT'D)

Biochemical Studies (Cont'd)


57. Uy-Yu, N. et al., Biochem. Pharmacol., 39(9):1492-1495 (1990) (NIOSH/00197121).

"Comparative studies on sex-linked difference in biochemical responses of livers to perfluorooctanoic acid between rats and mice."

58. Vanden Heuvel, J. P. et al., Chem. Biol. Interact., 82(3):317-328 (1992).

"Covalent binding of perfluorinated fatty acids to proteins in the plasma, liver, and testes of rats."

59. Whitehouse, M. W. and I. F. Skidmore, Biochem. Pharmacol., 16:911-915 (1967).

"Uncoupling of oxidative phosphorylation by some fluoro-compounds, notably perfluoropinacol."

60. Wigler, P. W. and Y. B. Shah, Toxicol. Appl. Pharmacol., 85(3):456-463 (1986).

"Perfluorodecanoic acid inactivation of a channel for 2-aminopurine in the L5178Y cell membrane and recovery of the channel."


Analytical Methods


61. Belisle, J. W., 3M Co. Data (1978).

"Analysis of serum for FC-143."

62. Belisle, J. and D. F. Hagen, Anal. Biochem., 101(2):369-376 (1980) (HEEP/80/08621).

"A method for the determination of perfluorooctanoic acid in blood and other biological samples."


S. W. Snyder
November 24, 1975

Updated by:
Kimberly A Iverson:md
April 16, 1981

Richard C. Graham:md:
July 23, 1984 - INF19.4
June 1, 1994 - DATA41.14

R. C. Graham

DUPONT
WASHINGTON WORKS
P.O. Box 1217
PARKERSBURG, WV 26102-1217

DU PONT POLYMERS

Office of Env. Enforcement
Div. of Env. Protection
2311 Ohio Avenue
Parkersburg, WV 26101

John G. Britvec
Office of Water Resources
Industrial Waste Section
1304 Goose Run Road
Fairmont, WV 26554

July 8, 1994

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Mr. Mark A. Scott, Chief
Office of Water Resources
Division of Environmental Protection
1201 Greenbrier Street
Charleston, West Virginia 25311

Attention: Industrial Waste Section

REF: WV/NPDES Permit Application WV0076244

Dear Mr. Scott:

Enclosed is the original and four copies of our WV Solid Waste/NPDES permit application for the Dry Run Landfill. This application is being submitted for renewal of the referenced permit issued on January 12, 1990.

The attached permit application does not include an approval letter from the Historical Preservation Office of the West Virginia Division of Culture and History. The results of an archaeological survey of the area were submitted to the Historical Preservation Office on June 10, 1994. The report indicates that there are no areas of a historical significance in the vicinity of the landfill. We will send you the approval letter from the Historical Preservation Office when it is received.

Also, we have been working to obtain the storm event NPDES samples required for the permit application, but weather conditions have delayed completion of the sampling at the landfill. The results will be forwarded to you when they are available.

A check for the $1,000.00 renewal application fee is attached. A signed affidavit for the Statement for Billing for the costs of publishing legal advertisement is also enclosed.

If you have any questions or need additional information, please call me on 863-4271.

Very truly yours,

W. M. Stewart

W. M. Stewart
Sr. Environmental Control Consultant
Washington Works

Attachments
/vlw (6858-1)

EID053308

I N T E R O F F I C E  M E M O R A N D U M

Date: 13-Sep-1994 09:51am EDT
From: RITCHERL
RITCHERL@ISCDCVM1@CDCIL1@M
Dept:
Tel No:

TO: PLAYTIS@A1

Subject: Ducatman

VMSmail To information: WWPS::PLAYTIS

Date: 09/13/94 09:30:49
To: ZIPFEL --WWPS MULHERFP--JLCL01
PLAYTIS --WWPS VALENTR --HSKVAX

From: Bob Ritchey, FLPR SHEA, CRP711/130, 999-2870
Subject: Ducatman

After our conference call yesterday, I called Judy Walrath back because I was curious for more information on the off-handed comment she had made that she had attended a conference and heard that "PFOA was on a fast-track as a potential carcinogen".
Judy indicates that one of the co-authors of the JOM article associating PFOA production at the 3M plant to prostate cancer (the death certificate study) presented that paper at the National Cancer Institue's late April '94 conference on Occupational and Urogenital Cancers. Judy attended. The pape was presented without any disclaimers or qualifiers or limitations and Judy had to bite her tongue to avoid challenging it on the flaws we saw in it for fear of being branded the industry response. Aaron Blair, epidemiologis with NCI and known to be very interested in occupational cancers, then acknowledged the paper in front of the audience as an excellent paper. A person in attendance was Alan Ducatman, who is with WVU. He called Dr. Karrh to and indicated to him his feeling that PFOA was on a fast-track to being identified as a potential carcinogen. Dr. Karrh alerted Judy (see attached old email), who called Dr. Zobel at 3M who indicated he would call Ducatman.
We don't know what came of that call, but have not heard anything more from Ducatman or anyone else.
I've asked Judy to contact Dr. Zobel or Jeff Mandel with 3M and see what came of the followup with Ducatman.
I pass this along to keep us all aware of the perceptions and linkages that are "out there" around C8.
Regards,
Bob Ritchey
*** Forwarding note from WALRATJ --ISCDCVM1 09/13/94 08:54 ***
To: RITCHERL--ISCDCVM1

From: Judy Walrath
Epidemiology/Haskell/CR&D, 36-66594
Subject: Ducatman
Bob, For your information. I'll follow-up with 3M and let you know what I learn. Judy
*** Forwarding note from KARRHBW --ISCDCVM1 05/27/94 14:44 ***
To: WALRATJ --ISCDCVM1

From: Bruce W. Karrh * 4-9513 * N-10460-6

EID073255

Subject: Ducatman
Alan Ducatman of University of WVa called me today. He was at the NCI conference where you gave the AN paper. His comment was that NCI has perfluorooctanoatic acid on a "fast track" and, if we are planning any work on this compound, he would like to be involved. He was aware that we made or used it at a "West Virginia" plant, something Ididn't confirm nor deny but told him I would get in touch with the people who may be involved with that chemical and let them know of his interest.
I tried to call Gerry Kennedy but he was not in. Will you please pass on to him Alan's interests?? Thanks

cc: REINHACF--HSKVAX

cc: KENNEDY --HSKVAX
PEETDL --ISCDCVM1 David L. Peet
SERINGCS--ISCDCVM5
HUDSONBL--WWPS
RITCHERL--ISCDCVM1 Robert L. Ritch
HOLMESDA--ISCDCVM1 Dave Holmes
PORTERJB--CSOC

EID073256

**INTEROFFICE MEMORANDUM**

**Date:** 09-Nov-1994 11:28am
**From:** Michael E. Gell
GELLME AT A1 AT ENGG
**Dept:** ENGR-SERO
**Tel No:** (704) 362-5943 Fax 8-362-5934

**TO:** See Below

**Subject:** C-8 MSDS (suspect - small "c" carcinogen ?)

Marshall,
The attached MSDS for C-8 indicates that the compound causes tumors in mammals and is a **suspect carcinogen**. This compound is not listed as a known and federally controlled carcinogen, but Dupont Haskell lab considers it a suspect (small c carcinogen). I asked Haskell lab about the exposure limits they have established for C-8 and they indicated that they considered it a potential carcinogen when they set the AEL and CEG(community exposure guideline). I am not in a position to comment on the carcinogenicity of the C-8, but Haskell Lab is and I suggest we discuss this compound with Haskell.

The CEG of 1 ug/l in drinking water may be lower than the concentrations seen in our customers WWTP effluent, but I don't know enough about how they process the fiber and how much of the finish they would wash off and discharge to the environment. I am , however, concerned that we could potentially discharge this same material from any plant that makes or processes the finish.

Will this compound find its way into the wastewater?
Looking at the notes on the K-7762 finish I get the impression that we will not detect C-8 in the wastewater and that our customers will not see it in their air or water systems, but will it be there at some concentration? I am concerned that the C-8 will be there at some concentration and I am not sure what our obligation is to inform the site and the customer of that presence. I suggest that we discuss the potential exposure of the our WWT system and that of our customers to this compound in light of its **potential** carcinogenicity.

I am concerned that we may have a product stewardship issue if we have a finish that contains a suspect carcinogen that is made and processed on a DuPont site and then goes out to a potentially uninformed customer. If that customer does not handle the product with adequate precautions there could be a backlash to DuPont as the supplier.

Please let me know if I can be of any assistance with the C-8 issue.

RJZ015850

EID089977

N? 0025

# QA LAB WORK REQUEST

REQUESTED BY: Jim Holbert DATE: 4-18-95

UNIT, PROJECT OR DEPT THIS WORK SUPPORTS: QA

REPORT RESULTS TO: (Name and phone number) Jim

DATE AND TIME RESULTS REQUIRED: Wednesday

SAMPLE DESCRIPTION: (Sample Point, Container, Contents, Date, Time)

Env. Sample Dry Run

SAFETY: (Circle One) Normal Hydrocarbon; Acidic; Caustic; Other Stinks

ANALYSIS REQUIRED:

COD & TSS & Surfactants

WHY IS WORK REQUIRED:

Neighboor complaint

Shell chemical & Delpre - Jiffy Fueling

ANALYTICAL RESULTS: (Attach additional pages if necessary)

Surfactant 12.5 ppm

COD 1010 mg/L

TSS

1426.7
1415.0
11.7
x4

46.8 mg/L

DATE COMPLETED: 4-19-95

ANALYST: [signature]

FORM FOLIO NUMBER: 9002 1065

## SAMPLE DISPOSAL

RETURN SAMPLE TO REQUESTER ☐

RETURN SAMPLE TO RETAIN ☐

DISPOSE OF SAMPLE ☐

EPC 5/94

000253

CA 002

**Water Sample from Dry Run, WV**

Sample received by Jim Holbert from EJ Tennant on April 18, 1995.

Sample was not preserved or cooled.

pH of sample was 7.8. Ohio River pH on April 12, 1995 was 8.0 thus consider pH of sample acceptable.

Chemical Oxygen Demand was 1010 mg/L. Shell's treated water seldom goes above 25 mg/L so 1010 is extremely high.

Surfactant level was 12.5ppm. This should be close to zero. Shows there are detergents present.

Total suspended solids was 46.8 mg/L. The Ohio river when not flooded has solids of less than 10 mg/L.

Given the smell and the detergents present. My guess is the sample is contaminated with sewage and is harmful.

CA 003

Caustic Chemical or Sulfuric Acid

THE SAMPLE WAS pulled 4/16/95

Rained & washed Rain out ~~5/12/95~~

000054 5/12/95

INTEROFFICE MEMORANDUM

08:08am

PLOEGER

AFFAIRS/LITIGATIONS

Date: 26-Apr-1995

From: GERALD A PLOEGER

Dept: EXT.

Tel No: 863-4248

TO: WALTER M. STEWART ( STEWART )
TO: GEORGE WOYTOWICH ( WOYTOWIG )
TO: LYNWOOD K. IRELAND ( IRELAND )
TO: RICHARD A KIRSCHNER, JR ( KIRSCHRA )

CC: DAWN D JACKSON ( JACKSODD )

Subject: ATTACHED REGARDING CITIZEN COMMENTS

INTEROFFICE MEMORANDUM

03:37pm

Date: 25-Apr-1995

From: D CHRIS KOELSCH
KOELSCDC

Dept: ZYTEL AUTOCLAVE

Tel No: 304-863-2291

TO: GERALD A PLOEGER ( PLOEGER )
TO: H. DAVID RAMSEY, JR. ( RAMSEY )

Subject: RE: DRY RUN LANDFILL

Jerry,

Rod Newell, a E&I mechanic in the Zytel* A/C area came to me yesterday morning asking me if I could help him in finding out some information about any trouble we might be having at the dry run landfill. Rod owns about 200 acres up by the landfill and raises about 80 to 90 head of cattle. There were basically three things that prompted him to seek further information; some of his cattle will start to graze in the fields next week, his neighbor's comments to him, and the fact that he has noticed large amounts of foam coming from the run off as far down as Lee creek, as much as 18 inches in stagnate pools. During the conversation he did mention that within the last year he's had two calves that were still born and his neighbor had one. These were the first two he had ever had. He was not trying to link that to the landfill but did want to register it's presents.

000459

EID007456

CW001370

His neighbor, Earl Tennant, Rod is quick to point out, is a bit on the reactionary side. Earl has made comments to Rod that he has seen and collected dead fish and frogs out of the run off and has them frozen, has complained to the DNR and the EPA, and has had a sample tested himself and the results showed "bad stuff in the water". Earl owns about 700 acres out and around the landfill and also raises cattle.

After Rod talked to me I contacted Ron Meloon and explained the situation. Ron was going to look into it and get back to me. We have missed each other several times in to last day and half. This morning I called George and relayed the same information to him. He assured me that surface water testing on about 28 different parameters are reported to the state each month and results from the 7 monitoring wells are reported quarterly. George stated to me there should not be a concern as far as the health and safety of his cattle.

After my discussion with George I talked to Rod and relayed George's comments to him and offered to gather any other information for him if he still had concerns. Rod was satisfied and thankful for the information and now has a better understanding of the extent of monitoring we perform. He then mentioned that he was turkey hunting this morning and foam all the way down to the creek was very heavy in the run off. I then passed that information onto George and asked Rod to let me know if he did not see an improvement in a couple of days.

That's about all I know. Let me know if there is anything else I should or can do. One other comment, Rod has approached this in a very calm and professional manner and is working with us. It gives us a great opportunity to improve our PR status with the abutting land owners to the landfill.

Thanks,
Chris

18.

INTEROFFICE MEMORANDUM

08:43am

Date: 04-May-1995

WOYTOWICH

From: GEORGE

000257

GW00157

EID007457

D.806

FILE 5-

Revised
3/93

STATE OF WEST VIRGINIA
DIVISION OF ENVIRONMENTAL PROTECTION
ENVIRONMENTAL ENFORCEMENT

Applic/Permit/Order No. ____
Expiration Date 1-11-96
Municipal ____
Industrial WV 0076244

## LANDFILL INSPECTION REPORT

Name of Facility EI DuPont - Dry Run Landfill

Weather Sunny Date of Last Inspection 23 Mar 95

Current Monthly Tonnage ____ Status of Operation: Active (X), Inactive ( ), Not Started ( ), Closed ( )

Permit Acreage ____ Estimated disturbed acreage at time of inspection ____

Rating: S - Satisfactory, U - Unsatisfactory, N/A - Not Applicable, N/O - Not Observed, N/D - Not Determined
M - Marginal

### I. LEACHATE MANAGEMENT

| | | | | | |
|---|---|---|---|---|---|
| Liner System Construction | N/A | Monitoring Wells | N/D | Sewer Hook-up | |
| Liner System Maintenance | N/A | Monitoring Reports | N/D | Treatment Plan | |
| Leachate Collection System | N/A | Leachate Treatment System | N/A | Other | |
| Leachate Detection System | N/A | Sprayback Recirculation | N/O | Other | |
| Leachate Storage System | N/A | Pumpout and Transport | N/A | Other | |

Is there a leachate discharge into receiving stream: Yes (X) No ( ) Receiving Stream Dry Run

Outlet Status:

| Outlet Number | 001 | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Rating | U | | | | | | | | |

Comments The bottom valve for the sediment pond was opened 1 day as to decrease the volume in the pond to be tested for sulfides. A heavy rain on 1-2 May has added additional flow. Pond is approx 3' lower than on 23 Mar 95. Mr. Isabel was advised to close the valve. Effluent is discoloring Dry Run. 90 degrees 4.5 mile

### II. SITE MANAGEMENT AND OPERATION

| | | | | | |
|---|---|---|---|---|---|
| Daily Cover | N/A | QA/QC | N/A | H.W. Exclusion Plan/Inspections | |
| Intermediate Cover | U | Certifications | N/A | Contaminated Soils Disposal | |
| Final Cover | N/A | Control of Wind Blown Material | S | Asbestos Disposal | |
| Erosion, Sediment Control, Diversion Ditches | U | Odor Control | S | Tire Disposal | |
| | | Dust Control | S | Drum Disposal | |
| Revegetation | S | Fire Control | S | Sludge Disposal | * |
| Access Roads | S | Vector Control | S | Shredder Fluff Disposal | |
| Access Control | S | Gas Management | N/A | Infectious Waste Disposal | |
| Operations Plan | | Records | | Other | |
| Placement and Compaction | U | Equipment | S | Other | |

Comments: Diversion ditches have been installed to minimize runon to work area. Three decon pads have been installed in the fill to aid in draining waste area. Leachate has odor but is not black as it was in Apr 95 Weeks are not being placed 1-2m in 2' lifts. Benches need defined. Erosion gullies filled/reclaimed

WARNING! You are hereby warned that the following remedial measures must be taken on or before the ____ day of ____, 19 ____

Comply with permit.

Number of N.O.V.'s issued on this date: None to Follow/None (/)

Company Representative and Title: William Stewart - Environmental Control Coordinator

Signed: [signature] (Inspector) Date: 3 May 95 Phone: 420-4435

White Copy - District Yellow Copy - Inspector Pink Copy - Charleston Goldenrod - Facility

EID012

000259

• Called down
• self control

C-8
TOC + /once per week -
D.O.
flow - at pipe
downstream at Toca
• Flyash
• Burning
• Landspreading
C-8

INTEROFFICE MEMORANDUM

Date: 02-May-1995 03:00pm
From: WALTER M. STEWART
STEWART
Dept: PPD-SHEA&EA
Tel No: (304) 863-4271

TO: Distribution List

Subject: MEETING - LANDFILL ISSUES

THERE WILL BE A MEETING ON THURSDAY, 5/4 FROM 10:00 A.M. TO 12:00 NOON IN MY OFFICE TO DISCUSS ISSUES CONCERNING THE DRY RUN AND LOCAL LANDFILLS. THE TOPICS WILL INCUDE:

DRY RUN

1) STATUS OF BLACKWATER IN THE POND AND STREAM

2) FOAM IN RUN BOTH ON AND OFF OUR PROPERTY

3) C-8 IN SLUDGE

LOCAL

1) NEW PERMIT REQUIREMENTS

2) COMPLIANCE ORDER

3) STRATEGY - ACCEPT OR APPEAL

LET ME KNOW IF OTHER ITEMS SHOULD BE ADDED TO THE AGENDA. THANKS.

000262

Copies to: R. Melon / L. Ireland
Alison Grant
DAW
GW
WMS

DATE RECEIVED
9/26/95
ENVIRONMENTAL
OFFICE

DIVISION OF ENVIRONMENTAL PROTECTION

GASTON CAPERTON
GOVERNOR

1304 Goose Run Road
Fairmont, WV 26554-1390

LAIDLEY ELI McCOY, Ph.D.
DIRECTOR

September 25, 1995

Mr. W. M. Stewart
Senior Environmental Control Consultant
Washington Works
E.I. DuPont de Nemours and Company
P.O. Box 1217
Parkersburg, WV 26102-1217

Re: Solid Waste/NPDES Water Pollution Control
Permit Application No. WV0076244,
Dry Run Landfill

This agency's May 16, 1994 field investigation revealed numerous deficiencies regarding the operation of the Dry Run landfill. Although your company was advised of these deficiencies at that time, March 23, 1995 and May 3, 1995 field investigations conducted by an inspector from the Environmental Enforcement group revealed measures had not been taken to correct the deficiencies. These deficiencies, which are discussed in detail below, are of concern not only because of non-compliance with the operational requirements of Title 47, Series 38, Solid Waste Management Regulations (SWMR), but also because the structural integrity of the landfill could be effected. Said deficiencies included but were not necessarily limited to the following: sparse vegetative cover, poorly defined benches which were not providing adequate routing of stormwater and leachate thus causing numerous erosional gulleys on the landfill surface, lack of interceptor channels to properly route stormwater and leachate to the surface impoundment, lack of a leachate collection system, lack of surface water diversion channels, and excessive solid waste lift thicknesses. Said deficiencies are primarily due to the design of the landfill which must be upgraded. For this purpose, this agency requests that your company contract the services of a consulting firm having expertise in landfill design.

Your letter dated June 9, 1995 indicates that in response to the above referenced Environmental Enforcement inspections, diversion channels have been dug to prevent run-on to the active landfill area and furthermore, drain pipes have been installed in the fill area to aid in draining water from the area. Although these efforts are commendable, be advised that diversion channels must meet the requirements of Section 4.5.2.b. of the SWMR as further referenced in Section C.2.b.1. below. Also be advised that the installation of drain pipes does not preclude the placement of a leachate collection system (see Section C.2.e. below).

Subsequent to the May 16, 1994 field review, a thorough review of Application No. WV0076244 was conducted. Based upon these reviews, and the March 23, 1995, and May 3, 1995 field investigations, said application has been found to be incomplete. Therefore, the following information is needed to continue the processing of Application No. WV0076244. Note that the sections referenced below correspond to these of the Class F application.

A.9 - Provide detailed information regarding the waste minimization program which shall include putrescible materials. Also provide details of the waste minimization programs which shall include a description of the technologies and methodologies of waste reduction, reuse, recycling, and energy recovery which are applicable to the wastes being disposed.

B.1.b. - The application reaveals that the sludge filter cake will consist of approximately 22% sludge, 28% filter aid and 50% water. As the filter press has subsequently been placed into operation, provide actual sludge, filter aid, and water percentages.

B.2. - As indicated in revised Attachment 7 submitted with your letter dated September 14, 1994, % bio-solids of the sludge have increased dramatically pursuant to the recently started operation of a filter press to dewater sludge from the wastewater treatment plant from 4.8% (referenced in A.C. Hustons' 3/1/88 letter to the writer) to 20%-30% as referenced in Attachment 7. The increase in biosludge concentration as well as increases in the amount of sludge disposed since the filter press became operational have apparently caused the presence of anaerobic bacteria and iron sulfide referenced in your April 13, 1995 and May 12, 1995 letters. Because of the dramatic increase in % bio-solids, your company should have requested that Permit No. WV0076244 be modified to incorporate said sludge as Condition G.1. of Permit No. WV0076244 allows only for the disposal of sludge having a biomass of the concentration referenced in the above referenced 3/1/88 letter. Concurrently with the modification request, your company should have submitted a leachability analysis of the filter aid/sludge mixture. Therefore, this agency requests that such be conducted by utilizing the Toxicity Characteristic Leaching Procedure (TCLP) for the parameters indicated in Mr. A.C. Huston's letter dated April 14, 1988. % weight dry solids of the filter aid/sludge mixture shall also be determined.

C.1.c. - As this agency has no record of Well MW-12B, please provide boring logs and well construction information for said well.

C.2.a.1. - It is unclear which contours indicate 1991 conditions and those which reference 1994 conditions. Therefore, please differentiate. Also, please explain the volume calculations and how they were derived.

Regarding Section A - A of Drawing M677 (Attachment 11), the interval between the solid black line and the dashed line labeled "ravine bottom", appears to indicate an area which was filled prior to waste disposal. If this assumption is correct, please indicate materials utilized for filling as well as placement procedures. If the assumption is incorrect, please label the solid black line and indicate the significance of the interval between the solid black line and the dashed line.

C.2.a.4. - Section 6.1.5. of the SWMR requires, unless otherwise approved by this agency in writing, that a cap consisting of a uniform and compacted one foot layer of clay that is no more permeable than 1 x 10-7 cm/sec shall be placed and graded over the entire surface of each final lift. As the landfill was not underlain by a leachate collection system prior to initial disposal operations, it is this agency's position that a one foot clay cap should be installed to minimize percolation of precipitation through the waste material.

In accordance with Section 3.12. of the SWMR, provide a borrow area study identifying materials to be utilized for the above referenced one foot clay cap and a two foot topsoil cover (a lesser topsoil cover thickness will be considered providing sufficient rationale in support of such is provided). Note that in accordance with Section 3.8.2. of the SWMR, test pits or test borings shall be utilized for this purpose. Refer to 3.8.2. to determine the required number of test pits or test borings and also to determine the information to be derived from test pits and borings. Said information shall be included in the borrow area study. Note that sufficient quantities of borrow materials must be available to provide the temporary cover referenced in this section but also the final cover requirements referenced in Section C.2.d.8.

C.2.a.5, C.2.a.6, E.3.f., E.3.g. - The application indicated that "the surface will be crowned to provide a 1% to 2% slope to eliminate ponding on the surface and to direct surface water to diversion ditches on either side of the fill" to "mininize surface water flow through the fill and over the face of the lift." However, the May 16, 1994, the March 23, 1995, and the May 3, 1995 field investigations revealed that most of the benches of the landfill surface are not well defined or are not sloped sufficiently to direct runoff toward the sides of the landfill thus allowing runoff to flow directly across the face of the landfill which is resulting in numerous erosional gulleys some of which have exposed waste materials. Those benches which are defined do not contain a sufficient slope to route leachate toward either side of the landfill as pools of leachate were observed on the benches. Therefore, the benches must be redesigned to provide routing of stormwater and leachate to interceptor channels which must also be developed to continue routing of runoff to the surface impoundment for treatment. It must be determined if slope drains should be utilized to

route runoff from the benches to interceptor channels. If it is determined that slope drains are needed, provide detailed drawings of such and calculations verifying that the proposed design will fulfill their intended purpose. If it is determined that slope drains are not needed, provide appropriate rationale and calculations accordingly.

During the May 16, 1994 field review, Mr. George Woytowich of your staff informed the writer that individual lifts of waste materials are not placed in accordance with surveyed elevations. As such is essential to provide proper drainage of the landfill surface, provide information detailing the implementation of surveying activities.

Describe measures to be undertaken to revamp existing benches to assure each existing bench is well defined and properly constructed to provide proper drainage. Provide a detailed plan view map of scale 1" = 100' (or a more expanded scale) clearly indicating each reconstructed bench. Each reconstructed bench shall be designated by letter. Indicate proposed elevations for the front and back of each reconstructed bench (note that benches shall be sloped from front to back to facilitate drainage), and also directions of flow for each reconstructed bench toward intercepter channels. Note that each reconstructed bench shall be routed to an interceptor channel. (see Section C.2.b.1. regarding interceptor channel sizing). The reconstructed benches shall be designated Phase I of the disposal area (see paragraph below regarding Phase I). Each successive series of benches overlying Phase I shall be designated Phase II, Phase III, etc., until the proposed elevation of the completed fill referenced on Drawing M677 is reached. For each Phase, provide the above requested information for Phase I. Note that Attachment 12 is insufficient as it does not provide the information previously referenced in this section.

The overall stability expressed as a factor of safety of 1) the existing disposal area and 2) the final configuration of the disposal area must be determined using an approved method. Indicate on a plan view map the landfill section utilized for this purpose which shall extend from the toe of the fill to the top of the fill. Provide in table form soil properties utilized for the analysis. Calculations utilized to genrate the factor of safety shall also be included.

The May 16, 1994 field review revealed that the upper surface impoundment was filled with sediment and therefore serving no useful purpose. At that time your company was advised that the impoundment should be drained to allow the sediment to thoroughly dry prior to disposing of waste materials thereupon. However, a representative of your company informed the Environmental Enforcement inspector present during the March 23, 1995 field investigation that no special measures were taken to drain the

impoundment (other than its natural drainage) prior to placement of waste materials thereupon which was being effected that date. As the May 16, 1994 field review revealed that the natural drainage of the pond was poor, the pond could not have been properly drained. Insufficient drainage of the impoundment sediments is a concern to this agency as the structural integrity of the landfill could be affected. Placement of waste materials within the area of the upper surface impoundment has expanded the ravine being utilized for disposal to its ultimate footprint. Said placement, in addition to the area previously utilized for disposal, for purposes of this letter, constitute Phase I of the landfill. The application references that a "slope of 1:2 will be maintained during filling." Note that Section 4.6.2.a.A. of the SWMR requires that slopes of working faces shall not exceed 3:1. Also note that Section 6.1.5.a.D. of the SWMR requires that the grade of the final surface shall not exceed 3:1. Benches must be spaced to assure that the slopes of working faces and the final surface do not exceed 3:1; this requirement must be taken into consideration when preparing the map referenced in the above paragraph. Provide the following bench details the locations of which shall be clearly indicated on the plan view map referenced in the above paragraph: cross section to scale of a typical bench which shall include its upslope working face; plan view to scale of typical bench drainage to interceptor channels which shall include erosion control materials (provide documention that erosion control protection will not be required if such is not proposed to be utilized); plan view to scale of a typical bench corner; and cross-section to scale of typical bench erosion protection (provide documentation that erosion control protection will not be required if such is not proposed to be utilized).

C.2.b.1. ~~As~~ the May 16, 1994 field investigation revealed that surface run-on has eroded a channel which in places is approximately three feet deep through the waste material on the southern side of the fill area, diversion channels are needed to route surface run-on around the site. Note that although Application No. WV0076244 dated August 19, 1987 indicated that a diversion channel would be constructed, the May 16, 1994 field investigation revealed that it had not. Note that diversion channels should also extend below the surface impoundment (see C.2.b.3.) The application incorrectly states that diversion channels are shown on Attachment 10 with details and profiles of the channels being shown on Attachment 11. Note that in accordance with Section 4.5.2.b.A. of the SWMR that your company must design, construct, operate and maintain a run-on control system capable of preventing flow onto any part of the disposal area during peak discharge from at least a 25-year, 24-hour rainfall event. Note that it is unclear whether the drainage swales indicated our Drawing M641 (Attachment 10) meet this requirement. Furthermore, Section 4.5.2.b.B. of the SWMR requires that diversion channels shall have the capacity to pass safely the peak discharge from the contributing watershed from a 25-year, 24-hour rainfall event. Therefore, your company must provide the information required by Section 4.5.2.b.C. of the

SWMR for the diversion channels referenced in your June 9 letter. Should your company determine that the diversion channels referenced in said letter do not meet the requirements of Section 4.5.2.b.C., submit the information required by 4.5.2.b.C. for alternative diversion channels.

Attachment 13 incorrectly references that a cross-section of the diversion ditch is shown on Attachment 11 as such is not indicated on Attachment 11 or any other Drawing contained within the application. Furthermore, a determination cannot be made whether Attachment 13 contains the required information to verify that the diverson channel has sufficient capacity to pass safely the 25-year, 24-hour rainfall event. Therefore, this agency requests that a procedure approved by the United States Soil Conservation Service be utilized to determine the location and sizing not only of channels to divert surface run-on, but also to determine the location and sizing of interceptor channels referenced in C.2.a.5. above. At a minimum, the following factors must be included in the analysis: surface drainage area contributing to surface run-on; maximum, minimum, and average slope of the area contributing to surface run-on as well as the area where diversion channels are to be located; run off curve numbers; rainfall amount from a 25-yr/24-hr. storm event with its corresponding run-off depth, and peak rates of discharge in cubic feet per second. Based upon the analysis, the location and shapes (triangular or trapezoidal) of interceptor and diversion channels shall be determined. Said channels shall be indicated on a plan view map of scale 1' = 100' (or a more expanded scale) to accurately reference their locations. Provide cross sections of typical triangular and trapezoidal channels. For each interceptor and diversion channel, which shall be numbered in plan view, provide in table form the following information: the acreage of the contributing watershed, the design velocity in cubic feet per second based upon a 25-yr, 24-hr storm event, the type of section (trapezoidal or triangular), depth of channel, type of lining (grass, fabricform or rock - indicate rock size and type), thickness of lining, maximum velocity (feet per second), minimum slope (ft/ft), maximum slope (ft/ft), bottom width, and width of side slopes. For channels utilizing fabricform lining, provide minimum thickness and filter point spacing for centerline slopes less than or equal to 5%, 5%-10%, and greater than 10%. All calculations utilized to determine the design of the interceptor and diversion channels shall be provided. It must also be determined if geotextile should be utilized in the interceptor and diversion channels. If it is determined that geotextile should be utilized, provide the type(s) to be utilized and rationale for choosing said type(s). If it determined that geotextile should not be utilized, provide sufficient rationale accordingly.

C.2.b.2. - Provide type and quantity (pounds per acre) of fertilizer to be utilized. Also note that Section 4.5.6.b.B. of the SWMR requires that for soils having a pH of less than 5.5,

lime shall be added to maintain a soil pH of 6.0. Therefore, based upon soil analyses, determine whether lime will be utilized and furthermore, indicate (if it is determined that lime is necessary) the amount to be utilized in pounds per acre.

C.2.b.3. - As the discharge from Outlet No. 001 typically exceeds its maximum daily Total Suspended Solids limitation, and furthermore, as elevated BOD, TOC, and COD concentrations have been observed (see Section C.2.h.), the surface impoundment is not fulfilling its intended purpose. Be advised that the surface impoundment must meet the design requirements of Sections 4.5.2.c.A.(c), 4.5.2.c.A.(g)(B), and 4.5.2.c.A.(g)(C) of the SWMR. To determine if said requirements are being met, a procedure approved by the United States Soil Conservation Service shall be utilized. Therefore, provide documentation (calculations, drawings, verbage) regarding such. As the Environmental Enforcement inspector's May 3, 1995 field investigation revealed that the discharges from the diversion channels referenced in your June 9 letter are routed to the surface impoundment, said discharges must be included when determining if the requirements of Sections 4.5.2.c.A.(c), 4.5.2.c.A.(g)(B), and 4.5.2.c.A.(g)(C) are being met. Note that diversion channels should instead be routed around the surface impoundment to provide improved sedimentation control. If it is determined that the requirements are not being met, provide detailed drawings clearly indicating changes which will be made to upgrade the surface impoundment to conform with said requirements. Concurrently, with the revised drawings, provide documentation (calculations, verbage) to indicate that the revised design of the surface impoundment meets the requirements of the SWMR.

It is the agency's understanding that the riser structure was changed concurrent with pond reconstruction in August 1993. Therefore, provide riser details.

C.2.b.5. - Attachment No. 13 indicates that the discharge structure is a 15" diameter steel pipe. If this is correct, please confirm such. If not, indicate its type and diameter.

C.2.c.1. - It is the writer's opinion that a haul road is required not only to provide access to the working face but also to provide protection for areas previously utilized for disposal. Therefore indicate haul road(s) in plan view on a map having a scale of 1" = 100' (or a more expanded scale). Station numbers (one per 100 feet minimum) shall also be indicated for each haul road on said plan view map. Provide haul road profile(s) referenced by station numbers. Also provide a typical cross-section of the haul road(s). Interceptor and diversion channels should be included in cross-sections of the haul road(s), if applicable.

Note that access roads must be designed in accordance with Section 4.5.3.b. of the SWMR. As required by Sections 4.5.3.b.D.

and 4.5.3.b.F. of the SWMR, provide calculations documenting that drainage ditches and culvert openings are capable of passing the peak discharge of a 1-year, 24-hour rainfall event. For each culvert, which shall be numbered in plan view, provide in table form the following information: the design velocity in cubic feet per second based upon a 25-yr, 24-hour storm event, the diameter expressed in inches, type (reinforced concrete pipe, bituminous coated corrugated metal pipe, HDPE), length, % slope, invert elevation of point of entry, site coordinates of point of entry, invert elevation of point of discharge, and site coordinates of point of discharge.

C.2.d.1., E.3.A. - The application correctly references, as was observed during the May 16, 1994, March 23, 1995, and May 3, 1995 field investigations, that solid waste is being placed in eight foot lifts. This procedure in not in compliance with Section 4.6.2.a.C. of the SWMR which requires that solid waste shall be placed in layers not exceeding two feet in depth. The above field investigations revealed that waste materials were being landfilled which are difficult to compact such as large cardboard boxes and metal products. Although the application indicates that a waste minimization program is in effect, greater waste miminization efforts are in order to substantiallly reduce (preferably eliminate) materials which are difficult to compact as said materials pose a threat to the structural integrity of the landfill. Provide the size of the bulldozer which is utilized as Section 4.6.2.a.C. requires that an 815 Caterpillar compactor or other equipment of equivalent weight be utilized.

C.2.d.2. - The application incorrectly states that layers up to two feet thick are disposed as field investigations referenced in C.2.d.1. revealed eight foot lifts were being utilized. Solid waste lift thickness must not exceed two feet. Describe measures which shall be implemented to comply with this requirement as the materials referenced in C.2.d.1. are difficult to compact to this thickness.

C.2.d.4. - A detailed narrative is needed as Attachment 12 is insufficient for this purpose. The narrative must refer to the development of the Phases of designated benches previously referenced in Section C.2.a.5.

C.2.d.6., E.3.j. - As the application references that the landfill is not generally utilized during inclement weather, indicate where waste is stockpiled during inclement weather conditions.

C.2.d.7. - Regarding the sludge fill area indicated on Attachment 9 (Drawing M688), although such was not observed during the May 16, 1994 field review, it is not marked inactive on said drawing. In addition to providing a time frame during which the sludge fill area was utilized, provide authorization by which such was effected, the quantity of sludge disposed and the type and quantity of materials mixed with the sludge prior to disposal.

C.2.d.8. - Provide sedimentation control measures to be undertaken at the sites on Drawing M688 designated "future cover material sites". During the May 16, 1994 field review, Mr. Woytowich of your staff indicated that archaeological investigations would be conducted upon said sites. Therefore, either submit report(s) summarizing the findings of said investigations or provide a date by which they will be submitted. As the application indicates that soil will only be used as cover on a temporarily closed area prior to seeding, provide an example of a temporarily closed area. Borrow area studies shall be conducted upon each "future cover material site" - see Section c.2.a.4. regarding borrow area study requirements. Note that sufficient quantities of borrow materials must be available to provide the final cover referenced in section C.2.a.4. but also the temporary cover requirements referenced in this section.

C.2.e. The May 16, 1994, March 23, 1995, and May 3, 1995 field investigations revealed the presence of numerous seeps on the landfill surface which are likely due to the lack of a leachate collection system underlying the fill area. Therefore, to alleivate this problem, Mr. Woytowich was advised by the writer during the May 16, 1994 field investigation that a leachate collection system must be placed upon the existing waste materials before an additional lift of waste materials is placed. Said system shall be designed in accordance with the requirements of Section 4.5.4.f. of the SWMR. To determine the amount of leachate flow from the site, which will be utilized in determining the proper design of the leachate collection system, the landfill modeling program entitled "Hydrologic Evaluation of Landfill Performance" created by the Waterways Experimental Station of the US Army Corps of Engineers shall be utilized. Leachate flow shall be determined from the existing site as it represents the ultimate footprint of the disposal area. The design properties of the leachate collection system, based upon the requirements of Section 4.5.4.f., shall be incorporated into the HELP model analysis. Leachate flow shall also be determined utilizing the final configuration having a one foot clay cap, topsoil, and vegetative cover in place. Input data sheets and computer outputs derived from the program shall be submitted. Note that although Section 4.5.4.f.A.(b) requires that the leachate collection system be eighteen inches in thickness, a lesser thickness may be utilized if justified by the program. A maximum daily leachate discharge must be calculated not only to determine the thickness of the leachate collection system but also the sizing of pipes located within the leachate collection system; note that Section 4.5.4.f.A.(g) requires that perforated pipes be located within the leachate collection system. Provide calculations indicating that leachate collection system piping is of sufficient strength to support anticipated loadings without crushing. Also provide calculations indicating that the leachate collection system is of sufficient capacity to continue to function effectively should settlement of the underlying waste material occur.

Routing of leachate away from the landfill area is critical to its structural integrity. A leachate collection system must overlie the entire areal extent of the series of Phase I benches

and associated working faces referenced in Section C.2.a.5. As disposal operations extend from the base of the disposal area to the top of the disposal area, the revampled Phase I benches referenced in Section C.2.a.5. shall be regraded immediately prior to the placement of the leachate collection system which shall placed immediately prior to the series of Phase II benches and their related working faces. Therefore on a plan view map of scale 1" = 100' (or a more expanded scale) indicate piping within the leachate collection system, its routing to interceptor and diversion channels, and culverts. Interceptor channels, diversion channels, and culverts shall be numbered with each having a reference to a detailed drawing. Detailed drawings (cross-sections) shall be submitted for each type of interceptor channel, diversion channel, and culvert. For each culvert, provide in table form the design velocity in cubic feet per second based upon a 25-yr, 24-hr storm event, its diameter, the type of material from which it is composed, its length, its slope, inverted elevation of incoming flow, and inverted elevation of outgoing flow. Also provide detailed drawings indicating the interface between the leachate collection system and the interceptor ditches. Also indicate the type of material to be utilized for the leachate collection system. It must be determined if geotextile is needed 1) between the leachate collection system and the underlying subgrade, and 2) the leachate collection system and the overlying waste materials be evaluating particle movement and head loss in the filter material. If it is determined that geotextile is needed, specify the type(s) to be utilized and provide rationale for choosing said type(s). If it is determined that geotextile is not needed, provide sufficient rationale accordingly. It must be determined if geotextile is needed for interceptor and diversion ditches. If it is determined that geotextile is needed, specify the type(s) to be utilized and provide rationale for choosing said type(s). If it is determined that geotextile is not needed, provide sufficient rationale accordingly.

C.2.h. - As the March 23, 1995, and May 3, 1995 field investigations indicated that the discharge from Outlet No. 001 was causing conditions not allowable in state waters referenced in Section 3.2. of Title 46, Series I, the surface impoundment is not providing sufficient leachate treatment. Insufficient treatment is further reflected by the elevated BOD, COD, and TOC concentrations observed at Outlet No. 001 during the first four months of 1995. Concentrations of this magnitude are not acceptable. As the surface impoundment does not contain a liner system, said concentrations will likely impact groundwater. Section 4.5.1. of Title 47, Series 58, Groundwater Protection

Regulations effective June 1, 1994 states "existing impoundments shall be evaluated for their potential to cause groundwater contamination. Where potential for contamination exists, action shall be taken to eliminate, to the degree practicable, the potential for groundwater contamination." Therefore, the surface impoundment must be retrofitted with a liner system which meets the design requirements of Section 4.8.3.C. of the SWMR. It must be determined if geotextile will be utilized in the liner system. It it is determined that geotextile will be utilized, provide the type(s) to be utilized and provide rationale for choosing said type(s). If it is determined that geotextile is not necessary, provide sufficient rationale accordingly. Your May 12, 1995 letter indicated that Sulf Control has been utilized to reduce the above referenced BOD, COD, and TOC concentrations. Therefore, provide a Material Data Safety sheet for the product. As use of such is merely a "stop gap" measure, provide detailed information regarding all steps being investigated - as was indicated in your May 12, 1995 letter - to eliminate the black water discharge from the fill area thereby resulting in the return of BOD, COD, and TOC to acceptable concentrations.

C.3.a.3. - Available soil cover shall be determined by conducting the soil borrow area studies referenced in Section C.2.a.4.

C.3.c. - It is this agency's belief that utilization of the HELP modelling program previously referenced in Section C.2.e. would have been more appropriate for providing the information required by this Section. Therefore, it is requested that said program be utilized taking into consideration the deficiencies referenced in the paragraph below. Should your company believe that the data generated by the method referenced in Attachment 19 is as reliable as data generated by the HELP modelling program, this agency will accept the utilization of said method provided that you company provide detailed rationale in support of said method. Note that if the method referenced in Attachment 19 is utilized, the deficiencies referenced in the paragraph below must be taken into account.

Attachment 19 indicates that "slope at the surface of each cell will be 1% - 2%". As the May 16, 1994, March 23, 1995, and May 3, 1995 field investigations revealed numerous areas of ponded water, the writer does not concur with this assumption. Attachment 19 also states that "the remaining 16.9 acres of fill area is covered with an impermeable clay (1x10-7 cm/sec) which is placed in a 6" compacted layer." However, Attachment 6 of the application indicates that the cover material instead exhibits a permeability of 1x10-6. Although Attachment 19 indicates that said cover contains deep rooted grass, the May 16 field review revealed a sparse vegetative cover. It should also be noted that surface run-on must also be considered in your evaluation.

E.2.e. - The application states that a 6" cover is applied as necessary "to maintain compaction and for dust and debris control during windy periods." Indicate factors which are considered when determining cover is to be applied. Also describe how cover material aids compaction.

E.4.a. - Describe use of each type of equipment utilized.

E.5.a.1. - Provide name, address, and qualifications of the landfill contractor.

E.7. - Sections E.7.b. and E.7.c. indicate that a six-inch thick soil cover is applied as necessary to maintain compaction and for dust and debris control in windy periods while E.7.h. indicates that wastes are covered as soon as possible with a minimum thickness of 6" of compacted cover material. Indicate if soil cover referenced in E.7.b. and E.7.c. is the same soil cover referenced in E.7.h.

E.10.a.2. - Section C.3. of Attachment 20 indicates PCBs having concentrations less than 50PPM are to be disposed. If PCBs have been disposed in the past, please provide the types and quantities of wastes containing PCBs which were disposed in addition to the dates they were disposed. If PCBs were not disposed in the past, please confirm such. Section C.4. of Attachment 29 indicates that maximum lift thicknesses of ten feet having a slope not exceeding 3:1 will be maintained. This is in conflict with Sections C.2.a.5. and C.2.d.6. of the application. Therefore, please reconcile.

Section C.14. states that a minimum thickness of 6" of compacted cover material will be applied to the entire solid waste area once each working day. This conflicts with Section E.7. of the application which states that a 6" soil cover is applied as necessary to maintain compaction and for dust and debris control in windy periods. Therefore, please reconcile.

Section F.3. of Attachment 20 indicates two feet of soil cover is to be placed over completed sections of the fill. Verify that "completed" sections refer to lifts which have reached final grade which will receive no additional waste materials.

Quarterly Monitoring Well Reports (QMWRs) submitted as required by Permit No. WV0076244 reference that statistical comparisons of the data provided for downgradient wells 6A, 12A, and 13A with background well 14 are not meaningful as 14 monitors a different aquifer. Therefore, your company contends that background groundwater quality in the aquifer monitored by wells 6A, 12A, and 13A should be defined as the average of each parameter measured for the first four quarters in the data base, specifically the period covering the second quarter 1990 through the first quarter 1991. Be advised that the SWMR require that background groundwater quality be determined by sampling wells that are hydrogeologically upgradient of the disposal area. However, the SWMR allow background groundwater quality to be based on sampling of wells that are not hydrogeologically upgradient of the disposal area where other wells will provide background groundwater quality that is as representative or



EID012683

more representative than that provided by hydrogeololically upgradient wells. Therefore, as wells 6A, 12A, and 13A are located immediately downgradient of the disposal area, they must not be utilized to determine background groundwater quality. Therefore, other well(s) must be utilized. For this purpose, this agency will consider the utilization of wells MW-1A and/or MW-4A as they are not located immediately downgradient of the disposal area. Should your company elect to utilize MW-1A or MW-4A (in lieu of constructing new wells), it must be demonstrated that said well(s) will adequately provide groundwater data in the aquifer monitored by MW-6A, MW-12A, and MW-13A.

Section 5.5.1. of the SWMR allows for the waiver or modification of various secions of the SWMR provided that sufficient documentation justifying the waiver or modification is provided. Based upon a review of Application No. WV0076244, waivers of Sections 4.4, 4.6.2.b.A, and 4.12.2 appear to be appropriate. Should your company desire to pursue waivers of these sections, provide sufficient justification.

As was indicated in this agency's February 4, 1994 letter, Application No. WV0076244 must include a Quality Assurance/ Quality Control Plan consistent with the requirements of Section 3.7.7. of the SWMR.

By letter dated November 30, 1990 to R.L. Weser, Chief, Compliance Division, Air Pollution Control Commission, you expressed a desire to incinerate solidified methacrylate sirup. Therefore, indiciate if methacrylate sirup ash has been disposed at the Dry Run landfill. If so, provide dates and quantities of disposal as well as all available leachability analyses. If methacrylate sirup ash has not been disposed, please indicate accordingly.

An 1991 inspection by this agency revealed the lack of surface seals around wells MW-6 and MW-6A. Therefore, provide measure to be implemented to install surface seals around these wells.

Attachment No. 14 references Stream Sampling Points 1 and 2. Indicate which location is utilized for collection of the sample obtained for semi-annual leachate analysis.

A detailed narrative, where appropriate, shall accompany the above requested information. Please submit the original copy of the above requested information to Chief, Office of Water Resources and four (4) copies of same to my attention at my Fairmont office in an expeditious manner but prior to January 11, 1996, so that the review of Application No. WV0076244 may continue.

Should you have any questions concerning the above, please contact me at (304) 367-2724.

Sincerely,

OFFICE OF WATER RESOURCES

John G. Britvec

John G. Britvec, Geologist
Industrial Branch

JGB/jb

cc: Cindy Musser, Insp., SW Dist.

000277

RAK

INTEROFFICE MEMORANDUM

Date: 05-Dec-1995 11:29am
From: CRAIG K DILLON
DILLONCK
Dept: Fluoroproducts
Tel No: 304-863-4972

TO: Distribution List

Subject: Meeting Notes - C-8 at Dry Run

A meeting was held on November 30 to discuss the acceptable C-8 levels for Teflon* waste that is proposed to be landfilled at Dry Run. Those in attendance were Walt Stewart and Dan Weber (Environmental), Woody Ireland (Power & Services), John Doughty (Central Lab), and Roger Zipfel, Ed James, and Craig Dillon (Teflon*).

Zipfel presented data on various Teflon* waste samples that were extracted by Doughty for C-8 levels. Details of the samples extracted are recorded in book No. E56538 page 123 (Doughty Log?). Dan Weber also provided as reference extraction data furnished by Quality Analytical Lab on bio-cake. Summary of the data is as follows:

| SAMPLE | DESCRIPTION | POLYMER C-8 LEVEL PPM | WATER EXTRACTION EXTRACTED C-8 LEVEL PPM** |
|---|---|---|---|
| 150-1 | Fine Powder Trench Scrap - undried | 59 | 0.44 |
| 150-2 | Granular Vacuum System Scrap - dry | 2 | 0.08 |
| 150-3 | PFA from Process K test dryer - dry | 11 | 0.04 |
| 150-4 | FEP-4100 from Line 3 - dry | 7 | 0.04 |
| 150-5 | 1 ppm blank | - | 1.04 |
| NA | Bio-cake sample (Quality Analytical) | 0.5-1.0 | 0.004 |

Doughty extracted the sample that Quality Analytical analyzed. Weber noted that the analysis method used by Quality Analytical differed from the method used by Doughty on the polymer samples. Based on the bio-cake analysis, Weber indicated that approximately 7 pounds per year of C-8 is currently landfilled at Dry Run due to bio-cake.

Using the test results as a guide, the group agreed that Granular scrap can be landfilled at Dry Run. In addition, any melted scrap PFA or FEP can be landfilled at Dry Run. For PFA, this would be scrap material generated at the exit side of the extruder and beyond (generally cubes). For FEP, this would be scrap material generated at the exit side of the humid heat treater and beyond (generally slabs, shred, cubes). It was the consensus of the group that the potential landfill amounts of these scrap materials would not significantly impact C-8 levels at Dry Run.

For Fine Powder/Dispersion, no process scrap will be allowed at Dry Run. This was based on the test results and also on process configuration. There is concern that the process configuration would not allow for distinct



EID016730

separation of C-8 and non-C-8 scrap. If a large amount of finished Fine Powder/Dispersion must be disposed, then this material should be tested for C-8 and handled on an individual basis. It was also the consensus of the group that Teflon* continue to drive the sale of all scrap to reduce the need for landfilling.



EID016731

GW000847

12/13/95 notes from Dawn Jackson

Jim Crum, Elkins DNR Office, called me today about a call that he had received from Earl Tennant about water flowing from DuPont's Dry Run Landfill poisoning the deer in the area. Tennant told Crum that he had also contacted Dr. Thomas, the State Veterinarian with the Dept. of Agriculture. Crum told Tennant that he (Tennant) should contact his local DNR office, which Tennant said he had done, but without satisfaction. Crum called the local office, and Jeff McCrady told him to call me, knowing that I had some background info on Mr. Tennant.

Tennant told Crum that numerous deer had been poisoned in the area recently. When Crum pinned him down on numbers, the total turned out to be five, between the months of April and October, with October being the last time that Tennant said that he had found one near Dry Run. Crum told Tennant that if he would contact the DNR office when he finds a fresh carcass, the DNR will do a tissue analysis. (Crum indicated to me that the DNR had done a number of such analyses when there was a virus killing whitetail deer in the Belleville area in 1988.)

Tennant also provided several very specific kinds of details to Dr. Thomas and Jim Crum. He said that his water analysis indicated the presence of mercury in the Dry Run stream, that there was an arsenic level of .0097 ppm, and that the pH was "not right." He also said that DuPont was treating the landfill ponds on Tuesdays and Thursdays with 5 gallons of hydrogen peroxide.

When Crum asked Tennant to share his water analysis info with the DNR, Tennant said that he wouldn't show the report to anyone until someone could give him some help with his cattle. Crum told him that the DNR couldn't address the cattle issue, but repeated that the DNR would do a tissue sample on a fresh deer carcass.

Jim Crum (with whom our WHEC has worked on at least one project -- the osprey reintroduction) sounded very sympathetic and told me about the number of rumors that the DNR struggles with. He said that contacting us was a courtesy call, and he said that he would call us if he hears from Mr. Tennant again.

He also asked me specifically if we <u>are</u> treating the ponds with something that Mr. Tennant may have seen and misinterpreted, and he asked me if we do an analysis of the materials that Mr. Tennant mentioned. I told him that I'd get back to him.

I talked with Ron Meloon and Woody Ireland, and Woody pointed out that arsenic, mercury, and pH are among the parameters reported monthly to the DEP. Woody also recalled one hydrogen peroxide treatment on April 11 to change the pH of the pond, but he was not aware of any treatment that may have occurred as routinely as every Tuesday/Thursday. Ron and Woody are checking with Boso to see if anything like antifoam or algae treatment may have been used on a schedule during the summer that may have led Tennant to conclude that it was being done every Tue/Thur.

In conclusion, Dawn loves her job, she hopes that these notes make sense, and she wishes Mr. Tennant a very Merry Christmas.



EID010289

DDJ001308

I N T E R O F F I C E  M E M O R A N D U M

Date: 27-Mar-1996 02:58pm EDT
From: Roger J. Zipfel
ZIPFEL
Dept: Fluoroproducts
Tel No: 304-863-2567

TO: 7 addressees

CC: GERALD L KENNEDY ( KENNEDY AT A1 AT HSKVAX )

Subject: Phone conversation with Gerry Kennedy

Attached are the notes of a phone conversation I recently had with Gerry Kennedy. Since I am not good with some of these words, I may have not got the story completely correnct.

By this note, I am asking Gerry to correct any error in note taking on my part.

Per Gerry the bottom line is that item 1 is not good news and items 2&3 are good news.

Roger.

EID073253

C-8 Toxicity

re: phone conversation with Gerry Kennedy on 3/20/96

1) 3M and Hoechst are running tests to fully expand our Geno Toxicity understanding. They ran an "Unscheduled DNA Synthesis" test. They found at high C-8 concentrations (1000 microg/ml) that C-8 gave equivocal results on geno toxicity. They are currently collaborating on why this happens.

Where this will go in the future is not clear, but this is the first indication that C-8 could be affecting DNA. The worst case scenario is that C-8 could be classified as a large "C" carcinogen.

2) 3M and Hoechst have proposed to the APME to look at the human relevance of the significance of the current rat pancreatic cancer concerns.

3) 3M has hired an epidemiologist to relook at 3M (1991) worker hormonal levels. Results of this effort are that the affects reported by the U.of Minn. can not be confirmed. The same worker population was also examined with data taken in 1993&1995 and again no relationships of C-8 exposure to hormonal affects were noted.

A relook at the prostrate cancers noted in the 1991 UofMinn study showed that only 1 of the 6 cases was a C-8 worker.

3M plans to publish this work.

AJP0006911

EID073254

000285

INTEROFFICE MEMORANDUM

Date: 10-Sep-1996 04:59pm
From: WALTER M. STEWART
STEWART
Dept: PPD-SHEA&EA
Tel No: (304) 863-4271

TO: See Below

Subject: DRY RUN LANDFILL COMPLAINT

I HAVE HAD TWO CONVERSATIONS SINCE YESTERDAY (9/9) WITH U.S. EPA, REGION III OFFICIALS REGARDING A COMPLAINT RECEIVED FROM MR. EARL TENANT. BOTH MARY BECK, REPRESENTING THE RCRA CORRECTIVE ACTION GROUP AND SARAH CASPAR, FROM THE SUPERFUND REMOVAL ENFORCEMENT AND OIL SECTION INDICATED THE COMPLAINT ALLEGED THAT 150 CATTLE DIED AS A RESULT OF WATER FLOW ACROSS HIS PROPERTY FROM OUR LANDFILL. HE OFFERED AS PROOF A STATEMENT FROM A VETERINARIAN (NO NAME) THAT TEETH EXAMINED FROM THE DEAD CATTLE SHOWED SIGNS OF FLUORIDE EXPOSURE.

THEY BOTH REQUESTED NAMES OF THE STATE PEOPLE WE WERE WORKING WITH AND I GAVE THEM THE NAMES OF JOHN BRITVEC, OUR PERMIT WRITER AND CINDY MUSSER, THE ENFORCEMENT INSPECTOR. MS. CASPAR INDICATED SHE WOULD BE FOLLOWING UP WITH BOTH OF THEM.

THE EPA IS TAKING THE COMPLAINT VERY SERIOUSLY AND IT WAS STATED BY MS. CASPAR THAT SHE EXPECTED TO VISIT THE SITE ABOUT TWO WEEKS FROM NOW. SHE INDICATED SHE WOULD LET ME KNOW WHEN, AND THAT THE STATE WOULD BE ASKED TO PARTICIPATE. I FILLED HER IN ON SOME OF THE BACKGROUND ABOUT THE SITUATION AND INDICATED THAT WE WOULD COOPERATE WITH THEM IN INVESTIGATING THE MATTER.

I'LL KEEP YOU INFORMED WHEN I HEAR MORE ABOUT THIS.

Distribution:

| | | |
|---|---|---|
| TO: | H. DAVID RAMSEY, JR. | ( RAMSEY ) |
| CC: | MAYNARD S EATON, JR | ( EATONMS ) |
| CC: | RONALD W MELOON | ( MELOONRW ) |
| CC: | LYNWOOD K. IRELAND | ( IRELAND ) |
| CC: | DAWN D JACKSON | ( JACKSODD ) |
| CC: | GERALD A PLOEGER | ( PLOEGER ) |
| CC: | Charles T. Alt | ( ALT ) |
| CC: | H RAY HODGES | ( HODGESHR ) |
| CC: | GARY W. KLESEL | ( KLESELGW ) |
| CC: | ROBERT L. RITCHEY | ( RITCHERL ) |
| CC: | DANIEL A WEBER | ( WEBERDA ) |
| CC: | GEORGE WOYTOWICH | ( WOYTOWIG ) |

000287

INTEROFFICE MEMORANDUM

Date: 16-Sep-1996 02:29pm
From: Thomas R. Waldron
WALDROTR
Dept: PPD-POWER/SERVICE
Tel No: 304-863-2489

TO: RONALD W MELOON ( MELOONRW )
TO: LYNWOOD K. IRELAND ( IRELAND )

CC: RICHARD A KIRSCHNER, JR ( KIRSCHRA )
CC: DANIEL A. WEBER ( WEBERDA )

Subject: Dry Run Landfill 09/16/96

I toured Dry Run Landfill today and made the following observations. 1) The newly sown grass seed is growing well and I saw no exposed areas. 2) The rip-rapped ditches were doing a good job of holding back most of the the run-off(it was raining at the time), the discharge from them was mostly clear. 3) The lower pond was still very green with some slight discoloration due to a small amount of solids in the runoff. 4) There was some foaming coming down into the lower pond from the upper pond and persisting downstream past the outfall. I instructed the operator to put a quart of anti-foam in the upper pond. 5) The waste was completely covered, except what had been deposited during the morning hours.

Any questions, give me a call.

Tom

000&89

I N T E R O F F I C E  M E M O R A N D U M

Date: 07-Oct-1996 01:55pm
From: Thomas R. Waldron
WALDROTR
Dept: PPD-POWER/SERVICE
Tel No: 304-863-2489

TO: RONALD W MELOON ( MELOONRW )
TO: LYNWOOD K. IRELAND ( IRELAND )

CC: RICHARD A KIRSCHNER, JR ( KIRSCHRA )
CC: DANIEL A. WEBER ( WEBERDA )

Subject: Dry Run Landfill

I toured the Dry Run landfill this afternoon and found a couple of things that I made corrections too.

1) The outfall had some considerable foam in and around the exit pipe. It also appeared to persist in the stream down to about the 500' foot level. I added about a quart of anti-foam to the pond via the air line and about a pint to the outfall exit itself. The foam began to immediately disipate.

2) I also found some small areas of exposed trash on the face of the active fill. Apparently the trash had lain uncovered all weekend. I instructed the landfill operator to cover the material before he left for the day and he told me he would get it done.

Most of the recently seeded areas have a good crop of grass growing on them and those places that have been exposed by the recent rains will be reseeded this week. The meadows below the ponds have lots of 4 wheeler tracks in them as it would appear that someone is still running that area when we are not present. Any questions, give me a call.

Tom

000231

INTEROFFICE MEMORANDUM

Date: 15-Oct-1996 03:49pm
From: WALTER M. STEWART
STEWART
Dept: PPD-SHEA&EA
Tel No: (304) 863-4271

TO: Distribution List

Subject: EPA AUDIT

I RECEIVED A PHONE CALL FROM MR. KEVIN SCOTT, PRC ENVIRONMENTAL MANAGEMENT THIS AFTERNOON (10/15) INFORMING ME THAT HE AND ANOTHER EPA PERSON WOULD BE CONDUCTING AN INSPECTION OF OUR DRY RUN LANDFILL FOR THE NEXT SEVERAL DAYS. HE INDICATED THAT THE INSPECTION WAS THE RESULT OF MR. TENNANT'S COMPLAINT AND THAT IT WOULD START THIS AFTERNOON.

THE INSPECTION WILL INVOLVE VIEWING THE SITE, EXTENSIVE SAMPLING AT THE LANDFILL, AND SAMPLING DRINKING WATER WELLS IN THE VICINITY. I INDICATED THAT WE WANTED TO SPLIT SAMPLES OF ANY TAKEN AT OUR LANDFILL.

MR. SCOTT DID NOT WANT TO COME TO THE PLANT BUT WAS GOING DIRECTLY TO THE LANDFILL. I WILL KEEP YOU INFORMED ABOUT THE INSPECTION. IF THERE ARE ANY QUESTIONS, PLEASE CALL.

000293

DIVISION OF ENVIRONMENTAL PROTECTION
10 McJunkin Road
Nitro, WV 25143

GASTON CAPERTON
GOVERNOR

LAIDLEY ELI McCoy, Ph.D.
DIRECTOR

October 15, 1996

Mr. W. M. Stewart
Senior Environmental Control Consultant
E.I. DuPont de Nemours and Company
Washington Works
P.O. Box 1217
Parkersburg, West Virginia 26102

Re: Dry Run Landfill (WV/NPDES Permit No. WV0076244) and Washington Works facility (WV/NPDES Permit No. WV0001279)

Dear Mr. Stewart:

E.I. DuPont de Nemours and Company has always been a valued citizen of West Virginia. However, over the past several months, inspectors with DEP have observed serious shortcomings in DuPont's operations of the Dry Run Landfill and the Washington Works facility. During numerous inspections, DEP employees noticed, among other violations, the following:

1. On several occasions, DuPont has allowed, and continues to allow, a large flow of black and odorous leachate to be released from its Dry Run Landfill into Dry Run, in violation of its NPDES Permit and the West Virginia Code of State Regulations;

2. On several occasions, DuPont discharged Teflon, Delron, Nylon and wax beads from its Washington Works facility into the Ohio River, in violation of its NPDES Permit and the West Virginia Code of State Regulations;

3. On several occasions, DuPont has failed, and apparently continues to fail, to prevent run-off from flowing across the Dry Run Landfill and has failed to collect and treat leachate generated by the Dry Run Landfill, in violation of the West Virginia Code of State Regulations;

4. On several occasions, DuPont has failed, and apparently continues to fail, to notify DEP that the quality of the leachate generated by the Dry Run Landfill has exceeded the design capacity of the treatment system, in violation of its NPDES Permit and the West Virginia Code of State Regulations;

5. On several occasions, DuPont has failed, and apparently continues to fail, to properly compact the wastes disposed at the Dry Run Landfill, in violation of the West Virginia Code of State Regulations;

6. On numerous occasions, DuPont has deposited, and continues to deposit, waste in the Dry Run Landfill in greater concentrations than the concentrations approved by DEP, in violation of its NPDES Permit and the West Virginia Code of State Regulations;

7. On several occasions, DuPont has failed to properly operate and maintain the Dry Run Landfill, in violation of its NPDES Permit.

8. DuPont has failed, and continues to fail, to retrofit the Dry Run Landfill surface impoundment with a liner system and leak detection system, in violation of the West Virginia Code of State Regulations;

9. On numerous occasions, DuPont has allowed the effluent from the Dry Run Landfill to exceed the water quality standards, in violation of the West Virginia Code of State Regulations;

10. On numerous occasions, DuPont has allowed the effluent from the Dry Run Landfill to exceed the discharge limitations established in its NPDES Permit, in violation of its NPDES Permit; and

11. On several occasions, DuPont has failed, and continues to fail, to comply with each and every condition of its NPDES Permit, in violation of its NPDES Permit.

Due to the foregoing violations at the Washington Works facility and Dry Run Landfill, DEP intends to file a civil action against DuPont in the Circuit Court of Wood County. The agency will seek injunctive relief to address current and prospective violations and will ask for the assessment of civil penalties for current and past violations. A copy of a draft complaint is attached.

I have instructed DEP's Office of Legal Services to initiate formal proceedings against DuPont in circuit court by no later than January 15, 1997. Should DuPont wish to enter into negotiations to explore potential resolution of the enforcement action before a complaint is filed, please contact DEP attorneys Matthew Crum or Scott Goldman at (304) 558-9160.

Very truly yours,

Laidley Eli McCoy, Ph.D.
Director



EID012734

IN THE CIRCUIT COURT OF WOOD COUNTY, STATE OF WEST VIRGINIA

LAIDLEY ELI McCOY, Director,
West Virginia Division of
Environmental Protection,

Plaintiff,

DRAFT

v. Civil Action _____

E. I. DuPONT DE NEMOURS & COMPANY, INC.,

Defendant.

---

**COMPLAINT**

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## INTRODUCTION

Plaintiff Laidley Eli McCoy, Director, West Virginia Division of Environmental Protection ("DEP"), by counsel, alleges the following:

1. This is a civil action brought under the Water Pollution Control Act, West Virginia Code §§ 22-11-1 to -28 (1995) ("WPCA") and the Solid Waste Management Act, West Virginia Code §§ 22-15-1 to -20 (1995) ("SWMA"), against Defendant E.I. DuPont De Nemours and Company, Incorporated, based on violations of the WPCA, SWMA, and the regulations promulgated thereunder for injunctive relief, the assessment of civil penalties, and attorneys fees.

## PARTIES

2. Plaintiff, Laidley Eli McCoy, for and on behalf of the State of West Virginia, is the duly appointed Director of DEP, and is thereby authorized by the WPCA, and the regulations promulgated thereunder, to enforce the water pollution control laws and regulations of this State. Specifically, W. Va. Code § 22-11-22 states:

> Any person who violates any provision of any permit issued under or subject to the provisions of [the WPCA] is subject to a civil penalty not to exceed ten thousand dollars per day of such violation, and any person who violates any provision of this article or of any rule . . . is subject to a civil penalty not to exceed ten thousand dollars per day of such violation. Any such civil penalty may be imposed and collected only by a civil action instituted by the director [of DEP] in the circuit court of the county in which the violation occurred or is occurring or of the county in which the waters thereof are polluted as the result of such violation.
>
> Upon application by the director, the circuit courts of this state or the judges thereof in vacation may by injunction compel compliance with and enjoin violations of the provisions of this article, the rules of the [environmental quality] board or director, effluent limitations, the terms and conditions of any permit granted under the provisions of this article, or any order of the director or board, and the venue of any such action shall be the county in which the violation or noncompliance exists or is taking place or in any county in which the waters thereof are polluted as the result of such violation or noncompliance.

3. Additionally, Director McCoy is authorized by the SWMA and the regulations promulgated thereunder, to enforce the solid waste management laws and regulations of this State. Specifically, W. Va. Code § 22-15-15(d) and (e) state:

> (d) Any person who violates any provision of [the SWMA], [a]ny permit or any rule or order issued pursuant to [the SWMA] is subject to a civil penalty not to exceed twenty-five thousand dollars for each day of such violation, which penalty shall be recovered in a civil action either in the court wherein the violation occurs or in the circuit court of Kanawha County.
>
> (e) The director may seek an injunction, or may institute a civil action against any person in violation of any provisions of this article or any permit, rule or order issued pursuant to this article.



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4. Defendant E. I. DuPont De Nemours & Company ("Defendant"), is a corporation duly authorized to conduct business in the State of West Virginia. Defendant owns and operates a facility ("Washington Works") in Wood County, West Virginia, which produces plastic and acrylic resins. Defendant also owns and operates a non-hazardous waste landfill ("Dry Run Landfill") in Wood County, West Virginia, that serves the Washington Works facility. At all pertinent times hereto, Defendant was and is doing business in Washington, Wood County, West Virginia.

5. Under the WPCA and SWMA, the definition of the term "person" includes "any industrial user, public or private corporation, institution, association, firm or company organized or existing under the laws of this or any other state or country." W. Va. Code §§ 22-11-3(15) and -15-2(22). Defendant is a "person" as defined under the WPCA and SWMA.

## JURISDICTION AND VENUE

6. This Honorable Court has jurisdiction over this action pursuant to W. Va. Code §§ 22-11-22 and 22-15-15(d).

7. Venue is proper in this court pursuant to W. Va. Code §§ 22-11-22 and 22-15-15(d) because Defendant's Washington Works facility and Dry Run Landfill are located in Wood County and because the violations referenced herein occurred or are occurring in Wood County.

## STATEMENT OF FACTS

8. Defendant was originally authorized to operate the Dry Run Landfill in 1982 through issuance of permit No. IWL-6282-82 by the Department of Natural Resources ("DNR"),



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predecessor to DEP. Such permit authorized Defendant to discharge a specific amount and type of pollutants into Dry Run, a tributary of the North Fork of Lee Creek, which is a tributary of the Ohio River. Pursuant to W. Va. Code §§ 22-11-3(23), Dry Run, the North Fork of Lee Creek, and the Ohio River are all "waters" of the State.

9. On January 12, 1990, DNR reissued IWL-6282-82 to Defendant as Solid Waste/National Pollutant Discharge Elimination System ("SW/NPDES") Permit No. WV0076244. As written, the SW/NPDES Permit was scheduled to expire on January 11, 1995. A copy of SW/NPDES Permit No. WV0076244 is attached hereto as Exhibit 1.

10. Solid Waste/National Pollutant Discharge Elimination System Permit No. WV0076244 incorporated by reference, as a term and condition thereof, information submitted by Defendant with SW/NPDES Application No. WV0076244, as well as information contained in correspondence from Defendant dated August 10, 1987; September 15, 1987; March 1, 1988; April 14, 1988; March 16, 1989; and May 9, 1989. See Exhibit 1, page 1.

11. Under the federal Clean Water Act, 33 U.S.C. § 1251, et seq. and the WPCA, the State of West Virginia, through DEP, is authorized to regulate discharges into the waters of the State of West Virginia through implementation of the National Pollutant Discharge Elimination System ("NPDES") Program. Specifically, the NPDES Program is "the [n]ational program for issuing, denying, modifying, revoking and reissuing, suspending, revoking, monitoring and enforcing permits, and imposing and enforcing pretreatment requirements under Sections 307, 318, 402, and 405 of [the federal Clean Water Act], including any approved [s]tate program." See National Pollutant Discharge Elimination System (NPDES) Program, ("NPDES Program"), West Virginia Code of State Regulations ("CSR"), Title 47, Series 10 § 2.27 (1993).

12. On February 16, 1990, Defendant appealed the terms and conditions of SW/NPDES Permit No. WV0076244 to the Water Resources Board, predecessor to the Environmental Quality Board. The appeal was resolved by the Board's entry of an agreed order dated March 12, 1991, and resulted in a corresponding modification of SW/NPDES Permit No. WV0076244. The agreed order is attached hereto Exhibit 2.

13. By letter dated January 11, 1995, DEP extended the expiration date of SW/NPDES Permit No. WV0076244 from January 11, 1995, to January 11, 1996.

14. By letter dated January 3, 1996, DEP conditionally extended the expiration date of SW/NPDES Permit No. WV0076244 to September 11, 1996. Such extension was conditioned upon Defendant providing DEP with additional information regarding its Dry Run Landfill.

15. By letter dated August 22, 1996, DEP extended the expiration date of SW/NPDES Permit No. WV0076244 to December 11, 1996.

16. In addition to the Dry Run Landfill, Defendant also owns and operates the Washington Works facility which produces plastic and acrylic resins. On September 30, 1994, DEP reissued NPDES Permit No. WV0001279 to Defendant for its Washington Works facility, such permit authorizing Defendant to discharge a specific amount and type of pollutants directly into the Ohio River. NPDES Permit No. WV0001279 is attached hereto as Exhibit 3.

## COUNT I
## Violations of Conditions Not Allowable in State Waters

17. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 16 above as if fully restated herein.

18. Pursuant to the Requirements Governing Water Quality Standards ("Water Quality Standards"), 46 CSR 1 § 3 (1996), entitled "Conditions Not Allowable in State Waters,"

> No sewage, industrial waste, or other wastes present in any of the waters of the State shall cause therein or materially contribute to . . . distinctly visible floating or settleable solids, suspended solids, scum, foam, or oily slicks . . . [d]eposits or sludge banks on the bottom . . . [o]dors in the vicinity of the waters . . . [or] [d]istinctly visible color.

19. On March 23, 1995, Cynthia J. Musser, Inspector, Environmental Enforcement, DEP, inspected the Dry Run landfill. During the inspection, a flow of very black and odorous leachate was emanating from the subject landfill and entering Dry Run, thus discoloring Dry Run and imparting an odor thereto.

20. On May 3, 1995, Inspector Musser conducted an inspection of Defendant's Dry Run Landfill. The leachate from the landfill remained odorous and black, and the discharge from the subject landfill into Dry Run was odorous, turbid, and green with algae, thus discoloring Dry Run and imparting an odor thereto.

21. On August 13, 1996, Inspector Musser conducted an inspection of Defendant's Dry Run Landfill. The leachate from the landfill remained odorous and black, and the discharge from the subject landfill into Dry Run was odorous, turbid, and green with algae, thus discoloring Dry Run and imparting an odor thereto.

22. On August 20, 1996, Inspector Supervisor Charles A. Moses, Inspector James C. Laine, Jr., and Inspector Musser conducted a river patrol of the Ohio River in the vicinity of Defendant's Washington Works facility. During the river patrol, the inspectors sampled deposits on the bottom of the Ohio River and along the river's banks immediately below Outfall 002 of the

Washington Works facility. The sample contained beads made of Nylon, Delron and Teflon - all materials manufactured at the Washington Works facility.

23. During the August 20, 1996, Ohio River patrol, the inspectors likewise sampled deposits along the banks of the Ohio River immediately below Outfall 003 of the Washington Works facility. The sample contained beads made of Nylon, Delron and Teflon - all materials manufactured at the Washington Works facility.

24. During the August 20, 1996, Ohio River patrol, the inspectors sampled deposits on the bottom of the Ohio River below Outfall 005 of the Washington Works facility. The bottom deposit sample yielded beads which contained Teflon and Delron - both materials manufactured at the Washington Works facility.

25. During the August 20, 1996, Ohio River patrol, the inspectors observed floating solids on the Ohio River and along the banks of the Ohio River which were determined to be a mixture of Teflon and wax and which had been discharged from Outfall 005 of the Washington Works facility. The floating solids were located along the West Virginia river bank of the Ohio River for approximately one-half mile below Outfall 005.

26. On March 23, 1995, May 3, 1995, and August 13, 1996, Defendant was in violation of Water Quality Standards 46 CSR 1§ 3, (1996) "Conditions Not Allowable in State Waters," by discharging a turbid flow from Dry Run Landfill into Dry Run, such flow constituting the discharge of distinctly visible colors, turbid conditions, and odors in waters of the State.

27. On August 20, 1996, Defendant was in violation of Water Quality Standards, 46 CSR 1 § 3, (1996) "Conditions Not Allowable in State Waters," by discharging beads of material

manufactured at Defendant's facility, from three separate discharge points, into the Ohio River, waters of the State. Such deposition of material constituted the illegal discharge of settleable solids into waters of the State as well as the impermissible deposition of materials on the bottom of the subject stream.

28. On August 20, 1996, Defendant was in violation of Water Quality Standards, 46 CSR 1 § 3, (1996) "Conditions Not Allowable in State Waters," by discharging a floating or suspended mixture of Teflon and wax, materials manufactured at Defendant's Washington Works facility, into the Ohio River. Such discharge constituted the illegal placement of distinctly visible floating or suspended solids into waters of the state.

## COUNT II
## Failure to Prevent Run-off and Collect and Treat Leachate

29. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 28 above as if fully restated herein.

30. Solid Waste Management Regulations ("SWMR") 47 CSR 38 §§ 4.8.1.b and d (1996), entitled "Leachate Management," state:

> 4.8.1.b Any liquid which comes in contact with waste or accumulates in a portion of the facility where active waste disposal operations are occurring must be handled as leachate and properly treated . . . unless otherwise approved by the director [of DEP] in writing.
>
> . . .
>
> 4.8.1.d In the case of an industrial landfill, the leachate collection and treatment facility must be in place and operable prior to the commencement of landfill operations.



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31. Solid Waste Management Regulation, 47 CSR 38 § 4.5.2.b.A. (1996), entitled "Run-on Control System," states:

> (a) Permittees of all SWLFs [solid waste landfills] must design, construct, operate, and maintain:
>
> (b) A run-on control system capable of preventing flow onto any part of the disposal area including the active portion of the SWLF during peak discharge from at least a 25-year, 24-hour storm.

32. During a May 16, 1994, field review of the Dry Run Landfill conducted by John G. Britvec, Geologist, Office of Water Resources, DEP, and Inspector Musser, Mr. Britvec and Inspector Musser observed that Defendant had not installed a system which would collect and treat leachate, as required by SWMR, 47 CSR 38 § 4.8.1.d (1996).

33. During the May 16, 1994, field review, Mr. Britvec and Inspector Musser observed that Defendant had not installed diversion ditches at the facility, as required by SWMR, 47 CSR 38 § 4.5.2.b.A. (1996).

34. During the May 16, 1994, field review and on other occasions, DEP personnel advised Defendant's managerial personnel that a leachate collection system and diversion ditches must be installed at the subject landfill.

35. During the March 23, 1995, inspection of the Dry Run Landfill, Defendant did not have a collection system or treatment facility to collect or treat leachate generated by the Dry Run Landfill, as required by SWMR, 47 CSR 38 § 4.8.1.d (1996).

36. During the March 23, 1995, inspection, Defendant did not have diversion ditches in place to re-direct surface flow or run-on away from the working surface of the landfill, as required by SWMR, 47 CSR 38 § 4.5.2.b.A (1996).

37. Upon information and belief, in April 1995, Defendant constructed two diversion ditches above the Dry Run Landfill to prevent run-on from flowing across the landfill.

38. During the August 13, 1996, inspection of the Dry Run Landfill, the diversion ditch on the upper side of the landfill was not properly maintained by Defendant, as required by SWMR, 47 CSR 38 § 4.5.2.b.A. (1996).

39. During the August 13, 1996, inspection of the Dry Run Landfill, the facility did not have a leachate collection system installed, as required by SWMR 47 CSR 38 § 4.8.1.d. (1996).

40. From May 16, 1994, until August 13, 1996, and upon information and belief, to the present date, Defendant has been in violation of SWMR 47 CSR 38 § 4.8.1.d. (1996), for failure to install a leachate collection system.

41. From May 16, 1994 until April 1995, Defendant was in violation of SWMR 47 CSR 38 § 4.5.2.b.A. (1996), for failure to design, construct and operate a run-on control system.

42. On August 13, 1996, and, upon information and belief, to the present date, Defendant was and is in violation of SWMR 47 CSR 38 § 4.5.2.b.A. (1996), for failure to maintain a run-on control system.

**COUNT III**
**Failure to Notify of Violations**

43. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 42 above as if fully restated herein.

44. Solid Waste Management Regulation 47 CSR 38 § 4.8.1.j (1996), specifying notification requirements, states:



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> The permittee must immediately notify the director [of DEP] and describe remedial steps to be taken if . . . [o]peration of the leachate treatment facility under this rule cannot prevent the facility from . . . [v]iolating the terms of its permit, [SWMR], the [federal] Clean Water Act and the rules and regulations promulgated thereunder, or W. Va. Code § 22-11 and the rules and regulations promulgated thereunder; or . . . [c]ausing surface water pollution or groundwater degradation, contamination, or pollution.

Further, Solid Waste Management Regulation 47 CSR 38 § 4.8.1.j (1996), states:

> The permittee must immediately notify the director [of DEP] and describe remedial steps to be taken if . . . [t]he facility is generating a quality or quantity of leachate that exceeds the design capacity of the treatment system.

45. Defendant has failed to notify DEP that the operation of the Dry Run Landfill exceeded the design capacity of the landfill's treatment system and that, as a result, such facility was in violation of the terms and conditions of the SW/NPDES permit, the WPCA, and regulations promulgated thereunder.

46. Upon information and belief, Defendant continues to fail to notify DEP when the quality of the leachate generated by the Dry Run Landfill exceeds the capacity of the treatment system, thus causing violations of established discharge limits into Dry Run.

47. On numerous occasions Defendant has been and, upon information and belief, still is in violation of SWMR 47 CSR 38 § 4.8.1.j (1996), for failure to notify DEP when operations of its facility exceeded the design capacity of its treatment system.

## COUNT IV
## Failure to Adequately Compact

48. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 47 above as if fully restated herein.



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49. Solid Waste Management Regulation 47 CSR 38 § 4.6.2.a.C, "Layering and Compaction," (1996) provides:

> Solid waste must be placed in layers not exceeding two (2) feet in depth and compacted with a minimum of three (3) passes with . . . equipment of [effective] compacting ability.

50. During the May 3, 1995, inspection, Defendant was compacting the wastes in the Dry Run Landfill in layers exceeding two feet in depth.

51. During the August 13, 1996, inspection, Defendant was continuing to compact the wastes in the Dry Run Landfill in layers exceeding two feet in depth.

52. On May 3, 1995, and August 13, 1996, and upon information and belief, to the present day, Defendant violated and continues to violate SWMR, 47 CSR 38 § 4.6.2.a.C. (1996), "Layering and Compaction," by failing to properly compact the wastes disposed in the Dry Run Landfill.

## COUNT V
## Changing Composition of Waste Without Proper Authorization

53. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 52 above as if fully restated herein.

54. Section G.1 of the SW/NPDES Permit No. WV0076244 states:

> Only the waste materials specified in [SW/NPDES] Permit Application No. WV0076244 and in the letter dated the 1st day of March 1988 may be disposed in the landfill.

55. Section C.4 of SW/NPDES Permit No. WV0076244 states:

> This Permit may be modified, revoked and reissued, suspended, or revoked for cause. The filing of a request by the permittee for a permit modification,



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revocation, and reissuance or revocation or a notification of planned changes or anticipated noncompliance, does not stay any permit condition.

(Emphasis added.)

56. By letter dated September 14, 1994, Defendant informed DEP that it had changed the composition of wastes being disposed at its Dry Run Landfill.

57. During mid-1994, Defendant did dramatically alter the composition of wastes being disposed at the Dry Run Landfill. not true

58. From mid-1994 through, upon information and belief, the present date, Defendant has disposed and continues to dispose of waste at the Dry Run Landfill, the composition of which differs substantially from that which was specified in Defendant's SW/NPDES Permit application and subsequently authorized in the SW/NPDES Permit No. WV0076244 without having obtained prior approval through a permit modification, in violation of SW/NPDES Permit No. WV0076244 § G.1.

## COUNT VI
## Failure to Properly Operate and Maintain Facility

59. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 58 above as if fully restated herein.

60. Section D.1 of SW/NPDES Permit No. WV0076244, entitled "Proper Operation and Maintenance," states:

> The permittee shall at all times properly operate and maintain all facilities and systems of treatment and control . . . which are installed or used by the permittee to achieve compliance with the conditions of this permit.



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61. On May 1, 1995, upon information and belief, a drain valve for the sediment/leachate pond located at the Dry Run Landfill was opened to lower the level of the pond. The drain valve remained open until May 3, 1995. From May 1 until May 3, 1996, as a result of Defendant opening the drain valve, an indeterminate amount of untreated leachate was discharged directly into Dry Run.

62. During the May 3, 1995, inspection, the discharge/sediment pond was approximately three (3) feet lower than the pond's normal elevation. This condition resulted from Defendant's failure to correctly operate such pond.

63. From May 1 to May 3, 1995, Defendant was in violation of SW/NPDES Permit No. WV0076244, § D.1, by failing to properly maintain and operate the drain valve on its sediment/leachate pond.

## COUNT VII
## Failure to Use a Leachate Liner and Leak Detection System

64. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 63 above as if fully restated herein.

65. Solid Waste Management Regulation 47 CSR 38 § 4.8.3.c.B. (1996), setting forth requirements for surface impoundment, states that surface impoundments, such as the sediment/leachate pond at the Dry Run Landfill:

> must be constructed with a liner system of a minimum of two (2) liners and a leak detection system. Surface impoundments currently in use that do not have liners and a leak detection system as prescribed in [SWMR] must either be closed or retrofitted to conform to this section within six (6) months following the effective date of this rule.



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66. The Defendant has failed to construct a lining for its sediment/leachate pond at the Dry Run Landfill.

67. The surface impoundment used by Defendant to treat leachate has not been closed or retrofitted to properly treat and collect the leachate and does not use a leak detection system.

68. Defendant has violated SWMR, 47 CSR 38 § 4.8.3.c.B. (1996), by operating the Dry Run Landfill without retrofitting the surface impoundment with a liner system and leak detection system.

69. Upon information and belief, Defendant continues to violate SWMR, 47 CSR 38 § 4.8.3.c.B. (1996), by operating the Dry Run Landfill without retrofitting the surface impoundment with a liner system and leak detection system.

## COUNT VIII
## Violations of Numeric Water Quality Standards

70. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 69 above as if fully restated herein.

71. Section C.12 of SW/NPDES Permit No. WV0076244, "Water Quality", states:

> The effluent covered by this permit may not cause a violation of applicable water quality standards adopted by the [Environmental Quality] Board.

72. Section G.8 of SW/NPDES Permit No. WV0076244 requires Defendant to sample Dry Run approximately one thousand feet (1000 ft.) downstream of Outlet 001 for several water quality standard parameters and submit such results on the "Monthly Stream Sampling Report Form."

73. The water quality standard for the concentration of total iron in Dry Run established in the Water Quality Standards, 46 CSR 1, App. E., § 8.15 (1995), is 1.5 milligrams of iron per 1.0 liter of water (1.5 mg/l).

74. The water quality standard for the concentration of manganese in Dry Run established in the Water Quality Standards, 46 CSR 1, App. E., § 8.17 (1995), is 1.0 mg/l.

75. Results submitted by Defendant on its Monthly Stream Sampling Report Forms for May, 1995, July 1995, September 1995, January 1996, and March 1996 showed the stream contained 4.8 mg/l, 1.7 mg/l, 1.9 mg/l, 6.0 mg/l, and 3.7 mg/l of total iron, respectively - all exceeding the 1.5 mg/l total iron water quality standard.

76. Results submitted by Defendant on its Monthly Stream Sampling Report Forms for February 1995, April 1995, and July 1995, showed the stream contained 2.5 mg/l, 1.1 mg/l, and 1.9 mg/l of manganese, respectively - all exceeding the 1.0 mg/l manganese water quality standard.

77. On at least eight separate occasions, Defendant has violated SW/NPDES Permit No. WV0076244, Section C.12, "Water Quality," by exceeding numeric water quality standards.

78. Upon information and belief, Defendant continues to occasionally violate SW/NPDES Permit No. WV0076244, Section C.12, "Water Quality," by continuing to exceed the numeric water quality standards.

**COUNT IX**
**Violations of Discharge Limitations**

79. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 78 above as if fully restated herein.

80. Section E.2.a of SW/NPDES Permit No. WV0076244, entitled "Reporting," states:

> Permittee shall submit each month according to the enclosed format, a Discharge Monitoring Report (DMR) indicating in terms of concentration, and/or quantities, the values of the constituents listed in Part A.1 [of the Permit] analytically determined to be in the plant effluent(s).

81. Section A.1 of SW/NPDES Permit No. WV0076244, entitled "Discharge Limitations and Monitoring Requirements," establishes a maximum daily discharge limit for total suspended solids ("TSS") as 60 mg/l for Outlet 001.

82. Section A.1 of SW/NPDES Permit No. WV0076244, entitled "Discharge Limitations and Monitoring Requirements," establishes a maximum daily discharge limit for total iron as 7.0 mg/l and an average monthly discharge limit for total iron as 3.5 mg/l.

83. Defendant's required Discharge Monitoring Reports ("DMRs") that were sent to DEP for January 1995, May 1995, and July 1995 show the effluent of the Dry Run Landfill contained TSS concentrations of 64 mg/l, 190 mg/l, and 75 mg/l, respectively - all exceeding the maximum daily discharge limit for TSS concentrations set forth in SW/NPDES Permit No. WV0076244 § A.1. Copies of the DMRs for 1995 and 1996 are attached hereto as Exhibit 5.

84. The DMRs submitted by Defendant for May 1995 and May 1996 showed the effluent of the Dry Run Landfill contained total iron concentrations of 8.7 mg/l and 3.6 mg/l, respectively - both exceeding the average monthly discharge limit for total iron concentrations set forth in SW/NPDES Permit No. WV0076244 § A.1. See Exhibit 5.

85. Upon at least five separate occasions, Defendant has violated SW/NPDES Permit No. WV0076244, § A.1, "Discharge Limitations and Monitoring Requirements," by exceeding the only two discharge limitations that were specifically established in SW/NPDES Permit No. WV0076244.

86. Upon information and belief, Defendant continues to violate SW/NPDES Permit No. WV0076244, § A.1, "Discharge Limitations and Monitoring Requirements," by exceeding the only two discharge limitations that were specifically established in SW/NPDES Permit No. WV0076244.

**COUNT X**
**Duty to Comply**

87. Plaintiff hereby realleges and incorporates the allegations set forth in paragraphs 1 - 86 above as if fully restated herein.

88. Section C.1.a of SW/NPDES Permit No. WV0076244, entitled "Duty to Comply" states:

> The permittee must comply with all conditions of this permit. Permit noncompliance constitutes a violation of the [federal Clean Water Act] and [State WPCA] and is grounds for enforcement action."

89. Issuance of SW/NPDES Permit No. WV0076244 was based, in part, upon information submitted by Defendant with SW/NPDES Application No. WV0076244, as well as information contained in correspondence from Defendant dated August 10, 1987, September 15, 1987, March 1, 1988, April 14, 1988, March 16, 1989, and May 9, 1989. A copy of SW/NPDES Application No. WV0076244, dated August 10, 1987, is attached hereto as Exhibit 6.



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90. In the information submitted by Defendant on August 10, 1987, Defendant stated that the surface of the Dry Run Landfill would be crowned to provide a 1% to 2% slope to eliminate ponding on the surface of the landfill and to direct surface water to diversion ditches on either side of the landfill. See Exhibit 6, page 7.

91. In the information submitted by Defendant on August 10, 1987, Defendant stated that compaction would be provided by a dozer operator who would spread the waste material in layers up to two feet thick. See Exhibit 6, page 7.

92. During the March 21, 1995, inspection, Defendant was not providing a 1% to 2% crown to prevent ponding on the surface of the landfill as stated in Defendant's SW/NPDES Application No. WV0076244.

93. During the March 21, 1995, inspection, Defendant was not adequately compacting the wastes at Dry Run Landfill, as stated in Defendant's SW/NPDES Application No. WV0076244.

94. During the May 3, 1995, inspection, Defendant was not providing a 1% to 2% crown to prevent ponding on the surface of the landfill, as stated in Defendant's SW/NPDES Application No. WV0076244.

95. During the May 3, 1995, inspection, Defendant was not adequately compacting the wastes at Dry Run Landfill, as stated in Defendant's SW/NPDES Application No. WV0076244.

96. During the August 13, 1996, inspection, Defendant was not providing a 1% to 2% crown to prevent ponding on the surface of the landfill, as stated in Defendant's SW/NPDES Application No. WV0076244.





000424

97. During the August 13, 1996, inspection, Defendant was not adequately compacting the wastes at Dry Run Landfill, as stated in Defendant's SW/NPDES Application No. WV0076244.

98. Upon at least six separate occasions, Defendant has violated SW/NPDES Permit No. WV0076244 § C.1, by failing to comply with every condition of the subject permit.

99. Upon information and belief, Defendant continues to violate SW/NPDES Permit No. WV0076244 § C.1, by failing to comply with every condition of the subject permit.

**PRAYER FOR RELIEF**

WHEREFORE, pursuant to West Virginia Code §§ 22-1-6, 22-11-4, 22-11-12, 22-11-15, 22-11-22, 22-15-5, 22-15-10, and 22-15-15, SW/NPDES Permit No. WV0076244 §§ C.1 and C.14, and otherwise, Plaintiff, Laidley Eli McCoy, Director, West Virginia Division of Environmental Protection, prays for an Order of Judgment against the Defendant, E. I. DuPont de Nemours & Company, that:

1. Preliminarily and permanently enjoins Defendant at its Dry Run Landfill from violating the Water Pollution Control Act, W. Va. Code §§ 22-11-1 to -28, the regulations promulgated thereunder, the Solid Waste Management Act, W. Va. Code §§ 22-15-1 to -20, the rules and regulations promulgated thereunder, SW/NPDES Permit No. WV0076244, and any and all other environmental laws, rules, or regulations which Defendant might be currently violating;

2. Preliminarily and permanently enjoins the Defendant from placing waste materials at the Dry Run Landfill until such time as the Defendant has installed a properly constructed

landfill and leachate treatment facility, including a a lined impoundment, to prevent the current and future degradation of the waters and soils of the State of West Virginia;

3. Orders Defendant to construct a system of ditches and other permanent, mechanical devices to prevent water from running onto the Dry Run Landfill and mixing with the waste deposited therein;

4. Levies a civil penalty not to exceed $10,000 (ten thousand dollars) per day against Defendant, as specifically authorized by the West Virginia Water Pollution Control Act, at W. Va. Code § 22-11-22, for each of the Defendant's violations of SW/NPDES Permit No. WV0076244, the West Virginia Water Pollution Control Act, and the accompanying rules and regulations;

5. Levies a civil penalty not to exceed $25,000 (twenty-five thousand dollars) per day against Defendant, as specifically authorized by the West Virginia Solid Waste Management Act, at W. Va. Code § 22-15-15(d), for each of the Defendant's violations of SW/NPDES Permit No. WV0076244, the West Virginia Solid Waste Management Act, and the accompanying rules and regulations;

6. Levies a civil penalty not to exceed $10,000 (ten thousand dollars) per day against Defendant, as specifically authorized by SW/NPDES Permit No. WV0076244 § C.14(a), for each of the Defendant's violations of SW/NPDES Permit No. WV0076244, the federal Water Pollution Control Act, the West Virginia Water Pollution Control Act, and the accompanying rules and regulations;



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7. Awards to Plaintiff its costs and disbursements, including reasonable attorneys fees, incurred in this action, as specifically provided by the West Virginia Solid Waste Management Act, at W. Va. Code § 22-15-15(g); and

8. Awards such further relief that this Honorable Court deems appropriate.

Respectfully submitted,

LAIDLEY ELI McCOY, Director,
West Virginia Division of
Environmental Protection

BY COUNSEL:

MATTHEW B. CRUM, DEPUTY CHIEF
SCOTT D. GOLDMAN, ASSISTANT CHIEF
OFFICE OF LEGAL SERVICES
WEST VIRGINIA DIVISION OF
ENVIRONMENTAL PROTECTION
1356 Hansford Street
Charleston, West Virginia 25301
Telephone: 304-558-9160



000847 EID012756

000415

DRY RUN LANDFILL LEACHATE COLLECTION AND TRANSPORT TO WASHINGTON WORKS

(Letter, W. M. Stewart to Barbara S. Taylor, dated 11/8/96)

BCC: P. McGee, Legal, D8065-1
In Turn:
J. H. Little
H. D. Ramsey
W. M. Stewart
R. W. Meloon / L. K. Ireland
R. A. Kirschner
In Turn:
C. T. Alt
J. J. Mentink
R. L. Ritchey
G. Woytowich
D. A. Weber

hcw: 15633-3

RAK007672

EID010842

000819

DuPont
Washington Works
P. O. Box 1217
Parkersburg, WV 26102-1217

DUPONT

November 8, 1996

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Ms. Barbara S. Taylor, Chief
Office of Water Resources
WV Division of Environmental Protection
1201 Greenbrier Street
Charleston, WV 25311-1088

RE: Letter, L. E. McCoy to W. M. Stewart, dated October 15, 1996

Dear Ms. Taylor:

This letter is to inform you of our plans to collect leachate generated at Dry Run Landfill and transport it to Washington Works for treatment in our bio-oxidation wastewater treatment facility. The interim system described in this letter will be used until design modifications specified in our Dry Run Landfill permit renewal application have been completed and are in operation.

Currently, approximately 15 - 20 thousand gallons per day of landfill leachate is collected in a leachate collection system servicing the bottom portion of the active fill area. This system drains through three separate pipe discharges to a surface impoundment (upper pond) for treatment. We propose to route the three existing discharge lines into a six-inch pipe that will gravity drain into an above-ground 50,000 gallon modular collection tank to be located above the upper pond (refer to Attachment 1 for location). Tank dimensions are 38' x 38' x 4'9" and the bolt-together design facilitates assembly at the site.

Prior to erecting the tank, the area above of the upper pond will be cleared and prepared. The tank will be set on a one-foot compacted clay sub-base approximately 42' x 42' in size. If necessary, gravel will be used to stabilize the area prior to placement of the clay base. The tank wall panels comprised of 16 gauge galvanized steel will be supported by galvanized 2" x 2" x 1/8" and 2" x 2" x 3/16" steel angle and galvanized tension cables. A 10 mil geotextile material will be placed over the tension cables prior to installing the 20 mil HDPE tank liner. A photograph of a typical modular tank is included as Attachment 2. Stormwater will be routed around the tank into the upper pond.

RAK007673

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A 3-HP submersible well pump will be mounted inside the tank to transfer the leachate through approximately 500 feet of two-inch underground plastic pipe to a 5,000 gallon tanker truck located at the top of the hill, just east of the closed asbestos fill area. The well pump will deliver between 50-60 gpm of flow and will take approximately 90 minutes to fill a tank truck. We estimate three to five tankers per day will be transported to Washington Works for treatment primarily during daylight hours.

The 50,000 gallon collection tank is sized to provide two feet of freeboard and will be pumped to its minimum capacity each day to ensure that it will not overfill during the night. The collection tank will be inspected for leaks on a daily basis.

An NPDES permit modification request to process the collected leachate through our site wastewater treatment facility will be submitted under separate cover letter.

We request written approval to proceed with installation of our proposed interim leachate pumping system so that we may begin leachate collection and transport to Washington Works.

If you have any questions or need additional information, please call me on 863-4271.

Very truly yours,

W. M. Stewart

W. M. Stewart
Sr. Environmental Control Consultant
Washington Works

Attachments

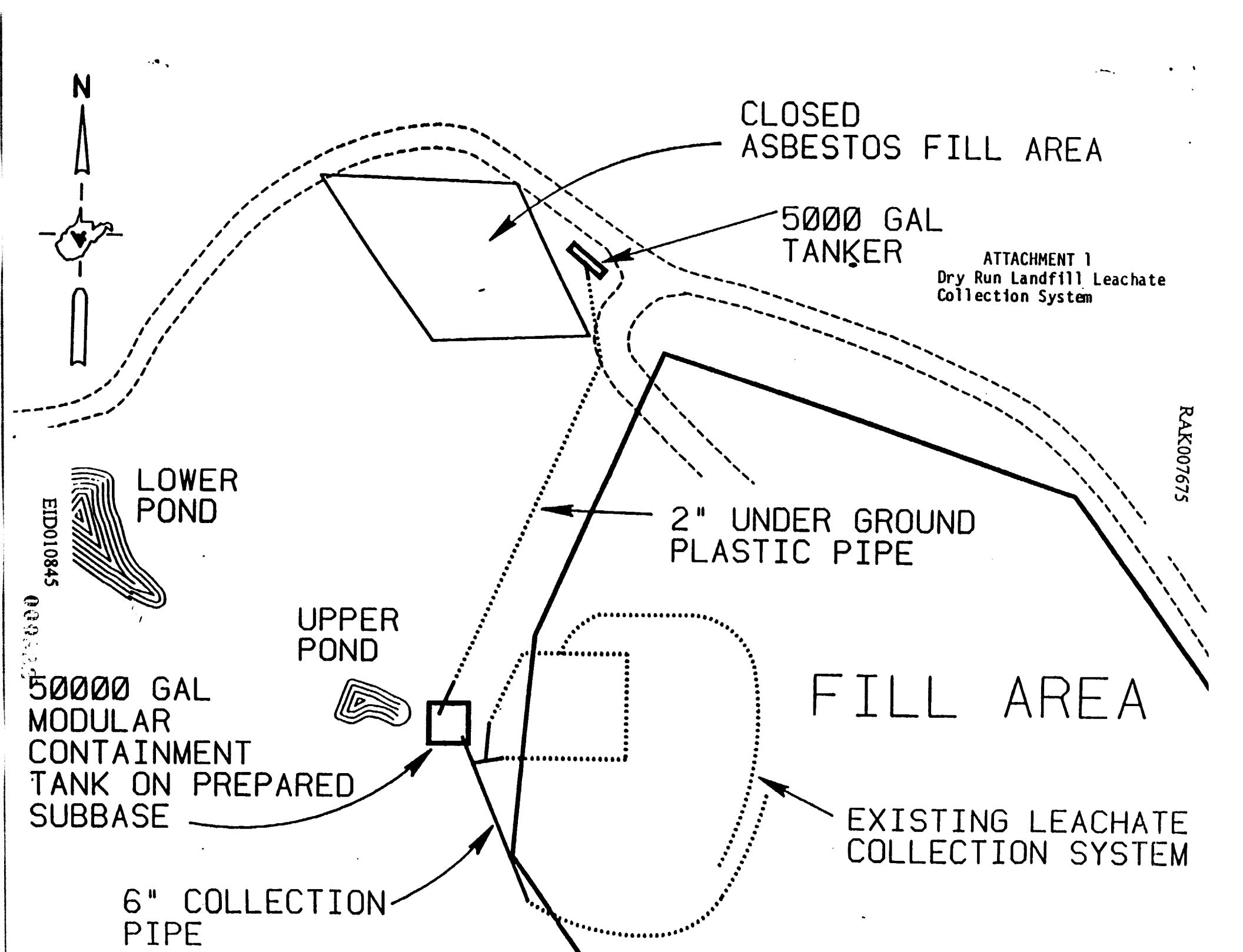
hcw: 15633-2

cc: Ms. Cindy Musser
Office of Env. Enforcement
Div. of Env. Protection
2311 Ohio Avenue
Parkersburg, WV 26101

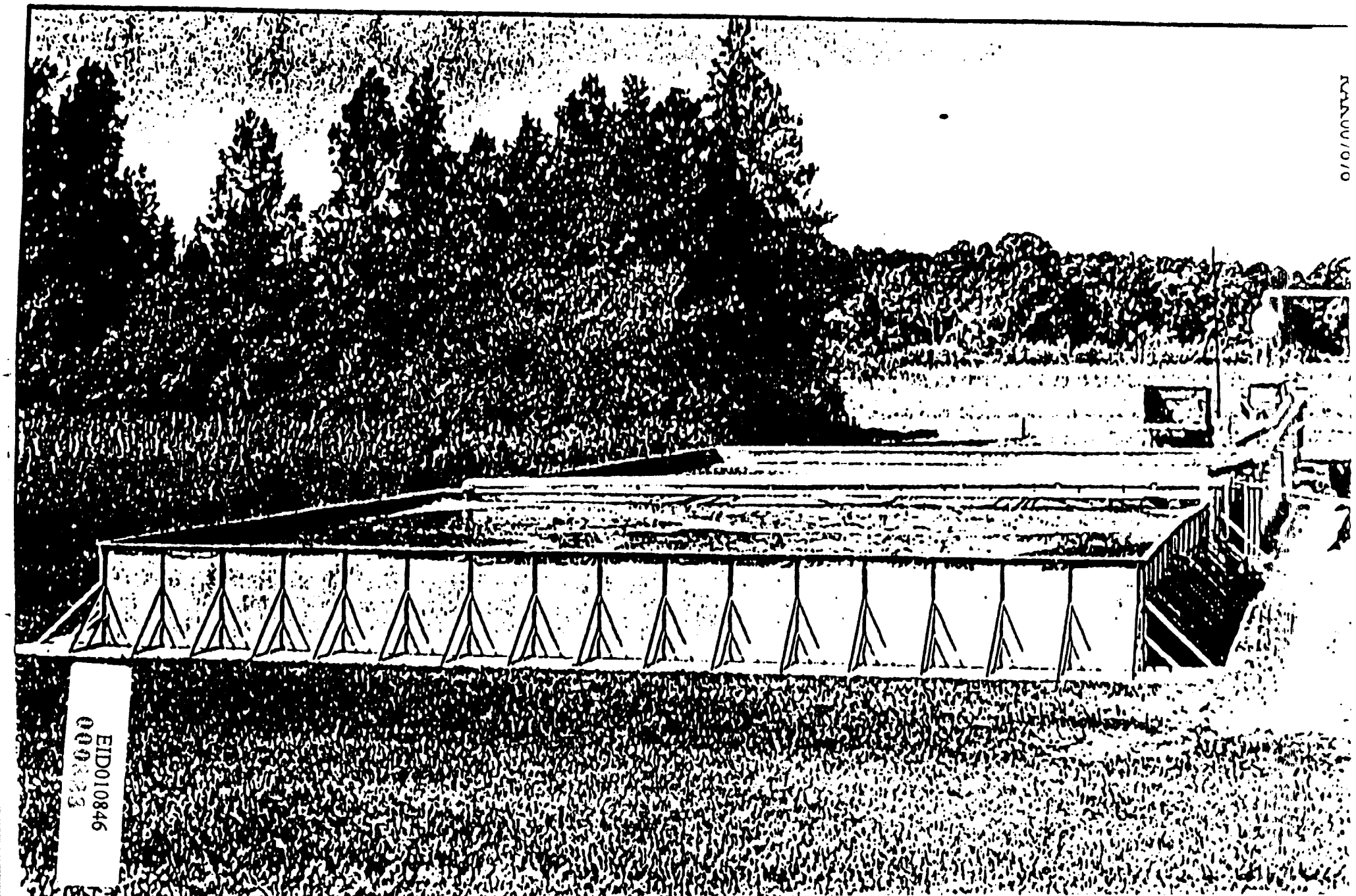
Michael A. Zeto
Water Resources/Waste Management
Environmental Enforcement
1356 Hansford Street
Charleston, WV 25301-1401

John G. Britvec
Office of Water Resources
Industrial Waste Section
1304 Goose Run Road
Fairmont, WV 26554

KAK007674


N
CLOSED
ASBESTOS FILL AREA
5000 GAL
TANKER
ATTACHMENT 1
Dry Run Landfill Leachate
Collection System
RAK007675
LOWER
POND
EID010845
2" UNDER GROUND
PLASTIC PIPE
UPPER
POND
50000 GAL
MODULAR
CONTAINMENT
TANK ON PREPARED
SUBBASE
FILL AREA
EXISTING LEACHATE
COLLECTION SYSTEM
6" COLLECTION
PIPE

Attachment 2 : Modular Tank Photograph



EID010846

GASTON CAPERTON
GOVERNOR

DIVISION OF ENVIRONMENTAL PROTECTION
10 McJunkin Road
Nitro, WV 25143-2506

LAIDLEY ELI MCCOY, Ph.D.
DIRECTOR

November 27, 1996

PLANT FILE COPY
DO NOT REMOVE
File 2:303

Mr. H. David Ramsey, Superintendent
DuPont
Washington Works
Post Office Box 1217
Parkersburg, West Virginia 26102

Dear Mr. Ramsey:

I was very pleased that we could reach a settlement in principal on the Dry Run Landfill issue. As soon as I have an opportunity, assuming I will, to discuss the small business loan program concept with Governor Underwood's staff, I will draft the body of the proposal. Once that occurs, I will call you and arrange a meeting.

As it stands now, the $50,000 SEP (Supplemental Environmental Project) will be available to capitalize the loan program. If we are successful in establishing the program, that will constitute an end to DuPont's commitment. In the event that this loan program should not be adopted, I will contact you to decide how to redirect the Supplemental Environmental Project funding in the manner we have discussed. In this case, I will work with you directly to develop the new SEP, including other funding opportunities from DuPont as per our discussions. I do appreciate your assistance in this matter. Should you have questions, please feel free to call me at 304-759-0515.

Sincerely,

Laidley Eli McCoy, Ph.D.
Director

LEM:jrb

cc: Matthew Crum, Deputy Chief, OLS

CI:000128

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000826

PRC Environmental Management, Inc.
1800 John F. Kennedy Boulevard
Sixth Floor
Philadelphia, PA 19103
215-972-0444
Fax 215-972-0484

December 13, 1996

Ms. Sarah Caspar
On-Scene Coordinator
U.S. Environmental Protection Agency
Region 3
841 Chestnut Building
Philadelphia, PA 19107

**Subject:** **Field Trip Report**
**Preliminary Assessment**
**Dry Run/N. Fork Lee Creek Bovine Site**
**Washington, Wood County, West Virginia**
**TDD Number 03-9609-0013**

Dear Ms. Caspar:

PRC Environmental Management, Inc. (PRC), is submitting the enclosed trip report for the Dry Run/N. Fork Lee Creek Bovine site under the Site Assessment Technical Assistance (SATA) contract labor hour pool, Contract Number 68-S5-3002. If you have any questions about this report or need additional information, please contact me at (215) 656-8703.

Sincerely,

Kevin Scott
Project Manager

Enclosure

cc: Bill Hagel, PRC
Cate Brock, PRC SATA Dedicated Team (cover letter only)

# FIELD TRIP REPORT
# PRELIMINARY ASSESSMENT
# DRY RUN/N. FORK LEE CREEK BOVINE SITE
# WASHINGTON, WOOD COUNTY, WEST VIRGINIA

Prepared for:

U.S. ENVIRONMENTAL PROTECTION AGENCY
Region 3
841 Chestnut Building
Philadelphia, PA 19107

| | | |
|---|---|---|
| EPA Region | : | 3 |
| Date Prepared | : | December 13, 1996 |
| Contract Number | : | 68-S5-3002 |
| TDD Number | : | 03-9609-0013 |
| Prepared by | : | PRC Environmental Management, Inc.<br>(Kevin Scott)<br>(215) 656-8703 |
| On-Scene Coordinator | : | Sarah Caspar |
| Telephone Number | : | (215) 566-3283 |

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USFW 015

## 1.0 INTRODUCTION

PRC Environmental Management, Inc. (PRC), was tasked by the U.S. Environmental Protection Agency (EPA) to conduct a preliminary assessment at the Dry Run/N. Fork Lee Creek Bovine site, in Washington, Wood County, West Virginia. The work was conducted under Contract Number 68-S5-3002, Technical Direction Document Number 03-9609-0013. The purpose of the preliminary assessment was to investigate a citizen's complaint that hazardous materials are present in the surface water and sediments of Dry Run, a tributary of the North Fork of Lee Creek, a tributary to the Ohio River.

## 2.0 BACKGROUND

Mr. Wilber Tennant, the citizen who placed the complaint, claims that contaminants are discharging into Dry Run from a landfill owned by the E. I. DuPont Company (DuPont). Dry Run flows across Mr. Tennant's land before it discharges to the North Fork of Lee Creek. Mr. Tennant raises cattle for beef on his land and his cattle drink from Dry Run. Mr. Tennant alleges that the numerous deaths, blindness, and other illnesses suffered by his herd are directly attributable to the contaminants in Dry Run. He also attributes some of the health-related problems experienced by members of his family to these contaminants.

## 3.0 SITE ACTIVITIES

The following summary describes field activities conducted to investigate Mr. Tennant's allegations.

<u>October 15, 1996</u>

At about 1000 hours, PRC representatives Mr. Kevin Scott and Ms. Alicia Shultz arrived at the West Virginia Department of Natural Resources (WVDNR) in Parkersburg, West Virginia, where they met with Ms.Cynthia Musser, an enforcement officer for the West Virginia Department of Environmental Protection (WVDEP). The PRC personnel reviewed the state's files pertaining to the Dry Run landfill while awaiting the arrival of EPA Region 3 representative, Ms. Sarah Caspar. The Dry Run Landfill, as determined by the file review, is an active industrial solid waste landfill owned and operated by DuPont and the potential source of contamination entering Dry Run. The landfill is used by DuPont to

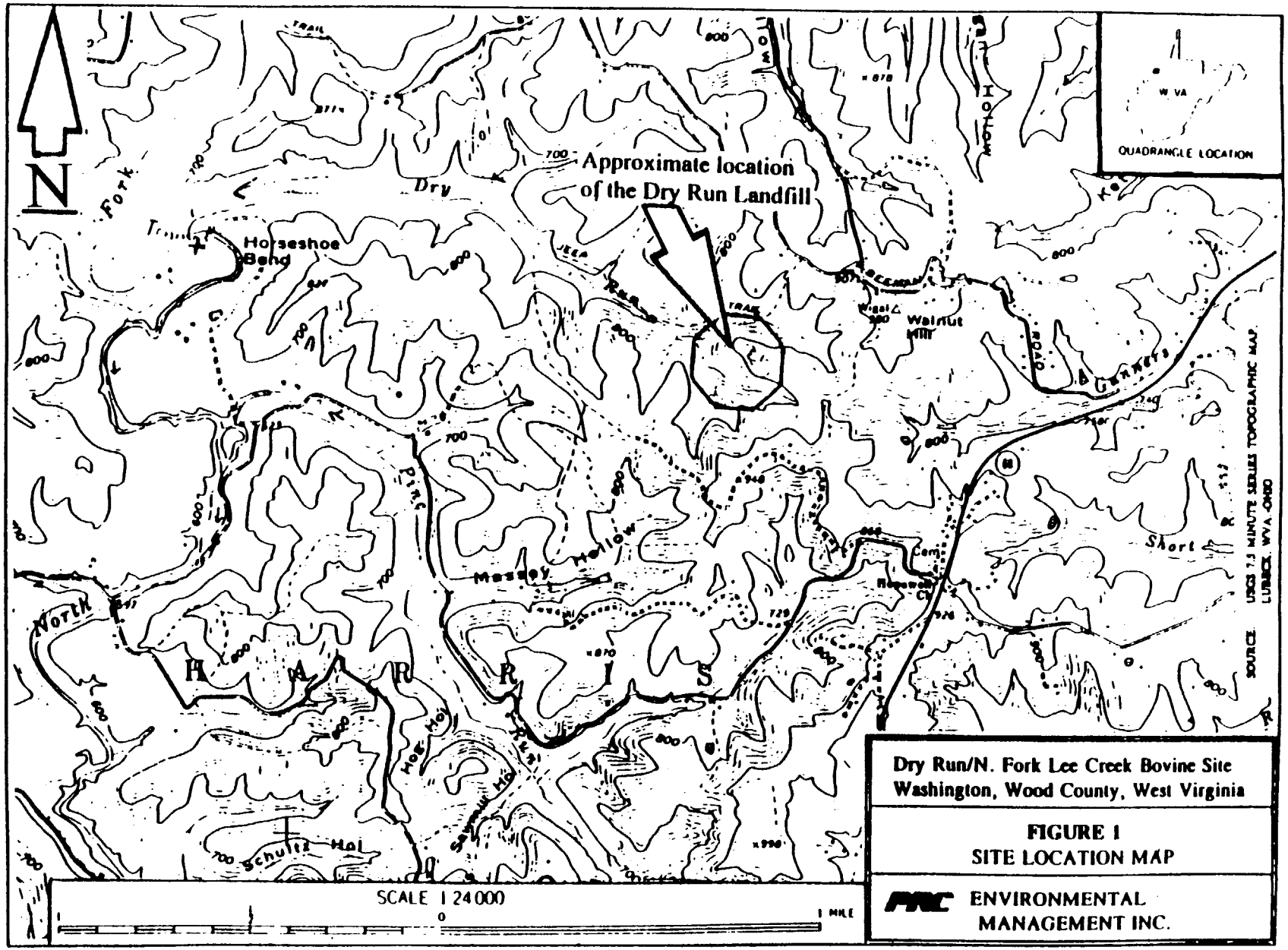
USFW 015

dispose of waste that DuPont identifies as nonhazardous waste generated at its Washington Works facility located in Washington Bottom, West Virginia. The landfill is located in Washington, West Virginia, about 10 miles south of the Washington Works facility. The approximate location of the Dry Run landfill is shown in the site location map provided as Figure 1. The landfill permit application filed with the WVDEP lists wastes deposited at the landfill as fly ash and bottom ash from the facility's nonhazardous waste incinerator, "biosolids" (filter cake) from the facility's industrial wastewater treatment operation, polyamides, acrylics, polyacetyl, polyvinyl butyral, polyethylene terephthalate, cardboard, and construction debris.

While at the WVDNR office, Mr. Scott placed a phone call to Mr. Tennant and arranged a meeting between EPA, PRC, and Mr. Tennant at the Tennant residence. Mr. Scott also contacted Mr. Walt Stewart, environmental manager for the DuPont Washington Works facility, to gain access to the Dry Run landfill. During the conversation between Mr. Scott and Mr. Stewart, Mr. Stewart indicated that DuPont would like to obtain split samples of all samples collected during the site assessment. He also requested a meeting at his office before EPA and PRC conducted the site assessment.

EPA representative Ms. Caspar arrived at the WVDNR Parkersburg office at about 1500 hours. After further review of the state files, EPA and PRC personnel departed the WVDNR office and traveled to the Tennant residence in Washington, West Virginia. During the meeting between EPA, PRC, and Mr. Tennant, Mr. Tennant indicated that more than 200 cattle from his herd have died in the past 2 years from unknown health problems. He also said that he observed other dead wildlife in or near Dry Run, including, minnows, frogs, crows, raccoons, and deer. Mr. Tennant attributes the deaths of his cattle and the wildlife to exposure to the contaminants in Dry Run through ingestion and direct contact. While at the Tennant residence, Mr. Tennant showed the EPA and PRC personnel a cow skull that he had saved. Most of the teeth in the skull showed black staining and were easily removed from the jaw bone. According to Mr. Tennant, the discoloration of the cow's teeth was indicative of fluoride contamination. He also indicated that most of the cattle in his herd were or are affected similarly.

EPA and PRC personnel traveled with Mr. Tennant and his daughter Amy to Dry Run, near its confluence with the North Fork of Lee Creek. Dead minnows were observed in Dry Run a short distance upstream from the confluence of the two water bodies. An oil-like sheen was also observed on the water when stream sediments were disturbed. A whitish-colored foam also was observed on the


N
Approximate location of the Dry Run Landfill
Horseshoe Bend
Dry
Fork
Pine
Massey Hollow
North
Short
Schultz Hol
Walnut Hill
Gunnets
Hollow
Road
SCALE 1 24000
0
1 MILE
W VA
QUADRANGLE LOCATION
SOURCE USGS 7.5 MINUTE SERIES TOPOGRAPHIC MAP LUBECK, WVA-OHIO
Dry Run/N. Fork Lee Creek Bovine Site
Washington, Wood County, West Virginia
FIGURE 1
SITE LOCATION MAP
ENVIRONMENTAL MANAGEMENT INC.

USFW 0161

surface of the water at several locations along Dry Run. PRC took photographs of locations along the run that showed visible signs of contamination. The photographs taken by PRC during the site assessment are included in Attachment A. Mr. Tennant used a video camera to collect additional photographic documentation of stream contamination. PRC observed that the condition of the stream continued to degrade as they moved upstream, closer to the landfill.

October 16, 1996

At about 0900 hours, EPA and PRC personnel met with Mr. Stewart at the DuPont Washington Works facility. Also present at the meeting were Mr. Daniel Weber, senior environmental engineer at the facility, Mr.Richard Kirschner, chemist and wastewater treatment plant engineer, and Tom Waldren, supervisor of operations at the Dry Run landfill. During the meeting, EPA and PRC informed the DuPont personnel of the site assessment sampling objectives and schedule. Mr. Steward suggested that EPA and PRC personnel be shown the landfill and leachate ponds in the morning and return after lunch to collect samples. Before traveling to the landfill, EPA and PRC personnel reviewed DuPont's files and analytical data pertaining to the landfill and Dry Run.

At about 1100 hours, EPA, PRC, and DuPont personnel departed the Washington Works facility for the landfill. The Dry Run landfill is located off interstate Route 68, about 10 miles south of the DuPont facility. While at the landfill, a tractor trailer load of fly ash was dumped onto the upper lift of the landfill. A heavy equipment operator, operating a bulldozer, spread the fly ash across the lift. The bulldozer operator appeared to be the only worker present at the landfill during our visit.

Failure to meet lift height and compaction specifications were noted as permit violations in several of the inspection reports prepared by Ms. Musser for the state. Based on PRC observations during the site visit, the current lift height appeared to be above the 2-foot permit limit. Additionally, the waste material did not appear adequately compacted.

A sample of the wastewater treatment plant sludge was not obtained as planned because this material had been covered with fly ash and compacted before EPA and PRC personnel's arrival. Downgradient from the landfill were two leachate collection ponds. A steady stream of leachate was discharging into the upper pond from a single outfall at the foot of the landfill. According to DuPont, the flow rate of the leachate was approximately 20,000 gallons per day, and the outfall accounts for

USFW 0162



193UN9609131\Trip RPT

nearly all of the flow in Dry Run. An aeration system was in operation in the upper leachate collection pond. The system was powered by a diesel-powered air compressor, located west of the lower leachate collection pond. DuPont indicated that the aeration system is presently alternated between the upper and lower collection ponds. Mr. Weber indicated that DuPont was installing a permanent electrical system for continuous operation of the aeration system. Photographs of the leachate collection ponds and leachate discharge outfalls are included in Appendix A.

At about 1245 hours, all personnel broke for lunch. DuPont agreed to meet EPA and PRC personnel back at the landfill at about 1400 hours.

After returning to the landfill, PRC personnel prepared to collect a sample of the leachate from the outfall at the foot of the landfill. At about 1500 hours, leachate sample L-1 was collected at this location. A sample location map is provided as Figure 2. A sufficient volume of the leachate was collected for multiple analytical parameters, including the target compound list (TCL) of volatile organic compounds (VOC) and semivolatile organic compounds (SVOC); and the target analyte list (TAL) of metals and cyanide; and fluoride, formaldehyde, and sulfate. The sample was collected in containers specific to each of the analytical parameters or laboratory conducting the analysis.

Initially, the PRC sampling team had difficulty obtaining a viable sample for the VOC analysis due to a reaction between the leachate and the hydrochloric acid preservative in the sample vials. The reaction produced gas bubbles, which prevented the samplers from obtaining a sample with no headspace (as required by sampling and analytical protocols). The preservative was removed from the sample vials, and a viable sample was obtained. Three separate laboratories were contracted to analyze the samples for the various analytical parameters. Two EPA contract laboratory program (CLP) laboratories, American Analytical and Technical Services, Inc., and TMA/Skinner and Sherman Labs, Inc., were contracted to analyze for the TCL organic compounds and TAL inorganic compounds, respectively. Lancaster Laboratories, a PRC-subcontracted laboratory, was contracted to analyze samples for fluoride, formaldehyde, and sulfate. A summary of the sample collection information is presented in Table 1.

In addition to the laboratory-contracted analytical parameters, the leachate samples were screened in the field by PRC for pH, temperature, and dissolved oxygen concentration. Due to an instrument

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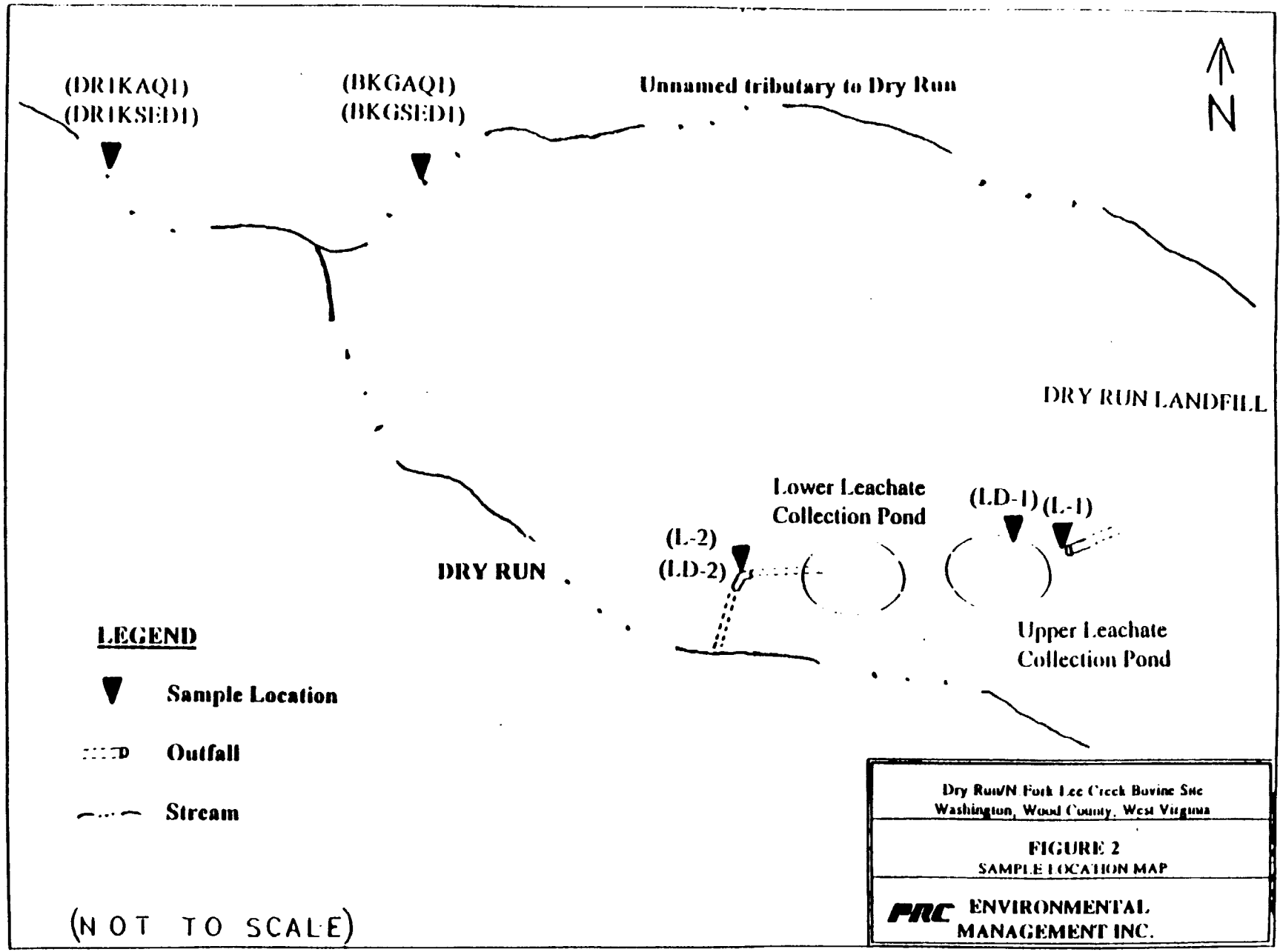


000225

USFW 0164

**TABLE 1**

**SAMPLING AND ANALYSIS SUMMARY**
**DRY RUN/N. FORK LEE CREEK BOVINE SITE**

(Page 1 of 3)

| Sample ID | Location | Matrix | Collection Date | Parameter | Method | Container/Preservative | Laboratory |
|---|---|---|---|---|---|---|---|
| L-1 | Discharge pipe at foot of landfill | Leachate (Aqueous) | 1507; 10/16/96 | TCL VOCs | CLP Protocol (3/90) | 3 x 40 ml, VOA vial, HCl to pH <2, cool to 4 °C | AATSLA |
| | | | | TCL SVOCs, Pesticides, and PCBs | CLP Protocol (3/90) | 2 x 80 oz., amber glass, cool to 4°C | AATSLA |
| | | | | TAL Metals | CLP Protocol (3/90) | 1 x 1 liter, poly bottle, $HNO_3$ to pH >2 | TMA/Skinner |
| | | | | TAL Cyanide | CLP Protocol (3/90) | 1 x 1 liter, poly bottle, NaOH to pH >12 | TMA/Skinner |
| | | | | Fluoride | EPA 340.2 | 100 ml, glass, cool to 4 °C | Lancaster |
| | | | | Formaldehyde | Draft EPA 8315 | 40 ml, glass, cool to 4 °C | Lancaster |
| | | | | Sulfate | EPA 375.4 | 100 ml, glass, cool to 4 °C | Lancaster |
| LD-1 | Northern bank of upper leachate collection pond | Sediment | 1537; 10/16/96 | TCL VOCs | CLP Protocol (3/90) | 2 x 4 oz, wide-mouth clear glass jar, cool to 4 °C | AATSLA |
| | | | | TCL SVOCs, Pesticides, and PCBs | CLP Protocol (3/90) | 2 x 8 oz, wide-mouth clear glass jar, cool to 4 °C | AATSLA |
| | | | | TAL Metals and Cyanide | CLP Protocol (3/90) | 1 x 8 oz, wide-mouth clear glass jar | TMA Skinner |
| | | | | Fluoride | CLP Protocol (3/90) | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| | | | | Formaldehyde | Draft EPA 8315 | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| | | | | Sulfate | Ion Chromatography | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| L-2 | Lower leachate collection pond discharge pipe | Sediment | 1647; 10/16/96 | TCL VOCs | CLP Protocol (3/90) | 3 x 40 ml, VOA vial, HCl to pH <2, cool to 4 °C | AATSLA |
| | | | | TCL SVOCs, Pesticides, and PCBs | CLP Protocol (3/90) | 2 x 80 oz., amber glass, cool to 4 °C | AATSLA |
| | | | | TAL Metals | CLP Protocol (3/90) | 1 x 1 liter, poly bottle, $HNO_3$ to pH >2 | TMA/Skinner |
| | | | | TAL Cyanide | CLP Protocol (3/90) | 1 x 1 liter, poly bottle, NaOH to pH >12 | TMA/Skinner |
| | | | | Fluoride | EPA 340.2 | 100 ml, glass, cool to 4 °C | Lancaster |
| | | | | Formaldehyde | Draft EPA 8315 | 40 ml, glass, cool to 4 °C | Lancaster |
| | | | | Sulfate | EPA 375.4 | 100 ml, glass, cool to 4 °C | Lancaster |



USFW 0165

**TABLE 1**

**SAMPLING AND ANALYSIS SUMMARY**
**DRY RUN/N. FORK LEE CREEK BOVINE SITE**



| Sample ID | Location | Matrix | Time and Date | TCL Analytical Parameters | Analytical Protocol | Container, Preservative | Laboratory |
|---|---|---|---|---|---|---|---|
| LD-2 | Discharge drainage swale downstream of lower leachate collection pond | Sediment | 1647; 10/16/96 | TCL VOCs | CLP Protocol (3/90) | 2 x 4 oz, wide-mouth clear glass jar, cool to 4° C | AATSLA |
| | | | | TCL SVOCs, Pesticides, and PCBs | CLP Protocol (3/90) | 2 x 8 oz, wide-mouth clear glass jar, cool to 4° C | AATSLA |
| | | | | TAL Metals and Cyanide | CLP Protocol (3/90) | 1 x 8 oz, wide-mouth clear glass jar | TMA/Skinner |
| | | | | Fluoride | CLP Protocol (3/90) | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| | | | | Formaldehyde | Draft EPA 8315 | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| | | | | Sulfate | Ion Chromatography | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| BKGAQ1 | Unnamed tributary of Dry Run, west of leachate collection ponds | Surface Water (Aqueous) | 1710; 10/16/96 | TCL VOCs | CLP Protocol (3/90) | 3 x 40 ml, VOA vial, HCl to pH < 2, cool to 4° C | AATSLA |
| | | | | TCL SVOCs, Pesticides, and PCBs | CLP Protocol (3/90) | 2 x 80 oz, amber glass, cool to 4 °C | AATSLA |
| | | | | TAL Metals | CLP Protocol (3/90) | 1 x 1 liter, poly bottle, $HNO_3$ to pH > 2 | TMA/Skinner |
| | | | | TAL Cyanide | CLP Protocol (3/90) | 1 x 1 liter, poly bottle, NaOH to pH > 12 | TMA/Skinner |
| | | | | Fluoride | EPA 340.2 | 100 ml, glass, cool to 4 °C | Lancaster |
| | | | | Formaldehyde | Draft EPA 8315 | 40 ml, glass, cool to 4 °C | Lancaster |
| | | | | Sulfate | EPA 375.4 | 100 ml, glass, cool to 4 °C | Lancaster |
| BKGSED1 | Unnamed tributary of Dry Run, west of leachate collection ponds | Sediment | 1727; 10/16/96 | TCL VOCs | CLP Protocol (3/90) | 2 x 4 oz, wide-mouth clear glass jar, cool to 4 °C | AATSLA |
| | | | | TCL SVOCs, Pesticides, and PCBs | CLP Protocol (3/90) | 2 x 8 oz, wide-mouth clear glass jar, cool to 4 °C | AATSLA |
| | | | | TAL Metals and Cyanide | CLP Protocol (3/90) | 1 x 8 oz, wide-mouth clear glass jar | TMA/Skinner |
| | | | | Fluoride | CLP Protocol (3/90) | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| | | | | Formaldehyde | Draft EPA 8315 | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| | | | | Sulfate | Ion chromatography | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |





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**TABLE 1**

**SAMPLING AND ANALYSIS SUMMARY**
**DRY RUN/N. FORK LEE CREEK BOVINE SITE**

(Page 3 of 3)

| Sample ID | Location | Matrix | Time and Date | TCL Parameters | Analytical Method | Bottle Type, Container, and Preservative | Lab |
|---|---|---|---|---|---|---|---|
| DRIKAQ1 | Dry Run, 1,000 feet downstream of Outfall No. 001 | Surface water (Aqueous) | 1807; 10/16/96 | TCL VOCs | CLP Protocol (3/90) | 3 x 40 ml, VOA vial, HCl to pH < 2, cool to 4° C | AATSLA |
| | | | | TCL SVOCs, Pesticides, and PCBs | CLP Protocol (3/90) | 2 x 80 oz, amber glass, cool to 4° C | AATSLA |
| | | | | TAL Metals | CLP Protocol (3/90) | 1 x 1 liter, poly bottle, $HNO_3$ to pH > 2 | TMA/Skinner |
| | | | | TAL Cyanide | CLP Protocol (3/90) | 1 x 1 liter, poly bottle, NaOH to pH > 12 | TMA/Skinner |
| | | | | Fluoride | EPA 340.2 | 100 ml, glass, cool to 4 °C | Lancaster |
| | | | | Formaldehyde | Draft EPA 8315 | 40 ml, glass, cool to 4 °C | Lancaster |
| | | | | Sulfate | EPA 375.4 | 100 ml, glass, cool to 4 °C | Lancaster |
| DRIKSED1 | Dry Run, 1,000 feet downstream of Outfall No. 001 | Sediment | 1827; 10/16/96 | TCL VOCs | CLP Protocol (3/90) | 2 x 4 oz, wide-mouth clear glass jar, cool to 4 °C | AATSLA |
| | | | | TCL SVOCs, Pesticides, and PCBs | CLP Protocol (3/90) | 2 x 8 oz, wide-mouth clear glass jar, cool to 4 °C | AATSLA |
| | | | | TAL Metals and Cyanide | CLP Protocol (3/90) | 1 x 8 oz, wide-mouth clear glass jar | TMA/Skinner |
| | | | | Fluoride | CLP Protocol (3/90) | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| | | | | Formaldehyde | Draft EPA 8315 | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |
| | | | | Sulfate | Ion Chromatography | 1 x 8 oz, wide-mouth clear glass jar | Lancaster |

Notes:

| | | | | | |
|---|---|---|---|---|---|
| TCL | Target compound list | SVOC | Semivolatile organic compounds | $HNO_3$ | Nitric acid |
| VOC | Volatile organic compounds | VOA | Volatile organic analysis | NaOH | Sodium hydroxide |
| CLP | Contract laboratory program | Hcl | Hydrochloric acid | PCB | Polychlorinated biphenyl |
| TAL | Target analyte list | C | Centigrade | oz | Ounce |
| ml | Milliliter | AATSLA | American Analytical and Technical Services, Inc. | | |
| TMA/Skinner | TMA/Skinner and Sherman Labs, Inc. | | | | |

USFW 016

malfunction, however, pH measurements were not obtained. Dissolved oxygen and temperature measurements for the samples were recorded in the logbook and are summarized in Table 2.

At about 1540 hours, sediment sample SD-1 was collected from the upper leachate collection bank. A sufficient volume of sample was collected for the same set of analytical parameters as described above.

At 1600 hours, PRC personnel collected leachate sample L-2 from the discharge pipe of the lower leachate collection pond. The outfall is a routine sample collection station for DuPont and is identified as No. 001 by a sign staked in the ground near the outfall. From this location, the leachate flows a short distance through an underground culvert and empties into Dry Run. Sediment sample LD-2 was collected in the same vicinity as L-2.

Background surface water and sediment samples were collected from a small unnamed tributary of Dry Run, which converged with Dry Run several hundred feet downstream from Outfall No. 001. Aqueous sample BKGAQ1 and sediment sample BKGSED1 were collected at about 1730.

Between 1800 and 1830 hours, PRC personnel collected aqueous sample DR1KAQ1 and sediment sample DR1KSED1 from a location along Dry Run about 1,000 feet downstream of Outfall No. 001. This location is a routine sampling station for DuPont. A thick film was observed on the surface of the creek at this location. A violent reaction occurred again in the sample bottle when PRC personnel attempted to collect a water sample for VOC analysis. PRC emptied the preservative from the sample vial and recollected the sample. Aqueous and sediment split samples were again provided to DuPont.

**TABLE 2**

**FIELD MEASUREMENTS**
**DRY RUN/N. FORK LEE CREEK BOVINE SITE**

| Sample ID | Temperature (°C) | Dissolved Oxygen (mg/L) |
|---|---|---|
| L-1 | 17.7 | 80 |
| L-2 | 16.2 | 73 |
| BKGAQ1 | 18.7 | 62 |
| DR1KAQ1 | 15.7 | 80 |

DuPont personnel were provided with split samples for each of the locations sampled by PRC. At about 1830 hours, all personnel departed the site. PRC personnel returned to the hotel to complete the sample paperwork. All samples were logged onto laboratory-appropriate chain-of-custody (COC) records, identified with sample-specific sample tags, and placed into coolers in preparation for shipment to the appropriate laboratory. Copies of the EPA organic and the inorganic traffic report and COC records are provided in Appendix B.

October 17, 1996

At about 0900 hours, EPA and PRC personnel returned to the DuPont Washington Works facility to meet with Mr. Stewart. Mr. Caspar updated Mr. Stewart on the sampling activities conducted the previous day. Ms. Shultz signed the completed COC record for the split samples, officially relinquishing the samples to DuPont. At about 1000 hours, EPA and PRC personnel departed the Washington Works facility and traveled to the WVDNR office in Parkersburg, West Virginia, to gather additional information about the site. Ms. Musser was unavailable, and the EPA and PRC personnel departed shortly after their arrival. PRC personnel completed sample shipment preparations and traveled to the local Federal Express office to ship the samples. At about 1230 hours, PRC personnel departed Parkersburg, West Virginia to return to Philadelphia, Pennsylvania.

## 4.0 DATA EVALUATION

Table 3 summarizes fluoride, formaldehyde, and sulfate analytical data from samples collected during the site assessment. The results of analyses of the samples were compared with EPA numeric removal action levels (RAL) for contaminated drinking-water sites, EPA Region 3 risk-based concentrations (RBC) for tap water (developed by Roy Smith, EPA toxicologist), and background sample concentrations. No concentrations of fluoride, formaldehyde, or sulfate exceeded either of the EPA action limits or guidelines. Fluoride was observed at concentrations above background in both of the leachate samples and in the downsteam sample. Fluoride was detected at concentrations of 220 and 160 micrograms per liter (ug/L) in leachate samples L-1 and L-2, respectively, and at a concentration of 130 ug/L in sample DR1AQ1. Fluoride was not detected in the background sample (BKGAQ1). The highest concentration of sulfate (34,500 ug/L) was detected in the background sample.

**TABLE 3**

**SUMMARY OF LABORATORY ANALYTICAL RESULTS (AQUEOUS SAMPLES)**
**DRY RUN/N. FORK LEE CREEK BOVINE SITE**

| Sample ID | EPA | EPA | | LD-1 | LD-2 | BKGAQ1 | DR1KAQ1 | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Limit of Quantitation |
| Fluoride | NA | 2,200 | ug/L | 220 | 160 | <100 | 130 | 100 |
| Formaldehyde | 5,000 | 7,300 | ug/L | <15 | <15 | <15 | <15 | 15 |
| Sulfate | 250,000 (aesthetics) | NA | ug/L | <4,000 | 3,300 | 34,500 | 24,800 | 4,000 |

Notes:

ID Identification

NA Not Applicable

ug/L Micrograms per liter

Table 4 presents a summary of fluoride, formaldehyde, and sulfate analytical data from samples of the sediment collected during the site assessment. The results of analyses of the samples were compared with EPA Region 3 RBCs for fish and to background sample concentrations. Fluoride was detected at concentrations of 396 and 411 milligrams per kilogram (mg/kg) in sediment samples LD-1 and LD-2, respectively; 335 mg/kg in the downstream sediment sample (DR1KSED1); and 198 mg/kg in the background sample (BKGSED1). The fluoride concentrations in these samples are significantly above the EPA guidance level of 81 mg/kg. Fluoride concentrations that exceeded EPA limits are shaded in Table 4.

Appendix C provides the Lancaster Laboratories Sample Analysis Report for the aqueous and sediment samples.

The analytical results for the samples submitted to the CLP laboratories are not included in this evaluation. Upon receipt and evaluation of the remaining sample analytical data, PRC will prepare and submit to EPA an addendum to this trip report.

TABLE 4

SUMMARY OF LABORATORY ANALYTICAL RESULTS (SEDIMENT SAMPLES)
DRY RUN/N. FORK LEE CREEK BOVINE SITE

| Sample ID | EPA [illegible] Risk-Based Concentration (RBC) | | LD-1 Sediment 10/18/96 | LD-2 Sediment 10/18/96 | BKGSED1 Sediment [illegible] | DRYKSED1 Sediment [illegible] | Limit of Quantitation |
|---|---|---|---|---|---|---|---|
| Chemical | | | | | | | |
| Fluoride | 81 N | mg/kg | [illegible] | [illegible] | [illegible] | [illegible] | 18 |
| Formaldehyde | 270 N | mg/kg | 6.8 | 10.8 | 9.4 | <0.7 | 0.7 |
| Sulfate | NA | mg/kg | 44.2 | 36.9 | 38.4 | 14.7 | 6.5 |
| Moisture | NA | % by wt. | 46.1 | 26.0 | 23.4 | 25.8 | 0.5 |

Notes:

Sediment sample analytical results are presented as dry weight results.
Fluoride concentrations that exceed EPA criteria are shaded.

ID Identification
N Noncarcinogenic effects
NA Not applicable
mg/kg Milligrams per kilogram
% by wt Percent by weight

## 5.0 RECOMMENDATIONS

Based on the limited sample analytical data available to PRC at the present time, PRC recommends that an in-depth evaluation of the CLP analytical data be conducted before conducting further sampling at the site. If the evaluation of the CLP data shows that additional sampling is warranted, PRC will make this recommendation to the EPA.

000343

DIVISION OF ENVIRONMENTAL PROTECTION
GASTON CAPERTON
GOVERNOR
1356 Hansford Street
Charleston, WV 25301-1401
LAIDLEY ELI McCOY, PhD.
DIRECTOR

# ORDER

ISSUED UNDER THE
WATER POLLUTION CONTROL ACT
WEST VIRGINIA CODE, CHAPTER 22, ARTICLE 11

and

THE SOLID WASTE MANAGEMENT ACT
WEST VIRGINIA CODE, CHAPTER 22, ARTICLE 15

RECEIVED
JAN 2 9 1997
INDUSTRIAL BRANCH
WATER RESOURCES

ORDER NO. 3850

DATE: December 31, 1996

TO: Mr. H. David Ramsey, Jr.
E. I. Du Pont de Nemours and Company
Washington Works
Post Office Box 1217
Parkersburg, West Virginia 26102-1217

Attention: Mr. Ramsey, Jr.

This order is issued by the Director of the West Virginia Division of Environmental Protection (or "WVDEP"), through his authorized representatives, under the authority of the West Virginia Code, as amended (hereinafter, the "Code"), Chapter 22, Article 11 and Chapter 22, Article 15 to E. I. Dupont de Nemours and Company (hereinafter "Dupont").

## FINDINGS OF FACT

As a result a result of inspections conducted at Dupont's Dry Run Landfill and the Washington Works facility by authorized representatives of the Director and in support of this Order, the Director of the West Virginia Division of Environmental Protection hereby finds the following:

A. Dupont is a person as defined in Chapter 22, Article 11, Section 3 of the Code and Chapter 22, Article 15, Section 2. Dupont is a corporation duly authorized to conduct business in the State of West Virginia; and owns and operates a facility ("Washington Works") in Wood County, West Virginia. Dupont also owns and operates a non-hazardous waste landfill ("Dry Run Landfill"), also in Wood County, West Virginia, which serves the Washington Works facility.

B. Dupont is authorized to operate the Dry Run Landfill under Solid Waste/West Virginia National Pollutant Discharge Elimination System ("SW/WVNPDES") Permit No. WV 0076244. The permit authorizes Dupont to discharge a specified amount and type of pollutant into Dry Run, a tributary of the North Fork of Lee Creek, which is a tributary of the Ohio River. Dupont also holds a WVNPDES Permit No. WV001279 for the Washington Works facility, which allows the facility to discharge a specified amount and type of pollutant into the Ohio River.

C. Authorized representatives of the Director conducted an inspection of the Dry Run Landfill on March 23, 1995, May 3, 1995 and August 13, 1996 and noted a discolored leachate flowing from the landfill into Dry Run.

D. Authorized representatives of the Director conducted an inspection of the Ohio River in the vicinity of the Washington Works facility on August 20, 1996. The inspectors noted solids of wax and polymeric materials of the type manufactured at the Washington Works facility in the Ohio River and its banks.

E. Authorized representatives of the Director inspected the Dry Run Landfill on May 16, 1994, March 23, 1995 and August 13, 1996 and noted the absence of a leachate collection system. Inspectors also noted an absence of diversion ditches during the May 16 and March 23 inspections.

F. Subsequent to the inspection, Dupont constructed two diversion ditches above the landfill to prevent run-on from flowing across the landfill. Dupont incorporated designs for a leachate collection system into the permit application for renewal of the permit for the landfill.

G. Authorized representatives of the Director inspected the compaction procedures used at the landfill on May 3 and August 13, 1996 and noted that the wastes were being compacted in layers exceeding two feet in depth.

H. Dupont notified the WVDEP in September 1994 of a change in the ratio of wastes being disposed at the landfill. Such wastes were disposed of at the landfill through September 1996.

I. Representatives of the Director have noted that the surface impoundment at the landfill does not use a leak detection system or a liner. Dupont incorporated designs for leak detection and liner systems into the permit application for renewal of the permit for the landfill.

J. Authorized representatives of the Director inspected the leachate pond located near the landfill on May 3, 1995 and noted that the water level was approximately three (3) feet below normal elevation. Dupont had opened a drain value in preparation for treatment of the pond.

K. Water quality standards were exceeded at Dry Run Landfill and permit discharge limits were exceeded for Total Suspended Solids, manganese, and iron as reported on Dupont's Monthly Discharge Monitoring Reports.

L. Authorized representatives of the Director inspected the landfill on March 21, 1995, May 3, 1995 and August 13, 1996 and noted that a 1-2% crown was not provided to prevent ponding on the surface of the landfill.

## REQUIREMENTS OF ORDER

Now, in accordance with Chapter 22, Article 15, Section 15 and Chapter 22, Article 11, Section 15 of the Code, it is hereby agreed between the parties and ordered by the Director as follows:

In settlement of all issues in the Findings of Fact, Dupont agrees to the following:

1. Immediately upon the effective date of this Order, Dupont shall cease disposal of filter cake from the Washington Works wastewater treatment system at the Dry Run Landfill. Dupont will refrain from future disposal of such filter cake at the Dry Run Landfill until such time as the installation and operation of all improvements to the landfill as required under the SW/WVNPDES Permit to be issued by the WVDEP for the subject landfill are completed.

2. Immediately upon the effective date of this Order, Dupont shall collect the leachate generated at the Dry Run Landfill and transport it for treatment to the Washington Works wastewater treatment facility upon approval of WVDEP of the necessary permit modifications of the Washington Works WVNPDES permit to allow such treatment. Dupont will continue to collect such leachate and transport it to the Washington Works water treatment facility until the installation and operation of all improvements to the landfill as

required under the SW/WVNPDES Permits to be issued by the WVDEP for the subject landfill are completed.

3. Immediately upon the effective date of this Order, Dupont shall perform such maintenance on the diversion ditches currently in place at the Dry Run Landfill as necessary for controlled flow of surface water through such ditches and away from the active face of the landfill.

4. Within thirty (30) days of the effective date of this order, Dupont will send via certified mail or shall hand deliver to the office of Environmental Enforcement for deposit in the Water Quality Management Fund a check of two-hundred thousand dollars ($200,000) payable to "West Virginia Division of Environmental Protection."

5. Dupont will perform a supplemental environmental project ("SEP") to accrue to the benefit of the environment but which shall not be directed towards facilitating Dupont's compliance with statutory or regulatory requirements. Dupont will make a contribution of fifty thousand dollars ($50,000) to an escrow account to be held for the agency responsible for developing and/or implementing a loan program designed to facilitate environmental compliance by small business. If by May 1, 1997 such a loan program has not been developed, the fifty thousand dollars ($50,000) held in escrow will be returned to Dupont and an alternative SEP shall be developed between the parties, which shall be valued at not less than fifty-thousand dollars ($50,000). In the event that the parties fail to negotiate a mutually acceptable SEP by July 1, 1997, Dupont will send via certified mail or will hand deliver to the office of Environmental Enforcement for deposit in the Water Quality Management Fund a check for fifty-thousand dollars ($50,000) payable to "West Virginia Division of Environmental Protection."

## GENERAL PROVISIONS

1. WVDEP reserves all rights and defenses which they may have pursuant to any legal authority, as well as the right to raise as a basis for supporting such legal authority or defenses, facts other than those enumerated in the Findings of Fact.

2. Dupont hereby waives its right to appeal this order under the provisions of Chapter 22, Article 15, Section 15 and Chapter 22, Article 11, Section 21 of the Code. Under this Order, Dupont agrees to undertake all actions required by the terms and conditions of this Order and consent to and will not contest the jurisdiction of the Director regarding this Order. However, Dupont does not admit any factual or legal determinations made in this Order and reserves all rights and defenses available regarding liability and

responsibility in any proceedings regarding these facilities other than proceedings, either administrative or civil, to enforce this Order.

3. This Order becomes effective on the date indicated and shall terminate upon notification from the Agency that Dupont has satisfactorily completed all tasks set forth in the requirements of the Order.

12/31/96
Effective Date

Name
West Virginia Division of
Environmental Protection

Chief
Title

Name
E. Il Dupont de Nemours
and Company

Plant Superintendent
Title

APO#3850
Water Quality Mgmt Fund

DU PONT

E.I. DU PONT DE NEMOURS AND COMPANY
FINANCE - VENDOR PAYMENT
WILMINGTON, DELAWARE 19898

62-20
311

| DATE | CHECK NUMBER | AMOUNT |
|---|---|---|
| 12/16/96 | 52892530 | $*****200,000.00 |

PAY TO THE ORDER OF
STATE OF WEST VIRGINIA
DEPT OF ENVIRONMENTAL PROT
10 MC GUNKIN RD
NITRO WV 25143

NOT VALID AFTER 90 DAYS

E.I. DuPont de Nemours & Co.

TO:
CITIBANK DELAWARE
A Subsidiary of Citicorp
One Penn's Way
New Castle, DE 19720

⑈52892530⑈ ⑆031100209⑆ 3878857?⑈

Better Things for Better Living . . . from Du Pont

DU PONT

E.I. DU PONT DE NEMOURS AND COMPANY
FINANCE - VENDOR PAYMENT
WILMINGTON, DELAWARE 19898

62-20
311

| DATE | CHECK NUMBER | AMOUNT |
|---|---|---|
| 12/16/96 | 52892531 | $*****50,000.00 |

PAY TO THE ORDER OF
STATE OF WEST VIRGINIA
DEPT OF ENVIRONMENTAL PROT
10 MC GUNKIN RD
NITRO WV 25143

NOT VALID AFTER 90 DAYS

E.I. DuPont de Nemours & Co.

TO:
CITIBANK DELAWARE
A Subsidiary of Citicorp
One Penn's Way
New Castle, DE 19720

⑈52892531⑈ ⑆031100209⑆ 3878857?⑈

Better Things for Better Living . . . from Du Pont

INTEROFFICE MEMORANDUM

Date: 03-Jan-1997 04:20pm
From: RONALD W MELOON
MELOONRW
Dept: EP P&S
Tel No: 304-863-4753

TO: Distribution List

Subject: Leachate to the BioPond

Early this afternoon we successfully brought our first full load of leachate from Dry Run back to the new dewatering facility.

All pumps, equipment and logistics worked well. At this point we intend to continue handling leachate over the weekend.

EID029946

000451

000852

DUPONT

January 9, 1997

DuPont Haskell Laboratory

# HAZARD CHARACTERIZATION FOR HUMAN HEALTH C8 EXPOSURE

## CAS REGISTRY NO. 3825-26-1

Prepared by: L. B. Biegel, Ph.D.
Senior Research Toxicologist

MAB000010



000053

EID080808

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# INTRODUCTION

This document is a Hazard Characterization of C8 for human health. C8 is also known as ammonium perfluorooctanoate (AFPO; CAS # 3825-26-1) and is the primary ingredient in FC-143 FLUORAD Brand Fluorochemical Surfactant. Within this document the chemical will be referred to as C8. However, it is acknowledged that many of the studies discussed actually tested the product FC143, which is a mixture of several straight-chain perfluorocarboxylic acids containing approximately 93.0-97.0% C8.

# I. MAMMALIAN TOXICOLOGY

## I.1. Acute Toxicity Studies

### I.1.a. Acute Oral Toxicity

Numerous acute oral toxicity studies, in several species (rats, mice, guinea pigs, dogs), have been conducted with C8 (see Table I-1). The results of the various studies have been consistent in their results. Administration of a single dose of 12 mg/kg to 3 rats produced no clinical signs of toxicity. Studies demonstrate that newborn and older adult rats appear to be more sensitive than weanlings and young adults. Additionally, while mice and rats appear to be equally sensitive to the acute toxicity of C8, guinea pigs are more sensitive than mice or rats. In the rat, acute oral exposure generally results in enlarged livers, elevations of liver enzyme levels, gastrointestinal irritation, and weight loss. C8 is considered to have moderate acute oral toxicity.

In addition to the numerous studies listed below, several other studies were conducted which investigated the effects of C8 alone or on animals pre-exposed to other chemicals or drugs. Pre-treatment of rats with phenobarbital sodium or proadifen hydrochloride does not result in an alteration of the $LD_{50}$ of C8 (478 mg/kg). Pre or post-dosing with Dowex® 1-X2-Cl Ion Exchange Resin at 1000 mg/kg reduced the mortality compared to rats dosed with C8 alone. A study was conducted to determine if pre-treatment with ethanol (a single dose of 60% or a 15% aqueous solution (v/v) in drinking water for 14 days) modifies the effects of C8 on liver weight. This study determined that pre-treatment with ethanol did not alter C8's effect on liver to body weight ratios.

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Table I-1

SUMMARY OF ACUTE ORAL TOXICITY STUDIES WITH C8

| Study Type | Species | #/sex/dose | Dose (mg/kg) | Vehicle | Results mg/kg | Reference |
|---|---|---|---|---|---|---|
| ALD | Rat | 10 Males | 200, 480, 670 | Corn oil | 480 | HL-565-81 |
| ALD | Rat | 1 Male | 1.5, 12, 40, 120, 200, 300, 450, 670, 1000, 1500, 2250 | Water | 670 | HL-55-61 |
| ALD | Rat | 1 Male | 1.0, 1.5, 2.3, 3.4, 5.1, 26, 40, 60, 77, 90, 120, 130, 170, 200, 300, 450, 670, 2250 | Water | 670 | HL-128-68 |
| $LD_{50}$ | Rat | 5 | 100, 215, 464, 1000, 2150 | Acetone(49%) Corn oil (60%) | Males: 680 Females: 430 | (Griffith and Long, 1980) |
| $LD_{50}$ | Rat | 10 | 200, 400, 450, 500, 670, 1000 | Corn oil | Males: 470 Females: 482 | HL-295-81 |
| $LD_{50}$ as a function of age | Rat | | | | | HL-788-82 |
| | | 10 Weanling males | 350, 400, 450, 525, 670, 710 | Corn oil | 573 | |
| | | 10 Weanling females | 350, 400, 450, 670 | | 580 | |
| | | 10 Young adult females | 350, 425, 500, 670 | | 453 | |
| | | 10 Mature adult males | 200, 240, 300, 350, 400, 500, 720 | | 336 | |
| | | 10 Mature adult females | 225, 350, 400, 450, 670 | | 343 | |
| | | 10 Newborn males | 130, 200, 240, 280, 330, 370 | | 243 | |
| | | 10 Newborn females | 130, 180, 200, 220, 240, 280, 320 | | 258 | |
| $LD_{50}$ castrated vs. intact adults | Rat | | | | | HL-600-81 |
| | | 10 intact males | 200, 480, 670 | Corn oil | 439 | |
| | | 10 intact females | 200, 480, 670 | | 491 | |
| | | 10 orchidectomized males | 200, 480, 670 | | 459 | |
| | | 10 ovariectomized females | 200, 480, 670 | | 400 | |
| $LD_{50}$ | Rat | 10 males | 400, 500, 650 | Corn oil | 478 | HL-567-81 |
| $LD_{50}$ | Rat | | | | 390 | Hazleton Laboratory America, Inc. 2-6-87 |
| $LD_{50}$ | Mice | 10 | 250, 500, 750, 1000, 2000, 4000 | Corn oil | 457 | HL-329-81 |
| Liver function | Dog | 3 Males | 450, 200 | Not stated | Lethal at 450 mg/kg by 48 hours Elevated GPT and GOT which normalized within 1 week at 200 mg/kg | HL-123-65 |
| $LD_{50}$ | Guinea Pig | 10 | 150, 200, 250, 300, 400, 670 | Corn oil | Males: 178 Females : 217 | HL-291-81 |

a). Weanling = 21-days old b). Young adult = 8-10 weeks old c). Mature adult = >10 weeks old d). Newborn = < 2 days old

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I.1.b. Acute Dermal Toxicity

Acute dermal toxicity and irritation studies in rats and rabbits have been conducted with C8. C8 is considered to be mild - moderately irritating to the skin and moderately toxic by the dermal route of exposure. Rat skin showed less irritation than rabbit and in general the effects were more pronounced in males than in females. In addition to dermal irritation several clinical signs of toxicity were observed in both rats and rabbits in response to C8 exposure. These observations included body weight loss, wet and/or stained perineal area, cyanosis (rabbits only), diarrhea (rabbits only), lethargy (rabbits only), labored breathing (rabbits only), and chromodacryorrhea (rats at 7500 mg/kg)

Table I-2

SUMMARY OF ACUTE DERMAL TOXICITY/IRRITATION STUDIES WITH C8

| Study Type | Species | #/sex/dose | Dose (mg/kg) | Results | Reference |
|---|---|---|---|---|---|
| $LD_{50}$ | Rat | 5 | 3000, 5000, 7500 | Male $LD_{50}$ = 6959 mg/kg<br>Female $LD_{50}$ = >7500 mg/kg | HL-659-79 (Kennedy, 1985) |
| Skin Absorption $LD_{50}$ | Rat | 5 Females | 5000 and 7500 | $LD_{50}$>7500 mg/kg<br>Mild skin irritation | HL-682-80 |
| Skin Absorption $LD_{50}$ | Rat | 5 Females | 5000 and 7500 | $LD_{50}$>7500 mg/kg<br>Mild skin irritation | HL-682-80 |
| $LD_{50}$ | Rabbit | 5 Males (2 at 7500) | 1500, 3000, 5000, 7500 | $LD_{50}$ = 4278 mg/kg | HL-659-79 (Kennedy, 1985) |
| | Rabbit | 4 | 100, 1000, 2000 | Lethal<br>4/4 at 2000<br>3/4 at 1000<br>0/4 at 100 | Riker Laboratories Report No. 09790AB0485 |
| Skin Irritation | Rabbit | 6 | 500 mg on intact and abraded sites | Non-irritating | (Griffith and Long, 1980) |
| Skin Irritation | Rabbit | 6 Males | 500 mg | Mild-moderate irritation at 24 hours<br>Slight-moderate irritation at 48 hours | HL-636-79 |

I.1.c. Acute Ocular Toxicity

Eye irritation studies in rabbits have been conducted with C8. C8 is considered to be moderately irritating to the eye. Instillation of solid C8 into rabbit eyes produced moderate corneal opacity, iritis, and conjunctivitis. These ocular effects gradually receded over time. Prompt washing of the eye reduced the effects and provided a more rapid recovery. In addition to the eye irritation studies that have been conducted, rats

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exposed to C8 during a 4-hour inhalation period exhibited corneal opacity and ulceration, which were microscopically evident 42 days post-exposure.

Table I-3

SUMMARY OF EYE IRRITATION STUDIES WITH C8

| Species | #/sex/dose | Dose (mg) | Results | Reference |
|---|---|---|---|---|
| Rabbit | 2 (1 unwashed, 1 washed) | 38.3 | Unwashed eye<br>Moderate-severe corneal opacity<br>Moderate iritis<br>Moderate conjunctivitis<br>At 21-28 days<br>Corneal opacity<br>Mild vascularization<br>Washed eye<br>Slight-moderate corneal opacity<br>Slight-moderate conjunctivitis<br>At 7 days<br>Mild conjunctival redness<br>At 14 days<br>Normal | HL-635-79 |
| Rabbit | 6 unwashed<br>6 washed | 100 | Unwashed eye<br>Moderate irritation<br>Conjunctivitis<br>Iritis<br>Washed eye<br>Conjunctivitis<br>At 7 days<br>4/6 eyes were free of irritation | Biosearch, Inc. Report No. T1395<br>(Griffith and Long, 1980) |

I.1.d. Acute Inhalation Toxicity

Acute inhalation toxicity studies in rats have been conducted with C8. Acute exposure to C8 by inhalation is considered to be highly toxic, with a 4-hour approximate lethal concentration (ALC) in rats of 0.8 mg/L. At concentrations of 2.2 mg/L C8 and higher, all rats died within 48 hours of exposure. At concentrations between 0.38 and 0.83 mg/L C8, rats experienced an initial weight loss following exposure and an increased liver-to-body weight ratio which returned to the high end of the normal range 42 days post-exposure. Additionally, all rats exposed to 0.81 mg/L C8 and higher showed corneal opacity and corrosion.

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Table I-4

SUMMARY OF ACUTE INHALATION STUDIES WITH C8

| Species | #/sex/dose | Concentration (mg/L) | Results | Reference |
|---|---|---|---|---|
| Rat | 6 Males | 4-hour exposure to: 0.38, 0.81, 0.83, 2.2, 4.8, 5.7 | 4-hour ALC = 0.8 mg/L $LC_{50}$ = 0.98 mg/L | HL-160-69 (Kennedy, et al., 1986) |
| Rat | 5 | 1-hour exposure to:18.6 mg/L | No deaths Eye and respiratory irritation | (Griffith and Long, 1980) |

I.1.e. Acute Injection Toxicity

Acute toxicity of C8 when administered by intraperitoneal injection was assessed in mice (3M, 1979). The $LD_{50}$ by intraperitoneal injection in mice is 192 mg/kg.

I.2. Subchronic Toxicity Studies

I.2.a. Subchronic Oral Toxicity

Numerous subchronic oral toxicity studies in several species (rats, mice, and monkeys), have been conducted with C8 (see Table I-5). The results of the various studies have been quite consistent in their results. Administration of C8 in the diet or by daily gastric intubation produced death at concentrations of 1000 ppm and higher for rats and mice and at 30 mg/kg/day for monkeys. The primary target organ for toxic responses in all species studied is the liver. C8 produces increased liver weights, increased liver enzyme activity, hepatocellular hypertrophy, and hepatic peroxisome proliferation.

In addition to the numerous studies listed below, other studies were conducted which investigated the mechanism of action of C8. These studies are summarized in Section III. Mechanisms of Action.

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Table I-5

SUMMARY OF SUBCHRONIC ORAL TOXICITY STUDIES WITH C8

| Study Type | Species | #/sex/dose | Concentration (ppm unless specified) | Results | Reference |
|---|---|---|---|---|---|
| 14-day feeding | Mice | 5 | 10, 30, 100, 300, 1000, 3000, 10000 | 100% mortality at ≥3000; deaths at 1000; increased liver weight/body weight ratio at ≥10 | HL 560-81 |
| 14-day feeding | Mice | 5 | 30, 300, 3000 | 100% mortality at 3000; deaths at 300; weight loss at ≥ 300 increased liver weight/body weight ratios at ≥ 30 | HL12-82 (Kennedy, 1987) |
| 21-day feeding | Mice | 5 | 0, 0.01, 0.03, 0.1, 0.3, 1, 3, 10, 30 | Significantly increased liver weight at 30 | HL 323-82 (Kennedy, 1987) |
| 14-day feeding | Mice | 5 | 30 | Increased liver weight; when C8 was combined with an equal amount of nonadecafluorodecanoic acid, a similar effect was produced | HL 537-82 |
| 9-dose gavage | Mice | 5 | 0.1, 1.0, 10 mg/kg | Weight loss, death in 10 mg/kg females, increased liver weight at 1 and 10 mg/kg | HL 138-83 |
| | Rat | 5 | 0.1, 1.0, 10 mg/kg | Weight loss, increased liver weight in 10 mg/kg males | |
| 14-day feeding with a 2-week recovery period | Rat | 6 Males | 25% Teflon® with C8 as the dispersing agent | Slightly increased liver weights following the recovery period | HL 56-61 |
| 14-day feeding With a 56 day recovery period | Rat | 5 Males | 30, 300 | Decreased body weights at 300; increased liver weights at the end of the feeding period at 30 and 300 and on recovery days 7 and 28 (300 ppm only; elevated blood fluoride levels out to recovery day 7 (final day tested) | HL 326-95 |

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Table I-5 (Con't)

SUMMARY OF SUBCHRONIC ORAL TOXICITY STUDIES WITH C8

| Study Type | Species | Sex (#/sex/dose) | Concentration (ppm unless specified) | Results | Reference |
|---|---|---|---|---|---|
| 28-day feeding | Rat | 5 | 30, 100, 300, 1000, 3000, 10000, 30000 | 100% mortality at ≥ 3000; decreased body weights at ≥ 1000 and 3000 for females; increased liver weight/body weight ratios at ≥ 30 for males and ≥ 300 for females | (Griffith and Long, 1980) |
| | Mice | 5 | 30, 100, 300, 1000, 3000, 10000, 30000 | 100% mortality at ≥ 1000; deaths at ≥ 30; decreased body weights at ≥ 30; cyanosis and muscle weakness at ≥ 3000; increased liver weight/body weight ratios at ≥ 30; panlobular diffuse hypertrophy of hepatocytes | |
| 90-day feeding | Rat | 5 | 10, 30, 100, 300, 1000 | Decreased body weights at ≥ 300; increased liver weights at ≥ 300; panlobular diffuse hypertrophy of hepatocytes at ≥ 1000 with males more affected than females; serum fluoride concentration increased 75 to 226 fold with higher concentrations observed in males | |
| 90-day gavage | Monkey | 2 | 3, 10, 30, 100 mg/kg/day | 100% mortality at ≥ 100; deaths at ≥ 30 (females only); decreased body weights at ≥ 30; no signs of toxicity at 3 mg/kg/day; dose dependent increases in serum and liver fluorine levels (no apparent sex difference) | |
| 90-day feeding with an 8-week recovery period | Rat | 55 Males | 1, 10, 30, 100 | Reduced body weigh at 100; increased palmitoyl CoA oxidase activity at ≥ 30 and transient increases at 10; palmitoyl CoA oxidase activity returned to normal after the 8 weeks of recovery; increased liver weights and hepatocellular hypertrophy at ≥ 10 which was reversible following the recovery period. Serum estradiol, testosterone and lutienizing hormone levels were not affected by dietary exposure to C8, while estradiol levels were slightly elevated at 100 ppm at week 5. The NOAEL = 100 ppm; the NOEL = 1 ppm | (Perkins, 1992) |

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I.2.b. Subchronic Inhalation Toxicity

Similar to the oral toxicity studies, inhalation exposure to C8 produces reduced body weight, increased liver weights, increases in plasma enzymes indicative of liver injury, and pathological lesions in the liver. Measurement of the blood fluoride levels (indicative of the presence of C8) determined that the blood half-life of C8 in the rat is 5-7 days following inhalation exposure.

Table I-6

SUMMARY OF SUBCHRONIC INHALATION TOXICITY STUDIES WITH C8 IN THE RAT

| Study Type | Sex (#/sex/dose) | Concentration | Results (mg/kg) | Reference |
|---|---|---|---|---|
| 10 exposure with a 42-day recovery | 20 | 11, 83 mg/m$^3$ for 6 hours/day | Dose related decrease in body weight, suppression of body weight maintained during the 42-day recovery period at 83; increased plasma enzymes indicative of liver injury present up to 28 days following the last exposure; granular degeneration of hepatocytes; increased liver weights, no ocular effects were observed. The liver effects were not observed after 14, 32, or 42 days of recovery. | HL 253-79 |
| 10 exposure with an 84-day recovery | 24 | 1, 8, 84 mg/m$^3$ for 6 hours/day | Deaths at 84; increased lung, liver and testes weights, no ocular effects observed; increased plasma enzymes indicative of liver injury; increased liver weights at $\geq$ 8 mg/m$^3$; panlobular and centrilobular hepatocellular hypertrophy and necrosis. The liver effects were reversible following a 28-day recovery period. Dose related presence of C8 in the blood, which decreased with time during the recovery period but was still detectable after 84 days of recovery. NOAEL = 1 mg/m$^3$, although 13 ppm organofluoride was detected immediately following exposure to 1 mg/m$^3$ | HL 205-81 (Kennedy, Hall et al., 1986) |

I.2.c. Subchronic Dermal Toxicity

The subchronic dermal toxicity of C8 has been studied in the rat and rabbit (see Table I-7). Similar to the oral toxicity studies, dermal exposure to C8 produces reduced body weight, increased liver weights, increases in plasma enzymes indicative of liver injury and lesions in the liver. Measurement of blood fluoride levels (indicative of the presence of C8) determined that the blood half-life of C8 in the rat is 5-7 days following dermal exposure. A comparison of the dermal exposure studies to the feeding studies leads to the conclusion that the rates of absorption of C8 by these two routes are not significantly different.

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Table I-6

SUMMARY OF SUBCHRONIC INHALATION TOXICITY STUDIES WITH C8 IN THE RAT

| Study Type | Species (#/sex/dose) | Concentration (mg/kg) | Results (mg/kg) | Reference |
|---|---|---|---|---|
| 10 dose with an 84 day recovery | 15 Male Rats | 20, 200, 2000 for 6 hours/day, 5 days/week | Skin irritation at ≥ 200; reversible reduction in body weight at ≥ 200; increased plasma enzymes indicative of liver injury; increased liver weights at ≥ 20; hepatocellular hypertrophy and necrosis at 20 ; no ocular effects observed<br>The liver effects were generally reversible following a 42-day recovery period at ≤ 200.<br>Dose related presence of C8 in the blood, which decreased with time during the recovery period but was still detectable after 42 days of recovery. | HL 589-80 (Kennedy, 1985) |
| Range-finder | 4 Rabbits (sex not specified) | 100, 1000, 2000 | Lethal to 4 of 4 at 2000, 3 of 4 at 1000, 0 of 4 at 100 | Riker Laboratories, Report 09790AB0485, March 15, 1981 |
| 10 exposure with a 14 day recovery | 10 Rabbits | 100 for 6 hours/day, 5 days/week | Reversible reduction in body weight; Blood fluorine levels were 5.4, 6.8, 4.6 ppm for males and 10.1, 12.1, and 3.5 for females at 7, 14, and 28 days of the study, respectively. | |

## I.3. Developmental Toxicity

Developmental toxicity studies have been conducted in rats and rabbits (See Table I-7). The original developmental toxicity study in rats indicated that C8 might be a teratogen in rats. However, because the results were questionable, additional studies were conducted to clarify the result. The additional studies did not confirm the original result. Overall, C8 is not considered to be uniquely hazardous to the conceptus.

The two areas of question were apparent lens abnormalities and skeletal alterations. In the original study, the lens alterations consisted of the following: large lens cleft, dark streak running ½ to ¾ of the way through the lens; or disorganized lens fibers. In the subsequent studies, the lens alterations were determined to be an artifact created in the lens during freehand sectioning. Processing Bouin's-fixed fetal heads that were trimmed on either side of the orbit, instead of through the center of the eye, essentially eliminated this artifact. Examination of the eyes of offspring using focal illumination, indirect ophthalmoscopy, and slitlamp microscopy were also used and did not detect any C8-related eye alterations. The skeletal alterations included ossification sites on the first lumbar vertebrae in rats and 13 ribs in rabbits. Both of these alterations are considered to represent stress-related changes indirectly related to C8-administration.

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Table I-7

SUMMARY OF DEVELOPMENTAL/REPRODUCTIVE TOXICITY STUDIES WITH C8[a]

| Species (#/dose) | Concentration | Results mg/kg | Reference |
|---|---|---|---|
| Rats (# not specified) | 25, 50, 75, 100, 150 mg/kg by gavage | Reduced maternal body weight gain and clinical signs of toxicity at 150; eye abnormalities at 25 and 150 | 3M Report M-601 (1981) |
| 25 Rats[b] | 100 mg/kg by gavage | Maternal deaths, decreased maternal body weight gain; no developmental toxicity or abnormalities observed. | HL 1-82 (Staples, et al., 1984) |
| 12 Rats[c] | 100 mg/kg by gavage | Maternal deaths, decreased maternal body weight gain; no alterations in postpartum viability, growth rate, or development. No ocular effects observed. | |
| Rats (# not specified) | 0.05, 1.5, 50, 150 mg/kg by gavage | Maternal deaths at 150; C8 was not embryotoxic, no abnormal gross findings, no malformations[d]. Fetal lens findings were observed in all groups. Determined to be a processing artifact. No effect on ovaries, reproductive tract, male/female ratio, implantation sites, corpora lutea, or fetal weights. | 3M Report 0681TR0110, 1981 |
| Rats | 0.14, 1.2, 9.9, 21 mg/m$^3$ by inhalation | Maternal deaths at 21; overt maternal toxicity at 9.9; No teratogenic effects were observed in any of the exposed groups; embryo-fetal toxicity was observed at 21; processing artifacts were observed on lens'. | HL 881-81 (Staples, et al., 1984) |
| 18 Rabbits | 1.5, 5, 50 mg/kg by gavage | Reduced maternal body weight gain at 50, C8 was not embryotoxic or teratogenic[e] | 3M Product Toxicity Sheet, May 24, 1996 |

a. Pregnant rats were dosed by gavage on days 6-15 of pregnancy. Pregnant rabbits were dosed by gavage on days 6-18 of pregnancy.
b. Sacrificed on Day 21 of gestation.
c. Pups sacrificed on day 35 postpartum.
d. A significantly higher incidence of the skeletal finding "one sternabrae missing", occurred in the high-dose group. This was a minor skeletal aberration and was not considered a malformation in this study. Furthermore, the incidence of this finding did not differ from the control group or the 3 lower-level treatment groups. The incidences of skeletal findings associated with delayed ossification and rib aberrations were not different among the treatment groups and controls.
e. There was a statistically significant increase in the incidence of 13 ribs in the high dose group and 13 ribs spurred in the mid-dose group. While the findings are significantly greater in the treated animals than in the controls, they are not considered to be teratogenic changes or malformations, rather they are considered to represent stress-related changes to compound administration.

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I.4. Reproductive Toxicity

No information is available on the reproductive toxicity of C8

I.5. Mutagenicity

It has been demonstrated that C8 is not mutagenic in a variety of mutagenicity tests (See Table I-9).

Table I-9

SUMMARY OF MUTAGENICITY STUDIES WITH C8 IN THE RAT

| Study Type | Study Description | Results | Reference |
|---|---|---|---|
| Mutagenicity assay | Assayed in *S. Typhimurium* (TA1535, TA1537, TA1538, and TA100) and *S. cerevisiae* D4 yeast, with and without metabolic activation. | Negative | Litton Bionetics; LBI Project 20838, Feb. 1, 1978 (Griffith and Long, 1980) |
| *In vivo* mouse micronucleus | 3 mice/sex were dosed with 200, 400, 600, 800, and 1000 mg/kg and bone marrow was evaluated at 24, 48 and 72 hours after dosing. | Negative | Corning Hazleton, 17388-0-455, May 16, 1996 |
| Chromosomal aberration | Assayed for ability to induce chromosomal aberrations in CHO cells with and without metabolic activation. | Negative | Corning Hazleton, 17388-0-437, April 25, 1996 |
| Mammalian cell transformation assay | Assayed for cell transformation potential and cytotoxicity in C3H 10T1/2 colony cells. | LD = 50 g/mL; low cytotoxicity<br>No evidence of cell transformation | University of Minnesota Environ. Path Lab, T2942, April 9, 1981 |

I.6. Chronic Toxicity and Oncogenicity

The chronic toxicity and oncogenicity of C8 has been investigated in two 2-year feeding studies in rats (see Table I-10).

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Table I-10

SUMMARY OF CHRONIC TOXICITY AND ONCOGENICITY STUDIES WITH C8 IN RATS

| #/sex/dose | Concentration (mean daily intake, mg/kg/day) | Results | Reference |
|---|---|---|---|
| 50 | 0, 30, 300 ppm (0, 1.5, and 15 mg/kg/day) | Decreased body weight gain and food consumption, increased ataxia. Decreased RBC counts, hemoglobin, and hematocrit values. Increased liver weights, liver cell hypertrophy, degeneration and necrosis. Not considered to be carcinogenic. | Riker Laboratory 0281CR001 (April 1981–May 1983) |
| 156 Males | 0, 0-pair-fed, 300 ppm | Decreased body weight gain and food consumption. Increased estradiol levels. Increased incidence of liver, Leydig cell and pancreatic acinar cell adenomas. | (Cook, et al., 1994) DuPont MR-5686 |

In the original study, in-life findings consisted of a dose dependent decrease in mean body weight gain and increase in food consumption in males, and a slight treatment-related increase in the incidence of ataxia in females. No increase in mortality was observed. C8-related hematologic alteration included decreased red blood cell counts, hemoglobin and hematocrit values observed at various times throughout the 2-year test period. However, the decreases in erythrocyte counts were observed early in the study and did not progress into generalized anemia. Histopathologically, C8-associated alterations were observed in the liver. These changes were characterized by increased liver weights, hypertrophy, hepatocellular degeneration, and necrosis. As with the erythrocyte counts, the hepatic alterations were observed early in the study and showed little progression over the remainder of the 2-year study. The incidence of tumors was relatively low, and the types of neoplasms found were not different from the tumor profiles commonly observed in geriatric rats. Hepatocellular tumors were slightly increased in the 300 ppm males, however, not to the extent that would be expected considering the morphological evidence of hepatocellular stimulation observed at the 1-year necropsy. The incidence of testicular Leydig cell adenomas (0/50, 3/50, and 7/50 at 0, 30, and 300 ppm, respectively) was suggestive of a compound-related effect. However, because the incidence was within the historical control range, it was not considered to be a compound-related effect. Based on the tumor incidence, types of tumors, time of tumor appearance, and the survival rate at the 2 year time point, the overall conclusion was that C8 was not carcinogenic in the rat (Riker Laboratory, 0281CR0012). However, in this original study, some of the pathological findings were equivocal (liver and Leydig cell tumors), even when evaluated by an outside laboratory, and therefore a second 2-year study was conducted to clarify some of these findings.

The second study included many mechanistic endpoints to help determine the mechanism of tumor formation (DuPont MR-5686, Cook, et al., 1994). In addition to the *ad libitum* control, a second control was pair-fed to the C8 group. Peroxisome proliferation (ß-oxidation activity) and cell proliferation (BrdU, 6-day osmotic pumps)

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were measured in the liver and testis. Serum hormone levels (testosterone, estradiol, lutienizing hormone (LH), follicle stimulating hormone (FSH) and prolactin) were also measured. Interim sacrifices were performed at 3-month intervals as well as at 1 month. Increased relative liver weights were observed in the C8-treated rats. Hepatic ß-oxidation activity was also increased in the C8-treated rats at all time points. In contrast, hepatic cell proliferation was not significantly increased in the C8-treated group. C8 did not significantly alter the rate of Leydig cell ß-oxidation or Leydig cell proliferation. Moreover, the rate of ß-oxidation in Leydig cells was approximately 20-times less than the rate of hepatic ß-oxidation, irrespective of treatment. Serum testosterone, FSH, prolactin, and LH levels were unchanged in the C8-treated rats when compared to the controls. There were, however, significant increases in serum estradiol levels in the C8-treated rats at 1, 3, 6, 9, 12, 15, 18 and 21 months. Histopathological evaluation revealed compound-related increases in liver, Leydig cell, and pancreatic acinar cell tumors in C8-treated rats. Based on the data, the Leydig cell tumors appear to be due to the combination of elevated estradiol levels and reduced prolactin levels. The pancreatic acinar cell tumors are related to an increase in serum cholecystokinin (CCK) levels.

## II. METABOLISM

Numerous studies have been conducted investigating the excretion and disposition of C8 in various species. Additionally, studies have been conducted with exposed workers at a manufacturing plant which produces C8. Sex and species differences have been noted, whereas reproductive status in females did not have an effect on excretion or disposition in rats. Rabbits (both sexes), female rats, and male hamsters rapidly excrete C8, while male rats and female hamsters excrete C8 more slowly. Mice (both sexes) excrete C8 even more slowly. C8 also has a long ½-life in humans. Measurement of C8 blood levels in an exposed worker showed that the ½-life in men is greater than 1.5 years.

### II.1. Animal Studies

The excretion and disposition of C8 has been investigated in rats, mice, hamsters and rabbits. Studies have also investigated the influence of route of exposure. These studies are summarized below.

II.1.a. Male and female rats were administered radiolabeled C8 by intravenous injection. Females excreted essentially 100% of the administered dose by 24 hours, while males had excreted only 20% of the administered dose. Radioactive tissue residues were not detectable after 17 days in the females, while at 36 days males had 2.8% of the $^{14}C$ in the liver, 1.1% in the plasma and lower but detectable levels in other organs (Riker Laboratory drug Metabolism Report 1-20 (1980)).

II.1.b. Sex differences in the excretion and disposition of radiolabeled C8 were observed in a study of rats, mice, hamsters, and rabbits. Male and female animals of each species were dosed by gavage with 10 mg/kg C8, and urine and feces were collected at 24, 48, 72, 96, and 120 hours post-dosing. Animals were then sacrificed, and blood and

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tissues were analyzed. The urine and feces of rabbits was also collected at 144 and 168 hours post-dosing, and rabbits were sacrificed at 168 hours.

The female rat and male hamster had excreted over 99% of the administered dose at the time of sacrifice. The male rat and the female hamster had excreted 39 and 60% of the administered dose, respectively, at the time of sacrifice. Both sexes of rabbits excreted the C8 as rapidly and completely as the female rat and male hamster. The male and female mice retained substantial amounts of the total administered radioactivity in their tissues at the time of sacrifice, only excreting 21% of the administered dose at 120 hours post-dosing (HL 62-82)

II.1.c. Cholestyramine, a non-absorbable anion-exchange resin, was demonstrated to protect rats from the acute lethal effect of C8 when administered within 2 hours of C8 dosing (HL 828-81).

A second study was conducted to investigate the effect of cholestyramine on the elimination of $^{14}$C-C8 (10 mg/kg by gavage) from rats and mice (HL 405-82). Adult male rats and mice were given cholestyramine (1000 mg/kg by gavage) 24 hours after dosing with $^{14}$C-C8. The cholestyramine did not enhance the elimination of C8 via the feces, urine, or exhaled air. Similarly, Dowex® Ion Exchange Resin was also able to reduce the acute lethal effect of C8. When rats and mice were given Dowex® resin 24 hours after dosing with C8 no signs of enhanced elimination of C8, via the feces, urine or exhaled air, were observed (HL 405-82).

To further investigate the use of cholestyramine to enhance C8 elimination, a third study was conducted in rats. In this study, rats were dosed with $^{14}$C-C8 (13.3 mg/kg, by iv.) and then were fed diets containing 4% cholestyramine for 14 days. The cholestyramine increased the elimination of C8 via the feces by 9.8 fold and decreased the concentration of C8 found in the liver, plasma, and red blood cells (Johnson, et al., 1984).

II.1.d. A series of experiments was conducted to evaluate the uptake and clearance of C8 from the blood of male and female (pregnant and non-pregnant) rats following oral exposure, and inhalation exposure.

The uptake and clearance of C8 from the blood of female rats following a single oral dose was rapid, with peak reached 1-2 hours post-treatment and virtual total clearance by 24 hours. A dose-response was demonstrated with no apparent changes in blood C8 levels following multiple oral dosing. The slower clearance rate in male rats was demonstrated following a single oral dose. The same general statements apply following inhalation exposure. A single 6-hour inhalation exposure resulted in: peak blood levels within 1 hour after cessation of exposure; the material rapidly cleared from the blood; the number of exposures did not affect blood levels; and male rats cleared the compound much more slowly. Pregnant and non-pregnant rats showed similar C8 blood

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levels following either oral or inhalation exposure (HL 593-91). Specifics of the experiments are summarized below.

II.1.e.1. Oral administration-Blood levels of C8 as a function of time post-dosing (female rats)

C8 levels of 14 ppm were seen 15 minutes following administration of C8. These levels peaked at 30 ppm at 1-2 hours, dropped to 26 ppm by 8 hours, and to 0.7 and 0.045 ppm at 24 and 168 hours, respectively. C8 is absorbed and rapidly cleared from the blood of female rats given a single oral dose.

II.1.e.2. Oral administration-Blood levels of C8 as a function of dose (female rats)

C8 levels 30 minutes following administration of 2.5 – 150 mg/kg ranged from 3 –162 ppm. The same dose response was observed at 24 hours with blood levels ranging from 0.12 - 18 ppm. The response was linear. The level of C8 in the blood is directly related to the amount of C8 administered.

II.1.e.3. Oral administration-Blood levels of C8 as a function of number of doses (female rats)

Blood levels in female rats given 1 versus 11 doses of C8 were not considerably different. Concentrations at 15 minutes following administration were 14 and 17 ppm for 1 and 11 doses, respectively. At 30 minutes C8 concentrations were 16 and 25 ppm; at 8 hours 26 and 13 ppm; at 24 hours, 0.7 and 0.8 ppm; and at 168 hours, 0.045 and 0.10 ppm for 1 and 11 doses, respectively. C8 does not appear to accumulate in the blood of female rats following repeated oral administration. The number of treatments does not appear to influence the C8 blood level.

II.1.e.4. Oral administration-Blood levels of C8 following a single 25 mg/kg dose (male and female rats)

| Time following single oral dose (hours) | Blood Levels of C8 (ppm) | |
|---|---|---|
| | Male Rats | Female Rats |
| ½ | 23 | 16 |
| 8 | 63 | 26 |
| 24 | 50 | 0.7 |
| 168 | 23 | 0.045 |

C8 is retained in the blood of male rats to a greater extent than female rats.

II.1.e.5. Inhalation exposure-Blood levels of C8 as a function of time post-exposure (female rats)

C8 levels of 96 ppm were observed 15 minutes following a single 6-hour exposure to 10 mg C8/$m^3$. The level was maintained through 1 hour, fell to approximately 70 ppm at 8 hours, 52 ppm at 24 hours, and dropped to 0.39 ppm at 168



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hours post-exposure. This same general pattern was observed in rats exposed to either 0.1 or 1 mg/m$^3$. The lag phase seen following oral exposure was not observed here due to blood sampling following a 6-hour inhalation exposure (rather than a single dose at a given time). C8 is absorbed and rapidly cleared from the blood of female rats following a single inhalation exposure.

II.1.e.6. Inhalation exposure -Blood levels of C8 as a function of dose (female rats)

| Time following single inhalation exposure dose (hours) | Blood Levels of C8 (ppm) Following exposure to C8 at | | |
|---|---|---|---|
| | 0.1 mg/m$^3$ | 1 mg/m$^3$ | 10 mg/m$^3$ |
| ½ | 2 | 7 | 109 |
| 2 | 2 | 17 | 69 |
| 8 | 0.85 | 4 | 71 |
| 24 | 0.14 | 0.56 | 52 |

The response is linear at 30 minutes. C8 blood levels are directly related to the amount of C8 inhaled. At the high concentration used, the clearance rate is somewhat slower than observed at the lower levels. This suggests massive overloads in the clearance system.

II.1.e.7. Inhalation exposure -Blood levels of C8 following a single 6 hour exposure to 10 mg/m$^3$ (male and female rats)

| Time following single oral dose (hours) | Blood Levels of C8 (ppm) | |
|---|---|---|
| | Male Rats | Female Rats |
| ½ | 137 | 109 |
| 2 | 157 | 69 |
| 8 | 182 | 71 |
| 24 | 147 | 52 |

C8 is retained in the blood of male rats to a greater extent than female rats following inhalation exposure.

II.1.e.8. Oral administration-Blood levels of C8 following a single 25 mg/kg dose (pregnant and non-pregnant female rats)

| Time following single oral dose (hours) | Blood Levels of C8 (ppm) | |
|---|---|---|
| | Pregnant Rats | Non-pregnant Rats |
| ½ | 16 | 10 |
| 2 | 33 | 39 |
| 8 | 26 | 31 |

C8 clearance following oral dosing is similar in pregnant and non-pregnant female rats.

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II.1e.9. Oral and Inhalation exposure -Blood levels of C8 as a function of number of exposure concentration (pregnant female rats)

| Time following single oral dose of 25 mg/kg | Blood Levels of C8 (ppm) Following | | |
|---|---|---|---|
| (hours) | 1 exposure | 6 exposures | 10 exposures |
| ½ | 18 | 12 | 12 |
| 2 | 39 | 37 | 15 |
| 8 | 31 | nd[a] | 11 |
| 24 | 2 | nd | 1 |

| Time following single inhalation exposure to 10 mg/m$^3$ | Blood Levels of C8 (ppm) Following | |
|---|---|---|
| (hours) | 1 exposure | 10 exposures |
| ½ | 77 | 53 |
| 2 | 90 | nd |

a. nd = not done

When comparing the C8 levels seen at 2 and 8 hours, following 10 consecutive oral doses, there appears to be a lowering of the C8 blood levels. Blood levels following 1 or 10 consecutive inhalation exposures (6 hours/day) were not different. C8 does not appear to accumulate in the blood of pregnant rats following repeated oral or inhalation exposures.

II1.f. The ability of $^{14}$C-C8 to transfer through the placenta was investigated in rats (HL 61-82). A single dose of 10 mg/kg $^{14}$C-C8 was administered to pregnant rats on the 19$^{th}$ day of pregnancy. Maternal blood and placental levels of $^{14}$C-C8 increased between 2 and 4 hours post-dosing, and decreased between 4 and 8 hours post-dosing.

| Time following single oral dose of 10 mg/kg | Levels of C8 | |
|---|---|---|
| | Maternal | Fetal |
| (hours) | (µg equivalents/mL blood) | (µg equivalents/mL tissue) |
| 2 | 12 | 0.7 |
| 4 | 20 | 3 |
| 8 | 12 | 3 |

## II.2. Human Exposure

Determinations of organic fluorine blood levels in workers exposed to C8 in an industrial environment were performed. Approximately 90% of the organic fluorine was composed of the C8 anion. The highest levels were found in workers with the longest work history in fluorochemical production. The majority of the values remained at approximately the same level throughout the 2 ½ year monitoring period. Monitoring of C8 blood levels of a worker who was removed from the fluorochemical production site due to high C8 blood levels (70 ppm) suggests that fluorochemicals are very slowly



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eliminated. From this limited data it is hypothesized that the ½-life of C8 is 1.5 years in men (Ubel, et al., 1980).

| Group | Number analyzed | Blood Organic Fluorine Levels (ppm) |
|---|---|---|
| Normal human sera | from published literature | 0.01-0.13 |
| Industrial controls | 4 | 0.01-0.08 |
| Laboratory personnel (>20 years exposure) | 8 | 0.04-2.00 |
| Plant workers | 49 | 1.00-71.00 |

## III. MECHANISMS OF ACTION

C8 is not metabolized in rats. C8 produces hepatomegaly, induces hepatic peroxisomes in mice and rats, and has been shown to produce hepatic, Leydig cell, and pancreatic acinar tumors in a 2-year feeding study in rats. The male rat is more susceptible to the toxic effects of C8 than the female rat, presumably due to the longer ½-life in males. Short-term studies have been conducted investigating the mechanisms of action responsible for the various effects.

### III.1. Investigation of C8s' Effect on the Liver.

III.1.a. Because C8 had been shown to induce a striking hepatomegaly in rats, a study was conducted to investigate the hepatic biochemical and morphological changes associated with C8-induced hepatomegaly in rats (Pastoor, et al., 1987). In this study male rats were dosed daily for 1, 3, or 7 days with 50 mg C8/kg body weight by intragastric intubation. The total cytochrome P450 content and activity of benzphetamine *N*-demethylase was increased in the livers of C8-treated rats, indicating the proliferation of smooth endoplasmic reticulum. In contrast, the soluble, cytoplasmic enzymes, glutathione *S*-transferase and UDP-glucuronyltransferase, were unaffected. Carnitine acetyltransferase activity was disproportionately increased relative to carnitine palmitoyl transferase activity, confirming the predominant proliferation of peroxisomes versus mitochondria. Electron microscopy confirmed the proliferative response of the endoplasmic reticulum, peroxisomes, and microsomes in the livers of the C8-treated rats. This study also demonstrated that C8 does not possess hypolipidemic activity.

III.1.b. C8 increased serum estradiol concentrations in 2-week gavage studies, and feeding studies at various time points up to 2 years. This was accompanied by increases in liver weights, and hepatic β-oxidation activity (Cook, et al., 1992; Cook, et al., 1994). Since peroxisome proliferators induce both β-oxidation activity and cytochrome P450 enzymes, an investigation was conducted to determine if C8 increases serum estradiol levels by stimulating aromatase activity (Liu, et al., 1996a). Fourteen days of treatment with up to 40 mg C8/kg/day produced dose-dependent increases in liver weights, serum estradiol, and hepatic aromatase activity. A significant linear correlation was established between estradiol and hepatic aromatase activity. *In vitro* experiments using cultured hepatocytes suggest that the increase in serum estradiol is at least partly



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due to a direct effect on the liver to increase synthesis of estradiol through induction of aromatase cytochrome P450 in the endoplasmic reticulum.

III.2. Investigation of C8s' Effect on Testicular Leydig Cells

Because C8 produced an increased incidence of testicular Leydig cell tumors in a 2-year feeding study in rats, and because C8 was negative in short-term tests for genotoxicity, a non-genotoxic (hormonal-mediated) mechanism for tumor formation was investigated. The studies summarized below support a hormonally-mediated mechanism of Leydig cell tumorigenesis: C8 produces an increase in hepatic aromatase activity, which elevates serum estradiol concentrations, which in turn modulates growth factors in the testis, which results in tumor formation.

III.2.a. Fourteen days of treatment with up to 50 mg C8/kg/day produced dose-dependent increases in hepatic β-oxidation activity, and serum concentrations of estradiol, and decreases in serum testosterone concentrations, body weights, and relative accessory sex organ weights in male rats (Cook, et al., 1992). Challenge experiments, using human chorionic gonadotropin (hCG), gondaotropin-releasing hormone (GnrH), or naloxone challenges, suggest that the decrease in testosterone may be due to a lesion at the level of the testes, due to a decrease in the conversion of 17α-hydroxyprogesterone to androstenedione.

III.2.b. Using *in vitro, in vivo* and *ex vivo* studies, C8 was examined for its ability to directly affect Leydig cells *in vitro* using isolated Leydig cells from untreated rats, and *ex vivo* using Leydig cells isolated from C8-treated rats. Additionally, the ability of C8 to affect testicular interstitial fluid hormone levels and induce aromatase activity was investigated (Biegel, et al., 1995). The *in vitro* studies demonstrated that C8 directly inhibits testosterone production, while the *ex vivo* studies demonstrated that this inhibition is reversible. In the *in vivo* study, serum and testicular interstitial fluid estradiol were increased and testicular interstitial fluid transforming growth factor α were increased. Additionally, hepatic aromatase activity was increased while aromatase activity levels were not affected in the testis, muscle, or fat. These data suggest that the increases in estradiol levels are primarily due to increases in aromatase activity.

III.2.c. Previous studies with C8 showed a direct effect on Leydig cells to alter steriodogenesis. It was therefore proposed that peroxisome proliferators, in general, may directly affect Leydig cell function to produce Leydig cell tumors. A study investigating whether several peroxisome proliferators (including C8), directly affect Leydig cell function *in vitro* was conducted. This study showed that peroxisome proliferators, as a class of compounds, directly modify the steroidogenic function of Leydig cells *in vitro*. This also suggests that compounds which directly affect Leydig cell function *in vitro* may also induce Leydig cell tumors *in vivo* (Liu, et al., 1996b).

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III.3. Investigation of C8s' Effect on the Pancreas

Several peroxisome proliferators have been shown to produce pancreatic acinar cell hyperplasia/adenocarcinomas in 2-year feeding studies, including C8. Therefore, *in vitro* and *in vivo* investigations of C8's (*in vitro* only) and Wyeth-14, 643's (a model peroxisome proliferator) mechanism of tumorigenesis in the pancreas were conducted. These mechanisms include cholecystokinin receptor agonism ($CCK_A$) trypsin inhibition, alterations in gut fat content, cholestasis and altered bile flow/composition. All of these mechanisms enhance pancreatic growth either by binding to the $CCK_A$ receptor or by increasing plasma CCK levels. C8 did not bind directly to the $CCK_A$ receptor and it failed to inhibit trypsin, a common mechanism for increasing plasma CCK levels. *In vivo* studies with Wyeth-14, 643 suggest that these peroxisome proliferators produce pancreatic tumors by cholestasis, which may be responsible for the decrease in bile acid output which contributes to the increase in plasma CCK levels. Therefore, for Wyeth-14, 643 (and perhaps C8), the pancreatic tumors may be secondary to hepatic cholestasis (Obourn, et al., 1997).

IV. CLINICAL REPORTS OF HUMAN EXPOSURE

IV.1.a. Health screening examinations were offered to employees of a 3M plant that produced C8, as well as other fluorochemicals. No health problems related to exposure were encountered among those examined. Additionally, no relationship was observed between deviations from normal laboratory test results and blood levels of organic fluorine (the liver enzyme SGGT was the most frequently encountered test result exceeding the normal range. C8 exposure levels ranged from 0.03 to 7.6 $mg/m^3$ (Ubel, et al., 1980).

IV.1.b. A study was made of Washington Works employees potentially exposed to C8. Results of blood chemistry testing (SGOT, LDH, AP, and bilirubin) indicated no conclusive evidence of an occupationally related health problem among workers exposed to C8 (Fayerweather, 1981).

IV.1.c. Although C8 is the major organofluorine compound found in humans, little information is available concerning human responses to C8 exposure. Therefore, a study was conducted among 115 workers exposed to C8 occupationally (serum fluorine levels varied between 0 and 26 ppm, with a mean of 3.3). In an examination of the cross-sectional associations between C8 and hepatic enzymes, lipoproteins, and cholesterol, there was no significant clinical hepatic toxicity of the C8 levels observed in this study (Gilliland and Mandel, 1996). Serum C8 levels were positively associated with estradiol and negatively associated with free testosterone and not associated with luteinizing hormone. The negative association between testosterone and C8 was stronger in older men. Thyroid stimulating hormone and C8 were positively associated. Prolactin and C8 were positively associated in moderate drinkers. The effect of adiposity on serum glutamyl oxaloacetic acid and glutamyl pyruvic transaminase decreased as C8 increased. The induction of gamma glutamyl transferase by alcohol was decreased as C8 increased. The effect of alcohol on HDL was reduced as C8 increased. A positive association

between hemoglobin, mean cellular volume, and leukocyte counts with C8 was observed. These results suggest that C8 affects male reproductive hormones and that the liver is not a significant site of toxicity in humans at the C8 levels observed in this study. However, C8 appears to modify hepatic and immune responses to xenobiotics (Gilliland and Mandel, 1993).

## V. EPIDEMIOLOGY

V.1.a. A retrospective cohort mortality study was made of employees at a 3M plant where C8 and other fluorocompounds are manufactured. Records on 4218 employees were reviewed. Only those who worked for 6 months or more (3688 workers) were included in the mortality follow-up. Of the 180 known deaths, 177 death certificates were obtained. Overall the number of deaths was significantly less than expected. The observed-to-expected ratio for cancer deaths was 1.0 (Ubel, et al., 1980).

V.1.b. In a retrospective cohort mortality study, a relationship between mortality and employment at a plant where C8 and other fluorocompounds are manufactured were investigated (Gilliland and Mandel, 1993). The cohort consisted of 2788 male and 749 female workers employed between 1947 and 1983. The all-causes standardized mortality rate (SMR) was 0.75 for males and 0.77 for females. There was no significantly increased cause-specific SMR for men or women. The SMRs for prostate cancer were 2.03 in the exposed group and 0.58 in the not-exposed group. In the exposed group there were 4 observed and 2 expected deaths from prostate cancer. Among men, 10 years of employment in C8 production was associated with a significant 3-fold increase in prostate cancer mortality when compared to no employment in production. Given the small number of prostate cancer deaths and the natural history of the disease, the association between production work and prostate cancer must be viewed as hypothesis generating and not over interpreted. If the prostate cancer mortality excess is related to C8, the results of this study and other clinical studies suggest that C8 may increase prostate cancer mortality through endocrine alterations.

## VI. DISCUSSION OF ENDPOINTS

### VI.1. Discussion of Target Organs

The primary target organ for C8-induced toxicity is the liver in mice, rats, and dogs, regardless of route of exposure. The hepatotoxicity manifests as increased liver weights, hepatocellular hypertrophy, liver degeneration, increases in liver enzymes, necrosis of the liver, and induction of peroxisomes (rats and mice only). Many of these effects were demonstrated to be reversible when animals were provided with a recovery period. Evidence of hepatotoxicity was not evident in studies in monkeys or humans.

In contrast with the rodent, the target organs in the monkey were the

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gastrointestinal tract and the reticuloendothelial system (Griffith and Long, 1980). While the liver does not appear to be a primary target organ in humans, exposure to C8 appears to modify the hepatic and immune response to xenobiotics (Gilliland and Mandel, 1996).

VI.2. Discussion of Differences in Species-Specific Sensitivities

The induction of peroxisome proliferation by xenobiotics is generally determined as an increase in the activities of certain peroxisome-specific enzymes, or as an increase in the numerical or volume density of peroxisomes in the affected organ. Peroxisome proliferation is associated with: increases in number and volume of peroxisomes; an increase in DNA synthesis and liver growth; and liver, Leydig cell, and pancreatic acinar cell tumors. The phenomenon of peroxisome proliferation is not uniform across all species. While rats and mice are particularly sensitive to this phenomenon, guinea pigs, cats, dogs and primates (including man), are predominantly non-responsive.

VI.3. Tumors Associated with C8 in the Rat

C8 has been demonstrated to be a peroxisome proliferator in the rat. C8 exposure in the rat was found to be associated with tumors in the liver, Leydig cell, and pancreatic acinar cell. Peroxisome proliferators, in general, were initially recognized to be associated with hepatocarcinogenesis in rats. However, more recently peroxisome proliferators have been associated with the induction of a triad of tumors in rats: liver, Leydig cell, and pancreatic acinar cell. Hyperplasia of these cell types is typically observed prior to, and along with, the occurrence of neoplasia. Several known peroxisome proliferators (clofibrate, HCFC-123, methylclofenapate, and Wyeth-14,643) are reported to induce this triad of tumors in rats. Hence, this tumor profile appears to be common phenomenon for at least a subset of compounds that are peroxisome proliferators.

VI.3.a. Significance of C8-Induced Rodent Tumor to Human Risk

VI.3.a.1. Liver Tumors

The abundance of data indicates that there is a hepatocarcinogenic hazard of peroxisome proliferators to responsive species (rats and mice) in chronic studies, whereas the carcinogenic hazard to non-responding species, such as humans, is clearly questionable. The epidemiology data, albeit limited, strongly support that the relevance of the hepatocarcinogenic effects of C8 and other peroxisome proliferators for human hazard assessment should be considered negligible.

VI.3.a.2. Leydig Cell Tumors

Leydig cell hyperplasia and adenomas are commonly observed in laboratory rats. The incidence of spontaneous Leydig cell adenomas in Crl:CD®BR rats ranges from approximately 0-12% by 2 years of age, and ranges from approximately 64 -100 % in F344 rats. In contrast, the rate in humans has been reported to be approximately 0.4 per



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million (0.00004%). Although a direct comparison is somewhat tenuous, the data suggest a substantial difference in the susceptibility of rodents and humans to Leydig cell tumorigenesis. This is supported by epidemiology data from compounds that clearly produce Leydig cell tumors in rodent studies but are commonly ingested by humans and are not associated with Leydig cell tumorigenesis in humans.

C8 and other peroxisome proliferators do not produce increases in peroxisomes in Leydig cells and are hypothesized to produce these tumors via a different mechanism than the liver tumors. The mechanism of tumorigenesis is not completely understood, and therefore relevance to humans can not be completely ruled out. However, it is known that non-genotoxic compounds (such as C8) produce Leydig cell tumors by altering the endocrine system. Therefore, a threshold for tumorigenesis is expected. If this is the case, use of a margin of safety approach is appropriate for the quantitative dose-response assessment. It is important to consider the slope of the dose-response at the low end of the observed range in determining an acceptable margin of safety.

VI.3.a.3. Pancreatic Acinar Cell Tumors

C8 and other peroxisome proliferators do not produce increases in peroxisomes in the pancreas and are hypothesized to produce these tumors via a different mechanism than the liver tumors. The mechanism of tumorigenesis is not understood, and therefore relevance to humans can not be completely ruled out. However there is a growing weight of evidence that the pancreatic acinar cell tumors are hormonally mediated, therefore they should be treated similarly to peroxisome-proliferator-induced Leydig cell tumors.

## VII. SUMMARY

C8 has moderate acute oral toxicity with $LD_{50}$'s ranging from 178 mg/kg in male guinea pigs to 680mg/kg in adult male rats. An aqueous paste of C8 produced mild to moderate dermal irritation in rabbits and clinical signs of toxicity were observed at doses as low as 1000 mg/kg. Instillation of solid C8 into the rabbit eye produced moderate corneal opacity, iritis, and conjunctivitis. These ocular effects gradually receded. C8 has high acute inhalation toxicity with a 4-hour ALC of 0.8 mg/L in the rat. Subchronic inhalation exposure to C8 produced reversible liver effects at concentrations as low as 8 $mg/m^3$ (measured as 7.6 $mg/m^3$). Oral and skin absorption subchronic studies confirmed the hepatotoxicity of C8 in the rat. In chronic feeding studies in rats, C8 produced an increased incidence of tumors in the liver, pancreas, and testis. C8 was found not to be a developmental toxic or mutagenic in several tests for mutagenicity.

The relevance to human health of tumors induced by peroxisome proliferators in rodents has been the focus of several investigators. Regarding the liver, there is a strong association and probable link between peroxisome-proliferator-induced liver growth and the subsequent development of rodent liver tumors. A combination of *in vivo* and *in vitro* studies as well as epidemiology data, has led several investigators to conclude that humans appear to be insensitive or unresponsive to peroxisome-proliferator-induced hepatic effects, and therefore these nongenotoxic agents pose little or no

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hepatocarcinogenic hazard to humans. Evidence is also accumulating that the initiating events, which lead to the development of Leydig cell and pancreatic acinar cell tumors are from changes in the liver. These hepatic changes appear to alter the hormonal control of the testis and pancreas. Although these relationships need to be confirmed, it is likely that these extrahepatic tumors pose little or no carcinogenic hazard to humans. Additionally, programs monitoring the health of C8-exposed workers and retrospective cohort studies of workers exposed to C8 provide no evidence of an association between C8 exposure and adverse human health effects.

Of primary concern in humans is the slow clearance of C8 from human blood, the opportunity for exposure in the work place, and the moderate-high acute toxicity, regardless of route of exposure.

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REFERENCES

DuPont Co., Haskell Laboratory Report Numbers(HL):

55-61, Acute Oral Test.1961, H. Sherman.

56-61, Subacute Feeding Study.1961, H. Sherman.

123-65, Effects of Fluorocarbon Disbursing Agents on the Livers of Rats and Dogs.1965, J. R. Barnes and H. Sherman.

124-65, Two-Week Feeding Study.1965, H. Sherman.

128-68, Acute Oral Test.1963, S. B. Fretz.

160-69, Acute Inhalation Dust Toxicity.1969, F. O. Tayfun and R. S. Waritz.

253-79, Inhalation Subacute.1979, G. T. Hall.

635-79, Eye Irritation Test in Rabbits.1979, D. F. Edwards.

636-79, Skin Irritation Test on Rabbits.1979, D. F. Edwards.

659-79, Skin Absorption $LD_{50}$ - Rabbit and Rat.1979, D. F. Edwards.

589-80, Rat Skin Absorption Subacute Study with FC-143.1980, L. S. Silber.

682-80, Skin Absorption $LD_{50}$-Female Rats.1980, J. E. Henry.

205-81, Subacute Inhalation Toxicity of Pentadecafluorooctanoic Acid, Ammonium Salt.1981, S. D. Nash and M. A. Britelli.

291-81, Oral $LD_{50}$ Test in Guinea Pigs.1981, J. A. Hall.

295-81, Oral $LD_{50}$ Test in Rats.1981, J. A. Hall.

329-81, Oral $LD_{50}$ Test in Mice.1981, J. A. Hall.

560-81, Mouse Feeding Study - 14 Day.1981, L. S. Ford.

565-81, Acute Oral Toxicity Comparison Test in Rats.1981, L. Hinckle.

567-81, Oral $LD_{50}$ Test in Rats.1981, J. E. Henry.

593-81, Ammonium Perfluorooctanoate Blood Levels in the Female Rat.1981, Kennedy, G. L.

MAB000036

600-81, Liver Weight Comparison in Castrated and Ovariectomized Rats vs. Normal Rats.1981, L. Hinckle.

537-82, Fourteen-Day Feeding Study in Male and Female Mice.1982, J. E. Henry.

788-82, Acute Oral Toxicity of FC-143 as a Function of Age.1983, L. Hinckle.

138-83, C-8 Liver Weights in Rats and Mice: Oral Administration to Males and Females.1983, C. N. Wyle.

DuPont MR 5686, Investigation of Hormonally-Mediated Mechanisms for Xenobiotic-Induced Leydig Cell Tumors in Male Rats. (1989).

3M Co. Reports:

Personal communication, J. Long to P.J. Gort, January 8, 1979.

3M Report M-601 (1981).

3M Report 0681TR0110, 1981.

3M Product Toxicity Sheet, May 24, 1996.

Biosearch, Inc. Report No. T1395, March 4, 1976.

Corning Hazleton, 17388-0-455, May 16, 1996.

Corning Hazleton, 17388-0-437, April 25, 1996.

Hazleton Laboratory America, Inc. 2-6-87 (Submitted to EPA, TSCA 8eCAP Submission, Fiche 536984).

Litton Bionetics; LBI Project 20838, February 1, 1978.
Riker Laboratory Drug Metabolism Report 1-20 (1980).

Riker Laboratories, Report 09790AB0485, March 15, 1981.

Riker Laboratory 0281CR0012, April 1981–May 1983.

University of Minnesota Environ. Path Lab, T2942, April 9, 1981.

Published References:

Biegel, L. B., R. C. M. Liu, M. E. Hurtt, and J. C. Cook (1995). Effects of ammonium perfluorooctanoate on Leydig cell function: *In vitro*, *in vivo*, and *ex vivo* studies. Toxicol. Appl. Pharmacol. 134: 18-25.

MAB000037

Cook, J. C., M. E. Hurtt, S. R. Frame, and L. B. Biegel (1994). Mechanisms of extrahepatic tumor induction by peroxisome proliferators in Crl:CD®BR (CD) rats. Toxicologist (abstract) **14**.

Cook, J. C., S. M. Murray, S. R. Frame, and M. E. Hurtt (1992). Induction of Leydig cell adenomas by ammonium perfluorooctanoate: A possible endocrine-related mechanism. Toxicol. Appl. Pharmacol. **113**: 209-217.

Fayerweather, W. E., Liver study of Washington Works employees exposed to C8: Results of blood biochemistry testing. DuPont, Epidemiology Division of Corporate Medical, January 15, 1981.

Gilliland, F. D. and J. S. Mandel (1993). Mortality among employees of a perfluorooctanoic acid production plant. J. Occup. Med. **35**: 950-954.

Gilliland, F. D. and J. S. Mandel (1996). Serum perfluorooctanoic acid and hepatic enzymes, lipoproteins, and cholesterol: A study of occupationally exposed men. American Journal of Industrial Medicine **29**: 560-568.

Griffith, F. D. and J. E. Long (1980). Animal toxicity studies with ammonium perfluorooctanoate. Am. Ind. Hyg. Assoc. J. **41**: 576-583.

Johnson, J. D., S. J. Gibson, and R. E. Ober (1984). Cholestyramine-enhanced fecal elimination of carbon-14 in rats after administration of ammonium [14C]perfluorooctanoate or potassium [ 14C]perfluorooctanesulfonate. Fundam. Appl. Toxicol. **4**: 972-976.

Kennedy, Jr., G.L. (1985). Dermal toxicity of ammonium perfluorooctanoate. Toxicol. Appl. Pharmacol. **81**: 348-355.

Kennedy, Jr., G.L., G. T. Hall, M. R. Brittelli, J. R. Barnes, and H. C. Chen (1986). Inhalation Toxicity of Ammonium Perfluorooctanoate. Fd. Chem. Toxic. **24**(12): 1325-1329.

Kennedy Jr., G. L. (1987). Increase in mouse liver weight following feeding of ammonium perfluorooctanoate and related fluorochemicals. Toxicol. Letters **39**: 295-300.

Liu, R. C. M., C. Hahn, and M. E. Hurtt (1996a). The direct effect of hepatic peroxisome proliferators on rat Leydig cell function *in vitro*. Fundam. Appl. Toxicol. **30**: 102-108.

Liu, R. C. M., M. E. Hurtt, J. C. Cook, and L. B. Biegel (1996b). Effect of the peroxisome proliferator, ammonium perfluorooctanoate (C8), on hepatic aromatase activity in adult male Crl:CDBR (CD) rats. Fundam. Appl. Toxicol. **30**: 220-228.



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Obourn, J. D., S. R. Frame, R. H. Bell Jr., D. S. Longnecker, G. S. Elliott, and J. C. Cook (1997). Mechanisms for the pancreatic oncogenic effects of the peroxisome proliferator Wyeth-14, 643. Toxicology and Applied Pharmacology **145**: 425-436.

Pastoor, T. P., K. P. Lee, M. A. Perri, and P. J. Gillies (1987). Biochemical and morphological studies of ammonium perfluorooctanoate-induced hepatomegaly and peroxisome proliferation. Experimental and Molecular Pathology **47**: 98-109.

Perkins, R. G. (1992). Investigation of ammonium perfluorooctanoate effect on hormone levels and peroxisomal proliferation in the rat. The Toxicologist **12**(1): 52.

Staples, R. E., B. A. Burgess, and W. D. Kerns (1984). The embryo-fetal toxicity and teratogenic potential of ammonium perfluorooctanoate (APFO) in the rat. Fundamental and Applied Toxicology 4: 429-440.

Ubel, F. A., S. D. Sorenson, and D. E. Roach (1980). Health status of plant workers exposed to fluorochemicals-a preliminary report. Am. Ind. Hyg. Assoc. J. **41**(8): 584-589.

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**Proposal to Conduct a**
**General Human Health and Environmental Effects**
**Risk Analysis on C-8**

Purpose: The purpose of this project is to evaluate the risks to human health and the environment from exposure to C-8 during manufacture, transport, product use, and disposal of C-8. The analysis will be conducted in a fashion that will provide semi-quantitative estimates of risks so that exposures yielding the highest risks can be identified and recommendations on reducing these risks can be developed. Risks from manufacture, transport and product use will developed in a way that will facilitate future comparisons of risks estimated for potential C-8 alternatives. The project will be conducted in three parts. The first two will be conducted in parallel, in which human health and ecological risks will be characterized. The final part will develop conclusions on exposures that contribute the highest risks so that recommendations for risk management strategies and alternatives can be developed. The project is estimated to take 12 months to complete from the time of initiation. The Exposure analyses listed below will require collaboration with appropriate plant personnel. *(The dates presented assume a Feb. 1, 1997 SBU approval date.)*

Scope:

| | | Time Line | Est. Cost($) |
|---|---|---|---|
| I. | Human Health Risk | | |
| A. | Hazard Identification | 4/18/97 | 8000 |

Hazards to human health will be reviewed and summarized in this section. The critical toxicity endpoints of relevance to human health risk will be identified and potential dosimeters to be used for interspecies extrapolation of risk will be discussed. The Haskell toxicity summary will be updated as part of this task.

B. Dose-Response Analysis — 9/30/97 — 43,200

The dose-response characteristics of C-8 will be evaluated. This may include conducting benchmark dose analyses to identify no-observed adverse effect levels where necessary. Appropriate dosimeters for interspecies extrapolation will also be developed based on the likely mode of action. The pharmacokinetics of C-8 will also be reviewed. If possible, rudimentary physiologically-based pharmacokinetics approaches will be developed to facilitate interspecies extrapolation of risk. Risks vs. dose relationships will be developed in this phase

C. Exposure Analysis — 9/30/97 — 16,000

Reasonable exposure scenarios for C-8 will be developed. This are likely to include airborne, drinking water, dermal, and other oral ingestion pathways. Intake rates and durations of exposure will be developed. Haskell will work with an assigned person(s) from the plant site to help characterize these exposure pathways for manufacturing, transport, product use, and waste disposal operations. The business will provide Haskell with data on concentrations of C-8 in the affected media (air, water, soil). These data will be tabulated. Monte Carlo techniques may be used to calculate expected upper confidence limits for these exposures, depending the availability of data. The cost associated with this task include only Haskell personnel time.

D. Risk Characterization — 12/15/97 — 16,000

Risks will be summarized according to the major routes of exposure (air, water, dermal, other oral) for each C-8 application (manufacture, transport, product use, disposal). The risks will be characterized by comparing the likely exposure concentrations to the dose-response relationship. This method is generally referred

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to as a Margin of Exposure. The characterization will provide the risk manager with information that will help identify the operations and exposure pathways that present the highest risk. The characterizations will also enable future comparisons to be made of potential risks posed by C-8 alternatives.

II Ecological Effects
- A. Hazard Identification
- B. Dose-Response Analysis
- C. Exposure Analysis
- D. Risk Characterization

III. Recommendations on Risk Management Strategies and Alternatives

12/15/97 4800

This section will evaluate collectively the risks identified to human health and ecological receptors. Based on these analyses, recommendations will be made as to which operations could be targeted to reduce the largest risks for the least cost. This will be a very subjective exercise (narrative) and will require some input from the plant people.

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# I N T E R O F F I C E  M E M O R A N D U M

Date: 09-Jun-1997 09:32am EDT
From: DANIEL A. WEBER
WEBERDA
Dept: ENGINEERING POLYMERS
Tel No: 8-863-4415

TO: 7 addressees

CC: 3 addressees

Subject: EPA REGION III ECOSYSTEM ASSESSMENT OF DRY RUN CREEK

EID017890

FYI,

THIS MORNING, WOODY IRELAND AND I MET WITH SARA CASPAR (REGION III EPA, PHILADELPHIA) FOR ABOUT A HALF HOUR TO REVIEW HER PLANS TO OBTAIN SAMPLES NEEDED TO CONDUCT AN ECOLOGICAL RISK ASSESSMENT OF THE DRY RUN CREEK. THE PURPOSE OF THIS ASSESSMENT IS TO LOOK FOR POTENTIAL CONTAMINATION IN THE DRY RUN CREEK DRAINAGE AND IDENTIFY POTENTIAL TOXINS WHICH MAY BE RESPONSIBLE FOR THE OBSERVED DECLINE AND PHYSIOLOGICAL ABNORMALITIES IN CATTLE WHICH USE THE DRY RUN CREEK AREA AS A SOURCE OF WATER AND FOOD.

SARA AND A TEAM OF 4 TO 5 PEOPLE WILL BE IN THE DRY RUN CREEK AREA FROM TODAY THROUGH FRIDAY TO COLLECT SAMPLES FOR THE RISK ASSESSMENT. SOME SAMPLES WILL BE TAKEN ON DUPONT PROPERTY AND SARA MENTIONED THAT WE WOULD RECEIVE A COPY OF THE DATA RESULTS. (AS AN ASIDE, SHE ALSO PLANS TO SEND US SOME INFORMATION ON RESULTS OF COW TISSUE ANALYSES PERFORMED AT MICHIGAN STATE.)

SARA PROVIDED ME A COPY OF A DRAFT WORK PLAN FOR COLLECTING SOIL, SEDIMENT, WATER AND BIOTA (ANIMAL SPECIMENS) FROM THE DRY RUN CREEK. A COPY OF THIS WORK PLAN HAS BEEN SENT TO THE ADDRESSEES AND CONTAINS ADDITIONAL INFORMATION NOT SUMMARIZED IN THIS NOTE.

BASED ON OUR DISCUSSIONS THIS MORNING WITH SARA AND A REVIEW OF THE DRAFT WORK PLAN, LISTED BELOW ARE THE TYPES OF SAMPLING WHICH WILL BE PERFORMED. VARIOUS TOXICITY ASSESSMENTS AND ANALYSES WILL BE MADE ON THESE SAMPLES. NOT ALL SAMPLES WILL BE TAKEN FROM DUPONT PROPERTY AND EXACT SAMPLING LOCATIONS HAVE NOT YET BEEN DETERMINED.

- OBTAIN FIVE SEDIMENT AND SURFACE WATER SAMPLES FOR TOXICITY TESTING.
    - FATHEAD MINNOWS, EARTHWORMS AND "HYALELLA" TO BE USED FOR TOXICITY TESTING.
- PERFORM BENTHIC SURVEYS WHERE SAMPLES ARE TAKEN.
- PERFORM ELECTROSHOCK FISH SAMPLING FOR TISSUE ANALYSIS AT SURFACE WATER SAMPLING LOCATIONS.
- OBTAIN FROG EGGS FOR ANALYSIS OR LIVE ADULT FROGS FOR BREEDING.
- COLLECT GRASS SAMPLES FOR ANALYSIS.
- COLLECT SMALL ANIMALS (DEER MOUSE AND MEADOW VOLE IMPLIED BASED ON WORK PLAN) FOR TISSUE ANALYSIS. EXAMINE JAW BONES AND TEETH FOR DISCOLORATION.
- USE DATA OBTAINED ABOVE AND APPLY TO A FOOD CHAIN MODEL TO ASSESS POTENTIAL HEALTH EFFECTS ON A ROBIN, RED-TAILED HAWK, AND RED FOX. SARA INDICATED THERE WOULD BE AN ATTEMPT TO CATCH THESE ANIMALS BUT I DIDN'T SEE THIS MENTIONED IN THE WORK PLAN.

THE WORK PLAN CONCLUDES WITH A LIST OF 17 TESTABLE HYPOTHESES, OR QUESTIONS, WHICH THE STUDY IS DESIGNED TO HELP ANSWER.

DAN

000389

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July 28, 1997

DO NOT REMOVE

File 3:86

Mr. Daniel Weber
Senior Engineer
E.I. DuPont DeNemours and Company
Washington Works, Building 1
P.O. Box 1217
Parkersburg, West Virginia 26102

Dear Mr. Weber:

RE: Response to Affiliated Construction Trades Foundation comments on the Dupont, Dry Run Landfill Permit Application, For Use at Public Hearing.

I was asked to offer my thoughts on the four comments made by the Affiliated Construction Trades Foundation regarding the Dry Run Landfill draft permit. These comments are offered without researching the specific federal and/or state statute or regulation, and are intended to be general in nature. My first thought is that DuPont should not attempt to respond to these questions at the hearing, but let the agency answer them. These are accusations that challenge the agency's integrity, which will be addressed in the agency's formal response to comments.

COMMENT 1

The draft allows for new landfill on top of existing. The existing landfill must be closed in accordance with state regulations, and state should not allow a landfill on the area, which is already impacted as is indicated by the elevated fluoride levels.

The facility has not been closed out, a new cell is being added. The state has allowed "piggy backing" of a new cell on or against an existing cell in other cases. There is no prohibition against it. The director is given a great deal of flexibility to control industrial wastes. The code and the regulations allow best engineering judgements to be made on facilities such as this. The existing area has been covered and graded and has an excellent stand of vegetation. It is not an open hole that encourages the infiltration of rainwater, which results in leachate formation. As the new cell expands the leachate collection system will serve the same purpose as a liner system. The Division of Environmental Protection (DEP) has approved the use of this system for future groundwater protection, so it should serve as an adequate cap of the old facility. The only negative issue here is the 10 years it will take to cover the old facility with the new cell and associated impervious layer.

If I understand the facts correctly, the fluoride value in the groundwater was a one time occurrence. The regulations specify what a facility is to do when monitoring wells show elevated levels. I would assume that the agency and the company are following the procedures as required. If elevated fluoride levels are shown to be a problem, the appropriate action will be taken.

Act is encouraging DuPont to move to a "green fields" site for the new cell. The agency should encourage the use of those existing industrial sites to the degree possible. The waste should logically be in the same place to affect the best control possible.

**COMMENT 2**

Additional treatment of leachate to meet water quality limits.

The available information on quality of the discharge into Dry Run is based upon the quantity and quality of the leachate, as well as the efficiency of the existing treatment system. Those performance levels cannot be used to estimate the effectiveness of the new system. The new system, with its larger area and baffled aeration area, is expected to have higher removal efficiencies. In the event that the limits cannot be met, DuPont can either truck the leachate to their plant on the Ohio River (I assume they have a permit modification in place to allow this) or add additional treatment to the existing site. There is no proof that the new system will not meet the limits. The bottom line is DuPont will have to meet them or apply to Environmental Quality Board (EQB) for a variance for Dry Run. It is a compliance issue if they fail to meet the limits.

**COMMENT 3**

Limits for fecal coliform, boron and formaldehyde.

The director must first determine that these materials are, or may be, discharged at a level to contribute to an excursion of the water quality standards. The director or his representatives have reviewed the information and determined that they do not need to be limited at this time. The agency should evaluate the effectiveness of the new system and, if additional limits are needed, modify the permit accordingly. There are no water quality standards promulgated by the EQB for boron or formaldehyde. EPA suggested ambient goals are not regulations until adopted by the EQB. The inclusion of these as limits are discretionary on the part of the agency. They can be added if they are felt to be a problem.

**COMMENT 4**

WET testing on a continuing basis.

The agency is requiring the initial whole effluent toxicity (WET) testing on the facility. It is standard practice to discontinue that requirement, should toxicity be shown not to be a problem. It is a waste of resources to require such testing when it does not appear to be a problem. The agency can at any time take WET tests on the effluent. The agency can require DuPont to do WET tests on the effluent. Should the agency decide that toxicity is an issue of concern, the permit can be modified to require it, but to require it where there is no history of toxicity problems is useless.

I am available to discuss these issues further, if you feel that would be helpful.

Sincerely,

Laidley Eli McCoy

LEM/srs

cc: Mr. Ronald R. Potesta
Mr. Mark Kiser

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CONFIDENTIAL

Dry Run Landfill Task Team
Anticipated Questions -- FINAL (7/29/97)

A. General Information / Physical Features / Operation

1. Why wasn't a liner installed when the landfill was constructed, and why is the WVDEP making you install one now?

   (Quoted material is from the draft permit.)

   The landfill permit predated the Solid Waste Management Regulations of 1990 and "the design requirements of the SWMR are now being incorporated into the design of the Dry Run landfill" to improve groundwater protection.

2. How long will you be operating the landfill?

   For the foreseeable future.

3. When the current cell is full, will you expand the landfill to another area, open another cell?

   Decisions about expansion in the future will depend upon economical considerations and the regulations/requirements in effect at the time.

4. Why were those new tanks installed at the landfill?

   To collect leachate for transport to the Site for disposal. This practice is required by the State in the interim until modifications required by the new permit are completed.

5. What about all the windblown trash that comes off Boso's trucks as he's hauling the waste down to the landfill?

   All trucks are covered, with the exception of truck loads of dirt from excavations during current construction activity. Blowing material is unusual -- we'd like to be contacted so that we can follow-up. Please call 863-2000.

6. What about illegal dumpings at night? Why isn't the landfill more secure?

   Protected by locked gate. No scheduled activity after dark. Operated only during daylight hours. If you witness any activity after hours, please contact us at 863-2000.

7. If you do such a good job, how come you had to close down your other landfills?

   Local landfill -- reached capacity.
   Letart -- The amount of plastic waste being landfilled has decreased over the years, and landfill operations are being consolidated for economic reasons.



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General Information, continued

8. Are you going to start dumping into Dry Run some of the bad actors that used to go elsewhere?

   Only nonhazardous solid waste materials are landfilled at Dry Run.

9. Why were materials taken to Letart in the first place?

   Teflon* wastes were segregated from other waste materials because of the potential at the time for a landfill fire resulting from the disposal of hot polymer waste. The involvement of Teflon* waste in a landfill fire had the potential to release dangerous fumes. Since hot polymer waste is no longer landfilled, there is no longer a need to segregate the Teflon* wastes.

10. What determines what is landfilled and what is incinerated?

    Two factors primarily influence the determination: permitting and handling. First, with our waste minimization effort, we try to reuse or recycle whatever we can. Next, we try to process as much nonhazardous waste material as possible that is acceptable and permitted for burning in our alternate fuel boiler (AFB) so that we can recover heat value for plant steam needs. Finally, nonhazardous waste materials which can't be processed through the AFB are landfilled.

11. Leachate

    a. What is leachate?

       Any liquid that might have contacted disposed waste.

    b. Why is the State requiring that you transport leachate to Washington Works for disposal? What is their concern?

       This practice is required by the State in the interim until modifications required by the new permit are completed.

       The concern is about such conditions as iron content, total suspended solids, and biochemical oxygen demand in unlined landfill operations. The State is interested in protecting the groundwater to the greatest extent possible, and we are complying.

Leachate, continued

c. How long will you be hauling leachate out?

We don't know -- at least until the new permit construction is complete.

d. Do you have DOH or DOT approval to haul leachate?

The operation for hauling the leachate meets all State requirements for hauling nonhazardous materials. Such requirements include licensed drivers, weight limit compliance, etc.

e. I saw it leak out on the road; did you report the leak?

No record of any leak; vehicles inspected for integrity. Material hauled is nonhazardous. However, if concerned, let us know. Call 863-2000.

f. Is your contractor licensed to transport the leachate?

Meets all requirements within his existing PSC license. Also, contract, not commercial hauler.

B. Black Water Issues

1. Why did the water turn black and smelly?

This was a temporary situation (about five months) that has been resolved for approximately two years. The color and odor were caused by the presence of iron sulfide and hydrogen sulfide. Aerating the ponds -- that is, bubbling air through the water -- eliminated the color and odor, and they have not reappeared since aeration began in June 1995.

2. What effect did it have on the ecology of the stream?

Frequent stream surveillance has provided no evidence of a negative impact on the wildlife or vegetation of the area.

3. If you've messed up in the past with that black water, how can we be sure you won't mess up again in some other way?

We are confident that our work with the WVDEP to upgrade the landfill to comply with new regulations, in combination with increased routine surveillance (monitoring) required by the State, will enhance the overall operation of the facility.

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C. Contents / Materials Landfilled

1. What carcinogens, hazardous wastes, toxic wastes, etc., are being placed in the "dump?"

   The landfill is a nonhazardous solid waste landfill. The only material requiring special handling is, as we described earlier, asbestos, which is considered a possible carcinogen to those directly exposed to it. However, asbestos is a hazard only when it is in the air and can be inhaled. When handled properly and buried or wet, it presents no hazard.

2. What else is in that landfill that is not on the list of 19 parameters? What aren't you reporting?

   We dispose of only those materials specified in the permit; we must report their amounts annually; we are subject to unannounced inspections by the WVDEP to monitor landfill operations.

3. Is there fertilizer or other chemicals in the landfill water going into Dry Run?

   Consistent with State regulations and recommendations, we do add fertilizer from time to time to establish vegetation growth. We use no pesticides or herbicides.

D. Toxicity / Other Harmful Effects

1. Is the antifoam additive toxic?

   No. EC-210 (Antifoam) is used in accordance with manufacturer specifications. (MSDS sheet is on file; mineral oil.)

2. Is the algae poisonous to cattle if they drink water with algae in it?

   The algae are not harmful.

   (Report faxed from Haskell Lab is on file regarding fresh water blue-green algae bloom that can cause lethal poisoning; most problems reported have occurred in northern plains states. Furthermore, it is likely that this question would be asked from this perspective: "If leachate from your landfill contacts algae in the stream, would the polluted algae in the stream be toxic to cattle?" Suggested answer: To-date, neither the DEP nor the EPA has established a connection between the landfill and poor animal health.)

D. Toxicity / Other Harmful Effects, continued

3. Is the groundwater near the landfill contaminated? If so, will contaminated groundwater get into my drinking water?

(Quoted material is from the draft permit.)

Analysis of quarterly groundwater data "has revealed that the Dry Run site has not caused a statistically significant impact upon groundwater relative to background groundwater concentrations."

4. Is it possible that some chemical that you don't know about is coming out of your landfill and causing harm?

It is unlikely, since the permit requires quarterly screening for a large number of possibilities in the landfill leachate. In addition, we do several nonspecific chemical and biological tests, monitoring such indicator parameters as BOD (biochemical oxygen demand), COD (chemical oxygen demand), and TOC (total organic carbons).

With regard to causing harm, we have no evidence that the landfill operation is causing harm. We have observed no impact on vegetation or wildlife.

We conducted the State's standard acute toxicity testing in 1993. The results were within established limits.

5. Can the nonhazardous materials that you put in your landfill degrade to other things that could be more hazardous?

Materials placed in the anaerobic environment of a landfill (that is, buried so that oxygen cannot penetrate) have been observed to degrade extremely slowly. Excavations at municipal landfills have uncovered such items as newspapers and various foodstuffs that have survived intact for decades. The materials that we put into the Dry Run Landfill are at least as durable. If any degradation products formed that were harmful, they would appear so slowly that their concentrations would remain too low to be detrimental.

6. Are there harmful bacteria in the water discharge from the landfill? If so, where does it come from -- the filtercake?

We have no reason to believe that there is a problem. The new permit requires monthly monitoring for fecal coliform, so we'll be checking for that as well as other parameters.

D. Toxicity / Other Harmful Effects, continued

7. Why don't you tell everyone about the C-8 coming out of your landfill and why you're so worried about it?

C-8, or ammonium perfluorooctanoate, is a surfactant used in our Teflon* area. Although C-8 is not regulated by any government agency, DuPont controls it voluntarily because of our high internal standards. Based on our 40 years of experience with handling it at the Plant, we are confident that the levels detected at the landfill are not harmful. No ill effect in our employees has been observed to-date.

Additional information for our use that may be too detailed or too alarming for the general public:

If C-8 were to have a negative health impact, the target organ would be the liver. We have collected epidemiologic data on our employees over the life of the plant, and the incidence of liver cancer is no higher than expected. C-8 is a weak animal carcinogen. We have reported the presence of C-8 in the leachate to the WVDEP.

E. Filtercake Issues

1. What exactly is filtercake?

Filtercake is a concentrated form of sludge that is removed from the site waste water treatment plant. It is composed of waste biosolids, microorganisms from the waste water treatment plant, fly ash, and water. It is different from sludge in that excess water is removed and fly ash is added.

The components of filtercake are similar to those produced by municipal waste water treatment plants and commonly either disposed of in municipal landfills or applied as fertilizer.

2. Why did you stop disposing of filtercake in the landfill and start taking it to the Northwestern Landfill?

This practice is required by the State in the interim until modifications required by the new permit are completed.

3. How much filtercake have you put in the landfill?

tons: 1994--568.57 1995--3,888.27 1996--3,952.81

4. How long did you dispose of filtercake in the landfill?

From late August 1994 to mid-November 1996

DRAFT REPORT

# DRY RUN CREEK
# WASHINGTON, WOOD COUNTY, WEST VIRGINIA

NOVEMBER 1997


PREPARED BY:

Mark D. Sprenger, Ph.D.
Environmental Response Team

AND

Michael T. Horne, Ph.D.
U.S. Fish & Wildlife Service/Environmental Response Team

IN CONJUNCTION WITH:

Mark Huston
REAC/ERT

Environmental Response Team Center
Office of Emergency & Remedial Response



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USFW 0579

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# SECTION I. TECHNICAL APPROACH, SUMMARY OF FIELD EFFORT RESULTS, AND PRELIMINARY RISK SCREEN

## 1.0 INTRODUCTION

### 1.1 Objective

The objective of this project was to provide technical support to the U.S. Environmental Protection Agency Region III Removal Program in conducting an evaluation of ecological risks from alleged contamination of soil, sediment, and water at a working beef production farm located down gradient of a landfill. The effort resulted in the collection of soil, sediment, surface water, and biota samples for contaminant analyses and soil, sediment, and surface water for laboratory toxicity testing. The primary goals of the project were to: 1) identify contaminants present, 2) determine the extent of contamination, and 3) produce an ecological risk assessment based on the collected data.

### 1.2 Site Background

The site is a working beef production farm located in Washington, Wood County, WV. The owner of the farm has filed numerous complaints with the West Virginia Department of Natural Resources and the U. S. EPA alleging that contaminants are being discharged from an industrial landfill owned by the DuPont corporation, into Dry Run. Dry Run flows through the farmer's property and is a primary source of water for his cattle. The farmer maintains that numerous deaths, blindness, and other unusual illnesses observed in his herd are directly attributable to the contaminants that are discharged into Dry Run from the DuPont landfill. It has also been reported that fish and wildlife kills have also occurred in the area, which may be associated with the abnormalities observed in the cattle.

## 2.0 METHODOLOGY

The approach used in this document followed current U.S. EPA guidance for designing and conducting ecological risk assessments (U.S. EPA 1997). Based on the problem formulation phase of the risk assessment design, the following field study was conducted to provide data needed to complete the assessment. A screening-level ERA was conducted after the field investigation, as little data on site contamination was available prior to the effort. Numerous fish and wildlife kills, in addition to problems in the cattle, had been reported prior to this activation.

### 2.1 Soil Sampling

Surface soil samples were collected at 4 sample areas along Dry Run and in one reference sample area (Figure 1). Sample areas were selected based on distance from the landfill outfall in an attempt to identify a contaminant concentration gradient. Sampling was concentrated in the meadows along the stream bed. Three replicate samples were taken in each sampling area. Replicate sampling locations were determined by gridding the sampling area and randomly choosing three grid nodes for sampling through the use of a random numbers table. Sampling grid nodes were determined by using a random numbers table.

Surface soil samples were collected using a decontaminated stainless steel trowel or spoon from the top 6 inches of the soil according to ERTC/REAC Standard Operating Procedure (SOP) #2012, *Soil Sampling*. All soil samples were analyzed for total organic carbon (TOC); grain size; target analyte



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list (TAL) metals; TCL pesticides/PCBs; TCL Base, Neutral, and Acid Extractable (BNAs) compounds; TCL volatile organic compounds (VOCs), total fluoride, and organofluoride compounds. Additional soil was collected from the sample node closest to the stream bed for use in an earthworm toxicity test. A vegetation sample was also taken at each of the soil sampling nodes.

## 2.2 Sediment Sampling

Sediment samples were collected at 5 sample areas on site in Dry Run, one reference sample area, and one area in Lee Creek. Sample areas were selected based on distance from the landfill outfall in an attempt to identify a contaminant concentration gradient. Sampling was concentrated in the depositional areas along the stream bed.

All sediment sampling was conducted according to ERTC/REAC SOP #2016, *Sediment Sampling*. At each sample station, sediment was collected from the top 6 inches using a decontaminated trowel. The sample was composited into a decontaminated 5-gallon stainless steel bucket, homogenized, and divided into the appropriate sample containers for chemical analyses. Additional sediment was collected in the reference area, Tributary A, Tributary B, Area II, and Area IV, for use in a *Hyalella azteca* whole sediment bioassay.

## 2.3 Surface Water Sampling

Surface water samples were collected at locations which corresponded to each of the seven sediment sample stations. Surface water samples were collected directly into two 1-liter polypropylene bottles for metals analyses and into 1-liter glass bottles for organic (i.e., BNAs, Pesticide/PCBs, VOCs) analyses as per ERTC/REAC SOP #2013, *Surface Water Sampling*. Water samples were collected prior to collecting sediment samples and upstream of any stream disturbances caused by the sampler. One sample at each location was filtered through a 0.45 micron ($\mu$m) filter in the field prior to TAL metals analysis; all the remaining TAL metals samples and all the organic samples were analyzed unfiltered. All samples analyzed for metals were preserved by adding 40 percent nitric acid until a pH of less than 2 in the sample was obtained. The filtered sample submitted for TAL metal analysis was preserved after the sample was filtered. All surface water samples were submitted for TAL metals, TCL BNAs, TCL Pesticide/PCBs, TCL VOCs, chloride, fluoride, bromide, nitrate, sulfate, and phosphate analyses. Additional sample was taken in the reference area, Tributary A, Tributary B, Area II, and Area IV, for use in a *Pimephales promelas* aquaeous phase toxicity test.

Water quality parameters were measured using a Horiba™ water quality meter. The meter was used to measure temperature in degrees Celsius (°C), pH, dissolved oxygen [milligrams per liter (mg/L)], conductivity [millimhos per centimeter (mmhos/cm)], oxidation reduction potential [volts (V)]. The meter was calibrated prior to and after data collection. In-situ water quality data was transcribed from the digital display of the Horiba™ into a field logbook at the time of collection. The Horiba™ was used in accordance the manufacturer's operating manual.

## 2.4 Drinking Water Well Sampling

Water was sampled from a drinking water well on the Tennant farm. Parameters were analyzed as outlined above. Samples to be analyzed were taken from a tap that was located directly on the pump head after the well had been purged for a period of approximately five minutes.



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## 2.5 Biological Sampling

### 2.5.1 Small Mammal Study

Small mammals were collected from the site to determine body burden levels of TAL metals and total fluoride and to evaluate histopathological effects of exposure to site contaminants. Tissue burdens of small mammals trapped on site were compared to animals collected from the reference area. All field trapping activities were conducted in accordance with ERTC/REAC Draft Standard Operating Procedure SOP #2029, *Small Mammal Sampling and Processing*.

Four trapping areas were established on site in areas corresponding to the soil sampling locations. A fifth grid was established on a reference area located just to the north of Dry Run in similar meadow habitat as that observed along the stream corridor (Figure 1). The reference area was chosen because the habitat present was similar to that in the meadows near Dry Run, and because it was outside the area that could be directly influenced by surface water from Dry Run. The length of the trapping period and the trapping effort varied among each of the trap areas and was based on the length of time and effort required to capture a sufficient number of mammals for statistical evaluation. Sampling was performed using Museum Special snap traps set in grids. All traps were spaced 10 feet apart and baited with a rolled oats and peanut butter mixture. The traps were checked twice daily, once in the morning and once in the evening. During trap checks, traps were rebaited and reset as necessary. Recovered animals were labeled with the trap area, trap number, species, and date of capture while in the field and then were transferred in coolers to the staging area for processing.

For each animal, prior to performing the necropsy, data from the specimen label was transferred to a small mammal data sheet (Appendix A). Body metrics including total body weight, body length, tail length, ear length, liver weight, and kidney weight were measured and recorded on the data sheet. During the necropsy any abnormalities were noted and the contents of the gastrointestinal tract were removed from each specimen. Sections of the liver and kidney (approximately 0.5 g each) were removed for histopathological analyses. The sections were placed in a labeled 40-mL glass vial and preserved with 10 percent neutral buffered formalin. Preserved liver and kidney sections were submitted to Animal Reference Pathology (ARP) for histopathological evaluation. The remaining tissue was submitted for homogenization and TAL metal, total fluoride, percent moisture, and percent lipid analysis.

### 2.5.2 Vegetation Sampling

Vegetation was collected by hand for residue analysis per ERTC/REAC SOP #2038 *Vegetation Assessment Field Protocol*. The most abundant grass taxa observed at all sampling locations was targeted for residue analysis. Grass samples were taken in each area at the same grid nodes as the soil samples were taken. The above ground portion of plants from the immediate vicinity of the soil sampling node were collected by cutting the stems at the soil surface with a decontaminated knife. All samples were analyzed for TAL metals, total fluoride, percent moisture, and percent lipids.

### 2.5.3 Aquatic Macroinvertebrate Sampling



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The infaunal macroinvertebrate community was sampled per Draft ERTC/REAC SOP #2032 *Benthic Macroinvertebrate Sampling* and U.S. EPA (1983, 1989, and 1990). Macroinvertebrate samples were collected for evaluation of community structure. In this investigation, macroinvertebrates were defined as organisms that impinged on a 0.5 millimeter (mm) sieve. A total of three replicates were collected from each of five sediment sampling locations (Figure 1).

A long-handled, D-frame kick net, measuring approximately 45 centimeters wide and 20 centimeters tall, with 0.5 mm mesh was used. The net was used to disturb submerged vegetation and debris and collect dislodged invertebrates. Each replicate collection was performed over a uniform area at each sampling location. Benthic invertebrate samples were transferred to 500 ml polyethylene jars and preserved with a 70 percent 2-propanol solution.

In the laboratory, the sample was rinsed in clean water and placed in a white 12 x 18-inch polyethylene pan with just enough water added to allow complete dispersion of the material within the pan. Large debris, stones, and other extraneous materials were removed from the tray and inspected for attached or clinging organisms. All organisms picked from the pan were identified to the lowest positively identified taxonomic level, enumerated, and recorded on a laboratory bench sheet. The size and life history stage of the organisms and state of taxonomic knowledge of the group determined the level of identification. The organisms were identified using appropriate taxonomic references and a representative subsample were identified by a second individual to meet the Quality Assurance/Quality Control (QA/QC) requirements of the taxonomic analysis.

### 2.5.4 Fish Collection

Fish were collected from Dry Run to determine body burden levels of TAL metals and total fluoride. A Coffelt™ battery powered backpack electroshocker was used and operated as per the manufacturer's instructions. The sampling team consisted of one individual operating the electroshocker and one individual collecting stunned fish with a dip net. Stunned fish were placed in a 5-gallon bucket filled with site water. Following collection, fish were identified to the lowest taxonomic level possible in the field and live specimens were released. Voucher and dead specimens were preserved with a dilute formaldehyde solution and returned to the ERT/REAC biological laboratory for confirmation of field taxonomic analyses. Fish tissue was homogenized and submitted to the laboratory for TAL metal, total fluoride, % lipid and % moisture analysis.

## 2.6 Toxicity Testing

### 2.6.1 *Eisenia foetida* (Earthworm) Toxicity Tests

Five soil samples were taken for evaluation in an earthworm toxicity test. Four of the samples were taken in the meadow sampling areas along Dry Run and one in the reference meadow area as outlined above. The test was run for a period of 28 days, at which time mortality and growth in each of the test soils was enumerated. Earthworm tissue resulting from each of the treatments was submitted for TAL metals, total fluoride, % lipid, and % moisture analysis. Figure 1 details the earthworm toxicity test soil sampling locations.

### 2.6.2 *Hyalella azteca* (Amphipod) Toxicity Tests

Five sediment samples were taken for evaluation in an amphipod toxicity test. Four of the samples were taken in Dry Run and one in a reference area stream. The test was run for a period of 10 days, at which time mortality and growth in each of the test sediments was enumerated. Figure 1 details the amphipod toxicity test sediment sampling locations.

2.6.3 *Pimephales promelas* (Minnow) Toxicity Tests

Five surface water samples were taken for evaluation in a fathead minnow toxicity test. Four of the samples were taken in Dry Run and one in a reference area stream. The test was run for a period of 7 days, at which time mortality and growth in each of the test waters was enumerated. Figure 1 details the fathead minnow toxicity test water sampling locations.

2.7 Sampling Equipment Decontamination

The following sampling equipment decontamination procedure was employed prior to and subsequent to sampling in the following numerical sequence:

1. physical removal
2. nonphosphate detergent wash
3. potable water rinse
4. 10 percent nitric acid rinse
5. distilled water rinse
6. solvent rinse [acetone]
7. air dry

2.8 Standard Operating Procedures

2.8.1 Documentation

Documentation was conducted in accordance with the following SOPs:

-ERTC/REAC SOP #2002, *Sample Documentation*
-ERTC/REAC SOP #4001, *Logbook Documentation*
-ERTC/REAC SOP #4005, *Chain of Custody Procedures*

2.8.2 Sample Packaging, Shipment, Storage, Preservation, and Handling

Sample packaging, shipment, storage, preservation and handling were conducted in accordance with the following SOPs:

-ERTC/REAC SOP #2003, *Sample Storage, Preservation and Handling*
-ERTC/REAC SOP #2004, *Sample Packaging and Shipment*

2.8.3 Field Sampling and Analytical Techniques

Field sampling activities and field analytics were conducted in accordance with the following SOPs:

-ERTC/REAC SOP #2001, *General Field Sampling Guidelines*



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-ERTC/REAC SOP #2005, *Quality Assurance/Quality Control Samples*
-ERTC/REAC SOP #2006, *Sampling Equipment Decontamination*
-ERTC/REAC SOP #2012, *Soil Sampling*
-ERTC/REAC SOP #2013, *Surface Water Sampling*
-ERTC/REAC SOP #2016, *Sediment Sampling*
-REAC SOP #2029, *Small Mammal Trapping and Processing*
-REAC SOP #2032, *Benthic Sampling*

2.8.4 Health and Safety

Health and Safety was conducted in accordance with the following SOPs:

-ERTC/REAC SOP #3001, *REAC Health and Safety Program Policy and Implementation*
-ERTC/REAC SOP #3012, *REAC Health and Safety Guidelines at Hazardous Waste Sites*
-ERTC/REAC SOP #3020, *Inclement Weather, Heat Stress and Cold Stress*

# 3.0 RESULTS

## 3.1 Water, Soil, and Sediment Analysis

### 3.1.1 BNAs

Surface Water

Analysis of the surface water samples from Dry Run, the reference stream, and Lee Creek produced only one detection on the standard BNA scan. A sample taken in the Upper Tributary B location contained an estimated concentration of 2 ug/L of Bis(2-Ethylhexyl)phthalate. In addition, numerous Tentatively Identified Compounds (TICs) including unknown alkane and alkene compounds were found in the surface water samples. Results for the BNA analysis of surface water samples taken in in Dry Run are presented in Table 1 and in Appendix B.

Well Water

The sample taken at the Tennant Farm well produced no detections from the standard BNA list. Several TICs were identified, however only one of the detected compounds could be tentatively characterized and identified as an alkene. Results for the BNA analysis of the well water sample taken at the Tennant farm is presented in Table 1 and in Appendix B.

Soil

Analysis of the surface soil samples from the meadows adjacent to the streambed and the reference meadow area produced a few isolated hits from the standard BNA list. Fluoranthene was detected at an estimated concentration of 23 ug/Kg in one of the three reference samples. Carbazole was detected at an estimated concentration of 41 ug/Kg in one of the three Area I soils. Di-n-butylphthalate was detected at concentrations of 22, 27, 26, and 30 ug/kg in one sample from area II, one sample from area three, and two of the three samples from area IV, respectively. Bis(2-Ethylhexyl)phthalate was detected at estimated concentrations of 27 and 62 ug/Kg in one sample from Area III and one sample



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from Area IV. Di-n-octylphthalate was detected at an estimated concentration of 180 ug/kg in one of the samples from area IV. In addition, numerous TICs including unknown alkane, cycloalkane, alkene, aldehyde, sterols, alcohols, PAH, acid, and other organic compounds were found in the surface soil samples. Results for the BNA analysis of soil samples is presented in Table 2 and in Appendix B.

Sediment

Analysis of the sediment samples from the five site and two off-site stream locations produced only a few isolated detections of BNA compounds. Di-n-butylphthalate was detected in the Area IV sediment sample at an estimated concentration of 30 ug/Kg. Bis(2-Ethylhexyl)phthalate was detected in the Area III sediment sample at an estimated concentration of 52 ug/Kg. No other standard list compounds were found in any of the Lee Creek, reference stream, or Dry Run sediment samples. Numerous TICs including unknown alkane, cycloalkane, alkene, aldehyde, sterols, alcohols, PAH, acid, and other organic compounds were found in the Dry Run, Lee Creek, and reference sediment samples. Results for the BNA analysis of sediment samples is presented in Table 3 and in Appendix B.

3.1.2 TAL Metals

Surface Water

Analysis of the surface water samples from Dry Run, the reference stream, and Lee Creek included both filtered and unfiltered samples for TAL metals analysis. Antimony, arsenic, beryllium, cadmium, cobalt, mercury, nickel, selenium, silver, thallium, and vanadium were not detected in any of the filtered or unfiltered water samples.

Aluminum, barium, calcium, copper, iron, magnesium, manganese, potassium, sodium, and zinc were detected in the filtered water samples. Of the list of detected metals in the filtered samples, it appears that aluminum, copper, and zinc are found in higher concentrations in the Dry Run Creek drainage, including the reference stream, than in Lee Creek.

Aluminum, barium, calcium, copper, iron, magnesium, manganese, potassium, sodium, and zinc were detected in the unfiltered samples. Of the list of detected metals in the unfiltered samples, concentrations of aluminum, iron, and zinc appear to be higher in Dry Run than those measured in Lee Creek.

Detailed results of the TAL metals analysis in filtered and unfiltered water samples are presented in Table 4 and in Appendix B.

Well Water

Well water sampled from the well on the Tennant farm was analyzed as an unfiltered sample. Antimony, arsenic, beryllium, cadmium, chromium, cobalt, mercury, nickel, selenium, silver, thallium, and vanadium were not detected in the well sample. Concentrations measured for the remaining list of TAL metals are presented in Table 4 and in Appendix B.



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Soil

Three replicate surface soil samples from the four Dry Run meadow areas and the reference meadow area were analyzed for TAL metals. Antimony, cadmium, mercury, selenium, silver, and thallium were not detected in any of the soil samples. One-way analysis of variance determined that soil manganese concentrations were significantly higher in Area II compared to the reference area, but the same as those noted in areas I, III, and IV ($p=0.094$). Area II had the highest mean manganese concentration with mean concentrations from the other areas ranging from 680 to 1310 mg/kg. Further results of the TAL metals analysis of site and reference soil samples are presented in Table 5 and Appendix B.

Sediment

Seven sediment samples were submitted for TAL metals analysis. Five of the samples were taken in the streambed of Dry Run, one was taken in Lee Creek, and one was taken in the reference stream. Antimony, cadmium, mercury, selenium, silver, and thallium were not detected in any of the sediment samples. In comparison to the levels measured in the Lee Creek sample, it appears the Dry Run Creek reach may be enriched in aluminum, arsenic, barium, calcium, chromium, cobalt, copper, iron, lead, magnesium, manganese, nickel, potassium, sodium, vanadium, and zinc. Based on the results of the aluminum, barium, chromium, cobalt, copper, iron, lead, manganese, nickel, vanadium, and zinc analysis, there also appears to be a general trend that metal concentrations decrease with increasing distance from the landfill. Further results of the TAL metals analysis of site and reference sediment samples are presented in Table 6 and Appendix B.

3.1.3 Pesticides/PCBs

Water

No pesticides or PCBs were detected in the Dry Run samples, the Lee Creek sample, or in the reference stream sample (Table 7; Appendix B).

Soil

No pesticides or PCBs were detected in the Dry Run meadow samples, or in the reference meadow samples (Table 8; Appendix B).

Sediment

No pesticides or PCBs were detected in the Dry Run samples, the Lee Creek sample, or in the reference stream sample (Table 9, Appendix B).

3.1.4 VOCs

Water

No volatile organic carbon compounds were detected in the Dry Run samples, the Lee Creek sample, or in the reference stream sample (Table 10; Appendix B).



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Soil

Trichlorofluoromethane was detected in every soil sample taken in the meadows adjacent to Dry Run at concentrations ranging from 0.9 to 3.6 ug/Kg. In addition, tetrachloroethene was detected in one replicate Area III soil sample at a concentration of 4.4 ug/Kg. No other volatile organic carbon compounds were detected in the Dry Run samples, the Lee Creek sample, or in the reference stream sample. Results of the VOC analysis of site and reference soil samples are presented in Table 11 and Appendix B.

Sediment

Acetone was detected in the Area IV sample at a concentration of 7.2 ug/Kg. Chloroform was detected at a concentration of 0.5 ug/kg in the Area III sample. No other volatile organic carbon compounds were detected in the Dry Run samples, the Lee Creek sample, or in the reference stream sample. Results of the VOC analysis of site and reference sediment samples are presented in Table 12 and Appendix B.

3.1.5 Total Fluoride

Water / Well Water

Fluoride was not detected in the Dry Run samples, the Lee Creek sample, the reference stream sample, or in the well sample taken on the Tennant farm (Table 13; Appendix B).

Soil

Fluoride was detected in the Dry Run meadow and in the reference meadow samples. Soil fluoride concentrations ranged from a low of 180 mg/kg in Area IV to a high of 370 mg/kg in Area III. There appear to be no statistically significant differences in total soil fluoride concentration. Results of the soil fluoride analysis are presented in Table 14 and Appendix B.

Sediment

Fluoride was detected in the Dry Run creekbed and in the reference creekbed samples, but not in Lee Creek. Fluoride concentrations ranged from a low of 290 mg/kg in the Area IV sampling area to a high of 450 mg/kg in the Upper Tributary A sampling area. Fluoride was not detected in Lee Creek. Overall, fluoride concentrations tend to decrease with increasing distance from the landfill. Sediments sampled in the Dry Run Creek reach appear to be enriched with fluoride, which is not found in Lee Creek. Results of the sediment fluoride analysis are presented in Table 15 and in Appendix B.

3.1.6 Organofluorides

Sediment

Because of methodology problems, specifically in obtaining appropriate standards, and the high volatility of some standards, only a limited suite of organofluoride compounds could be scanned for in the sediment samples. These compounds are presented in Table 16. Of the list that was analyzed for (Tetrafluoroethylene, hexafluoropropylene,



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chlorodifluoromethane, perfluorocyclobutane, 1-Chloro-1,1,2,2, tetrafluoroethane, 2-Chloro-1,1,1,2,3,3,-hexafluoropropane, and Perfluoroisobutylene), none of the organofluoride compounds were detected in site sediments. Results of the organofluoride analysis is reported in Table 16 and in Appendix B.

3.1.7 Total Organic Carbon and Grain Size of Soil and Sediment

Summaries of total organic carbon and grain size analysis are presented in Tables 17-20 and in Appendix B. TOC in the soil ranged from an average low of 5.6% in Area III soils to an average high of 9.2 in Area IV soils. Soil grain size determinations are summarized in Table 18. TOC in the sediment ranged from a low of 1.9% in Lee Creek to a high of 4.5 % in Area IV. Sediment grain size determination is presented in Table 20.

3.1.8 Water Quality Parameters

Water quality parameters including pH, conductivity, turbidity, dissolved oxygen, temperature, bromide, chloride, nitrate, phosphate, and sulfate was measured in the Dry Run Creek reach, the reference stream, and in Lee Creek. The most notable observations were that conductivity and sulfate concentration decreased with increasing distance from the landfill. Other parameters appeared to be in the expected range. Results of these measurements and analyses are presented in Tables 21 and 22.

3.1.9 Bovine Fecal Samples

Six fecal samples were taken to determine if environmental contaminants were showing up in the digestive products of the affected cattle.

BNAs

Phenol was detected in all six fecal samples at concentrations ranging from 2.6 to 8.0 mg/kg. Additionally, 4-methylphenol was detected in all six samples in concentrations ranging from 45 to 110 mg/kg. Benzoic acid was detected in two of the samples at a concentration of 30 mg/kg. Additional information is presented in Table 23 and in Appendix B.

TAL Metals

Aluminum, barium, calcium, copper, iron, lead, magnesium, manganese, potassium, sodium, vanadium, and zinc were detected in the fecal samples. Additional information presented in Table 24 and in Appendix B.

Fluoride

Fluoride was not detected in any of the fecal samples (Table 25; Appendix B).

3.2 Biotic Sampling and Tissue Analysis

3.2.1 Benthic Macroinvertebrates



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A total of 27 taxonomic groups were collected from the 5 locations sampled (Table 26). Of these, there was 1 Oligochaete, 1 Mollusc, 1 Turbellarian, 3 Crustaceans, and 21 Insect taxa. Of the latter, the dominant group, in terms of taxonomic diversity, were the Coleopteria which were represented by 7 taxa. The Dipteria were represented by 4 taxa and the Ephemeropteria were represented by 3 taxa. The Plecopteria, Hemipteria Tricopteria, and Megaloptera were the least diverse groups and were represented by one or two taxa. The greatest taxonomic diversity was observed at the reference location, where 19 taxa were collected. Fewer taxa were observed at locations I through IV, and the lowest diversity was observed at location IV where 11 taxa were collected. The difference in taxonomic diversity between the reference location and locations I, II, III, and IV was primarily due to the presence of a greater number of rare taxa at the former location.

The number of individuals collected per replicate ranged from 203 at the reference location (replicate A) to 24 at location IV (replicate C). The observed density of individuals throughout the study area is primarily the result of the numerical abundance of only several taxa (Table 26). The numerically dominant taxa collected from the study area includes *Leucrocuta* and *Asellidae*. When present, these taxa were typically the most numerically abundant organisms and were represented by 221 and 391 individuals, respectively. Other taxa, including *Perlista*, *Chironomidae*, *Hyalella*, the Turbellaria, and to a lesser extent, *Leptophlebia*, *Baetis*, and *Pseudolimnophilia*, were present at most locations in consistently significant proportions. In general, most taxa collected were relatively rare and were represented by five or fewer individuals at most locations. For example, of the 129 total taxonomic observances, 54 were represented by one individual, 36 were represented by two to five individuals, 11 by six to 10 individuals, 15 by 11 to 20 individuals, and 13 by greater than 21 individuals.

Several taxa were collected from all locations sampled including *Leucotricia*, *Perlesta*, Chironomidae, and Asellidae (Table 26). Several taxa were not collected from all locations but were broadly represented throughout the drainage including *Leptophlebia*, *Agabus*, *Hyalella*, and Turbellaria. Of the nine taxa observed at only one location, four, including Elmidae, Scirtidae, *Pseudolimnophila*, and Stratiomyidae were collected only from the reference location. The most common distribution observed was one where a taxa was collected in relatively low numbers, and at few locations. For example, Elmidae, *Hydropsyche*, Limnophilidae, *Nigronia*, and Ceratoponidae were collected infrequently and in low numbers. Similarly, *Lipogomphus*, Dytiscidae, Curculidae, Elmidae, Scirtidae, Histeridae, *Pseudolimnophila*, Stratiomyidae, and *Physa* were collected in low numbers at only one location.

Five functional feeding groups were collected from the Dry Run drainage (Table 26). Resulting from the presence of Asellidae and *Hyalella*, omnivores were the dominant functional group at most locations. Although less dominant, collector-gatherers and scrapers were consistently collected from all locations in the study area and included the mayflies *Leucrocuta* and *Leptophlebia*. The dominant scraper was the mayfly *Leucrocuta*. Predators were dominant at locations II and III and were represented primarily by the stonefly *Perlesta*.

The overall assessment of ecological condition first focuses on the evaluation of habitat quality, and secondly on the analysis of biological components in light of habitat. Habitat,



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as the principal determinant of biological potential, sets the stage for interpreting biosurvey data and can be used as a general predictor of biological condition. High quality habitat will support high quality biological communities and responses to minor alterations will be subtle and of little consequence. However, as a habitat declines in quality, discernable biological impairment results. When habitat and biological data are systematically collected together, empirical relationships can be quantified and subsequently used for screening impact and discriminating water quality effects from habitat degradation. The watershed that drains the Dry Run study area has been modified as a result of past and present land use, particularly with respect to cattle grazing and other agricultural practices, as well as the siting of commercial and industrial facilities. The loss of riparian vegetation, through replacement by species resistant or adapted to grazing, or elimination by grazing has several consequences that should be considered when evaluating the distribution of benthic macroinvertebrates and macroinvertebrates in the current study.

The attainable biological potential of a stream or river is primarily determined by the quality of the habitat at a particular location. The Dry Run study area is situated in a rural area utilized primarily for grazing cattle and, although historic indications of grazing are evident, significant portions of the riparian area remain vegetated, and there are few areas with a completely open canopy and exposed soil. Portions of the Dry Run drainage, though somewhat degraded, support a surprisingly diverse and apparently robust aquatic community. The taxonomic diversity and numerical abundance of the macroinvertebrate was relatively high at the reference area. In contrast, the diversity and abundance at locations I, II, III, and IV was reduced substantially. Since habitat considerations at all locations in Dry Run are similar, the presence of contamination at the latter locations may be significant.

### 3.2.2 Mammal

Four species of small mammals (meadow voles, short-tailed shrews, white-footed mice, and meadow jumping mice) were caught during the trapping effort. Whole bodies were submitted for lipid, TAL metal and total fluoride analysis.

The trapping effort revealed at least one important field observation, which was there was extremely low trapping success in Area I, the area nearest the landfill outfall, as compared to the other areas. This is highly irregular given the similar habitats present site-wide, and may indicate an ecological threat. Field necropsies identified several significant problems with the small mammals collected in the meadow areas adjacent to Dry Run. Short-tail shrews sampled from all areas showed blackened and degenerating teeth. Shrews commonly have what is known as chestnut tipped teeth, where the extreme points of the dentitia are a light brown color. The black, mottled, and degenerating teeth observed in this study are not normally observed in shrews. One meadow vole sampled from the area was missing the left kidney, and another appeared to have and extra kidney or an extra lobe on the right kidney, independent of the adrenals.

Sufficient numbers of meadow voles were caught from the Reference Area, Area II, Area III, and Area IV for statistical comparisons of tissue concentrations. Lipid concentration of meadow voles was significantly depressed in the Reference Area, Area II, and Area III, compared to that observed in Area IV ($p < 0.001$). Barium concentration was significantly lower in the Reference Area, Area II, and Area III, compared to that observed in Area IV



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($p=0.067$).

Sufficient numbers of short-tail shrews were caught from the Reference Area and Area III for statistical comparisons. There were no differences in body concentration of lipid, TAL metals, and fluoride in shrews taken from these two areas.

Results of the trapping success, TAL metals, and fluoride analysis are presented in Tables 27-29, and in Appendix B. Results are presented by species and trapping location.

3.2.3 Fish

Four species of fish were collected from Dry Run in Areas II, III, and IV. No fish were observed in Area I or the Reference Area. Creek chubs and fantail darters were collected in Area IV. Creek chubs and river chubs were collected in area III. Creek chubs, river chubs, fantail darters, central stone rollers, and black-nose dace were collected in Area II. Fish sampled during the electrofishing effort in Dry Run were submitted for whole body lipid, TAL metal and fluoride analysis. A composite sample that was taken during a historical fish kill in Dry Run was also analyzed.

Since creek chubs were the only species common to all three sampling locations, statistical analysis concentrated on differentiating between tissue concentrations in this species. Aluminum, arsenic, barium, beryllium, cobalt, iron, lead, manganese, thallium, and vanadium were significantly higher in creek chubs from area II than those sample in Areas III and IV. Likewise, concentrations of these metals in Area III creek chubs were higher than those in Area IV. Conversely, cadmium and silver concentrations showed the reverse trend, with tissue concentrations in Area IV significantly higher than those measure in Areas II or III. In spite of this result, it is clear that fish inhabiting upper reaches of Dry Run, nearer to the landfill are being dosed with a significantly higher amount of metals than those in the lower reaches. There were no difference in lipid or fluoride concentration.

Results of the chemical analyses are presented in Tables 30-32 and in Appendix B.

3.2.4 Earthworm

Three replicate earthworm samples were produced from each of the earthworm toxicity test soil samples. One-way analysis of variance was used to look for significant differences in tissue concentrations between earthworms exposed to each of the five soil treatments. Cadmium and thallium concentrations in the earthworm tissue was significantly higher in the Area I exposures than those in Areas II, III, IV, or the Reference soils. Cobalt levels were lower in worms taken from the reference area, but tended to increase with increasing distance from the landfill. Copper and nickel concentrations were higher in worms taken from the reference soils than those observed in worm taken from the other soil area exposures.

Further results of the earthworm tissue analyses for lipid, TAL metals and fluoride concentration are presented in Tables 33-35 and in Appendix B.

3.2.5 Vegetation



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Three replicate samples of meadow grass were taken in each of the five soil sampling locations. One way analysis of variance was used to look for differences in plant tissue concentrations across the five sampling areas. Barium concentration was significantly higher in the Area I vegetation than in the Area III and IV vegetation, but similar to that observed in plants take in the Reference and in Area II. Manganese concentration was significantly higher in the Area I and reference vegetation than in the Area II, III and IV vegetation. Cobalt was significantly higher in Area IV vegetation than that taken in any of the other areas. There was no difference in the fluoride or lipid concentration.

Results of the TAL metals and fluoride analysis are presented in Tables 36-38 and in Appendix B.

3.3 Histological assay of small mammal liver and kidney

Histological analysis of liver and kidney sections of meadow voles, short-tail shrews, meadow jumping mice, and white-footed mice trapped in each of the five trapping areas concluded that there were no substantive changes in liver or kidney morphology. The absence of a kidney in one animal, and the presence of an extra lobe on the right kidney of another provide anecdotal evidence of an effect, however we are unable to ascertain the importance of these observations. Full summaries of the histopathological work are presented in Table 39 and in Appendix B.

3.4 Toxicity Testing

Earthworm

Based on the toxicity evaluation of soils, there is no evidence for growth or survival effects on earthworms tested in any of the soil samples collected at the Dry Run Creek site. Survival was 100% in all treatment replicates and growth ranged from 32.4 to 54.3%. Further results of the earthworm toxicity test are presented in Table 40 and in Appendix C.

Fathead minnow

Based on the toxicity evaluation of surface water samples to the fathead minnow, it appears that surface water taken in the Upper Tributary A location induced significant mortality. Survival in the Upper Tributary A sample was 58% while survival in all other samples, including the reference location, ranged from 87 to 100%. There appear to be no growth related effects water, on the minnows in any of the water samples taken from Dry Run Creek. Survival was negatively correlated potassium concentrations, however these correlations are not statistically significant at the 0.10 level. There was a significant positive correlation between fathead survival and iron concentrations in the filtered water samples. Further results of the fathead minnow toxicity test are presented in Table 40 and in Appendix C.

Amphipod

Based on the results of the 10 day solid phase whole sediment toxicity test with the amphipod, *Hyalella azteca*, growth of the organisms was inhibited in the sediment samples taken at the Upper Tributary A, and Area II locations. There were no effects observed on survival in any of the samples taken. The observed negative growth effect was significantly negatively correlated with



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fluoride, aluminum, calcium, magnesium, nickel, potassium, and sodium. Further, there were strong negative associations between the growth endpoint and chromium, copper, lead, and zinc concentrations, although the relationships were not significant at the 0.10 level. Further results of the amphipod toxicity test are presented in Table 40 and in Appendix C.

## 4.0 SUMMARY OF PRELIMINARY ECOLOGICAL RISK ASSESSMENT SCREEN

Sediment, soil and water concentrations were compared against Region III BTAG screening values (U.S. EPA 1995). Hazard quotients were generated by dividing the maximum site concentration measured in each matrix by the corresponding Region III benchmark values. All contaminants for which maximum concentrations exceeded benchmarks for sediment, soil, and water in the initial screening-level risk assessment are listed in the following sections. Contaminants that failed the initial screening process will be further evaluated in a final risk assessment for the site.

### Sediment

Table 1 lists maximum concentrations, screening criteria, and quality criteria factors for sediment contaminants. The maximum concentration recorded at the site exceeded the benchmark values for the following compounds: arsenic, chromium, copper, manganese, and nickel. Because of the lack of a screening benchmark, the following compounds are still considered as risk factors as well: fluoride, aluminum, barium, beryllium, cobalt, iron, and vanadium.

### Water

Table 1 lists maximum concentrations, benchmarks, and quality criteria factors for water contaminants. The maximum concentration recorded at the site exceeded the benchmark values for the following compounds: aluminum, copper, and iron. Because of the lack of a screening benchmark, fluoride is considered to be a potential risk factor.

### Soil

Table 1 lists maximum concentrations, screening criteria, and quality criteria factors for soil contaminants. The maximum concentration recorded at the site exceeded the benchmark values for the following compounds: aluminum, beryllium, chromium, copper, iron, lead, manganese, vanadium, and zinc. Because of the lack of a screening benchmark, the following compounds are still considered as risk factors as well: fluoride and trichlorofluoromethane.

## 5.0 DISCUSSION

The data generated during the field effort suggests that there are potential problems associated with conditions at the Dry Run Creek site. Minimally, the results of the sediment and water toxicity tests suggest potential problems in the stream. Some metals appear in higher tissue concentrations in biota sampled nearest the outfall of the landfill, with those levels progressively dropping with increasing distance from the landfill area. Likewise, sediments sampled in Dry Run near the landfill outfall appear to have higher fluoride and metals concentrations than those sampled further downstream. A preliminary screen of potential risk factors suggests that other problems, specifically elevated levels of fluorides, organofluorides, and some metals, may be present as well. Data gathered during the field effort will be further analyzed through a base-line ecological risk assessment for the Dry Run Creek site



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## SECTION II ECOLOGICAL RISK ASSESSMENT

### 1.0 INTRODUCTION

#### 1.1 Objective

The objective of this risk assessment was to provide technical support to the U.S. Environmental Protection Agency Region III in conducting an evaluation of potential ecological threat due to existing contaminant levels in soil, sediment, and water at a working beef production farm located down gradient of a landfill. Soil, sediment, surface water, and biota samples were collected for contaminant analyses and soil, sediment, and surface water were collected for laboratory toxicity testing. The information gathered during this field effort was incorporated into in an ecological risk assessment for the Dry Run Creek site.

#### 1.2 Site Background

The site is a working beef production farm located in Washington, Wood County, WV. The owner of the farm has filed numerous complaints with the West Virginia Department of Natural Resources and the U. S. EPA alleging that contaminants are being discharged from an industrial landfill owned by the Dupont corporation, into Dry Run. Dry Run flows through the farmer's property and is a primary source of water for his cattle. The farmer maintains that numerous deaths, blindness, and other unusual illnesses observed in his herd are directly attributable to the contaminants that are discharged into Dry Run from the Dupont landfill. In addition to these abnormalities, numerous fish and wildlife kills have also been reported in Dry Run since the construction of the landfill.

### 2.0 PROBLEM FORMULATION

This risk assessment was designed to evaluate the potential threats to ecological receptors from exposure to site contaminants. During the preliminary risk assessment, the problem formulation process included the identification of COCs and a comparison of the maximum concentration of COCs with accepted benchmarks. This information was then used to identify complete exposure pathways of compounds exceeding benchmarks to ecological receptors and their appropriate measurement endpoints.

The first step of the preliminary risk assessment process compared all chemicals analyzed in soil, sediment, and water during the field with established toxicological benchmarks. Benchmarks for sediment and soil were used to identify potential contaminants of concern for the protection of aquatic biota (U.S. EPA 1995, Long and Morgan 1990, Long et al. 1995, Persuad et al. 1992, U.S. EPA 1992, Suter and Mabrey 1994). Compounds exceeding benchmarks were retained for further evaluation using ingestion-based exposure models for higher vertebrates, and direct exposure assays for fish, benthic and terrestrial invertebrates.

#### 2.1 Ecological Risk Assessment

This ecological risk assessment was written to determine the risk associated with the exposure of biota to site-related contaminants. The following steps were completed for this preliminary risk assessment:

(1) A literature search was conducted to locate life history information for selected indicator species, to determine ecotoxicological effects of site contaminants, and to locate bioconcentration factors for site contaminants.

(2) An evaluation of ecological receptors was prepared. This consisted of the following:

- Exposure scenarios were determined based on site contaminant levels, the extent and magnitude of contamination, and the toxicological mechanisms of the



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contaminants.

- Indicator species were selected based on species present and/or potentially present on site, the availability of toxicity information from the literature, and the potential for exposure to site contaminants based on habitat use or behavior.
- Exposure pathway(s) were determined for each indicator species.
- Exposure and effect profiles were written for each indicator species and each site contaminant.
- A risk characterization was conducted which involved the calculation of hazard quotients (HQs) for each species for a range of exposure scenarios.

Based on the results of this evaluation, the COCs identified in the initial screen were further evaluated through the use of conservative risk models.

## 2.2 Identification of the Contaminants of Concern

The contaminants of potential concern were identified using the initial contaminant screen. The COCs for this site that were retained through the preliminary screen include metals, fluoride, and organofluoride compounds.

## 2.3 Exposure Characterization

The objective of the exposure assessment is to determine the pathways and media through which receptors may be exposed to site contaminants. Potential exposure pathways are dependant on habitats and receptors present on site, extent and magnitude of contamination, and environmental fate and transport of COCs. In this base-line ecological risk assessment, it will be concluded that "a potential risk" exists if the HQ calculated from the maximum site concentration and the No Observed Apparent Effect Level (NOAEL) equals or exceeds 1.

Exposure to COCs present in forage and prey species via ingestion could cause toxicity in higher trophic level organisms. In addition to exposure via consumption of contaminated forage, ecological receptors may also be exposed through ingestion of water and incidental ingestion of soil/sediment. The exposure of benthic invertebrates, terrestrial invertebrates, and fish was determined by examining results of the toxicity tests.

## 2.4 Hazard Characterization/Toxicity Assessment

To determine the effects of contaminants on biota, it is necessary to understand the mechanisms of toxicity of the chemicals and the systems that they affect. Knowledge of the fate, effects, and mode of action of the COCs allows for the selection of appropriate assessment endpoints. Next is a review of the general toxicological information for the COCs identified in Section 2.4.

### 2.4.1 Fluoride

Inorganic fluoride compounds are ubiquitous in nature. However, industrial processes such as manufacturing and mining have contributed to the environmental load of fluoride, primarily through atmospheric deposition. In low doses, it is accepted that fluoride is



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protective of teeth in humans as well as other animals. However in higher levels it is generally accepted that fluoride can be toxic to both plant and animal life. Dental and skeletal lesions, lameness, stiffness of gait, appetite impairment, decreased weight gain, decreased milk production, posture abnormalities, tremors, stillbirths, overgrowth of hooves, severe diarrhea, and death have been associated with mammalian fluoride toxicity (Suttie, 1977; Shupe et al, 1992). In addition to the effects known in mammals, birds are also susceptible to fluoride toxicity. Mortality, decreased growth rates, depressed appetite, and decreased eggshell quality have been reported as toxicological endpoints of fluoride exposure in birds (Fleming and Schuler 1988; Fleming et al. 1987; Guenter and Hahn 1986).

### 2.4.2 Organofluorides

Organofluorides are used in a variety of industrial processes including the production of Teflon™, propellants, and refrigerants. Available toxicological data generally concentrates on inhalation exposure and dermal absorption. Acute (10 day) exposure of rats to chlorodifluormethane produced decreased maternal and fetal weights, as well as an increased frequency of anopthalmia and subsequent blindness in newborn fetuses (IARC 1986). Hexafluoropropene exposure induced an increased incidence of hamster ovary cell aberrations and increased frequency grossly abnormal cells (HSDB 1997).

### 2.4.3 Aluminum

Because of its strong reactivity, aluminum (Al) is not found as a free metal in nature. Aluminum has only one oxidation state (+3), thus its behavior in the environment depends on its ordination chemistry and the surrounding conditions. In soils, a low pH generally results in an increase in aluminum mobility. In water, an equilibrium with a solid phase is established that controls the extent of aluminum dissolution (ATSDR 1990).

Plants vary in their ability to remove aluminum from soils, although bioconcentration factors for plants are generally less than one. Biomagnification of aluminum in terrestrial food chains does not appear to occur. There is no data on the biomagnification of aluminum in aquatic food chains (ATSDR 1990).

The nervous system may be a target area for aluminum. Aluminum accumulates in neurofibrillary tangles in humans with Alzheimer's disease. Aluminum may also interact with neuronal DNA to alter gene expression and protein formation. Mammalian studies do not indicate that aluminum affects reproduction although some developmental effects have been reported in mammals (ATSDR 1990). Aluminum is known to interfere with gill transport of oxygen and carbon dioxide in fish, and has also been identified in ionoregulatory disruption.

### 2.4.4 Arsenic

Several review articles are available which discuss the toxic effects of As (Eisler 1988a, Nriagu 1994). Arsenic tends to be widespread in the environment (Woolson 1975) and is constantly being oxidized, reduced, or mobilized (Eisler 1988a). Physical processes are important in determining As bioavailability in aquatic environments. For example, arsenates are readily adsorbed onto sediments with high organic matter, and arsenates are more strongly adsorbed onto sediments than other As forms. However, absorption depends on the As concentration, sediment characteristics, pH, and ionic concentration of other compounds



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(Eisler 1988a; U.S. EPA 1981). The U.S. EPA (1981) noted that arsenate (pentavalent) is the predominant As form in oxygenated water and that arsenite (trivalent) is the predominant As form in anaerobic conditions.

Arsenic is not significantly concentrated in aquatic invertebrates; whole body concentration factors for invertebrates range from 3 to 17 for exposure to arsenic trioxide (trivalent) and from 0 to 7 for arsenic pentoxide (pentavalent). Arsenic may be bioconcentrated by organisms at the bottom of the food chain; however, data do not indicate that significant biomagnification occurs (U.S. EPA 1985).

### 2.4.5 Beryllium

The majority of the beryllium (Be) in the environment is the result of coal and oil combustion. Beryllium naturally enters waterways through the weathering of rock and soil, and through deposition of atmospheric beryllium. Upon reaching water and soil, beryllium is most likely retained as an insoluble form that is generally immobile. However, beryllium chloride, fluoride, nitrate, phosphate, and sulfate (tetrahydrate) are all water-soluble forms (ATSDR 1993a).

Due to its geochemical similarity to aluminum, beryllium may be expected to adsorb onto clay surfaces at low pHs, and it may remain precipitated as insoluble complexes at higher pHs. Therefore, beryllium is expected to have limited mobility in soil (ATSDR 1993a).

Beryllium is not expected to bioconcentrate in aquatic animals and no evidence for significant biomagnification within food chains has been found. Beryllium is extremely toxic to warmwater fish in soft water. The degree of toxicity decreases with increasing hardness (ATSDR 1993a).

Major exposure routes for aquatic ecological receptors include ingestion of contaminated soil and sediment. Although several studies point out the negative effects of beryllium in mammalian systems, no studies that evaluated the relationship between sediment beryllium concentration and observed toxicity to benthic organisms could be found (ATSDR 1993a).

### 2.4.6 Chromium

Chromium (Cr) can exist in oxidation states ranging from -2 to +6, but is most frequently converted to the relatively stable trivalent (+3) and hexavalent (+6) oxidation states (Eisler 1986a). In both freshwater and marine systems, hydrolysis and precipitation are the most important processes that determine the fate and effects of Cr, whereas adsorption and bioaccumulation are relatively minor. Precipitated $Cr^{+3}$ hydroxides remain in sediments under aerobic conditions. However, under anoxic and low pH conditions, $Cr^{+3}$ hydroxides may solubilize and remain as ionic $Cr^{+3}$ unless oxidized to $Cr^{+6}$ through mixing and aeration (Eisler 1986a). In soils, the solubility and bioavailability of Cr are governed by soil pH and organic complexing substances, although organic complexes play a more significant role (James and Bartlett 1983a; James and Bartlett 1983b).

The trivalent state is the form usually found in biological materials. This form functions as an essential element in mammals by maintaining efficient glucose, lipid, and protein metabolism (Stevens et al. 1976). Chromium is beneficial but not essential to higher plants (Eisler 1986a). The biomagnification and toxicity of $Cr^{+3}$ is low relative to $Cr^{+6}$ because of



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its low membrane permeability and its noncorrosivity. However, a large degree of accumulation by aquatic and terrestrial plants and animals in the lower trophic levels has been documented (Eisler 1986a), although, the mechanism of accumulation remains largely unknown.

Chromium is mutagenic, carcinogenic, and teratogenic, with $Cr^{+6}$ exhibiting the greatest toxicity; relatively less is known about the toxicity of $Cr^{+3}$. At high concentrations, $Cr^{+6}$ is associated with abnormal enzyme activity, altered blood chemistry, lowered resistance to pathogenic organisms, behavioral modifications, disrupted feeding, histopathology, osmoregulatory upset, alterations in population structure, and inhibition of photosynthesis.

Rabbits fed dietary Cr accumulated hyaluronates, chondroitin sulfates, and neutral mucopolysaccharides in the soft tissues, causing pericapillary sclerosis (Kucher and Shabanov 1967). This accumulation blocked blood tissue barriers, which are permeable under normal conditions, preventing the normal transport of metabolites. One manifestation of this condition was the inhibition of insulin production in the pancreatic islets due to damage to the beta-cells contained therein.

Chromium also leads to nephron damage via swelling and loss of microvilli, the formation of intracellular vacuoles, mitochondrial swelling, and cytoplasmic liquefication and loss of cells lining the nephron surface (Evan and Dail 1974).

The preliminary step in Cr-induced respiratory cancer is speculated to be the scarring of alveolar tissue, followed by the elicitation of inflammatory reactions in lung tissue leading to bronchopneumonia, alveolar epithelial changes, atrophy, and benign tumor formation. Direct skin contact with highly corrosive chromic acid and its anhydride produces skin ulcers and necrosis by a mechanism independent of any allergic response (Steven et al. 1976).

### 2.4.7 Copper

Copper (Cu) does not appear to have mutagenic properties (IRIS 1990), but it is a teratogen (RTECS 1991) and a possible carcinogen (Venugopal and Luckey 1978). Copper is caustic, and acute toxicity is primarily related to this property (Hatch 1978).

Copper is an essential element for animals and is a component of many metalloenzymes and respiratory pigments (Demayo et al. 1982). It is also essential to iron (Fe) utilization and functions in enzymes for energy production, connective tissue formation, and pigmentation (Venugopal and Luckey 1978). Excess Cu ingestion leads to accumulation in tissues, especially in the liver. High levels of Cu modify hepatic metabolism (Brooks 1988), which may lead to inability of the liver to store and excrete additional Cu. When liver concentration exceeds a certain level, the metal is released into the blood, causing hemolysis and jaundice. High Cu levels also inhibit essential metabolic enzymes (Demayo et al. 1982). Toxic symptoms appear when the liver accumulates 3 to 15 times the normal level of Cu (Demayo et al. 1982).

Although the exact mechanism of toxicity is not known, the following mechanisms have been proposed: formation of stable inhibitory complexes with cytochrome P-450 (Wiebel et al. 1971); impairment of function of NADPH-cytochrome c reductase and alteration of mixed function oxidations (Reiners et al. 1986); and inhibition of heme biosynthesis (Martell 1981). Intranuclear inclusions may act as a detoxifying mechanism where Cu is complexed by



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protein ligands, protecting cytoplasmic organelles (Demayo et al. 1982).

Ruminants are the most sensitive mammal species to Cu toxicosis. Young animals retain more dietary Cu than older animals and are more sensitive to Cu toxicity (Venugopal and Luckey 1978).

2.4.8 Iron

Iron (Fe) is commonly detected in concentrations of 5 percent or more in soil. It is used primarily in the production of steel and other alloys as well as a major source of hydrogen. Iron is a constituent of hemoglobin and is essential to plant and animal life as well as being an important component in cellular oxidative processes. The disposition of ingested iron is regulated by a complex mechanism to maintain homeostasis. Therefore, bioconcentration in biota is not expected to be a significant process for iron. Generally, about 2 to 15 percent of ingested iron is absorbed from the gastrointestinal tract, and elimination is approximately 0.01 percent of the body burden per day. Adverse effects of iron toxicity may include renal failure and hepatic cirrhosis. The mechanism of toxicity begins with acute mucosal cell damage and absorption of ferrous ions directly into circulation, resulting in capillary endothelial cell damage to the liver (Shacklette and Boerngen 1984).

2.4.9 Lead

Lead does not biomagnify to a great extent in food chains, although accumulation by plants and animals has been extensively documented (Wixson and Davis 1993, Eisler 1988b). Older organisms typically contain the highest tissue Pb concentrations, with the majority of the accumulation in the bony tissue of vertebrates (Eisler 1988b).

Predicting the accumulation and toxicity of Pb is difficult since its effects are influenced to a very large degree, relative to other metals, by interactions among physical, chemical, and biological variables. In general, organolead compounds are more toxic than inorganic Pb compounds, and young, immature organisms are most susceptible to its effects (Eisler 1988b). In plants, Pb inhibits growth by reducing photosynthetic activity, mitosis, and water absorption. The mechanism by which photosynthetic activity is reduced is attributed to the blocking of sulfhydryl groups, inhibiting the conversion of coproporphyrinogen to protoporphyrinogen (Holl and Hampp 1975).

The toxic effects of Pb on aquatic and terrestrial organisms are extremely varied and include mortality, reduced growth and reproductive output, blood chemistry alterations, lesions, and behavioral changes. However, many effects exhibit general trends in their toxic mechanism. Generally, Pb inhibits the formation of heme, adversely affects blood chemistry, and accumulates at hematopoietic organs (Eisler 1988b). At high concentrations near levels causing mortality, marked changes to the central nervous system occur prior to death (Eisler 1988b).

Plants can uptake Pb through surface deposition in rain, dust, and soil, or by uptake through the roots. The ability of a plant to uptake Pb from soils is inversely related to soil pH and organic matter content. Lead can inhibit photosynthesis, plant growth, water absorption.

2.4.10 Manganese



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Manganese (Mn) does not occur as a free metal in the environment but is a component of numerous minerals. Elemental manganese and inorganic manganese compounds have negligible vapor pressures, but may exist in air as suspended particulate matter derived from industrial emissions or the erosion of soil. Removal from the atmosphere is mostly through gravitational settling. The transport and partitioning of manganese in water is controlled by the solubility of the specific chemical form present. The metal may exist in water in any of four oxidation states (2+, 3+, 4+, or 7+). Divalent manganese ($Mn^{+2}$) predominates in most waters (pH 4 to 7), but may become oxidized at a pH greater than 8 or 9. Manganese is often transported in moving water as suspended sediments. The tendency of soluble manganese compounds to adsorb to soils and sediments depends mainly on the cation exchange capacity and the organic composition of the soil. Manganese in water may be significantly bioconcentrated at lower trophic levels. However, biomagnification in the food chain may not be significant (ATSDR 1990).

The amount of manganese absorbed across the gastrointestinal tract is variable. There does not appear to be a marked difference between manganese ingested in food or in water. One of the key determinants of absorption appears to be dietary iron intake, with low iron levels leading to increased manganese absorption. This is probably because both iron and manganese are absorbed by the same transport system in the gut (ATSDR 1990).

### 2.4.11 Nickel

Pure nickel (Ni) is a hard, white metal that is usually used in the formation of alloys (such as stainless steel) and Ni combined with other elements is found in all soils. Nickel is the 24$^{th}$ most abundant element and is found in the environment as oxides or sulfides. Nickel may be released into the environment through mining, oil-burning power plants, coal-burning power plants, and incinerators. Nickel will attach to soil or sediment particles, especially those containing Fe or manganese (Mn). Under acidic conditions, Ni may become more mobile and seep into the groundwater. The typical Ni concentration reported in soils is from 4 - 80 milligrams per kilograms (mg/kg). The speciation and physicochemical state of Ni is important in considering its behavior in the environment and its availability to biota.

The most probable exposure routes of Ni are through dermal contact, inhalation of dust, and ingestion of Ni-contaminated soil. The respiratory system is the primary target of Ni exposure following inhalation. Manifestations such as inflammation of the lungs, fibrosis, macrophage hyperplasia, and increased lung weight have been noted in animals exposed to Ni. Animals exposed to Ni through oral exposure were noted to have lethargy, ataxia, irregular breathing, salivation, and squinting (ATSDR 1996).

### 2.4.12 Vanadium

Elemental vanadium does not occur naturally but it can exist in 50 different ores and fossil fuels. Other anthropogenic sources include acid-mine leachate, sewage sludge, and fertilizers. The principal use of vanadium is as an alloy constituent, especially in steel. The addition of vanadium to steel removes oxygen and nitrogen, which improves the strength. The average concentration of vanadium in the earths crust is 150 mg/kg and in the U.S. soils are 200 mg/kg (Byerum et al. 1974).

The release of vanadium to water and soil occurs as a result of the weathering of rocks and from soil erosion. This process usually converts the less-soluble trivalent form to the more-



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soluble pentavalent form. The mobility of vanadium in soil is affected by pH, redox potential, and the presence of particulates. Relative to other minerals, vanadium is mobile in neutral or alkaline soils and its mobility decreases in acidic soils (ATSDR 1991; Van Zinderen Bakker and Jaworski 1980).

In the terrestrial systems, bioconcentration is more common in lower plant species. In addition, vanadium concentrations in plants are dependent on the amount of water-soluble vanadium, pH, and growing conditions. Vanadium appears to be present in all terrestrial mammals but the concentrations are usually below the detection limits. The highest concentration of vanadium is usually found in the liver and skeletal tissues (ATSDR 1991).

Vanadium is very poorly absorbed into the gastrointestinal tract and the toxic mechanism of vanadium on the respiratory system is similar to other metals (Castronova et al. 1984). Vanadium damages the alveolar macrophages by decreasing the macrophage membrane integrity. Damaged macrophages inhibit the ability of the respiratory system to clear itself of other particles. In vitro experiments indicate that the mechanism of toxicity of vandium is by inhibiting sodium-potassium ATPase activity, which inhibits the sodium-potassium pump. This pump is necessary for the transport of material across cell membranes (Nechay and Saunders 1978).

### 2.4.13 Zinc

Zinc (Zn) is essential for normal growth and reproduction in plants and animals and is regulated by metallothioneins. Metallothioneins act as temporary Zn storage sites and aid in reducing the toxicity of Zn to both vertebrates and invertebrates (Olsson et al. 1989). Zinc is not known to bioaccumulate in food chains, because it is regulated by the body and excess Zn is eliminated.

Zinc has its primary metabolic effect on Zn-dependant enzymes that regulate the biosynthesis and catabolic rate of ribonucleic (RNA) acid and deoxyribonucleic acid (DNA). High levels of Zn induce Cu deficiency and interfere with metabolism of calcium (Ca) and Fe (Goyer 1986). The pancreas and bone seem to be the primary targets of Zn toxicity in birds and mammals. Pancreatic effects include cytoplasmic vacuolation, cellular atrophy, and cell death (Lu and Combs 1988, Kazacos and Van Vleet 1989). Zinc preferentially accumulates in bone, and induces osteomalacia (a softening of bone caused by a deficiency of Ca, phosphorus and other minerals) (Kaji et al. 1988). Gill epithelium is the primary target site in fish. Zinc toxicosis results in destruction of gill epithelium and tissue hypoxia (Spear 1981).

## 2.5 Selection of Assessment Endpoints

The information gathered during a site reconnaissance and during the field work, and subsequent discussions with the U.S. EPA on-scene coordinator and the Region III Biological Technical Assistance Group, allowed for the selection of assessment endpoints that corresponded to the habitat types present at the Dry Run Creek site. The site is composed of a variety of habitats including forested and old-field uplands, grassy meadows, the creek, and associated riparian areas. A variety of birds, mammals, and fish may use the site for feeding and nesting. Likewise, terrestrial and benthic invertebrates are key elements in the functions of these systems. Therefore, the assessment endpoints focused toward these faunal groups. Viability of terrestrial, avian, and aquatic populations and organism survivability were selected as assessment endpoints for this risk assessment. Listed next are



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the specific assessment endpoints selected for this ecological risk assessment.

Ten assessment endpoints were chosen to evaluate the risk of contaminants at the Dry Run Creek site:

1) protection of benthic invertebrate community structure and function

2) protection of soil invertebrate community structure and function

3) protection of fish communities to ensure that direct exposure to contaminants does not have a potential negative impact on growth, survival, or reproductive success.

4) protection of worm-eating birds to ensure that ingestion of contaminants in forage does not have a potential negative impact on growth, survival, or reproductive success.

5) protection of carnivorous birds to ensure that ingestion of contaminants in forage does not have a potential negative impact on growth, survival, or reproductive success.

6) protection of carnivorous mammals to ensure that ingestion of contaminants in forage does not have a potential negative impact on growth, survival, or reproductive success.

7) protection of piscivorous mammals to ensure that ingestion of contaminants in forage does not have a potential negative impact on growth, survival, and reproductive success.

8) protection of omnivorous mammals to ensure that ingestion of contaminants in forage does not have a potential negative impact on growth, survival, and reproductive success.

9) protection of insectivorous mammals to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success.

10) protection of herbivorous mammals to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success.

## 2.6 Production of Testable Hypotheses

The testable hypotheses are specific risk questions that are based on the assessment endpoints. Based on the mechanism of contaminant toxicity, the number of exposure pathways that may exist for an assessment endpoint, or other factors, there may be more than one question for each assessment endpoint.

Are levels of site contaminants sufficient to have negative effects on benthic invertebrate community structure and function?

Are levels of site contaminants sufficient to have negative effects on soil invertebrate community structure and function?

Are levels of site contaminants sufficient to cause direct toxicity to fish growth, survival, and reproductive success?

Are levels of site contaminants sufficient to cause negative impacts on growth, survival, or reproductive success of worm-eating birds due to the ingestion of contaminated forage, soil, and water



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on the site?

Are levels of site contaminants sufficient to cause negative impacts on growth, survival, and reproductive success of carnivorous birds due to the ingestion of contaminated forage, soil, and water on the site?

Are levels of site contaminants sufficient to cause negative impacts on growth, survival, and reproductive success of carnivorous mammals due to the ingestion of contaminated forage, soil, and water on the site?

Are levels of site contaminants sufficient to cause negative impacts on growth, survival, and reproductive success of piscivorous mammals due to the ingestion of contaminated forage, soil, and water on the site?

Are levels of site contaminants sufficient to cause negative impacts on growth, survival, and reproductive success of omnivorous mammals due to the ingestion of contaminated forage, soil, and water on the site?

Are levels of site contaminants sufficient to cause negative impacts on growth, survival, and reproductive success of insectivorous mammals due to the ingestion of contaminated forage, soil, and water on the site?

Are levels of site contaminants sufficient to cause negative impacts on growth, survival, and reproductive success of herbivorous mammals due to the ingestion of contaminated forage, soil, and water on the site?

## 2.7 Conceptual Model

The conceptual model relies on contaminant and habitat characteristics to identify critical exposure pathways to the selected measurement endpoints. At the Dry Run Creek site, contaminants in the soil may come in contact with subsurface (earthworms) and above-ground terrestrial receptors (small mammals) inhabiting the wooded, wetland, and open field areas of the site. Subsurface terrestrial receptors in these areas may be exposed to site contaminants through direct contact with the soil, and in some cases, the intentional ingestion of soil. Above-ground terrestrial receptors may be exposed to contaminants through direct contact with the soil, the ingestion of subsurface terrestrial organisms, the ingestion of other above-ground terrestrial receptors, the incidental ingestion of soil adhered to food items, and the intentional ingestion of surface water from any of the on-site surface drainages.

The wooded areas, riparian area, and meadow areas provide distinct habitat types that may support a variety of terrestrial and avian receptors. For example, a small omnivorous mammal may occupy one or all the habitat types, whereas, an individual carnivorous mammal may regularly traverse all three habitats daily in search of food items. Avian piscivores and carnivores may be exposed to site contaminants in much the same way as an above-ground terrestrial receptor. The consumption of contaminated prey, the incidental ingestion of soil/sediment, and the consumption of surface water may transfer contaminants to these receptors.

The conceptual model relies on contaminant and habitat characteristics to identify critical exposure pathways to the selected measurement endpoints. The preliminary risk screen identified metals, fluoride, and trichlorofluoromethane as the primary contaminants exceeding benchmarks in site sediment, soil, and water. Benthic macroinvertebrates, fish, and terrestrial invertebrates may be



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exposed to contaminated sediment, water, or soil through direct toxicity. For the purposes of this risk assessment, the concentration of the contaminants of concern found in the sediment, water, or soil were correlated with toxicity levels identified in the corresponding toxicity tests to determine if benthic invertebrates fish, or terrestrial invertebrates may be at risk. Terrestrial receptors may be exposed to contaminants by feeding on organisms which have accumulated COCs in their tissues. Higher trophic level receptors may also be exposed to contaminants from food ingestion and via incidental ingestion of soil/sediment and water. The pathway to the reference area meadow is unknown, however the pathway to the reference area stream may involve groundwater transport. The following pathways were evaluated in this risk assessment:

I. Benthic invertebrates
   a) Direct exposure to sediment

II. Soil invertebrates
   a) Direct exposure to soil

III. Fish
   a) Direct exposure to water

IV. Worm-eating bird
   a) Ingestion of earthworms
   b) Incidental ingestion of soil
   c) Incidental ingestion of water

V. Carnivorous bird
   a) Ingestion of small mammals
   b) Incidental ingestion of soil
   c) Incidental ingestion of water

VI. Carnivorous mammal
   a) Ingestion of small mammals
   b) Incidental ingestion of soil
   c) Incidental ingestion of water

VII. Piscivorous mammal
   a) Ingestion of forage fish
   b) Incidental ingestion of sediment
   c) Incidental ingestion of water

VIII. Omnivorous mammal
   a) Ingestion of forage fish
   b) Incidental ingestion of sediment
   c) Incidental ingestion of water

IX. Insectivorous mammal
   a) Ingestion of earthworms
   b) Incidental ingestion of sediment
   c) Incidental ingestion of water

X. Herbivorous mammal



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a) Ingestion of vegetation
b) Incidental ingestion of sediment
c) Incidental ingestion of water

2.8 Selection of Measurement Endpoints

Measurement endpoints are measurable ecological characteristics that are related to the valued characteristics selected as assessment endpoints. Measurement endpoints should be linked to the assessment endpoints by the mechanism of toxicity and the route of exposure. Measurement endpoints are used to derive a quantitative estimate of potential effects, and form a basis for extrapolation to the assessment endpoints.

Measurement endpoints were selected on the basis of potential presence of receptors on site, and the potential for exposure to contaminants of concern. The availability of the appropriate toxicity information on which risk calculations could be based was also an important consideration. Endpoints selected were determined to be representative of exposure pathways and assessment endpoints identified for the site.

Next is a list of specific measurement endpoints that correspond to the assessment endpoints identified in Section 2.5.

**Measurement endpoints for assessment endpoint:**

- protection of benthic invertebrate communities structure and function

To evaluate the structure and function of the benthic community, benthic macroinvertebrates were collected from five locations in Dry Run. Existing community structure was evaluated at each of the five locations by determining taxonomic diversity and through an evaluation of functional feeding groups.

Sediment was also collected in each of the five areas for toxicity testing using the amphipod, *Hyallela azteca*. The endpoints of these tests were survival and growth. Collocated sediment samples were also collected and analyzed for target analyte list (TAL) metals, BNA's, Pest/PCBs, VOCs, fluoride, grain size, and total organic carbon (TOC). The chemistry results were then correlated with observed adverse biotic responses in the toxicity tests in order to determine risk potential.

**Measurement endpoints for assessment endpoint:**

- protection of soil invertebrate community structure and function

To evaluate the structure and function of the benthic community, soil was collected from each of the meadow locations and tested using the earthworm, *Eisenia foetida* in toxicity tests. The endpoints of these tests were survival and growth. Collocated soil samples were also collected and analyzed for target analyte list (TAL) metals, BNA's, Pest/PCBs, VOCs, fluoride, grain size, and total organic carbon (TOC).

**Measurement endpoints for assessment endpoint:**

- protection of fish communities to ensure that direct exposure to contaminants does not have a negative impact on growth, survival, and reproductive success.



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Fathead minnow, *Pimephales promelas*, toxicity tests were used to determine the toxicity of the water in Dry Run. The endpoints of these tests were survival and growth. Collocated water samples were also collected and analyzed for target analyte list (TAL) metals, BNA's, Pest/PCBs, VOCs, fluoride, grain size, and total organic carbon (TOC). The chemistry results were then correlated with observed adverse biotic responses in the toxicity tests in order to determine risk potential.

**Measurement endpoints for assessment endpoint:**

- Protection of worm-eating birds to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success.

Food chain accumulation studies were selected to evaluate risk to avian species which utilize the site as a feeding area. The selected measurement endpoint receptor species is the American robin, *Turdus migratorius*. Appropriate forage species (earthworms) were identified for the above receptor, and the dietary exposure of receptors to contaminants was quantified and compared to existing toxicity data for these, or other closely related species.

**Measurement endpoints for assessment endpoint:**

- Protection of carnivorous birds to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success.

Food chain accumulation studies were selected to evaluate risk to avian species which utilize the site as a feeding area. The selected measurement endpoint receptor species is the red-tailed hawk, *Buteo jamaicensis*. Appropriate forage species (small mammals) were identified for the above receptor, and the dietary exposure of receptors to contaminants was quantified and compared to existing toxicity data for these, or other closely related species.

**Measurement endpoints for assessment endpoint:**

- Protection of carnivorous mammals to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success.

Food chain accumulation studies were selected to evaluate risk to mammalian species which utilize the site and adjacent areas. The selected measurement endpoint receptor species is the red fox, *Vulpes vulpes*. Appropriate forage species (small mammals) were identified for the above receptors and the dietary exposure of receptors to contaminants was quantified.

**Measurement endpoints for assessment endpoint:**

- Protection of piscivorous mammals to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success.

Food chain accumulation studies were selected to evaluate risk to mammalian species which utilize the site and adjacent areas. The selected measurement endpoint receptor species are the mink, *Mustela vison*. Appropriate forage species (fish) were identified for the above receptors and the dietary exposure of receptors to contaminants was quantified.

**Measurement endpoints for assessment endpoint:**



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- Protection of omnivorous mammals to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success.

Food chain accumulation studies were selected to evaluate risk to mammalian species which utilize the site and adjacent areas. The selected measurement endpoint receptor species is the raccoon, *Procyon lotor*, as a model for omnivorous mammalian species. Appropriate forage species (fish) were identified for the above receptors and the dietary exposure of receptors to contaminants was quantified.

**Measurement endpoints for assessment endpoint:**

- Protection of insectivorous mammals to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success

Food chain accumulation studies were selected to evaluate risk to mammalian species which utilize the site and adjacent areas. The selected measurement endpoint receptor species is the short-tail shrew, *Blarina brevicauda*, as a model for insectivorous mammalian species. Appropriate forage species (earthworms) were identified for the above receptors and the dietary exposure of receptors to contaminants was quantified.

**Measurement endpoints for assessment endpoint:**

- Protection of herbivorous mammals to ensure that ingestion of contaminants in forage does not have a negative impact on growth, survival, and reproductive success

Food chain accumulation studies were selected to evaluate risk to mammalian species which utilize the site and adjacent areas. The selected measurement endpoint receptor species is the meadow vole, *Microtus pennsylvanicus*, as a model for herbivorous mammalian species. Appropriate forage species (vegetation) was identified for the above receptors and the dietary exposure of receptors to contaminants was quantified.

## 2.9 Life History/Exposure Profile Information

Receptor species were selected from several trophic levels. Organisms which were likely to be exposed to contaminants because of specific behaviors, patterns of habitat use, or feeding habits were selected for evaluation in this risk assessment. The availability of appropriate toxicity information on which risk calculations could be based was also an important consideration. The terrestrial invertebrate receptor selected for this assessment is the earthworm. The terrestrial vertebrate receptor species selected for this risk assessment are: meadow vole, short-tail shrew, raccoon, mink, and red fox. The avian receptor species selected for this risk assessment are: American robin and red-tailed hawk. The aquatic vertebrate receptor species for this risk assessment is the fathead minnow. The aquatic invertebrate receptor is *H. azteca*.

### 2.9.1 The amphipod (*Hyallela azteca*) as Representative of Benthic Invertebrates

Justification

*Hyallela azteca* was selected as representative of benthic invertebrates due to their direct contact with sediment for a significant portion of their life cycle, ubiquitous distribution in aquatic systems, importance as a food item for aquatic-invertebrate consumers, and ease of



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use in laboratory toxicity evaluations. These species are also likely to occur in the surface sediment at the Dry Run Creek site.

Life History (*Hyallela azteca*)

The amphipod, *Hyallela azteca*, is commonly found in freshwater lakes, streams, ponds, and rivers throughout North and South America. In preferred habitats, they are known to reach densities in excess of 10,000 per square meter. They may also be found in sloughs, marshes, and ditches, but generally in lower numbers (U.S. EPA 1994).

*Hyallela azteca* are epibenthic detritivores that feed on coarse particulate organic material. They typically burrow into surface sediment, and avoid bright light. Because of their feeding and behavioral characteristics, they are ideal test organisms for toxicological evaluation of freshwater sediments. Avoidance of light by movement into the sediment keeps these organisms almost constantly in contact with sediment contaminants (U.S. EPA 1994).

Reproduction in this crustacean is sexual. Males are larger than females and have larger front gnathopods that are presumably used for holding the female during amplexus and copulation. During amplexus, the male and female feed together for a period of up to one week. The pair separates temporarily while the female goes through a molting period. Immediately after the molt, the two rejoin and copulation begins. During copulation, the male releases sperm near the female's marsupium. The female sweeps the sperm into her marsupium, and simultaneously releases eggs from her oviducts, into the marsupium, where fertilization takes place. The average brood size for female *Hyallela azteca* is 18 eggs per brood, but this number can vary with environmental conditions and physiological stress (U.S. EPA 1994).

Developing embryos and hatched young are kept inside the female's marsupium until she undergoes a second molt. At that time, the juvenile *Hyallela azteca* are released into the surrounding environment. Under favorable conditions, each female produces approximately one brood during every ten day time period (U.S. EPA 1994).

*Hyallela azteca* have a minimum of 9 instars, with 5 to 8 pre-reproductive stages. The first five stages are juvenile stages; instars 6 and 7 form the adolescent stages; and stages 8 and higher are considered adult (fully reproductive) stages (U.S. EPA 1994).

Exposure Profile for *Hyallela azteca*

Since direct contact with contaminated sediment in the toxicity evaluation is the primary route of exposure for *Hyallela azteca* in this risk assessment, the results of the test will be used to indicate exposure.

2.9.2 Earthworm (*Eisenia foetida*) as Representative of Terrestrial Invertebrates

Justification

Earthworms were selected as representative of terrestrial invertebrates due to their feeding habits, ubiquitous distribution throughout many habitats and soil conditions, and importance in providing a food base for many small- to medium-sized predators. A diet of detritus, microflora, and microfauna, combined with direct contact with the surrounding soil, presents a potential link between soil contaminants and soil-invertebrate consumers. In addition,



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earthworms were observed in both the wooded and open field areas of the Dry Run Creek site.

Life History

Earthworms feed on dead and decaying plant and animal remains and on free-living soil microflora and microfauna. Their primary source of food is dead plant material, especially plant litter. Next to food, their most important requirement is adequate moisture. Water conservation mechanisms are poorly developed: respiration depends on diffusion of gases through the body wall which must be kept moist. Earthworms are generally absent or rare in soils with very coarse texture, in soils with high clay content in regions of high rainfall, and in soils with a pH of less than 4 (Lee 1985).

Earthworms are hermaphroditic and most species reproduce by cross-fertilization, although many species can also produce cocoons parthenogenetically. Sexual reproduction cannot occur without a clitellum, ovaries, oviducts, and possibly the ovisacs, but male organs are not essential. The population of an earthworm species at any one time consists of young immature, well-grown immature (adolescent), mature, and senescent individuals (Edwards and Lofty 1977).

Earthworms have several ways of surviving adverse environmental conditions such as soil desiccation and ambient cold and heat. In terms of population survival, the cocoons can resist desiccation and temperature extremes much more easily than mature individuals. Worms may also migrate to deeper soil or undergo states of inactivity until environmental conditions become favorable once again (Edwards and Lofty 1977).

Some species of worms grow throughout their lives by continually adding segments proliferated from a growing zone located just in front of the anus. Other species, such as *E. foetida*, possess the adult number of segments upon hatching and increase in size without increasing the number of segments. The life span of *Eisenia foetida* was reported to be approximately 4.5 years under laboratory conditions (Edwards and Lofty 1977).

Exposure Profile

Direct contact with contaminated soil is the primary route of exposure for earthworms in this risk assessment. Survival and growth endpoints following exposure to site soils will be used to evaluate risk to these organisms. Tissue residue analysis will also be conducted on the worms to determine exposure to higher trophic level organisms.

2.9.3 Fathead Minnow (*Pimephales promelas*) as Representative of Fish Community

Justification

The fathead minnow was selected as representative of omnivorous fish due to its dietary composition, direct contact with water throughout the life cycle, ubiquitous distribution in aquatic systems, importance as a food item for fish-eating consumers, and ease of use in laboratory toxicity evaluations.

Life History



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The fathead minnow, *P. promelas*, is widely distributed in North America and is found in a variety of habitats such as small streams, ponds, and small lakes. It is uncommon or absent in streams of moderate and high gradients. It is tolerant of high temperature, high turbidity, and low oxygen concentrations (U.S. EPA 1985).

The fathead minnow is primarily omnivorous. Young typically feed on detritus, algae, and zooplankton. Adults feed on aquatic insects, worms, small crustaceans, and other animals. This species is considered an important food source for other fish and birds (U.S. EPA 1985).

Adult fathead minnows spawn in the spring and continue to spawn throughout most of the summer. The minimum spawning temperature appears to be approximately 16°C. The ovaries of the females contain eggs in all stages of development, and they spawn repeatedly as the eggs mature. The average number of eggs per spawn per female is 100 to 150. Larger females may lay 400 to 500 eggs per spawn. Hatching times depend on temperature and average about six days. In warm water with an ample food supply, spawning may occur as early as the first year. In cooler water with a moderate food supply, spawning usually occurs during the second year. Survival to the third year is relatively uncommon (U.S. EPA 1985).

Exposure Profile

Since direct contact with contaminated water in the toxicity evaluation is the primary route of exposure for fathead minnows in this risk assessment, the results of the test will be used to indicate exposure.

2.9.4 American Robin (*Turdus migratorius*) as Representative of Worm-eating Birds

Justification

The American robin was selected as representative of omnivorous and carnivorous birds because of its ubiquitous distribution and dietary composition. The preference for soil invertebrates in its omnivorous diet allows this species to be used as both an omnivorous and carnivorous receptor in this risk assessment. This species is also likely to occur at the Dry Run Creek site.

Life History

The American robin (*Turdus migratorius*) occurs throughout most of the continental U.S. and Canada, wintering in the southern half of the U.S., Mexico, and Central America. Given the increase in open habitat and lawns, the robin's breeding range has expanded in the recent times. Habitat requirements for breeding robins include access to fresh water, protected nesting sites, and productive foraging areas. These requirements are commonly met in moist forests, swamps, open woodlands, and other open areas. Non-breeding robins occupy similar habitats although proximity to fruit bearing trees is of more importance.

The primary foraging technique for robins is to hop along the ground in search of ground-dwelling invertebrates, although they commonly search for insects and fruit in tree branches as well. The robin's diet during the breeding season consists mainly of invertebrates and some fruit, but fruit is the primary food consumed outside of the breeding season. As robins exhibit a low digestive efficiency for fruit, they often consume more than their own body weight in fruit to meet their metabolic needs (U.S. EPA 1993).



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Breeding territories are established by male robins. Most foraging occurs close to these territories during the breeding season; however, if densities of robins are high in a given area or if food resources are limited, adult robins will leave to temporarily forage elsewhere. Outside of the breeding period, robins typically return to the same foraging sites and roost within 1 to 3 kilometers (km) of these areas (U.S. EPA 1993).

Exposure Profile

Adult American robins are reported to weigh from 77.3 to 133.8 g (U.S. EPA 1993). Territory sizes vary from 0.3 to 1 acre, with foraging home ranges reported up to 2 acres (U.S. EPA 1993). The lowest reported body weight (77.3 g) and the smallest reported home range of (0.3 acres) were assumed for this risk assessment.

A food ingestion rate of 0.89 to 1.52 g/g BW/day and a water ingestion rate of 0.14 g/g BW/day are reported for this species (U.S. EPA 1993). Assuming a 77.3 g body weight, an American robin can be expected to consume 117.5 g/day of food and 10.8 g/day of water.

The diet of the American robin consists of seasonally variable proportions of invertebrates (e.g., earthworms, snails, beetles, caterpillars, spiders) and fruit (e.g., dogwood, cherry, sumac, hackberries, raspberries) (U.S. EPA 1993, Ehrlich et al. 1988). During spring, summer, and fall, the dietary composition is reported to change from 93 percent invertebrates and 7 percent fruit in the spring (nesting season) to 92 percent fruit and 8 percent invertebrates in fall (migratory season). The summer dietary proportion is reported as 68 percent fruit and 32 percent invertebrates (U.S. EPA 1993). For the purposes of this risk assessment, a diet of 100% earthworms will be assumed.

An incidental soil ingestion rate for the American robin could not be found in the literature. However, a soil ingestion rate of 10.4 percent of the diet reported for the American woodcock will be used as a substitute ingestion rate for the American robin (Beyer et al. 1994). Assuming a food ingestion rate of 117.5 g/day, the soil ingestion rate for the American robin is 12.2 g/day.

2.9.5 Red-tailed Hawk (*Buteo jamaciensis*) as Representative of Carnivorous Birds.

Justification

The red-tailed hawk was selected as representative of a carnivorous bird due to its dietary composition, relative abundant distribution, and likelihood of occurrence at the Dry Run Creek site. Its diet allows for the evaluation of contamination in site soils. In addition, the concentration of contaminants found in small mammal tissue will also provide an accurate dose to the red-tailed hawk which allows for the evaluation of contaminants in the food source.

Life History

Red-tailed hawks are the most common and widespread American *Buteo* (Bull and Farrand 1977). Their habitat is highly variable, but they are commonly found in wooded areas near open land. They also inhabit plains, prairie groves, and deserts in the western United States (NGS 1987). This species is absent, however, from tundra, and rare in extensive unbroken forest. An opportunistic feeder, the red-tailed hawk hunts from a perch or on the wing for



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food items such as small mammals (e.g., mice, chipmunks, rabbits), birds (usually ground-dwelling species), reptiles, insects, and occasionally, prey species that are too heavy to lift off the ground (Burton 1989).

The breeding season starts with aerial courtship displays, commonly followed by mating on a perch and nest-building by both sexes. Nests are placed in tall trees, high rock ledges, or tall cacti and are often refurbished annually for use in consecutive years. Incubation of two to three eggs is carried out by both sexes and lasts for approximately 30 days. The young are able to feed themselves at 4 to 5 weeks and fledge in about 45 days (Bull and Farrand 1977; Burton 1989).

Exposure Profile

Adult male and female red-tailed hawks are reported to weigh 960 g and 1,235 g, respectively (DeGraaf and Rudis 1983; U.S. EPA 1993). Home ranges vary from 148.26 to 395.36 acres (Kirkwood 1980). The lowest reported body weight of 0.960 kg and the smallest reported home range of 148.26 acres were assumed for this risk assessment.

The diet of a red-tailed hawk consists of mammals, birds, reptiles, and insects which vary in importance with season and availability (U.S. EPA 1993). For the purposes of this risk assessment, the hawk will be assumed to consume 100% small mammals. Food ingestion rates are reported to range from 136 to 400 g/day (Kirkwood 1980). The highest reported food ingestion rate of 400 g/day was assumed for this risk assessment. A water ingestion rate of approximately 0.059 g/g BW/day has been estimated for this species (U.S. EPA 1993). To express this value in units of g/day, the water ingestion rate was multiplied by the lowest reported body weight of 960 g to yield a water ingestion rate of 56.64 g/day (56.64 mL/day).

A soil ingestion rate for the red-tailed hawk could not be found in the literature; therefore, the amount of soil predicted to be entrained in the digestive tract of a white-footed mouse was used to calculate this value. A soil ingestion rate of less than 2 percent of the total diet has been reported (Beyer et al. 1994) for the white-footed mouse. From this value, a conservative soil ingestion rate of 1.9 percent of the total diet was assumed for the white-footed mouse. To express this value in units of g/day, the soil ingestion rate of 1.9 percent was multiplied by the food ingestion rate of the white-footed mouse (4.50 g/day) (U.S. EPA 1993) to yield a soil ingestion rate of 0.09 g/day. This value was assumed to represent the amount of soil entrained in the digestive tract of the white-footed mouse that remains constant over time. To express 0.09 g in units of grams of soil per gram of mouse body weight, this value was divided by the lowest reported body weight (13 g) of the white-footed mouse (Merritt 1987) to yield a value of 0.007 g/g BW. This value was then multiplied by the food ingestion rate of the red-tailed hawk (400 g/day) to yield a soil ingestion rate of 2.8 g/day.

2.9.6 Red Fox (*Vulpes vulpes*) as Representative of Carnivorous Mammals

Justification

The red fox was selected as representative of a carnivorous mammal due to its dietary composition, relative abundant distribution, and likelihood of occurrence at the Dry Run Creek site. Its diet allows for the evaluation of contamination in site soils. In addition, the concentration of contaminants found in small mammal tissue will also provide an accurate



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dose to the red fox which allows for the evaluation of contaminants in the food source.

Life History

Red fox inhabit open meadows, ditch banks, field and wood edges, fencerows, stream and lake borders, and farmlands (Hoffmeister 1989; Jones and Birney 1988; Merritt 1987). With the exception of the breeding season, red fox have no permanent home but sleep on the ground (Schwartz and Schwartz 1981). A den, usually modified from an existing woodchuck or fox den, is dug during the breeding season and exceptionally cold winters (Barbour and Davis 1974). These scent-marked dens have multiple rooms, entrances, and trails leading to and from hunting areas (Schwartz and Schwartz 1981). In addition to their dens, both males and females will defend their scent-marked hunting territory from intruders (Jones and Birney 1988).

The red fox is primarily an opportunistic carnivore, consuming food items such as rabbits, opossums, muskrats, skunks, rodents, birds, eggs, carrion, invertebrates, snakes, and frogs (Barbour and Davis 1974; Merritt 1987). Some vegetable matter such as fruits and nuts are also consumed when in season (Jones and Birney 1988). During times of abundant food supply, the red fox will bury surplus food to return to for consumption at a later time (Schwartz and Schwartz 1981).

Male and female foxes pair for life, remaining together from midwinter to summer. Females bear one litter per year usually between March and April (Merritt 1987). Gestation periods last from about 49 to 56 days, with most averaging 53 days (Schwartz and Schwartz 1981). The pups are weaned at about 60 days, leave the den in the autumn, and are sexually mature by their first winter (Merritt 1987). Natural predators of the red fox are few but include large hawks and owls, and possibly coyotes (Merritt 1987; Schwartz and Schwartz 1981). Red fox may live from six to ten years in the wild (Schwartz and Schwartz 1981).

Exposure Profile

Adult red fox weigh from 2.7 to 7 kg (Barbour and Davis 1974; Jones and Birney 1988). Home ranges vary from 245 to 1,235 acres (Merritt 1987).

The food ingestion rates of the red fox range from 0.069 g/g BW/day for a nonbreeding adult to 0.16 g/g BW/day for a juvenile (U.S. EPA 1993). The water ingestion rate for an adult red fox is estimated to be approximately 0.086 g/g BW/day (U.S. EPA 1993). To express these values in units of g/day, the highest reported food ingestion rate of 0.16 g/g BW/day and the water ingestion rate of 0.086 g/g BW/day were multiplied by the lowest reported body weight of 2.7 kg (2,700 g) to yield a food ingestion rate of 432 g/day and a water ingestion rate of 232.2 g/day (232.2 mL/day). For the purposes of this risk assessment, a diet of 100% small mammals will be assumed.

A soil ingestion rate of 2.8 percent of the total diet has been reported (Beyer et al. 1994) for the red fox. To express this value in units of g/day, the soil ingestion rate of 2.8 percent was multiplied by the food ingestion rate of 432 g/day to yield a soil ingestion rate of 12.1 g/day.

2.9.7 Mink (*Mustela vison*) as Representative of Carnivorous Mammals

Justification



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The mink was selected as representative of a carnivorous mammal due to its dietary composition, relative abundant distribution, and likelihood of occurrence at the Dry Run Creek site. Its diet allows for the evaluation of contamination in site soils. In addition, the concentration of contaminants found in clams and fish tissue will also provide an accurate dose to the mink which allows for the evaluation of contaminants in the food source.

Life History

Mink are distributed over much of boreal North America, southward throughout the eastern United States and in the west to California, New Mexico, and Texas (Jones and Birney 1988). They can be found in virtually any habitat containing permanent water thus, they are not commonly found in upland areas (Jones and Birney 1988). Although primarily nocturnal, their activity often extends into midday (Hoffmeister 1989).

Dens are always near water, and they are usually an old muskrat burrow or constructed by the mink itself (Jones and Birney 1988). Males tend to live in their own burrows which are less elaborate than ones occupied by females (Barbour and Davis 1974). Home ranges tend to be linear since mink often follow a shoreline (Jones and Birney 1988). Mink are solitary and mark their territories by spraying (Merritt 1987).

Seasonal food availability governs the dietary composition (Barbour and Davis 1974). Their diets may consist of crayfish, frogs, fish, snakes, rodents, rabbits, and plants among other items (Jones and Birney 1988; Schwartz and Schwartz 1981). Crayfish are a major portion of the summer diet in many regions of North America (Barbour and Davis 1981; Jones and Birney 1988; Merritt 1987).

Breeding occurs from January to early April with highly variable gestation periods ranging from 40 to 75 days (Merritt 1987; Schwartz and Schwartz 1981). A highly variable single litter of 1 to 17 young may be produced (Schwartz and Schwartz 1981). Average litter sizes vary among regions (Barbour and Davis 1974; Hoffmeister 1989; Jones and Birney 1988; Merritt 1987; Schwartz and Schwartz 1981). Young are weaned at about five to six weeks of age and are sexually mature by ten months (Merritt 1987; Schwartz and Schwartz 1981). Occasionally great horned owls, foxes, coyotes, bobcats, and dogs will prey on mink (Merritt 1987; Schwartz and Schwartz 1981). Although some individuals have lived up to six years, mink seldom exceed two years of age in the wild (Schwartz and Schwartz 1981).

Effects Profile

Adult mink weigh from 520 to 1,730 g (Merritt 1987; U.S. EPA 1993). Home ranges vary from 19 to 1,900 acres (U.S. EPA 1993).

A year-round food ingestion rate of 0.22 g/g BW/day has been estimated for both male and female mink (U.S. EPA 1993). To express this value in units of g/day, the food ingestion rate was multiplied by the lowest reported body weight (520 g) to yield a food ingestion rate of 114 g/day. An estimated water ingestion rate of 0.11 g/g BW/day was reported for farm-raised females (U.S. EPA 1993). To express this value in units of g/day, this water ingestion rate was multiplied by the lowest reported body weight of 520 g to yield a water ingestion rate of 57.2 g/day (57.2 mL/day). For the purposes of this risk assessment, a diet of 100% fish will be assumed.



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An incidental sediment ingestion rate was not available from the literature; therefore, a predicted incidental ingestion rate for sediment that may be entrained in the digestive system of the prey item (fish) was used for this risk assessment. Consumption of this prey item was assumed to be the primary mechanism by which mink may incidentally ingest sediment. The derivation of the predicted level of incidental sediment ingestion via consumption of fish is described next.

Life history information for the bluegill (*Lepomis machrochirus*) was used to predict the amount of sediment that may be ingested by mink via consumption of fish. Adult bluegills range in size from 100 to 230 mm (Pflieger 1975; Smith 1985). In keeping with the conservative approach of this risk assessment, the amount of sediment entrained in the lowest body size of 100 mm in length was predicted. The weight of a 100 mm bluegill was calculated to be 18.11 g based on the following algorithm relating length to weight (Hillman 1982):

log Weight (g) = -5.374 + 3.316 log Length (mm)

A daily food ingestion rate of 1.75 percent BW/day has been reported for the bluegill (Kolehmainen 1974). This provides a predicted intake rate of 0.32 g of food per day for a 18.11 g fish. A study evaluating the stomach contents of 153 bluegills reported an average content of detritus and sediment to be 9.6 percent of the total diet (Kolehmainen 1974). If a conservative assumption is made that 9.6 percent of the food ingested is entirely sediment, it can be predicted that a fish of this size may contain 0.03 g of sediment in its digestive system.

For the purpose of this model, it was assumed that the level of sediment contained in the digestive system of a fish remains constant over time. This value (0.03 g) was divided by the predicted fish body weight (18.11 g) to express sediment entrained in fish digestive systems in units of grams of sediment per gram of fish body weight. This provided a value of 0.0017 g sediment/g body weight. When this value is multiplied by the food ingestion rate of the mink (114 g/day), the predicted sediment ingestion rate for the mink through consumption of fish is 0.2 g/day.

2.9.8 Raccoon (*Procyon lotor*) as Representative of Omnivorous Mammals

Justification

The raccoon was selected as representative of a omnivorous mammal due to its dietary composition, relative abundant distribution, and likelihood of occurrence at the Dry Run Creek site. Its diet allows for the evaluation of contamination in site sediment. In addition, the concentration of contaminants found in forage fish tissue and clams will also provide an accurate dose to the raccoon which allows for the evaluation of contaminants in the food source.

Life History

Raccoons are medium-sized omnivores and are abundant throughout North America. Raccoons prefer aquatic habitats, particularly hardwood swamps, flood plains, freshwater wetlands, and salt marshes (Kaufmann 1982). Raccoons have also adapted well to residential areas and farmlands. Raccoons rely heavily on surface waters for foraging and as a source



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of drinking water (Stuewer 1943). Raccoons are active primarily from dusk to dawn (Stuewer 1943) but will alter their activities to opportunistically feed on whatever is available (Sanderson 1987). For example, raccoons living near a salt marsh may become active during the day to take advantage of feeding opportunities during low tide (Ivey 1948). Raccoons feed primarily on fruits, nuts, acorns, grains, insects, frogs, crayfish, and eggs (Palmer and Fowler 1975).

Raccoons in the southern regions of the United States are active year round (Goldman 1950). Adult raccoons are normally solitary but will come together for short periods of time during mating (Kaufman 1982). Mating occurs from March to June in southern areas and each male may mate with several females during each season (Sanderson 1987; Kaufman 1982). Young males are normally not sexually mature in the first breeding season but mature later in the summer, while females mature in the first year (Sanderson 1951).

The home range of a raccoon depends on the animal's age, habitat, food resources, and season (Sanderson 1987). Home ranges are typically a few hundred hectares (ha) but ranges as large as a few thousand ha have been reported (Sanderson 1987). Population densities also depend strongly on the amount of resources in the area. Numbers of 0.1 to 0.2 animals per ha are common (Hoffman and Gottschang 1977).

Raccoons are found near every aquatic habitat. During the last 50 years raccoon populations have increased greatly (Sanderson 1987). In Alabama, adult male raccoons weighed up to 8.8 kilograms (kg) (mean 4.31 kg) while adult females can weigh up to 5.9 kg (mean 3.67 kg) (Johnson 1970). Adult raccoons weigh between 2 and 12 kg (Nowak 1991), and consume 0.5 kg of food per day (Newell et al. 1987).

Raccoons feed primarily on fruits, nuts, acorns, grains, insects, frogs, crayfish, eggs (Palmer and Fowler 1975). In a Maryland forested bottom land, the dietary composition of raccoons during the summer was principly made up of insects (39 percent), wild cherry (17 percent), blackberries (16 percent), crayfish (8 percent), snails (5 percent), herptiles (5 percent), fish (2 percent), rodents (2 percent), corn (1 percent), and trace amounts of *Smilax*, acorns and pokeberry (Llewellyn and Uhler 1952). At Washington state tidewater area raccoons displayed the following dietary composition: molluscs, mussels and oyster (44 percent), Crustacea, shrimp and crabs (25 percent), fish (9 percent), marine worms (20 percent), and Echiurida worms (1 percent) (Tyson 1950).

The home range of a raccoon depends on the animal's age, habitat, food resources, and season (Sanderson 1987). Home ranges are typically a few hundred hectares but ranges as large as a few thousand hectares have been reported (Sanderson 1987). The home range for adult male raccoon found in coastal Georgia raccoons is approximately 65 ha (± 18 SE) while the home range for adult females in the same area is approximately 39 ha (± 16 SE) (Lotze 1979). Population densities also depend strongly on the amount of resources in the area. Numbers of 0.1 to 0.2 animals per hectare is common (Hoffman and Gottschang 1977).

Exposure Profile

For the purposes of this risk assessment, a body weight of 2 kg, an ingestion rate of 0.5 kg/day, and a diet of 80 percent forage fish and 20 percent clams were assumed. A soil ingestion rate of 9.4 percent of the diet has been reported for raccoons (Beyer et al. 1991). Multiplying the ingestion rate by 9.4 percent yields a sediment ingestion rate of 0.047 kg/day.



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A daily water ingestion rate of 0.18 Liters per day (L/day) was calculated using an allometric equation derived by Calder and Braun (1983). A diet of 100% fish will be assumed.

2.9.3 Short-tailed Shrew (*Blarina brevicauda*) as Representative of Insectivorous Mammals

Justification

The short-tailed shrew was selected as representative of insectivorous mammals because of its dietary composition, relative abundant distribution in both moist and dry habitats, and likelihood of occurrence at the Dry Run Creek site. Although their diets may consist of plants as well as insects, they tend to favor soil invertebrates when they are in abundance. Hence, by assuming that their dietary composition comprises solely invertebrates in this risk assessment, this species may represent an insectivorous mammal.

Life History

The short-tailed shrew is an extremely active, large, and heavy-bodied shrew common within its range (Jones and Birney 1988). It occupies a variety of moist and dry habitats such as marshes, bogs, moist forest floors with ample decaying matter, brushland, fencerows, weedfields, and pastures (Barbour and Davis 1974; Jones and Birney 1988). Short-tailed shrews are active both day and night throughout the year, although most of this activity is subnivean (Merritt 1987). During harsh winters, this species may undergo a period of torpor (Hoffmeister 1989).

The home range of this species varies with their dramatic population cycles. In peak years, animal density may be greater than 25 individuals per acre (Schwartz and Schwartz 1981). In other years, this species may have an animal density of one individual per acre (Merritt 1987).

Although short-tailed shrews strongly prefer animal matter, they are opportunistic omnivores and will voraciously consume whatever food items are in ample supply (Barbour and Davis 1974). These food items include earthworms, slugs, snails, insects, arthropods, fungi, vegetable matter, seeds, snakes, salamanders, small mammals, and young birds (Barbour and Davis 1974; Jones and Birney 1988; Schwartz and Schwartz 1981). Plant matter is generally consumed to a greater extent in winter (Schwartz and Schwartz 1981). In some regions, plant matter may constitute up to 20 percent of the shrew's diet (Barbour and Davis 1974). Submaxillary glands produce a venom that quickly immobilizes their prey (Merritt 1987). Prey items that are not consumed immediately are stored in a cache (Merritt 1987).

Using echolocation and scent-marking, short-tailed shrew rely heavily on their hearing and sense of smell to locate food and to move about (Hoffmeister 1989). An elaborate system of runways and tunnels are constructed usually just a few inches below the ground surface (Schwartz and Schwartz 1981). Two types of nests are built by this species, a breeding nest and a resting nest. Both nests are built underground beneath a log, rock, or other cover, and have multiple entrances. The breeding nest is typically larger than the resting nest (Merritt 1987).

Breeding appears to commence in early spring and extend into the fall, although in some regions, breeding may subside in early and midsummer but peak again in early fall (Hoffmeister 1989; Jones and Birney 1988). Gestation periods are approximately 21 to 22



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days with litter sizes of approximately four to ten young (Jones and Birney 1988; Schwartz and Schwartz 1981). The young are fully mature from one to three months of age (Barbour and Davis 1974; Schwartz and Schwartz 1981). Both sexes may breed their first spring (Schwartz and Schwartz 1981).

Natural predators of the short-tailed shrew include fish, snakes, owls, hawks, shrikes, opossums, raccoons, foxes, weasels, bobcats, skunks, and feral cats, although most of these predators do not consume the shrew (or at least all of the shrew) because of their distasteful musk glands (Barbour and Davis 1974; Jones and Birney 1988; Merritt 1987; Schwartz and Schwartz 1981). The life expectancy of a short-tailed shrew in the wild is approximately one year (Schwartz and Schwartz 1981).

Exposure Profile

Adult short-tailed shrews weigh from 12 to 30 grams (g) (Jones and Birney 1988; Merritt 1987). Home ranges vary from 0.5 to 1 acre (Merritt 1987). Therefore, it was assumed that a short-tailed shrew could obtain 100 percent of its diet from the contaminated area (area use factor of 1), since the area comprising the on-site sampling locations was approximately 20 acres.

Food ingestion rates ranging from 0.49 to 0.62 gram per gram of body weight per day (g/g BW/day ) have been reported (U.S. EPA 1993). An average food ingestion rate of 7.95 g/day has also been reported (U.S. EPA 1993). To express the former food ingestion rates in units of g/day for comparison to the latter ingestion rate, the former ingestion rates were multiplied by the lowest reported body weight of 12 grams to yield food ingestion rates of 5.88 to 7.44 g/day. Of these values, the highest food ingestion rate of 7.95 g/day will be used for the purposes of this risk assessment.

A water ingestion rate of 0.223 g/g BW/day has been reported (U.S. EPA 1993). To express this value in units of g/day, the water ingestion rate was multiplied by the lowest reported body weight of 12 g to yield a water ingestion rate of 2.7 g/day (2.7 milliliters per day [mL/day]).

A soil ingestion rate for the short-tailed shrew was not available from the literature, therefore, the soil ingestion rate of the opossum was used. The opossum's diet is similar to that of the short-tailed shrew since they are both opportunistic omnivores with a strong preference for animal matter (Schwartz and Schwartz 1981). A soil ingestion rate of 9.4 percent of the diet was reported for the opossum (Beyer et al. 1994). This value was multiplied by the highest food ingestion rate of the short-tailed shrew (7.95 g/day) to yield a soil ingestion rate of 0.74 g/day. For the purposes of the food chain model in this risk assessment, it was assumed that 100 percent of the diet of the short-tailed shrew was comprised of earthworms.

2.9.10 Meadow Vole (*Microtus pennsylvanicus*) as Representative of Herbivorous Mammals

Justification

The meadow vole was selected as representative of herbivorous mammals because of its dietary composition, abundance in North America, preference for moist areas, and likelihood of occurrence at the Dry Run Creek site.



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Life History

The meadow vole is one of the largest and most abundant voles in North America (Jones and Birney 1988; Merritt 1987). Although they are more commonly found in habitats such as moist meadows, bogs, swamps, stream banks, and lakeshores, they have also been known to inhabit cultivated fields, roadside ditches, and fencerows (Barbour and Davis 1974; Jones and Birney 1988; Merritt 1987; Schwartz and Schwartz 1981). Dense vegetative cover appears to be one of the major prerequisites for habitation (Hoffmeister 1989; Jones and Birney 1988).

The home range of the meadow vole varies in size with season, habitat, and population size (Jones and Birney 1988). Populations tend to fluctuate drastically every two to five years, with peak population density levels exceeding 100 voles per acre (Barbour and Davis 1974; Jones and Birney 1988). Activity occurs during both day and night, and throughout the year, although it is greatest at dawn and dusk (Barbour and Davis 1974). Well-worn intersecting runways under vegetative cover are distinctive of meadow vole inhabitation (Jones and Birney 1988). Elaborate spherical nests are commonly built aboveground in the center of a tussock of grass, although underground nests are also built in drier areas (Barbour and Davis 1974; Jones and Birney 1988).

The meadow vole is herbivorous, feeding primarily on grasses, sedges, legumes, tubers, and roots (Merritt 1987); however, insectivory and cannibalism have been reported in some individuals (Barbour and Davis 1974; Hoffmeister 1989). Bluegrass (*Poa* sp.) is a major component of the diet in some regions (Jones and Birney 1988; Hoffmeister 1989). This species hoards food for the winter in above- and below-ground caches (Merritt 1987).

The meadow vole is one of the most prolific mammals, producing litter after litter in rapid succession (Barbour and Davis 1974). Breeding occurs during the warmer months of the year (Jones and Birney 1988). The gestation period is about 21 days with litter sizes ranging from 1 to 11 young (averaging four to seven) (Barbour and Davis 1974; Jones and Birney 1988). The helpless young mature rapidly and may breed by 25 days of age (Barbour and Davis 1974).

Meadow voles are preyed upon by nearly all species of predatory birds and mammals (Barbour and Davis 1974). These predators include owls, hawks, shrikes, bluejays, crows, foxes, weasels, mink, cats, raccoons, skunks, opossums, shrews, and snakes (Barbour and Davis 1974; Merritt 1987). Due to heavy predation, only a small proportion of the population exceeds sixty days of age (Schwartz and Schwartz 1981).

Exposure Profile

Adult meadow voles weigh from 20 to 65 grams (Merritt 1987; U.S. EPA 1993). The home range of this species varies from less than one acre to 3.2 acres (Merritt 1987). Therefore, it was assumed that a meadow vole could obtain 100 percent of its diet from the contaminated area (area use factor of 1), since the area comprising the on-site sampling locations was approximately 20 acres.

A food ingestion rate ranging from 0.30 to 0.35 g/g BW/day, and a mean water ingestion rate of 0.21 g/g BW/day is reported for this species (U.S. EPA 1993). To express these values in units of g/day, the highest reported food ingestion rate of 0.35 g/g BW/day and the water



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ingestion rate of 0.21 g/g BW/day were multiplied by the lowest reported body weight of 20 g to yield a food ingestion rate of 7.0 g/day and a water ingestion rate of 4.2 g/day (4.2 ml/day).

A soil ingestion rate of 2.4 percent of the total diet has been reported (Beyer *et al.* 1994) for the meadow vole. To express this value in units of g/day, the soil ingestion rate of 2.4 percent was multiplied by the food ingestion rate of 7.0 g/day to yield a soil ingestion rate of 0.17 g/day.

For the purposes of the food chain model in this risk assessment, it was assumed that 100 percent of the diet was comprised of plants.

## 3.0 ASSUMPTIONS

This risk assessment evaluates exposure to contaminants through food and incidental sediment/soil ingestion. The following conservative assumptions were made to conduct this risk assessment in the absence of site-specific data:

- The maximum of the contaminant levels measured in sediment, soil, or water collected on site was used in risk calculations.
- The maximum concentrations of COCs reported in sediment, soil, water, and biota were assumed to be present site-wide.
- An area use factor (AUF) of 1 was assumed for all species using the site for feeding.
- Contaminants were assumed to be 100 percent bioavailable.
- Dietary composition information was obtained from the literature for the receptor species. However, simplifications of complex diets were performed for the receptors.
- A literature search was conducted to determine the chronic toxicity of the contaminants of concern when ingested by the indicator species. If no toxicity values could be located for the receptor species, values reported for a closely related species were used. All studies were critically reviewed to determine whether study design and methods were appropriate. When values for chronic toxicity were not available, $LD_{50}$ (median lethal dose) values were used. For purposes of this risk assessment, a factor of 100 was used to convert the reported $LD_{50}$ to a No Observed Apparent Effect Level (NOAEL). A factor of 10 was used to convert a reported Lowest Observed Adverse Effect Level (LOAEL) to a NOAEL. and a factor of 10 was used to convert a reported $LD_{50}$ to a LOAEL. If several toxicity values were reported for a receptor species, the most conservative value was used in the risk calculations regardless of toxic mechanism. Toxicity values obtained from long-term feeding studies were used in preference to those obtained from single dose oral studies. No other safety factors were incorporated into this risk assessment.
- In some cases, contaminant doses were reported as part per million contaminant in diet. These were converted to daily intake (in milligrams per kilogram body weight per day; mg/kg-day) by using the formula:

  Intake (mg/kg/day)=Contaminant Dose (mg/kg diet) x Ingestion Rate (kg/day) x 1/Bodyweight (kg)



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This conversion allows dietary toxicity levels cited for one species to be converted to a daily dose for a different species based on body weight. For the purposes of this risk assessment, incidental soil/sediment ingestion was also included in the calculation to determine the total daily intake for the receptor species. This daily dose may then be used to evaluate the risk to other species if no specific toxicity data are available for a [illegible] receptor.

- Some contaminants of concern (e.g. aluminum) are not food chain accumulators, but instead are direct toxins when ingested at the presented levels.

## 4.0 EFFECTS PROFILE

Many contaminants detected at the Dry Run Creek site do not have benchmarks. This excluded them from further consideration in this risk assessment, but does not exclude them as potential contaminants of concern. Based on the results of the preliminary risk assessment, the following compounds were considered COCs and their toxic effects are presented next: fluoride, trichlorofluoromethane, aluminum, arsenic, beryllium, chromium, copper, iron, lead, manganese, nickel, vanadium, and zinc. Based on the chemistry results, these compounds will be further evaluated using food chain accumulation models. Contaminants exceeding their respective benchmarks are assumed to be affecting receptor species and negatively impacting species, populations, and communities in the aquatic and terrestrial ecosystems at the Dry Run Creek site.

### 4.1 Fluoride

Maurer et al (1990) identified skeletal and dental abnormalities in rats that were exposed to sodium fluoride for a period of 99 weeks. The LOAEL identified in this study was 4 mg Fl/kg BW/day. A NOAEL was calculated from the LOAEL using an accepted conversion factor of 10. Based on these results, a LOAEL of 4mg/kg BW/day and an estimated NOAEL of 0.4/kg BW/day will be used to evaluate the risk posed by fluoride mammalian receptors

Fleming et al. (1987) found significant growth rate reduction in European starling fed a diet containing as low as 13 mg Fl/kg BW/day. No effects were observed at 10 mg Fl/kg BW/day. As such, this risk assessment will estimate fluoride related risk using a LOAEL of 13 mg/kg BW/day and a NOAEL of 10 mg/kg BW/day.

### 4.2 Organofluorides

No studies pertaining to the dietary toxicity of trichlorofluoromethane or any other fluorinated organic compound was found in the literature.

### 4.3 Aluminum

Dixon et al. (1979) conducted a study that evaluated the reproductive success of rats exposed to aluminum in drinking water for 90 days prior to breeding. The highest dose administered was 77.5 milligrams per kilogram body weight per day (mg/kg BW/day) and did not result in reproductive abnormalities. Lal et al. (1993) conducted a 180-day drinking water study in which rats were exposed to 55 mg/kg BW/day of aluminum. At this dose, behavioral effects were observed, including a significant reduction in spontaneous locomotor activity and significant deficits in acquisition and retention of learned responses. Based on these results, a LOAEL of 55 mg/kg BW/day and an estimated NOAEL of 5.5 mg/kg BW/day will be used to evaluate the risk posed by aluminum to mammalian receptors (Table 1).



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No effects were observed when Japanese quail were fed a diet containing 0.05 percent (84 mg/kg BW/day) aluminum for four weeks (Hussein et al. 1988). When quail were fed a diet containing 0.1 percent (165 mg/kg BW/day) aluminum, a decrease in egg shell breaking strength was observed. Finally, when quail were fed a diet containing 0.15 percent (257 mg/kg BW/day) aluminum, a decrease in body weight, egg shell strength, and egg shell production was observed. A 48-day feeding study using chickens concluded that dietary levels of 28.4 mg/kg BW/day aluminum resulted in a decrease in weight gain, feed intake, and plasma inorganic phosphorus, as well as an increase in plasma calcium (Hussein 1990). However, only the altered metabolism of calcium and phosphorus could be attributed to the direct effects of aluminum. The associated NOAEL for this effect is 22.8 mg/kg BW/day. Because a range of concentrations were used and the endpoints were ecologically significant and related to the dose, the study by Hussein et al. (1988) was used to the develop the NOAEL and LOAEL values. A NOAEL of 84 mg/kg BW/day and a LOAEL of 165 mg/kg BW/day will be used to evaluate the risk posed by aluminum to avian receptors (Table 1).

4.4 Arsenic

Several studies were located which determined the effects of As to mammals. A study conducted on cats indicated that a chronic oral toxicity dose was 1.5 mg/kg BW/day (Pershagen and Vahter 1979). In addition, National Resources Council of Canada (1978) states that mammals in general have oral $LD_{50}$s that range from 10 to 50 mg/kg of lead arsenate. A study conducted on mice indicated an oral dose $LD_{50}$ of 39.4 mg/kg BW/day and an oral dose $LD_0$ of 10.4 mg/kg BW/day after 96 hours (NAS 1977). For the purposes of this risk assessment, the chronic value for the cat was used to calculate HQs for mammals (1.5 mg/kg BW/day). This value was converted to a NOAEL by dividing by a factor of 10.

Eisler (1988a) reviewed several studies in which the toxicity of inorganic arsenicals were measured. Inorganic As is more mobile than organic As and may pose greater risk by leaching into surface water. Studies were also described in which organoarsenical compounds were measured. Studies indicate that sensitive species include the California quail (single oral dose $LD_{50}$ of 47.6 mg/kg BW/day) (Hudson et al. 1984) and chicken (single oral dose $LD_{50}$ of 33 mg/kg BW/day) (NAS 1977). For the purposes of this risk assessment, a value of 3.3 mg/kg BW/day was used to determine the HQ to birds. This value was converted to a NOAEL by dividing by a factor of 10.

4.5 Beryllium

Two separate chronic dietary exposure studies using rats reported similar musculoskeletal effects. Guyatt et al. (1933) fed large amounts of beryllium carbonate to rats at concentrations of 10, 20, 40, 80, 160, and 240 mg/kg BW/day. Rats from all exposure levels developed rickets, with the fragility of the bones varying directly with the exposure concentration. Similar results were reported by Jacobson (1933) who reported severely weakened bones in rats fed beryllium carbonate at dietary levels of 121 and 242 mg/kg BW/day.

For this risk assessment, a dietary exposure level of 10 mg/kg BW/day was used to estimate risk of beryllium to the short-tailed shrew. A NOAEL of 0.10 mg/kg BW/day was derived from this LOAEL using an accepted conversion factor of 10.

No studies pertaining to the dietary toxicity of beryllium to avian receptors were found in the literature.

4.6 Chromium



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Heinz and Haseltine (1981) exposed 2- to 3-year old breeding pairs of black ducks (*Anas rubripes*) to a diet containing 0, 20, or 200 mg/kg, wet weight, (0. 2.77, or 27.77 mg/kg BW/day) of $Cr^{+3}$ as chromium potassium sulfate [$CrK(SO_4)_2 \cdot 12H_20$] for a period of approximately five months, until the onset of egg-laying by the females. Hatched ducklings were then fed a mash diet containing the same Cr concentrations that the parents were fed. Seven-day old chicks were tested for avoidance behavior in response to a fright stimulus. None of the Cr concentrations resulted in alteration of avoidance behavior.
However, Haseltine et al. (1985), in an unpublished study reported by Eisler (1986a) notes that black duck ducklings suffered reduced survival and altered growth patterns when exposed to 10 mg/kg and 50 mg/kg of an unspecified $Cr^{+3}$ compound in their diets. The percent reduction in survival and a detailed explanation of the altered growth patterns were not available in this unpublished study.

For the purposes of this risk assessment, dietary levels of 10 mg/kg (1 mg/kg BW/day) of Cr in prey was used as a LOAEL for the avian species. However, due to the conflicting results, a NOAEL was derived from the same study in which the LOAEL was selected to maintain a degree of consistency regarding the Cr species evaluated. A NOAEL of 0.1 mg/kg BW/day was derived from the LOAEL using a conversion factor of 10.

A study conducted with dogs indicated that 2.5 mg/kg/day of $Cr^{+6}$ ingested in the diet caused death (Steven et al. 1976). For the purposes of this risk assessment, a LOAEL of 0.25 and a NOAEL of 0.025 were used for the red fox, raccoon, and mink.

4.7 Copper

One study was located which determined the effects of ingestion of Cu to mammalia species. An oral dose of 100 mg/kg/day to a dog caused death (OHMD 1987). For the purposes of this risk assessment, a LOAEL of 10 mg/kg/day was used and a NOAEL of 1 mg/kg/day were used for the exposure of mammals.

Several studies were located which determined the effects of Cu on chickens. A dose of 350 mg/kg (61.3 mg/kg/day) caused a significant decrease in growth and food consumption (Smith 1969). Another study found that a dose of 325 mg/kg (23.5 mg/kg/day) caused respiratory problems (Hatch 1978). Assuming that respiratory problems are an acute effect, a LOAEL of 2.35 mg/kg/day and a NOAEL of 0.235 mg/kg/day were used to determine risk to avian species.

4.8 Iron

No studies pertaining to the dietary toxicity of iron to mammalian or avian receptors were found in the literature.

4.9 Lead

The gastric motility of adult male and female red-tailed hawks fed 0.82 and 1.64 mg Pb/kg BW/day in a single oral dose was evaluated through the use of surgically implanted transducers for a period of three weeks following the dose. Neither concentration had any effect on gastric contractions or egestion of undigested material pellets (Lawler et al. 1991).

A study conducted on red-tailed hawk found that 3 mg/kg/day of Pb caused the clinical symptoms of Pb poisoning (Reiser and Temple 1981). A similar study found that 3 mg/kg/day fed to starlings caused a reduction in muscle condition and altered their feeding activity (Osborne et al. 1983). For



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the purposes of this risk assessment, a LOAEL of 3 mg/kg/day was used to determine risk to avian species and a NOAEL of 0.3 was used.

Several studies were located which determined the effects of Pb ingestion to mammals. A study conducted on mice indicated that 1.5 mg/kg/day of Pb caused a reduction in success of implanted ova (Clark 1979). Another study found that 2.2 mg/kg/day caused a reduction in the frequency of pregnancy when the dose was administered 3 to 5 days following mating (Clark 1979). For the purposes of this risk assessment, a NOAEL of 0.15 mg/kg/day and a LOAEL of 1.5 mg/kg/day were used to determine risk to mammals.

4.10 Manganese

The effects levels for manganese toxicity vary widely, most likely attributable to the form of manganese tested. Rats exposed to 13 mg/kg BW/day of manganese as Mn3O4 in their diet for 224 days exhibited reduced testosterone levels (Laskey et al. 1982). In mice, a dietary level of 140 mg/kg BW/day, also of Mn3O4 for 90 days resulted in decreased activity (Gray and Laskey 1980). A much higher exposure concentration of 2,300 mg/kg BW/day of manganese as MnCl2 resulted in reduced dopamine levels (Gianutsos and Murray 1982).

In contrast, levels as high as 930 mg/kg BW/day of manganese as MnSO4 for 103 weeks had no effect on the respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, dermal, and ocular systems of mice (Hejtmancik et al. 1987).

For this risk assessment, a dietary exposure level of 13 mg/kg BW/day will be used as a LOAEL to estimate risk of manganese to the selected mammalian receptor. A NOAEL of 1.3 mg/kg BW/day was derived from this LOAEL using an accepted conversion factor of 10.

No studies pertaining to the dietary toxicity of manganese to an avian receptor were found in the literature.

4.11 Nickel

Several studies were available which determined the effects of Ni ingestion to mammals. Wistar rats fed Ni sulfate indicated a NOAEL of 187.5 mg/kg/day to most systems except for body weight. This level of Ni sulfate caused a 27 to 29 percent decreased body weight (Ambrose et al. 1976). In a similar study with a beagle, a NOAEL of 62.5 mg/kg/day was noted (Ambrose et al. 1976). For the purposes of this risk assessment a NOAEL of 62.5 mg/kg/day was used to determine risk to mammals. This value was converted to a LOAEL of 625.0 mg/kg/day by multiplying the NOAEL by a factor of 10.

No studies were available that determined the dose of Ni to avian species. Therefore, the risk to avian species from ingested Ni will not be determined.

4.12 Vanadium

Gavage studies in mice have found an LC50 of 31 mg Vn/Kg diet (Schroeder and Balassa 1967). This dose was converted to a LOAEL of 3.1 mg/kg and a NOAEL of 0.31 using an accepted factor of 10 conversion. This food dose was converted to a daily dose by multiplying the LOAEL or NOAEL concentration by an ingestion rate commonly observed in mice (0.003 kg of food/day) and then by the inverse of the body weight (0.025 kg)(RTECS 1985). This calculation resulted in a LOAEL of 0.372



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mg V/kg BW/day and a NOAEL of 0.0372 mg V/kg BW/day. These values will be used to estimate risk to mammalian receptors in this risk assessment.

Rosomer (1960) exposed chickens to varying concentrations of vanadium. The study involved feeding 4 replicates of 13 chickens varying concentrations of vanadium for a period of 21 days. The study found that a dietary level of 40 mg/kg in the diet resulted in a marked depression in weight gain and efficiency of food utilization. At levels of 200 mg/kg, mortality was noted in all test chickens. The authors reported that a dietary level of 20 mg/kg could be tolerated with no resultant toxic effects. This dietary level was converted to a daily dose as above by multiplying the dietary concentration by a representative chicken ingestion rate (0.140 kg/day) and then by the inverse of the body weight (0.800 kg)(RTECS 1985). This calculation resulted in LOAEL of 7 mg V/kg BW/day and a NOAEL of 3.5 mg V/kg BW/ day. These values will be used to estimate risk to avian receptors.

4.13 Zinc

Several studies were available which determined the effects of ingested Zn to birds. A concentration of 144.5 mg/kg/day caused a decrease in growth and anemia in chickens (Stahl et al. 1989). In a similar study conducted on chickens, a concentration of 361 mg/kg/day caused a reduction in body weight (Dean et al. 1991). In a study conducted on Japanese quail, a concentration of 139 mg/kg/day caused 7 percent mortality in chicks and reduced food intake (Hill and Camardese 1986). For the purposes of this risk assessment, a LOAEL of 139 mg/kg/day was used to determine the effects to avian species. This value was converted to a NOAEL of 13.9 mg/kg/day by dividing the LOAEL by a factor of 10.

A study conducted on dogs, indicated that 1,000 mg/kg (25 mg/kg/day) caused no effects after one year (NAS 1979). For the purposes of this risk assessment, a LOAEL of 250 and a NOAEL of 25 were used to determine risk to the fox and the mouse. In a study conducted on ferrets, a dose of 370 mg/kg day caused a decrease in food intake and weight loss. Because the ferret is similar to the mink, a LOAEL of 370 mg/kg/day was used and a NOAEL of 37 was used to determine risk to the mink.

## 5.0 RISK CHARACTERIZATION

The following method was used to calculate risk. To estimate the risk to wildlife in the model systems utilizing the Dry Run Creek site, implications of the exposure concentrations need to be determined. The HQ method (U.S. EPA 1989, Barnthouse et al. 1986) compares exposure concentrations to ecological endpoints such as reproductive failure or reduced growth. The comparisons are expressed as ratios of potential intake values to population effect levels, or:

$$\text{Hazard Quotient (HQ)} = \frac{\text{Mean Exposure Concentration}}{\text{No Observed Adverse Effect Level (NOAEL)}}$$

A HQ greater than one indicates that exposure to the contaminant has the potential to cause adverse effects in the organism. A HQ less than one does not indicate a lack of risk. The HQ should be interpreted based on the severity of the effect reported. The results of the risk characterization are presented next.

### 5.1 Benthic Invertebrate Community Structure and Function

The benthic invertebrate community in Dry Run appears to be at risk for two reasons. The benthic community survey showed a decrease in community taxonomic diversity and abundance in Dry Run as compared to the Reference stream. Since land use and available habitat are the same along both



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streams, the decrease in diversity and abundance in Dry Run may be attributed to contamination present in sediments in Dry Run. In addition, the amphipod toxicity test clearly demonstrates that acute exposure sub-lethal effects can be produced in the benthic community, especially under conditions present in Tributary A and in Area II. The observed negative growth effect was significantly negatively correlated with fluoride, aluminum, calcium, magnesium, nickel, potassium, and sodium. Further, there were strong negative associations between the growth endpoint and chromium, copper, lead, and zinc concentrations, although the relationships were not significant at the 0.10 level. Since the sediments closer the landfill along the whole Dry Run reach appear to be enriched with metals, the observed toxicity may represent a significant threat.

## 5.2 Soil Invertebrate Community Structure and Function

The soil invertebrate community does not appear to be at risk based on current soil conditions at Dry Run. The earthworm toxicity test identified no problems with survival or growth.

## 5.3 Fish Communities

The fish community at Dry Run may be at risk. Results of the fathead minnow toxicity bioassay show that water conditions in Upper Tributary A induce mortality to larval fish. This mortality could not directly be associated with a suite of contaminants as in the amphipod test, but survival was negatively correlated with potassium concentrations, however this correlation was not statistically significant at the 0.10 level. There was a significant positive correlation between fathead survival and iron concentrations in the filtered water samples. Low species diversity and abundance observed during the electroshocking effort may be reflected by the results of the toxicity test.

## 5.4 Worm-eating Birds

A conservative risk assessment model based on wet-weight concentrations of contaminants for the Dry Run Creek site has determined that worm-eating birds may be at risk due to ingestion of contaminated forage, soil, and water. The model predicts that aluminum, chromium, copper, lead, vanadium, zinc, and fluoride are risk factors based on conservative inputs. By default, beryllium, iron, manganese, nickel, and trichlorofluoromethane are risk factors due to lack of toxicological benchmarks for these compounds. Food chain risk calculations and resultant hazard quotients are presented in Table 42.

## 5.5 Carnivorous Birds

A conservative risk assessment model based on wet-weight concentrations of contaminants for the Dry Run Creek site has determined that carnivorous birds may be at risk due to ingestion of contaminated forage, soil, and water. The model predicts that aluminum, chromium, copper, lead, zinc, and fluoride are risk factors based on conservative inputs. By default, beryllium, iron, manganese, nickel, vanadium, and trichlorofluoromethane are risk factors due to lack of toxicological benchmarks for these compounds. Food chain risk calculations and resultant hazard quotients are presented in Table 42.

## 5.6 Carnivorous Mammals (Terrestrially feeding)

A conservative risk assessment model based on wet-weight concentrations of contaminants for the Dry Run Creek site has determined that carnivorous mammals may be at risk due to ingestion of contaminated forage, soil, and water. The model predicts that aluminum, chromium, copper, lead,



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manganese, vanadium, and fluoride are risk factors based on conservative inputs. By default, iron and trichlorofluoromethane are risk factors due to lack of toxicological benchmarks for these compounds. Food chain risk calculations and resultant hazard quotients are presented in Table 42.

5.7 Piscivorous Mammals

A conservative risk assessment model based on wet-weight concentrations of contaminants for the Dry Run Creek site has determined that piscivorous mammals may be at risk due to ingestion of contaminated forage, soil, and water. The model predicts that chromium, manganese, and fluoride are risk factors based on conservative inputs. Trichlorofluoromethane is not considered a risk factor because it was not detected in site sediments. Food chain risk calculations and resultant hazard quotients are presented in Table 42.

5.8 Omnivorous Mammals

A conservative risk assessment model based on wet-weight concentrations of contaminants for the Dry Run Creek site has determined that omnivorous mammals may be at risk due to ingestion of contaminated forage, soil, and water. The model predicts that arsenic, chromium, copper, manganese, vanadium, and fluoride are risk factors based on conservative inputs. Trichlorofluoromethane is not considered a risk factor because it was not detected in site sediments. Food chain risk calculations and resultant hazard quotients are presented in Table 42.

5.9 Insectivorous Mammals

A conservative risk assessment model based on wet-weight concentrations of contaminants for the Dry Run Creek site has determined that insectivorous mammals may be at risk due to ingestion of contaminated forage, soil, and water. The model predicts that aluminum, chromium, copper, lead, manganese, vanadium and fluoride are risk factors based on conservative inputs. By default, iron, and trichlorofluoromethane are considered risk factors due to lack of toxicological benchmarks for these compounds. Food chain risk calculations and resultant hazard quotients are presented in Table 42.

5.10 Herbivorous Mammals

A conservative risk assessment model based on wet-weight concentrations of contaminants for the Dry Run Creek site has determined that herbivorous mammals may be at risk due to ingestion of contaminated forage, soil, and water. The model predicts that aluminum, chromium, lead, manganese, and fluoride are risk factors based on conservative inputs. By default, iron, vanadium, and trichlorofluoromethane are considered risk factors due to lack of toxicological benchmarks for this compound. Food chain risk calculations and resultant hazard quotients are presented in Table 42.

## 6.0 UNCERTAINTY ANALYSIS

There are factors inherent in the risk assessment process which contribute to uncertainty and need to be considered when interpreting results. Major sources of uncertainty include natural variability, error, and insufficient knowledge.

Error can be introduced by use of invalid assumptions in the conceptual model. Conservative assumptions were made in light of the uncertainty associated with the risk assessment process. This was done to minimize the possibility of concluding that no risk is present when a threat actually does exist (e.g., elimination of false negatives). Whenever possible, risk calculations were based on conservative values. For example, NOAELs



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used to calculate HQs were the lowest values found in the literature, regardless of toxic mechanism.

An important contributor to uncertainty is the incompleteness of the data or information upon which the risk assessment is based. Risk calculations are based on maximum COC levels in sediment, water, and soil samples.

Literature values for the toxicity of COCs were not available for all receptor species. An attempt was made to identify studies using closely related species to make risk estimates for the selected receptors. Species respond differently to exposure to toxins; responses to COCs by the indicator species may be different from species for which the toxicity data are reported. Methodological problems were also apparent in several of the studies from which NOAELs were obtained. Unfortunately, studies which were more suitable for this assessment were not found for some of the selected receptors.

A literature search was conducted to identify appropriate NOAELs and LOAELs for this risk assessment. The values used to calculate HQs were the lowest values found in the literature. In many of the studies reviewed, adverse effects were observed at the lowest exposure concentration. This made it impossible to identify appropriate NOAELs for some receptors. In these cases, a factor of 10 was used to convert the LOAEL to a NOAEL, which adds uncertainty to the NOAEL-based calculations.

Doses in toxicological studies can be reported in units of mg contaminant/kg diet, or in units of mg contaminant/kg body weight/day. All doses reported as mg/kg in diet were converted to units of mg/kg BW/day. If body weights were reported for the test animals in a given study, these values were used for making this conversion. Otherwise, the body weight and ingestion rate for the species reported in other literature sources were used.

Another source of uncertainty arises from the use of toxicity values reported in the literature which are derived from single-species, single-contaminant laboratory studies. Prediction of ecosystem effects from laboratory studies is difficult. Laboratory studies cannot take into account the effects of environmental factors which may add to the effects of contaminant stress. NOAELs were generally selected from studies using single contaminant exposure scenarios. Species utilizing the Dry Run Creek site are exposed to a variety of contaminants.

There is very little information available in the literature regarding the rates of incidental soil/sediment ingestion for wildlife species. In this risk assessment, most of these values were based on estimates reported for species similar to the indicator species.

Exposure concentrations were calculated for each target receptor species based on levels of contaminants detected in site media, daily food ingestion rates, incidental soil/sediment ingestion rates, and body weight reported in the literature.

This ecological risk assessment was conducted with the intent of completing a baseline risk assessment. In this risk evaluation it is concluded that a "potential ecological risk" exists if the HQ calculated from the maximum area concentration and the NOAEL equals or exceeds one. Within the calculation spreadsheets, alternate calculations were made using LOAEL toxicity benchmarks.

## 7.0 CONCLUSIONS

### 7.1 Benthic Invertebrate Community Structure and Function

Data from both the benthic survey and the toxicity tests indicate that fluoride and metal contamination may be a significant problem in Dry Run. Numerous fish kills historically reported in Dry Run also



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provide evidence for potential effects on the benthic community.

7.2 Soil Invertebrate Community Structure and Function

The structure and function of the soil invertebrate community does not appear to be at risk under current conditions found at the Dry Run Creek site. However, since earthworms comprise a significant amount of the forage base of some organisms (e.g. American robins, short-tail shrews, etc.), food chain problems may result from contaminants being tied up in the earthworm tissue. Based on our food chain models, it appears that this may be the case.

7.3 Fish Communities

It was shown through the results of the fathead minnow bioassay, that larval fish were susceptible to contamination currently present near the landfill outfall at Dry Run. This finding is further supported by the results of the benthic invertebrate toxicity tests, where toxicity was observed at the same location. Negative effects of contaminants on the benthic community may directly affect fish communities, in that a portion of the fish food base in Dry Run (i.e. benthic invertebrates) may also be removed from the system. Reports of historical fish kills are also an important piece of evidence that suggests an ecological risk. In addition, high levels of metals were noted in the fish, which may present problems to upper level consumers due to dietary toxicity.

7.4 Worm-eating Birds

Results of the food chain model for worm-eating birds such as the American robin indicate a potential risk due to metals, fluoride, and trichlorofluoromethane. This risk is associated with these contaminants in the soil and/or in earthworm tissue. Reports of historical wildlife kills also suggest ecological risk to avian receptors.

7.5 Carnivorous Birds

Results of the food chain model for carnivorous birds such as the red-tail hawk indicate a potential risk due to metals, fluoride, and trichlorofluoromethane. This risk is associated with these contaminants in the soil and/or in small mammal tissue. Reports of historical wildlife kills also suggest ecological risk to avian receptors.

7.6 Carnivorous Mammals

Results of the food chain model for terrestrially feeding carnivorous mammals such as the red fox indicate a potential risk due to metals, fluoride, and trichlorofluoromethane. This risk is associated with these contaminants in the soil and/or in small mammal tissue. Reports of historical wildlife kills also suggest ecological risk to mammalian receptors.

7.7 Piscivorous Mammals

Results of the food chain model for piscivorous mammals such as the mink indicate a potential risk due to metals, fluoride, and trichlorofluoromethane. This risk is associated with these contaminants in the soil and/or in fish tissue. Reports of historical wildlife kills also suggest ecological risk to mammalian receptors.

7.8 Omnivorous Mammals



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Results of the food chain model for omnivorous mammals such as the raccoon indicate a potential risk due to metals, fluoride, and trichlorofluoromethane. This risk is associated with these contaminants in the soil and/or in fish tissue. Reports of historical wildlife kills also suggest ecological risk to mammalian receptors.

### 7.9 Insectivorous Mammals

Results of the food chain model for insectivorous mammals such as the short-tail shrew indicate a potential risk due to metals, fluoride, and trichlorofluoromethane. This risk is associated with these contaminants in the soil and/or in earthworm tissue. Physiological abnormalities, specifically the tooth structure of the shrews taken on site, further suggest ecological risk. In addition to the direct potential risk for the shrews, some of these animals had high concentrations of metals and fluoride in their tissues. This could present problems to organisms that feed on shrews and other small mammals on the site due to dietary toxicity. Reports of historical wildlife kills also suggest ecological risk to mammalian receptors.

### 7.10 Herbivorous Mammals

Results of the food chain model for herbivorous mammals such as the meadow vole indicate a potential risk due to metals, fluoride, and trichlorofluoromethane. This risk is associated with these contaminants in the soil and/or in plant tissue. In addition to the direct potential risk for the voles; some of these animals had high concentrations of metals and fluoride in their tissues. This could present problems to organisms that feed on voles and other small mammals on the site due to dietary toxicity. Reports of historical wildlife kills also suggest ecological risk to mammalian receptors.

## 8.0 SUMMARY

During the past several years, a farmer who grazes his cattle along the reach of Dry Run Creek, has reported severe abnormalities in his herd. These abnormalities have included an increased incidence of stillborn calves, blindness in newborn and adult cattle, erratic behavior, stiffness of gait in adult cattle, abnormal posture, mottled teeth, and a high mortality rate across all age classes of his herd. In addition to problems with his herd, the farmer and others have also reported numerous fish kills in Dry Run, and wildlife kills (e.g. deer) for animals drinking from Dry Run. The results of this risk assessment support his assertion that effluent from the Dry Run Creek landfill may be having adverse effects on the ecological communities that inhabit the old field, deciduous forest, meadow, stream, and riparian habitats that are present on the site. These effects may be related to enriched levels of metals, fluoride, and trichlorofluoromethane that appear to be resultant of the landfill drainage. At a minimum, the symptoms manifest by the herd are characteristic of fluoride toxicity, and consistent with the conclusions of the risk assessment. In addition to the compounds that were studied in this risk assessment, numerous other compounds were present in Dry Run (specifically those identified as TICs or Tentatively Identified Compounds in the BNA scan) that could not be accurately identified. These compounds may also present a threat to the system, an certainly merit further investigation. The DuPont landfill that drains into Dry Run is the only apparent source of trichlorofluoromethane in soils adjacent to the stream.



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## LITERATURE CITED


Agency for Toxic Substances and Disease Registry (ATSDR). 1990. *Toxicological Profile for Aluminum.* Prepared by Sciences International Inc. Under U.S. Department of Health and Human Services Contract No. 205-93-0606. Research Triangle Park, NC.

Agency for Toxic Substances and Disease Registry (ATSDR). 1990. *Toxicological Profile for Manganese.* Prepared by Sciences International Inc. Under U.S. Department of Health and Human Services Contract No. 205-93-0606. Research Triangle Park, NC.

Agency for Toxic Substances and Disease Registry (ATSDR). 1991. *Toxicological Profile for Vanadium.* Prepared by Sciences International Inc. Under U.S. Department of Health and Human Services Contract No. 205-93-0606. Research Triangle Park, NC.

Agency for Toxic Substances and Disease Registry (ATSDR). 1993. *Toxicological Profile for Beryllium.* Prepared by Sciences International Inc. Under U.S. Department of Health and Human Services Contract No. 205-93-0606. Research Triangle Park, NC.

Agency for Toxic Substances and Disease Registry (ATSDR). 1996. *Toxicological Profile for Nickel.* Prepared by Sciences International Inc. Under U.S. Department of Health and Human Services Contract No. 205-93-0606. Research Triangle Park, NC.

Ambrose, A.M., P.S. Larson. and J.F. Borzelleca. 1976. "Long-term toxicological assessment of nickel in rats and dogs." *J. Food. Sci. Technol,.*13:181-187.

Barbour, R.W. and W.H. Davis. 1974. *Mammals of Kentucky.* Lexington, KY: University of Kentucky Press. 322p.

Barnthouse, L.W., G.W. Suter, S.M. Bartell, J.J. Beauchamp, R.H. Gardner, E. Linder, R.V. O'Neill and A.E. Rosen. 1986. *User's Manual for Ecological Risk Assessment.* Publication Number 2679. ORNL-6251. Environmental Services Division, Oak Ridge National Laboratory, Oak Ridge, TN.

Beyer, W.N., E.E. Conner, and S. Gerould. 1994. "Estimates of Soil Ingestion by Wildlife." *J. Wildl. Manage.*, 58(2):375-382.

Brooks, L. 1988. "Inhibition of NADPH-cytochrome c reductase and attenuation of acute diethylnitrosamine hepatotoxicity by copper." Ph.D. Dissertation, Rutgers University, New Brunswick, N.J

Bull, J. And J. Farrand, Jr. 1977. *The Audubon Society Field Guide to North American Birds, Eastern Region.* New York, NY: Alfred A. Knopf, Inc.

Burton, P. 1989. *Birds of Prey of the World.* New York, NY: W.H. Smith Publishers.

Byerrum, R.U., R.E. Eckardt, L.L. Hopkins. 1974. *Vanadium.* Washington, D.C. National Academy of Sciences.




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Calder, W.A., and E.J. Braun. 1983. "Scaling of osmotic regulation in mammals and birds." *American Journal of Physiology*, 244: R601-R606.

Castranova, V., L. Bowman, and J.R. Wright. 1984. "Toxicity of Metallic Ions in the Lung: Effects on Alveolar Macrophages and Alveolar Type II Cells." *J. Toxicol. Environ. Health* 13:845-856.

Clark, D.R. Jr. 1979. "Lead Concentrations: Bats vs. Terrestrial Mammals Collected near a Major Highway." *Environ. Sci. Tech.*, 13:338-341.

Dean, C.E., B.M. Hargis and P.S. Hargis. 1991. "Effects of Zinc Toxicity on Thyroid Function and Histology in Broiler Chicks." *Toxicol. Letters*, 57:309-318.

DeGraaf, R.M. and D.D. Rudis. 1983. *New England Wildlife: Habitat, Natural History, and Distribution.* Amherst, MA. The University of Massachusetts Press.

DeMayo, A., M.C. Taylor and K.W. Taylor. 1982. "Effects of Copper on Humans, Laboratory and Farm Animals, Terrestrial Plants and Aquatic Life." *CRC Critical Reviews in Environmental Control*, 12(3):183-255.

Dixon, R.L., R.J. Sherins, and I.P. Lee. 1979. "Assessment of Environmental Factors Affecting Male Fertility." Environmental Health Perspectives. 30:53-68.

Edwards, C.A. and J.R. Lofty. 1977. *The Biology of Earthworms.* John Wiley and Sons, New York, NY.

Ehrlich, P.R., D.S. Dobkin, and D. Wheye. 1988. *The Birders Handbook.* Simon and Schuster, Fireside. New York. 785 pp.

Eisler, R. 1986. "Chromium Hazards to Fish, Wildlife, and Invertebrates: a Synoptic Review." *U.S. Fish and Wildlife Service Biological Report*, 85(1.86). 60p.

Eisler, R. 1988a. "Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." *U.S. Fish and Wildlife Service Biological Report* 85(1.12).

Eisler, R. 1988b. "Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review." *United States Fish and Wildlife Biological Report.* 85(1.14).

Evan, A.P. and W.G. Dail. 1974. "The Effects of Sodium Chromate on the Proximal Tubules of the Rat Kidney." *Lab. Invest.*, 30:704-715 In: Steven, J.D., L.J. Davies, E.K. Stanley, R.A. Abbott, M. Inhat, L. Bidstrup, and J.F. Jaworski. 1976. "Effects of Chromium in the Canadian Environment." *Nat. Res. Counc. Can.*, NRCC No. 15017. 168p.

Fleming, W.J. and C.A. Schuler. 1988. "Influence of the Method of Fluoride Administration on Toxicity and Fluoride Concentrations in Japanese Quail." *Environmental Toxicology and Chemistr.* 7(10):841-846.

Fleming, W.J., C.E. Grue, C.A. Schuler, and C.M. Bunck. 1987. "Effects of Oral Doses of Fluoride on Nestling European Starlings." *Arch. Env. Cont. Toxicol.* 16(4): 483-490.

Gianutsos, G. and M.T. Murray. 1982. "Alterations in Brain Dopamine and GABA Following Inorganic or Organic Manganese Administration." *Neurotoxicol.* 3:75-81.

Goldman, E.A. 1950. *Raccoons of North and Middle America.* Washington, DC: U.S. Fish and Wildl. Service.



000103

USFW 0641

*Effects on Aquatic Biota: 1994 Revision.* ES/ER/TM-96/R1, Martin Marietta Energy Systems, Inc.

Suttie, J.W. 1977. "Effects of Fluoride on Livestock." *Journal of Occupational Medicine.* 19(1):40-48.

Tyson, E.L. 1950. "Summer Food Habits of the Raccoon in Southwest Washington." *J. Mammal.*, 31:448-449.

U.S. Environmental Protection Agency (U.S. EPA). 1981. *An exposure and risk assessment for arsenic.* Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA-440/4-85-005.

U.S. Environmental Protection Agency (U.S. EPA). 1985. *Ambient water quality criteria for arsenic.* Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C.

U.S. Environmental Protection Agency (U.S. EPA). 1985. *Methods for Measuring the Acute Toxicity of Effluents to Freshwater and Marine Organisms.* United States Environmental Protection Agency. EPA/600/4-85/013.

U.S. Environmental Protection Agency (U.S. EPA). 1989. *Risk Assessment Guidance for Superfund. Volume I.* Washington, D.C. EPA/540/1-89/002.

U.S. Environmental Protection Agency (U.S. EPA). 1992. *Ambient Water Quality Criteria for the Protection of Aquatic Life. Federal Register.* Volume 57. No. 246. December 22.

U.S. Environmental Protection Agency (U.S. EPA). 1993. *Wildlife Exposure Factors Handbook, Volume I of II.* United States Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/R-93/187a.

U.S. Environmental Protection Agency (U.S. EPA). 1994. *Methods for Measuring the Toxicity and Bioaccumulation of Sediment-Associated Contaminants with Freshwater Invertebrates.* United States Environmental Protection Agency. EPA/600/R-94/024.

U.S. Environmental Protection Agency (U.S. EPA). 1995. *Revised Region III BTAG Benchmark Values.* U.S. EPA Region III BTAG. Technical Support Section. Philadelphia, PA.

Van Zinderen Bakker and J.F. Jaworski. 1980. *Effects of Vanadium in the Canadian Environment.* Ottawa, Canada: National Research Council of Canada, Associate Committee Scientific Criteria for Environmental Quality.

Venugopal, B. and T.D. Luckey. 1978. *Metal Toxicity in Mammals: 2. Chemical Toxicity of Metals and Metalloids.* Plenum Press, New York, NY.

Wiebel, F.J., J.C. Leutz, L.Diamond and H.V. Gelboin. 1971. "Aryl Hydrocarbon (Benzo(a)pyrene) Hydroxylase in Microsomes from Rat Tissues: Differential Inhibition and Stimulation by Benzoflavones and Organic Solvents." *Arch. Biochem. Biophys.*, 144:78-86.

Wixson, B.G. and B.E. Davis. 1993. "Lead in Soil." *Lead in Soil Task Force, Science Reviews,* Northwood. 132 pp.

Woolson, E.A. 1975. Arsenical pesticides. ACS Ser 7:1 - 176 (*as cited in Eisler, 1994*).



009184

USFW 0642

FIGURE 1
Sampling Site Map
Dry Run Creek Site
Washington, Wood County, WV
November 1997
U.S. EPA Environmental Response Team Center
Response Engineering and Analytical Contract
68-C4-0022
W.O. # 03547.142.001.2273.01
Area 1
mammal
sediment
soil
vegetation
benthos
Area 2
mammal
sediment
soil
vegetation
water
fish
benthos
Area 3
mammal
sediment
soil
vegetation
water
fish
benthos
Area 4
mammal
sediment
soil
vegetation
water
fish
benthos
Ref.
mammal
soil
vegetation
Ref.
sediment
water
benthos
Trib. A
sediment
water
Trib. B
sediment
water
Landfill
Area
Lee Creek
sediment
water
Dry Run
Horseshoe Bend
Pine
Lamps Hollow
JEEP TRAIL
JEEP
2273fig1.cdr

99:600

# I N T E R O F F I C E  M E M O R A N D U M

Date: 14-Nov-1997 02:11pm EDT
From: DANIEL A. WEBER
WEBERDA
Dept: ENGINEERING POLYMERS
Tel No: 8-863-4415

TO: LYNWOOD K. IRELAND ( IRELAND )
TO: RONALD W MELOON ( MELOONRW )
TO: ROBERT L. RITCHEY ( RITCHERL )
TO: H. DAVID RAMSEY, JR. ( RAMSEY )
TO: David C. Harrison ( HARRISDC )

**Subject:** C-8 DATA FOR DOMESTIC WATER WELLS

FYI,

C-8 LEVELS IN THE EAST FILED DOMESTIC WATER SERVICE WELLS AND WEST FIELD (OLD LUBECK WELLFIELD) WELL #1 ARE SUMMARIZED BELOW:

**WEST WELLFIELD (OLD LUBECK WELLFIELD)**

WELL #1

| | | | |
|---|---|---|---|
| 5/93 | 0.6 PPB | 8 - 11 % | SURROGATE RECOVERY |
| | 2.1 PPB (RETEST) | 60 - 75 % | SURROGATE RECOVERY |
| 8/94 | 1.5 PPB | 30 - 38 % | SURROGATE RECOVERY |
| 5/97 | 7.9 PPB | 120 - 126 % | SURROGATE RECOVERY |

**EAST WELLFIELD**

WELL # 336

| | | | |
|---|---|---|---|
| 6/93 | 0.5 PPB | 52 - 76 % | SURROGATE RECOVERY |
| 4/96 | 0.48 PPB | 29 - 85 % | SURROGATE RECOVERY |
| 5/97 | 0.79 PPB | 36 - 41 % | SURROGATE RECOVERY |

WELL # 331

| | | | |
|---|---|---|---|
| 6/93 | 2.9 PPB | 55 - 78 % | SURROGATE RECOVERY |
| 4/96 | 0.52 PPB | 40 - 101 % | SURROGATE RECOVERY |
| 5/97 | 0.55 PPB | 43 - 45 % | SURROGATE RECOVERY |

WELL # 332

| | | | |
|---|---|---|---|
| 6/93 | 3.3 PPB | 69 - 87 % | SURROGATE RECOVERY |

SURROGATE RECOVERY PERTAINS TO THE PRACTICE OF ADDING COMPOUNDS WITH SIMILAR C8 ORGANIC STRUCTURE TO THE SAMPLE TO GAGE THE PER CENT RECOVERY OF THE ANALYTICAL METHOD USED TO DETERMINE THE C8 RESULT (IE, AN INDICATION OF HOW MUCH OF THE C-8 PRESENT IN THE SAMPLE WAS IDENTIFIED).

DAW000638

EID091600

KENNEDGL
12/08/97 09:41 AM

To: ZIPFEL, KOENINMC, CHAPMAGA
cc: COOKJC
Subject: C-8 MONKEY STUDY

MET WITH APME APFO GROUP LAST WEEK-MINUTES WILL COME WITH OFFICIAL ITEMS BUT HERE IS A QUICK PREVIEW:
1)D.FARRAR WILL WORK TO GET CONTRACTS ON PLACE BY YEARS END
2)6 1/2 SHARES (650,000) COMMITTED TO MONKEY AND RAT WORK
3)FINAL COST TO BE IN THAT REGION, 2 DETAILS INCLUDE ANALYTICAL WORK BY 3-M (EST 50,000) AND BIOCHEMICAL DETERMINATIONS BY HASKELL (AROUND 20,000). THIS WILL BE DETAILED THROUGH BY PAUL LEIDER--PERHAPS THE DOLLAR AMOUNTS CAN BE ADJUSTE. IT IS THE DESIRE OF THE TOX WORKING GROUP TO NOT GO ASK FOR MORE MONEY.
4)TIMING-MONKEY PILOT TO START IN FEB, 1998, RESULTS AVAILABLE MAY,1998.

MONKEY MAIN-TO START JUNE 1998, RESULTS AVAILABLE MAY 1999. WE DECIDED THAT THERE WAS LITTLE CHANCE THAT THE STUDY WOULD GENERATE ENOUGH SOLID INFORMATION TO ALLOW CONCLUSION AFTER 3 MONTHS--SO IT IS A 6 MONTH STUDY.
5)COMMUNICATION INTERFACE WILL NEED BE MAINTAINED AND EXPEDITED-ALL TOX MEMBERS MADE TIMING COMMITMENTS TO MEET THE ABOVE SCHEDULE-- THIS WILL INCLUDE 2 US AND 2 EUROPEAN MEETINGS DURING AND AFTER COMPLETION OF THE IN LIFE PHASES.

FURTHER DEAILS TO COME--WORKING GROUP WAS THE MOST COOPERATIVE TO DATE- LOOKS LIKE EVERYONE IS IN LINE TO GET AFTER THE ISSUE. CO NOTE THAT THE RISK ASSESSMENT WILL RELY HEAVILY ON THIS MONKEY, AND TO A LESSER EXTENT THE RAT, STUDIES AND HENCE WILL NOT BE COMPLETED IN 1998.

INTEROFFICE MEMORANDUM

Date: 17-Feb-1998 11:29am EDT
From: ROBERT L. RITCHEY
RITCHERL
Dept: EP - Env
Tel No: 863-4271

TO: See Below

Subject: EPA Visit to Dry Run Landfill

We hosted today a pre-scheduled visit by Mike Taurino, EPA Region III, to Dry Run Landfill.

Mike was assigned to EPA's Dry Run efforts several weeks ago. He is a Fund On-Scene Coordinator. As such, he gets involved under Superfund/CERCLA when there is a potential "removal" project. He explained that his role is dealing with situations that require immediate attention (thus removal) as opposed to their group that handles remedial responses which are longer term in nature. He would define what response is required to a given immediate need. Mike is a Civil Engineer with an Environmental Masters and has been with EPA five years.

What drew Mike into this Dry Run effort is the perception by EPA that our landfill may be having an impact on Dry Run Creek. The basis for this is obviously influenced by claims of historical cattle, fish, and other wildlife impacts which are reflected in the EPA risk assessment report on Dry Run. Mike indicated that he had read the conclusions (only) of this report and also has available to him the 104e information we submitted on Dry Run. With that background, he was predisposed to believe that there are problems at Dry Run Creek.

Presentations and Q&A sessions with Woody Ireland and Dan Weber were successful in casting serious doubt on the science and credibility of the EPA risk assessment. Mike at one point stated something like "so if I am deciding what actions are needed, it sounds like I really can't rely on this report." We did acknowledge that in 1995 we had an episode of "black water", but explained the cause, disassociated it from fluorides, explained our corrective actions and that it has not reoccurred since. We also discussed the numerous design improvements and additional monitoring and permit requirements which will be incorporated in our new permit when it is finally issued. We indicated our desire to begin working on those ASAP.

I believe Mike is of the opinion that what we have already done and what we plan to do under the new permit will resolve the issue. He reserved the right to decide otherwise, including a decision to do more study. However, he was clearly having trouble thinking of what else we could do and what else would be justified.

We took Mike to the Landfill and he got a good tour. He does not have a background in dealing with landfills, so we did most of the talking and he had few questions. Everything looked good. Dry Run Creek did not have the appearance of being adversely effected at all. As Dan summarized afterwards, Mike came to see ugly and he didn't.

Mike will be at our meeting with EPA next Monday in Philadelphia. I believe he can help stabilize EPA's unattached emotional reaction to



KLK001725

EID067733

some of the assertions and conclusions of the study. I believe based on this interaction today that it will be important to hit hard on the aquatic and mammalian tox issues and when we visit EPA.

Distribution:

TO: WILLIAM H. HOPKINS ( HOPKINWH )
TO: BARRY L. HUDSON ( HUDSONBL )
TO: JOHN H. LITTLE ( LITTLEJH )
TO: H. DAVID RAMSEY, JR. ( RAMSEY )

CC: LYNWOOD K. IRELAND ( IRELAND )
CC: JOHN R. MATHEWS ( MATHEWS )
CC: RICHARD A KIRSCHNER, JR ( KIRSCHRA )
CC: DAWN D JACKSON ( JACKSODD )
CC: RALPH G. STAHL, JR. ( STAHLRG AT A1 AT CSOC )
CC: KEITH B. PIERSON ( PIERSOKB AT A1 AT ESVAX )
CC: ANDREW S. HARTTEN ( HARTTEAS AT A1 AT CSOC )
CC: Charles T. Alt ( ALT )
CC: JOHN J MENTINK ( MENTINJJ )
CC: JOHN M MIGLIORE ( MIGLIOJM )
CC: DANIEL A. WEBER ( WEBERDA )
CC: GEORGE WOYTOWICH ( WOYTOWIG )

DUPONT

May 14, 1998

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Ms. Sarah L. Caspar (3HW32)
U.S. Environmental Protection Agency
841 Chestnut Building
Philadelphia, PA 19107

RE: EPA Draft Report, Dry Run Creek, November 1997

Dear Ms. Caspar:

Thank you for providing DuPont Washington Works a copy of the subject report and for meeting with DuPont on February 23, 1998, to hear our concerns regarding the validity of the report's conclusions.

The purpose of this letter is to document those concerns and others which time prohibited reviewing in our meeting. A summary of key comments is provided, followed by a more detailed discussion.

We recognize that the report we reviewed is a "Draft" copy. While our discussion on February 23 raised the prospect of making changes to this draft report to remove some of the implications that the co-authors indicated they did not intend, upon review we believe that a major rewrite would be necessary to accomplish this. We do not believe that prospect was offered. Therefore, we have elected instead to limit our comments to this letter.

We share an interest in determining whether Dry Run Creek has been or is being adversely affected, and if so, determining the cause. However, it is our opinion that the conclusions of the draft report with respect to these questions are not supported by available data. As indicated at our February 23 meeting, it is our intent to work with you to design and consider a collaborative next-step to advance a scientific understanding of the issues behind the risk assessment.

Sincerely yours,

Robert L Ritchey

Robert L. Ritchey
Sr. Environmental Control Consultant
Washington Works

Attachment

hcw: 19675-1

USEPA 062

"Roger J. Zipfel" <ZIPFEL@WWPS-A1.EMAIL.DUPONT.COM> on 09/14/99 04:38:51 PM

To: Roger J Zipfel/AE/DuPont
cc:
Subject: (C-8 ENVIROMENTAL SAMPLING/24-Jun-1998)FC-143 Results Availab

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Date: Wed, 24 Jun 1998 13:38:00 EDT
From: VERA L WIGAL <WIGALVL@wwps-a1.email.dupont.com>
Subject: FC-143 Results Available To-Date (Woytowich)
To: "ANDREW S. HARTTEN" <HARTTEAS@CSOC-A1.EMAIL.DUPONT.COM>, "ROBERT L. RITCHEY" <RITCHERL@wwps-a1.email.dupont.com>, "DANIEL A. WEBER" <WEBERDA@wwps-a1.email.dupont.com>, "LYNWOOD K. IRELAND" <IRELAND@wwps-a1.email.dupont.com>, "Roger J. Zipfel" <ZIPFEL@wwps-a1.email.dupont.com>, "RICHARD A KIRSCHNER, JR" <KIRSCHRA@wwps-a1.email.dupont.com>, GEORGE WOYTOWICH <WOYTOWIG@wwps-a1.email.dupont.com>
MIME-version: 1.0
Content-type: MULTIPART/MIXED; BOUNDARY="Boundary_(ID_2NbORBOXIUKUMFSQRO/NFA)"
Delivery-date: Wed, 24 Jun 1998 13:44:00 EDT
Posting-date: Wed, 24 Jun 1998 13:42:00 EDT
Importance: normal
Al-type: MAIL

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Date: Wed, 24 Jun 1998 11:07:00 EDT
Subject: FC-143 Data from Landfills
MIME-version: 1.0
Content-type: text/plain
Importance: normal
Al-type: DOCUMENT

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June 24, 1998

Details of FC-143 Sample Data From CH2M Hill Available To-Date:

| Location | Date Site Sampled | Site Location | Result ug/L |
|---|---|---|---|

EID082057

| | | | |
|---|---|---|---|
| Dry Run Landfill | 5/19/98 | Stream I | 1.0 |
| " " | " | Stream II | 4.6 |
| " " | " | 001 Outlet | 17 |
| " " | " | Leachate | 56 |
| | | FBLK21 | 0.1 |
| Letart Landfill | 5/28/98 | Lower Pond | 1100 |
| " | " | Upper Pond | 480 |
| " | " | WWKLTLMW6 | 30 |
| " | " | WWKLTLMW2A | 990 |
| " | " | WWKLTLMW7 | 260 |
| " | " | WWKLTLMW1 | 24000 |
| " | " | WWKLTLMW8 | 2700 |
| " | " | Trip Blank | 0.1 |
| | | FBLK02 | 0.1 |
| Local Landfill | 6/2/98 | Outlet 101 | 54 |
| " | " | Stream 2 | 15 |
| " | " | Outfall 004 | 12 |
| " | " | Outfall 005 | 39 |
| | | FBLK05 | 0.1 |
| Plant Site | " | West WellFld | 16 |

16058 (vlw)



EID082058

INTEROFFICE MEMORANDUM

Date: 08-Aug-1998 11:33am
From: Thomas R. Waldron
WALDROTR
Dept: PPD-POWER/SERVICE
Tel No: 304-863-2489

TO: LYNWOOD K. IRELAND ( IRELAND )
TO: JOHN R. MATHEWS ( MATHEWS )

CC: Gary W. Klesel ( KLESELGW )
CC: RICHARD A KIRSCHNER, JR ( KIRSCHRA )

Subject: Dry Run Landfill

I came in this morning at about 0830 to look at Dry Run Landfill. The pond is being drained to the stream, at a very slow rate. The level of the pond had dropped about a foot or so. On my way out, I ran into one of Richard's hands and he said he had opened the valve a little, earlier this moring, and was just checking on the progress. He also said he was told to turn it off before dark.(?) I took some water samples (just for visual comparison) at #001, one at the 2,000 ft level and one at the property line. Richard Construction has installed a couple of check dams at 001 and also installed silt fences and hay bales in the outfall itself. The water was running through them both but not all the solids were staying behind. The stream was turbid down to about a thousand foot or so but beyond that, was fairly clear. There is still a lot of activity down near the property line, and Wilbur Earl has installed a metal gate at the line fence, my guess would be to give him easier access on to our property when he so desires. Someone is running the fill from his end quite a lot. If I get a chance, I may check on things down there tomorrow.

Tom



KAK008851

EID030998

82

000480

INTEROFFICE MEMORANDUM

Date: 09-Aug-1998 03:57pm
From: Thomas R. Waldron
WALDROTR
Dept: PPD-POWER/SERVICE
Tel No: 304-863-2489

TO: LYNWOOD K. IRELAND ( IRELAND )
TO: JOHN R. MATHEWS ( MATHEWS )

CC: Gary W. Klesel ( KLESELGW )
CC: RICHARD A KIRSCHNER, JR ( KIRSCHRA )

Subject: DRY RUN LANDFILL

I came in today at 1400 to check on Dry Run. The pond has now drained down about 3 to 4 feet. I again took samples (for visual inspection only), just below the #001 Outfall, as I did yesterday, at about the 2,000' level and again at the property line. The outfall is still pretty turbid and the sample at the 2,000' level is also slightly turbid. The sample at the property line is still clear. Wilbur's cattle were wallowing in the stream just beyond the fence at the property line. I got several pictures both yesterday and today. You can notice an odor from the sample at the outfall but it does not smell like leachate. I will bring them down in them in the morning and let you all have a look. I talked to the Security Guard at the fill and he said the same couples as last week came down on 4-wheelers and went back up the old road just beyond the old 1,000' sample point. I am going to check that out soon and see if I can find some wacky weed perhaps being grown up there. Problem is, I don't know what the stuff looks like. I may have to take Roger with me!! I hear they grow a lot of that stuff over in Meigs County. As I write this message, looking out my office window, it sure looks a lot like rain on the way! Just what we need at the landfill... More solids in the stream...

Tom

AGENDA

8/11/98

DRY RUN LANDFILL

- Commitment to work together
- Purpose of Collaborative Effort
- Materials Potentially Landfilled
- DuPont Inputs on what to look for
  - * Mammalian effects
  - * Organofluorides
  - * TICs
- 7/22/98 Split Sample Results
- Collaborative Program Proposal



USEPA 0821

PURPOSE STATEMENT FOR COLLABORATIVE PROGRAM:

A collaborative effort between EPA and DuPont, including scope development, data collection, and interpretation, will be undertaken to characterize environmental conditions in order to:

1) investigate potential causes of environmental effects

2) determine if causes are connected to landfill constituents and exposure pathways

8/11/98



EID017942

JMM002629

Group 1: Materials known or suspected to have been sent to Dry Run Landfill in significant quantities at some time in history include the following. This listing represents a good faith effort to evaluate disposal history for materials meeting this criteria.

flyash (coal-fired boilers)
boiler bottom ash (coal-fired and alternate fuels boilers)
dirt/rubble from plant excavations
absorbed biosludge
biocake
bio-waste treatment settlings (equalization tank)
supernate pond dirt
polyacetal resins
nylon resins
polyester resins
elastomeric polymers
acrylic polymers
polyvinyl butyral
fluoropolymers
polyethylene
wood
cardboard
scrap metal
glass
asbestos

8/11/98

Group 1 Appendix: Major monomers and constituents of Group 1 resins.

| RESIN | MAJOR MONOMERS/CONSTITUENTS |
|---|---|
| polyacetal resins | formaldehyde |
| nylon resins | adipic acid<br>hexamethylene diamine<br>caprolactam<br>dodecanedioic acid |
| polyester resins | teraphthalic acid*<br>ethylene glycol*<br>butylene glycol* |
| elastomeric polymers | ethylene-propylene-hexadiene elastomer*<br>ethylene/methacrylic acid copolymers*<br>butylene/poly(alkylene ether)phthalate* |
| acrylic polymers | methyl methacrylate<br>ethyl acrylate<br>ethyl methacrylate<br>n-butyl methacrylate<br>isobutyl methacrylate<br>methacrylic acid |
| polyvinyl butyral | butyraldehyde<br>polyvinyl alcohol<br>tetra ethylene glycol diheptanoate |
| fluoropolymers | tetrafluoroethylene<br>hexafluoropropylene<br>ethylene |
| polyethylene | ethylene |

* = not handled at manufacturing site as monomers (already polymers)

9/1/98

Group 2: Low and trace volume chemicals of interest which could possibly be present at the Dry Run Landfill include the following chemicals used as solvents, plasticizers, refrigerants, surfactants, etc., which could be present in de minimis amounts on filters, gaskets, insulation, rags, absorbents, etc., or as laboratory or maintenance chemicals. The list is not complete as to create such a list would be an exhaustive exercise at a facility as diverse as ours; however, a good faith effort has been made to include those chemicals of greatest interest given the intent of the EPA request.

acetic acid
formic acid
methanol
acetone
chloroform
carbon tetra chloride
heptanes mixed isomers
heptanol mixed isomers
stearic acid and salts
N-butyl benzene sulfonamide
biphenyl and biphenyl oxide
difluorochloromethane (F-22)
trichlorotrifluoroethane (F-113)
methylene chloride
tetrachloroethylene
trichloroethylene
methyl chloroform
phenol
m-cresol
benzyl alcohol
hydroxyethyl cellulose
methyl ethyl ketone
toluene
hydrocarbon oils
silicon oils
polyethylene glycol ethers
succinic acid
ammonium perfluorooctanoate
ethylene glycol
gasoline
di-2 ethylhexoate of polyethylene glycol
numerous hindered phenol compounds
d-limonene
phthalic anhydride
alkaline batteries
Alternate Fuels Boiler flyash (one time event)
diesel fuel

8/12/98

# DRAFT

**Proposal to Conduct a**
**General Human Health and Environmental Effects**
**Risk Analysis on C-8**

Purpose: The purpose of this project is to evaluate the risks to human health and the environment from exposure to C-8 during manufacture, transport, product use, and disposal of C-8. The analysis will be conducted in a fashion that will provide semi-quantitative estimates of risks so that exposures yielding the highest risks can be identified and recommendations on reducing these risks can be developed. Risks from manufacture, transport and product use will developed in a way that will facilitate future comparisons of risks estimated for potential C-8 alternatives. The project will be conducted in three parts. The first two will be conducted in parallel, in which human health and ecological risks will be characterized. The final part will develop conclusions on exposures that contribute the highest risks so that recommendations for risk management strategies and alternatives can be developed. The project is estimated to take 12 months to complete from the time of initiation. The Exposure analyses listed below will require collaboration with appropriate plant personnel. *(The dates presented assume a Feb. 1, 1997 SBU approval date.)*

Scope:

| | | Time Line | Est. Cost($) |
|---|---|---|---|
| I. | Human Health Risk | | |
| A. | Hazard Identification | 4/18/97 | 8000 |

Hazards to human health will be reviewed and summarized in this section. The critical toxicity endpoints of relevance to human health risk will be identified and potential dosimeters to be used for interspecies extrapolation of risk will be discussed. The Haskell toxicity summary will be updated as part of this task.

| | | | |
|---|---|---|---|
| B. | Dose-Response Analysis | 9/30/97 | 43,200 |

The dose-response characteristics of C-8 will be evaluated. This may include conducting benchmark dose analyses to identify no-observed adverse effect levels where necessary. Appropriate dosimeters for interspecies extrapolation will also be developed based on the likely mode of action. The pharmacokinetics of C-8 will also be reviewed. If possible, rudimentary physiologically-based pharmacokinetics approaches will be developed to facilitate interspecies extrapolation of risk. Risks vs. dose relationships will be developed in this phase

| | | | |
|---|---|---|---|
| C. | Exposure Analysis | 9/30/97 | 16,000 |

Reasonable exposure scenarios for C-8 will be developed. This are likely to include airborne, drinking water, dermal, and other oral ingestion pathways. Intake rates and durations of exposure will be developed. Haskell will work with an assigned person(s) from the plant site to help characterize these exposure pathways for manufacturing, transport, product use, and waste disposal operations. The business will provide Haskell with data on concentrations of C-8 in the affected media (air, water, soil). These data will be tabulated. Monte Carlo techniques may be used to calculate expected upper confidence limits for these exposures, depending the availability of data. The cost associated with this task includes only Haskell personnel time.

| | | | |
|---|---|---|---|
| D. | Risk Characterization | 12/15/97 | 16,000 |

Risks will be summarized according to the major routes of exposure (air, water, dermal, other oral) for each C-8 application (manufacture, transport, product use, disposal). The risks will be characterized by comparing the likely exposure concentrations to the dose-response relationship. This method is generally referred to as a Margin of Exposure. The characterization will provide the risk manager with

Are the issues relevant?

information that will help identify the operations and exposure pathways that present the highest risk. The characterizations will also enable future comparisons to be made of potential risks posed by C-8 alternatives.

II Ecological Effects

A. Hazard Identification

Time Line | Est. Cost($)

Hazards to key environmental health indicator species will be reviewed and summarized in this section. The critical toxicity endpoints will be identified. Based on the current literature search, chronic studies in aquatic species will need to be undertaken. Avian reproduction and avian food-chain accumulation will need assessment; however this may be extrapolated using mammalian toxicology and PB-PK data and E Fate information. The Haskell toxicity summary will be updated as part of this task.

Are eco issues relevant?

1.) Hazard Assessment
2.) Testing (Fish ELS and Daphnid Chronic) 9/15/97 $10,000
(This is with static renewal and analytical) 8/15/97 $80,000
3.) Avian (if needed) 8/15/97 ~~$80,000~~ SCRATCH

B. Exposure Analysis

Eco costs

Eco toxicologist

Reasonable exposure scenarios for C-8 will be developed for the higher environmental exposure areas at the three manufacturing sites, including their respective disposal sites and a customer site. Intake rates and durations of exposure will be developed. Haskell will work with an assigned person(s) from the plant site to help characterize these exposure pathways for manufacturing, transport, product use, and waste disposal operations. The business will provide Haskell with data on concentrations of C-8 in the affected media (air, water, soil). These data will be tabulated. Monte Carlo techniques may be used to calculate expected upper confidence limits for these exposures, depending on the availability of data. The cost associated with this task includes only Haskell personnel time.

1.) Exposure Assessment - Washington Works 6/15/97 8,000
2.) Exposure Assessment - Dordrecht 8/15/97 6,000
3.) Exposure Assessment - Shimizu 9/15/97 2,000
4.) Exposure Assessment - Customor 10/15/97 2,000

John Mishue Engr 2/1/98

D. Risk Characterization

Risks will be summarized according to the major media of environmental exposure (water, soil, air) for each C-8 application (manufacture, transport, product use, disposal). The risks will be characterized by comparing the likely Predicted Environmental Concentrations (PEC) to the Predicted No Effect Concentrations (PNEC). This method is generally referred to as Hazard Quotient. The characterization will provide the risk manager with information that will help identify the operations and exposure pathways that present the highest risk. The characterizations will also enable future comparisons to be made of potential risks posed by C-8 alternatives.

12/15/97 16,000

III. Recommendations on Risk Management Strategies and Alternatives

12/15/97 4800

This section will evaluate collectively the risks identified to human health and ecological receptors. Based on these analyses, recommendations will be made as to which operations could be targeted to ensure risks are appropriately managed in the most cost effective manner. This will be a very subjective exercise (narrative) and will require some interaction with the appropriate people from the business.

5

I. 83

II. 204

III 5

"Roger J. Zipfel" <ZIPFEL@WWPS-A1.EMAIL.DUPONT.COM> on 09/14/99 08:51:12 PM

To: Roger J Zipfel/AE/DuPont
cc:
Subject: (C-8 REVIEWS/10-Sep-1998)C-8 Review Date

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Date: Thu, 10 Sep 1998 11:28:00 EDT
From: "ROBERT L. RITCHEY" <RITCHERL@wwps-a1.email.dupont.com>
Subject: C-8 Review Date
To: Bernard J Reilly <REILL2BJ@NANOTES1.EMAIL.DUPONT.COM>, "ISIDOROS J. ZANIKOS, P.E." <ZANIKOIJ@CSOC-A1.EMAIL.DUPONT.COM>, TRINI GARZA <GARZAOT@wwps-a1.email.dupont.com>, "Roger J. Zipfel" <ZIPFEL@wwps-a1.email.dupont.com>, "ROBERT R. MATTSON" <MATTSORR@wwps-a1.email.dupont.com>, "DANIEL A. WEBER" <WEBERDA@wwps-a1.email.dupont.com>, DAWN D JACKSON <JACKSODD@wwps-a1.email.dupont.com>
Cc: "H. DAVID RAMSEY, JR." <RAMSEY@wwps-a1.email.dupont.com>, JOHN M MIGLIORE <MIGLIOJM@wwps-a1.email.dupont.com>, Andrea V Malinowski <MALIN2A@NANOTES1.EMAIL.DUPONT.COM>, L Craig Skaggs <SKAGG2LC@NANOTES1.EMAIL.DUPONT.COM>, Maurice Astorga <ASTORGM@ISCDCVM5>, Robert F Pinchot <PINCH2RF@NANOTES1.EMAIL.DUPONT.COM>
MIME-version: 1.0
Content-type: text/plain
Delivery-date: Thu, 10 Sep 1998 11:56:00 EDT
Posting-date: Thu, 10 Sep 1998 11:49:00 EDT
Importance: normal
Al-type: MAIL

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The C-8 review has been set for 12:30-4:30 October 5 in the B21 upstairs small conference room. Tentative Agenda appears below.

Visitors: I will be arranging your passes and you should plan to have receptionist call me. We can go to cafeteria for lunch if you arrive early enough.
Craig and Andrea: Please feel free to attend. Let me know.

AGENDA: 10/5/98 C-8 Review

* C-8: What is it ?
  - Function and use
  - Vendor and competitor information
  - Molecular structure and biopersistence
  - Toxicology



EID082020

* FLPR Management System
  - Global team structure
  - Goals, Program Summary
  - Reduction and replacement effort progress
  - Product Stewardship view; mass balance

* Dry Run Landfill
  - Fluoropolymers disposal history
  - Sampling data

* Government Communications
  - 104e
  - RFI
  - Permits
  - Drinking Water

* Risk Assessment Program

* Crisis Management Plans

000194

DuPont
Washington Works
P. O. Box 1217
Parkersburg, WV 26102-1217

September 11, 1998

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Mr. John G. Britvec, Geologist
Industrial Waste Section
Office of Water Resources, WV-DEP
Goose Run Road
Fairmont, WV 26554

Dear John,

During our September 10, 1998 conference call with EPA regarding Dry Run Landfill, you asked me to send you the seven items I summarized as our answer to EPA's question about changes we had made in the 1993-1994 timeframe.

As indicated when I covered this material, we are simply reporting changes in the nature of the waste or the physicals of the landfill, without attempting to correlate these changes to any effects. It would be our judgment that most of these changes would have low probability of an eco-effect. This would be further supported by EPA's revelation during the conference call that the allegations of effect date to at least the mid-1980's. EPA also commented that the question about the 1993-1994 timeframe predated their understanding of that history of allegation, and that any question about changes during that or other timeframes is probably not appropriate. However, since you had asked for the summary, I am including it below.

1) Some grades of polyacetal resins were transitioned beginning around 1992 to a different stabilizer...a polyacrylamide stabilizer... to improve product performance at processing temperatures. Only finished resins have the stabilizer, and maximum concentration of polyacrylamide present in them is .5wt%, which would be incorporated in the resin. This would probably not present an issue since resin is stable.

2) At the same time as the stabilizer change and for those same products, we changed to a better anti-oxidant, irganox 245. This material is also incorporated in the stable polymer. The amount in the finished product is less than .1wt%.

3) As the result of a global program to reduce polymer to landfill by finding new outlets for sale of off-spec material and through incineration for recovery of heat value, significant reductions in the quantity of polyacetal, nylon, polyester, and elastomers being landfilled at Dry Run occurred each year from 1993 through 1995.



Printed on Recycled Paper

4) As indicated previously to OWR and EPA, in 1994, in preparation for installing the new biosolids filter press at the plant site, the amount of sorbed solids being sent to Dry Run landfill was significantly increased in the Spring of 1994.

5) Also as indicated previously, in August 1994, we began disposing of biosolids in filter cake form, which increased biomatter concentration and put more total biomass to the landfill because the new filter press was more efficient in removing solids.

6) Because of solids accumulation, the lower pond was drained and sediments were removed in August, 1993 to maintain pond capacity requirements. As you know, this was done in communication with WV-OWR. Steps were taken to prevent the release of sediment to the receiving stream.

7) We began landfilling at the lower section of the landfill in September, 1994, having completed the first pass on the upper section.

I also indicated during the September 10, 1998 conference call that we do not have information on what changes the neighboring farmer may have introduced in this same time period and that we would hope that EPA is investigating this area.

Sincerely,

Robert L. Ritchey
Sr. Environmental Control Consultant
Washington Works

20770-2

000487

# INTEROFFICE MEMORANDUM

**Date:** 30-Sep-1998 09:26am EDT
**From:** DANIEL A. WEBER
WEBERDA
**Dept:** ENGINEERING POLYMERS
**Tel No:** 8-863-4415

TO: DANIEL A. WEBER ( WEBERDA )

Subject: C-8 INFO SLIDES FOR RLR MEETING: 10/5/98

DAW000631

EID091595

000189

DRY RUN LANDFILL: FC-143 HISTORY SUMMARY

- SUPERNATE POND SOIL DISPOSAL:

  - 7100 TONS OF SOIL CONTAINING ~ 85 PPM FC-143 DISPOSED OF NEAR THE BOTTOM OF THE LANDFILL IN 11/88.

  - THIS SOIL WAS LATER MOVED TO THE TOP OF THE LANDFILL AND BURIED IN A CLAY-LINED SEPARATE CELL IN 10/91.

- FC-143 GROUNDWATER AND SURFACE WATER MONITORING:

  - BEGAN MONITORING ANNUALLY FOR FC-143 IN 1991.

  - GROUNDWATER CONCENTRATIONS RANGE <1 - 15 PPB.

  - LEACHATE CONC. AT O.F. 001 RANGES 30 - 200 PPB.

    * PROPERTY BOUNDARY STREAM CONCENTRATION RANGES 2 - 25 PPB.

  - DATA REPORTED ANNUALLY TO THE WVDEP BEGINNING WITH THE 1991 ANNUAL REPORT. STILL MONITORING AND REPORTING ANNUALLY.

    * 1998 RESULTS:

| | | |
|---|---|---|
| LEACHATE | = 56 | PPB |
| OUTLET 001 | = 17 | PPB |
| STREAM #1 | = 1 | PPB |
| STREAM #2 | = 4.6 | PPB |
| PROP. BND. | = 0.88 | PPB |

- ~ 7 POUND/YR FC-143 DISPOSED OF WITH BIOCAKE (930 PPB).

- 600M POUNDS/YR OF FLUOROPOLYMER WASTE DISPOSAL BEGAN 1/96; DOES NOT CONTAIN FC-143.

EID091596

DAW000632

000300

INTEROFFICE MEMORANDUM

Date: 13-Oct-1998 05:11pm
From: DANIEL A. WEBER
WEBERDA
Dept: ENGINEERING POLYMERS
Tel No: 8-863-4415

Subject: POTENTIAL FOAMING MATERIALS AT DRY RUN LANDFILL

FYI,

BASED ON A REVIEW OF GROUP 1 & 2 LISTED MATERIALS, BELOW IS A LIST OF MATERIALS WHICH MAY HAVE GONE TO DRY RUN LANDFILL THAT ARE KNOWN TO CAUSE FOAM.

N-butyl benzene sulfonamide - this compound was identified in a sample of foam taken from a Dry Run Landfill leachate discharge.

stearic acid and salts
polyethylene glycol ethers
ammonium perfluorooctanoate

Triton X-100 (tm) - not specifically listed but contained in supernate dirt and possibly empty containers
ucon oil - not on group 1 & 2 list

biological foams - from biocake and bacteria growth in ponds



EID016841

009502

INTEROFFICE MEMORANDUM

Date: 29-Oct-1998 10:29am
From: DANIEL A. WEBER
WEBERDA
Dept: ENGINEERING POLYMERS
Tel No: 8-863-4415

TO: See Below

Subject: FOAM AT DRY RUN LANDFILL

JOHN,

I WENT DOWN TO DRY RUN LANDFILL TODAY WITH TOM WALDRON.

I NOTICED FOAM FORMING DOWN IN THE LOWER POND BELOW THE LEACHATE DISCHARGE PIPE. THERE IS CURRENTLY NO POND DISCHARGE TO DRY RUN CREEK.

BARBARA DEEM WILL BE TAKING A SAMPLE TODAY OF THE FOAM AND THE LEACHATE DISCHARGE.

PLEASE TAKE A LOOK AT THESE SAMPLES AND LET ME KNOW WHAT YOU CAN DETERMINE IS CAUSING THE FOAM - SAME MATERIALS AS LAST TIME ? CAN THE LEVELS BE QUANTIFIED ?

PLEASE REFRIGERATE THE UNUSED PORTION OF THE SAMPLES IN CASE WE WANT TO DO ADDITIONAL ANALYSES LATER.

THANKS FOR YOUR HELP,

DAN

Distribution:

| | | |
|---|---|---|
| TO: | JOHN F. DOUGHTY | ( DOUGHTJF ) |
| CC: | ROBERT L. RITCHEY | ( RITCHERL ) |
| CC: | LYNWOOD K. IRELAND | ( IRELAND ) |
| CC: | RICHARD A KIRSCHNER, JR | ( KIRSCHRA ) |
| CC: | Thomas R. Waldron | ( WALDROTR ) |
| CC: | Gary W. Klesel | ( KLESELGW ) |

RAK008791

EID030763

000503

000504

C-8 PACE

**INTEROFFICE MEMORANDUM**

**Date:** 02-Nov-1998 02:36pm
**From:** GERRY KENNEDY
KENNEDGL
**Dept:** AG
**Tel No:** 366-5259

**TO:** Oscar T Garza ( GARZA2OT AT CDCLNM3 )
**TO:** Matthew C Koenings ( KOENI2MC AT CDCLNM1 )
**TO:** Roger J. Zipfel ( ZIPFEL AT A1 AT WWPS )
**TO:** WILLIAM J BROCK ( BROCKWJ )

**Subject:** C-8 MONKEY STUDY

PROGRESS REPORT THROUGH FIRST 30 DAYS OF MONKEY STUDY:
1)TOP DOSE 30MG/KG NOT TOLERATED BY 2 OF 6 MONKEYS, DOSING WAS DISCONTINUED AFTER 11 DOSES OF 30 FOR 11 DAYS THEN WAS RESTARTED AT 20 MG/KG. AFTER THIS LOWERING, 1 OF THE 6 (AFFECTED AT 30) WAS SACRIFICED IN DISTRESS AFTER 7 DOSES. A NUMBER OF CLINICAL PARAMETERS WERE ALTERED IN THIS ANIMAL ALONG WITH MARKED HYPOCHOLESTEROLEMIA, MILD HYPERBILIRUBINEMIA, AND MARKED INCRESE IN HEPATIC ENZYMES. THE MOST APPARENT EXPLANATION FOR THE RESPONSE IN THIS ANIMAL IS LIVER DEGENERATION/NECROSIS WITH IMPAIRED LIVER FUNCTION, POSSIBLE BLOOD LOSS, AND INFLAMMATION IN EITHER THE LIVER OR GI TRACT. PATHOLOGY WILL ADD DETAIL TO THIS.
2)3 AND 10 MG/KG MONKEYS TO DATE LOOK FINE.
4)REMAINING 5 MONKEYS AT 20 MG/KG DOING FINE; CLINICALS DO NOT SEEM TC INDICATE ANY PROBLEMS INCLUDING THE LIVER.
NOTE)NO EFFECTS SEEN IN 4 WEEK PILOT AT 20 MG/KG. IT IS OBVIOUS THAT AT LEAST SOME OF THE POPULATION CAN NOT TOLERATE 30/20 MG/KG, 3 AND 10 MG/KG COULD PRODUCE EFFECTS AS THE TIME OF DOSING EXTENDS FROM 1 TO 6 MONTHS. REMEMBER THE CUMULATIVE POTENTIAL HERE IS WHAT WE ARE LOOKING AT/FOR.
NEXT REPORT WILL BE A 2 MONTHS UNLESS STRIKING FINDING INTERVENE.

GWJ00189

EID088762

000506

INTEROFFICE MEMORANDUM

Date: 04-Nov-1998 08:10am
From: JOHN F. DOUGHTY
DOUGHTJF
Dept: PPD
Tel No: (304) 863-2801

TO: See Below

Subject: SAMPLES FROM DRY RUN LANDFILL

Paul Rowan and I have spent several man days on the two samples using the FT-IR, the Mass Spec and the x-ray on the electron microscope.

The foam sample collected from the Lower Pond on 10-29-98 at 11:39AM was analyzed to identify the cause of the foam.

Because the foam was very concentrated and stable, we were able to isolate material by drying at room temperature and a simple methanol wash. The major material in the foam is the sodium salt of an acrylic polymer with a high acid content. As you know these resins have become a major product in recent years. We have never identified this material before so to confirm the identity, I made some in the laboratory using Elvacite 2627 and sodium hydroxide. The scans were a match. Later we did our normal extraction procedure and isolated enough total material to be very roughly 3500 mg/L in concentration.

I speculate the high acid acrylic resins which are insoluble as they are made are being converted to the sodium salt in the landfill and become soluble. We know there is sodium in the fly ash and the fly ash is basic. I am not aware of any other major sources of sodium. The resin itself could originate either from the filter cake in Acrylics or from acrylic polymers that accumulate in the biosludge and the bottom of the equalization tank.

Mass Spec work done on the extracts also identified smaller amounts of ortho and para toluene sulfonamide and butyl benzene sulfonamide, nylon oligomers, a fluorocarbon that we believe is C8 and a complex mixture that we believe is biological in origin. Lots of microbiological organisms were evident under the microscope.

X-ray work also detected sodium, chlorine, magnesium, calcium, potassium, aluminum, silicon, sulfur, iodine and iron. Most of these could be from dirt or the fly ash but the iodine in particular is not expected. The source must be the potassium iodide and cuprous iodide used as stabilizers in Zytel products. One of our Mass Spec scans also seems to indicate an iodine containing compound.

The other sample was taken from the discharge of the 4 inch pipe at the Lower Pond at the same time.

We did our normal extraction procedure to isolate the organics in the sample. We weighed the recovered material to determine the concentration which was roughly 35 mg/L. We found the major component was nylon oligomers. Also present was some material that we believe is biological in origin as well as ortho and para toluene

sulfonamide and butyl benzene sulfonamide.

Distribution:

| | | |
|---|---|---|
| TO: | DANIEL A. WEBER | ( WEBERDA ) |
| TO: | ROBERT L. RITCHEY | ( RITCHERL ) |
| TO: | GEORGE WOYTOWICH | ( WOYTOWIG ) |



EID030774

CC: PAUL E ROWAN ( ROWANPE )
CC: CHARLES J REFSHAUGE ( REFSHACJ )
CC: PAULA G CRAWLEY ( CRAWLEPG )
CC: CYNTHIA H KIRSCHNER ( KIRSCHCH )
CC: RICHARD A KIRSCHNER, JR ( KIRSCHRA )

000516

I N T E R O F F I C E  M E M O R A N D U M

Date: 13-Jan-1999 10:26am
From: Thomas R. Waldron
WALDROTR
Dept: PPD-POWER/SERVICE
Tel No: 304-863-2469

TO: JON F. MOORE ( MOOREJF )

CC: LYNWOOD K. IRELAND ( IRELAND )
CC: RICHARD A KIRSCHNER, JR ( KIRSCHRA )

Subject: Dry Run Landfill

While touring the facility yesterday, I witnessed quite a bit of solids in the stream. I talked to Melvin Boso and we took steps to contain as much as we could. This would be to add fresh bales of hay/straw in the stream and the ditch line itself. The pond was running out the outfall and appeared to be in very good shape.

Tom

JI:M000400



EID030784

000312

KOENI2MC@CDCLN05.LVS.DUPONT.COM on 01/26/99 09:52:00 AM

To: PINCH2RF@CDCLN05.LVS.DUPONT.COM@SMTP
cc: ASTOR2M@CDCLN05.LVS.DUPONT.COM@SMTP , BROCKWJ, ZIPFEL, GARZA2OT@CDCLN05.LVS.DUPONT.COM@SMTP
Subject: Re: C-8 Human Risk Assessment and Analysis

Rob,

I like your outline for risk assessment. It may not prove to be perfect, but it is an excellent start. I don't see any flaws at this point in time, but I have experienced evolution in these type of processes once they are put in use for the first time.

I would suggest that we use at least one other alternative case. We exit the use of C-8 in all product lines that do not "heat treat" the resin in such a way that the C8 levels are dramatically reduced in our product and the C8 used in the process can be aggressively contained/destroyed. Dispersions are my first concern, but there may be a few other types/grades that do not receive adequate heat treatment in this respect. The attempt here is to eliminate situations where we distribute a product with "high" levels of C8 that go outside of our control, but manage the use of C8 where the levels are low and the applications are known to either adequately destroy and or control the ultimate fate. It is an attempt to mitigate the risk where practical and eliminate the risk where we loose control. There may be a few of these intermediate positions to discuss/explore.

Thanks.

Matt

Robert F Pinchot 01/22/99 02:50 PM
(Embedded image moved to file: PIC23720.PCX)

To: Oscar T Garza/AE/DuPont@DuPont, "Roger J. Zipfel" <ZIPFEL@wwps-a1.email.dupont.com>, Matthew C Koenings/DuPont@DuPont, "WILLIAM J BROCK" <BROCKWJ@esvax-a1.email.dupont.com>
cc: Maurice Astorga/AE/DuPont@DuPont
Subject: C-8 Human Risk Assessement and Analysis

I've been putting a lot of thought into and talking to a lot of people about how we might use the output of the C-8 monkey study and the subsequent risk assessment. In this thought process I've come to some conclusions as to what I think will be the outcome based on intuition. Hopefully, the data will uphold my intuitive thoughts but we'll have to wait until the data is available. But that isn't the purpose of this note.

What I want to do with this note is to get you thinking about the approach that I'm proposing and giving me feedback on how to upgrade the process. Comments are welcome and needed...Here goes.

Assumptions:

Output of the Risk assessment will be a series of results that will be in the form of "for toxic endpoint X, the risk is Y cases/1000yr at Z exposure."

We will have exposure data that will allow calculation the risk of the exposed population in the form of Y' cases/1000 yr.

Any C-8 replacements will have at least 10X less risk for toxic endpoints as C-8.

Proposed Approach:

Select a base case and 4 alternative cases as options for our C-8 strategy.

Compare the financials of the five cases ( using NPV as the best indicator) with the risk calculation for these cases.

Utilize the following data measures as the comparison points:

Absolute change in the risk for human health effects

i.e. strategy A reduces risk by X cases/1000yr vs. strategy B

Relative change in the risk for human health effects

i.e. strategy A reduces risk by X% vs. strategy B

Relative change in the risk for human health effects on a financially weighted basis

i.e. utilize the metric of X cases reduced/1000yr/$MM NPV change vs. the base case

Consider the DuPont image vs. the competitor's image based on the various strategies. Would the strategies hold up to public scrutiny?

Consider the relative risk of the various C-8 strategies with other risks of industrial occupation and every day life. (just for your information, some risk numbers are listed at the end of this note. I thought that these are enlightening)

Consider the premise that the goal for exposures and emissions is ZERO.

Consider the relative risk reduction of the C-8 strategies vs. other risk reduction activities in our business..

Options to Consider

Base Case:

Replace C-8 where technically feasible and market conditions allow.

Aggressively minimize exposure/emissions at our manufacturing plants and customer locations

Alternate Case 1:

Do not replace C-8 in any application.

Aggressively minimize exposure/emissions at our manufacturing plants and customer locations

Alternate Case 2:

Replace all C-8 use

Aggressively minimize replacement emissions/exposure

RJZ012342

Exit fine powder market

Alternate Case 2a:

Replace all C-8 use
Aggressively minimize replacement emissions/exposure
Develop Fine powder replacement surfactant

Alternate Case 3:

Do nothing beyond emission reduction/exposure minimization beyond that in place year end 1999.

RFP Thoughts

One may think that a decision could be made based solely on the relative risk reduction ( i.e. the metrics above) of the various options. I contend that the most likely outcome of the risk study will be that the metrics won't matter much and the decision on the best strategy will have to be made based on the last four considerations above. The human risk hierarchy (from highest to lowest) is AC3, BC, AC2a, AC2.

When you also consider environmental risk the picture gets even fuzzier and can be very site specific. In that case the risk numbers will only tell us if we have a definite problem with our past or current practices.

Comments/Upgrades Requested

Please comment on the following aspects of this plan. Note that this is very preliminary and will probably go through many iterations prior to getting the data.

Metrics to be used
Assumptions made
Considerations to take into account
Cases to be studied

Risk Numbers

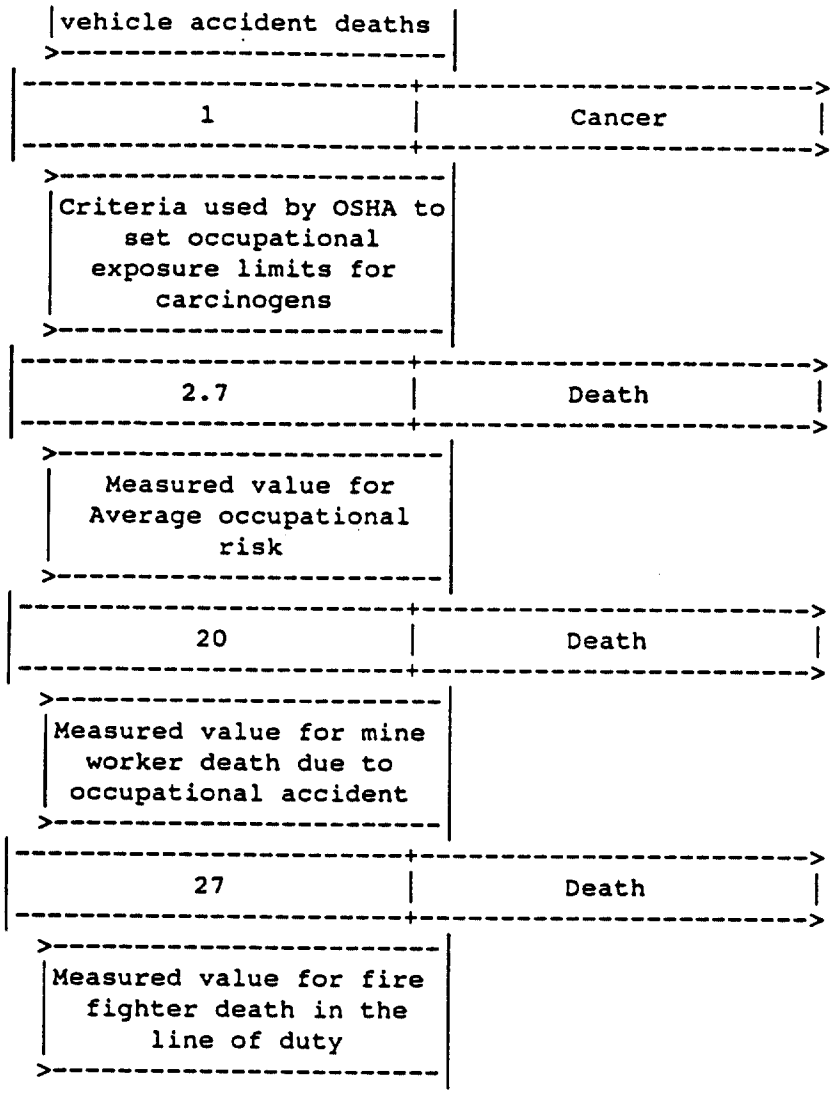
| Number of endpoints/1000 people | Endpoint considered |
|---|---|
| 0.001 | Cancer |
| 0.02 | Death |

Event/Criteria

California Prop 65 Labeling Law

Measured value for motor

EID087031


vehicle accident deaths
1
Cancer
Criteria used by OSHA to set occupational exposure limits for carcinogens
2.7
Death
Measured value for Average occupational risk
20
Death
Measured value for mine worker death due to occupational accident
27
Death
Measured value for fire fighter death in the line of duty

Dry Run Landfill - weber

- EPA found 3-4 PPB of Freon® 11 in soil

attorney/c
Prevale

- No mention of C-8
- Stat has been asking about organo-fluorine compounds because EPA is sensitive to this
- EPA is sensitive to organo fluorine due to effects of Fluoride on cattle teeth - definite connection to Teflon®
- Plant Environmental group wants - attorney client priv

1) what wastes going to Dry Run contain organofluorine
   organic fluori
   a) how much by type - Bills off Waste Connections, Chemfil, Inc., ASP, etc. } waiver - detail; waste - det.
   b) how long

2) Technical position on landfill deposits - migrat
   a) degradation
   b) ~~leach~~ dissolution in water or Soil
   c) Vaporization
   d) Mechanical carry off due to wind, etc
   e) Paper, etc

3) By 2/3.

4)

- Inc. or C. de. ↓ due for organic
- Dry Run

who do all our wastes - Emmelle

CC. Hudson

EID090029

000547

FP/D - Diesel op/Supp ✓ Brown - Ken Cunningham
Area - Gen - P.O. Jill Rogers Casey Cole
FEP - P.O. ✓ Reade Casey Clowes
PFA/TZ - P.O. ✓ John Goldeen, Bob Smith
Manager - Morris Jim Frey

Weights - Steven adams - 2532
B-5
- Mark Jackson - 4763

✓ Pallets - Wooden, or Other (12) Containers
✓ ~~Packing material~~ shields
✓ Special shipping structures
✓

RJZ016397

EID090030

000548

KENNEDGL
02/24/99 12:43 PM

To: BROCK2WJ, KOENI2MC, ZIPFEL, GARZAOT
cc:
Subject: C8 MONKEY STUDY

CURRENTLY IN 21ST WEEK OF 26 WEEK STUDY. LAST WEEK A MONEY (1 OF 4) IN THE LOW DOSE (3MG/KG) SHOWED RAPID DETERIORATION AND WAS SACRIFICED. NO OUTSIDE CAUSE FOR THE DEATH WAS DETECTED; TREATMENT RELATIONSHIP IS A STRONG POSSIBILITY. THIS IS BEING FOLLOWED CLOSELY WITH THE CONTRACTOR (COVANCE-MADISON). THE OTHER 3 ANIMALS IN THIS GROUP ARE FINE AS ARE ALL 6 MONKEYS RECEIVING 10 MG/KG. THERE SEEMS TO BE AN AFFECTS 1 ANIMAL, NOT ANOTHER PHENOMENON GOING ON. ANALYSIS OF EFFECTS VS BLOOD C-8 LEVELS WILL BE INSTRUCTIVE-PRELIMINARY FINDINGS SHOW LITTLE DIFFERENCES IN BLOOD LEVELS BETWEEN MONKEYS RECEIVING 3, 10, OR 20 MG/KG. RECALL THAT MORTALITIES HAVE OCCURRED AT THE 30/20 MG/KG GROUP--THE TOX FOLKS WILL BE GATHERING IN MADISON AT DOSING EBD TO REVIEW DATA-LOOK AT RECOVERY PARAMETERS. PLEASE CALL WITH ANY QUESTIONS (366-5259), I'M CONCERNED ABOUT THE LACK OF DOSE RESPONSE AND SERIOUS FINDINGS IN 1 OF 4 LOW DOSE ANIMALS.

KJZ012241

000520

# Cattle Team Kickoff Meeting
## March 9, 1999
## Summary

The "Cattle Team" met for the first time on March 9 at the Univ of Pennsylvania's New Bolton Center. Many thanks to New Bolton Center for hosting the meeting and providing the conference room.

**Attendees**

Sarah Caspar (EPA Region III), Mike Horne (USFWS), Mark Sprenger (EPA OERR), Perry Habecker (U of Pa), Bob Poppenga (U of Pa), Peter Moisan (Rollins Laboratory), Lisa Heller (private practice), Greg Sykes (DuPont Pharma), Paul Ross (DuPont Haskell Lab), Rudy Valentine (DuPont Haskell Lab), and Ralph Stahl (DuPont Corp. Remediation Group).

**Expectations**

The Cattle Team is expected to be an objective, independent body that will advise EPA and DuPont as to the existing condition of Mr. Tennant's herd and any associated exposures or parameters that might impact the overall health of the herd. The Team is expected to be technically sound in its approach and reach a consensus on the herd's condition and recommendations to mitigate possible future impacts to the herd's health.

**Team Structure and Logistics**

- The Cattle Team is a subset of the Dry Run Technical Team and is composed of veternarians and scientists with expertise in large animals, especially cattle. The Team will conduct detailed evaluations of Mr. Tennant's herd and report back to the Technical Team in approximately 2-3 months. A written report that details the findings of the Team and recommendations for action will be prepared. The Team members are: Habecker, Heller, Moisan Poppenga, and Sykes. Where needed, Caspar, Horn, Sprenger, Stahl and Valentine will support the Team.

- Dr. Greg Sykes was elected to be the coordinator of the Cattle Team, with Dr. Perry Habecker acting as the Scientific Leader. Greg will be responsible for handling logistics and other coordinator duties, while Perry will be responsible for leading the herd evaluation, interpretation of the results, and the drafting of the findings and recommendations.

- Dr. Lisa Heller will act as the local veterinarian and will be involved with all the Team's activities. In addition, because of her proximity to Mr. Tennant's farm, she will be responsible, where needed, for examining any of the herd that might exhibit untoward signs or that is found dead or moribund. Mr. Tennant

GPS000986

will be responsible for notifying Dr. Heller when such animals are noted or found.

- Sarah Caspar will act as the interface with Mr. Tennant on the initial site visit and all communications. No member of the Cattle Team is expected to respond to any questions from Mr. Tennant or others regarding findings, conclusions or recommendations on the herd. Sarah will be responsible for fielding any questions from the media and responding on behalf of EPA. Ralph Stahl will be responsible for referring the same questions to Ms. Dawn Jackson at DuPont Washington Works who will respond on behalf of DuPont.

- EPA will be responsible for supporting the work of Drs. Habecker and Poppenga; DuPont will be responsible for supporting Drs. Heller and Moisan. Analytical work conducted on the herd including blood analysis, etc., administrative support (copying, mailing, etc.) will be billed 50% to EPA, 50% to DuPont. The Team agreed that Mr. Tennant would not be compensated for cattle subjected to sacrifice since he was gaining substantial benefit from this effort at little or no cost.

- While contacted on March 9 by Sarah Caspar and Mike Horne, Mr. Tennant noted that he currently has 40 head of cattle, 36 of which are expected to calve this spring. Two cows are currently exhibiting signs of distress and may require examination by Dr. Heller in the near term. He has a cinder-block barn foundation that can be used as a corral, and a steel head-gate that can be used to hold cattle during examination.

- EPA determined that they would fund and undertake a study to examine white-tailed deer in Wood County and other areas this spring. EPA may use the Cattle Team for consultation on this study, including its design and the interpretation of the results.

**Action Items**

1. The visit to Mr. Tennant's farm is April 8, 1999. Mr. Tennant will be informed of this date by Sarah and Mike. The Team will conduct an evaluation on all 40 head of cattle, obtain a detailed herd history from Mr. Tennant, and may visit the grazing areas including those on or near Dry Run Landfill.

2. Dr. Heller will visit Mr. Tennant's farm to ensure that the corral and head gate are safe and appropriate for use in examining the herd. She will report back to Greg Sykes and Perry Habecker on this point in the next few weeks.

3. The Team will hold a conference call to review the 13 potential problems and differential diagnostic indicators produced during the March 9 meeting. Greg and Perry are responsible for setting this up.

GPS000987

4. Mike Horne will provide the Team with a copy of the Woodward Clyde small mammal sampling effort.

5. Ralph Stahl will provide the Team with the lists of substances and materials deposited in Dry Run Landfill.

6. The majority of the Team's correspondence will occur by email. Dr. Heller will get an email account so that she can communicate electronically with the Team.

7. Home phone numbers of the Team and supporting individuals were distributed so everyone could be notified in the event of an animal exhibiting morbidity or mortality prior to or after the April 8 site visit.

8. The next meeting of the Team and supporting individuals will be sometime after the April 8 visit to Mr. Tennant's farm. The exact date will depend on when analytical results are obtained from blood samples, etc. and whether or not the Team has drafted its findings and recommendations. Ralph and Sarah will check with Greg and Perry after the April 8 visit and determine when this meeting may take place.

Phone, fax, email information


Sarah Caspar

| | | |
|---|---|---|
| Wo: | 215 814-3283 | cellphone 215-370-9170 |
| Fax: | 215 814-3254 | |
| Home: | 610 873-1237 | pager 215-319-6329 |
| Email: | caspar.sarah@epamail.epa.gov | |

Perry Habecker

| | |
|---|---|
| Wo: | 610 444-5800, ext. 2385 |
| Fax: | 610 444-0892 |
| Home: | 610 384-0452 |
| Email: | habecker@vet.upenn.edu |

Mike Horne

| | |
|---|---|
| Wo: | 732 321-6717 |
| Fax: | 732 321-6724 |
| Home: | 610 923-9701 |
| Email: | horne.michael@epamail.epa.gov |

Peter Moisan

| | |
|---|---|
| Wo: | 919 733-3986 |
| Fax: | |
| Home: | 919 363-3523 |
| Email: | |


GPS000988

Greg Sykes
Wo: 302 366-6070
Fax: 302 366-6065
Home: 610 869-8469
Email: greg.p.sykes@dupontpharma.com

Bob Poppenga
Wo: 610 444-5800, ext. 2217
Fax: 610 444-4617
Home: 610 268-4112
Email: poppenga@vet.upenn.edu

Mark Sprenger
Wo: 732 906-6826
Fax: 732 321-6724
Home:
Email: sprenger.mark@epamail.epa.gov

Lisa Heller
Wo: 304 684-9495
Fax:
Home: 304-684-7404
Email: ladh1@jvnc.com

Ralph Stahl
Wo: 302 892-1369
Fax: 302 892-7641
Home: 302 478-9253
Email: ralph.g.stahl-jr@usa.dupont.com

Rudy Valentine
Wo: 302 366-5315
Fax: 302
Home:
Email: rudolph.valentine@usa.dupont.com

GPS000989

# I N T E R O F F I C E  M E M O R A N D U M

**Date:** 10-May-1999 08:08am EDT
**From:** DANIEL A. WEBER
WEBERDA
**Dept:** ENGINEERING POLYMERS
**Tel No:** 8-863-4415

**TO:** ROBERT L. RITCHEY ( RITCHERL )
**TO:** JOHN M MIGLIORE ( MIGLIOJM )

**CC:** ALISON A CRANE ( CRANEAA )

**Subject:** C8 IN EAST FIELD WELLS

BOB/JOHN,

FYI,

PER P&S, OUR DRINKING WATER SUPPLY WELLS ARE 331 AND 336. 332 IS ALSO OCCASIONALLY USED FOR DRINKING WATER.

C8 CONCENTRATIONS MEASURED IN THESE WELLS BY CH2MHILL ARE AS FOLLOWS:

| WELL | JUNE 1993 | APRIL 1996 | MAY 1997 |
|---|---|---|---|
| 331 | 2.9 ug/L | 0.52 ug/L | 0.55 ug/L |
| 336 | 0.5 ug/L | 0.48 ug/L | 0.79 ug/L |
| 332 | 3.3 ug/L | | |

DAN

DAW000627

EID091591

000036

INTEROFFICE MEMORANDUM

**Date:** 21-May-1999 10:11am
**From:** ANTHONY J. [TONY] PLAYTIS
PLAYTIS
**Dept:** POLYMERS SHE&EA
**Tel No:** 304-863-2228

**TO:** ROBERT L. RITCHEY ( RITCHERL )

**CC:** DAWN D JACKSON ( JACKSODD )
**CC:** Roger J. Zipfel ( ZIPFEL )

**Subject:** C-8 In Drinking Water Communications

Bob,

I checked my files for any communications that we might have issued on C-8 in drinking water. The only pertinent documents I could find are attached. I don't believe we ever actually released any communication on the subject.

I'll stop by your office on Tuesday morning at 8:00 to discuss the Thursday meeting in Wilmington. Roger may or may not be able to make it.

Tony

EID089418

From: Dawn D Jackson on 06/23/99 07:55 AM

To: Robert L Ritchey/CL/DuPont@DuPont, Anthony J Playtis/CL/DuPont@DuPont, Roger J Zipfel/AE/DuPont@DuPont

cc:

Subject: Outline of communications with GE Plastics regarding C-8

Gentlemen,

I have attached a draft of an outline of points to be made in communications with GE Plastics. If there are omissions or errors, please let me know. I will attempt to send you a revision before I leave today at 4:30. I will be out of town until Monday.

Dawn

GE Communication

6/22/99
Bob Ritchey, Tony Playtis, Roger Zipfel, Dawn Jackson
Planning for Communications with GE Plastics regarding C-8

Scenario 1: Results of early June testing of GE drinking well show <3 ppb

Recommended communication: John Little calls Dallas Silverthorne to report test results and speak from talking points such as those below; John also offers to send MSDS on C-8 and Community Exposure Guideline (CEG) information.

Outline of talking points (in brief form; to be developed further):

- Why ? – Why we did the sampling (RFI; C-8 in groundwater; need to know if it crosses site boundary; model says no; WW wanted to sample to be sure.)
- What ? -- What is C-8? (Explain what it is: detergent-like material; surfactant; used in manufacture of Teflon® fluoropolymer resins)
- Where? – Where is it coming from? (old supernate ponds – anaerobic digestion ponds constructed in mid-1950's (one) and mid-1970's (two); decommissioned in 1988.)
- Re: concentration -- represents dose lower than TLV would allow; it's ___% of the TLV; below the CEG.
- Toxicity piece: Refer to MSDS; offer to provide copy of MSDS; at these levels, no acute health effects ever observed; however, it does persist in the bloodstream; in animal testing at high doses, liver is the target organ.
- Path forward: Emphasize that concentration in GE well is comparable to concentration in WW drinking water; WW will continue to monitor; if there is an increase at WW, we will contact GE; Site EA will prepare standby statement and Qs/As, in the event of media inquiries.

Scenario 2: Test results – 3-25 ppb

Recommended communication: John Little calls Dallas Silverthorne to request a meeting (1/2-day?) with Silverthorne and his Playtis, Ritchey counterparts; to attend from WW – Little, Ritchey, Playtis, Zipfel.

Outline of talking points (to be more detailed than for first scenario):

- Why? – Same info as above, + some detail about its being the only chemical in the RFI above EPA screening levels; assurance that WW will work with EPA to define levels acceptable to EPA and methods/controls to reach those levels.
- What? – Same info as above + a review of the C-8 molecule itself; possible other applications outside DuPont
- Where? – Same info as above + hydrogeology, including river recharge, plume in groundwater

- Re: concentration: Provide MSDS; discuss toxicologic information in detail, such as major exposure routes (inhalation, skin absorption) vs. ingestion; discuss CEG and intent to reduce or eliminate human health exposure route.
- Toxicity piece: Talk through elements of MSDS, vs. just providing a copy; provide occupational health background – animal studies details, human health-related information, U. of Minnesota paper (flawed study); review what we've done with our own employees since 1981, including employee communications; share Pace Team strategies?
- Path forward: DuPont will take necessary action to protect human health and meet regulatory requirements; Site EA will prepare standby statement and Qs/As, in the event of media inquiries.

Anticipated Qs from GE:

1. What kind of engineering controls are effective to handle elevated levels of C-8?
2. What exposure routes are there besides drinking water?
3. Do the levels in GE drinking water indicate a probability of measurable levels in employees' blood?
4. ...

DuPont
Washington Works
P O Box 1217
Parkersburg, WV 26102 1217

DUPONT

CERTIFIED MAIL –
RETURN RECEIPT REQUESTED

June 24, 1999

Mr. Martin Kotsch
Project Manager
U.S. EPA, Region III
1650 Arch Street
Philadelphia, PA 19103-2029

RE: Permit WVD045875291

Dear Mr. Kotsch:

Please find enclosed the RCRA Facility Investigation (RFI) Report of Findings for your review and comment.

If you have any questions or comments, please contact me at (304) 863-4271.

Very truly yours,

R L Ritchey

R. L. Ritchey
Sr. Environmental Control Consultant
Washington Works

Attachment

CC: Mr. Mark Priddy
Office of Waste Management
WV-DEP
1356 Hansford Street
Charleston, WV 25301

Ms. Barbara Taylor, Chief*
Office of Water Resources
WV-DEP
1201 Greenbrier Street
Charleston, WV 25311

Mr. B. F. Smith, Chief*
Office of Waste Management
Charleston, WV 25301
1356 Hansford Street

* cover letter only

EID073527

ACC005340

# TABLE 6.8
# HEALTH-BASED SCREENING
# FOR PRODUCTION WELL WATER

| Constituent | Units | Potable Water Well (1) | Process Water Wells (2) | Screening Level | Type | Max > SL? |
|---|---|---|---|---|---|---|
| Arsenic | mg/L | 0.01 | 0.0068 | 0.05 | MCL | No |
| Barium | mg/L | 0.07 | 0.16 | 2 | MCL | No |
| Cadmium | mg/L | ND (<0.01) | ND (<0.01) | 0.005 | MCL | No |
| Lead | mg/L | ND (<0.003) | 0.0022 | 0.015 | MCL | No |
| Nickel | mg/L | ND (<0.05) | ND (<0.05) | 0.14 | MCL | No |
| FC-143 | ug/L | 1.9 | 16.2 | 3 | PSL | Potable - No Process - Yes (3) |
| Tetrachloroethylene | ug/L | ND (<5) | 5 | 5 | MCL | No |
| Trichloroethylene | ug/L | ND (<5) | 22 | 5 | MCL | Potable - No Process - Yes (4) |
| Methyene Chloride | ug/L | ND (<5) | - | 5 | MCL | No |
| Freon 113 | ug/L | ND (<10) | 270 | 59000 | RBC | No |

**Legend:**
MCL = Maximum Contaminant Level for drinking water.
PSL = Preliminary Screening Level; see Table 6.6 and Section 6.4.2.
RBC = Risk-based concentration for tap water (USEPA Region III, 1999)
- = Not analyzed.

**Notes:**
(1) Well 336 (AM07-PW01)

(2) Process Water Wells:
K16-PW01
K16-PW01
L17-PW01
L17-PW01
V05-PW01
V05-PW01
L04-PW01
L04-PW01

(3) Trichloroethylene exceeded the MCL in production well V05-PW01.
(4) FC-143 exceeded the preliminary SL in production wells K16-PW01, V05-PW01, and L04-PW01.

GLK002756

EID109784

[redacted]

**William R Berti**

08/11/99 02:11 PM

To: Roger J Zipfel/AE/DuPont@DuPont
cc: Guat-Lian C Kreamer/AE/DuPont@DuPont
Subject: Estimated C-8 in river

Hi Roger,

Attached is the Excel file that I put together on Ohio River flows and estimated C-8 concentrations that we talked about earlier.

Estimated C-8 concentrations in Ohi

Best regards,

*Bill*

DuPont Central Research & Development
Voice: (302) 366-6762
Fax: (302) 366-6602
Pager: (888) 548-9264
Internet e-mail: William.R.Berti@usa.dupont.com

Ohio River monthly average flows at Pittsburgh, PA
River flow data from <ftp://www.lrd-wc.usace.army.mil/Monthly_htm/>
Assume: 1 ft3/sec = 0.0283m3/sec; 1 m3 = 1*10^6 cm3; 1 cm3 = 1 mL

| Mo-Yr | Measured flow, ft3/sec | Long-term of normal, % | Estimated normal flow, ft3/sec | Estimated normal flow, m3/sec | Estimated normal flow, m3/mo | Estimated normal flow, L/mo | C8 discharge to Ohio River in 1996, lbs |
|---|---|---|---|---|---|---|---|
| Jun-98 | 28,480 | 120 | 23733 | 6.72E+02 | 1.74E+09 | 1.74E+12 | |
| Jul-98 | 17,192 | 124 | 13865 | 3.92E+02 | 1.05E+09 | 1.05E+12 | |
| Aug-98 | 9,111 | 76 | 11988 | 3.39E+02 | 9.09E+08 | 9.09E+11 | |
| Sep-98 | 7,312 | 72 | 10156 | 2.87E+02 | 7.45E+08 | 7.45E+11 | |
| Oct-98 | 7,966 | 66 | 12070 | 3.42E+02 | 9.15E+08 | 9.15E+11 | |
| Nov-98 | 6,144 | 28 | 21943 | 6.21E+02 | 1.61E+09 | 1.61E+12 | |
| Dec-98 | 9,440 | 25 | 37760 | 1.07E+03 | 2.86E+09 | 2.86E+12 | |
| Jan-99 | 53,865 | 124 | 43440 | 1.23E+03 | 3.29E+09 | 3.29E+12 | |
| Feb-99 | 46,314 | 94 | 49270 | 1.39E+03 | 3.37E+09 | 3.37E+12 | |
| Mar-99 | 52,898 | 77 | 68699 | 1.94E+03 | 5.21E+09 | 5.21E+12 | |
| Apr-99 | 60,741 | 105 | 57849 | 1.64E+03 | 4.24E+09 | 4.24E+12 | |
| May-99 | 23,053 | 61 | 37792 | 1.07E+03 | 2.86E+09 | 2.86E+12 | |
| Total flow Jun-98 to May-99, L | | | | | | 2.88E+13 | 40600 |

11-Aug-99

| C8 discharge to Ohio River in 1996, kg | Estimated C8 concentration in Ohio River, kg/L | Estimated C8 concentration in Ohio River, mg/L | **Estimated C8 concentration in Ohio River, ug/L** |
|---|---|---|---|
| 18416 | 6.39E-10 | 6.39E-04 | **0.639** |



EID082039

Ohio River monthly average flows at Belleville Dam, WV

River flow data from <http://waterdata.usgs.gov/nwis-w/WV/data.components/hist.cgi?statnum=03159530>

Based on USGS mean daily flows from 10/1/1974 to 9/30/1985

| Mo | Measured flow, ft3/sec | Estimated normal flow, ft3/sec | Estimated normal flow, m3/sec | Estimated normal flow, m3/mo | Estimated normal flow, L/mo | C8 discharge to Ohio River in 1996, lbs | C8 discharge to Ohio River in 1996, kg | Estimated C8 concentration in Ohio River, kg/L |
|---|---|---|---|---|---|---|---|---|
| Jan | 64000.79 | 64001 | 1.81E+03 | 4.69E+09 | 4.69E+12 | | | |
| Feb | 88908.68 | 88909 | 2.52E+03 | 6.74E+09 | 6.74E+12 | | | |
| Mar | 111235.8 | 111236 | 3.15E+03 | 8.43E+09 | 8.43E+12 | | | |
| Apr | 94016.67 | 94017 | 2.66E+03 | 6.90E+09 | 6.90E+12 | | | |
| May | 67107.33 | 67107 | 1.90E+03 | 5.09E+09 | 5.09E+12 | | | |
| Jun | 47378.91 | 47379 | 1.34E+03 | 3.48E+09 | 3.48E+12 | | | |
| Jul | 30989.47 | 30989 | 8.77E+02 | 2.35E+09 | 2.35E+12 | | | |
| Aug | 30501.91 | 30502 | 8.63E+02 | 2.31E+09 | 2.31E+12 | | | |
| Sep | 25339.3 | 25339 | 7.17E+02 | 1.73E+09 | 1.73E+12 | | | |
| Oct | 33029.62 | 33030 | 9.35E+02 | 2.50E+09 | 2.50E+12 | | | |
| Nov | 42329.7 | 42330 | 1.20E+03 | 3.11E+09 | 3.11E+12 | | | |
| Dec | 78985.92 | 78986 | 2.24E+03 | 5.99E+09 | 5.99E+12 | | | |
| Total | 7.14E+05 | 7.14E+05 | 2.02E+04 | 5.33E+10 | 5.33E+13 | 40600 | 18416 | 3.45E-10 |

| Estimated C8 concentration in Ohio River, mg/L | Estimated C8 concentration in Ohio River, ug/L |
|---|---|
| 3.45E-04 | 0.345 |

Ohio River monthly average flows at Huntington, WV

River flow data from <ftp://www.lrd-wc.usace.army.mil/Monthly_htm/>

Assume: 1 ft3/sec = 0.0283m3/sec; 1 m3 = 1*10^6 cm3; 1 cm3 = 1 mL

| Mo-Yr | Measured flow, ft3/sec | Long-term of normal, % | Estimated normal flow, ft3/sec | Estimated normal flow, m3/sec | Estimated normal flow, m3/mo | Estimated normal flow, L/mo | C8 discharge to Ohio River in 1996, lbs |
|---|---|---|---|---|---|---|---|
| Jun-98 | 97,300 | 172 | 56570 | 1.60E+03 | 4.15E+09 | 4.15E+12 | |
| Jul-98 | 68,742 | 190 | 36180 | 1.02E+03 | 2.74E+09 | 2.74E+12 | |
| Aug-98 | 28,484 | 94 | 30302 | 8.58E+02 | 2.30E+09 | 2.30E+12 | |
| Sep-98 | 17,733 | 78 | 22735 | 6.43E+02 | 1.67E+09 | 1.67E+12 | |
| Oct-98 | 19,710 | 80 | 24638 | 6.97E+02 | 1.87E+09 | 1.87E+12 | |
| Nov-98 | 18,233 | 41 | 44471 | 1.26E+03 | 3.26E+09 | 3.26E+12 | |
| Dec-98 | 28,710 | 36 | 79750 | 2.26E+03 | 6.04E+09 | 6.04E+12 | |
| Jan-99 | 141,810 | 126 | 112548 | 3.19E+03 | 8.53E+09 | 8.53E+12 | |
| Feb-99 | 108,500 | 84 | 129167 | 3.66E+03 | 8.84E+09 | 8.84E+12 | |
| Mar-99 | 142,350 | 86 | 165523 | 4.68E+03 | 1.25E+10 | 1.25E+13 | |
| Apr-99 | 112200 | 82 | 136829 | 3.87E+03 | 1.00E+10 | 1.00E+13 | |
| May-99 | 57484 | 62 | 92716 | 2.62E+03 | 7.03E+09 | 7.03E+12 | |
| Total flow Jun-98 to May-99, L | | | | | | 6.90E+13 | 40600 |

| C8 discharge to Ohio River in 1996, kg | Estimated C8 concentration in Ohio River, kg/L | Estimated C8 concentration in Ohio River, mg/L | **Estimated C8 concentration in Ohio River, ug/L** |
|---|---|---|---|
| 18416 | 2.67E-10 | 2.67E-04 | **0.267** |

Ohio River monthly average flows at Cincinnati, OH

River flow data from <ftp://www.lrd-wc.usace.army.mil/Monthly_htm/>

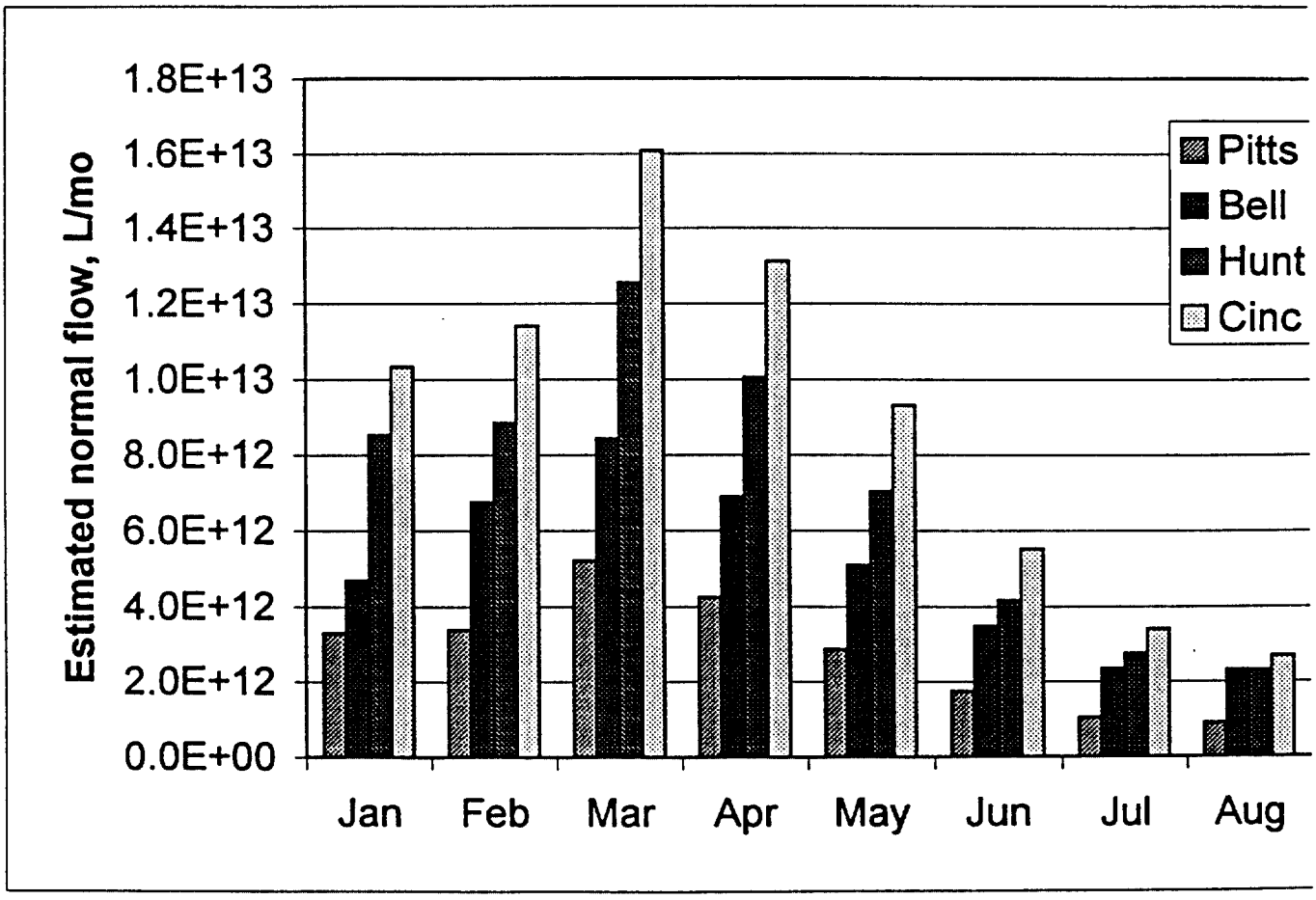
Assume: 1 ft3/sec = 0.0283m3/sec; 1 m3 = 1*10^6 cm3; 1 cm3 = 1 mL

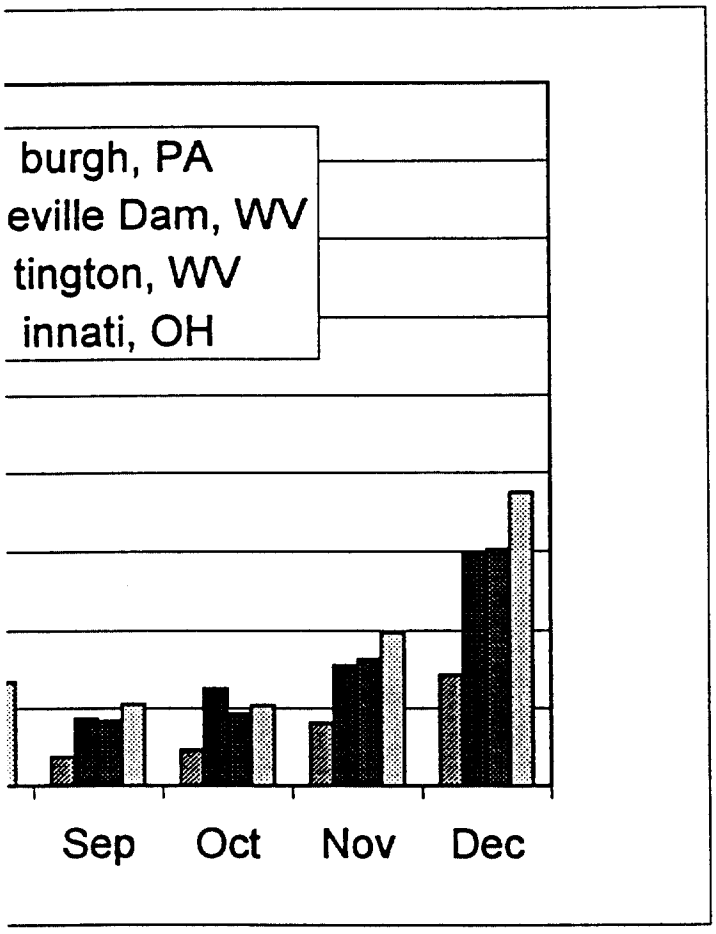
| Mo-Yr | Measured flow, ft3/sec | Long-term of normal, % | Estimated normal flow, ft3/sec | Estimated normal flow, m3/sec | Estimated normal flow, m3/mo | Estimated normal flow, L/mo | C8 discharge to Ohio River in 1996, lbs |
|---|---|---|---|---|---|---|---|
| Jun-98 | 131,400 | 175 | 75086 | 2.12E+03 | 5.51E+09 | 5.51E+12 | |
| Jul-98 | 94,452 | 211 | 44764 | 1.27E+03 | 3.39E+09 | 3.39E+12 | |
| Aug-98 | 28,387 | 80 | 35484 | 1.00E+03 | 2.69E+09 | 2.69E+12 | |
| Sep-98 | 21,200 | 74 | 28649 | 8.11E+02 | 2.10E+09 | 2.10E+12 | |
| Oct-98 | 21,065 | 77 | 27357 | 7.74E+02 | 2.07E+09 | 2.07E+12 | |
| Nov-98 | 21,400 | 40 | 53500 | 1.51E+03 | 3.92E+09 | 3.92E+12 | |
| Dec-98 | 34,548 | 35 | 98709 | 2.79E+03 | 7.48E+09 | 7.48E+12 | |
| Jan-99 | 178,190 | 131 | 136023 | 3.85E+03 | 1.03E+10 | 1.03E+13 | |
| Feb-99 | 134,860 | 81 | 166494 | 4.71E+03 | 1.14E+10 | 1.14E+13 | |
| Mar-99 | 180,260 | 85 | 212071 | 6.00E+03 | 1.61E+10 | 1.61E+13 | |
| Apr-99 | 118000 | 66 | 178788 | 5.06E+03 | 1.31E+10 | 1.31E+13 | |
| May-99 | 60226 | 49 | 122910 | 3.48E+03 | 9.32E+09 | 9.32E+12 | |
| Total | 1.02E+06 | | 1.18E+06 | 3.34E+04 | 8.74E+10 | 8.74E+13 | 40600 |

| C8 discharge to Ohio River in 1996, kg | Estimated C8 concentration in Ohio River, kg/L | Estimated C8 concentration in Ohio River, mg/L | **Estimated C8 concentration in Ohio River, ug/L** |
|---|---|---|---|
| 18416 | 2.11E-10 | 2.11E-04 | **0.211** |

Ohio River monthly average flows at Pittsburgh, PA, Huntington, WV, and Cincinnati, OH
River flow data from <ftp://www.lrd-wc.usace.army.mil/Monthly_htm/>

| No. | Mo | Pittsburgh, PA | Belleville Dam, WV | Huntington, WV | Cincinnati, OH |
|---|---|---|---|---|---|
| 1 | Jan | 3.29E+12 | 4.69E+12 | 8.53E+12 | 1.03E+13 |
| 2 | Feb | 3.37E+12 | 6.74E+12 | 8.84E+12 | 1.14E+13 |
| 3 | Mar | 5.21E+12 | 8.43E+12 | 1.25E+13 | 1.61E+13 |
| 4 | Apr | 4.24E+12 | 6.90E+12 | 1.00E+13 | 1.31E+13 |
| 5 | May | 2.86E+12 | 5.09E+12 | 7.03E+12 | 9.32E+12 |
| 6 | Jun | 1.74E+12 | 3.48E+12 | 4.15E+12 | 5.51E+12 |
| 7 | Jul | 1.05E+12 | 2.35E+12 | 2.74E+12 | 3.39E+12 |
| 8 | Aug | 9.09E+11 | 2.31E+12 | 2.30E+12 | 2.69E+12 |
| 9 | Sep | 7.45E+11 | 1.73E+12 | 1.67E+12 | 2.10E+12 |
| 10 | Oct | 9.15E+11 | 2.50E+12 | 1.87E+12 | 2.07E+12 |
| 11 | Nov | 1.61E+12 | 3.11E+12 | 3.26E+12 | 3.92E+12 |
| 12 | Dec | 2.86E+12 | 5.99E+12 | 6.04E+12 | 7.48E+12 |




burgh, PA
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tington, WV
innati, OH
Sep
Oct
Nov
Dec

ATTACHMENT 101

is TSCA CBI.
DCN 6301000014
No Sanitized Version
was provided.

DuPont
Washington Works
P. O. Box 1217
Parkersburg, WV 26102-1217

August 31, 1999

Mr. Dallas Silverthorne, Plant Manager
GE Plastics
P. O. Box 68
Washington, WV 26181

Dear Dallas:

As we discussed by telephone on August 30, I have enclosed the lab report on the water sample taken at your facility on June 3, 1999, for analysis of levels of ammonium perfluorooctanoate. I have also enclosed the DuPont Material Safety Data Sheet. Both documents refer to the material as FC-143, but it is commonly known as C-8.

I'd like to reiterate that the 0.552 ug/l concentration detected in your site's sample represents a dose lower than the TLV would allow. In fact, it's approximately 1% of the TLV and is in the same range as what we see in our site's drinking water wells. No human health effects have been observed at concentrations many times this level.

I hope that this information is useful to you. If you have any (further) questions, you can reach me at 863-4305.

Sincerely,

John H. Little
Plant Manager

Enclosures

EID089407

COPY

E. I. du Pont de Nemours and Company

Printed on Recycled Paper

Lancaster Laboratories
Where quality is a science.

LLI Sample No. WW 3167122 by GW
Collected: 6/ 3/99

Submitted: 6/ 5/99 Reported: 6/16/99
Discard: 7/17/99

WWK-G-R-2 Water Sample

ANNUAL C8 SAMPLING 1999

Account No: 07032
CRG-E.I.DuPont de Nemours & Co
P.O. Box 47
Old Hickory TN 37138-0047

P.O. LBIO-62248-1
Rel. LE458

AS RECEIVED

| CAT NO. | ANALYSIS NAME | RESULTS | METHOD DETECTION LIMIT | UNITS |
|---|---|---|---|---|
| 2324 | FC-143 in Water | 0.552 | 0.029 | ug/l |

The stated QC limits are advisory only until sufficient data points can be obtained to calculate statistical limits.

QUALITY CONTROL REPORT

| | SAMPLE MDL | SAMPLE UNITS | BLANK | DUP RPD | MS | MSD | MS RPD | LCS | LCS DUP | LCS RPD | LCS LIMITS LOW | LCS LIMITS HIGH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 2324 | FC-143 in Water | | | | | | | | | | | |
| | 0.029 | ug/l | 0.032 J ug/l | | 96 | -38 | 36 | 21 | | | 50 | 150 |

SURROGATE SUMMARY

| | TRIAL ID | SURROGATE | RECOVERY % | SURROGATE LIMITS LOW | SURROGATE LIMITS HIGH |
|---|---|---|---|---|---|
| 2324 FC-143 in Water | | PFNA | 35 | 9 | 81 |
| | | 11H-PFUA | 0 | 13 | 88 |
| | | PFDOA | 5 | 50 | 150 |

LABORATORY CHRONICLE

| CAT NO | ANALYSIS NAME | METHOD | | TRIAL | ANALYSIS ID | DATE AND TIME | ANALYST |
|---|---|---|---|---|---|---|---|
| 2324 | FC-143 in Water | FC-143 by GC/ECD | 10/91 | 1 | | 06/10/99 0328 | Carolyn J. Koch |
| 2325 | Pesticide Alternate Water Extr | FC-143 10/91 | | 1 | | 06/08/99 0730 | Deborah M. Zimmerman |

EID089408

RECEIVED
JUN 18 1999

1 COPY TO Woodward Clyde Diamonds ATTN: Ms. Sara Seastrom

CED Load Date 6/18/99 Time 13:07:30
Accuracy Check by [signature] Date 6/21/99
Holding Time Check [signature] Date 6/21/99

Questions? Contact your Client Services Representative
Nancy Bornholm at (717) 656-2300
07:17:34 D 0001 1 135803 668971
0.00 00022500 ASR000

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax: 717 656 2681

Lancaster Laboratories is a subsidiary of Thermo TerraTech Inc., a Thermo Electron Company.
See reverse side for explanation of symbols and abbreviations.

7216 Rev.

FC-143 FLUORAD BRAND FLUOROCHEMICAL SURFACTANT (3M)
V0003045 Revised 15-JAN-1997 Printed 5-AUG-1999

## CHEMICAL PRODUCT/COMPANY IDENTIFICATION

Tradenames and Synonyms

FLUORAD FC-143

Company Identification

MANUFACTURER/DISTRIBUTOR
Revised 23-AUG-1996
3M General Offices
3M Center
St. Paul
MN
USA
55144-1000

PHONE NUMBERS
Product Information : 612-733-1110
Transport Emergency : 800-424-9300

## COMPOSITION/INFORMATION ON INGREDIENTS

Components

| Material | CAS Number | % |
|---|---|---|
| Ammonium Perfluorooctanoate | 3825-26-1 | 93-97 |
| Ammonium perfluoroheptanoate | 6130-43-4 | 1-3 |
| Ammonium perfluoropentanoate | 68259-11-0 | 1-3 |
| Ammonium perfluorohexanoate | 21615-47-4 | 0.1-1 |

## EXPOSURE CONTROLS/PERSONAL PROTECTION

Exposure Guidelines

Applicable Exposure Limits
Ammonium Perfluorooctanoate
PEL (OSHA) : None Established
TLV (ACGIH) : 0.01 mg/m3, 8 Hr. TWA, Skin, A3
AEL * (DuPont) : 0.01 mg/m3, 8 Hr. TWA, Skin

* AEL is DuPont's Acceptable Exposure Limit. Where governmentally imposed occupational exposure limits which are lower than the AEL are in effect, such limits shall take precedence.

EID089409

OTHER INFORMATION

Additional Information

**********
* Information in the above Sections are provided by DuPont*
* and should be used as the primary source for these *
* categories. *
**********

Vendor MSDS Text

3M MATERIAL SAFETY DATA SHEET

3M General Offices
3M Center
St. Paul, MN 55144-1000
612-733-1110

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2) Neither the copy nor the original is resold or otherwise distributed with the intention of earning profit thereon.

Abbreviations: N/D - Not Determined
N/A - Not Applicable

DIVISION: SPECIALTY CHEMICALS DIVISION

TRADE NAME: FC-143 FLUORAD Brand Fluorochemical Surfactant

3M ID NUMBER/U.P.C.: ZF-0002-0378-4 98-0211-0008-0 00-51135-09138-8

98-0211-0891-9 00-51135-09365-8 98-0211-5489-7
00-51135-02941-1

98-0211-6581-0 00-51135-10405-7 98-0211-7394-7
00-51135-10762-1

| 1. INGREDIENT | C.A.S. NO. | PERCENT |
|---|---|---|
| AMMONIUM PERFLUOROOCTANOATE | 3825-26-1 | 93 - 97 |
| AMMONIUM PERFLUOROHEPTANOATE | 6130-43-4 | 1 - 3 |
| AMMONIUM PERFLUOROPENTANOATE | 68259-11-0 | 1 - 3 |
| AMMONIUM PERFLUOROHEXANOATE | 21615-47-4 | 0.1 - 1 |

2. PHYSICAL DATA

Boiling Point: N/A
Vapor Pressure: N/A
Vapor Density: N/A

EID089410

(Continued)

| | |
|---|---|
| Evaporation Rate: | N/A |
| Solubility in Water: | Apprec. |
| Sp. Gravity: | 0.4 - 0.5 Water=1 (Bulk) |
| Percent Volatile: | N/A |
| pH: | ca. 5 (0.5% Aqueous) |
| Viscosity: | N/D |
| Melting Point: | N/A |
| Appearance and Odor: | Light colored powder; slight odor |

3. FIRE AND EXPLOSION HAZARD DATA

| | |
|---|---|
| Flash Point: | Non-flammable |
| Flammable Limits - LEL: | N/A |
| Flammable Limits - UEL: | N/A |
| Autoignition Temperature: | N/A |
| Extinguishing Media: | Water, carbon dioxide, dry chemical, foam |

Special Fire Fighting Procedures:

Wear full protective clothing, including helmet, self-contained, positive pressure or pressure demand breathing apparatus, bunker coat and pants, bands around arms, waist and legs, face mask, and protective covering for exposed areas of the head.

Unusual Fire and Explosion Hazards:

See Hazardous Decomposition section for products of combustion.

4. REACTIVITY DATA

Stability: Stable

Incompatibility - Materials to Avoid: Not applicable

Hazardous Polymerization: Will not occur

Hazardous Decomposition Products:

Carbon monoxide and carbon dioxide, oxides of nitrogen, hydrogen fluoride, ammonia.

5. ENVIRONMENTAL INFORMATION

EID089411

Spill Response:

Observe precautions from other sections. Collect spilled material. Use wet sweeping compound or water to avoid dusting. Clean up residue. Place in a closed container.

Recommended Disposal:

Incinerate in an industrial or commerical facility in the presence of a combustible material. Combustion products will include HF. Disposal

alternative: Dispose of waste product in a facility permitted to accept chemical waste.

Environmental Data:

Chemical Oxygen Demand (COD) = Nil(.00070 g/g); 20-day Biochemical Oxygen Demand (BOD20) = Nil; Theoretical Oxygen Demand (ThOD) = 0.32 g/g; Fathead Minnow (Pimephales promelas) 96-hr LC50 = 740 mg/L; Water flea (Daphnia magna) 48-hr EC50 = 460 mg/L; Green Algae (Selenastrum capricornatum) 14-day EC50 (cell dry weight) = 73 mg/L; Green Algae (Selenastrum capricornutum) 14-day EC50 (cell count) = 43 mg/L

Bioconcentration factor (BCF) for ammonium perfluorooctanoate (PFO = 1.8)

Regulatory Information:

Volatile Organic Compounds: N/A
VOC Less H2O & Exempt Solvents: N/A

Since regulations vary, consult applicable regulations or authorities before disposal. U.S. EPA Hazardous Waste Number = None (Not U.S. EPA Hazardous).

The components of this product are in compliance with the chemical registration requirements of TSCA, EINECS, CDSL, AICS, MITI and KTCCL.

EPCRA Hazard Class:

Fire Hazard: No Pressure: No Reactivity: No Acute: Yes
Chronic: Yes

6. SUGGESTED FIRST AID

Eye Contact:

Immediately flush eyes with large amounts of water for at least 15 minutes. Ge immediate medical attention.

Skin Contact:

Flush skin with large amounts of water. If irritation persists, get medical attention.

Inhalation:

EID089412

If signs/symptoms occur, remove person to fresh air. If signs/symptoms continue, call a physician.

If Swallowed:

Do not induce vomiting. Drink two glasses of water. Call a physician.

7. PRECAUTIONARY INFORMATION

Eye Protection:

Avoid eye contact. Wear vented goggles.

Skin Protection:

Avoid skin contact. Wear appropriate gloves when handling this material. A pair of gloves made from the following material(s) are recommended: Butyl rubber. Use one or more of the followig personal protection items as necessary to prevent skin contact: head covering, coveralls. Protective garments (other than gloves) should be made of either of the following materials: Polyethylene/polyvinylidene chloride (Saranex).

Ventilation Protection:

Use with appropriate local exhaust ventilation. Provide sufficient ventilation to maintain emissions below recommended exposure limits. If exhaust ventilatio is not adequate, use appropriate respiratory protection.

Respiratory Protection:

Avoid breathing of airborne material. Select one of the following NIOSH approved respirators based on airborne concentration of contaminants and in accordance with OSHA regulations: full-face high-efficiency filter respirator, full-face supplied air respirator.

Prevention of Accidental Ingestion:

Do not eat, drink or smoke when using this product. Wash exposed areas thoroughly with soap and water. Wash hands after handling and before eating.

Recommended Storage:

Do not store containers on their sides. Store at room temperature. Keep container dry. Keep container closed when not in use.

**PREVENT MOISTURE CONTAMINATION TO KEEP POWDER FREE FLOWING.** EID089413

Fire and Explosion Avoidance:
Keep container tightly closed. No smoking while handling this material.

Other Precautionary Information:

HMIS HAZARD RATINGS: HEALTH: 3 FLAMMABILITY: 0 REACTIVITY: 0
PERSONAL PROTECTION: X (See precautions, section 7.)

EXPOSURE LIMITS

| Ingredients | Value | Unit | Type | Auth | Skin* |
|---|---|---|---|---|---|
| Ammonium Perfluorooctanoate | 0.01 | mg/m3 | TWA | ACGIH | Y |
| Ammonium Perfluoroheptanoate | 0.1 | mg/m3 | TWA | 3M | Y |
| Ammonium Perfluoropentanoate | 0.1 | mg/m3 | TWA | 3M | Y |

(Continued)

| | | | | | |
|---|---|---|---|---|---|
| Ammonium Perfluorohexanoate | 0.1 | mg/m3 | TWA | 3M | Y |

* Skin Notation: Listed substances indicated with "Y" under SKIN refer to the potential contribution to the overall exposure by the cutaneous route including mucous membrane and eye, either by airborne or, more particularly, by direct contact with the substance. Vehicles can alter skin absorption.

Source of Exposure Limit Data:
- ACGIH: American Conference of Governmental Industrial Hygienists
- AIHA: American Industrial Hygiene Assoc. Workplace Environmental Exposure Level
- 3M: 3M Medical Department Guideline

3. HEALTH HAZARD DATA

EYE CONTACT:

Moderate eye irritation: signs/symptoms can include redness, swelling, pain, tearing, and hazy vision.

Airborne product may cause eye injury consisting of corneal opacity.

SKIN CONTACT:

Product is not expected to be irritating to the skin.

Mild skin irritation (after prolonged or repeated contact): signs/symptoms can include redness, swelling, and itching.

May be absorbed through the skin and persist in the body for an extended time.

INHALATION:

Illness requiring medical attention may result from a single exposure by inhalation to moderate quantities of this material.

May be absorbed by inhalation and persist in the body for an extended time.

Repeated inhalation of airborne product above the exposure guideline can result in elevated organofluoride levels in the blood. Single overexposure, above recommended guidelines, may cause:

Irritation (upper respiratory): signs/symptoms can include soreness of the nose and throat, coughing and sneezing.

Prolonged or repeated overexposure, above recommended guidelines, may cause:

Liver Effects: signs/symptoms can include yellow skin (jaundice) and tenderness of upper abdomen.

IF SWALLOWED: EID089414

Ingestion is not a likely route of exposure to this product.

Illness may result from a single swallowing of a moderate quantity of this material.

CANCER: A mixture of ammonium perfluorooctanoate, ammonium perfluoroheptanoate ammonium perfluoropentanoate and ammonium perfluorohexanoate, that was 93 to 9 AMMONIUM PERFLUOROOCTANOATE (3825-26-1) was fed to albino rats for 2 years, no compound induced carcinogenicity was found in the study. There were statistically significant compound realted benign testicular tumors. In a second two-year study there were statisticallly significant compound related benign tumors in the liver, pancreas, and testis when compared to ad libitum a pair-fed controls. Based on the current knowledge, these findings have no huma health implications. (1983 and 1993 studies conducted jointly by 3M and DuPont).

MUTAGENICITY: Not mutagenic in invitro mutagenicity assays. Did not cause cell transformation in a mammalian cell transformation assay.

REPRODUCTIVE/DEVELOPMENTAL TOXINS:
Not teratogenic in rabbits by oral administration. Not teratogenic to rats by gavage or inhalation exposures.

OTHER HEALTH HAZARD INFORMATION:
A 3M Product Toxicity Summary Sheet is available.

SECTION CHANGE DATES

HEADING Section changed since May 20, 1996 issue MSDS: FC-143
FLUORAD Brand Fluorchemical Surfactant
August 23, 1996

The information in this Material Safety Data Sheet (MSDS) is believed to be correct as of the date issued. 3M MAKES NO WARRANTIES, EXPRESSED OR IMPLIED, INCLUDING, BUT NOT LIMITED TO, ANY IMPLIED WARRANTY OF MERCHANTABILITY OR FITNESS FOR A PARTICULAR PURPOSE OR COURSE OF PERFORMANCE OR USAGE OF TRADE. User is responsible for determining whether the 3M product is fit for a particular purpose and suitable for user's method of use or application. Given the variety of factors that can affect the use and application of a 3M product, some of which are uniquely within the user's knowledge and control, it is essential that the user evaluate the 3M product to determine whether it is fit for a particular purpose and suitable for user's method of use or application.

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The data in this Material Safety Data Sheet relates only to the specific material designated herein and does not relate to use in combination with any other material or in any process.

End of MSDS EID089415

Fl-C8

Kris Hansen
3M Environmental Lab
Fluorine Analytical Chemistry Team
2-3E-09

# 3M Environmental Lab

September 2, 1999

Paul Lieder
3M Toxicology Services
220-2E-02

Dear Paul Lieder:

Here are preliminary results for POAA levels in sera and liver for TOX026 (POAA Monkey Study). As I said in my original email message, please do not make these results public. We provide them only to help you make better decisions about the status of current and future projects. I understand you need to use the data to ascertain trends, but please do not publish or release specific values.

As stated in an earlier communication, this data has all been QC'd, but not QA'd. These are interim results that have been through peer review by the scientific staff in the Environmental Lab. By definition, interim results may change before they are entered in final reports if errors are discovered as the data move through the review process, including pending review by the Environmental Lab QAU. I trust you understand the potential risks involved with interim data.

Sincerely,

Kris Hansen
3M Environmental Lab

cc Dale bacon 2-3E-09 John Butenhoff 220-2E-02

GWJ000050

EID088631

## Covance# 6329-231

| | |
|---|---|
| Study: | 6 Month Capsule T |
| Product Number(Test Substance): | T-6889.2 (POAA) |
| Matrix: | Monkey Liver |
| Method/Revision: | FACT-M-1.0 & FA |
| Analytical Equipment System Number: | Soup020199 |
| Instrument Software/Version: | MassLynx 3.2 |
| Date of Extraction/Analyst: | 7/13/99 SEE |
| Date of Analysis/Analyst: | 07/21/99 DRB |
| Date of Data Reduction/Analyst: | 07/22/99 DRB |

**Sample Data**

**MONKEY LIVER Week 20 and 40**

| Group Dose | Sample # | POAA Calc. Conc. ng/g | Amount of POAA ug/g |
|---|---|---|---|
| Method Blk | H2O Blk-1 | 0.0 | <LOD |
| | H2O Blk-2 | 0.0 | <LOD |
| Matrix Blk | Rabbit Liver Blk-1 | 2.8 | <LOD |
| | Rabbit Liver Blk-2 | 0.0 | <LOD |
| QC - 250 ppb | I05718M-MS-11 | 297 | 99% |
| | I05718M-MSD-12 | 278 | 92% |
| Group 1, Wk40 | I05718M | 12.3 | 0.0123 |
| 0.0 mg/kg/day | I05720M | 11.8 | 0.0118 |
| Group 2, Wk20 | I05721M | 17874 | 17.9 |
| Group 3, Wk40 | I05722M | 286 | 0.286 |
| 10 mg/kg/day | I05716M | 84.4 | 0.0844 |

POAA = Perfluorooctanoate
Limit of Quantitation (LOQ) = 30 ppb
Limit of Detection (LOD) = 15 ppb

Date Entered/By: 07/26/99 LAC
Date Verified/ By: 08/03/99 GML

EID088632

GW10000051

## Covance# 6329-231

| | |
|---|---|
| Study: | 6 Month Capsule T |
| Product Number(Test Substance): | T-6889.2 (POAA) |
| Matrix: | Monkey Liver |
| Method/Revision: | FACT-M-1.0 & FA |
| Analytical Equipment System Number: | Soup020199 |
| Instrument Software/Version: | MassLynx 3.2 |
| Date of Extraction/Analyst: | 06/21/99 SAH |
| Date of Analysis/Analyst: | 06/23/99, 07/28/99 |
| Date of Data Reduction/Analyst: | 06/25/99, 07/29/99 |

**Sample Data**

**MONKEY LIVER Week 27**

| Group Dose | Sample # | POAA Calc. Conc. ng/g | Amount of POAA ug/g |
|---|---|---|---|
| Method Blk | H2O Blk-1 | 10.7 | <LOD |
| | H2O Blk-2 | 0.0 | <LOD |
| Matrix Blk | Rabbit Liver Blk-1 | 11.8 | <LOD |
| | Rabbit Liver Blk-2 | 14.0 | <LOD |
| QC - 250 ppb | I05709M-MS-3 | 219 | 74% |
| | I05709M-MSD-3 | 184 | 62% |
| Group 1 | I05709M Wk27 | 93.5 | 0.0935 |
| Control | I05714M Wk27 | 54.3 | 0.0543 |
| 0.0 mg/kg/day | I05715M Wk27 | 235 | 0.235 |
| | I05725M Wk27 | 41.8 | 0.0418 |
| Group 2 | I05702M Wk27 | 15723 | 15.7 |
| Mid Dose | I05706M Wk27 | 19209 | 19.2 |
| 3.0 mg/kg/day | I05717M Wk27 | 11629 | 11.6 |
| 5721M replacement | I05723M | 16873 | 16.9 |
| Group 3 | I05707M Wk27 | 22764 | 22.8 |
| Mid-High Dose | I05708M Wk27 | 6488 | 6.49 |
| 10 mg/kg/day | I05710M Wk27 | 9069 | 9.07 |
| | I05719M Wk27 | 19444 | 19.4 |
| Group 4 | I05703M Wk27 | 1350 | 1.35 |
| High Dose | I05704M Wk27 | 16582 | 16.6 |
| 30 mg/kg/day | I05711M Wk27 | 224 | 0.224 |
| | I05713M Wk27 | 86933 | 86.9 |
| | I05722M Wk27 | 0 | 0.00 |
| | I05724M Moribund | 160808 | 161 |

POAA = Perfluorooctanoate
Limit of Quantitation (LOQ) = 30 ppb
Limit of Detection (LOD) = 15 ppb

Date Entered/By: 06/24/99, 06/25/99, 07/29/99 LAC
Date Verified/ By: 08/03/99 GML

EID088633
GW/0000052

| | |
|---|---|
| Study | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance) | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | FACT-M-3 1 & FACT-M-4 1 |
| Analytical Equipment System Number: | Amelia 062498 |
| Instrument Software/Version: | MassLynx 3 1 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 11/13/98 IAS |
| Date of Analysis/Analyst: | 11/16/98, 11/24/98, 12/03/98 HOJ |
| Date of Data Reduction/Analyst: | 11/17/98, 11/30/98, 12/07/98 HOJ |

## Sample Data

### Day 9 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL, | Average POAA ug/mL, | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 500 ppb | RBS11138-MS | 109% | | |
| | RBS11138-MSD | 100% | 104% | 8% |
| Group 1 | 105709M | 0.0372 | | |
| Control | 105714M | 0 1158 | | |
| 0 0 mg/kg/day | 105715M | <MDL | | |
| | 105718M | 0.0638 | | |
| | 105720M | <MDL | | 69 5 |
| | 105725M | 0 0214 | 0 0595 | 0 0414 |
| Group 2 | 105702M | 86 1 | | |
| Low Dose | 105706M | 173 | | |
| | 105717M | 110 | | |
| | 105721M | NR | | 29.5 |
| | 105723M | 124 | 123 | 36.4 |
| Group 3 | 105707M | 224 | | |
| Mid-High Dose | 105708M | 91.5 | | |
| 10 mg/kg/day | 105710M | 162 | | |
| | 105712M | 172 | | |
| | 105716M | 222 | | 28 2 |
| | 105719M | 213 | 181 | 50 9 |
| Group 4 | 105703M | 6656 | | |
| High Dose | 105704M | 291 | | |
| 30 mg/kg/day | 105711M | 1741 | | |
| | 105713M | 208 | | |
| | 105722M | 164 | | 152 |
| | 105724M | 883 | 1657 | 2522 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL.

POAA = Perfluorooctanoate

Tentative value, sample evaporated to dryness

Date Entered/By: 11/21/98, 12/07/98 LAC
Date Verified/ By: 06/21/99 EAD

EID088634

GWJ0000053

## Covance# 6329-231

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | FACT-M-3 1 & FACT-M-4 1 |
| Analytical Equipment System Number: | Amelia 062498 |
| Instrument Software/Version: | MassLynx 3.1 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 11/13/98 IAS |
| Date of Analysis/Analyst: | 11/16/98, 11/24/98, 12/03/98, 01/04/99 HOJ |
| Date of Data Reduction/Analyst: | 11/17/98, 11/30/98, 12/07/98, 01/07/99 HOJ |

### Sample Data

**Week 4 MONKEY SERA**

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 500 ppb | RBS11138-MS | 109% | | |
| | RBS11138-MSD | 100% | 104% | 8% |
| Group 1 | I05709M | 0 313 | | |
| Control | I05714M | 0 240 | | |
| 0 0 mg/kg/day | I05715M | 0 0819 | | |
| | I05718M | 0 362 | | |
| | I05720M | 0 174 | | 51 1 |
| | I05725M | 0 119 | 0.215 | 0 110 |
| Group 2 | I05702M | 89.7 | | |
| Low Dose | I05706M | 3820 * | | 43 4* |
| | I05717M | 135 | 93.1* | 40.4* |
| | I05721M | 54 5 | | 182 |
| | I05723M | NR | 1025 | 1864 |
| Group 3 | I05707M | 206 | | |
| Mid-High Dose | I05708M | 87 8 | | |
| 10 mg/kg/day | I05710M | 279 | | |
| | I05712M | E | | |
| | I05716M | 124 | | 41 8 |
| | I05719M | 203 | 180 | 75 3 |
| Group 4 | I05703M | 297 | | |
| High Dose | I05704M | 264 | | |
| 30 mg/kg/day | I05711M | 4962 | | |
| | I05713M | 69 4 | | |
| | I05722M | 58 3 | | 178 |
| | I05724M | 836 | 1081 | 1922 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL

* Outlier, was not included in these values.
NR = Sample not received not reported.
E = Sample evaporated, not analyzed.

POAA = Perfluorooctanoate

Date Entered/By: 11/21/98, 12/07/98, 01/12/99 LAC
Date Verified/ By: 06/21/99 EAD

EID088635
GWJ0000054

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4 1 and ETS-8-5 1 |
| Analytical Equipment System Number: | Amelia 062498, Soup 020199 |
| Instrument Software/Version: | MassLynx 3 2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 05/12/99 SAH |
| Date of Analysis/Analyst: | 06/03/99, 06/07/99, 06/10/99, 06/16/99 HOJ/SAH/MEE/LAC |
| Date of Data Reduction/Analyst: | 06/04/99, 06/08/99, 06/11/99, 06/17/99 HOJ/LAC |

## Sample Data

### Week 6 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | RBS05129-MS-3 | 78% | | |
| | RBS05129-MSD-3 | 78% | 78% | 0% |
| | RBS05129-MS-4 | 87% | | |
| | RBS05129-MSD-4 | 82% | 84% | 5% |
| Group 1 | 105709M | 0 166 | | |
| Control | 105714M | 0.160 | | |
| 0 0 mg/kg/day | 105715M | 0 093 | | |
| | 105718M | 0 133 | | |
| | 105720M | 0 092 | | 27.9 |
| | 105725M | 0 096 | 0.123 | 0 0343 |
| Group 2 | 105702M | 70 5 | | |
| Low Dose | 105706M | 120 | | |
| | 105717M | 87 3 | | |
| | 105721M | 149 | | 32 8 |
| | 105723M | NR | 107 | 35.1 |
| Group 3 | 105707M | 88 | | |
| Mid-High Dose | 105708M | 82 2 | | |
| 10 mg/kg/day | 105710M | 107 4 | | |
| | 105712M | 92 | | |
| | 105716M | 90 | | 21.5 |
| | 105719M | 140 | 100 0 | 21.5 |
| Group 4 | 105703M | 169 | | |
| High Dose | 105704M | 115 | | |
| 30 mg/kg/day | 105711M | 167.9 | | |
| | 105713M | 161 | | 14 9 |
| | 105722M | 143 | 151 | 23 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL. NR = Sample not received nor reported.

POAA = Perfluorooctanoate

Date Entered/By: 06/08/99, 06/14/99, 06/17/99 LAC
Date Verified/ By: 06/21/99 EAD

EID088636
GW/0000055

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889.2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 and ETS-8-5.1 |
| Analytical Equipment System Number: | Amelia 062498 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 05/12/99 SAH |
| Date of Analysis/Analyst: | 06/03/99, 06/07/99, 06/10/99 HOJ |
| Date of Data Reduction/Analyst: | 06/04/99, 06/08/99, 06/11/99 HOJ |

## Sample Data

### Week 8 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | RBS05129-MS-3 | 78% | | |
| | RBS05129-MSD-3 | 78% | 78% | 0% |
| | RBS05129-MS-4 | 87% | | |
| | RBS05129-MSD-4 | 82% | 84% | 5% |
| Group 1 | 105709M | 0.242 | | |
| Control | 105714M | 0.162 | | |
| 0.0 mg/kg/day | 105715M | 0.0726 | | |
| | 105718M | 0.0166 | | |
| | 105720M | 0.129 | | 65.5 |
| | 105725M | 0.0925 | 0.119 | 0.0780 |
| Group 2 | 105702M | 79.8 | | |
| Low Dose | 105706M | 106 | | |
| | 105717M | 73.5 | | |
| | 105721M | 136 | | 28.8 |
| | 105723M | NR | 99 | 28.5 |
| Group 3 | 105707M | 101 | | |
| Mid-High Dose | 105708M | 70.7 | | |
| 10 mg/kg/day | 105710M | 92.4 | | |
| | 105712M | 86 | | |
| | 105716M | 107 | | 24.1 |
| | 105719M | 138 | 101.9 | 24.6 |
| Group 4 | 105703M | 183 | | |
| High Dose | 105704M | 122 | | |
| 30 mg/kg/day | 105711M | 106.8 | | |
| | 105713M | 105 | | 59.6 |
| | 105722M | 349 | 173 | 103 |

Limit of Quantitation (LOQ) 10 ng/mL
Method Detection Limit (MDL) 5.0 ng/mL.

NR = Sample not received nor reported.

POAA = Perfluorooctanoate

Date Entered/By: 06/08/99, 06/14/99 LAC
Date Verified/ By: 06/21/99 EAD

EID088637

GW10000056

| | |
|---|---|
| Study | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4 1 and ETS-8-5 1 |
| Analytical Equipment System Number | Madeline 041098 |
| Instrument Software/Version: | MassLynx 3 2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 05/13/99 SRP/JCP |
| Date of Analysis/Analyst: | 05/17/99, 05/19/99, 06/10/99 MEE/КЛИЮЛ |
| Date of Data Reduction/Analyst: | 05/18/99, 05/20/99, 06/11/99 КЛИ/MEE/ЮЛ |

## Sample Data

**Week 10 MONKEY SERA**

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <LOQ | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | 0 03174 | | |
| | Rabbit Sera Blk-2 | 0 03454 | 0 03314 | 0 |
| Matrix Blk | Monkey Sera Blk-1 | 0 08671 | | |
| | Monkey Sera Blk-2 | 0 04897 | 0 06784 | 0 |
| QC - 250 ppb | RBS05129-MS-7 | 103% | | |
| | RBS05129-MSD-7 | 105% | 104% | 2% |
| | RBS05129-MS-8 | 103% | | |
| | RBS05129-MSD-8 | 127% | 115% | 20% |
| Group 1 | 105709M | 0 202 | | |
| Control | 105714M | 0.111 | | |
| 0 0 mg/kg/day | 105715M | 0.0976 | | |
| | 105718M | 0 137 | | |
| | 105720M | 0 136 | | 27.5 |
| | 105725M | 0 118 | 0 134 | 0 0367 |
| Group 2 | 105702M | 79 2 | | |
| Low Dose | 105706M | 119 | | |
| | 105717M | 78 0 | | |
| | 105721M | 161 | | 36 0 |
| | 105723M | NR | 109 | 39 4 |
| Group 3 | 105707M | 102 | | |
| Mid-High Dose | 105708M | 76 0 | | |
| 10 mg/kg/day | 105710M | 91.7 | | |
| | 105712M | 107 | | |
| | 105716M | 107 | | 14 6 |
| | 105719M | 112 | 97.7 | 14 3 |
| Group 4 | 105703M | 327 | | |
| High Dose | 105704M | 134 | | |
| 30 mg/kg/day | 105711M | 70 3 | | |
| | 105713M | 217 | | 66 8 |
| | 105722M | 488 | 247 | 165 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL

NR = Sample not received nor reported
E = Sample evaporated, not analyzed

POAA = Perfluorooctanoate

Date Entered/By: 05/20/99, 05/25/99, 06/14/99 LAC

EID088638
GW1000057

| | |
|---|---|
| Study | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance) | T-6889 2 (POAA) |
| Matrix | Monkey Sera |
| Method/Revision: | FACT-M-3 1 & FACT-M-4 1 |
| Analytical Equipment System Number: | Madeline 041098 |
| Instrument Software/Version | MassLynx 3 1 |
| Filename | See Attachments |
| R-Squared Value: | See Attachments |
| Slope | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 01/08/99 SAH/JCP |
| Date of Analysis/Analyst | 01/21/99, 01/27/99 HOJ/MEE |
| Date of Data Reduction/Analyst | 01/25/99, 01/28/99 KJH/MEE |

## Sample Data

### Week 12 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | RBS01089-MS | 101% | | |
| | RBS01089-MSD | 99% | 100% | 2% |
| Group 1 | 105709M | 0 214 | | |
| Control | 105714M | 0 101 | | |
| 0 0 mg/kg/day | 105715M | 0 0619 | | |
| | 105718M | 0 139 | | |
| | 105720M | 0 143 | | 43 5 |
| | 105725M | 0 0852 | 0 124 | 0 0540 |
| Group 2 | 105702M | 67 9 | | |
| Low Dose | 105706M | 90 8 | | |
| | 105717M | 58 9 | | |
| | 105721M | 121 | | 32 8 |
| | 105723M | NR | 84 7 | 27 8 |
| Group 3 | 105707M | 100 | | |
| Mid-High Dose | 105708M | 60 2 | | |
| 10 mg/kg/day | 105710M | 83 8 | | |
| | 105712M | 142 | | |
| | 105716M | 89 9 | | 27 9 |
| | 105719M | 105 | 96.7 | 27 0 |
| Group 4 | 105703M | 240 | | |
| High Dose | 105704M | 110 | | |
| 30 mg/kg/day | 105711M | 34 3 | | |
| | 105713M | 193 | | |
| | 105722M | 148 | | 54 3 |
| | 105724M | NR | 145 | 78 9 |

Limit of Quantitation (LOQ) 10 ng/mL
Method Detection Limit (MDL) 5 0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 01/27/99, 01/28/99 LAC
Date Verified/ By: 06/21/99 EAD

EID088639
GWJ00058

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | FACT-M-3.1 & FACT-M-4 1 |
| Analytical Equipment System Number: | Madeline 041098 |
| Instrument Software/Version: | MassLynx 3 1 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 01/08/99 SAH/JCP |
| Date of Analysis/Analyst: | 01/21/99 HOJ |
| Date of Data Reduction/Analyst: | 01/25/99 KJH |

## Sample Data

**Week 14 MONKEY SERA**

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | RBS01089-MS | 101% | | |
| | RBS01089-MSD | 99% | 100% | 2% |
| Group 1 | 105709M | 0.288 | | |
| Control | 105714M | 0 159 | | |
| 0 0 mg/kg/day | 105715M | 0.0973 | | |
| | 105718M | 0 157 | | |
| | 105720M | 0 182 | | 41 9 |
| | 105725M | 0.105 | 0.165 | 0 0689 |
| Group 2 | 105702M | 77.4 | | |
| Low Dose | 105706M | 116 | | |
| | 105717M | 62 0 | | |
| | 105721M | 124 | | 31 6 |
| | 105723M | NR | 94.9 | 30 0 |
| Group 3 | 105707M | 70 0 | | |
| Mid-High Dose | 105708M | 105 | | |
| 10 mg/kg/day | 105710M | 65 2 | | |
| | 105712M | 84 5 | | |
| | 105716M | 139 | | 29 5 |
| | 105719M | 115 | 96.4 | 28 4 |
| Group 4 | 105703M | 89.6 | | |
| High Dose | 105704M | 116 | | |
| 30 mg/kg/day | 105711M | 72 1 | | |
| | 105713M | 38 5 | | |
| | 105722M | 105 | | 36 1 |
| | 105724M | NR | 84 2 | 30 4 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 01/27/99 LAC
Date Verified/ By: 06/21/99 EAD

EID088640

GW10000059

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance) | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4 1 and ETS-8-5 1 |
| Analytical Equipment System Number: | Madeline 041098 |
| Instrument Software/Version: | MassLynx 3.1 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept. | See Attachments |
| Date of Extraction/Analyst: | 05/13/99 SRP/JCP |
| Date of Analysis/Analyst | 05/17/99, 05/19/99, 06/10/99 MEE |
| Date of Data Reduction/Analyst: | 05/18/99, 05/20/99, 06/11/99 KJH/MEE/HOJ |

## Sample Data

### Week 16 MONKEY SERA

| Group Dose | Sample # | POAA Reported ng/mL | Average POAA ng/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <LOQ | | |
| | H2O Blk-2 | <MDL | #DIV/0! | #DIV/0! |
| Matrix Blk | Rabbit Sera Blk-1 | 0 03174 | | |
| | Rabbit Sera Blk-2 | 0 03454 | 0 03314 | 0 |
| Matrix Blk | Monkey Sera Blk-1 | 0 08671 | | |
| | Monkey Sera Blk-2 | 0 04897 | 0 06784 | 0 |
| QC - 500 ppb | RBS05129-MS-7 | 104% | | |
| | RBS05129-MSD-7 | 106% | 105% | 2% |
| | RBS05129-MS-8 | 104% | | |
| | RBS05129-MSD-8 | 128% | 116% | 20% |
| Group 1 | I05709M | 0 298 | | |
| Control | I05714M | 0.152 | | |
| 0 0 mg/kg/day | I05715M | 0 0840 | | |
| | I05718M | 0 168 | | |
| | I05720M | 0 168 | | 48 8 |
| | I05725M | 0 0870 | 0.159 | 0 0778 |
| Group 2 | I05702M | 57.7 | | |
| Low Dose | I05706M | 75.7 | | |
| | I05717M | 45.3 | | |
| | I05721M | 108 | | 37.9 |
| | I05723M | NR | 71 6 | 27.1 |
| Group 3 | I05707M | 104 | | |
| Mid-High Dose | I05708M | 62 8 | | |
| 10 mg/kg/day | I05710M | 76 4 | | |
| | I05712M | 188 | | |
| | I05716M | 95 2 | | 43.0 |
| | I05719M | 90 3 | 103 | 44 1 |
| Group 4 | I05703M | 71.2 | | |
| High Dose | I05704M | 118 | | |
| 30 mg/kg/day | I05711M | 138 | | |
| | I05713M | 174 | | |
| | I05722M | 60 3 | | 69.7 |
| | I05724M | NR | 87 6 | 61.1 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL

NR = Sample not received nor reported.
E = Sample evaporated, not analyzed

POAA = Perfluorooctanoate

Date Entered/By: 05/20/99, 06/14/99 LAC
Date Verified/ By: 06/21/99, 06/22/99 EAD/LAC

EID088641
GW1000060

| | |
|---|---|
| Study | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance) | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4 1 and ETS-8-5.1 |
| Analytical Equipment System Number: | Madeline 041098, Amelia 062498 |
| Instrument Software/Version: | MassLynx 3 2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 05/14/99 SEE |
| Date of Analysis/Analyst: | 05/18/99, 05/24/99, 05/25/99, 06/07/99 MEE/HOJ/SAH |
| Date of Data Reduction/Analyst: | 05/19/99, 05/25/99, 05/27/99, 06/08/99 MEE/HOJ |

## Sample Data

### Week 18 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <LOQ | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | RBS05149-MS-11 | 105% | | |
| | RBS05149-MSD-11 | 84% | 94% | 22% |
| | RBS05149-MS-12 | 88% | | |
| | RBS05149-MSD-12 | 71% | 80% | 22% |
| Group 1 | 105709M | 0 275 | | |
| Control | 105714M | 0 254 | | |
| 0 0 mg/kg/day | 105715M | 0.0991 | | |
| | 105718M | 0 172 | | |
| | 105720M | 0 202 | | 34 8 |
| | 105725M | 0 144 | 0.191 | 0 0665 |
| Group 2 | 105702M | 11 6 | | |
| Low Dose | 105706M | 16.7 | | |
| | 105717M | 69.7 | | 86 5 |
| | 105721M | 24 9 | 30.7 | 26 6 |
| Group 3 | 105707M | 14 0 | | |
| Mid-High Dose | 105708M | 10 1 | | |
| 10 mg/kg/day | 105710M | 48 4 | | |
| | 105712M | 14 3 | | |
| | 105716M | 10 5 | | 82 1 |
| | 105719M | 11 8 | 18 2 | 14 9 |
| Group 4 | 105703M | 71 8 | | |
| High Dose | 105704M | 20 9 | | |
| 30 mg/kg/day | 105711M | 19 8 | | |
| | 105713M | 28 5 | | 58 5 |
| | 105722M | 47 9 | 37 8 | 22 1 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 05/19/99, 05/25/99, 05/27/99, 06/08/99 LAC
Date Verified/ By: 06/21/99 EAD

EID088642

GW1000061

## Covance# 6329-231

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 and ETS-8-5.1 |
| Analytical Equipment System Number: | Madeline 041098, Amelia 062498, Soup 020199 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 05/14/99 SEE |
| Date of Analysis/Analyst: | 05/18/99, 05/24/99, 05/25/99, 06/10/99, 06/16/99 MEE/IOJ/SAH/LAC |
| Date of Data Reduction/Analyst: | 05/19/99, 05/25/99, 05/27/99, 06/11/99, 06/17/99 MEE/IOJ/LAC |

### Sample Data

**Week 20 MONKEY SERA**

| Group Dose | Sample # | POAA Reported ug/ml. | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| Method Blk | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| Matrix Blk | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | RBS05149-MS-11 | 104% | | |
| | RBS05149-MSD-11 | 83% | 94% | 22% |
| QC - 250 ppb | RBS05149-MS-12 | 87% | | |
| | RBS05149-MSD-12 | 70% | 79% | 22% |
| Group 1 | I05709M | 0.357 | | |
| Control | I05714M | 0 221 | | |
| 0 0 mg/kg/day | I05715M | 0 120 | | |
| | I05718M | 0.234 | | |
| | I05720M | 0 253 | | 32 6 |
| | I05725M | 0 216 | 0 234 | 0 0762 |
| Group 2 | I05702M | 18.6 | | |
| Low Dose | I05706M | 11 9 | | |
| | I05717M | 34 8 | | 78 8 |
| | I05721M | 72 6 | 34 5 | 27 2 |
| Group 3 | I05707M | 112 | | |
| Mid-High Dose | I05708M | 64.3 | | |
| 10 mg/kg/day | I05710M | 78 5 | | |
| | I05712M | 143 | | |
| | I05716M | 95.3 | | 27 6 |
| | I05719M | 112 | 101 | 27 8 |
| Group 4 | I05703M | 40.6 | | |
| High Dose | I05704M | 96.6 | | |
| 30 mg/kg/day | I05711M | 2 49 | | |
| | I05713M | 336 | | 132 |
| | I05722M | 34.4 | 102 | 135 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 05/20/99, 05/25/99, 05/27/99, 06/14/99, 06/17/99 LAC
Date Verified/ By: 06/21/99, 06/22/99 EAD/LAC



EID088643
GWJ00062

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889.2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 and ETS-8-5.1 |
| Analytical Equipment System Number: | Amelia 062498, Madeline 041098 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 05/14/99 MCH |
| Date of Analysis/Analyst: | 05/20/99, 05/26/99, 05/28/99, 06/10/99 HOJ/MEE |
| Date of Data Reduction/Analyst: | 05/24/99, 05/27/99, 06/01/99, 06/11/99 HOJ/MEE |

## Sample Data

### Week 22 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-9 | <MDL | | |
| | H2O Blk-10 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-9 | <MDL | | |
| | Rabbit Sera Blk-10 | <MDL | <MDL | <MDL |
| QC - 250 ppb | RBS05149-MS-9 | 103% | | |
| | RBS05149-MSD-9 | 108% | 106% | 4% |
| Group 1 | I05709M | 0.410 | | |
| Control | I05714M | 0.164 | | |
| 0.0 mg/kg/day | I05715M | 0.111 | | |
| | I05718M | 0.280 | | |
| | I05720M | 0.385 | | 49.5 |
| | I05725M | 0.169 | 0.254 | 0.126 |
| Group 2 | I05702M | 72.9 | | |
| Low Dose | I05706M | 110 | | 32.1 |
| | I05717M | 60.0 | 81.0 | 26.0 |
| Group 3 | I05707M | 127 | | |
| Mid-High Dose | I05708M | 66.5 | | |
| 10 mg/kg/day | I05710M | 91.6 | | |
| | I05712M | 175 | | |
| | I05716M | 111 | | 34 |
| | I05719M | 88.9 | 109.9 | 37.9 |
| Group 4 | I05703M | 17.2 | | |
| High Dose | I05704M | 96.8 | | |
| 30 mg/kg/day | I05711M | 1.91 | | |
| | I05713M | 100 | | 77.8 |
| | I05722M | 89.7 | 61.7 | 47.6 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5.0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 05/25/99, 05/27/99, 06/02/99, 06/14/99 LAC
Date Verified/ By: 06/21/99, 06/22/99 EAD/LAC

EID088644
GW1000063

## Covance# 6329-231

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4 1 and ETS-8-5 1 |
| Analytical Equipment System Number: | Amelia 062498 |
| Instrument Software/Version: | MassLynx 3 2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 05/14/99 MCH |
| Date of Analysis/Analyst: | 05/20/99, 05/26/99, 05/28/99 HOJ/SAH/MEE |
| Date of Data Reduction/Analyst: | 05/24/99, 05/27/99, 06/01/99 HOJ/MEE |

### Sample Data

**Week 24 MONKEY SERA**

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-9 | <MDL | | |
| | H2O Blk-10 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-9 | <MDL | | |
| | Rabbit Sera Blk-10 | <MDL | <MDL | <MDL |
| QC - 250 ppb | RBS05149-MS-9 | 103% | | |
| | RBS05149-MSD-9 | 108% | 106% | 4% |
| Group 1 | I05709M | Ext | | |
| Control | I05714M | Ext | | |
| 0 0 mg/kg/day | I05715M | 0 171 | | |
| | I05718M | 0 190 | | |
| | I05720M | 0 362 | | 46 4 |
| | I05725M | 0 139 | 0 216 | 0 0999 |
| Group 2 | I05702M | 10 6 | | |
| Low Dose | I05706M | 153 | | 95 3 |
| | I05717M | 62 8 | 75 4 | 71 8 |
| Group 3 | I05707M | 93 0 | | |
| Mid-High Dose | I05708M | 64 1 | | |
| 10 mg/kg/day | I05710M | 85 4 | | |
| | I05712M | 121 | | |
| | I05716M | 85 8 | | 23 3 |
| | I05719M | 120 | 94 9 | 22 1 |
| Group 4 | I05703M | 21 25 | | |
| High Dose | I05704M | 94 3 | | |
| 30 mg/kg/day | I05711M | 4 48 | | |
| | I05713M | 189 | | 119 |
| | I05722M | 17 8 | 65 4 | 77 6 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5 0 ng/mL

Ext: Samples lost during sample extraction.

POAA = Perfluorooctanoate

Date Entered/By: 05/25/99, 05/27/99, 06/02/99 LAC
Date Verified/ By: 06/21/99 EAD

EID088645
GW1000064

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4 1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Amelia 062498 |
| Instrument Software/Version: | MassLynx 3 2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 04/29/99, 05/14/99 SAH |
| Date of Analysis/Analyst: | 05/17/99, 05/20/99, 05/26/99, 06/10/99 HOJ/SAH/MEE |
| Date of Data Reduction/Analyst: | 05/19/99, 05/24/99, 05/27/99, 06/11/99 HOJ/MEE |

**Sample Data**

**Week 26 MONKEY SERA**

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| Method Blk | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| Matrix Blk | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | MKS05149-MS | 80% | | |
| | MKS05149-MSD | 86% | 83% | 7% |
| Group 1 | I05709M | 0 458 | | |
| Control | I05714M | 0.102 | | |
| 0.0 mg/kg/day | I05715M | 0.0956 | | |
| | I05718M | 0 215 | | |
| | I05720M | 0 322 | | 75.1 |
| | I05725M | 0 057 | 0.208 | 0 156 |
| Group 2 | I05702M | 130 | | |
| Mid Dose | I05706M | 147 | | 23 3 |
| | I05717M | 91 4 | 123 | 28 7 |
| Group 3 | I05707M | 81 3 | | |
| Mid-High Dose | I05708M | 57 8 | | |
| 10 mg/kg/day | I05710M | 101 | | 21 8 |
| | I05719M | 83 1 | 80 8 | 17.6 |
| Group 4 | I05703M | 11.6 | | |
| High Dose | I05704M | 64 4 | | |
| 30 mg/kg/day | I05711M | 1.47 | | |
| | I05713M | 312 | | 162 |
| | I05722M | 16 5 | 81.1 | 131 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5.0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 05/19/98, 05/27/99, 06/14/99 LAC
Date Verified/ By: 06/21/99, 06/22/99 EAD/LAC

EID088646
GWJ00006S

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889.2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Amelia 062498 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 04/29/99 SAH |
| Date of Analysis/Analyst: | 04/30/99, 05/03/99, 05/04/99, 05/05/99 KJH/MEE/HOJ |
| Date of Data Reduction/Analyst: | 05/03/99, 05/04/99, 05/05/99, 05/25/99 HOJ/KJH |

## Sample Data

### Week 26, 27 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| Method Blk | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| Matrix Blk | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | I05709M-MS | 79% | | |
| | I05714M-MS | 108% | 94% | 17% |
| Group 1 | I05709M Wk27 | 0.393 | | |
| Control | I05714M Wk27 | 0.229 | | |
| 0.0 mg/kg/day | I05715M Wk27 | 0.099 | | |
| | I05718M Wk26 | 0.215 | | |
| | I05720M Wk26 | 0.322 | | 47.1 |
| | I05725M Wk27 | 0.142 | 0.233 | 0.110 |
| Group 2 | I05702M Wk27 | 49.7 | | |
| Mid Dose | I05706M Wk27 | 66.9 | | |
| | I05717M Wk27 | 45.7 | | |
| | I05721M Moribund | 47.7 | | 17.4 |
| | I05723M | 62.2 | 54.5 | 9.48 |
| Group 3 | I05707M Wk27 | 126 | | |
| Mid-High Dose | I05708M Wk27 | 33.3 | | |
| 10 mg/kg/day | I05710M Wk27 | 49.4 | | |
| | I05712M Wk26 | 104 | | |
| | I05716M Wk26 | 81.0 | | 44.7 |
| | I05719M Wk27 | 67.6 | 76.9 | 34.4 |
| Group 4 | I05703M Wk27 | 7.76 | | |
| High Dose | I05704M Wk27 | 60.8 | | |
| 30 mg/kg/day | I05711M Wk27 | 0.909 | | |
| | I05713M Wk27 | 192 | | |
| | I05722M Wk27 | 6.90 | | 151 |
| | I05724M Moribund | 508 | 53.7 | 81.0 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5.0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 05/04/99, 05/05/99, 05/25/99 LAC
Date Verified/ By: 06/21/99 EAD

EID088647

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889.2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Soup 020199, Amelia 062498 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 06/11/99 RWW |
| Date of Analysis/Analyst: | 06/22/99, 06/23/99 DRJ/MEE |
| Date of Data Reduction/Analyst: | 06/23/99, 06/24/99 DRJ/MEE |

## Sample Data

### Week 28 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Monkey Sera Blk-1 | <MDL | | |
| | Monkey Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | MKS06119-MS-1 | 106% | | |
| | MKS06119-MSD-1 | 104% | 105% | 1% |
| Group 1 | I05718M | 0.160 | | 21.6 |
| Control, 0.0 mg/kg/day | I05720M | 0.218 | 0.189 | 0.0408 |
| Group 3 | I05712M | 32.4 | | 27.26 |
| 10 mg/kg/day | I05716M | 21.9 | 27.1 | 7.40 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5.0 ng/mL

POAA = Perfluorooctanoate

| | |
|---|---|
| Date Entered/By: | 06/23/99, 06/25/99 LAC |
| Date Verified/ By: | 08/04/99 GML |

EID088648

GW1000067

| | |
|---|---|
| Study | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4 1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Soup 020199, Amelia 062498 |
| Instrument Software/Version: | MassLynx 3 2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 06/11/99 RWW |
| Date of Analysis/Analyst: | 06/22/99, 06/23/99 DRH/MEE |
| Date of Data Reduction/Analyst: | 06/23/99, 06/24/99 DRH/MEE |

## Sample Data

### Week 30 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Monkey Sera Blk-1 | <MDL | | |
| | Monkey Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | MKS06119-MS-1 | 106% | | |
| | MKS06119-MSD-1 | 104% | 105% | 1% |
| Group 1 | 105718M | 0 133 | | 16 6 |
| Control, 0.0 mg/kg/day | 105720M | 0 168 | 0 150 | 0 0248 |
| Group 3 | 105712M | 15 6 | | 45.2 |
| 10 mg/kg/day | 105716M | 8 05 | 11.8 | 5.35 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5.0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 06/23/99, 06/25/99 LAC
Date Verified/ By: 08/04/99 GML

EID088649
GW1000068

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Soup 020199, Amelia 062498 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 06/11/99 RWW |
| Date of Analysis/Analyst: | 06/22/99, 06/23/99 DRB/MEE |
| Date of Data Reduction/Analyst: | 06/23/99, 06/24/99 DRB/MEE |

## Sample Data

### Week 32 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Monkey Sera Blk-1 | <MDL | | |
| | Monkey Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | MKS06119-MS-1 | 106% | | |
| | MKS06119-MSD-1 | 105% | 106% | 1% |
| Group 1 | 105718M | 0.0984 | | 19.8 |
| Control, 0.0 mg/kg/day | 105720M | 0.130 | 0.114 | 0.0226 |
| Group 3 | 105712M | 10.8 | | 51.3 |
| 10 mg/kg/day | 105715M | 5.07 | 7.95 | 4.08 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5.0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 06/23/99, 06/25/99 LAC
Date Verified/ By: 08/04/99 GML

EID088650

GW1000069

| | |
|---|---|
| Study: | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889.2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Soup 020199, Amelia 062498 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 06/11/99 RWW |
| Date of Analysis/Analyst: | 06/22/99, 06/23/99 DRB/MEE |
| Date of Data Reduction/Analyst: | 06/23/99, 06/24/99 DRB/MEE |

## Sample Data

### Week 34 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Monkey Sera Blk-1 | <MDL | | |
| | Monkey Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | MKS06119-MS-1 | 106% | | |
| | MKS06119-MSD-1 | 105% | 106% | 1% |
| Group 1 | 105718M | 0.0710 | | 29.7 |
| Control, 0.0 mg/kg/day | 105720M | 0.109 | 0.0899 | 0.0267 |
| Group 3 | 105712M | 5.69 | | 52.9 |
| 10 mg/kg/day | 105716M | 2.60 | 4.14 | 2.19 |

Limit of Quantitation (LOQ): 10 ng/mL
Method Detection Limit (MDL): 5.0 ng/mL

POAA = Perfluorooctanoate

Date Entered/By: 06/23/99, 06/25/99 LAC
Date Verified/ By: 08/04/99 GML

6329-231

EID088651

GWJ0000070

| | |
|---|---|
| Study | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Soup 020199 |
| Instrument Software/Version | MassLynx 3.2 |
| Filename | See Attachments |
| R-Squared Value | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 07/13/99 MCH |
| Date of Analysis/Analyst | 07/14/99, 07/21/99 GML/DRB |
| Date of Data Reduction/Analyst: | 07/15/99, 07/22/99 GML/DRB |

## Sample Data

### Week 38 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Monkey Sera Blk-1 | <MDL | | |
| | Monkey Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | MKS07139-MS-1 | 92% | | |
| | MKS07139-MSD-1 | 84% | 88% | 9% |
| Group 1 | 105718M | 0 0757 | | 34 1 |
| Control, 0 0 mg/kg/day | 105720M | 0 124 | 0 0998 | 0 0341 |
| Group 3 | 105712M | 2 99 | | 77.2 |
| 10 mg/kg/day | 105716M | 0.88 | 1 93 | 1.49 |

Limit of Quantitation (LOQ) 25 ng/mL
Method Detection Limit (MDL): 12 ng/mL

POAA = Perfluorooctanoate

| | |
|---|---|
| Date Entered/By: | 07/20/99, 07/23/99 LAC |
| Date Verified/ By: | 08/04/99 GML |

EID088652

| | |
|---|---|
| Study | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889 2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Soup 020199 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 07/13/99 MCH |
| Date of Analysis/Analyst | 07/14/99, 07/21/99 GML/DRB |
| Date of Data Reduction/Analyst: | 07/15/99, 07/22/99 GML/DRB |

## Sample Data

### Week 38 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/ml, | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Monkey Sera Blk-1 | <MDL | | |
| | Monkey Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | MKS07139-MS-1 | 92% | | |
| | MKS07139-MSD-1 | 84% | 88% | 9% |
| Group 1 | I05718M | 0 0757 | | 34 1 |
| Control, 0 0 mg/kg/day | I05720M | 0.124 | 0 0998 | 0 0341 |
| Group 3 | I05712M | 2.99 | | 77.2 |
| 10 mg/kg/day | I05716M | 0 88 | 1 93 | 1 49 |

Limit of Quantitation (LOQ): 25 ng/mL
Method Detection Limit (MDL): 12 ng/mL

POAA = Perfluorooctanoate

| | |
|---|---|
| Date Entered/By: | 07/20/99, 07/23/99 LAC |
| Date Verified/ By: | 08/04/99 GML |

EID088653

GWJ0000072

| | |
|---|---|
| Study | 6 Month Capsule Toxicity Study with APFO in Cynomolgus Monkeys |
| Product Number(Test Substance): | T-6889.2 (POAA) |
| Matrix: | Monkey Sera |
| Method/Revision: | ETS-8-4.1 & ETS-8-5.1 |
| Analytical Equipment System Number: | Soup 020199 |
| Instrument Software/Version: | MassLynx 3.2 |
| Filename: | See Attachments |
| R-Squared Value: | See Attachments |
| Slope: | See Attachments |
| Y-Intercept: | See Attachments |
| Date of Extraction/Analyst: | 07/13/99 MCH |
| Date of Analysis/Analyst: | 07/14/99, 07/21/99 GML/DRB |
| Date of Data Reduction/Analyst: | 07/15/99, 07/22/99 GML/DRB |

## Sample Data

### Week 40 MONKEY SERA

| Group Dose | Sample # | POAA Reported ug/mL | Average POAA ug/mL | RSD Std. Dev. |
|---|---|---|---|---|
| Method Blk | H2O Blk-1 | <MDL | | |
| | H2O Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Rabbit Sera Blk-1 | <MDL | | |
| | Rabbit Sera Blk-2 | <MDL | <MDL | <MDL |
| Matrix Blk | Monkey Sera Blk-1 | <MDL | | |
| | Monkey Sera Blk-2 | <MDL | <MDL | <MDL |
| QC - 250 ppb | MKS07139-MS-1 | 92% | | |
| | MKS07139-MSD-1 | 84% | 88% | 9% |
| Group 1 | I05718M | 0.0751 | | 5.1 |
| Control, 0.0 mg/kg/day | I05720M | 0.081 | 0.0779 | 0.0040 |
| Group 3 | I05712M | 1.84 | | 69.7 |
| 10 mg/kg/day | I05716M | 0.63 | 1.23 | 0.86 |

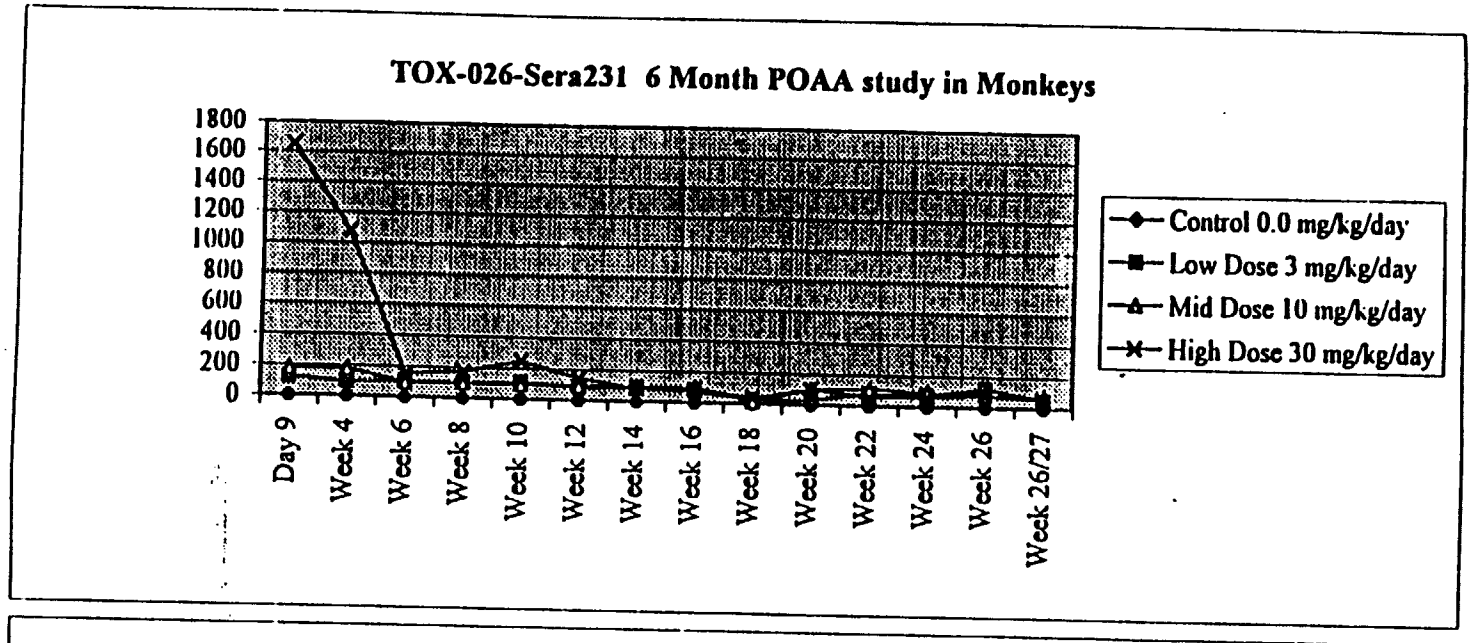
Limit of Quantitation (LOQ): 25 ng/mL
Method Detection Limit (MDL): 12 ng/mL

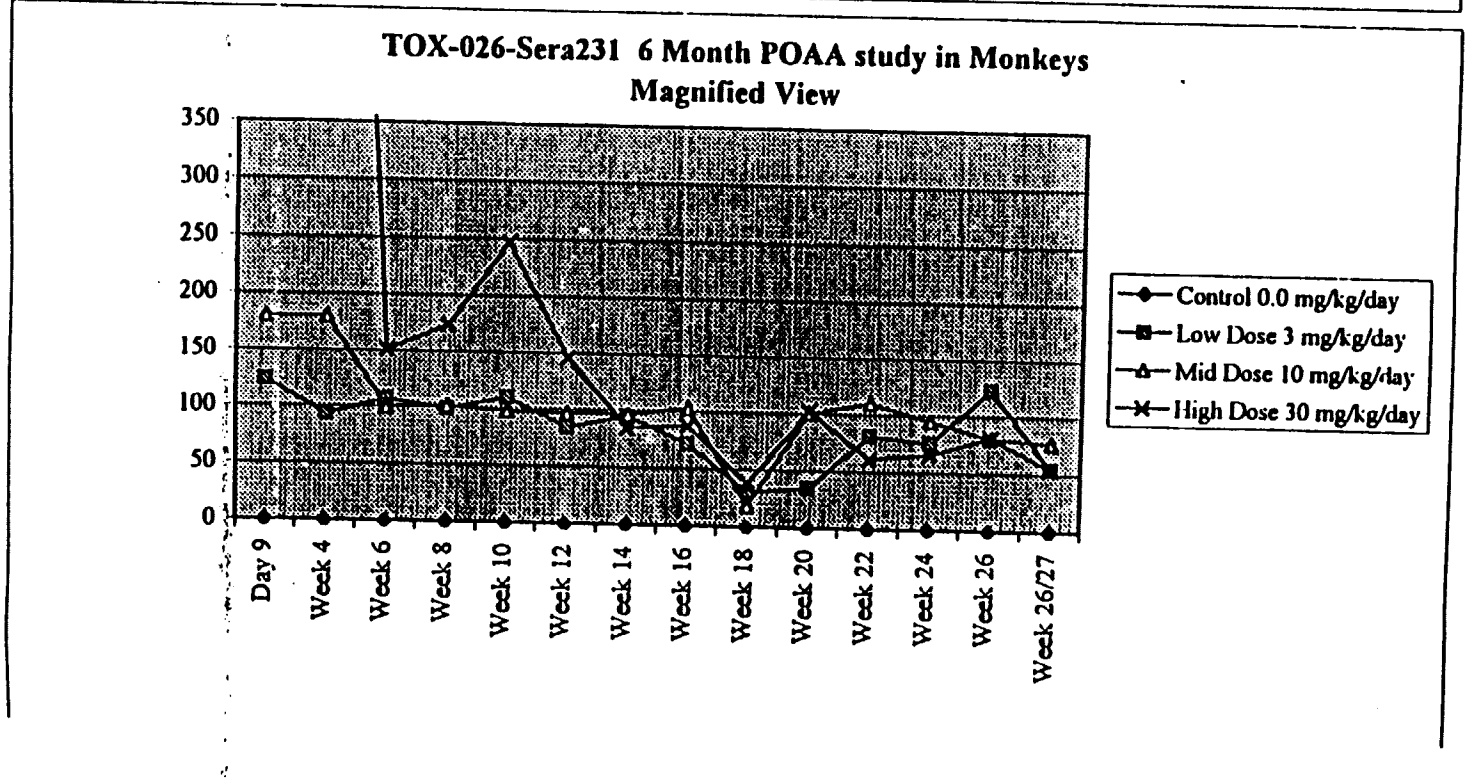
POAA = Perfluorooctanoate

Date Entered/By: 07/20/99, 07/23/99 LAC
Date Verified/ By: 08/04/99 GML

EID088654

GW/0000073


TOX-026-Sera231 6 Month POAA study in Monkeys
1800
1600
1400
1200
1000
800
600
400
200
0
Day 9
Week 4
Week 6
Week 8
Week 10
Week 12
Week 14
Week 16
Week 18
Week 20
Week 22
Week 24
Week 26
Week 26/27
Control 0.0 mg/kg/day
Low Dose 3 mg/kg/day
Mid Dose 10 mg/kg/day
High Dose 30 mg/kg/day


TOX-026-Sera231 6 Month POAA study in Monkeys
Magnified View
350
300
250
200
150
100
50
0
Day 9
Week 4
Week 6
Week 8
Week 10
Week 12
Week 14
Week 16
Week 18
Week 20
Week 22
Week 24
Week 26
Week 26/27
Control 0.0 mg/kg/day
Low Dose 3 mg/kg/day
Mid Dose 10 mg/kg/day
High Dose 30 mg/kg/day

EID088655

Andrew S Hartten 09/03/99 02:48 PM

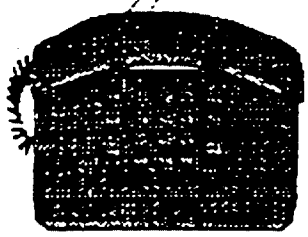
To: Robert L Ritchey/CL/DuPont@DuPont
cc: Robert F Pinchot/DEV/AE/DuPont@DuPont, Isidoros J Zanikos/AE/DuPont@DuPont, Andrea Malinowski/DPL/DUP@DUP, Bernard J. Reilly/DPL/DUP@DUP, Michael J Lukas/AE/DuPont@DuPont, Alison A Crane/AE/DuPont@DuPont, Michael A Turco/AE/DuPont@DuPont, H David Ramsey/AE/DuPont@DuPont, John M Migliore/AE/DuPont@DuPont, Roger J Zipfel/AE/DuPont@DuPont, Dawn D Jackson/CL/DuPont@DuPont, Anthony J Playtis/CL/DuPont@DuPont, George Woytowich/CL/DuPont@DuPont, "LYNWOOD K. IRELAND" <IRELAND@wwps-a1.email.dupont.com>, John E Auger/AE/DuPont@DuPont, Jon F Moore/CL/DuPont@DuPont, David C Harrison/CL/DuPont@DuPont, Timothy S Bingman/AE/DuPont@DuPont
Subject: Re: Plant Manager communication to GE

Bob,

Great to hear that additional communication has occurred with GE. Related to this subject, we need to be prepared to address EPA questions concerning this issue during their Sept. 23 site visit, especially if Martin Kotsch (EPA PM) visits the GE site first and arrives "enlightened". As you know, he will also be visiting GE during his trip to Parkersburg as they are also currently immersed in the RFI process. Hopefully, we can defer most questions to the formal "Wash Works RFI Results Summary" presentation he wants me (and Tim) to give to his project team (i.e., toxicologist, hydro) in mid-October.

Andrew

Pat 1:00 pm
mtg today



***FACSIMILE COVER SHEET***
***DU PONT FLUOROPRODUCTS***

**TO:** Irma Garza

**FAX:** 563-4391

**DATE:** **PHONE:**

---

**FROM:** Pat Dimick / K. Kennedy

**LOCATION:** CRP-711/ **PHONE:**

**FAX:** (302) 999-5123

**No. of Pages including cover sheet:**

**Message:**

**Deliver Immediately** **Confidential**

EID087238

22 monkeys

# *C-8 Monkey Study*

- Goal: A) to determine toxicologic effects of C-8 in the primate following an extended exposure period
  B) to determine potency for producing change (NOEL-LOEL-EL)

- Surprises: - Potency at lower dose (3 mg/kg)
  - Failure of all animals in group to respond similarly
  - Quick plateau of C-8 in blood
    A) Quick clearance from blood
    B) Lack of proportional response (exposure of x, 3x, 6x did not lead to blood concentrations of x, 3x, 6x)

- Subtle vs major toxicity end points

- Hormones unchanged

- Pathology unremarkable, especially in severely affected monkeys

# *C-8 Monkey Study*

Sponsor: APME Ad-Hoc APFO
Toxicology Working Group

Testing Facility: Covance Laboratories
Madison, WI

Study ID: 6329-231

Study Director: Peter Thomford
Study Monitor: Paul Lieder - 3M
Study Representatives: David Farrar - ICI
Reinhart Jung - Clariant
Gerry Kennedy - DuPont
[Giovanni Costa - Mitani]
[George Lin - Daikin]

In Life 9/23/98 - 7/2/99

OTG000258

# *C-8 Monkey Study*

## Experimental Details

- Oral dosing - gelatin capsules
- Diet - primate diet, 1 or 2 x/day
  - supplemented fruit/vegetables
- Young adult/adult - 3 ↔ 5 kg

OTG000259

# C-8 Monkey Study

monkeys - all male → e.g., male rats sensitive than fem

**Design**

Group 1 Control - 6 cynomolgus males
4 6 months
2 6 months & 3 months recovery

Group 2 3 mg/kg - 4 males - 6 months
(1 monkey - died day 137)

Group 3 10 mg/kg - 6 males
4 6 months
2 6 months & 2 months recovery

22 monkeys

Group 4 20/30 mg/kg - 6 males
30 - day 1-11
0 - day 12-21
20 - day 22 → 6 months
3 monkeys - dosing discontinued Days 43 ↔ 81
(1 monkey - died day 29)
All sacrificed at 6 months

stoppages due to animals not tolerating high doses

EID087242

# *C-8 Monkey Study*

## Parameters

I. *In Vivo*

Observed 2x/day
Body weight - weekly
Food consumption - estimate daily
Ocular exams - pre-test/weeks 27 & 40

II. Clinical Pathology

Timing - Pre-test, days 30, 60, 90, 180
Recovery days 30, 60, 90
Hematology - RBC, Hb, PcV, platelets
WBC & diet, reticulocytes, cell-indices
Coagulation - APTT, pro time, fibrinogen
Clin Chem - glucose, UN, creatinine, prot, bilirubin, cholesterol, triglycerides, ALT, AP, AST, GGT, SDH, ions (Ca, etc), amylase, lipase
Urine - standard & urobilinogen, bilirubin

# *C-8 Monkey Study*

III. Blood Hormones
- Timing - 3x pre-dosing, day 30, 60, 90, 180
  Recovery day 30, 60, 90
- Estradiol
  Estrone
  Estriol
  Thyroid stimulate hormone
  Total & free iodothyronine (T3)
  Total & free thyroxin (T4)
  Testosterone
  Cholecystokinin

IV. Exposure Indices
- Serum APFO - 7 days & every 2 weeks thereafter
- Urine APFO - as serum
- Feces APFO - as serum
- Liver APFO - at sacrifice

C1G00262

# *C-8 Monkey Study*

V. Pathology

Complete nescropsy

Organ weights

adrenal, brain, epididymis, kidney,
liver, pancreas, testes, thyroid (parathyroid)

Histopathology (36 tissues)

Additional parameters

- Palmitoyl CoA oxidase
- Cell proliferation
- Bile acid determination

(receptor level determinations)

(bone marrow smear)

# *C-8 Monkey Study - Results*

I. Clinical Observations

Control - none

3 mg/kg - 1 monkey - week 18 - ataxic, hypoactive (not seen in any other animals)
no food consumption, limited use
of hind limbs (animal was not going to)
week 20 - sacrificed, lost 9.5% bwt

3 monkeys - none

10 mg/kg - 6 monkeys - none

30 mg/kg - week 1 - low food consumption
lost 3-7.5% bwt

↓

20 mg/kg - week 3 - 3 monkeys - same as above without
marked wt loss, treatment
discontinued week 7, 10, 12
1 monkey died week 4
2 monkeys - no clinical signs
after week 2

OTG000264

# *C-8 Monkey Study - Results*

II. Ophthalmology - no findings

III. Body Weight
0/3/10 mg/kg - no differences
→ 20 mg/kg - lower wk 7, 9, 24 (14.3% wt gain of controls)
[30 mg/kg - lower wk 1]

→ exclude 1 monkey that died
→ 2 animals
wt loss started at this dose

IV. Food Consumption
0/3/10 mg/kg - no differences
20/30 mg/kg - lower (some no food consumption)

V. Blood Hormone
No significant effects (0 30 mg/kg)
Unexplained total thyroxin lower 20 mg/kg
free triiodothyronine lower 20 mg/kg

VI. Clinical Pathology
0/3/10 mg/kg - no differences
30/20 mg/kg - mild ↑ triglycerides
mild ↓ neutrophil, protein, albumin
- 2 distressed - ↑ marked ALT, AST, SDH, (creatine kinase)
↑ mild bile acids
- recovery in off-treatment monkeys

OTG000265

EID087247

# *C-8 Monkey Study - Results*

VII. Palmitoyl CoA oxidase - expect <4 wks
Cell proliferation - expect <2 wks
Bile acid - no differences

VIII. Pathology
Gross pathology - unremarkable
Organ weight - liver weights elevated
0/3/10/20-30
60/82/83/90 grams Mean (all test*)
1.5/1.8/1.9/2.4 Organ/bwt (all test*)

Some liver sites but not overwhelm stress

Histopathology - unremarkable
Cause of death - unclear in both moribund animals

OTG000266



EID087248

# C-8 Monkey Study
# Urinary C-8 Levels (ppm)

6 months

| Group (mg/kg) | Week 2 | 6 | 10 | 14 | 18 | 26 | 28 | 36 | 40 |
|---|---|---|---|---|---|---|---|---|---|
| 0 | 0/0 | 0/0.2 | 0/0 | 0/0 | 0/0 | 0/0 | 0/0.2 | 0/0 | 0/0 |
| 3 | 76/57 | 69/138 | 42/35 | 66/61 | 67/38 | 54/39 | | -- | -- |
| 10 | 184/430 | 133/164 | 182/284 | 127/129 | 191/159 | 114/269 | 3/3 | 0.05/0.01 | 0.01/0.01 |
| 20/30 | 949/548 | 283/369 | 342 | 156 | 177 | 61 | -- | -- | -- |

110's for 20/30 show anomalous hi-to-lo behavior?

OTG000267

EID087249

000387

# *C-8 Monkey Study*
# *Liver C-8 Levels (ppm)*

| Group (mg/kg) | [Liver] |
|---|---|
| 0 | 0.04 (0-0.04) |
| 3 | 5.9 (4.1-6.8) |
| 10 | 5.9 (3.6-8.7) |
| 20/30 | 17.2 (8-28) |
| Recovery | 0.8 (0.03-1.2) |

very similar

OTG000268

EID087250

# *C-8 Monkey Study - Conclusions*

- Monkeys do not tolerate 20 mg/kg or higher
  - Non-specific response involves liver
  - Recovery complete in off-dosing monkeys
  - Effects include body weight, liver weight
  - No specific histopathological changes
    hormonal changes
- Effects at 10 mg/kg only liver weights
- Effects at 3 mg/kg - 1 death (relationship to treatment?)
  liver weights
- C-8 cleared quickly from urine (proportional to exposure
- C-8 clears quickly from blood (not proportional)
  - Reaches plateau quickly
  - Leaves system quickly
- C-8 in liver proportional to exposure,
  recovery quick and complete
- C-8 in feces - will get "matrix information"

← don't
this d
in d

OTG000269

EID087251

# C-8 Monkey Study - Conclusion

- No observed effect level not attained (<3 mg/kg)

previously reported in earlier ... that was <10 ... This ...

- Potential serious effect in 1/4 monkeys at low dose (liver effect unexplained currently; best explanation could be enzyme induction)

unsatisfying situation

- At AEL/TLV of 0.01 mg/m$^3$, daily exposure to man is 0.001 mg/kg

existing industrial hygiene controls in DuPont appear to have been right more in protecting our people.

OTG000270

EID087252

000603

# *C-8 Monkey Study - Issues*

- Liver effect (will answer) serious liver damage is not occurring, however
- Death of low dose monkey
- Lack of slow elimination as seen in man
  (does human data reflect multi-phase clearance)
- Evaluation of monkey-by-monkey data (in progress)

OTG000271

# *Thanks for Hanging in there.....*

OTG000272

EID087254

000604

Increased Liver Weight - In context to Clinical Exposure - It is an Adaptive Response and Sometimes A toxic Response

→ Ben Kuby; Talk to Larry K.

Need to call Bernie → Dry Rm. Team

↳ Ext. Comm. 12/6/99

New Lubeck Wells - 1 PPB ??

G.E. Well - 0.5 PPB

1:30 PM Mig 11/18

JMM008028

EID073309

009609

Roger J Zipfel
11/17/99 11:27 AM

To: Robert F Pinchot/DEV/AE/DuPont@DuPont
cc: Oscar T Garza/AE/DuPont@DuPont
Subject: Re: C-8 Monkey Study and Allowable Exposure Limit

Rob, I don't fully support the first burger dot as it is stated. I don't think that we can rule out death due to C-8 exposure. Thus I believe it should read similar to the second burger dot.

Roger.
Robert F Pinchot 11/15/99 03:06 PM

**Robert F Pinchot** 11/15/99 03:06 PM

To: Robbin Banerjee/AE/DuPont@DuPont, Oscar T Garza/AE/DuPont@DuPont, Anthony J Playtis/CL/DuPont@DuPont, Stephen J Gissy/AE/DuPont@DuPont, Roger J Zipfel/AE/DuPont@DuPont, Matthew C Koenings/EUR/DuPont@DuPont, Arie J Vermeulen/EUR/DuPont@DuPont, Seiichi Tanaka/PO/DuPont@DuPont, Norifumi Ohtsuka/PO/DuPont@DuPont, Barbara J Gayda/AE/DuPont@DuPont, Barbara J Gayda/AE/DuPont@DuPont, Thomas J Cavanaugh/AE/DuPont@DuPont, Romain Van_Aken/EUR/DuPont@DuPont, Barbara J Dawson/AE/DuPont@DuPont, Susan S Mileti/DuPont@DuPont, Richard J Angiullo/AE/DuPont@DuPont, Maurice Astorga/AE/DuPont@DuPont, Marianne Marsi/DuPont@DuPont, David M Rurak/AE/DuPont@DuPont, Michael E McCord/AE/DuPont@DuPont, Robert J Cavanaugh/DuPont@DuPont, Clarence P Mihal/AE/DuPont@DuPont, Earl W. MacFarlane/DPL/DUP@DUP, Andrea V Malinowski/AE/DuPont@DuPont @ DUP, Sharron Laas/AE/DuPont@DuPont
cc: Diane R Shomper/AE/DuPont@DuPont, Gary W Jepson/AE/DuPont@DuPont, Gerald L Kennedy/AE/DuPont@DuPont
Subject: C-8 Monkey Study and Allowable Exposure Limit

Most of the results of the 6 month monkey study are available. Due to some of the findings of the study, the study is reportable to the US EPA under TSCA Section 8(e) by the 3M company. This notification will be made by Friday, November 19th and the letter will become a public document shortly thereafter. Since employees potentially exposed to C-8 have a right to know about the findings of the study, Diane Shomper and I are developing an employee communication framework for you to use for your communications. This communication package should be available for you to use by Monday November 22.

To give you a preview of what we will be in the communication, here are the key points:

- One monkey in the low dose group died for reasons that are unclear.
- One monkey in the highest dose group died for reasons that are unclear but may be related to C-8 exposure.
- All monkeys showed some liver effects (liver weight increases in all dosage groups, liver enzyme increases in the high dose group monkeys that were in distress. The recovery animals did not show these effects suggesting that the liver effects were reversible.
- No other clinical or pathological changes were observed other than the deaths and the liver effects.
- The study results are not complete. We won't have a full analysis of the data until January.
- The AEL (0.01mg/m3 8 and 12 hr TWA with a skin notation) and Community Exposure Guideline (3micrograms/day) will, for now, stay the same. The Committee will revisit this

after the full results are in and analyzed.

Please wait for the formal communication package to communicate this broadly. The purpose of this note is to give you a heads up that this communication is coming.

Regards,
Rob

# C-8 Monkey Study Communication Plan

Communication to Employees

DuPont Legal opinion is that communication is required to US employees. No specific time frame is given in the OSHA Hazard Communication Standard.

To be conducted on Thursday, December 9 to all DuPont and employees potentially exposed to C-8 (Sites include WW, Parlin, Spruance, CW, DW, Mechelen, Shenzhen, Madurai, and Shimizu). Summary of study made public on November 22 through TSCA 8(e) filing by 3M.

Messages to be conveyed at all sites

1. Current AEL is protective of human health
2. Liver effects seen at all doses, effects were reversible after ceasing exposure
3. Two deaths occurred in the test groups. One at low dose and one at high dose. The cause of the deaths is unknown.
4. More data will be communicated when analysis is complete (early 2000)

Some sites adding site specific information on exposure levels at their site (all of which are less than the AEL).

WW communicating that C-8 is in drinking water at the site, at levels less than the Community Exposure Guideline.

WW is communicating the results to GE Plastics management.

Communication to Customers

No MSDS changes have been made at this time. No need to communicate to all dispersion customers. May have to change the MSDS once the final results are analyzed and will need to communicate to customers

Chem Fab and Gore have been told about the monkey study. We will share the results with these two customers in the near future. (Gore meeting set up in January).

# Tennant Farm Herd Health Investigation

# Cattle Team Report

EPA/DuPont Cattle Team

Dr. Perry L. Habecker
Dr. Lisa A. Davis-Heller
Dr. Peter G. Moisan
Dr. Robert J. Munson
Dr. Robert H. Poppenga
Dr. Greg P. Sykes

December 23, 1999

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DUP 244

# 1.0 SUMMARY

**Introduction**: The objective of this investigation was to evaluate the health status of Mr. Earl Tennant's cattle herd and determine possible causes of any problems. The investigation included an examination of relevant historical data as well as the collection and evaluation of new data. The investigation terminated in the production of this report, which outlines study findings and makes recommendations to improve herd health.

**Methods**: The Tennant farm herd health investigation was conducted by a team of six veterinarians with collective experience in bovine diseases, herd health management, toxicology, pathology, and wildlife diseases. In addition to numerous meetings and discussions, the cattle team visited the Tennant Farm on April 7-8, 1999 in order to collect relevant data. The cattle team reviewed historical data (e.g., videotapes, diagnostic laboratory reports) and contemporary data (e.g., clinical examinations, blood tests). Diagnoses and recommendations were based upon the data collected.

**Results**: The multifaceted disease investigation of the adult cattle in the Tennant beef herd revealed health problems that were related to endophyte toxicity, infectious keratoconjunctivitis (pinkeye), malnutrition, and copper deficiency. Clinical and historical data, with prolactin values of some animals, were consistent with endophyte mycotoxicosis. An examination of videotapes made during the summer months and clinical examinations of previously affected adult animals during the herd visit indicated prolonged severe enzootics of facefly (*Musca autumnalis*) infestation and concomitant pinkeye. Hay analysis, cattle body condition scoring, and an evaluation of the mineral and grain rations fed were consistent with protein-energy malnutrition and macromineral/trace nutrient deficiencies. Earlier laboratory data, clinical signs, and serum testing at the time of the herd visit were indicative of severe copper deficiency in the cattle.

**Conclusion**: There was conclusive evidence that the Tennant cattle herd was, and continues to be, suffering from four major disease entities, some of which were potentially interrelated: endophyte toxicity (fescue mycotoxicosis), pinkeye, malnutrition, and copper deficiency. As substantiated by the clinical and laboratory findings, and historical data, these four conditions readily account for the chronic herd health problems on the Tennant farm.

The herd health investigation revealed deficiencies in herd management, including poor nutrition, inadequate veterinary care, and lack of fly control. The lack of vaccination and internal parasite control programs did not appear to have a substantial impact on this relatively isolated herd.

Despite an exhaustive review of historical and contemporary herd data, there was no evidence of toxicity associated with chemical contamination of the environment.

## 1.1 Signatures

Perry L. Habecker, VMD, Dipl. ACVP — Date: 12/23/99

Lisa A. Davis-Heller, DVM — Date: 12/30/99

Peter G. Moisan, DVM, Dipl. ACVP, ABVP — Date: 12/26/99

Robert J. Munson, VMD — Date: 1/3/00

Robert H. Poppenga, DVM. PhD, Dipl. ABVT — Date:

Greg P. Sykes, VMD, Dipl. ACVP, ABT — Date:

# 2.0 INTRODUCTION

The objective of this investigation was to evaluate the health status of Mr. Earl Tennant's cattle herd and determine possible causes of any problems. The investigation included an examination of relevant historical data as well as the collection and evaluation of new data. The investigation terminated in the production of this report, which outlines study findings and makes recommendations to improve herd health

# 3.0 METHODS

## 3.1. Cattle Team

### 3.1.1. Members

The Tennant Farm Herd Health Investigation was assigned to a team of six veterinarians ("cattle team"). The team was constituted to include expertise in bovine diseases, herd health management, toxicology, pathology, and wildlife diseases. Representatives of the DuPont Company (DuPont) selected three members (Drs. Sykes, Davis-Heller, Moisan); representatives of the United States Environmental Protection Agency (EPA) selected three members (Drs. Habecker, Poppenga, Munson). Abbreviated *Curriculum Vitae* of the cattle team members are included in this report (Appendix A).

Cattle Team

| Members | Associations |
|---|---|
| Perry Habecker, VMD, Dipl. ACVP | Chief, Large Animal Pathology, Laboratory of Pathology and Toxicology, Univ. of Penn. School of Veterinary Medicine, New Bolton Center, Kennett Square, PA. |
| Lisa Davis-Heller, DVM | Private Practitioner, St. Mary's Veterinary Clinic, St. Mary's, WV |
| Peter Moisan, DVM, Dipl. ACVP, Dipl. ABVP | Veterinary Pathologist, Rollins Animal Disease Diagnostic Laboratory (state laboratory), Raleigh, NC. |
| Robert Munson, VMD | Field Investigator, Center for Animal Health and Productivity, Univ. of Penn. School of Veterinary Medicine, Kennett Square, PA. |
| Robert Poppenga, DVM, PhD, Dipl. ABVT | Chief, Toxicology, Laboratory of Pathology and Toxicology, Univ. of Penn. School of Veterinary Medicine, New Bolton Center, Kennett Square, PA. |
| Greg Sykes, VMD, Dipl. ACVP, Dipl. ABT | Pathologist, Safety Assessment, DuPont Pharmaceuticals Company, Stine Research Center, Newark, DE. |

DVM, VMD = Doctor of Veterinary Medicine
Dipl. ACVP = Diplomate, American College of Veterinary Pathologists
Dipl. ABVP = Diplomate, American Board of Veterinary Practice (Food Animal and Beef Cattle Specialist)
Dipl. ACVT = Diplomate, American College of Veterinary Toxicologists
Dipl. ABT = Diplomate, American Board of Toxicology

At the first meeting of the cattle team (March 9, 1999), Perry Habecker was elected Scientific Leader (i.e., chairman) and Greg Sykes was elected Coordinator. The

chairman and coordinator were instructed to communicate with the "steering committee" leaders, Drs. Sarah Caspar (EPA, Region III) and Ralph Stahl (DuPont Corporate Remediation Group), as needed. This "steering committee" included Drs. Caspar, Stahl, Mike Horne (USFWS), Mark Sprenger (EPA-OERR), and Rudy Valentine (DuPont - Specialities Chemicals toxicologist).

### 3.1.2. Cattle Team Meetings

The cattle team met formally on four occasions, including the Tennant Farm site visit.

**Cattle Team Meetings**

| Date | Location |
|---|---|
| March 9, 1999 | New Bolton Center, Kennett Square, PA |
| March 23, 1999 | New Bolton Center, Kennett Square, PA |
| April 7-8, 1999 | Tennant Farm, Wood County, WV* |
| July 28-29, 1999 | New Bolton Center, Kennett Square, PA |

* including pre-meeting planning session at Holiday Inn, Parkersburg, WV

In addition, several in-person discussions took place at New Bolton Center between and among Drs. Habecker, Munson, Poppenga, and Sykes between March 1999 and the issuance of this report. All cattle team members utilized the telephone, internet email, and US postal systems for pair and group discussions and information sharing.

## 3.2. Animal Data

### 3.2.1. Videotapes

Two videotapes were supplied to the cattle team by the steering committee. Each of the tapes was a collection of videotapes made, and narrated, by Mr. Earl Tennant on his farm and the adjacent landfill area. Although the tapes were edited and include material from different seasons, it appeared that these tapes were all produced between 1995 and 1997.

**Tennant Farm Videotapes**

| Tape | Title | Animal Case Numbers |
|---|---|---|
| #1 | "Tennant Farm: New England, Wood County, WVa – January and February, 1997" | 1 - 18 |
| #2 | "Dry Run: Harris PC, Wood Co.: Off North Fork of Lee Creek, New England, WVa" | 19 - 60 |

Both of these tapes were viewed, in their entirety, by the cattle team members individually. They were also reviewed at a cattle team meeting on July 28th (Dr. Davis-Heller not present). A total of 60 animal cases, ranging from a dead crayfish to groups of sick cows, were evaluated. Items on the tapes that were not animal related (e.g., water treatment) were noted but not evaluated. Table 1 is a compilation of the relevant animal health data derived from these tapes. This table does not include many assertions and



000619 DUP 248

interpretations, made by the narrator in the tapes, which the team members considered to be incomplete or erroneous.

### 3.2.2. Diagnostic Pathology Reports

Two pathology reports (Appendix B) were supplied to the cattle team by the steering committee. Both were issued by state animal diagnostic laboratories in response to the submission of dead animals or tissues from the Tennant Farm. These reports were reviewed by the cattle team.

A third pathology report (Appendix B) was produced subsequent to the elective sacrifice of one animal (# 37) on June 10, 1999. The cattle committee decided, at a meeting on May 28, 1999, that it would be informative to have a toxicology screen on tissues from one or two older cows. Mr. Tennant was asked to select one or two of the cattle that he considered to be in the worst condition. He chose to sacrifice cow # 37, a 7-year old red cow that had freshened a few months earlier. This animal was euthanized by gunshot by Mr. Tennant on the farm on June 10, 1999. Mr. Tennant performed the dissection in the presence of Dr. Davis-Heller. Tissues were collected (Dr. Davis-Heller) for histopathology and analytical toxicology.

**Individual Animal Pathology Reports**

| Pathology Report Date | Name of Diagnostic Laboratory | Case Material (case number) |
|---|---|---|
| March 10, 1997 | Animal Disease Diagnostic Laboratory, Ohio Department of Agriculture, Reynoldsburg, OH | dead 6-month-old bull calf (#4977-97) |
| March 12, 1997 | Animal Health Diagnostic Laboratory, College of Veterinary Medicine, Michigan State University, Lansing MI | tissues 4-year-old Holstein cow (#1792571)<br>tissues 9-year-old Holstein cow (#1792571) |
| July 5, 1999 | Laboratory of Large Animal Pathology and Toxicology, New Bolton Center, University of Pennsylvania, Kennett Square, PA | tissues 7-year-old cow (#UP9902702; UP9901437) |

### 3.2.3. April 1999 Site Visit Data

The cattle team visited the Tennant Farm and adjacent Dry Run landfill site on April 7 and 8, 1999 in order to collect data and biological samples relevant to the herd health investigation. Before proceeding to the site on April 7th, a brief safety meeting was held at the Parkersburg Holiday Inn during which the Dry Run Safety Plan (Appendix C) was reviewed. The 6 cattle team members, 2 of the steering committee members (Caspar, Horner) and 2 USFWS employees were present.

April 7, 1999

The cattle team had planned to inspect the farm and adjacent landfill, view the herd grazing, and interview Mr. Tennant on the afternoon of Wednesday April 7th. However,

due to a misunderstanding, Mr. Tennant had corralled his entire herd on Wednesday morning. When the cattle team and EPA/USFWS personnel arrived at approximately 3:15 PM, the herd had been contained in a small enclosure, without food or water, for several hours. The team therefore elected to conduct the individual animal examinations and specimen collections immediately. A new head-hold restraint device had been installed next to the enclosure to facilitate animal handling. Mr. Earl Tennant and his brother were very helpful in handling the cattle and moving them into the restraint device.

The 41 adult cattle were individually run into the chute, secured with the head hold and a halter, and examined. Each adult (38 cows, 2 bulls, and 1 steer) was given a general physical examination including observation of gross abnormalities, grading of body condition, and estimation of age based on the incisors. Blood was collected by jugular venipuncture (2 red top, 1 green top, and 1 lavender top tubes) from each cow and fecal samples were rectally collected from each adult. Cows were rectally palpated to determine their pregnancy status. All examined adult animals received two ear tags, one with an identification number and one with an impregnated insecticide. One Hereford cow (i.e., the 39$^{th}$ cow, 42$^{nd}$ adult) escaped the chute prior to examination, aging, uterine palpation, blood collection, and ear tagging.

Each adult bovine was individually photographed and identified (Appendix D, photographs 1-42).

Only 1 cow (animal #22) received treatment – a mass, most consistent with a dermoid cyst, was incised and drained (Appendix D, photographs 49-51).

Additional photographs of the farm (Appendix D, photographs 43, 54-58), individual animal "lesions" observed on April 7, 1999 (Appendix D, photographs 44-48, 52, 53), photographs supplied to the team by Mr. Tennant (Appendix D, photographs 60-65), and a photograph of cattle team and steering committee members (Appendix D, photograph 66) are also included in this report.

**Adult Cattle Procedures**

| | |
|---|---|
| physical examination | fecal sample collection |
| body condition scored | blood collection |
| age estimation | ear tagged: animal identification |
| rectal pregnancy examination | ear tagged: insecticide |
| girth measurement | photographed |

Twelve calves were examined as a group. The smaller seven received insecticidal ear tags; the larger five received no ear tags as Mr. Tennant indicated that they were to be sold in the very near future.

Although the cattle team was prepared to do a post-mortem examination on one or more cattle, the team decided that there was no animal sufficiently ill to justify euthanasia and necropsy at the time of the visit.



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All animal biological samples (blood and feces) were taken back to New Bolton Center by Dr. Habecker. Blood samples were subjected to hematology, serum chemistry, and serology. Randomly selected fecal samples were subjected to fecal flotation.

The following clinical pathology parameters were evaluated:

Hematology

| | |
|---|---|
| leukocyte (total and differential) count | *hematocrit |
| erythrocyte count | *mean corpuscular hemoglobin |
| hemoglobin concentration | *mean corpuscular hemoglobin concentration |
| mean corpuscular volume | *red cell distribution width |
| platelet count | *platelet distribution width |
| mean platelet volume | |

*These parameters were calculated using the measured data.

Serum Chemistry

| | |
|---|---|
| aspartate aminotransferase | potassium |
| gamma glutamyl transferase | calcium |
| creatine kinase | chloride |
| blood urea nitrogen | magnesium |
| creatinine | phosphorus |
| total protein | copper |
| albumin | selenium |
| *globulin | pepsinogen |
| sodium | **prolactin |

* globulin was calculated from the measured albumin and total protein data.
** prolactin was measured from blood plasma derived from EDTA tubes (lavender top).

Serology

| | |
|---|---|
| bovine leukemia virus (e) | epizootic hemorrhagic disease of deer (agid) |
| bovine virus diarrhea virus (sn) | *Mycobacterium paratuberculosis* (Johne's) (e) |
| bovine virus diarrhea virus (mp) | leptospirosis (sn) |
| blue tongue virus (agid) | brucellosis (sn) |

(e) = elisa test; (sn) = serum neutralization test; (mp) = microplate assay; (agid) = agarose gel immunodiffusion test

April 8, 1999

Three members of the cattle team (Drs. Moisan, Munson, and Davis-Heller) met with Mr. Earl Tennant and recorded his recollection of the 1998-1999 herd health history (Appendix E).

Drs. Caspar and Horne gave all of the cattle team members a driving tour of the property between the Tennant barn and the Dry Run landfill, including a drive within the landfill. During those two trips, the team members were able to see the connecting pastures.

adjacent creeks, neighboring cattle grazing, and wildlife. The landfill was open and being utilized at the time of the site visit.

While on site, the cattle team made general observations regarding the environment, including the flora, fauna, and man-made structures. Core samples of three large round hay bales were collected (3 samples/bale) from bales in the barn yard. Mr. Tennant gave the team some receipts (dates ranged from 2/98 to 12/98) from complete, mixed feed, and mineral supplement he had purchased.

All plant biological samples (bailed hay core samples and grain sample) were subjected to nutritional analysis by the Forage Testing Laboratory of the North Carolina Department of Agriculture (Table 7). These data were used in an Excel spreadsheet program (CNCPS v 3.1) from Cornell University to evaluate the diets in two cow models under two feeding situations (Appendix F, models 1-4).

The following nutritional parameters were evaluated:

Hay and Grain Analysis

| | |
|---|---|
| dry matter | magnesium |
| crude protein | sulphur |
| unavailable protein | potassium |
| adjusted crude protein | copper |
| acid detergent fiber | iron |
| total digestible nitrogen | manganese |
| NE (lactation) | zinc |
| calcium | nitrate ion |
| phosphorus | selenium |
| sodium | |

3.2.4. Miscellaneous Data

During the site visit in April, Mr. Tennant was asked to notify Dr. Lisa Davis-Heller or Dr. Sarah Caspar if there were any future signs of disease problems in his herd. The team wished to collect whatever data might be relevant to their assessment of the health status of the Tennant herd.

Between April 8 and December 23, 1999, Mr. Tennant consulted twice with Dr. Davis-Heller. Subsequent to notifying Dr. Caspar of a newborn calf which was born with a "cloudy eye", Mr. Tennant brought the calf to Dr. Davis-Heller's clinic (April 21, 1999). Dr. Davis-Heller examined the calf and made a diagnosis. Mr. Tennant left with the calf. On May 26, 1999, Dr. Davis-Heller examined cow #30 for a lump under the right mandible.



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### 3.2.5. Wildlife

EPA and DuPont each sponsored environmental studies that included the trapping of small mammals in the grazing fields of Mr. Tennant's cattle. These reports were supplied to the cattle team by the steering committee. The data included in these reports, as it relates to small mammal pathology, were evaluated by the cattle team.

**Small Mammal Studies**

| Report Date | Report Title | Animal Data Section |
|---|---|---|
| November, 1997 (draft) | Dry Run Creek: Washington, Wood County, West Virginia. USEPA Environmental Response Team | Table 39: Results of Histopathology for the Meadow Vole |
| December 3, 1998 (letter) | Small Mammal Trapping Effort: Dry Run Landfill, Washington, WV (URS Greiner Woodward Clyde study) | Table 1: Data Summary, Small Mammal Investigation |

The cattle team was also made aware of periodic deer reproduction surveys conducted by Dr. Crum and associates of the West Virginia Department of Natural Resources (WV DNR) in the Dry Run area, however no reports were available.

## 4.0 RESULTS

### 4.1. Cattle

#### 4.1.1. Videotapes (Table 1)

Two videotapes of several hours duration were reviewed. Forty cattle scenes and 20 wildlife scenes were presented, depicting numerous lesions. The individual scenes (i.e., cases), along with the cattle team's diagnosis/comment, are presented in Table 1 in the order in which they were presented in the videotapes. The herd health problems identified in these videos were summarized as follows:

1. Keratitis: Corneal ulcers, scars and opacities, as well as blepharospasm, were observed in many cattle. These were all considered to be manifestations of "pinkeye", an infectious keratoconjunctivitis caused by bacteria such as *Moraxella bovis*. The facefly (*Musca autumnalis*) problem observed in these videos and the lack of effective fly control were consistent with the diagnosis of severe "pinkeye" in these cattle. Affected young and adult cattle were observed to have 2-3 to 25-50 or more faceflies feeding at the medial canthus of each eye and on the lacrimal secretions. Figures 1 and 2 are prints from videotape #2, illustrating the facefly problem on a calf and her dam, respectively. Both of these animals were blind from chronic keratoconjunctivitis. Figure 3 (videotape #2) demonstrates an early central corneal ulcer on the left eye of a calf; faceflies are seen by the lower eyelid.

2. Hair loss, neck and tail: The cervical alopecia was most consistent with a lice problem. The alopecia observed on the tails of some cattle (e.g., loss of "switch") may also have been due to lice, or may have been related to fescue mycotoxicity.

3. Poor shedding of hair coats: This clinical sign, most prominent in the late spring and summer segments of the videotapes, is consistent with multiple nutritional deficiencies as well as fescue mycotoxicosis. Figure 4 demonstrates a cow from videotape #2 that was described by Mr. Tennant as having delayed shedding of the winter coat.

4. Hair depigmentation: The discoloration of the cattle hair coats is a change most often associated with a nutritional deficiency, and is especially common in cases of copper deficiency.

5. Coronitis: Alopecia and erythema above the coronary band may be seen with fescue mycotoxicosis, mechanical trauma, or as a non-specific dermatitis. Figures 5 and 6 (videotape #2) are representative of the erythematous foot lesions observed by Mr. Tennant in many of his cattle. Figures 7 and 8 (videotape #2) ) demonstrate the cattle's preference for standing in the water when these lesions were present. Fescue mycotoxicosis ("fescue foot") was considered the most likely diagnosis of this condition.

6. "Hunched up" or painful stance: This is a clinical sign often associated with abdominal or foot pain, but has been specifically described as a manifestation of the fescue endophyte toxicity complex.

7. Thin body condition: Poor body condition is a non-specific finding and the cases presented most likely represented different etiologies. The emaciated dead calf with serous atrophy appears to have starved while others appear to have had diarrhea or mastication problems. Considering the other findings in this herd (e.g., feed analysis), nutritional insufficiency (protein-energy malnutrition) was also a likely contributing factor to poor body condition.

Some findings described in these tapes were difficult to evaluate by video although they probably were real. For example, the described "lumps" in a cow udder (case # 54) was difficult to see but would be considered an incidental finding in any case. A few cattle were described as "hunched-up" or "humped-up" (cases # 1, 5, 17) – these may have been related to lameness, hardware disease (traumatic reticulopericarditis), fescue mycotoxicosis, or other condition. An individual cow that was slobbering, losing cud, and urinating (case # 13) may also have had hardware disease, although the diagnosis was not clear. Another individual cow (case # 45) which was panting was consistent with fescue mycotoxicosis (i.e., "summer fescue" hyperthermia), although this presentation was certainly non-specific.

### 4.1.2. Diagnostic Pathology Reports (Appendix B)

Only four of Mr. Tennant's cattle were submitted for post-mortem diagnostic evaluation of some type. The most significant finding was moderate to marked copper deficiency in the three cows analyzed for heavy metals. Keratitis was observed in the only animal that was formally necropsied. This 6-month old bull calf apparently died from winter starvation (i.e., negative energy balance or protein-energy malnutrition) complicated by untreated intestinal coccidiosis. These reports are included in Appendix B; summaries are presented below:

a. Dead 6-month old bull calf submitted to the Animal Disease Diagnostic Laboratory, Ohio Department of Agriculture, Reynoldsburg, OH on 2/27/97; Laboratory report dated 3-10-97 (Appendix B)

| | |
|---|---|
| History: | 6-month old bull calf; underweight; diarrhea for one month; received no medication; corneal opacities; died. |
| Gross diagnosis: | Emaciation with serous atrophy of fat<br>Intestinal parasitism (trichuriasis) |
| Histopathologic diagnosis: | Enteric coccidiosis<br>Keratitis, moderate, chronic, focally extensive |

b. Tissues from a 4-year old Holstein cow submitted to the Animal Health Diagnostic Laboratory, College of Veterinary Medicine, Michigan State University, Lansing MI on 3/4/97; Laboratory report dated 3-12-97 (Appendix B)

| | |
|---|---|
| History: | 4-year old Holstein cow; died 2-18-97; dissected by owner; tissues submitted by EPA |
| Heavy metal screen: | Copper deficiency in liver (1.9 ppm vs. 25-150 reference range) and kidney (2.37 ppm vs. 4 – 6 ppm reference range).<br>Manganese was marginally low in liver; cadmium was slightly increased in the kidney.<br>No heavy metals found in urine (urine fluoride was 6.6 ug/ml)[reference range: toxic if ≥ 14 µg/mL] |

c. Tissues from a 9-year old Holstein cow submitted to the Animal Health Diagnostic Laboratory, College of Veterinary Medicine, Michigan State University, Lansing MI on 3/4/97; Laboratory report dated 3-12-97 (Appendix B)

| | |
|---|---|
| History: | 9-year old Holstein cow; died 3-2-97; dissected by owner; tissues submitted by EPA |

| | |
|---|---|
| Histopathologic diagnosis: | (only heart, liver, and kidneys submitted: autolysis and freeze/thaw artifact)<br>Myocardial sarcocysts |
| Heavy metal screen: | Copper deficiency in liver (2.03 ppm vs. 25-150 reference range) and kidney (2.33 ppm vs. 4 – 6 ppm reference range).<br>Manganese was marginally low in liver; iron was elevated in the liver; cadmium was slightly increased in the kidney.<br>Urine heavy metals were normal (urine fluoride was 5.55 ug/ml)[reference range: toxic if ≥ 14 µg/mL] |
| Clinical pathology: | No significant findings |

d. Tissues from a 7-year old red cow (euthanized on 6/10/99 and dissected by Mr. Tennant) submitted to the Laboratory of Large Animal Pathology and Toxicology, School of Veterinary Medicine, University of Pennsylvania, Kennett Square, PA on 6/27/99: Laboratory report dated 7-5-99 (Appendix B)

| | |
|---|---|
| History: | 7-year old red cow; euthanized 6-10-99; dissected by owner: tissues submitted by Dr. Davis-Heller. |
| Histopathologic diagnosis: | Enteric lesions of minimal significance (forestomach abscesses: intestinal coccidiosis; myocardial sarcocysts). |
| Heavy metal screen: | Copper deficiency in liver (7.71 ppm vs. 25-150 reference range). |

### 4.1.3. April 1999 Site Visit Data

#### a. Herd History (Appendix E)

The herd history (Appendix E) was based on Mr. Tennant's recollections during the interview on April 8, 1999. A written record of herd health was conspicuously absent. Much of this can be attributed to the lack of outside intervention. There is no record of veterinary care or consultation with an animal nutritionist. Minimal medications have been used on these animals, and there has been no use of vaccines or modern dewormers. Except for the few animals noted in this document (see Diagnostic Pathology Reports, above), no diagnostic laboratory tests were done on dead animals. The level of cattle herd management has not changed since the installation of the landfill.

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## b. Physical Examination (Table 2)

Age, Pregnancy Status

On April 7, 1999, the cattle team examined 41 animals, including 38 adult cows, 2 adult bulls, and 1 adult steer (Table 2). Estimated ages for the adult cows ranged from 4 to greater than 9 years. This is a herd of aged cattle; 32 of the 38 cows were estimated to be older that 9 years. Twenty-seven (27/38) cows were pregnant.

Body Condition Scores

Body condition scores (BCS) were based on a 1-9 scale. Scores ranged from 2 to 7; the average score was 3.5 (3=thin, 4=borderline, 5-7 is considered optimum).

Clinical Signs

Haircoat abnormalities were evident in 9 of the 41 cattle. One animal had an epidermal inclusion cyst that was lanced and emptied. Mammary gland lumps, consistent with chronic mastitis, were interpreted as abscesses or accumulations of fibrous connective (scar) tissue. Rectal examination of one cow suggested the presence of intra-abdominal fat necrosis.

## c. Clinical Pathology (Tables 3 – 6; Graph 1)

Hematology

Erythron (Table 3): Red blood cell indices were generally in the low-normal range. No animals were considered anemic.

Leukon (Table 4): The total white blood cell counts were within the normal range. Differential cell counts were considered invalid due to the 36 hour delay between collection and testing.

Clinical chemistry (Table 5)

Most electrolyte values were in the high-normal to slightly elevated range, consistent with mild dehydration. Since the cattle were penned during the daylight hours of April 7, which was unseasonably warm, and had no access to water, clinical dehydration was not unexpected. Dehydration is also the most likely explanation for the elevated creatinine in 40/41 cattle.

Most (27/41) of the cattle had elevated total protein, which also correlates highly with a diagnosis of dehydration (total protein = globulin fraction + albumin fraction). In all cattle (41/41) the globulin fraction was elevated while the albumin fraction was within the reference range.

Gamma glutamyltransferase (GGT), a sensitive indicator of active liver disease, was consistently within the normal reference range. Creatine kinase (CK), an indicator of muscle necrosis, was also normal in all of the cattle. However, aspartate aminotransferase (AST), a less sensitive indicator of liver and muscle degeneration, was minimally elevated in 33/41 cattle. This might be from leukocyte dissolution, the result of the 36 hour interval between blood collection and testing.

Special chemistry

Prolactin: Plasma prolactin levels (Table 4 and Graph 1) were significantly depressed. According to the testing laboratory (Dr. Neal Schrick, University of Tennessee), an average prolactin level for cattle on fescue-free pasture (for April) was 171.6 micrograms/liter (ng/mL) (i.e., reference mean = 171.6 ng/mL). The average of the 41 Tennant samples was 110.1 ng/mL and 36/41 of the Tennant cattle were below the reference mean (171.6 ng/mL). In contrast, an average prolactin level for cattle on endophyte-infected fescue (simulated by ergotamine tartrate administration) was 105.8 ng/mL (data from Dr. Schrick). This average was similar to the Tennant herd average (110.1 ng/mL) and 24/41 of the cattle were below this reference level (105.8 ng/mL). According to Dr. Schrick, these findings were highly supportive of endophyte toxicity in the Tennant herd.

Copper: Blood copper levels were in the deficient range for 26 of 41 animals; one copper assay was below the limit of detection.

Selenium: Blood selenium levels were normal for 41 of 41 cattle.

Pepsinogen: Pepsinogen, a blood enzyme indicator of parasite damage to the abomasum, was measured in 10 cows, one steer and one bull. All values were within the normal reference range. These data supported the conclusion that abomasal ostertagiasis was not a problem in adult cattle in this herd.

Serology (Table 6)

| | |
|---|---|
| BLV (Bovine Leukemia Virus) | 1/41 positive |
| Johne's (*Mycobacterium paratuberculosis* antigen) | 1/14 positive |
| Brucella | 0/41 |
| BVD (Bovine Virus Diarrhea antigen) | 2/41 positive |
| BVD Microplate Assay (viremia detection) | 0/41 |
| Bluetongue | 0/41 |
| Leptospira interrogans (5 subvarieties) | 3/41 positive |
| EHD type 2 (Epizootic Hemorrhagic Disease of deer) | 2/12 positive |

None of the serology values suggested a herd-wide problem. Positive titers for BVD and Leptospira might reflect residual vaccination titers in purchased animals or natural exposure to these diseases. The discovery of two positive EHD titers was evidence that this disease has been in the local deer population.

Parasitology

Routine fecal flotation (Table 4) revealed parasite ova in 11 of 14 randomly selected fecal samples. The magnitude of ova shedding could be categorized as rare to moderate. Parasite groups included coccidia, tapeworms, and strongyle-type nematodes. Intestinal parasitism was not considered to be a major herd problem in the adult cattle. The shedding of small numbers of oocysts and helminth ova is expected of mature cattle.

## d. Hay and Grain Analysis (Table 7)

During the cattle team's visit to the Tennant farm (4/8/99), Mr. Tennant was interviewed regarding herd feeding practices. Three large round bales of hay in the barnyard, identified as representative of the herd's forage, were visually inspected and core samples (3/bale) were collected for analysis. This forage (i.e., hay, grass/fescue) was fed *ad libitum*.

During the site visit, forage quality was subjectively evaluated as poor. The fiber level of the hay appeared to be high and forage analysis (Table 7) later substantiated that observation. Fiber levels of forage play an important role in determining how much of a feed can be consumed. Dry matter intake (DMI) is inversely related to fiber levels.

According to Mr. Tennant, mature animals, on the Tennant farm, were also fed five pounds per head per day of a grain mix composed of dry ear corn, soybean meal, and minerals (rate could not be validated, despite the existence of grain receipts). A sample of this feed was taken and analyzed.

An Excel spreadsheet program (CNCPS v 3.1) from Cornell University was used to evaluate the diets. Using the forage and grain nutritional analysis conducted by the Forage Testing Laboratory of the North Carolina Department of Agriculture (Table 7), two models for mature cows (early lactation and dry pregnant) were selected to examine the current feeding program (Appendix F).

According to the computer models, a cow in early lactation (model #1) in the spring of 1999 fed five pounds of this grain and free choice hay was in severe negative energy balance (72.5% of requirement) and less severe protein deficiency (90.9% of requirement). The limited nutrients available would have a negative impact on milk production, persistency of production, and calf growth. A cow under this feeding system would require 2.4 times the corn currently being fed to be isocaloric (model #2). Similarly, a dry pregnant cow (model #3) in the same feeding system would also be in negative energy balance (75.2% of requirement) but not deficient in protein (125%). The same dry pregnant cow maximizing dry matter intake (model #4) would still be in negative energy balance (87.7%).

The body condition scores (BCSs) of the herd were consistent with the energy deficit of the diets. The impact of the poor quality forage on this farm and subsequent energy deficits were considered to have chronic effects well after the cows return to adequate pasture.

## 4.2. Miscellaneous Data

Following the site visit, Mr. Tennant consulted with Dr. Davis-Heller regarding a suspected sick animal. Mr. Tennant brought a calf with a "cloudy eye" to Dr. Davis-Heller's clinic (April 21, 1999). Dr. Davis-Heller examined the calf and determined that there was nothing wrong with the eye, except for a slight mucoid exudate on the cornea which was easily wiped away.

On May 26, 1999, Dr. Davis-Heller examined cow #30 for a lump under the right mandible. The lesion was aspirated (pus) and subsequently lanced.

## 4.3. Wildlife

### 4.3.1. Videotapes (Table 1)

The 20 wildlife cases presented in the videotapes were all dead and, except for one case (case # 49), of no diagnostic value. These were all considered to be incidental deaths since there did not appear to be any consistency in the species, location, or timing of the deaths. In fact, these deaths appeared to be most consistent with the random wildlife carcasses which would be found in a healthy ecosystem. The individual dead deer (case # 49) which presented with hemorrhage from the nostrils may have died from epizootic hemorrhagic disease (EHD). This diagnosis would correspond with the known existence of this disease in local deer (personal communication: Dr. Crum to Dr. Sykes) and the finding of some EHD seropositive cattle in the Tennant herd.

### 4.3.2. Small Mammal Data

Two pathologists reviewed the liver and kidney histology from 45 small rodents (voles, shrews, and mice) captured on the Tennant property in 1997 (USEPA Environmental Response Team draft report: Dry Run Creek, Washington, Wood County, West Virginia; Table 39). No lesions characteristic of hepatotoxicity or nephrotoxicity were evident. Although several anomalies were identified, none were considered suggestive of toxic teratogenesis.

The 1998 small mammal trapping (URS Greiner Woodward Clyde study, December 3, 1998) observed no gross abnormalities in the captured animals (voles, shrews, and mice).

### 4.3.3. Deer Studies

The cattle team was made aware of the periodic deer reproduction surveys conducted by the West Virginia Department of Natural Resources (WV DNR) in the Dry Run area. Although the team reviewed some of the data sheets from these studies, no reports were available. One team member (Sykes) had a telephone conversation with Dr. Crum of the

WV DNR regarding deer herd health in the Dry Run area in order to acquire some additional information. From this conversation, it was the cattle team's understanding that:

- evidence of low fertility rates in young does was probably attributable to deer overpopulation;
- epizootic hemorrhagic disease (EHD) has been diagnosed clinically and serologically in dead and captured deer, respectively;
- bovine virus diarrhea (BVD) has been diagnosed serologically in deer not far from Dry Run;

In addition, the team has learned that outbreaks of EHD in West Virginia deer were reported between 1980 and 1989 [Southeastern Cooperative Wildlife Disease Study publication, Field Manual of Wildlife Diseases in the Southeastern U.S., 2nd ed.].

Since there was no evidence of an EHD or BVD outbreak in the Tennant herd, the deer population was not considered to be a contributing factor in the cattle herd health problems. Also, there was no evidence that the deer herd was a useful sentinel species for any of the disease entities identified in the Tennant herd.

## 5.0 DISCUSSION

The six veterinarians comprising the cattle team agreed that there was conclusive evidence that the Tennant herd was suffering from four major disease entities: endophyte toxicity, pinkeye, malnutrition, and copper deficiency. The clinical, laboratory, and historical data substantiate that these four conditions can readily account for the chronic herd health problems on the Tennant farm.

Endophyte toxicity

According to the history acquired on April 8, 1999, the herd has been fed a diet of KY31 fescue hay in the winter and pasture that approaches 100% KY31 fescue during the summer. There has been no supplemental pasture or other forage fed to these animals. The clinical signs in the Tennant herd that highly suggest endophyte toxicity (fescue mycotoxicosis) include:

- swelling and pain at the coronary band (coronitis) and above, with cattle that stand in the creek during hot weather (Figures 5-8);
- poor shedding of winter coats (Figure 4);
- patchy tail alopecia with loss of tail switch;
- birth of undersized calves;
- poor conception and calving rates;
- numerous vague diseases suggestive of immune dysfunction;

Other clinical signs that are consistent with endophyte toxicity include:

- fat necrosis, presumptive, as diagnosed by rectal palpation in cow #15;
- concomitant presence of copper deficiency;
- panting in cow #45, suggestive of hyperthermia;
- general failure of the cattle to thrive.

Vasoconstriction is seen as a result of ingestion of a number of ergot-like alkaloids. The endophyte fungus associated with fescue mycotoxicosis is *Acremonium coenophialum* which produces perloline, perlolidine, N-acetyl loline, and N-formyl loline (Stuedemann, et al., 1985). These alkaloids accumulate in the fescue grass seed during growth, acquiring the highest concentrations at the greatest maturity of the grass. Additionally, the alkaloids may be found in stored hay.

The coronitis problem observed in the videotapes (Figures 5 and 6) was typical of that produced by endophyte toxicity in the "fescue foot" syndrome. Signs of fescue foot generally start with reduced weight gain, or loss of weight, rough hair coat, arched back, and soreness in one or both rear limbs. Hyperemia of the coronary band occurs between the dewclaws and hooves and is generally accompanied by some swelling (Hemken, et al., 1984). Arching of the back was reported by the owner in the videotapes as "hunched" and "humped" posture in some cattle.

Peripheral vasoconstriction from these vasoactive alkaloids results in decreased ability to dissipate heat during hot weather. This phenomenon is known as "summer slump" (Osborne, et al., 1992). The panting and drooling by a red cow (#45) in videotape #2 was suggestive of the summer slump syndrome.

The birth of undersized calves to dams ingesting a diet high in endophyte-infested fescue has also been documented (Bolt, et al., 1986). This may be by a mechanism similar to the vasoconstriction caused by the ergot-like alkaloids. Vasoconstriction of the uterine blood vessels may result in decreased circulation to the growing fetus. The effect has been documented in mice that were exposed to the endophyte during gestation. Mouse pups from females fed the material during gestation were a lighter birth weight, were delayed in post-partum development, and grew slower than control mice (Varney, et al., 1991).

Evidence that endophyte toxicity in fescue grazing cattle may be responsible for the loss of the tail switch is largely anecdotal, from veterinary practitioners that have experience with similarly affected cattle. The most severe forms of endophyte-induced vasoconstriction result in dry gangrene of the tail and digits (Radostits, et al., 1994; Hemken, et al., 1984).

Reproductive effects from the ingestion of endophyte-infested fescue include incrementally depressed conception rates in cattle grazing infected pasture (Paterson, et al., 1995). In the Tennant herd, the grazing of pastures with 100% KY31 fescue could potentially play a major role in the unacceptable conception rate. A poor conception rate

could also be secondary to a lack of intensive breeding management at the Tennant farm, where breeding occurs year round. The advanced age of most animals in the breeding herd could also be expected to be a cause for depressed fertility and an extended calving interval. Likely, a combination of all three factors have contributed to suboptimal fertility.

The poor growth of the nursing calves could be multifactorial as well. The advanced age of the cows or a limited genetic potential in the herd could lead to poor milk production and poor calf growth. Similarly, lack of proper nutrition or a lack of a balanced diet among the brood cows could depress calf growth. The ingestion of endophyte-infested fescue could lead to depressed milk production due to prolactin inhibition (Hurley, et al., 1980; Paterson, et al., 1995). The mechanism is considered to be due to the dopaminergic effects of the endophyte ergopeptides on prolactin production in the bovine pituitary gland (Schultze, et al., 1999).

Endophyte intoxication is also considered to have an immunosuppresive effect by an apparently indirect route. Although anecdotal evidence exists to suggest that fescue intoxication is responsible for depressed immune function, there appears to be no apparent lack of immune response to vaccinations of cattle fed diets high in fescue (Rice, et al., 1997). Indirectly, however, there is a relationship between immune function, endophyte intoxication and copper status of cattle on a high fescue diet (Dennis, et al., 1998; Saker, et al., 1998). Steers grazing endophyte-infested tall fescue have lower serum copper concentrations than those not on a high-endophyte tall fescue diet, resulting in depressed monocyte-macrophage functions. This is apparently an effect of the decreased uptake of copper by the infected tall fescue plants. Another source has recorded depressed globulin levels in animals grazing infected pastures (Schultze, et al., 1999).

Fat necrosis is a herd problem of cattle grazing tall fescue with high endophyte levels. The necrotic fat accumulations are mostly within the omental and retroperitoneal fat depots, and consist of hard masses of partly mineralized fat. Cow #15 had palpable masses within the abdominal cavity that were identified during the herd exam on April 7, 1999. These masses were most suggestive of masses of necrotic fat. The biochemical reaction causing the fat necrosis is unclear, however, in affected animals that are on a diet high in N-formyl and N-acetyl loline, there is a consistent reduction of circulating levels of cholesterol (Stuedemann, et al., 1985). Animals with increased dietary levels of endophyte-infected fescue had decreased body condition and increased fat necrosis.

Pinkeye

Faceflies were a serious problem in the cattle in the videotaped recordings taken on the Tennant farm (Figures 1 and 2). Additionally, they have also been a problem in other years, according to the history provided by Mr. Tennant during the April 8, 1999 interview. Faceflies were seen in small numbers at the eyes of the cattle during the April 7, 1999 herd visit, representing an early spring infestation. The facefly, *Musca autumnalis*, is a vector for *Moraxella bovis*, the etiology of most bovine pinkeye. Control



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of these insects is based on preventing the flies from feeding on the lacrimal secretions of the cattle. Insecticides and insecticidal ear tags, as applied during the visit, are effective controls. Secondarily, the insecticides tend to limit the spread of *M. bovis* between susceptible cattle during outbreaks of pinkeye.

Severe facefly infestations can result in poor feed consumption and conversion among affected cattle. Some of the poor weight gain described in the history of the herd and the videos may be due to "fly worry" The manic stampeding of the cattle, that was described in one of the videotapes, was likely due to the large number of flies.

Malnutrition

Malnutrition was considered to be a significant problem in the Tennant herd. The owner had identified poor growth rates and infertility as specific problems among his cattle. This was considered a manifestation of protein-energy insufficiency and was reflected in the low body condition scores. Dietary requirements of beef cattle change dramatically during the life cycle of the animals and during the year, particularly with respect to such environmental and physiological stressors as fluctuating ambient temperatures and moisture conditions, lactation, growth, and breeding (Rice, 1991). The clinical impression of malnutrition that followed the visits of April 7 and 8 were confirmed by the results of the feed analysis of the hay and grain mix rations taken during the visits.

Copper deficiency

As indicated by the heavy metal analysis performed by two laboratories, on tissues from three animals, copper deficiency is a problem in the Tennant herd (Appendix B). In 1997 copper deficiency was diagnosed in a 4-year old Holstein cow and a 9-year old Holstein cow by the Animal Health Diagnostic Laboratory of Michigan State University in East Lansing, Michigan. In 1999, copper deficiency was revealed in tissues from a sacrificed 7-year old red cow by the Laboratory of Large Animal Pathology and Toxicology of the University of Pennsylvania, Kennett Square, Pennsylvania. Furthermore, serum samples taken from 41 adult animals at the farm on April 7, 1999 revealed that 26 of the 41 (63%) had serum copper levels below that considered deficient at the reference laboratory (Table 4).

Clinical signs of copper deficiency seen on the Tennant farm were lightening of the hair coats, poor quality hair on the cows, and overgrown hooves. This was reported by Mr. Tennant and shown in videotapes made of the herd.

The clinical effects of copper deficiency are multiple, and the clinical and physiological effects of such deficiency are well documented (NRC, 1996). The effects include immune deficiencies, poor quality hair coats, increased fragility of bone and sudden death in extreme cases. During disease, copper deficiency can result in depressed levels of tumor necrosis factor (a cytokine) with resulting abnormal temperature responses to infections and depressed feed intake (Gengelbach, et al., 1997).

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Immune deficiencies related to copper deficiency are multiple and include depressed acute phase protein response and lymphocyte responsiveness to mitogen stimulation (Arthington, et al., 1996; Gengelbach, et al., 1997).

As there has been no documentation of any copper supplementation of the herd, the deficiency is expected to be present at this time and to continue. The relationship between high endophyte fescue grazing and copper status in the feed has been described (Dennis, et al., 1998). Copper deficiency is widespread throughout the United States (Dargatz, et al., 1999).

Toxicology issues

Based upon the draft report entitled Dry Run Creek, 1997 from the Environmental Response Team of the US EPA, carnivorous, piscivorous, omnivorous, insectivorous and herbivorous mammals in the Dry Run Creek study area are at increased health risk due to exposure to metals, fluoride and trichlorofluoromethane. At least with regard to metals of concern, diagnostic evaluation of tissue and fluids collected from three animals from the Tennant farm (two submitted to Michigan State University and one submitted to New Bolton Center) do not suggest elevated concentrations of any metal. In addition, urine (two samples submitted to Michigan State University) and bone (one sample submitted to Michigan State University from the cow evaluated at New Bolton Center) samples for fluoride analysis did not measure concentrations above expected or "background" values. The urinary fluoride values were 6.60 ppm and 5.55 ppm and one bone fluoride value was 1090 ppm (expressed on a fat free, dry weight basis). Urine and bone fluoride concentrations in adult cattle consistent with fluorosis are 15 to 20 ppm or greater and 3000 ppm or greater (expressed on a fat free, dry weight basis), respectively (Osweiler et al., 1985). In addition, there was no clinical evidence of chronic fluorosis in the Tennant herd.

Exposure to trichlorofluoromethane was considered to be a default risk factor based upon a lack of toxicologic benchmarks for this compound. However, available toxicity data derived from inhalation studies using common laboratory animals indicate that chlorofluorocarbons such as trichlorofluoromethane have low acute and chronic toxicity (Magda, 1999). Signs associated with acute toxicity are reversible effects on the central nervous system such as lethargy and incoordination. Chlorofluorocarbons are not developmental toxicants, do not affect reproductive performance and are not genotoxic.

## 6.0 RECOMMENDATIONS

The most important general recommendation from the cattle team, after completion of the herd visit and review of the investigatory data, would be for the owner of the Tennant herd to engage veterinary and nutritional consultants in the design of a herd health program.

Endophyte toxicity: With dietary forage that contains nearly 100% KY31 fescue, fed during all seasons, the immediate goal would be for the dilution of the endophyte in the diet by mixing the hay and pasture with either non-endophyte infested fescue or another different forage source. A short-term goal would be to reduce the content of endophyte-infested hay and pasture to 50% or less of the diet, primarily by supplementing the diet with higher quality forage. An acceptable long-term plan would be to replace the fescue pasture and hay fields with a non-endophyte-infested forage crop or crops over time, again with the goal of diluting the dietary endophyte consumption. Crop management should be attempted under the supervision of specialists in forage and grazing management.

Pinkeye: Prevention of pinkeye (keratoconjunctivitis) in the immediate future should be attempted by an approach that limits the spread of the infectious organism, *Moraxella bovis*, from carrier cattle to susceptible cattle. Effective fly control, through the use of insecticide-impregnated eartags or some other proven method, is perhaps the most effective control method that should be practiced during the warm months. The insecticide-laden eartags should be rotated every year in order to circumvent the development of insecticide resistance by the local facefly population. An additional long-term approach that may be employed is the breeding for cattle that have dark faces. By selecting for dark-faced cattle, the corneal damage that is caused by the ultraviolet light of the sun is minimized. However, while minimizing the damage to the corneas of the cattle may prevent overt lesions of keratoconjunctivitis, the loss of condition from severe fly infestations will not be avoided by selecting for face color in cattle. Thus regardless of the type of cattle in the herd, insecticide fly repellants should be used every year.

Malnutrition: Rations and pasture nutrition should be planned with the assistance of a specialist in beef cattle nutrition. In the Tennant herd, there was evidence of gross protein-energy malnutrition, as well as concerns about macromineral (calcium, phosphorus, and magnesium) nutrition. Trace mineral and vitamin deficiencies are common to many beef herds and should also be addressed. In the development of a ration for this herd, particular attention should be paid by the nutritionist to the local trace mineral and macromineral deficiencies.

Copper deficiency: Copper is a trace mineral that is crucial to the health of cattle yet is missing from the diet of cattle in many areas of the North America. As copper deficiency was suspected clinically and confirmed biochemically in the Tennant herd, the provision of a high copper trace mineral supplement, again under the supervision of a beef cattle nutritionist, should be a priority year round. This is especially critical considering the prevalence of endophyte-infested fescue on the Tennant farm.

## 7.0 CONCLUSION

There was conclusive evidence that the Tennant cattle herd was, and continues to be, suffering from four major disease entities, some of which were potentially interrelated: endophyte toxicity (fescue mycotoxicosis), pinkeye, malnutrition, and copper deficiency. As substantiated by the clinical and laboratory findings, and historical data, these four conditions readily account for the chronic herd health problems on the Tennant farm.

The herd health investigation revealed deficiencies in herd management, including poor nutrition, inadequate veterinary care, and lack of fly control. The lack of vaccination and internal parasite control programs did not appear to have a substantial impact on this relatively isolated herd.

Despite an exhaustive review of historical and contemporary herd data, there was no evidence of toxicity associated with chemical contamination of the environment.

## 8.0 REFERENCES

Endophyte Toxicity References

1. Bolt DJ, Bond J: Effects in pregnant beef heifers grazing fungus-infected tall fescue on calf birth weight, milk yield, and calf growth. J Anim Sci 63(suppl):297, 1986.

2. Dennis SB, Allen VG, Saker KE, Fontenot JP, Ayad JYM, Brown CP: Influence of *Neotyphodium coenophialum* on copper concentration in tall fescue. J Anim Sci 76:2687-2693, 1998.

3. Hemken RW, Jackson JA, Boling JA: Toxic factors in tall fescue. J Anim Sci 58:1011-1016, 1984.

4. Hurley WL, Convey EM, Leung K, Hemken RW: Bovine prolactin, TSH, T4 and T3 concentrations as affected by tall fescue summer toxicosis and temperature. J Anim Sci 51:374-379, 1980.

5. Osborne TG, Schmidt SP, Marple DN, Rahe CH, Steenstra JR: Effect of consuming fungus-infected and fungus-free fescue and ergotamine tartrate on selected physiological variables of cattle in environmentally controlled conditions. J Anim Sci 70:2501-2509, 1992.

6. Paterson J, Forcherio C, Larson B, Samford M, Kerley M: The effects of fescue toxicosis on beef cattle productivity. J Anim Sci 73:889-898, 1995.

7. Radostits OM, Blood DC, Gay CC: Diseases caused by toxins in plants, fungi, cyanobacteria, clavibacteria, insects, and animals. In: Veterinary Medicine: A

textbook of the diseases of cattle, sheep, pigs, goats, and horses, 8th ed., Radostits OM, Blood DC, Gay CC, editors. Baillière Tindall, Philadelphia 1532-1611, 1994.

8. Rice RL, Blodgett DJ, Schurig GG, Swecker WS, Fontenot JP, Allen VG, Akers RM: Evaluation of humoral immune responses in cattle grazing endophyte-infected or endophyte-free fescue. Vet Immun Immunopathol 59:285-291, 1997.

9. Saker KE, Allen VG, Kalnitsky J, Thatcher CD, Swecker, Jr. WS, Fontenot, JP: Monocyte immune cell response and copper status in beef steers that grazed endophyte-infected tall fescue. J Anim Sci 76:2694-2700, 1998.

10. Schultze AE, Rohrbach BW, Fribourg HA, Waller JC, Oliver JW: Alterations in bovine serum biochemistry profiles associated with prolonged consumption of endophyte-infected tall fescue. Vet Human Toxicol 41:133-139, 1999.

11. Stuedemann JA, Rumsey TS, Bond J, Wilkinson SR, Bush LP, Williams DJ, Caudle AB: Association of blood cholesterol with occurrence of fat necrosis in cows and tall fescue summer toxicosis in steers. Am J Vet Res 46:1990-1995, 1985.

12. Varney DR, Varney LA, Zavos PM, Wiglesworth MD, Siegel MR: Tall fescue endophyte: effect on congenital development and pup growth in mice. J Dairy Sci 74:460-466, 1991.

Malnutrition References

13. Rice LE: The effects of nutrition on reproductive performance in beef cattle. Vet Clin North Amer – Food Anim Pract 7:1-26, 1991.

Copper Deficiency References

14. Arthington JD, Corah LR, Blecha F: The effect of molybdenum-induced copper deficiency on acute-phase protein concentrations, superoxide dismutase activity, leukocyte numbers, and lymphocyte proliferation in beef heifers inoculated with bovine herpesvirus-1. J Anim Sci 74:211-217, 1996.

15. Dargatz DA, Garry FB, Clark GB: Serum copper concentrations in beef cows and heifers. JAVMA 215:1828-1832, 1999.

16. Dennis SB, Allen VG, Saker KE, Fontenot JP, Ayad JYM, Brown CP: Influence of *Neothyphodium coenophialum*_on copper concentration in tall fescue. J Anim Sci 76:2687-2693, 1998.

17. Gengelbach GP, Ward JD, Spears JW, Brown TT: Effects of copper deficiency and copper deficiency coupled with high dietary iron or molybdenum on phagocytic cell function and response of calves to a respiratory disease challenge. J Anim Sci 75:1112-1118, 1997.

18. National Research Council: Minerals. in: Nutrient Requirements of Beef. 7th ed. National Academy Press. Washington, D. C. 54-74, 1996.

Toxicology Issues References

19. Magda S: Fluorocarbons. In: Toxicology, 1st ed., Marquardt H, Schafer SG, McClellan R and Welsch F, editors., Academic Press, San Diego, 659-662, 1999.

20. Osweiler GD, Carson TL, Buck WB, Van Gelder GA, editors: Fluoride. Clinical and Diagnostic Toxicology. Kendall/Hunt Publishing, Dubuque, IA, 183-188, 1985.

21. U.S.E.P.A. Environmental Response Team: Dry Run Creek, 1997 (draft).

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# 9.0 GRAPHS

Graph 1: Individual Cattle Plasma Prolactin (April 7, 1999).

Heparinized blood samples taken from 41 adult cattle during the cattle team's visit to the Tennant farm were analyzed for the endophyte-responsive hormone, prolactin. Graph illustrates the laboratory's reference means for endophyte-free (171.6 ng/mL) and endophyte-infested (105.8 ng/mL) April pasture. The average plasma prolactin value for the Tennant herd (110.1 ng/mL) was similar to the endophyte-infested reference. These results were highly supportive of the diagnosis of endophyte mycotoxicosis in the Tennant herd. (Testing laboratory: Dr. Neil Schrick, University of Tennessee)

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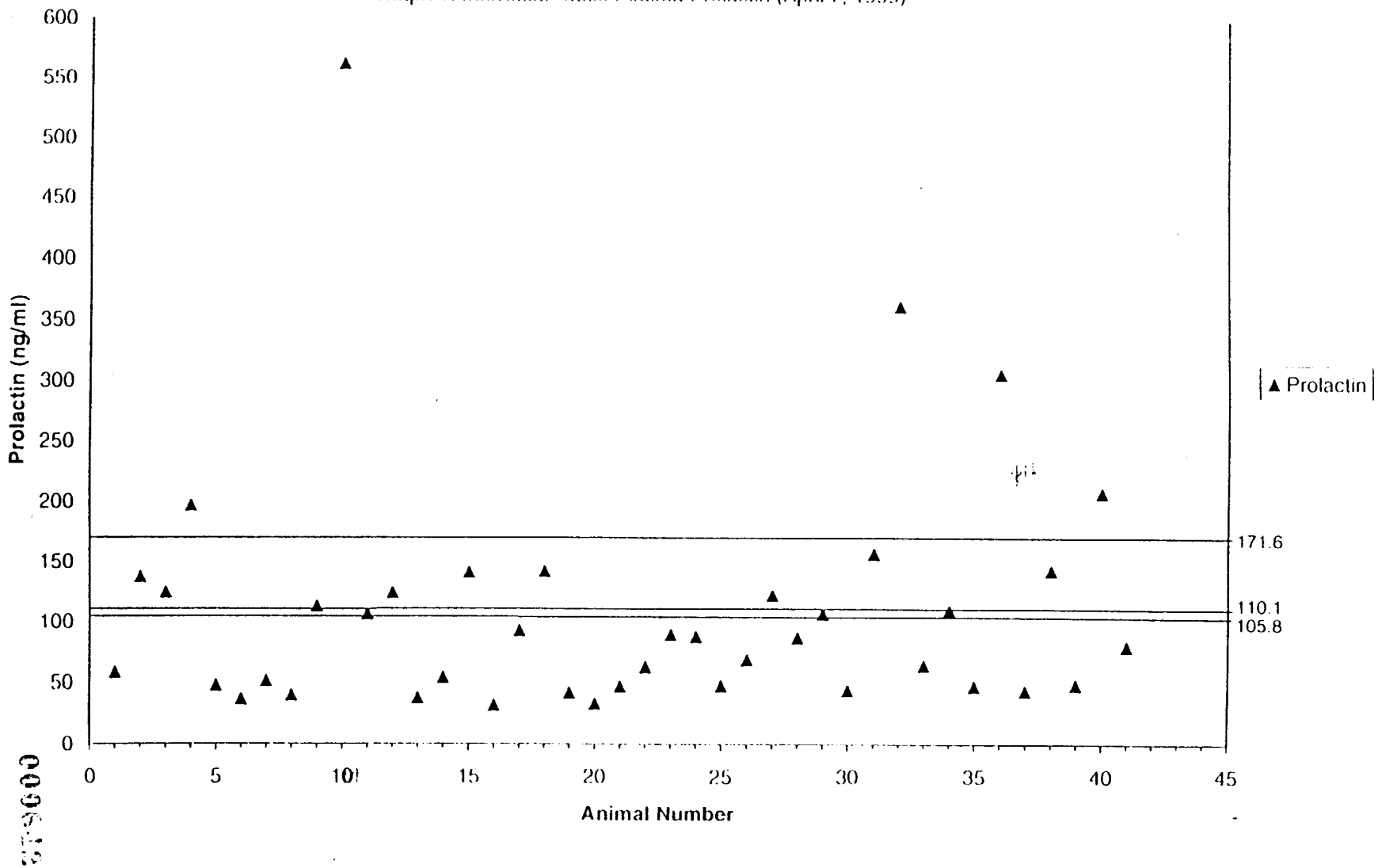
Graph 1: Individual Cattle Plasma Prolactin (April 7, 1999)

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# 10.0 FIGURES

Figure 1: Faceflies on the head of a calf.
Facefly (*Musca autumnalis*) infestation was considered to be a major disease problem on the Tennant farm. In addition to the constant harassment by flies, this calf had severe bilateral keratoconjunctivitis and appeared to be clinically blind on the videotape (photo was taken from Tennant videotape #2).

Figure 2: Faceflies on the head of a cow.
This cow was the dam of the calf in figure 1. Like her calf, she had severe bilateral keratoconjunctivitis. (photo was taken from Tennant videotape #2).

Figure 3: Left eye of a calf with faceflies and central corneal opacity.
Faceflies are a vector for the bacterial agents of pinkeye (keratoconjunctivitis) such as *Moraxella bovis*. Central corneal opacities may progress to diffuse corneal ulceration and clinical blindness. (photo was taken from Tennant videotape #2).

Figure 4: Delayed shedding of the winter coat on a beef cow.
The videotapes presented multiple cattle with delayed shedding of winter coats. This is a clinical sign often associated with endophyte toxicity as well as nutritional deficiencies. (photo was taken from Tennant videotape #2).

Figure 5: Alopecia and erythema above the coronary band (coronitis).
This lesion was present in several cattle in the videotapes. It is a common sign of endophyte toxicity and is generally referred to as "fescue foot". (photo was taken from Tennant videotape #2).

Figure 6: Alopecia and erythema above the coronary band (coronitis).
This example of "fescue foot" is similar to that presented in figure 5. (photo was taken from Tennant videotape #2).

Figure 7: Three cattle standing in the creek.
A unusual predilection for standing in water was described for the Tennant cattle in the summer. This behavior is known to be associated with endophyte toxicity as it provides some relief for both coronitis ("fescue foot") and hyperthermia ("summer fescue"). (photo was taken from Tennant videotape #2).

Figure 8: Two cattle standing in a puddle.
As described for figure 7, standing in water provides some clinical relief to cattle with coronitis. Although non-specific, a predilection for this behavior is associated with "fescue foot". (photo was taken from Tennant videotape #2).

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Figure 1

Figure 2

Figure 3

Figure 4

Figure 5

Figure 6



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Figure 7

Figure 8

# 11.0 TABLES

Table 1: Review of Tennant Farm Videotapes

Table 2: Individual Animal Data: Clinical Signs

Table 3: Individual Animal Data: Hematology (erythron, platelets)

Table 4: Individual Animal Data: Hematology (leukon), and Special Chemistry

Table 5: Individual Animal Data: Clinical Chemistry

Table 6: Individual Animal Data: Serology and Fecal Exam

Table 7: Tennant Farm Grain and Hay: Nutritional Analysis

# Table 1: Review of Tennant Farm Videotapes

## Tape #1 (cases 1 – 18): Tennant Farm: New England, Wood Co., WVa - January & February, 1997

| # | Subject Animal(s) | Presentation | Diagnosis/Comment |
|---|---|---|---|
| 1 | Cow, Hereford | • neck: alopecia, scaling,<br>• described as "humped-up",<br>• (snowing) | • probable lice,<br>• "humped-up" difficult to appreciate on tape. |
| 2 | Cow, Hereford | • corneal opacity,<br>• (snowing) | • corneal scar, probably secondary to pinkeye. |
| 3 | 2 black bulls | • possible discoloration of hair (not clear),<br>• (snowing) | • not clear from tape. |
| 4 | Cow, Hereford | • thin,<br>• described as poor teeth,<br>• (snowing) | • very thin, probably due to decreased food consumption and mastication problem, although teeth not seen on video,<br>• age unknown; cause of possible teeth problem unknown. |
| 5 | Cow, Hereford | • possible hunched back (not clear),<br>• (snowing) | • not clear from tape. |
| 6 | Cow (tan) | • alopecia, tail switch,<br>• (snowing) | • tail hair loss differential diagnosis would include mechanical, lice, fescue toxicosis, and selenium toxicosis. |
| 7 | Cow | • alopecia, tail switch,<br>• (snowing) | • tail hair loss differential diagnosis would include mechanical, lice, fescue toxicosis, and selenium toxicosis. |
| 8 | Calf, black | • dead (dated 2/9/97) in snow,<br>• overgrown hooves,<br>• bilateral lens opacities,<br>• black/brown teeth,<br>• mucous on feces,<br>• necropsy: no other lesions,<br>• stated: had diarrhea | • hooves a little longer than normal, but not unusual,<br>• "cold cataracts" (post mortem),<br>• teeth normal,<br>• fecal mucous normal for stagnant feces in rectum,<br>• necropsy: lack of fat; serous atrophy of fat: emaciation, probably due to starvation. |

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## Table 1 (continued): Review of Tennant Farm Videotapes

### Tape #1 (cases 1 – 18): Tennant Farm: New England, Wood Co., WVa - January & February, 1997

| # | Subject Animal(s) | Presentation | Diagnosis/Comment |
|---|---|---|---|
| 9 | Calf, black/white face | • neck: alopecia,<br>• diarrhea,<br>• lens or corneal opacities | • probable lice,<br>• cause of diarrhea unknown,<br>• probable corneal scar (hard to tell). |
| 10 | Cow, Hereford | • thin,<br>• corneal opacity,<br>• above hooves: alopecia and hyperemia,<br>• diarrhea | • corneal opacity probably secondary to pinkeye,<br>• differential diagnosis of hoof lesions include mechanical dermatitis, fescue toxicosis, and moist dermatitis due to unknown<br>• cause of diarrhea and thinning unknown, |
| 11 | Cow, black | • neck: alopecia | • probable lice. |
| 12 | Cow, red | • neck: alopecia | • probable lice. |
| 13 | Cow, red | • slobbering, losing cud (on ground),<br>• urinating,<br>• tail switch alopecia | • slobbering/urinating differential diagnosis includes hardware and cholinesterase inhibition (less likely due to individual cow affected),<br>• alopecia differential diagnosis includes mechanical, lice, and fescue or selenium toxicosis. |
| 14 | Cow, red | • neck: alopecia,<br>• tail switch: short hair | • neck: probable lice,<br>• tail hair differential diagnosis includes mechanical, lice, and fescue or selenium toxicosis. |
| 15 | Cow, red | • neck: alopecia | • probable lice. |
| 16 | Cow, red | • odd chewing behavior | • not clear from tape. |
| 17 | Cow, Hereford | • possible hunched back | • not clear from tape; differential diagnosis includes hardware disease (traumatic reticulopericarditis). |

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Table 1 (continued): Review of Tennant Farm Videotapes

Tape #1 (cases 1 – 18): Tennant Farm: New England, Wood Co., WVa - January & February, 1997

| # | Subject Animal(s) | Presentation | Diagnosis/Comment |
|---|---|---|---|
| 18 | Cow, red (same as #13) | • dead in barn,<br>• corneal opacity,<br>• necropsy: no other lesions | • (is this the 9-year-old cow submitted to Michigan?),<br>• corneal opacity may be postmortem artifact,<br>• necropsy: serous atrophy of fat,<br>• noted: normal postmortem changes include moderate to severe autolysis, pseudomelanosis of intestines, interlobular emphysema (agonal),<br>• also noted: normal front teeth<br>• large gallbladder suggests period of anorexia,<br>• cause of death unknown. |
| | | | |



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## Table 1 (continued): Review of Tennant Farm Videotapes

### Tape #2 (cases 19 – 60): Dry Run Harris PC. Wood Co. - Off North Fork of Lee Creek, New England, WVA

| # | Subject Animal(s) | Presentation | Diagnosis/Comments |
|---|---|---|---|
| 19 | Calves, multiple | • corneal opacities of varying severity; some with exudate and/or blepharospasm,<br>• fly problem (summer),<br>• some with poorly shedded and/or light-colored coats | • fly problem with secondary pinkeye,<br>• hair coat shedding and coloring probably due to a nutritional problem. |
| 20 | Fish (sucker?) | • dead | • incidental death: no diagnosis possible. |
| 21 | Small mammal (raccoon?) | • skeleton | • incidental death: no diagnosis possible. |
| 22 | Cow | • corneal opacity | • corneal scar secondary to pinkeye. |
| 23 | Calf | • corneal opacities,<br>• possible head tilt (not clear) | • corneal scar secondary to pinkeye,<br>• head tilt not clear on tape. |
| 24 | Calf | • corneal opacities,<br>• flies | • corneal scar secondary to pinkeye. |
| 25 | Calf | • dead (~1 month-old),<br>• corneal opacity,<br>• (burning carcass) | • cause of death unknown,<br>• corneal scar secondary to pinkeye. |
| 26 | Snake | • dead | • incidental death: no diagnosis possible. |
| 27 | Calf | • corneal opacities,<br>• fly problem | • fly problem with secondary pinkeye. |
| 28 | Fish | • dead | • incidental death: no diagnosis possible. |
| 29 | Calf | • corneal opacity | • corneal scar secondary to pinkeye. |
| 30 | Calf | • corneal opacity | • corneal scar secondary to pinkeye. |

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## Table 1 (continued): Review of Tennant Farm Videotapes

### Tape #2 (cases 19 – 60): Dry Run Harris PC. Wood Co. - Off North Fork of Lee Creek, New England, WVA

| # | Subject Animal(s) | Presentation | Diagnosis/Comments |
|---|---|---|---|
| 31 | Cow | • dead,<br>• worn incisors,<br>• diarrhea,<br>• emaciated, sunken eyes (dehydrated?), serous atrophy of fat (heart),<br>• agonal lung emphysema | • cause of diarrhea, emaciation and death unknown,<br>• worn incisors may be age or feed related,<br>• lung emphysema was agonal and insignificant . |
| 32 | Crow | • dead | • incidental death: no diagnosis possible. |
| 33 | Crow | • dead | • incidental death: no diagnosis possible. |
| 34 | Calves, multiple | • corneal opacity | • corneal scar secondary to pinkeye. |
| 35 | Cow | • corneal opacity,<br>• thin | • corneal scar secondary to pinkeye,<br>• diagnosis of the cause of thin condition not possible. |
| 36 | Fish | • dying | • incidental death: no diagnosis possible. |
| 37 | Crayfish | • dead | • incidental death: no diagnosis possible. |
| 38 | Salamander | • dead | • incidental death: no diagnosis possible. |
| 39 | Toad | • dead | • incidental death: no diagnosis possible. |
| 40 | Newborn calf | • contracted tendons,<br>• big hocks | • difficult to evaluate from tape; may be congenital contracted tendons. |
| 41 | Deer | • dead | • incidental death: no diagnosis possible. |
| 42 | Cow | • dead (rotten) | • no diagnosis possible. |
| 43 | Deer | • rotten carcass | • incidental death: no diagnosis possible. |
| 44 | Deer | • bones | • incidental death: no diagnosis possible. |



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## Table 1 (continued): Review of Tennant Farm Videotapes

### Tape #2 (cases 19 – 60): Dry Run Harris PC. Wood Co. - Off North Fork of Lee Creek, New England, WVA

| # | Subject Animal(s) | Presentation | Diagnosis/Comments |
|---|---|---|---|
| 45 | Cow | • panting; increased respiration | • differental diagnosis includes hyperthermia due to summer fescue toxicosis,<br>• diagnosis uncertain. |
| 46 | Cow | • diarrhea | • cause of diarrhea unknown. |
| 47 | Raccoon | • skull | • incidental death: no diagnosis possible. |
| 48 | Deer | • skeleton | • incidental death: no diagnosis possible. |
| 49 | Deer | • dead (fresh),<br>• bloody nose | • death possibly due to epizootic hemorrhagic disease of deer (EHD). |
| 50 | Rabbit | • dead | • incidental death: no diagnosis possible. |
| 51 | Turkey | • dead | • incidental death: no diagnosis possible. |
| 52 | Hawk | • dead | • incidental death: no diagnosis possible. |
| 53 | Fish | • dead | • incidental death: no diagnosis possible. |
| 54 | Cow | • lumpy udder | • unclear from videotape. |
| 55 | Cows, multiple | • above hooves: alopecia, possibly erythema,<br>• overgrown hooves | • differential diagnosis includes mechanical dermatitis, fescue toxicosis, and moist dermatitis due to unknown,<br>• hoof length not significant. |
| 56 | Bull, cows, multiple | • corneal opacities,<br>• alopecia and erythema above hooves | • corneal scar secondary to pinkeye,<br>• hoof differential diagnosis includes mechanical dermatitis, fescue toxicosis, and moist dermatitis due to unknown. |
| 57 | Cow | • lumpy udder | • unclear from videotape. |

## Table 1 (continued): Review of Tennant Farm Videotapes

### Tape #2 (cases 19 – 60): Dry Run Harris PC. Wood Co. - Off North Fork of Lee Creek, New England, WVA

| # | Subject Animal(s) | Presentation | Diagnosis/Comments |
|---|---|---|---|
| 58 | Cows, multiple | • above hooves: alopecia, possibly erythema.<br>• poor shedding of coat in some animals | • hoof differential diagnosis includes mechanical dermatitis, fescue toxicosis, and moist dermatitis due to unknown,<br>• poor hair coat shedding suggests nutritional problem or fescue mycotoxicosis. |
| 59 | Cow (tan) | • unusual gait | • lame due to unknown cause. |
| 60 | Cows, heifers, multiple | • thin.<br>• delayed shedding | • definitive diagnosis not possible from tape – probably a nutritional deficiency. |
| | | | |

Note: This table excludes many of the clinical signs and pathological conditions that were proposed by the videotapes' narrator but were considered by the cattle team to be inaccurate. For example, the team did not agree with the narrator's assertion that blackened teeth or patchy melanosis were abnormal. Also numerous speculative comments regarding normal organs, during the dissections shown in the videotapes, were not included in this table.



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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 2: Clinical Signs*

| Animal Number | Age | Preg. Status | Description | Body Condition Score (1 - 10) | | Clinical Signs | Photo # |
|---|---|---|---|---|---|---|---|
| | years | | | Girth (in.) | BCS (1-10) | | |
| 1 | >9 | 4 m | yellow | | 3 | possible lice | 1 |
| 2 | >9 | open | red, white face | - | 3 | lice, corneal scar (r) | 2 |
| 3 | >9 | open | yellow, white face | - | 3 | (large uterus) | 3 |
| 4 | >9 | 7 m | red, white face | 70 | 4 | lice | 4 |
| 5 | >9 | 6 m | red, white face | 75 | 4 | corneal scar (l) | 5 |
| 6 | >9 | 3 m | red, white face | - | 4 | - | 6 |
| 7 | >9 | 6 m | red, white face | 71 | 4 | hair loss (switch) | 7 |
| 8 | >9 | open | red, white face | - | 4 | | 8 |
| 9 | >9 | open | red, white face | 70 | 4 | (muzzle: melanin considered normal) | 9 |
| 10 | >9 | term | red, white face | 70 | 3 | - | 10 |
| 11 | >9 | 8.5 m | red, white face | - | 3 | - | 11 |
| 12 | >9 | term | red, white face | 72 | 3 | light coat color; small corneal scar (r) | 12 |
| 13 | >9 | open | red, white face | 66 | 2 | slight corneal scar (l), | 13 |
| 14 | >9 | calf | red, white face | 78 | 4 | alopecia (brisket) | 14 |
| 15 | >9 | open | yellow | 71 | 2 | palpation difficult (fat necrosis?) | 15 |
| 16 | 6 | open | black | 68 | 3 | - | 16 |
| 17 | >9 | 4 m | red, white face | 78 | 7 | - | 17 |
| 18 | >9 | 4 m | red, white face | 78 | 7 | mammary gland lumps (right front)(abscesses?) | 18, 53 |
| 19 | >9 | Calf | red, brockleface | 68 | 3 | slight corneal scars (l and r) | 19 |
| 20 | >9 | 6 m | red, white face | 74 | 3 | - | 20 |
| 21 | >9 | 5 m | red, white face | 69 | 3 | small corneal scar (l)(5 mm) | 21 |
| 22 | >9 | 5 m | red, brockleface | 76 | 4 | mass (12 cm) prescap. neck (r) - lanced: brown serum + hair | 22, 49-51 |
| 23 | - | bull | black | - | 4 | - | 23 |
| 24 | >9 | 6 m | red, white face | 78 | 4 | hair loss (neck) | 24 |

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 2: Clinical Signs (continued)*

| Animal Number | Age | Preg. Status | Description | Body Condition Score (1 - 10) | | Clinical Signs | Photo # |
|---|---|---|---|---|---|---|---|
| | years | | | | | | |
| | | | | Girth (in.) | BCS (1-10) | | |
| 25 | 4 | 5 m | black | 72 | 4 | corneal scar (l) | 25, 44 |
| 26 | 6 | bull | black | - | 5 | corneal opacity (l) | 26, 45 |
| 27 | >9 | 8 m | red, brockleface | 64 | 3 | - | 27 |
| 28 | 7 | 8 m | black, white face | 74 | 4 | - | 28 |
| 29 | >9 | term | red, brockleface | 77 | 2 | mammary gland lumps (left rear) (abscesses?) | 29 |
| 30 | >9 | 8 m | red, white face | 72 | 3 | lice, hair loss (abd.), extra teat, corneal opacity (l=3mm; r=5mm) | 30, 46 |
| 31 | 8 | 6 m | red, white face | 77 | 5 | corneal scar (r) (2 mm); (tongue: melanin considered normal) | 31, 52 |
| 32 | >9 | 6 m | black, white face | 70 | 3 | lice | 32 |
| 33 | >9 | 8 m | red, white face | 72 | 3 | - | 33 |
| 34 | >9 | 8 m | red, brockleface | 73 | 2 | - | 34 |
| 35 | >9 | 5 m | red, white face | 72 | 3 | - | 35 |
| 36 | >9 | 8 m | red, white face | 75 | 3 | corneal scar (l), severe | 36 |
| 37 | 7 | calf | red, brockleface | 77 | 4 | corneal scar (r) (1 cm) | 37, 47 |
| 38 | >9 | 7 m | red, white face | 73 | 4 | - | 38 |
| 39 | >9 | calf | red, white face | 68 | 2 | - | 39 |
| 40 | 4 | 6 m | black | 75 | 4 | light hair color, corneal opacity (l) (2mm) | 40, 48 |
| 41 | 4 | steer | black, white face | 71 | - | lame (lr), corneal opacity (r) severe; phthysical eye (l) | 41 |
| 1 cow | - | - | red, whiteface | - | - | cow escaped run before being tagged or examined | 42 |
| calves | < 6 m | - | 12 misc. calves | - | - | 5 bigger calves not tagged; 7 smaller calves tagged | - |

Cattle examinations conducted on April 7, 1999. Age determined by teeth examination (Dr. Moisan); Pregnancy Status determined by rectal palpation (Dr. Munson); Girth measured by tape (Dr. Habecker); BCS assigned by Drs. Moisan and Munson; Clinical Signs identified by cattle team (Drs. Davis-Heller, Habecker, Moisan, Munson, Poppenga, Sykes); Photos taken by Dr. Sykes.

Cow #22: Gross diagnosis (no histology) of skin lesion: epidermal inclusion cyst (incidental lesion of no significance).

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 3: Hematology (Erythron, Platelets)*

| Animal Number | Age | Preg. Status | RBC | Hgb | Hct | MCV | MCH | MCHC | RDW | PLT | MPV | PCT | PDW | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | years | | $x10^6$/ul | g/dl | % | fl | pg | g/dl | % | $x10^3$/ul | fl | | % | |
| | | Reference Range: | 5-10 | 8-15 | 24-46 | 40-60 | | | | | | | | |
| 1 | 4 | 4 m | 5.85 | 9.91 | 28.8 | 49.3 | 16.9 | 34.4 | 18.3 | 378 | 7.04 | .266 | 15.9 | |
| 2 | 3 | open | 4.68 | 8.82 | 26.0 | 55.6 | 18.8 | 33.8 | 19.5 | 186 | 9.10 | .170 | 19.0 | |
| 3 | >9 | open | 6.22 | 12.0 | 34.8 | 55.9 | 19.3 | 34.6 | 17.8 | 233 | 8.50 | .198 | 18.9 | |
| 4 | >9 | 7 m | 5.81 | 10.4 | 29.7 | 51.1 | 17.9 | 35.1 | 19.3 | 259 | 8.08 | .209 | 18.4 | |
| 5 | >9 | 6 m | 5.86 | 9.87 | 29.2 | 49.8 | 16.9 | 33.8 | 18.6 | 142 | 8.82 | .126 | 19.7 | |
| 6 | >9 | 3 m | 5.21 | 10.1 | 28.8 | 55.2 | 19.4 | 35.1 | 19.3 | 201 | 8.98 | .180 | 18.7 | |
| 7 | >9 | 6 m | 5.06 | 10.6 | 29.3 | 58.0 | 21.0 | 36.1 | 17.8 | 378 | 6.49 | .246 | 16.9 | |
| 8 | >9 | open | 4.44 | 9.58 | 26.6 | 59.9 | 21.6 | 36.0 | 19.2 | 448 | 6.42 | .287 | 17.2 | |
| 9 | >9 | open | 4.48 | 8.66 | 25.2 | 56.3 | 19.3 | 34.3 | 20.2 | 184 | 13.1 | .240 | 23.4 | |
| 10 | >9 | term | 5.23 | 10.5 | 29.7 | 56.9 | 20.1 | 35.3 | 17.1 | 369 | 8.02 | .296 | 18.1 | |
| 11 | >9 | 8.5 m | 4.81 | 9.84 | 28.2 | 58.6 | 20.4 | 34.9 | 17.8 | 142 | - | - | - | |
| 12 | >9 | term | 4.99 | 9.41 | 27.9 | 55.9 | 18.9 | 33.7 | 19.0 | 113 | 10.9 | .123 | 21.8 | |
| 13 | >9 | open | 4.29 | 8.62 | 23.7 | 55.2 | 20.1 | 36.4 | 20.4 | 595 | 6.73 | .400 | 17.7 | |
| 14 | >9 | calf | 6.04 | 12.0 | 33.7 | 55.8 | 19.9 | 35.7 | 18.8 | 361 | 8.91 | .321 | 19.2 | |
| 15 | >9 | pregn. | 4.65 | 8.41 | 24.9 | 53.6 | 18.1 | 33.7 | 20.6 | 238 | 7.73 | .184 | 19.2 | |
| 16 | 6 | open | 5.47 | 9.58 | 28.2 | 51.5 | 17.5 | 34.0 | 20.0 | 284 | 8.27 | .235 | 18.3 | |
| 17 | >9 | 4 m | 6.16 | 13.1 | 37.7 | 61.1 | 21.2 | 34.7 | 19.2 | 87.4 | - | - | - | |
| 18 | >9 | 4 m | 6.30 | 12.5 | 35.2 | 55.9 | 19.9 | 35.6 | 19.4 | 368 | 7.71 | .283 | 17.2 | |
| 19 | >9 | calf | 4.92 | 9.98 | 29.1 | 59.2 | 20.3 | 34.3 | 17.3 | 317 | 5.75 | .182 | 16.1 | |
| 20 | >9 | 6 m | 4.85 | 9.51 | 28.0 | 57.7 | 19.6 | 34.0 | 18.0 | 275 | 7.55 | .207 | 18.6 | |
| 21 | >9 | 5 m | 5.80 | 10.7 | 30.9 | 53.2 | 18.5 | 34.8 | 19.0 | 320 | 8.30 | .266 | 19.4 | |
| 22 | >9 | 5 m | 5.55 | 10.5 | 30.3 | 54.7 | 18.9 | 34.6 | 19.1 | 391 | 7.24 | .283 | 18.2 | |
| 23 | - | bull | 6.13 | 12.4 | 34.8 | 56.7 | 20.3 | 35.7 | 19.0 | 283 | 8.74 | .248 | 20.9 | |
| 24 | >9 | 6 m | 6.02 | 11.8 | 33.5 | 55.7 | 19.6 | 35.2 | 21.8 | 270 | 8.23 | .222 | 17.9 | |

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 3: Hematology (Erythron, Platelets) (continued)*

| Animal Number | Age | Preg. Status | RBC | Hgb | Het | MCV | MCH | MCHC | RDW | PLT | MPV | PCT | PDW | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | years | | x10⁶/ul | g/dl | % | fl | pg | g/dl | % | x10³/ul | fl | | % | |
| | | Reference Range: | 5-10 | 8-15 | 24-46 | 40-60 | | | | | | | | |
| 25 | 4 | 5 m | 6.93 | 12.5 | 35.9 | 51.8 | 18.0 | 34.8 | 18.8 | 231 | 8.33 | .193 | 18.1 | |
| 26 | 6 | bull | 6.13 | 11.4 | 33.1 | 54.0 | 18.5 | 34.3 | 18.4 | 278 | 7.71 | .215 | 18.2 | |
| 27 | >9 | 8 m | 4.90 | 9.88 | 28.3 | 57.8 | 20.1 | 34.9 | 18.3 | 406 | 7.05 | .286 | 17.8 | |
| 28 | 7 | 8 m | 6.12 | 11.7 | 34.1 | 55.7 | 19.2 | 34.4 | 17.8 | 180 | 7.18 | .129 | 17.6 | |
| 29 | >9 | term | 5.37 | 9.93 | 28.7 | 53.4 | 18.5 | 34.6 | 19.2 | 220 | 9.57 | .211 | 24.4 | |
| 30 | >9 | 8 m | 5.08 | 10.2 | 28.6 | 56.3 | 20.0 | 35.6 | 20.1 | 407 | 7.09 | .288 | 16.9 | |
| 31 | 8 | 6 m | 6.03 | 12.6 | 36.0 | 59.7 | 20.9 | 35.1 | 19.5 | 217 | 8.07 | .175 | 18.2 | |
| 32 | >9 | 6 m | 6.17 | 11.3 | 32.4 | 52.5 | 18.2 | 34.7 | 19.2 | 138 | 7.33 | .101 | 19.5 | |
| 33 | >9 | 8 m | 5.42 | 10.1 | 30.1 | 55.5 | 18.7 | 33.6 | 17.8 | 186 | 7.77 | .145 | 20.2 | |
| 34 | >9 | 8 m | 5.41 | 11.6 | 33.0 | 60.9 | 21.4 | 35.1 | 18.2 | 361 | 7.20 | .260 | 17.8 | |
| 35 | >9 | 5 m | 5.86 | 11.1 | 32.2 | 55.0 | 19.0 | 34.6 | 21.2 | 246 | 6.33 | .156 | 16.8 | |
| 36 | >9 | 8 m | 4.34 | 9.44 | 26.3 | 60.6 | 21.7 | 35.9 | 18.1 | 315 | 8.00 | .252 | 18.0 | |
| 37 | 7 | calf | 5.48 | 10.9 | 30.9 | 56.4 | 19.8 | 35.1 | 18.8 | 363 | 8.56 | .310 | 19.5 | |
| 38 | >9 | 7 m | 5.88 | 11.5 | 32.2 | 54.8 | 19.6 | 35.7 | 18.3 | 397 | 5.82 | .231 | 16.7 | |
| 39 | >9 | calf | 4.68 | 8.34 | 24.5 | 52.4 | 17.8 | 34.1 | 18.5 | 345 | 6.28 | .217 | 16.4 | |
| 40 | 4 | 6 m | 6.94 | 12.4 | 34.4 | 49.6 | 17.9 | 36.1 | 19.1 | 449 | 6.65 | .298 | 17.4 | |
| 41 | 4 | steer | 6.78 | 12.2 | 34.7 | 51.1 | 18.0 | 35.2 | 20.0 | 298 | 6.94 | .207 | 17.6 | |

Blood collected by jugular venipuncture during examination while cattle restrained in a head-hold device. Blood refrigerated for approximately 36 hours before hematology was run on an Abbott Cell-Dyne 3500.
Reference values are for cattle (New Bolton Center), but not from the Cell-Dyne 3500.

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 4: Hematology (leukon), Special Chemistry, and Fecal Exam*

| Animal Number | Age | Preg. Status | WBC | Neut | Lymph | Mono | Eos | Baso | Prolactin | Cu | Se-AA | Peps | Fecal Exam |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | years | | x10³/ul | % | % | % | % | % | plasma ng/ml | serum ppm | blood ppm | serum odu | |
| | Reference Range: | | 4-12 | 15-45 | 45-75 | 2-7 | 2-20 | 0-2 | 171.6 (mean) | 0.6-1.20 | > 0.080 | 0.0-0.3 | |
| 1 | 4 | 4 m | 8.74 | 35.9 | 28.7 | 7.48 | 27.9 | .016 | 58.70 | .282 | .121 | - | t; s (f) |
| 2 | 3 | open | 8.46 | 32.0 | 44.2 | 9.36 | 14.4 | .037 | 137.60 | .305 | .091 | .276 | - |
| 3 | >9 | open | 7.10 | 63.6 | 5.39 | 10.0 | 21.0 | .024 | 124.68 | .518 | .120 | - | neg. |
| 4 | >9 | 7 m | 6.32 | 42.5 | 28.9 | 7.39 | 21.1 | .109 | 197.19 | .593 | .121 | - | - |
| 5 | >9 | 6 m | 6.69 | 31.6 | 29.4 | 7.41 | 31.6 | 0.00 | 48.34 | .693 | .091 | - | c(r); t |
| 6 | >9 | 3 m | 8.48 | 35.6 | 25.1 | 7.07 | 32.3 | 0.00 | 37.01 | .667 | .110 | - | - |
| 7 | >9 | 6 m | 6.80 | 58.9 | 6.15 | 7.42 | 27.5 | .029 | 51.61 | .473 | .138 | - | - |
| 8 | >9 | open | 7.38 | 53.7 | 16.3 | .207 | 29.8 | 0.00 | 40.21 | .633 | .105 | - | - |
| 9 | >9 | open | 4.81 | 43.9 | 33.6 | 9.93 | 12.6 | .054 | 113.08 | .518 | .105 | - | - |
| 10 | >9 | term | 6.54 | 62.2 | 15.3 | 10.5 | 11.8 | .113 | 562.10 | .525 | .105 | - | - |
| 11 | >9 | 8.5 m | 6.25 | 34.8 | 35.8 | 9.72 | 19.6 | .061 | 106.50 | .344 | .102 | - | - |
| 12 | >9 | term | 6.13 | 45.7 | 33.0 | 7.75 | 13.5 | .037 | 124.43 | .462 | .104 | - | - |
| 13 | >9 | open | 5.37 | 61.4 | 18.0 | 10.0 | 10.4 | .151 | 38.18 | .586 | .100 | .304 | s (f) |
| 14 | >9 | calf | 5.21 | 31.2 | 41.9 | 8.25 | 18.5 | .137 | 54.40 | .549 | .128 | - | - |
| 15 | >9 | pregn. | 4.29 | 56.5 | 25.0 | 12.1 | 5.89 | .428 | 141.36 | .244 | .115 | .302 | s (f) |
| 16 | 6 | open | 7.38 | 49.7 | 33.7 | 10.7 | 5.76 | .240 | 32.04 | .487 | .101 | - | s (f) |
| 17 | >9 | 4 m | 6.83 | 44.8 | 27.6 | 10.2 | 17.5 | 0.00 | 92.87 | .838 | .156 | .334 | s (f) |
| 18 | >9 | 4 m | 8.44 | 45.1 | 23.7 | 5.23 | 26.0 | 0.00 | 142.25 | .773 | .174 | .390 | - |
| 19 | >9 | calf | 5.28 | 57.2 | 19.4 | 11.3 | 11.9 | .058 | 42.28 | .414 | .091 | - | - |
| 20 | >9 | 6 m | 3.59 | 53.4 | 19.2 | 8.19 | 19.2 | 0.00 | 33.35 | .205 | .134 | - | c(r);s(f) |
| 21 | >9 | 5 m | 7.90 | 32.0 | 22.6 | 4.68 | 40.7 | 0.00 | 47.37 | .623 | .135 | - | - |
| 22 | >9 | 5 m | 5.58 | 44.6 | 14.3 | 12.1 | 28.9 | .167 | 62.95 | .415 | .151 | - | - |
| 23 | - | bull | 6.02 | 59.1 | 21.2 | 7.31 | 12.4 | 0.00 | 89.60 | .437 | .121 | - | c(r);s(m) |
| 24 | >9 | 6 m | 5.84 | 42.5 | 25.5 | 10.6 | 21.4 | .028 | 87.94 | .649 | .135 | - | - |

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 4: Hematology (leukon), Special Chemistry, and Fecal Exam (continued)*

| Animal Number | Age | Preg. Status | WBC | Neut | Lymph | Mono | Eos | Baso | Prolactin | Cu | Se-AA | Peps | Fecal exam |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | years | | x10³/ul | % | % | % | % | % | plasma ng/ml | serum ppm | blood ppm | serum odu | |
| | | Reference Range: | 4-12 | 15-45 | 45-75 | 2-7 | 2-20 | 0-2 | 171.6 (105.8) | 0.6-1.20 | > 0.080 | 0.0-0.3 | |
| 25 | 4 | 5 m | 8.98 | 54.8 | 6.61 | 7.76 | 30.8 | .026 | 47.76 | .643 | .161 | - | - |
| 26 | 6 | bull | 6.09 | 64.6 | 17.4 | 6.87 | 10.9 | .168 | 68.95 | .778 | .142 | .184 | s (m) |
| 27 | >9 | 8 m | 5.69 | 56.4 | 16.2 | 8.79 | 18.4 | .160 | 122.27 | .443 | .116 | - | - |
| 28 | 7 | 8 m | 5.92 | 69.9 | 19.1 | .525 | 10.5 | 0.00 | 86.90 | .396 | .114 | - | - |
| 29 | >9 | term | 5.49 | 53.5 | 13.7 | 7.72 | 25.1 | 0.00 | 106.37 | <0.20 | .146 | .224 | neg. |
| 30 | >9 | 8 m | 3.58 | 64.8 | 14.5 | 5.79 | 15.0 | 0.00 | 44.53 | .289 | .114 | - | - |
| 31 | 8 | 6 m | 5.41 | 59.2 | 18.2 | 3.61 | 19.0 | 0.00 | 157.00 | .683 | .139 | .098 | - |
| 32 | >9 | 6 m | 6.86 | 59.0 | 19.8 | 3.36 | 17.8 | 0.00 | 361.97 | .488 | .132 | - | neg. |
| 33 | >9 | 8 m | 6.76 | 70.4 | 4.69 | 6.72 | 18.2 | .026 | 64.19 | .533 | .105 | - | - |
| 34 | >9 | 8 m | 5.19 | 57.6 | 19.5 | 9.81 | 13.0 | .056 | 109.60 | .434 | .161 | .324 | - |
| 35 | >9 | 5 m | 5.40 | 38.0 | 24.0 | 12.8 | 25.1 | .055 | 47.45 | .679 | .140 | - | - |
| 36 | >9 | 8 m | 4.39 | 57.1 | 17.5 | 13.5 | 11.9 | 0.00 | 306.10 | .596 | .101 | - | s (f) |
| 37 | 7 | calf | 5.89 | 54.0 | 14.4 | 10.2 | 21.3 | .090 | 43.89 | .732 | .142 | - | - |
| 38 | >9 | 7 m | 6.49 | 53.8 | 10.7 | 9.62 | 25.9 | .022 | 143.13 | .681 | .124 | - | - |
| 39 | >9 | calf | 4.72 | 35.5 | 35.5 | 7.47 | 21.5 | 0.00 | 48.70 | .762 | .109 | qns | - |
| 40 | 4 | 6 m | 9.05 | 26.7 | 29.7 | 12.6 | 30.9 | .055 | 207.43 | .607 | .146 | - | s (f) |
| 41 | 4 | steer | 9.57 | 52.3 | 31.3 | 2.73 | 13.7 | 0.00 | 80.12 | .582 | .143 | .304 | - |

Blood collected by jugular venipuncture during examination while cattle restrained in a head-hold device. Blood refrigerated for approximately 36 hours before hematology was run on an Abbott Cell-Dyne 3500. WBC (WIC values) considered valid; WBC differential (neutrophils, lymphocytes, monocytes, eosinophils, basophils) considered invalid due to 36-hour storage of blood.

Hematology reference values are for cattle (New Bolton Center), but not from the Cell-Dyne 3500.

Selenium values of 0.050 ppm are considered marginal; below 0.050 ppm are selenium deficient.

Prolactin (heparin tubes)(Dr. N. Schrick, Univ. of Tenn., Dept. An. Sci.). Ref. means: April non-fescue (171.6) and ergotamine tartrate (105.8 ng/ml).

Cu = copper; Se-AA = selenium. (New Bolton Center)

Peps = serum pepsinogen; odu = optical density units (Texas Veterinary Diagnostic Laboratory, College Station, TX)

Fecal exam: c = coccidia oocysts; s = strongyle eggs; t = Monezia tapeworm eggs; (r) = rare; (f) = few; (m) = moderate. (New Bolton Center).

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 5: Clinical Chemistry*

| Animal Number | Age | Preg. Status | BUN | Creat | Na+ | K+ | Cl- | Ca | Mg | Phos | Tot Prot | Alb | Glob | AST | CK | GGT |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | years | | mg/dl | mg/dl | mmol/l | mmol/l | mmol/l | mg/dl | mg/dl | mg/dl | g/dl | g/dl | g/dl | U/L | U/L | U/L |
| | | Reference Range: | 6 - 22 | 0.5 - 1.1 | 134 - 144 | 4.0 - 5.7 | 96 - 104 | 8.2 - 10 | 2.0 - 2.8 | 4.7 - 9.0 | 5.8 - 7.5 | 2.4 - 3.5 | 3.0 - 3.5 | 58 - 100 | | 22 - 64 |
| 1 | 4 | 4 m | 14.7 | 1.68 | 145 | 5.14 | 102 | 9.8 | 2.3 | 5.73 | 7.2 | 2.79 | 4.41 | 126 | 89 | 40 |
| 2 | 3 | open | 10.5 | 1.23 | 143 | 4.95 | 106 | 8.75 | 1.9 | 7.02 | 7.6 | 2.70 | 4.90 | 98 | 77 | 36 |
| 3 | >9 | open | 11.5 | 2.00 | 144 | 6.00 | 105 | 9.42 | 2.2 | 5.80 | 7.8 | 3.20 | 4.60 | 138 | 110 | 32 |
| 4 | >9 | 7 m | 10.8 | 1.59 | 144 | 4.78 | 106 | 9.21 | 2.1 | 7.24 | 7.3 | 2.92 | 4.38 | 84 | 57 | 29 |
| 5 | >9 | 6 m | 7.9 | 1.82 | 146 | 5.06 | 107 | 7.71 | 2.0 | 9.31 | 7.8 | 2.96 | 4.84 | 98 | 71 | 45 |
| 6 | >9 | 3 m | 8.1 | 1.73 | 145 | 4.55 | 107 | 9.87 | 2.2 | 5.09 | 7.6 | 3.17 | 4.43 | 126 | 181 | 37 |
| 7 | >9 | 6 m | 13.5 | 2.30 | 143 | 6.27 | 102 | 9.00 | 1.7 | 6.66 | 7.6 | 3.15 | 4.45 | 109 | 155 | 30 |
| 8 | >9 | open | 10.0 | 1.47 | 145 | 6.13 | 106 | 9.58 | 1.9 | 5.30 | 8.0 | 3.03 | 4.97 | 141 | 187 | 49 |
| 9 | >9 | open | 8.5 | 1.49 | 145 | 5.60 | 104 | 9.39 | 2.1 | 5.35 | 7.6 | 2.85 | 4.75 | 111 | 83 | 34 |
| 10 | >9 | term | 17.0 | 2.15 | 150 | 4.91 | 109 | 9.93 | 2.2 | 6.11 | 7.0 | 3.04 | 3.96 | 99 | 49 | 35 |
| 11 | >9 | 8.5 m | 11.6 | 1.86 | 149 | 4.81 | 111 | 10.24 | 2.3 | 5.02 | 7.1 | 2.86 | 4.24 | 101 | 105 | 27 |
| 12 | >9 | term | 21.0 | 1.62 | 146 | 4.64 | 109 | 9.56 | 1.7 | 4.76 | 7.6 | 2.86 | 4.74 | 97 | 76 | 37 |
| 13 | >9 | open | 15.9 | 1.06 | 145 | 5.52 | 105 | 9.10 | 1.8 | 5.20 | 7.8 | 2.74 | 5.06 | 141 | 97 | 42 |
| 14 | >9 | calf | 19.6 | 1.72 | 146 | 5.55 | 103 | 9.83 | 1.8 | 7.35 | 7.9 | 3.30 | 4.60 | 127 | 86 | 35 |
| 15 | >9 | pregn. | 21.2 | 1.83 | 144 | 5.02 | 104 | 8.42 | 2.0 | 6.37 | 7.0 | 2.46 | 4.54 | 106 | 52 | 34 |
| 16 | 6 | open | 17.8 | 1.78 | 140 | 5.44 | 101 | 9.44 | 2.1 | 5.49 | 6.8 | 2.82 | 3.98 | 96 | 85 | 30 |
| 17 | >9 | 4 m | 12.0 | 1.87 | 151 | 6.46 | 109 | 10.29 | 2.1 | 5.31 | 8.3 | 3.35 | 4.95 | 109 | 87 | 31 |
| 18 | >9 | 4 m | 8.2 | 1.73 | 144 | 4.85 | 102 | 9.52 | 2.0 | 6.76 | 7.9 | 3.42 | 4.48 | 141 | 298 | 39 |
| 19 | >9 | calf | 14.3 | 1.69 | 147 | 4.72 | 105 | 9.34 | 2.0 | 5.07 | 7.1 | 3.01 | 4.09 | 126 | 79 | 28 |
| 20 | >9 | 6 m | 11.3 | 2.09 | 146 | 5.36 | 107 | 10.32 | 2.1 | 5.95 | 8.1 | 3.07 | 5.03 | 113 | 92 | 35 |
| 21 | >9 | 5 m | 17.4 | 1.47 | 146 | 4.77 | 105 | 10.25 | 2.3 | 6.07 | 7.9 | 3.06 | 4.84 | 103 | 96 | 30 |
| 22 | >9 | 5 m | 15.6 | 2.09 | 145 | 5.42 | 108 | 10.40 | 2.2 | 4.93 | 7.8 | 3.25 | 4.55 | 101 | 90 | 34 |
| 23 | - | bull | 8.5 | 2.55 | 143 | 4.55 | 102 | 9.42 | 2.2 | 5.55 | 7.6 | 3.08 | 4.52 | 109 | 97 | 33 |
| 24 | >9 | 6 m | 9.6 | 1.98 | 148 | 4.67 | 110 | 8.19 | 2.1 | 7.03 | 7.5 | 3.03 | 4.47 | 109 | 116 | 37 |

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 5: Clinical Chemistry (continued)*

| Animal Number | Age | Preg. Status | BUN | Creat | Na+ | K+ | Cl- | Ca | Mg | Phos | Tot Prot | Alb | Glob | AST | CK | GGT |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | years | | mg/dl | mg/dl | mmol/l | mmol/l | mmol/l | mg/dl | mg/dl | mg/dl | g/dl | g/dl | g/dl | U/L | U/L | U/L |
| | | Reference Range: | 6 - 22 | 0.5 - 1.1 | 134 - 144 | 4.0 - 5.7 | 96 - 104 | 8.2 - 10 | 2.0 - 2.8 | 4.7 - 9.0 | 5.8 - 7.5 | 2.4 - 3.5 | 3.0 - 3.5 | 58 - 100 | | 22 - 64 |
| 25 | 4 | 5 m | 10.0 | 1.99 | 149 | 4.93 | 108 | 9.90 | 2.4 | 7.12 | 7.7 | 3.34 | 4.36 | 122 | 138 | 29 |
| 26 | 6 | bull | 7.6 | 2.08 | 147 | 4.69 | 107 | 9.72 | 2.2 | 5.31 | 7.7 | 3.13 | 4.57 | 139 | 150 | 54 |
| 27 | >9 | 8 m | 14.3 | 1.72 | 149 | 4.97 | 113 | 10.18 | 1.8 | 3.67 | 7.4 | 2.94 | 4.46 | 138 | 133 | 39 |
| 28 | 7 | 8 m | 14.7 | 2.07 | 147 | 5.44 | 110 | 9.83 | 2.4 | 5.04 | 7.6 | 3.20 | 4.40 | 102 | 123 | 33 |
| 29 | >9 | term | 12.9 | 2.16 | 146 | 5.26 | 109 | 10.19 | 2.2 | 5.35 | 7.3 | 2.83 | 4.47 | 98 | 50 | 37 |
| 30 | >9 | 8 m | 14.1 | 1.64 | 143 | 5.42 | 103 | 8.80 | 2.3 | 6.77 | 7.5 | 2.99 | 4.51 | 132 | 90 | 35 |
| 31 | 8 | 6 m | 11.3 | 1.66 | 148 | 4.55 | 109 | 10.33 | 2.2 | 5.24 | 7.3 | 3.25 | 4.05 | 111 | 99 | 29 |
| 32 | >9 | 6 m | 18.8 | 1.72 | 143 | 4.36 | 106 | 9.80 | 2.2 | 4.72 | 7.3 | 2.98 | 4.32 | 89 | 126 | 43 |
| 33 | >9 | 8 m | 13.0 | 1.95 | 152 | 4.50 | 112 | 9.61 | 1.4 | 7.14 | 7.9 | 3.14 | 4.76 | 150 | 137 | 38 |
| 34 | >9 | 8 m | 19.6 | 2.56 | 149 | 6.27 | 110 | 10.79 | 2.5 | 6.75 | 7.9 | 3.52 | 4.38 | 139 | 99 | 35 |
| 35 | >9 | 5 m | 7.9 | 1.81 | 148 | 5.77 | 107 | 9.82 | 2.3 | 5.30 | 8.0 | 3.27 | 4.73 | 120 | 106 | 30 |
| 36 | >9 | 8 m | 15.1 | 2.09 | 147 | 5.56 | 110 | 9.50 | 2.3 | 5.43 | 7.7 | 3.05 | 4.65 | 112 | 92 | 29 |
| 37 | 7 | calf | 9.8 | 1.92 | 146 | 5.55 | 106 | 9.43 | 2.0 | 4.12 | 8.2 | 3.43 | 4.77 | 119 | 94 | 36 |
| 38 | >9 | 7 m | 13.0 | 1.97 | 149 | 4.35 | 112 | 9.65 | 2.1 | 5.34 | 8.2 | 3.47 | 4.73 | 206 | 173 | 39 |
| 39 | >9 | calf | 6.4 | 2.03 | 145 | 4.90 | 102 | 9.44 | 1.9 | 5.31 | 8.1 | 3.01 | 5.09 | 135 | 79 | 33 |
| 40 | 4 | 6 m | 15.0 | 1.94 | 147 | 5.05 | 108 | 10.20 | 2.2 | 6.28 | 7.6 | 3.12 | 4.48 | 147 | 79 | 37 |
| 41 | 4 | steer | 8.3 | 1.72 | 143 | 6.37 | 106 | 9.95 | 2.3 | 6.99 | 7.4 | 3.10 | 4.30 | 110 | 192 | 26 |

Blood collected by jugular venipuncture during examination while cattle restrained in a head-hold device. Blood refrigerated for approximately 36 hours before serum chemistry was run on a Kodak Ektachem.

Cattle were coralled for several hours on a warm day, without water, before blood samples were collected.

Reference values are for cattle (New Bolton Center, Kodak Ektachem), except for globulin (Duncan and Prasse).

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 6: Serology*

| Animal Number | Age (yrs) | Preg. Status | BLV | Johne's | Brucella | BVD | BVD | Blue Tongue | Lepto | EHD (type 2) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | serum test: | elisa | elisa | sn | sn | microplate | AGID | sn | AGID | | |
| serum samples | | Reference Range: | | | | | | | | | | |
| 1 | 4 | 4 m | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 2 | 3 | open | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 3 | >9 | open | neg | neg | neg | neg | neg | neg | neg | - | | |
| 4 | >9 | 7 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 5 | >9 | 6 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 6 | >9 | 3 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 7 | >9 | 6 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 8 | >9 | open | neg | neg | neg | neg | neg | neg | neg | - | | |
| 9 | >9 | open | neg | neg | neg | neg | neg | neg | neg | - | | |
| 10 | >9 | term | neg | neg | neg | neg | neg | neg | neg | - | | |
| 11 | >9 | 8.5 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 12 | >9 | term | neg | neg | neg | neg | neg | neg | neg | - | | |
| 13 | >9 | open | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 14 | >9 | calf | neg | neg | neg | neg | neg | neg | neg | - | | |
| 15 | >9 | pregn. | neg | neg | neg | 1:512 | neg | neg | neg | >1:80 | | |
| 16 | 6 | open | neg | neg | neg | neg | neg | neg | neg | - | | |
| 17 | >9 | 4 m | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 18 | >9 | 4 m | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 19 | >9 | calf | neg | neg | neg | neg | neg | neg | neg | - | | |
| 20 | >9 | 6 m | neg | neg | neg | neg | neg | neg | 1:100 | - | | |
| 21 | >9 | 5 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 22 | >9 | 5 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 23 | - | bull | neg | neg | neg | neg | neg | neg | neg | - | | |
| 24 | >9 | 6 m | neg | neg | neg | neg | neg | neg | neg | - | | |

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## Individual Animal Data: Cattle from Tennant Farm (Washington, West Virginia)

*Table 6: Serology (continued)*

| Animal Number | Age (yrs) | Preg. Status | BLV | Johne's | Brucella | BVD | BVD | Blue Tongue | Lepto | EHD (type 2) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | serum test: | | elisa | elisa | sn | sn | microplate | AGID | sn | AGID | | |
| serum samples | Reference Range: | | | | | | | | | | | |
| 25 | 4 | 5 m | neg | (neg)[1] | neg | neg | neg | neg | neg | - | | |
| 26 | 6 | bull | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 27 | >9 | 8 m | POS | neg | neg | 1:256 | neg | neg | neg | - | | |
| 28 | 7 | 8 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 29 | >9 | term | neg | neg | neg | neg | neg | neg | 1:400 | >1:80 | | |
| 30 | >9 | 8 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 31 | 8 | 6 m | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 32 | >9 | 6 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 33 | >9 | 8 m | neg | neg | neg | neg | neg | neg | 1:100 | - | | |
| 34 | >9 | 8 m | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 35 | >9 | 5 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 36 | >9 | 8 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 37 | 7 | calf | neg | neg | neg | neg | neg | neg | neg | - | | |
| 38 | >9 | 7 m | neg | neg | neg | neg | neg | neg | neg | - | | |
| 39 | >9 | calf | neg | neg | neg | neg | neg | neg | neg | neg | | |
| 40 | 4 | 6 m | neg | POS | neg | neg | neg | neg | neg | - | | |
| 41 | 4 | steer | neg | neg | neg | neg | neg | neg | neg | neg | | |

BLV = bovine leukemia virus antibody

Johne's = *Mycobacterium paratuberculosis* antibody (technique has a 1% false positive error rate)

Brucella = *Brucella abortus* antibody

Lepto = antibody to *Leptospira spp.* (*L pomona, L. icterohemorrhagiae, L hardjo, L. grippotyphosa, L canicola*).

BLV, Johne's, Brucella, and Lepto assays conducted by the Animal Diagnostic Laboratory, Penn. State Univ., University Park, PA.

EHD = 2 of 12 tested cattle were "positive" for EHD-type 2 (Alberta strain); (Texas Veterinary Diagnostic Laboratory, College Station, TX).

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## Tennant Farm (Washington, West Virginia): Grain and Hay Analysis

*Table 7: Nutritional Analysis*

| Analyte | units | Hay | | Grain | |
|---|---|---|---|---|---|
| | | as sampled basis | dry matter basis | as sampled basis | dry matter basis |
| Dry Matter | % | 86.34 | - | 89.66 | - |
| Crude Protein | % | 6.77 | 7.84 | 10.21 | 11.39 |
| Unavailable Protein | % | 1.90 | 2.20 | - | - |
| Adj. Crude Protein | % | 5.55 | 6.42 | - | - |
| Acid Detergent Fiber | % | 40.44 | 46.84 | 7.59 | 8.46 |
| TDN | % | 47.67 | 55.21 | 69.35 | 77.35 |
| NE (Lactation) | Mcal./lb | 0.33 | 0.38 | 0.72 | 0.81 |
| Calcium | ppm | 0.40 | 0.46 | 0.95 | 1.07 |
| Phosphorus | ppm | 0.10 | 0.12 | 0.30 | 0.33 |
| Sodium | % | 0.02 | 0.02 | 0.37 | 0.48 |
| Magnesium | % | 0.13 | 0.15 | 0.30 | 0.34 |
| Sulfur | % | 0.18 | 0.21 | 0.13 | 0.14 |
| Potassium | % | 0.87 | 1.01 | 0.60 | 0.67 |
| Copper | ppm | 5 | 6 | 6 | 7 |
| Iron | ppm | 206 | 239 | 305 | 340 |
| Manganese | ppm | 331 | 384 | 22 | 24 |
| Zinc | ppm | 43 | 50 | 39 | 43 |
| Nitrate Ion | % | 0.00 | 0.00 | | |
| Selenium | ppm | | 0.12 | | 0.19 |

Hay and grain feed analysis performed by NC Dept. Agric., Food and Drug Protection Division, Forage Testing Laboratory, 4000 Reedy Creek Rd, Raleigh, NC 27607.

Hay and grain samples collected by cattle team during April 1999 site visit to Tennant Farm. Hay sample consisted of 3 core samples each from 3 large round bales of 1998 cut hay.

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# 12.0 APPENDICES

Appendix A: Abbreviated Curriculum Vitae of Cattle Team Members

Appendix B: Diagnostic Pathology Reports

Ohio Department of Agriculture: #4977-97

Michigan Animal Health Diagnostic Laboratory: #1792571

University of Pennsylvania Laboratory of Large Animal Pathology and Toxicology: #UP9902702, #9901437

Appendix C: Dry Run Safety Plan

Appendix D: Figures 1-42 : Photographs of the 42 adult cattle;

Figures 43-66 : Miscellaneous photographs of the Tennant Farm and cattle

Appendix E: Herd Health History (April 8, 1999 interview)

Appendix F: Diet Analysis: Computer Software Modelling



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# Appendix A: Abbreviated *Curriculum Vitae* of the Cattle Team Members

| Name: | Dr. Perry L. Habecker |
|---|---|
| Degrees, Certifications: | VMD, Diplomate ACVP |
| Employment: | Chief, Large Animal Pathology, Laboratory of Pathology and Toxicology, Univ. of Penn. School of Veterinary Medicine, New Bolton Center, Kennett Square, PA. |
| Education: | Juniata College (BS, 1972-1976)<br>University of Pennsylvania, School of Veterinary Medicine (VMD 1969-1973)<br>University of Pennsylvania, School of Veterinary Medicine (resident, 1989-1992) |
| Affiliations: | American College of Veterinary Pathologists<br>Pennsylvania Veterinary Medical Association<br>American Association of Veterinary Laboratory Diagnosticians |

| Name: | Dr. Lisa Davis-Heller |
|---|---|
| Degrees, Certifications: | DVM |
| Employment: | Private Practitioner, St. Mary's Veterinary Clinic, St. Mary's, WV. |
| Education: | Ohio State University<br>Ohio State University, College of Veterinary Medicine (DVM, 1979-1983) |
| Affiliations: | American Veterinary Medical Association<br>Ohio Veterinary Medical Association<br>West Virginia Veterinary Medical Association<br>International Veterinary Acupuncture Society<br>Holistic Veterinary Society |

| Name: | Dr. Peter G. Moisan |
|---|---|
| Degrees, Certifications: | DVM, Diplomate ACVP., Diplomate ABVP (food animal specialist: beef cattle specialist) |
| Employment: | Veterinary Pathologist and Field Investigator, Rollins Animal Disease Diagnostic Laboratory, North Carolina Department of Agriculture, Raleigh, NC. |
| Education: | Cleveland State Community College (AS, 1974)<br>East Tennessee State University (BS, Microbiology, 1975)<br>East Tennessee State University (MS, Microbiology, 1978)<br>University of Tennessee, College of Veterinary Medicine (DVM, 1981)<br>Michigan State University (Resident, Veterinary Pathology, 1995)<br>Kansas State University (Research, Veterinary Pathology, 1995-1998 |
| Affiliations: | American Board of Veterinary Practitioners<br>American College of Veterinary Pathologists<br>American Association of Bovine Practitioners<br>American Association of Swine Practitioners<br>Academy of Veterinary Consultants<br>American Veterinary Medical Association<br>American Association of Veterinary Laboratory Diagnosticians |

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bcc: Andrew Hartten
Rob Pinchot
Sharron Laas
J.H.Little
H.D.Ramsey
D.D.Jackson
J.M.Migliore
R.Banerjee
R.J.Zipfel
A.J.Playtis

*ANDREA V. MALINOWSKI, ESQ.*
**Senior Counsel**
DuPont Legal, Room D-7078
1007 Market Street
Wilmington, DE 19898
Phone: (302) 774-6443
Fax: (302) 774-4812

January 12, 2000

**VIA FAX (cover letter only) [413-448-6553]**
**VIA OVERNIGHT MAIL (with attachments)**

Douglas Johns, Esquire
Legal
General Electric Plastics
One Plastics Avenue
Pittsfield, MA 01201

Dear Mr. Johns:

This letter is further to our conversation of last week and more specifically in reply to items 1-3 of Mr. Dale Van De Velde's January 5, 2000 e-mail to Bob Ritchey of DuPont concerning FC-143 (also referred to as C-8 or ammonium perfluorooctanoate).

**Item 1: TSCA 8(e) Decision**

Regarding item 1, DuPont did not submit a TSCA 8(e) notification to EPA concerning the presence of FC-143 in environmental media. The 8(e)-reportability of the presence of FC-143 in environmental media was reviewed within DuPont and determined to not be reportable. The reasons for that decision are as follows:

- The toxicology of FC-143 has been reported to EPA by both DuPont and 3M in numerous TSCA 8(e) submissions. I faxed to you last week a copy of the latest TSCA 8(e) submission, dated November 19, 1999 and made by 3M. Other submissions were made both before and during the TSCA 8(e) Compliance Audit Program.

- The discharge of FC-143 into the Ohio River has been reported to EPA Region III and the West Virginia Division of Water Resources (WVDWR). For example, in a June 9, 1981 letter (attached Document 1) to the WVDWR (copy to EPA Region III), it is stated that FC-143 is present in outfall 005 (permit WV0001279) in a concentration of about 0.1 mg/L. Outfall 005 discharges into the Ohio River. Note it



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was later determined that the defects observed in the study referenced in this letter were not caused by FC-143.

- The presence of FC-143 in the aquifer underlying the Washington Works site has been reported to EPA Region III and the WVDWR. For example, attached Document 2 is an excerpt from Washington Works' Solid Waste Management Unit Survey submission made in 1985 to EPA Region III (copy to the WVDWR). In section B-4, paragraph entitled "Releases, Spills, etc.", it is stated that FC-143 has been detected in the aquifer at ppb levels.

- The presence of FC-143 in the Lubeck public supply wells in ppb levels has been reported to EPA Region III and the West Virginia Department of Natural Resources (WVDNR). For example, attached Document 3 is an excerpt from Washington Works' Verification Investigation Work Plan (VI) report submitted in 1990 to EPA Region III (copy to WVDNR and the West Virginia Pollution Control Division). On page numbered 18 of the VI, it is stated that the Lubeck public supply wells have detectable levels (ppb) of ammonium perfluorooctanoate (also called C-8).

Based upon the above correspondences, it was determined that the EPA Administrator had knowledge of the presence of FC-143 in various environmental media, both at the plant site and outside the boundary of the plant site.

In addition, as you know, EPA guidance on the criteria for environmental TSCA 8(e) reporting is vague, uncertain, and currently (for the past several years) being rewritten. In its last Notice of Clarification on TSCA 8(e) reporting criteria (58 FedReg 37735; July 13, 1993), EPA stated that

> "With regard to non-emergency environmental contamination information, EPA interprets section 8(e) to require reporting of information that provides evidence of widespread environmental distribution of a chemical substance or mixture, and which because of the extent, pattern, and amount of the contamination seriously threatens or may seriously threaten: (1) Humans with cancer, birth defects, mutation, death or serious or prolonged incapacitation ... or (2) non-human organisms with large-scale or ecologically significant population destruction. Thus, the mere presence of a chemical substance in an environmental media, absent some other relevant information as noted above, would not trigger reporting under section 8(e)."

At the levels FC-143 is present in environmental media, DuPont concludes that FC-143 does not pose the threat or potential threat described above.

In making the decision on 8(e) reportability, DuPont recognized that the guidance on 8(e) environmental reporting was in flux and it was decided that this decision would be reviewed again once EPA issues the new environmental guidance.

EID089384

**Items 2 and 3: Correspondences to the Town and Test Data on Town Wells**

We have located the following correspondences about FC-143 to the town (Lubeck) and test data on the town wells:

- June 13, 1989 letter (attached Document 4) to the Lubeck Public Service Division (LPSD) in which it is stated on page 2 of the cover letter that FC-143 was detected in LPSD water taps in concentrations ranging from 1.0 to 2.2 ppb. We are not aware of any written response to this letter from the LPSD. Note that DuPont subsequently purchased these wells and that the LPSD now draws its drinking water from different wells, samples of which have also been tested by DuPont (see next bullet).

- March 23, 1992 letter (attached Document 5) to the LPSD providing results of analyses performed on samples taken from three of the new LPSD wells. Results ranged from 0.09JX ppb to 0.4 ppb. Note that JX means estimate. We are not aware of any written response to this letter from the LPSD.

**Follow-up**

If you need any additional information or further clarification, please do not hesitate to contact me. If GE should decide to submit an 8(e) notification, we would appreciate receiving a copy of it.

Very truly yours,

Andrea V. Malinowski

Attachments

cc: Steve Austin [Fax: 413-448-6590]
GE Plastics
One Plastics Avenue
Pittsfield, Mass 01201

Dale Van De Velde [Fax: 304-863-7128]
Manager, Environmental Programs
General Electric Company, Plastics Mfg. Div.
P.O. Box 68
Washington, WV 26181

Robert Ritchey, DuPont Washington Works
Bernard Reilly, DuPont Legal, Wilmington, DE

EID089385

Katherine E. Reed, Ph.D.
Executive Director

3M Environmental Technology
and Safety Services

900 Bush Avenue
Building 42-2E-26
PO Box 33331
St Paul, MN 55133-3331
651 778 4331

8EHQ-1199-14596

Doubles to CDI

3M

November 19, 1999

MR 24451

CERTIFIED MAIL

RECEIVED
OPPT CBIC
1999 NOV 23 AM 11:24

Document Processing Center (7407)
(Attn: Section 8(e) Coordinator)
Office of Toxic Substances
US EPA
401 M Street, SW
Washington, DC 20460

Re: TSCA 8(e) SUBSTANTIAL RISK NOTICE ON:
Ammonium Perfluorooctanoate CAS# 3825-26-1

Dear Sir:

3M has recently completed a review of the draft report on a 6-month primate feeding study performed with ammonium perfluorooctanoate (PFOA) by Covance Laboratories, Madison, Wisconsin. Male cynomologus monkeys were given daily doses by intragastric capsule deposition of 0 (n=6), 3 (n=4), 10 (n=6) and 30 (n=6) mg/kg ammonium perfluorooctanoate. Significant illness occurred in the 30 mg/kg dose group within several weeks. Dosing was stopped while the animals recovered. Dosing was resumed at 20 mg/kg. At the end of the six month dosing period, two animals in the control, mid and high dose groups were allowed to recover for 90 days. One high dose animal was sacrificed in moribund condition on day 29. This animal suffered hepatic lesions which are felt to be compound-related. Other high dose animals also exhibited liver toxicity.

The following findings are being reported by 3M under TSCA 8(e):

- Significant increased liver-to-body weight ratios were observed in all dose groups. There was no corresponding adverse pathology in the two lowest dose groups (3 mg/kg/day, 10 mg/kg/day) at terminal sacrifice. Liver weight increases were reversed after a 90 day recovery period.

- Preliminary analytical results indicate the serum levels of PFOA for these two lower dose groups are similar to those found among the highest PFOA serum levels observed in a small number of 3M Cottage Grove chemical production employees. Workplace medical surveillance activities have been conducted at 3M since the 1970's. Employees' fluorochemical levels, whether measured as total organic fluorine or as PFOA, have not been associated with clinical hepatic toxicity.

99 DEC -8 AM 9:11
RECEIVED
OPPT NCIC

EID102775

AVM000209

8EHQ-99-14596

88000000036

- In addition, one low dose (3 mg/kg/day) test animal was sacrificed in moribund condition on study day 139. An independent pathology review of this case did not reveal a specific cause of morbidity. The effects observed in this animal were not consistent with the significant hepatoxicity observed at the high dose and may not be compound-related. There were no effects, other than increased liver weight, in the remaining three animals tested at the low dose.

This chemical is produced by 3M and is used by industrial customers as a process aid. 3M has and will continue to conduct medical monitoring of its chemical production workers and to reduce potential for worker exposure.

A copy of the final study report will be sent to the EPA when received.

Please contact Dr. William Weppner, 651-733-6374, for further information.

Sincerely,

Katherine E. Reed

Dear Gerry,

As discussed on April 13 by phone, EPA needs to review complete copies of final reports and any other relevant information in your possession or control on carboxylated and sulfonated perfluorochemicals, with initial focus on PFOA (perfluoro octanoic acid). The requested information includes health effects studies (including pharmacokinetics), environmental effects studies, environmental fate studies and information, and human and environmental exposure studies and information, including monitoring. If final reports are not available, we request that you clarify the status of any ongoing studies with regard to projected completion date and provide us with the latest and most complete version of any preliminary reports in your possession or control.

We request that you provide us with the above information on PFOA by May 26, 2000. Regarding the sulfonated perfluorochemicals, our initial interest is focused on perfluorooctyl sulfonic acid (PFOS) and we request that you provide the above information on PFOS by April 28, 2000.

If you have any of the above information on other carboxylated or sulfonated perfluorochemicals, we request that, by April 28, 2000, you initially identify the chemicals to allow us to confirm our interest, and then provide the requested information in your possession or control on other sulfonated perfluorochemicals by May 19, 2000 and on other carboxylated perfluorochemicals by a week later, May 26, 2000. The information on these additional chemicals can most usefully be provided in chemical specific increments as they are completed, with completion of the full submissions by the dates requested.

A few points of clarification regarding this request:

>duPont need not resubmit copies of studies already provided to EPA (e.g., under TSCA §8(e), as FYIs, under FIFRA) although we do request that you provide a comprehensive bibliography of the studies already submitted so we can check this against our holdings.

>copies of acute systemic toxicity studies can be limited to the key or critical studies as described in the Data Adequacy and robust summary guidance developed for the HPV Challenge Program. Under this approach all studies will be cited in a bibliographic listing although full copies can be limited to studies which (1) serve to identify lowest (i.e., most toxic) lethal dose values by different routes or (2) report observed effects which differ from those seen in studies (using the same route) submitted under (1).

>skin and eye irritation studies can be handled in a manner consistent with that outlined for acute systemic toxicity (i.e., submit copies of the key or critical studies which serve to set out the irritation potential of the chemical and other irritation studies which show effects which differ from those reported in submitted studies).

>the initial request is generally limited to studies conducted during or after 1976. For studies conducted prior to 1976, we ask for a bibliography listing all studies and for full copies of studies in the following endpoint areas: genetic toxicity; subchronic toxicity; reproductive toxicity; developmental toxicity; immunotoxicity; uptake/metabolism; chronic toxicity/carcinogenicity; neurotoxicity.

>regarding exposure information, we are most interested in receiving information and data of the types found in the Use and Exposure Information Profile (UEIP; the UEIP has been used to support SIDS assessments in the US) and information on results of exposure or monitoring studies (human and environmental), including full copies of studies where available and including details on procedures and methods used for sampling and analysis. We are also interested in studies relating to the ultimate environmental fate and distribution of these chemicals.

In order to maintain the integrity of your submissions, please submit all information on a given chemical as a separate "For Your Information" (FYI) submission. You may also submit the information as supplements to any prior submissions by your company under TSCA §8(e) relating to these chemicals. As always, any CBI must be submitted in accordance with applicable procedures. If you have any questions with regard to the submission procedures, or you wish to work out the arrangements in advance, please contact Terry O?Bryan (202-260-3483).

If you have any general questions regarding this request, please contact me by email or phone (202-260-3749). I thank you in advance for your cooperation.

Charles M. Auer, Director
Chemical Control Division
Office of Pollution Prevention and Toxics/OPPTS/EPA

AJP001522

EID080128

000630

H David Ramsey
05/19/2000 12:42 PM

To: John L Granquist/AE/DuPont@DuPont, Robert L Ritchey/CL/DuPont@DuPont, Lynwood K Ireland/CL/DuPont@DuPont, John E Auger/AE/DuPont@DuPont, John M Migliore/AE/DuPont@DuPont, Robbin Banerjee/AE/DuPont@DuPont, Dawn D Jackson/CL/DuPont@DuPont, Anthony J Playtis/CL/DuPont@DuPont, Roger J Zipfel/AE/DuPont@DuPont, Oscar T Garza/AE/DuPont@DuPont
cc:
Subject: EPA Side of PFOS Story - NY Times

---------- Forwarded by H David Ramsey/AE/DuPont on 05/19/2000 12:41 PM ----------

From: Bernard J Reilly on 05/19/2000 06:32 AM

To: Robert F Pinchot/DEV/AE/DuPont@DuPont, Michael E McCord/AE/DuPont@DuPont, Richard J Angiullo/AE/DuPont@DuPont, John S Sieg/EUR/DuPont@DuPont, H David Ramsey/AE/DuPont@DuPont, Paul J Bossert/AE/DuPont@DuPont
cc:
Subject: EPA Side of PFOS Story - NY Times

May 19, 2000

E.P.A. Says It Pressed 3M for Action on Scotchgard Chemical

By DAVID BARBOZA
CHICAGO, May 18 -- The Environmental Protection Agency said today that it had pressed Minnesota Mining and Manufacturing to come up with a solution after the company's own tests had shown that a chemical compound used in Scotchgard and an array of household products could pose a risk to human health and the environment.
The E.P.A. account differs from that of 3M, which said on Monday that it had voluntarily decided to stop making the chemical used in Scotchgard and many other products by the end of the year because the tests showed that the chemical compounds failed to decompose in the environment. Officials of 3M say they have no evidence that the chemicals pose a long-term threat to human health.

While the E.P.A. said it did not see an immediate safety risk for consumers using products now on the market, agency officials said that if 3M had not acted they would have taken steps to remove the product from the market. The officials said they grew concerned about potential long-term health risks to humans after a 3M study showed that the chemical, perfluorooctanyl sulfonate, lingered for years in human blood and animal tissue and that high doses were known to kill laboratory rats.

"The results raised a number of concerns," said Stephen Johnson, who works in the office of prevention, pesticides and toxic substances at the E.P.A. "What it suggests to us is that there are potentially long-term consequences. But we don't have evidence it is unsafe now."

Officials of 3M, however, say they are absolutely confident that their products are safe, and that there are no long-term consequences to human health.

"This isn't a health issue now, and it won't be a health issue," said Larry Zobel, the medical director at 3M., which is based in St. Paul.

"To the question of whether this builds up in humans, it would have to be a long time, like hundreds of thousands of years, to be a threat," he said.

Many scientists have praised 3M's decision to stop production of perfluorooctanyl and related chemicals.

"The real issue is this stuff accumulates, and if it accumulates in the ecosystem, you have to be worried about that because it could be trouble," said John Doull, a retired professor of clinical toxicology at the University of Kansas Medical Center in Kansas City. "No chemical is totally innocuous, and it seems inconceivable that anything that accumulates would not eventually become toxic."

In addition to Scotchgard products, the chemical is used as stain repellant and protective coating on textiles, carpets and leather, and in paper packing, snack-food bags, pet- food bags, firefighting foam and pesticides.

The E.P.A. said its decision to press 3M rested on four concerns: the compound is persistent in the environment; it appears in wildlife and human tissue around the world; it appears in human blood samples taken from around the world; and, based on the study of laboratory rats, it has the potential to harm humans.

The E.P.A. said it was first alerted to the study of laboratory rats shortly after it was conducted in 1998. In that study, male and female rats were given doses of the chemical and then mated. When a pregnant rat continued to get regular doses of about 3.2 milligrams per kilogram of body weight, most of the offspring died within four days.

"With all that information, we finally talked to 3M and said that raises a number of concerns. What are you going to do?" said Mr. Johnson at the E.P.A.

There is still a difference of interpretation, however. Officials of 3M said the doses given to the rats were extremely high, but E.P.A. officials said that few other chemicals would have as severe an effect.

"This is fairly toxic stuff in rats," one E.P.A. official said. "There's clear evidence it presents a problem in rats."

But 3M said it had not yet determined the cause of death in the rats nor how humans or animals ingested the chemicals so that it appeared in tissue or blood samples.

"That's a very interesting question," Mr. Zobel said. "We can't say how it gets into anybody's blood."

As a result of that uncertainty, and the persistence of the compound in the environment, 3M said it would do away with the chemistry by the end of the year. The company said it negotiated with the E.P.A. but its decision was voluntary and there was never a discussion of a recall of the products.

3M, which is the only company known to make perfluorooctanyl, has been searching for alternatives. But in recent weeks, after the E.P.A. asked 3M to come up with a solution, the company decided to discontinue using the chemistry behind most of its Scotchgard products, which account for about $300 million in sales, or less than 2 percent of the company's $16 billion in annual revenue.

Since the announcement on Monday, the company's shares have gained $1.125, closing at $87.0625 today.

3M, which also produces Post-it notes and Scotch tape, will stop supplying the chemical to other companies that use it for their products.

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H David Ramsey
05/19/2000 12:46 PM

To: Dawn D Jackson/CL/DuPont@DuPont, Robert L Ritchey/CL/DuPont@DuPont, John M Migliore/AE/DuPont@DuPont, Anthony J Playtis/CL/DuPont@DuPont, Roger J Zipfel/AE/DuPont@DuPont, Oscar T Garza/AE/DuPont@DuPont, Lynwood K Ireland/CL/DuPont@DuPont, John E Auger/AE/DuPont@DuPont
cc:
Subject: EPA and Canada on PFOS and Related Perfluoroalkyls - BNA News Clip

---------- Forwarded by H David Ramsey/AE/DuPont on 05/19/2000 12:45 PM ----------

From: Bernard J Reilly on 05/19/2000 06:48 AM

To: Robert F Pinchot/DEV/AE/DuPont@DuPont, John S Sieg/EUR/DuPont@DuPont, Paul J Bossert/AE/DuPont@DuPont, H David Ramsey/AE/DuPont@DuPont, Andrea V Malinowski/AE/DuPont@DuPont, Jim E Pesek/AE/DuPont@DuPont, Susan S Mileti/DuPont@DuPont, Robert C Buck/DuPont@DuPont, Richard J Angiullo/AE/DuPont@DuPont, Martha L Rees/AE/DuPont@DuPont@DuPont, John R Bowman/AE/DuPont@DuPont@DuPont, Earl W MacFarlane/AE/DuPont@DuPont, James K Higgins/AE/DuPont@DuPont, Erin Mariani/AE/DuPont@DuPont, Gerald L Kennedy/AE/DuPont@DuPont, Marjorie W Doyle/AE/DuPont@DuPont, Isidoros J Zanikos/AE/DuPont@DuPont, Hugh J Campbell_Jr/AE/DuPont@DuPont, Michael S Parr/AE/DuPont@DuPont, Darwin G Wika/AE/DuPont@DuPont
cc:
Subject: EPA and Canada on PFOS and Related Perfluoroalkyls - BNA News Clip

No. 98
Friday, May 19, 2000 Page A-14
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News

Chemical Safety
EPA Has No Regulatory Plans on PFOS;
3M Has Major Research, Testing Under Way

It is too early to know whether regulatory action is needed on a group of chemicals being pulled from the market by 3M, an Environmental Protection Agency official told BNA May 17.
At the same time, the company has vowed to conduct further health and environmental tests to better understand how perfluorooctanyl sulfonate (PFOS) chemicals, which are used in certain stain-resistant Scotchgard products, persist and move through the environment, even though the company said the chemicals posed no health threat.

The St. Paul, Minn.-based company announced plans May 16 to phase out a profitable product line that uses PFOS-based chemicals after sending EPA a substantial risk report and boxes of follow-up information. The Toxic Substances Control Act requires submittal to EPA of information about the adverse effects of industrial chemicals (96 DEN A-1, 5/17/00).

EPA Searches for 'Cousins.'

JMM008163

EID072413

EPA is trying to determine how many discrete PFOS chemicals are being made and how may related "cousin chemicals" are being made, Charles Auer, director of EPA's Chemical Control Division in the Office of Pollution Prevention and Toxics, told BNA May 17. The agency is particularly interested in identifying the polymers that are being made with a similar chemistry, he said, although most polymers are not measured in the TSCA reporting and tracking mechanisms.
Because PFOS is an old product--its production began more than 50 years ago--EPA has not required manufacturers to conduct environmental or health tests on it. However, as one top 3M official said, the company has conducted virtually every toxicity test known on PFOS, and some studies have been done more than once.

EPA expects to identify "tens of substances" in commerce that may need to be reviewed for possible regulatory controls, Auer said.

Auer said that while 3M was the predominant manufacturer of PFOS materials, it was not the only company making or handling the substance in the United States. He said data collected periodically by EPA on chemicals made or imported into the United States indicate that there are other companies that at least have the capability of making the materials. The technology used to make the substance--electrochemical fluorination or ECF--is used in other countries, he said.

Because the technology for making PFOS-related chemicals exists in other countries, U.S. officials will lead a discussion about whether worldwide controls are needed at an upcoming working group meeting of chemical regulators from the Organization for Economic Cooperation and Development, Auer said.

PFOS are not reported to the annual Toxic Release Inventory, he said. However, chemical companies that handle more than 10,000 pounds of the material are required to report production volume and other information to EPA every four years in the "TSCA Inventory Update" of chemicals in commerce, Auer said.

He also said PFOS chemicals are not among the chemicals currently being looked for in any of the federally funded exposure studies that seek to measure and identify levels chemicals and pesticides in human blood and tissue.

Some agency staff have described PFOS as "sailing under the agency regulatory radar screen," Auer said.

"It's too early to know whether a regulation is needed" under TSCA that would require companies to report or control their production, or whether letters to companies encouraging action on their part, such as 3M's voluntary withdrawal from the market, would address this issue, Auer said.

## Company's Responsibility

Top 3M officials interviewed by BNA said the company was undertaking research and testing for the chemical because the company has a responsibility to understand how its products interact with the environment, even after a product's commercial life has ended. Bill Coyne, senior vice president for research and development at 3M, told BNA "the reason we are phasing out this product is that it is not sustainable," even though it has been shown to be safe in dozens of toxicity tests. The fact that minute quantities of this material have been identified in the environment and persist in the environment is reason enough to act, he said.

3M has turned in several boxes of data on this chemical, and more boxes are expected to arrive shortly, an EPA official said. The information is housed in the TSCA public docket in Washington. The 3M materials are identified as HQ-04-00373 in the TSCA Section 8(e) substantial risk and "for your information" reports, the official said. The primary PFOS chemical that has been the subject of most of 3M's testing and analysis is potassium perfluorooctane sulfonate (CAS No. 2795-39-3), an EPA official said.

No Substitutes Yet

Researchers at 3M have said they are three to five years away from finding a substitute for PFOS in most of its Scotchgard products, Coyne said. He noted that a few Scotchgard products will remain in commerce because they are formulated differently.
Coyne said that by the end of 2000, production will be halted at both 3M manufacturing sites--one in Alabama, the other in the Netherlands.

The decision to abandon a $300 million product line without a substitute ready was "not all that hard" once the data showed the presence of PFOS in places where it was not expected, he said.

The company began efforts to reduce releases of the substance when it was discovered in minute quantities in blood from a blood bank two years ago, Katherine Reed, a top 3M scientist, explained.

3M scientists have developed and refined the laboratory methods that were used to detect the chemical, she said. The methodology, which has good laboratory practices associated with it, will be submitted for publication in a scientific journal soon, she said.

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Canada Welcomes 3M Decision

OTTAWA--The Canadian government welcomes the decision by 3M to phase out by the end of 2000 the use of perfluorooctanyl chemistry in some of the company's specialty materials, Environment Minister David Anderson said May 16.
Perfluorooctanyl chemicals were among the commercial chemicals in the perfluoroalkyl (PFA) class of substances identified in 1999 under the Canadian Environmental Protection Act (CEPA) as requiring further review as potential threats to the environment or human health, Anderson said in joint statement with Health Minister Allan Rock.

The Canadian government learned in March 2000 that the U.S. Environmental Protection Agency was also studying that class of chemicals, the statement said. "In the coming weeks, Health Canada and Environment Canada officials will use the authorities in CEPA to identify all manufacturers and importers of the chemicals and gather information on Canadian usage and toxicological information," it said.

By Peter Menyasz

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3M Phasing Out Some of its Specialty Materials

ST. PAUL, Minnesota – May 16, 2000 – 3M today announced it is phasing out of the perfluorooctanyl chemistry used to produce certain repellents and surfactant products.

The affected product lines represent about two percent of 3M's nearly $16 billion in annual sales. These include many Scotchgard products, such as soil, oil and water repellent products; coatings used for oil and grease resistance on paper packaging; fire-fighting foams; and specialty components for other products. 3M said it plans to substantially phase out production by the end of the year and will work with customers to accomplish a smooth transition. "Our decision anticipates increasing attention to the appropriate use and management of persistent materials," said Dr. Charles Reich, executive vice president, Specialty Material Markets. "While this chemistry has been used effectively for more than 40 years and our products are safe, our decision to phase out production is based on our principles of responsible environmental management."

"We're reallocating resources to accelerate innovation in more sustainable opportunities and technologies. This decision is not only in the public interest, it's in the best interests of all our constituencies ... our employees, customers, communities and investors," Reich said.

Sophisticated testing capabilities – some developed in only the last few years – show that this persistent compound, like other materials in the environment, can be detected broadly at extremely low levels in the environment and in people. All existing scientific knowledge indicates that the presence of these materials at these very low levels does not pose a human health or environmental risk. 3M expects to meet consensus earnings estimates for the rest of 2000. This excludes a one-time charge on the order of $200 million, that will be taken sometime this year.

"Our growth engines are more powerful than ever and we're confident in our ability to continue delivering on expectations," said L.D. DeSimone, chairman and CEO. "Many of our new technology platforms directly address the fastest-growing segments of the new economy such as electronics, telecommunications and flat-panel displays." "We expect the positive momentum in our financial performance to continue into 2001 with earnings somewhat above current analyst estimates," DeSimone said.

3M is a leading manufacturer of innovative products for industrial, consumer, trans-portation, safety, health care and other markets, with operations in more than 60 countries worldwide. The company is well known for its "Pollution Prevention Pays" environmental initiative, and its emission reduction programs including water-based replacement of solvents in manufacturing and replacements for ozone-depleting chlorofluorocarbons (CFCs).

Forward-Looking Statements Certain portions of this news release that do not relate to historical financial information constitute forward-looking statements. These forward-looking statements are subject to certain risks and uncertainties. Actual future results and trends may differ materially from historical results or those expected depending on a variety of factors, including: (1) worldwide economic conditions; (2) foreign exchange rates and fluctuations in those rates; (3) the timing and acceptance of new product offerings; (4) raw materials, including shortages and increases in the costs of key raw materials; and (5) legal proceedings.

Scotchgard is a trademark of 3M company.

From:
3M Public Relations
3M Center, Building 225-1S-15
St. Paul, MN 55144-1000
Phone: (651) 733-8805
E-mail: www.3m.com

[3M Home] [News and Profile] [Press Box]

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Legal Guidelines

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DuPont Haskell Laboratory
for Toxicology and Industrial Medicine
Elkton Road, P.O. Box 50
Newark, DE 19714-0050

1) ~~Bernie~~ Copy to Paula Gi
2) AVM and John Bo

DuPont Haskell Laboratory

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May 25, 2000

Mr. Charles M. Auer, Director
U. S. Environmental Protection Agency
Office of Pollution Prevention & Toxics
Chemical Control Division
401 M Street, N.W. – Room 403
Washington, D.C. 20460

Dear Mr. Auer:

As requested, enclosed are the summaries of studies conducted at DuPont Haskell Laboratory with Ammonium Perfluorooctanoate and Perfluorononanoate.

If you have questions, please contact me at (302) 366-5259.

Sincerely,

Gerald L. Kennedy
Director – Applied Toxicology
and Health

GLK:jhh
Enclosures

EID091521

E. I. du Pont de Nemours and Company

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# SUMMARY OF A STUDY CONDUCTED WITH POTASSIUM PERFLUOROALKYL SULFONATE (PFOS) AT DUPONT

| | |
|---|---|
| **Type:** | **Biopersistence Screening Study** |
| Species/Strain: | Rats/Crl:CD®SD(IGS)BR |
| Sex/Number: | Male/5/group |
| Exposure Period: | 5 or 10 days; 94 days of recovery |
| Frequency of Treatment: | *Ad libitum* for 5 or 10 days |
| Exposure Levels: | Ammonium perfluorooctanoate: 20 mg/kg<br>Potassium perfluoroalkyl sulfonate (PFOS): 10 mg/kg |
| Method: | Six groups of rats were administered ammonium perfluorooctanoate, 1 group received the test substance for 5 consecutive days and the other 5 groups received the test substance for 10 days. Approximately 2 hours after the first dose, blood was collected from each rat from the group designated for 5 doses. These same rats were sacrificed on test day 5 approximately 2 hours post-dosing, and blood and livers were collected. The remaining groups received the test substance for 10 days. Five rats were sacrificed and had blood and livers collected on test days 10 (2 hours post-dose), 13, 24, 52, or 93/94. The total fluorine content of the blood was determined by using a Wickbold torch combustion method, followed by analysis with a fluoride ion selective electrode. Body weights and clinical signs were recorded throughout the test. Liver weights were recorded, but not reported.<br><br>Six groups of rats were administered potassium perfluoroalkyl sulfonate as described above. Food and water were available *ad libitum* throughout the test. Corn oil was used as the vehicle for the ammonium perfluorooctanoate test groups.<br><br>Potassium perfluoroalkyl sulfonate was dissolved in acetone before suspending it in corn oil. The ratio of acetone to corn oil was 20:80. Negative controls of corn oil and corn oil/acetone were also tested as described above. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity 93-97%<br><br>Potassium perfluoroalkyl sulfonate, purity 82-86% CAS #2795-39-3, 3-8% CAS #3871-99-6, 3-7% CAS #29420-49-3, 1-3% CAS #60272-25-1 |
| Results: | Rats dosed with ammonium perfluorooctanoate exhibited wet perineum and diarrhea during the dosing period. Alopecia was observed during the recovery period. The normalized μM equivalents (blood organofluoride levels) in rat blood peaked after |

5 days of dosing and then decreased throughout the testing period. The Cmax was 518.12 ± 44.89 ppm with a terminal half-life of 8.3 days. The AI (Accumulation Index) was 12.5 and the BI (Bioaccumulation) was 6497.5. An area under the curve (estimated to infinity) (AUCINF/D) was calculated and normalized for fluorine content. The AUCINF/D was calculated as 70,789.6.

Rats dosed with potassium perfluoroalkyl sulfonate exhibited diarrhea, salivation, alopecia, black ocular discharge, and staining of various parts of the body during the dosing period. Alopecia was observed during the recovery period. The normalized μM equivalents (blood organofluoride levels) in rat blood continued to rise throughout the dosing period and may not have reached steady-state. The Cmax was 989.85 ± 116.90 ppm with a terminal half-life of 40.5 days. The AI was 59.0 and the BI was 58382.2. The AUCINF/D was calculated as 566,479.1.

Reference: DuPont Co. (2000). Unpublished Data, Haskell Laboratory Report No. 2922.

## SUMMARY OF STUDIES CONDUCTED WITH AMMONIUM PERFLUOROOCTANOATE AT DUPONT

### Ecotoxicity

| | |
|---|---|
| **Type:** | **Biodegradation** |
| Value: | 13% at Day 28 |
| Breakdown products: | $CO_2$ |
| Method: | Modified Strum Test (OECD 301 B). In this test, biodegradability was measured as $CO_2$ evolution. Activated sludge was used as the inoculum. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity assumed 100% |
| Results: | Ammonium perfluorooctanoate (30% solution) did not demonstrate "Ready Biodegradability." Note that failure to pass a "Ready Biodegradability Test" does not imply that the test substance will persist in the environment for an extended period of time. |

The toxicity test, which included both the test substance and the reference chemical (sodium benzoate) in the same flask, yielded < 25% biodegradation within 14 days; therefore, ammonium perfluorooctanoate was considered inhibitory to microorganisms in the inoculum.

EID091523



000822

Reference: DuPont Co. (1997). Unpublished Data, AEM Laboratory Report No. 24-97.

**Type:** **Adsorption-Desorption Screening Studies**

Method: Ammonium perfluorooctanoate adsorption/desorption screening studies were performed on several test materials, including two clays, a washed sand, peat moss, and an agricultural soil from Delaware, USA. The adsorption/desorption screening studies were also performed using two surface soils and two sediments from the Ohio River collected in the vicinity of the DuPont Washington Works plant near Parkersburg, West Virginia.

The adsorption/desorption procedure used in this study was adapted from a method developed by the Office of Prevention, Pesticides and Toxic Substances (OPPTS), United States Environmental Protection Agency for use in the testing of pesticides and toxic substances, and the development of test data (US EPA (1998). Fate, transport and transformation test guidelines: OPPTS 835.1220 sediment and soil adsorption/desorption isotherm, United State Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, EPA712-C-98-048).

Except for the clays, all samples were screened through a 2-mm (10 mesh) stainless steel sieve in preparation for the absorption/desorption screening test. Furthermore, the sand was washed using DI (deionized) water through 2-mm and 50-µm stainless steel sieves. Material retained on the 50 µm sieve was used in the study. The sand, soils, and sediments used in the studies were air-dried prior to use. Data calculations, however, were based on oven-dry weights of the samples. Oven-dry weights were determined after drying a subsample of material in an oven at 105°C for a minimum of 24 hours.

The pH of the samples was determined in a ratio of 1:1 (test substance to water). Percent organic matter was determined using the loss on ignition method.

Screening Test: Adsorption Studies

The adsorption test was performed in duplicate on each material. A blank containing 0.01M $CaCl_2$ solution with test materials and no ammonium perfluorooctanoate and a single control at each ammonium perfluorooctanoate concentration but no material were also included. Each material was equilibrated with the aqueous phase of a solution of the test substance at 50, 500, and 5000 µg/L

prepared in 0.01M $CaCl_2$

Sterile 50 mL polypropylene centrifuge tubes were used as the test vessels. One part air-dried material (4.0 g) was weighed and 5 parts test solution (20 mL) were decanted into centrifuge tubes, except the control tube. The centrifuge tubes were secured on an end-over-end mixer and agitated at approximately 30 rpm for 24 hours. Samples were centrifuged for 20 minutes at 4000 x g, and then filtered through a 0.2 µm nylon syringe filter into a new centrifuge tube. The volume of aqueous supernatant was measured and refrigerated at approximately 5°C until analyzed for the parent compound using an LC-MS. The blank and the control tubes were subjected to the same steps as the test systems, including filtering.

Screening Test: Desorption Studies

A fresh volume of 20 mL of 0.01M $CaCl_2$ solution without the test substance was added to each solid phase (pellet). The sample was mixed, centrifuged, filtered, stored, and analyzed as was done in the adsorption studies. This desorption step was repeated a second time, resulting in two washings that were analyzed separately.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity not applicable

Results: Five of 9 materials tested for the adsorption/desorption characteristics of ammonium perfluorooctanoate (peat moss, Upstream sediment, East Wood soil, Garden Area soil, and Kaolin clay) exhibited significant adsorption (> 25% of ammonium perfluorooctanoate added) at one or more of the added concentrations of ammonium perfluorooctanoate. Sand, Montmorillonite clay, Downstream sediment, and Sassafras soil did not adsorb a significant amount of ammonium perfluorooctanoate at any concentration.

Only peat moss, however, adsorbed a significant amount at all three concentrations of added ammonium perfluorooctanoate. Kaolin clay adsorbed a significant amount of ammonium perfluorooctanoate at 500 and 5000 µg/L. The Garden Area soil collected at Washington Works also adsorbed significant amounts of ammonium perfluorooctanoate at 500 µg/L. Upstream sediments from the Ohio River and East Wood soil adsorbed a significant amount of ammonium perfluorooctanoate at 50 µg/L only.

Once adsorbed on peat moss, most of the ammonium perfluorooctanoate (> 76%) did not desorb after two washings with a solution of 0.01M $CaCl_2$. For the Kaolin clay, most of the

ammonium perfluorooctanoate adsorbed when added at 5000 µg/L desorbed with washing (> 60%). At ammonium perfluorooctanoate added at 500 µg/L, however, an average of 98% did not desorb from the Kaolin clay after two washings. The East Wood soil, which adsorbed 46% of the ammonium perfluorooctanoate added at 50 µg/L desorbed only about 2%. For the Garden Area soil 57% of the ammonium perfluorooctanoate desorbed when adsorbed from the 500 µg/L solution. No measurable desorption of ammonium perfluorooctanoate from the Upstream sediment after two washings was observed when the ammonium perfluorooctanoate was added at 50 µg/L.

Reference: DuPont Co. (2000). Unpublished Data, Report No. EMSE-053-00.

**Acute Toxicity to Fish**

**Type:** **96-hour $LC_{50}$**
Species: *Lepomis macrochirus* (bluegill sunfish)
Value: 634 mg/L (95% fiducial interval, 567-725 mg/L)
Method: A rangefinding study was conducted using nominal test concentrations of 0, 0.1, 1, 10, 100, and 1000 mg/L.

For the definitive study, nominal test concentrations were 0, 262, 328, 410, 512, 640, 800, and 1000 mg/L. Glass aquaria (20 L) containing 10 L of test solution were employed. Positions of test chambers in the water bath used to maintain constant temperature were assigned using random numbers. Ten fish were added to each replicate using random numbers (2 replicates per concentration; total 20 fish per concentration). Control fish ranged from 1.2-2.8 cm standard length (mean 2.1 cm) and 0.0451-0.524 g wet weight (mean 0.228 g). Control loading was 0.23 g/L at test conclusion. Fish were not fed approximately 24 hours prior to and during the test. A photoperiod of 16 hours light (312-344 Lux) versus 8 hours darkness was employed with 25 minutes of transitional light (<2.15 Lux) preceding and following the 16-hour light interval. Observations for mortality and behavioral effects were made daily.

Dissolved oxygen, pH, and temperature were measured in each replicate before addition of fish at test start, daily, at total fish mortality, and/or at test end. Total alkalinity, EDTA hardness, and conductivity of the control water were measured before fish were added at test start. A continuously recording thermometer in a water-control replicate was used to check temperature variations during the 96-hour test. Chemical analyses of the test substance concentrations in water were not performed.

The $LC_{50}$ value and its 95% fiducial interval were calculated.

GLP: Yes

Test Substance: Ammonium perfluorooctanoate, purity 99%

Results: Mortality in the rangefinding study after 96 hours was 0, 0, 0, 0, 0, and 80% at 0, 0.1, 1, 10, 100, and 1000 mg/L, respectively.

Total mortality was 0, 5, 10, 15, 30, 40, 65, and 95% in the 0, 262, 328, 410, 512, 640, 800, and 1000 mg/L concentrations, respectively. All deaths occurred within 24 hours, with the exception of 1 death at 48 hours at 1000 mg/L. No behavioral effects were noted at ≤ 640 mg/L. Surviving fish at 800 and 1000 mg/L were lethargic.

All chemical and physical parameters were within expected ranges. Total alkalinity, EDTA hardness, and conductivity of the dilution water control were 79 mg/L $CaCO_3$, 76 mg/L $CaCO_3$, and 165 µmhos/cm, respectively. During the test, dissolved oxygen concentrations ranged from 6.7-8.5 mg/L, pH ranged from 6.9-7.4, and temperature ranged from 21.4-22.1°C.

Reference: DuPont Co. (1994). Unpublished Data, Haskell Laboratory Report No. 61-94.

**Type:** **96-hour $LC_{50}$**

Species: *Oncorhynchus mykiss* (rainbow trout)

Value: 4001 mg/L (95% confidence interval, 3327-4932 mg/L)

Method: A static rangefinding study with 5 fish per concentration was conducted using a dilution water control and nominal concentrations of 50, 100, 500, 1000, and 5000 mg/L.

Nominal concentrations of 625, 1250, 2500, 5000, and 10,000 mg/L (20% solution of ammonium perfluorooctanoate in water) were chosen for the definitive test based on the results of the preliminary rangefinding study.

Test chambers were stainless steel aquaria that held approximately 9 L of test solution. Two replicate test chambers were used per test concentration with 10 fish in each chamber (total of 20 fish per concentration). Each chamber was covered with a glass plate to prevent fish from escaping. Random numbers were used to assign test concentrations to the test chambers and position of test concentrations in the water bath.

Rainbow trout used in this study were not fed approximately 29 hours prior to and during the test, and were assigned to the test chambers using random numbers. Addition of fish to the test solutions was initiated approximately 41 minutes after mixing of

the test solutions was completed. Mortality and behavioral observations were made at test start, every 24 hours thereafter, and at test end. At test conclusion, all surviving fish were sacrificed.

A recirculating water bath was used to maintain mean temperature in the test chambers during the 96-hour test. In addition, a continuously recording thermometer was used to check for temperature variation in 1 replicate of the dilution water control. A photoperiod of 16 hours light (approximately 199-450 Lux) and 8 hours darkness was employed, which included 30 minutes of transitional light (11-157 Lux) preceding and following the 16-hour light interval.

Dissolved oxygen, pH, and temperature were measured in all replicate chambers of the control and test substance concentrations. These measurements were taken before fish were added at test start, every 24 hours thereafter, and at test end or at total mortality in a concentration. Total alkalinity, EDTA hardness, and conductivity of the water were measured before fish were added at the beginning of the test. Test solutions were not aerated during the test.

Concentrations of ammonium perfluorooctanoate were measured directly by high performance liquid chromatography/tandem mass spectrometry (LC/MS/MS).

At test conclusion, fish from the water control ranged from 2.5-3.2 cm in standard length (mean 2.8 cm) and 0.15-0.30 g in wet weight, blotted dry (mean 0.21 g). Standard length of the longest fish was not more than twice the length of the shortest fish in the control. Loading in the water control was 0.23 g/L at test conclusion.

The $LC_{50}$ value was calculated using the moving average-angle method.

GLP: Yes

Test Substance: Ammonium perfluorooctanoate, purity 99.4%

Results: At the end of 96 hours in the rangefinding study, the mortality was 0, 0, 0, 0, 0, and 60% at 0, 50, 100, 500, 1000, and 5000 mg/L, respectively. Test substance solutions were clear and colorless with no insoluble test substance present during the study.

In the definitive study, mean measured concentrations of ammonium perfluorooctanoate were 554, 1090, 2280, 4560, and 9360 for the 625, 1250, 2500, 5000, and 10,000 mg/L dose levels, respectively. Control solutions showed no detectable

concentrations of ammonium perfluorooctanoate. All test substance solutions were clear and colorless with no insoluble test substance present during the test.

All chemical and physical parameters for the definitive test were within expected ranges. Total alkalinity, EDTA hardness, and conductivity of the dilution water control were 49 mg/L $CaCO_3$, 122 mg/L $CaCO_3$, and 240 µmhos/cm, respectively. During the test, dissolved oxygen concentrations ranged from 7.5-11.2 mg/L, pH ranged from 7.1-7.2, and mean temperature was 11.8°C (range 11.6-12.1°C).

No mortality or sublethal effects were seen in the dilution water control fish. No mortality or sublethal effects were observed at concentrations ≤ 2500 mg/L. At 5000 mg/L, mortality was 8/20, 11/20, 14/20, and 15/20 at 24, 48, 72, and 96 hours, respectively. At 10,000 mg/L all fish were dead by 24 hours.

Surviving fish exposed to 5000 mg/L exhibited sublethal effects such as swimming at the surface, labored respiration, dark coloration, lethargy, partial loss of equilibrium, rapid respiration, and erratic swimming.

Reference: DuPont Co. (1999). Unpublished Data, Haskell Laboratory Report No. Dupont-3381.

**Mammalian Toxicity**

**Acute Toxicity**

**Type:** **Oral $LD_{50}$**
Species/strain: Male rats/Crl:CD®
Value: Male rats receiving ammonium perfluorooctanoate alone: 478 mg/kg (95% confidence limits, lower 361 mg/kg, upper 571 mg/kg, slope 7.9)

Male rats receiving ammonium perfluorooctanoate and pre-treatment of phenobarbital sodium: 547 mg/kg (95% confidence limits, lower 517 mg/kg, upper 582 mg/kg, slope 22.1)

Male rats receiving ammonium perfluorooctanoate and pre-treatment of proadifen hydrochloride: 520 mg/kg (95% confidence limits, lower 450 mg/kg, upper 618 mg/kg, slope 9.8)

Method: Ammonium perfluorooctanoate was administered by intragastric intubation as a suspension in corn oil in single doses to 10 rats/group at concentrations of 400, 500, or 650 mg/kg. Another 30 rats, 10/group, were treated by single intraperitoneal injections

with aqueous solutions of phenobarbital sodium at 80 mg/kg/day for 3 days. One day after the final treatment, the rats were administered ammonium perfluorooctanoate by intragastric intubation using the same treatment as described above. An additional group of 30 rats, 10/group, were treated with an intraperitoneal injection of aqueous solutions of proadifen hydrochloride at 50 mg/kg. One hour after this treatment, ammonium perfluorooctanoate was administered by intragastric intubation, using the same treatment as described above. The surviving rats from all treatments were weighed and observed during a 14-day recovery period and then sacrificed. The $LD_{50}$ values were calculated from the mortality data.

The phenobarbital sodium phase of the study was repeated. The rats from both phases were combined and the $LD_{50}$ value was based on all 60 rats.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: There were no significant differences in the $LD_{50}$ values of ammonium perfluorooctanoate, either when tested alone or following pre-treatment with either phenobarbital sodium or proadifen hydrochloride.

Mortality ratios of 2/10, 7/10, and 8/10 were observed for the rats treated with 400, 500, and 650 mg/kg ammonium perfluorooctanoate, respectively. Wet and/or stained perineal area and weight loss were observed at all levels tested. Other clinical signs observed included stained face (500 and 650 mg/kg), weakness (500 and 650 mg/kg), and chromodacryorrhea (500 mg/kg). All deaths occurred within 6 days after dosing.

Mortality of 0/20, 4/20, and 19/20 was observed for the rats pretreated with phenobarbital sodium and then treated with 400, 500, and 650 mg/kg ammonium perfluorooctanoate, respectively. Wet and/or stained perineal area, stained face, diarrhea, and weight loss were observed at all levels tested. Other clinical signs observed included weakness (500 and 650 mg/kg) and lethargy (650 mg/kg). All deaths occurred within 4 days after dosing.

Mortality of 1/10, 5/10, and 8/10 was observed for the rats pretreated with proadifen hydrochloride and then treated with 400, 500, and 650 mg/kg ammonium perfluorooctanoate, respectively. Stained face, wet and/or stained perineal area, weight loss, and weakness were observed at all levels tested. Other clinical signs observed included tremors (400 mg/kg), chromodacryorrhea (400 mg/kg), diarrhea (500 and 650 mg/kg), and lacrimation

(650 mg/kg). All deaths occurred within 5 days after dosing.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 567-81.

**Type:** **Oral $LD_{50}$**

Species/strain: Male and female rats/Crl:CD®

Value: Male rats: 470 mg/kg (95% confidence limits, lower 403 mg/kg, upper 536 mg/kg, slope 8.6)

Female rats: 482 mg/kg (95% confidence limits, lower 438 mg/kg, upper 541 mg/kg, slope 12.3)

Method: The test substance, as a suspension in corn oil, was administered by intragastric intubation in single doses to 6 groups of male and 6 groups of female young adult rats. Ten male and female rats/group were administered 200, 400, 450, 500, 670, or 1000 mg/kg of the test substance. The surviving rats were weighed and observed during a 14-day recovery period and then sacrificed. The $LD_{50}$ values were calculated from the mortality data.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: Mortality ratios of 2/10, 3/10, 4/10, 6/10, 9/10, and 10/10 were found in the 200, 400, 450, 500, 670, and 1000 mg/kg male groups, respectively. Mortality ratios of 0/10, 0/10, 5/10, 7/10, 9/10, and 10/10 were found in the 200, 400, 450, 500, 670, and 1000 mg/kg female groups, respectively.

Clinical signs observed in male rats included stained and/or wet perineal area, weakness, and weight loss at all levels tested. Other clinical signs observed in the male rats included stained face (450 mg/kg and above), chromodacryorrhea (500 and 1000 mg/kg), hunched posture (500 mg/kg), morbundity (670 mg/kg), eyes half closed (670 mg/kg), and gasping (670 mg/kg). All deaths occurred within 4 days of dosing.

Clinical signs observed in female rats included stained and/or wet perineal area and weight loss at all levels tested. Stained face was observed at all levels except 200 mg/kg. Other clinical signs noted in female rats included alopecia (400 mg/kg), weakness (400, 450, 670, and 1000 mg/kg), hunched posture (450 and 670 mg/kg), chromodacryorrhea (670 mg/kg), eyes half closed (670 mg/kg), and ataxia (670 mg/kg). All deaths occurred within 3 days after dosing.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 295-81.

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| **Type:** | **Oral $LD_{50}$** |
| Species/strain: | Male and female rats/Crl:CD® |
| Value: | Male newborn rats: 243 mg/kg (95% confidence limits, undefined, slope 55.7) |
| | Female newborn rats: 258 mg/kg (95% confidence limits, lower 244 mg/kg, upper 276 mg/kg, slope 19.9) |
| | Male weanling rats: 573 mg/kg (95% confidence limits, lower 514 mg/kg, upper 664 mg/kg, slope 8.0) |
| | Female weanling rats: 580 mg/kg (95% confidence limits, lower 470 mg/kg, upper 2258 mg/kg, slope 4.7) |
| | Male young adult rats: 470 mg/kg (95% confidence limits, lower 403 mg/kg, upper 536 mg/kg, slope 8.6) |
| | Female young adult rats: 453 mg/kg (95% confidence limits, lower 413 mg/kg, upper 503 mg/kg, slope 15.0) |
| | Male older rats: 336 mg/kg (95% confidence limits, undefined) |
| | Female older rats: 343 mg/kg (95% confidence limits, lower 285 mg/kg, upper 390 mg/kg, slope 8.0) |
| Method: | The test substance, as a suspension in corn oil, was administered by intragastric intubation in single doses to groups of rats. The groups (10 rats/group) were male and female newborn rats (< 2 days old), male and female weanling rats (21 days old), male and female young adults (~8-10 weeks old), and male and female older rats (age undefined). Male newborn rats were administered 130, 200, 240, 280, 330, or 370 mg/kg. Female newborn rats were administered 130, 160, 200, 220, 240, 280, or 320 mg/kg. Male weanling rats were administered 350, 400, 450, 525, 670, or 710 mg/kg. Female weanling rats were administered 350, 400, 450, or 670 mg/kg. Young adult rats were administered 350, 425, 500, or 670 mg/kg. Young adult male rat data are reported in DuPont Report No. 295-81. Older male rats were administered 200, 240, 300, 350, 400, 500, or 720 mg/kg. Older female rats were administered 225, 350, 400, 450, or 670 mg/kg. The surviving rats were weighed and observed during a 14-day recovery period and then sacrificed. The $LD_{50}$ values were calculated from the mortality data. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Mortality ratios for male newborn rats were 0/10, 0/10, 0/10, 10/10, |

10/10, and 10/10 for the 130, 200, 240, 280, 330, and 370 mg/kg groups, respectively. No clinical signs were reported. Deaths occurred up to 6 days after dosing.

Mortality ratios for female newborn rats were 0/10, 0/10, 0/10, 0/10, 5/10, 6/10, and 10/10 for the 130, 160, 200, 220, 240, 280, and 320 mg/kg groups, respectively. No clinical signs were reported. Deaths occurred up to 4 days after dosing.

Mortality ratios for male weanling rats were 0/10, 2/10, 2/10, 3/10, 7/10, and 8/10 for the 350, 400, 450, 525, 670, and 710 mg/kg groups, respectively. Stained and wet perineal area, weakness, and weight loss were observed. Deaths occurred up to 3 days after dosing.

Mortality ratios for female weanling rats were 1/10, 3/10, 3/10, and 6/10 for the 350, 400, 450, and 670 mg/kg groups, respectively. Stained and wet perineal area, stained face, weakness, and weight loss were observed. Deaths occurred up to 3 days after dosing.

Mortality ratio data for the young adult males are covered in DuPont Report No. 295-81.

Mortality ratios for female young adult rats were 1/10, 2/10, 8/10, and 10/10 for the 350, 425, 500, and 670 mg/kg groups, respectively. Stained and wet perineal area, stained face, nasal discharge, diarrhea, and weight loss were observed. Deaths occurred up to 4 days after dosing.

Mortality ratios for the older male rats were 0/10, 0/10, 0/10, 9/10, 10/10, 10/10, and 10/10 for the 200, 240, 300, 350, 400, 500, and 720 mg/kg groups, respectively. Stained face, stained and wet perineal area, weakness, tremors, lethargy, chromodacryorrhea, diarrhea, and weight loss were observed. Deaths occurred up to 9 days after dosing.

Mortality ratios for the older female rats were 1/10, 5/10, 6/10, 9/10, and 10/10 for the 225, 350, 400, 450, and 670 mg/kg groups, respectively. Stained face, stained and wet perineal area, weakness, tremors, lethargy, and weight loss were observed. Deaths occurred up to 5 days after dosing.

References:

DuPont Co. (1982). Unpublished Data, Haskell Laboratory Report No. 788-82.

DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 295-81.

EID091533



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| **Type:** | **Oral $LD_{50}$ Rangefinder and Liver Weight Comparison Test** |
| Species/strain: | Male and female rats/Crl:CD® |
| Value: | Male rats: 439 mg/kg (confidence limits, lower 294 mg/kg, upper 554 mg/kg, slope 6.1)<br><br>Castrated male rats: 491 mg/kg (confidence limits, lower 276 mg/kg, upper 619 mg/kg, slope 6.7)<br><br>Female rats: 459 mg/kg (confidence limits, lower 315 mg/kg, upper 607 mg/kg, slope 5.2)<br><br>Overiectomized female rats: 400 mg/kg (confidence limits, lower 259 mg/kg, upper 494 mg/kg, slope 7.2) |
| Method: | In an $LD_{50}$ rangefinder test, the test substance, as a suspension in corn oil, was administered by intragastric intubation in single doses to normal male rats, castrated male rats, normal female rats, and ovariectomized female rats. Dose levels of 200, 480, and 670 mg/kg were tested in each of the test systems mentioned above (10 rats/group).<br><br>In the liver weight comparison test, 5 rats/group were administered the test substance, as a suspension in corn oil, as a single dose. Male groups were defined as male control, male 100 mg/kg, male 200 mg/kg, male castrated control, and male castrated 200 mg/kg. Female groups were defined as female control, female 100 mg/kg, female 200 mg/kg, female ovariectomized control, and female ovariectomized 200 mg/kg. All rats were weighed and observed during a 14-day recovery period. The rats were then sacrificed and the livers were weighed. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | In the $LD_{50}$ study, mortality in the male rats occurred in 0/10, 7/10, and 8/10 rats in the 200, 480, and 670 mg/kg groups, respectively. Mortality in the castrated male rats occurred in 0/10, 5/10, and 8/10 rats in the 200, 480, and 670 mg/kg groups, respectively. Mortality of the female rats occurred in 0/10, 7/10, and 7/10 rats in the 200, 480, and 670 mg/kg groups, respectively. Mortality in the ovariectomized female rats occurred in 0/10, 8/10, and 9/10 mg/kg at 200, 480, and 670 mg/kg, respectively.<br><br>In the liver weight comparison study tested at dose levels of 0, 100, and 200 mg/kg, no changes in liver weight or in liver to body weight ratios were seen in the female rats given single doses of up to 200 mg/kg (ovariectomized or normal). A single oral dose of either 100 or 200 mg/kg produced an increase in liver weight of |

male rats. Castration reduced the magnitude of the liver weight increase, but rats castrated and given 200 mg/kg had heavier livers than did the controls. Clinical signs seen in the ammonium perfluorooctanoate-treated rats included stained face and perineal area, and sporadic weight loss. No deaths occurred in this phase of the experiment.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 600-81.

**Type:** **Acute Oral Toxicity**
Species/strain: Male rats/Crl:CD®
Method: The test substance, ammonium perfluorooctanoate (3M) and ammonium perfluorooctanoate (Rimar Co.), as suspensions in corn oil, were administered by intragastric intubation in single doses to young adult male rats. Rats (10/group) were administered 200, 480, or 670 mg/kg of the test substance. The surviving rats were weighed and observed during a 14-day recovery period and then sacrificed.
GLP: No
Test Substance: Ammonium perfluorooctanoate (3M), purity approximately 100%

Ammonium perfluorooctanoate (Rimar Co.), purity 95% minimum

Results: Mortality ratios of 0/10, 6/10, and 10/10 were found in the 200, 480, and 670 mg/kg ammonium perfluorooctanoate (3M) groups, respectively. Mortality ratios of 0/10, 10/10, and 10/10 were found in the 200, 480, and 670 mg/kg ammonium perfluorooctanoate (Rimar Co.) groups, respectively.

Clinical signs observed in ammonium perfluorooctanoate (3M) rats included stained and/or wet perineal area, stained face, and weight loss at all levels tested. Other clinical signs observed included weakness (480 and 670 mg/kg), diarrhea (480 mg/kg), and alopecia (200 mg/kg). All deaths occurred within 4 days after dosing.

Clinical signs observed in ammonium perfluorooctanoate (Rimar Co.) rats included stained and/or wet perineal area, stained face, and weight loss at all levels tested. Other clinical signs noted included weakness (480 and 670 mg/kg) and lethargy (670 mg/kg). All deaths occurred within 4 days after dosing.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 565-81.

**Type:** **Oral ALD (Approximate Lethal Dose)**
Species/strain: Male rats/ChR-CD
Value: 670 mg/kg
Method: The test substance, as an aqueous solution (pH ~6), was

administered by intragastric intubation to young adult male rats (1 rat/group) in single doses. Concentrations tested were 1, 1.5, 2.3, 3.4, 5.1, 26, 40, 60, 77, 90, 120, 130, 170, 200, 300, 450, 670, and 2250 mg/kg. Survivors were sacrificed 14 days later, and body weights and liver weights were recorded.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity not specified

Results: Deaths did not occur at dose levels of 1-450 mg/kg. The rat dosed with 670 mg/kg died 2 days after dosing, and the rat dosed with 2250 mg/kg died 1 day after dosing.

Slight initial weight losses occurred at 26 mg/kg and above for 1-2 days. At 90 mg/kg and above, the feces were small and irregularly-shaped for 1-6 days after dosing, and the rats were uncomfortable for 1-5 days after dosing. The compound caused liver enlargement in rats with single doses as low as 60-90 mg/kg. Based on clinical signs, it also acted as a gastrointestinal irritant. Additional toxic signs at the lethal doses (670 and 2250 mg/kg) included chewing motions, polyuria, increased respiration rate, and face-pawing on the day of dosing. Shovel-nosing was observed at 2250 mg/kg and slight salivation after dosing occurred at 670 mg/kg.

Reference: DuPont Co. (1968). Unpublished Data, Haskell Laboratory Report No. 128-68.

**Type:** **Oral ALD**

Species/strain: Male rats/ChR-CD

Value: 670 mg/kg

Method: The test substance was administered by intragastric intubation to male rats (1 rat/group) in single doses. Concentrations tested were 1.5, 12, 40, 120, 200, 300, 450, 670, 1000, 1500, and 2250 mg/kg. Survivors were sacrificed 14-15 days later, and body weights and liver weights were recorded.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity 99%

Results: Mortality occurred at 670 mg/kg and above. Clinical signs of toxicity included inactivity (120, 1000, 1500, and 2250 mg/kg), red discharge around the nose (670, 1500, and 2250 mg/kg), perineal discoloration (300 and 450 mg/kg), ruffled fur (300 and 450 mg/kg), irritability (670 mg/kg), and weight loss (40, 200, 300, 450, 670, and 1000 mg/kg). Animals sacrificed 14-15 days after having received doses down to and including 200 mg/kg had enlarged livers when compared to control rats weighing between 500-550 g with liver weights of 22.6, 20.7, 19.1, and 21.7 g and liver weight/body weight percentages of 4.2, 3.8, 3.6, and 3.8, respectively. Liver weights at 200, 300, and 450 mg/kg were 30.6,

25.2, and 28.2 g, with liver weight/body weight percentages of 5.6, 5.3, and 6.2, respectively. Liver enlargement was also possible in rats dosed below 200 mg/kg, but additional test and control rats would have to be compared in order to establish this.

Reference: DuPont Co. (1961). Unpublished Data, Haskell Laboratory Report No. 55-61.

**Type:** **Acute Oral Effects, Does pretreatment with ethanol modify the toxicity of ammonium perfluorooctanoate?**

Species/strain: Male rats/Crl:CD®

Method: Young adult male rats (6/group) were administered ammonium perfluorooctanoate as a 60% aqueous solution at dose levels of 200, 480, and 670 mg/kg. Additional groups of rats were pre-treated with ethanol (15% v/v) in drinking water for 14 days and then dosed with ammonium perfluorooctanoate at the same concentrations as listed above on day 15. Three other groups of rats were pretreated with a single 6000 mg/kg dose of ethanol via intragastric intubation followed 24 hours later with the ammonium perfluorooctanoate treatment at the levels listed above. An additional group of untreated rats served as controls. Groups of rats were also treated with ethanol alone at either 6000 mg/kg or 15% v/v in drinking water for 14 days. Clinical signs and body weights were recorded. Liver weights were recorded at the 14-day sacrifice.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: All rats died within 8 days after dosing with ammonium perfluorooctanoate at 670 mg/kg. Liver weight/body weight ratios showed an increase in all rats that received ammonium perfluorooctanoate when compared to the untreated controls and the animals that received only ethanol. There were no significant differences between the untreated controls and the animals that received only ethanol. There were no significant differences between rats that received ethanol and ammonium perfluorooctanoate and the rats that received only ammonium perfluorooctanoate.

Reference: DuPont Co. (1984). Unpublished Data, Haskell Laboratory Report No. 79-84.

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**Type:** **Oral $LD_{50}$**

Species/strain: Male and female mice/CD-1

Value: Male and female mice: 457 mg/kg (95% confidence limits, lower 344 mg/kg, upper 560 mg/kg, slope 6.6)

Method: The test substance, as a suspension in corn oil, was administered by intragastric intubation in single doses to 6 groups of male and 6 groups of female young adult mice. Ten male and female



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mice/group were administered 250, 500, 750, 1000, 2000, or 4000 mg/kg of the test substance. The surviving rats were weighed and observed during a 14-day recovery period and then sacrificed. The $LD_{50}$ values were calculated from the mortality data. The male and female 4000 and 2000 mg/kg dose levels were not calculated in the $LD_{50}$ data due to limitations of the computer.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: Mortality ratios of 1/10, 4/10, 10/10, 10/10, 10/10, and 10/10 were found in the 250, 500, 750, 1000, 2000, and 4000 mg/kg male and female groups, respectively.

Clinical signs observed in male mice included stained and/or wet perineal area, weakness, and weight loss in all surviving mice. Other signs included eyes half closed (250 and 1000 mg/kg), ruffled fur (250 and 500 mg/kg), ataxia (250, 500, and 1000 mg/kg), and tremors (500 and 750 mg/kg). All deaths in male mice occurred within 6 days after dosing.

Clinical signs observed in female mice included stained and/or wet perineal area, weakness, and weight loss in all surviving mice. Other signs included eyes half closed (750 mg/kg), stained face (750 mg/kg), tremors (750 mg/kg), ataxia (500 mg/kg), and ruffled fur (250 and 750 mg/kg). All deaths in female mice occurred within 7 days after dosing.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 329-81.

**Type:** **Oral $LD_{50}$**

Species/strain: Male and female guinea pigs/Duncan Hartley

Value: Male guinea pigs: 178 mg/kg (95% confidence limits, lower 144 mg/kg, upper 202 mg/kg, slope 8.9)

Female guinea pigs: 217 mg/kg (95% confidence limits, lower 187 mg/kg, upper 246 mg/kg, slope 8.6)

Method: The test substance, as a suspension in corn oil, was administered by intragastric intubation in single doses to 6 groups of male and 6 groups of female young adult guinea pigs. Ten male and female guinea pigs/group were administered 150, 200, 250, 300, 400, or 670 mg/kg of the test substance. The surviving guinea pigs were weighed and observed during a 14-day recovery period and then sacrificed. The $LD_{50}$ values were calculated from the mortality data.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: Mortality ratios of 3/10, 6/10, 9/10, 10/10, 10/10, and 10/10 were

found in the 150, 200, 250, 300, 400, and 670 mg/kg male groups, respectively. Mortality ratios of 2/10, 1/10, 8/10, 9/10, 10/10, and 10/10 were found in the 150, 200, 250, 300, 400, and 670 mg/kg female groups, respectively.

Clinical signs observed in male guinea pigs included stained and/or wet perineal area and weakness at all levels tested. Other clinical signs observed in the male guinea pigs included stained face (150, 200, and 670 mg/kg), eyes half closed (200 and 300 mg/kg), lacrimation (150 and 200 mg/kg), ataxia (670 mg/kg), tremors (150 mg/kg), and hunched posture (200 mg/kg). Weight loss occurred at all levels tested except 400 and 670 mg/kg. All deaths occurred within 4 days after dosing.

Clinical signs observed in female guinea pigs included stained and/or wet perineal area and weakness at all levels tested except 400 and 670 mg/kg. Other clinical signs noted in female guinea pigs included convulsions (670 mg/kg), ataxia (670 mg/kg), gasping (670 mg/kg), eyes half closed (150 and 250 mg/kg), tremors (400 mg/kg), lacrimation (250 mg/kg), stained face (200 mg/kg), and lethargy (150 mg/kg). Weight loss occurred at all levels tested except for 670 mg/kg. All deaths occurred within 6 days after dosing.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 291-81.

**Type:** **Acute Toxicity**
Species/strain: Male dogs/beagle
Method: Dogs (2/group) received either 200 or 450 mg/kg of the test substance. The following biochemical measurements were made on the blood: sugar, urea nitrogen, total cholesterol, and alkaline phosphatase. When the 450 mg/kg dose was administered, the level of activity of lactic dehydrogenase (LDH), isocitric dehydrogenase (ICDH), aldolase, glutamic oxalacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) were also measured. A routine hematological examination and an analysis of a 24-hour urine specimen was made at intervals on these animals. The level of various components of the blood following dosing was compared with an average value observed prior to exposure and with a similar measurement made at the same time on specimens from stock colony dogs. For the enzyme activities, a normal range was established from measurements made on stock dogs. The activity was also measured at least once prior to treatment.
GLP: No
Test Substance: Ammonium perfluorooctanoate, purity 99%
Results: The GPT and GOT levels were elevated within 48 hours in both

dogs that received 200 mg/kg. One week later, they were in the normal range. When 450 mg/kg was administered, all of the enzymes measured were markedly elevated 24 to 48 hours later. The greatest change occurred in the GPT and ICDH. Both animals that received 450 mg/kg died within 48 hours after dosing.

Reference: DuPont Co. (1965). Unpublished Data, Haskell Laboratory Report No. 123-65.

**Type:** **Inhalation ALC (Acute Lethal Concentration)**
Species/strain: Rats/ChR-CD
Exposure Time: 4 hours
Value: 0.8 mg/L
Method: Male rats (6/exposure level), weighing 250-270 grams were exposed to dusts of ammonium perfluorooctanoate at exposure levels of 0.38, 0.81, 0.83, 2.2, 4.8, or 5.7 mg/L. Exposures were head-only, except for the 2.2 mg/L exposure level where rats had whole-body exposure. Two different methods of dust generation were used to generate ammonium perfluorooctanoate aerosols. In the first method, a heavy cloud was generated by blowing approximately 30 L/minute of air through a high velocity Cu-tubing jet submerged in a mechanically stirred reservoir of the test substance. The air carried the dust particles into an 8 L exposure chamber. Suitable diluting air was introduced between the generator and the chamber to achieve lower concentrations. The generator did not fractionate the dust, and all particle sizes, large and small, were delivered into the exposure chamber. In the second method, a generator was used for low atmospheric dust concentrations. A falling stream of dust particles from a stirred reservoir impinged on a pneumatic jet. The air stream from this jet carried the particles to a cyclone head where the larger ones were returned to the reservoir. This generator achieved some particle size fractionation. The generator was run under constant conditions and dust concentrations were lowered by diluting the stream with air between the generator and the exposure chamber. The airborne concentrations were determined 5 times during each 4-hour exposure. At the mid-point of each exposure, a particle size distribution measurement was made.

Clinical signs were recorded. The eyes of the rats were stained with fluoroscein immediately after exposure. Rats were sacrificed for histopathologic examination at 1, 7, 14, 27, or 42 days post-exposure. Tissues from two rats dying during exposure were also examined microscopically.

GLP: No
Test Substance: Ammonium perfluorooctanoate, purity approximately 100%
Results: The mortality ratios for the 0.38, 0.81, 0.83, 2.2, 4.8, and 5.7 mg/L

exposure groups were 1/6 (not test substance-related), 2/6, 1/6, 6/6, 6/6, and 6/6, respectively. All deaths occurred either during exposure or within 48 hours after exposure.

Clinical signs observed during exposure included gasping (all exposure levels), irregular breathing (all exposure levels), red discharge around the eyes and nose (all exposure levels), and salivation (2.2, 4.8, and 5.7 mg/L). Clinical signs observed post-exposure included some rupture of eyes, all other eyes opaque (2.2 and 4.8 mg/L), and > 80% rats having opaque and corroded-appearing eyes (0.81 and 0.83 mg/L). External damage of the eyes was confirmed with fluorescein stain (0.81, 0.83, and 2.2 mg/L), with the eyes appearing normal after 20 days (0.81 mg/L).

Inhalation of the test substance caused liver enlargement which reached a maximum (2 times normal) 7-14 days after exposure. Liver weights returned to the normal range 42 days after exposure. No changes in liver cell morphology were observed. Microscopic examination indicated that acute pulmonary edema developed promptly, but disappeared in approximately 1 week, leaving no residual injury. No dust particles were seen in the lungs microscopically. There was irritation of the stomach that cleared in 2 weeks. Ammonium perfluorooctanoate also caused corneal opacity and ulceration that were still microscopically evident 42 days after exposure.

References:

DuPont Co. (1969). Unpublished Data, Haskell Laboratory Report No. 160-69.

Kennedy, G. L., Jr. et al. (1983). The Toxicologist, 3:22.

Kennedy, G. L., Jr. et al. (1986). Food Chem. Toxicol., 24:1325-1329.

**Type:** **Dermal $LD_{50}$**
Species/strain: Male rats/ChR-CD
Exposure Time: 24 hours
Value: 6959 mg/kg (95% confidence limits undefined)
Method: Rats were clipped free of hair over the back and trunk area. Five male rats/dose level were dosed with 3000, 5000, and 7500 mg/kg ammonium perfluorooctanoate. The test substance, as a 50/50 aqueous suspension, was applied to the back of each rat under a square of aluminum foil and held in place with elastic bandages. After 24 hours of exposure, the rats were unwrapped, sponged off with a mild detergent, rinsed, dried, and returned to their cages for 13 days observation or until death. During the

observation period, the rats were weighed and observed for clinical signs. Gross pathology was done on all survivors and 1 animal that was found dead at 7500 mg/kg. Liver weights were recorded in all rats except 1. The $LD_{50}$ value was calculated from the mortality data.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: Mortality ratios were 0/5; 0/5, and 4/5 at 3000, 5000, and 7500 mg/kg, respectively. Death occurred within 2 days of dosing. All rats dosed at 3000, 5000, and 7500 mg/kg had initial weight loss. Clinical signs observed included lethargy (5000 and 7500 mg/kg), wet perineal area (5000 and 7000 mg/kg), stained perineal area (7500 mg/kg), stained nose (7500 mg/kg), and chromodacryorrhea (7500 mg/kg). Gross pathology showed an increase in liver weights in all surviving rats examined at 14 days post-treatment.

References: DuPont Co. (1979). Unpublished Data, Haskell Laboratory Report No. 659-79.

Kennedy, G. L., Jr. (1985). Toxicol. Appl. Pharmacol., 81:348-355.

**Type: Dermal $LD_{50}$**

Species/strain: Female rats/ChR-CD

Exposure Time: 24 hours

Value: > 7500 mg/kg (the maximum feasible dose)

Method: Rats were clipped free of hair over the back and trunk area. Five female rats/dose level were dosed with 5000 or 7500 mg/kg ammonium perfluorooctanoate. The test substance, as a 50/50 aqueous suspension, was applied to the back of each rat under a square of aluminum foil and held in place with elastic bandages. After 24 hours of exposure, the rats were unwrapped, sponged off with a mild detergent, rinsed, dried, and returned to their cages for 13 days observation or until death. During the observation period, the rats were weighed and observed for clinical signs. Surviving rats were sacrificed.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: The mortality was 0/5 and 1/5 at 5000 and 7500 mg/kg, respectively. Death occurred 3 days after treatment. Ammonium perfluorooctanoate caused mild skin irritation and weight loss at both dose levels. In addition, stained face and wet and stained perineal area were observed at 7500 mg/kg.

References: DuPont Co. (1980). Unpublished Data, Haskell Laboratory Report No. 682-80.

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Kennedy, G. L., Jr. (1985). Toxicol. Appl. Pharmacol., 81:348-355.

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| **Type:** | **Dermal $LD_{50}$** |
| Species/strain: | Male rabbits/New Zealand White |
| Exposure Time: | 24 hours |
| Value: | 4278 mg/kg (95% confidence limits, lower 2369 mg/kg, upper 9814 mg/kg, slope 6.2504) |
| Method: | Rabbits were clipped free of hair over the back and trunk area, and were fitted with plastic collars. Five, 5, 5, and 2 male rabbits were dosed with 1500, 3000, 5000, and 7500 mg/kg ammonium perfluorooctanoate, respectively. The test substance was made into a slurry with water, was applied to the back of each rabbit, and the test site was occluded (wrapped with plastic wrap, gauze, and elastic bandages). After 24 hours of exposure, the animals were unwrapped, washed with water, dried, and returned to their cages for 14 or 15 days observation or until death. During the observation period, the animals were weighed and observed for clinical signs. Gross pathology was done on all survivors and 1 animal that was found dead at 3000 mg/kg. The $LD_{50}$ value was calculated from the mortality data. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Mortality ratios of rabbits were 0/5, 1/5, 3/5, and 2/2 at 1500, 3000, 5000, and 7500 mg/kg, respectively. Death occurred within 4 days of dosing. Gross pathological examination showed no compound-related changes; however, there was skin irritation at all dose levels. Clinical signs included weight loss and labored breathing at all dose levels. In addition, lethargy (3000 mg/kg), nasal discharge (5000 mg/kg), shallow breathing (5000 mg/kg), pallor (5000 mg/kg), diarrhea (5000 mg/kg), weakness (5000 mg/kg), wet underneath the body (5000 and 7500 mg/kg), and cyanosis (7500 mg/kg) were observed. |
| References: | DuPont Co. (1979). Unpublished Data, Haskell Laboratory Report No. 659-79. |

Kennedy, G. L., Jr. (1985). Toxicol. Appl. Pharmacol., 81:348-355.

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| **Type:** | **Dermal Irritation** |
| Species/strain: | Male rabbits/New Zealand White |
| Method: | Six male rabbits were clipped free of hair on the trunk and lateral areas, and placed in FDA-type stocks. Doses of 0.5 g solid ammonium perfluorooctanoate were applied as an aqueous paste to the intact skin under gauze squares, and the test site was semi-occluded (rubber sheeting was loosely wrapped around the |

trunk and secured with adhesive tape). After 24 hours, the rabbits were removed from the stocks, the patches removed, and the reactions observed. Observations were also made at 48 hours.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: Ammonium perfluorooctanoate caused mild to moderate skin irritation in 24 hours, and slight to moderate irritation in 48 hours when tested on the shaved intact skin of rabbits under semi-occluded conditions.

Reference: DuPont Co. (1979). Unpublished Data, Haskell Laboratory Report No. 636-79.

**Type:** **Dermal Irritation**

Species/strain: Male rabbits/New Zealand White

Method: Twenty-one male rabbits were clipped free of hair over the back and trunk areas, and fitted with plastic collars. Ammonium perfluorooctanoate impregnated Teflon® strips or ammonium perfluorooctanoate impregnated Kevlar® strips were applied to the back of each rabbit. Three rabbits served as controls and were wrapped in non-ammonium perfluorooctanoate-treated material. The trunk of each rabbit was then occluded (wrapped with a layer of plastic wrap, gauze stretch bandage, and adhesive stretch tape). After 4-, 8-, or 24-hour exposure periods, the wrappings were removed and the exposed areas were wiped with gauze pads that were soaked in a 50:50 ethanol:water solution. The animals were observed after unwrapping for skin irritation.

GLP: No

Test Substance: Ammonium perfluorooctanoate impregnated Teflon® (48% Teflon® fibers, 38% Teflon® resin that included 2.9% Triton® X-100 and 0.061% ammonium perfluorooctanoate, and 14% dimethylsilicone oil)

Ammonium perfluorooctanoate impregnated Kevlar® (40.5% Kevlar® fibers, 42% Kevlar® resin that included 3.2% Triton® X-100 and 0.067% ammonium perfluorooctanoate, and 17.5% dimethylsilicone oil)

Results: No skin irritation was observed during the study.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 736-81.

**Type:** **Eye Irritation**

Species/strain: Rabbits/New Zealand White

Method: One-tenth mL (38.3 mg) of solid test substance was placed into the right conjunctival sac of each of 2 rabbits (sex not specified). After 20 seconds, 1 treated eye was washed with tap water for 1 minute. The treated eye of the other rabbit was not washed. Observations

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| | of the cornea, iris, and conjunctiva were made with a hand-slit lamp at 1 and 4 hours, and at 1, 2, 3, 7, 14, 21, and 28 days. Fluor-I-strip® stain and a biomicroscope were used at examinations after the day of treatment. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity 100% |
| Results: | Ammonium perfluorooctanoate caused generalized moderate corneal opacity with a small area of severe opacity, intermittent moderate iritis, and moderate conjunctivitis when tested in a rabbit eye that was unwashed after treatment. The ocular effects gradually receded; however, the small area of corneal opacity persisted, and at 21-28 days was mild with vascularization (sign of healing). An eye dosed with the test substance and promptly washed had a small area of slight to moderate corneal opacity and moderate to slight conjunctivitis with no iritic effect. The eye was normal within 7 days, except for mild conjunctival redness, which was normal within 14 days. |
| Reference: | DuPont Co. (1979). Unpublished Data, Haskell Laboratory Report No. 635-79. |

**Repeat Dose Toxicity**

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| **Type:** | **Repeated Dose Oral Toxicity Study** |
| Species/Strain: | Mice/CD-1<br>Rats/Crl:CD® |
| Sex/Number: | Male and female/5/group |
| Exposure Period: | 9 doses (3/week) |
| Frequency of Treatment: | 3 weeks |
| Exposure Levels: | 0, 0.1, 1.0, 10 mg/kg |
| Method: | The test substance, as aqueous solutions, was administered by intragastric intubation to rats and mice. Control rats and mice received water only. Body weights and clinical signs were recorded. Animals were sacrificed 3 days after the final dose and the livers were removed and weighed. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | A significant liver weight increase was noted in both male and female mice at the 1 and 10 mg/kg levels. Significantly increased liver weights were noted in male rats, but only at 10 mg/kg. There were no significant changes in the liver weights of female rats at any dose. |

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| | Mortality occurred in 2/5 female mice at 10 mg/kg. No mortality occurred in male mice, male rats, or female rats at any dose level tested. Sporadic weight loss occurred in the 0.1, 1.0, and 10 mg/kg female rats and in the 0.1, 1.0, and 10 mg/kg male and female mice. Weakness was observed in the 10 mg/kg female mice. |
| Reference: | DuPont Co. (1983). Unpublished Data, Haskell Laboratory Report No. 138-83. |
| **Type:** | **Repeated Dose Oral Toxicity Study** |
| Species/Strain: | Rats/Crl:CD |
| Sex/Number: | Male/12 |
| Exposure Period: | 12 days; 14 days of recovery |
| Frequency of Treatment: | 10 daily doses (5 doses, 2 non-dose days, 5 doses) |
| Exposure Levels: | 0, 6.7 mg/kg |
| Method: | Each of 6 rats received 10 daily doses of ammonium perfluorooctanoate by intragastric intubation as an aqueous solution. Body weights were recorded. Three rats were killed 4 hours after receiving the $10^{th}$ dose, and the remainder were killed 14 days later. Organ weights were recorded. Six undosed rats served as controls. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity 99% |
| Results: | The most prominent effect of the test substance was enlargement of the liver, which, at the end of the dosage regimen, was about 45% heavier than that of the control rats. This change persisted after cessation of dosage, but the weight discrepancy was somewhat less since 14 days later the livers were only 20% heavier than those of control rats.<br><br>The renal weights were 20% heavier than those of corresponding control rats, and this increase persisted after cessation of dosage, being 22% above that of the control rats 14 days later. These weight changes were not accompanied by morphological changes.<br><br>The pancreatic weights were slightly depressed, being 8% and 12% lower than those of controls at the end of the test and recovery phases, respectively. The adrenals and testes were slightly increased in weight after the last dose, +14% and +11%, respectively, but returned to normal 14 days later. |
| Reference: | DuPont Co. (n.d.). Unpublished Data. |

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| **Type:** | **14-Day Feeding Study** |
| Species/Strain: | Rat/Crl:CD®BR |
| Sex/Number: | Male/20/group |
| Exposure Period: | 14 days; 56 days of recovery |
| Frequency of Treatment: | *ad libitum* for 14 days |
| Exposure Levels: | 0, 30, 300 ppm |
| Method: | Rats were approximately 6 weeks old at arrival. At study start, the body weights ranged from 196 to 240 g. Rats were housed singly and were fed food and water *ad libitum*. During the test period each group were fed diet that contained 0, 30, or 300 ppm of the test substance. Diets were prepared once for the 2-week feeding period and were stored refrigerated until used. All rats were weighed and observed for clinical signs of toxicity. Rats not sacrificed at the end of the feeding period were weighed and observed during the recovery period. Cageside examinations to detect moribund or dead rats were conducted twice daily. Five rats/group were sacrificed at the end of the 14-day feeding period, and on recovery days 7, 28, and 56. Livers were removed and weighed at each sacrifice period. Blood samples were taken for organofluoride concentration analysis from rats sacrificed at the end of the feeding period and on recovery day 7.<br><br>Appropriate statistical methods were used to analyze body weights, body weight gains, and organ weight data. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Diets were not analyzed.<br><br>No mortalities were observed during the study. Clinical signs of toxicity noted in the 30 and 300 ppm groups during the feeding period included irregular respiration, rapid breathing, red nasal discharge, and hunched posture. Statistically significant decreases in mean body weights were observed in rats from the 300 ppm dose group on days 7 and 14 of the feeding period and on recovery day 7. Mean body weight gains from this same group were significantly depressed during the first week of feeding.<br><br>Significant increases in mean absolute liver weights were observed in rats from the 30 and 300 ppm dose groups sacrificed at the end of the feeding period and on recovery days 7 and 28. Significantly increased mean relative liver weights were observed in rats from the 30 and 300 ppm dose groups sacrificed at the end of the feeding |

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| | period and on recovery day 7, and in the 300 ppm dose group rats sacrificed on recovery day 28.<br><br>Mean blood organofluoride concentrations of rats sacrificed on recovery day 0 were 0.3 ppm in control rats, 33.2 ppm in the 30 ppm rats, and 71.5 ppm in the 300 ppm rats. On recovery day 7, mean blood organofluoride concentrations were 0.9 ppm for controls, 19.3 ppm for the 30 ppm group, and 22.2 ppm for the 300 ppm group. |
| Reference: | DuPont Co. (1995). Unpublished Data, Haskell Laboratory Report No. 326-95. |
| **Type:** | **14-Day Feeding Study** |
| Species/Strain: | Mice/Crl®:CD-1 |
| Sex/Number: | Male and female/5/group |
| Exposure Period: | 14 days |
| Frequency of Treatment: | *ad libitum* for 14 days |
| Exposure Levels: | 0, 30, 300, 3000 ppm |
| Method: | Male and female mice (age 43 and 44 days, weighing 21 to 38 g) were fed diets containing ammonium perfluorooctanoate for 14 consecutive days. Male and female controls were observed concurrently and fed only ground chow. Individual body weights, food consumption, and clinical signs were recorded. Liver weights were recorded at the 14-day sacrifice. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | All male and female mice died after dosing with 3000 ppm. One female mouse died at 300 ppm. Body weight loss occurred at 300 and 3000 ppm. Liver weight/body weight ratios showed a dose-response increase at ≥ 30 ppm in male and female mice. Clinical signs observed included unkempt head area (300 and 3000 mg/kg) and weakness (300 mg/kg). |
| References: | DuPont Co. (1982). Unpublished Data, Haskell Laboratory Report No. 12-82.<br><br>Kennedy, G. L., Jr. (1987). Toxicol. Lett., 39:295-300. |
| **Type:** | **14-Day Feeding Study** |
| Species/Strain: | Mice/Crl®:CD-1 |
| Sex/Number: | Male and female/5/group/gender |
| Exposure Period: | 14 days |

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| Frequency of Treatment: | *ad libitum* for 14 days |
| Exposure Levels: | 0, 10, 30, 100, 300, 1000, 3000, 10,000 ppm |
| Method: | Male and female mice (age 41 and 42 days, weighing 19 to 30 g) were fed diets containing ammonium perfluorooctanoate for 14 consecutive days. Male and female controls were observed concurrently and fed only ground chow. Individual body weights, food consumption, and clinical signs were recorded. Liver weights were recorded at the 14-day sacrifice. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Death occurred at 1000, 3000, and 10,000 ppm. All female mice at 3000 ppm and all male mice at 10,000 ppm were dead at the 2-week time point. All 10,000 ppm female mice and 3000 ppm male mice were dead at the 1-week time point. Although death occurred in the 1000 ppm mice, details on the number of deaths was not reported.<br><br>Body weight loss occurred at the end of each week at 300, 1000, 3000, and 10,000 ppm male and female mice. Liver weight/body weight ratios showed a dose-response increase at ≥ 10 ppm in male and female mice. Clinical signs observed included weakness (100 – 3000 ppm), tremors (300 and 1000 ppm), piloerection (100, 1000, and 3000 ppm), pallor (10,000 ppm), stained perineal area (100 – 1000 ppm), weight loss (100, 1000, 3000, and 10,000 ppm), and unkempt appearances (300 – 10,000 ppm). |
| Reference: | DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 560-81. |

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| **Type:** | **14-Day Feeding Study** |
| Species/Strain: | Mice/Crl®:CD-1 |
| Sex/Number: | Male and female/5/group |
| Exposure Period: | 14 days |
| Frequency of Treatment: | *ad libitum* for 14 days |
| Exposure Levels: | Ammonium perfluorooctanoate: 0, 30 ppm<br>Nonadecafluorodecanoic acid: 3, 10, 30, 300, 3000 ppm<br>Mixtures of nonadecafluorodecanoic acid/ammonium perfluorooctanoate: 15/15, 5/25, 25/5 ppm |
| Method: | Male and female mice (age 44 days, weighing 24 to 35 g) were fed diets containing either ammonium perfluorooctanoate, nonadecafluorodecanoic acid, or mixtures of the two test substances for 14 consecutive days. Male and female controls were observed concurrently and fed only ground chow. Individual body |

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| | weights and clinical signs were recorded. Liver weights were recorded at the 14-day sacrifice. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Liver weight/body weight ratio showed an increase at all levels tested. Mixing the 2 test substances did not alter the effects. Liver enlargement appeared to be dose-related with nonadecafluorodecanoic acid appearing the more potent of the two substances. |
| Reference: | DuPont Co. (1982). Unpublished Data, Haskell Laboratory Report No. 537-82. |
| **Type:** | **21-Day Feeding Study** |
| Species/Strain: | Mice/Crl®:CD-1 |
| Sex/Number: | Male and female/5/group |
| Exposure Period: | 21 days |
| Frequency of Treatment: | *ad libitum* for 21 days (Mice were mistakenly fed ground chow, without compound, for 2 days) |
| Exposure Levels: | 0, 0.01, 0.03, 0.1, 0.3, 1, 3, 10, 30 ppm |
| Method: | Male and female mice (age 41 and 42 days, weighing 21 to 33 g) were fed diets containing ammonium perfluorooctanoate (suspended in 1% corn oil before being mixed in ground chow) for 21 consecutive days. Male and female controls were observed concurrently and fed only ground chow. Individual body weights and clinical signs were recorded. Liver weights were recorded at the 21-day sacrifice. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Livers were significantly heavier at 30 ppm, slightly heavier at 3 ppm, and appeared normal at 10 ppm in male and female mice. No changes in liver weight were seen at feeding levels of 1 ppm or lower, and the significance of the change observed at 3 ppm, in light of no differences at 10 ppm, is questionable. The only clinical sign observed was sporadic weight loss. |
| References: | DuPont Co. (1982). Unpublished Data, Haskell Laboratory Report No. 323-82. |
| | Kennedy, G. L., Jr. (1987). Toxicol. Lett., 39:295-300. |
| **Type:** | **2-Year Feeding Study** |
| Species/Strain: | Rat/Crl:CD®BR |
| Sex/Number: | Male/156/group |
| Exposure Period: | 2 years |

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Frequency of Treatment: *ad libitum* for 2 years

Exposure Levels: 0, 0 (pair-fed), 300 ppm

Method: Male rats were administered either 0 (*ad libitum* control group (control)), 0 (control pair-fed to the ammonium perfluorooctanoate group (CP-C8)), or 300 ppm ammonium perfluorooctanoate in the diet for approximately 2 years. After assignment to treatment groups each rat was designated for either hormonal evaluation (10/group/time point), cell proliferation evaluation (6/group/time point), or evaluation of peroxisome proliferation (6/group/time point). All rats were provided food and tap water *ad libitum*. Stability of ammonium perfluorooctanoate was confirmed by analyses at the beginning, middle, and end of the study. Throughout the study, concentration of the test compound in the diet and the homogeneity of the test diets were determined.

All rats were approximately 49 days of age on the day of study start. Body weights, food consumption, and clinical signs were monitored throughout the study.

Blood was collected for hormonal analyses at approximately 1, 3, 6, 9, 12, 15, 18, and 21 months after initiation of the study. Serum was analyzed for testosterone, estradiol, luteinizing hormone (LH), follicle stimulating hormone (FSH), and prolactin concentrations.

Rats were sacrificed at the following interim time points: 1, 3, 6, 9, 12, 15, 18, and 21 months for cell proliferation and peroxisome proliferation evaluations. The following organs were weighed: testes, epididymides, accessory sex gland (ASG) unit with fluid, coagulating gland/seminal vesicle with fluid removed, prostate, and liver. Immediately after weighing, the liver and testes from animals selected for peroxisome proliferation evaluation were placed in ice-cold homogenization buffer for peroxisomal preparation. The following tissues were collected from the rats selected for cell proliferation evaluation: right and left testes, epididymides, ASG, liver, duodenum, pituitary, and all organs with gross lesions. All rats surviving the 24-month test period were sacrificed and necropsied. Brain, heart, liver, spleen, kidneys, ASG unit, coagulating gland/seminal vesicles with fluid removed, prostate, epididymides, and testes were weighed at necropsy. The liver, testes, epididymides, pancreas, and organs with gross lesions were examined microscopically.

Data were analyzed by appropriate statistical methods.

GLP: No

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| Test Substance: | Ammonium perfluorooctanoate, purity 98-100% |
| Results: | Increased relative liver weights were observed in the ammonium perfluorooctanoate-treated rats. Hepatic ß-oxidation activity was also increased in the ammonium perfluorooctanoate-treated rats at all time points. Ammonium perfluorooctanoate did not significantly alter the rate of Leydig cell ß-oxidation or Leydig cell proliferation. Moreover, the rate of ß-oxidation in Leydig cells was approximately 20-times less than the rate of hepatic ß-oxidation. Serum testosterone, FSH, prolactin, and LH levels were unchanged. There were, however, significant increases in serum estradiol levels in the ammonium perfluorooctanoate-treated rats at 1, 3, 6, 9, 15, 18, and 21 months. At 12 months, the ammonium perfluorooctanoate-treated rats had elevated serum estradiol levels when compared to the pair-fed control. Histopathological evaluation revealed compound-related increases in liver, Leydig cell, and pancreatic acinar cell tumors. Based on these data, the Leydig cell tumors appear to be due to the combination of elevated estradiol levels and reduced prolactin levels. |
| Reference: | DuPont Co. (2000). Unpublished Data (Draft Manuscript). |

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| **Type:** | **Repeated Exposure Inhalation Study** |
| Species/Strain: | Rats/ChR-CD |
| Sex/Number: | Male/20/group |
| Exposure Period: | 2 weeks; 42 days of recovery |
| Frequency of Treatment: | 6 hours/day, 5 days/week |
| Exposure Levels: | 0, 8, 80 mg/m$^3$ |
| Method: | Houseline air (approximately 20 L/min) was passed through a cyclone-head dust generator connected to a particle agitator-reservoir that contained ammonium perfluorooctanoate. The resulting airborne particulate was passed into a 30 L battery jar exposure chamber. The atmospheric concentration of ammonium perfluorooctanoate in the exposure chamber was monitored at 30-minute intervals. |

Rats were exposed, head only, for 5 consecutive days, 6 hours per day. After 5 days, the rats were given a 2-day recovery (weekend), which was followed by 5 daily, consecutive 6-hour exposures. All rats were weighed and observed daily (except weekends) during the exposure and recovery periods. Food and water were available *ad libitum* at all times other than during the actual exposure.

Clinical laboratory examinations were performed on 10 rats from each group at 0, 14, and 28 days post-exposure. After a total of

10 exposures, 5 rats from each group were pathologically evaluated at 0, 14, 32, and 42 days post-exposure. Organs were weighed, and absolute and relative organ weights were calculated.

On the 5th and 9th days of exposure, 10 rats were selected from each group for eye examinations.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: Overall mean concentrations were 0, 11 ± 5, 83 ± 17 for the 0, 8, and 80 mg/m$^3$ exposure levels, respectively.

Rats exposed to ammonium perfluorooctanoate showed a suppression of body weight throughout the test. The 83 mg/m$^3$ group was more severely affected during exposure and for 14 days of recovery. During each exposure, sporadic cases of blinking, pawing, chewing, and red eye and nasal discharge were seen in all groups.

Some rats exposed to ammonium perfluorooctanoate dust displayed elevated alkaline phosphatase activity. Effects were seen in glutamic-pyruvic transaminase and glutamic-oxalacetic transaminase after 0, 14, and 28 recovery days. The incidence was related to dose; higher activities were found at the higher exposure level. However, only the latter activity at 83 mg/m$^3$ was statistically different from controls.

Pathologic evaluations showed cloudy swelling or granular degeneration of hepatocytes in the livers of rats exposed to ammonium perfluorooctanoate for 10 days with no recovery. This effect was not seen after a 14-day or longer recovery period. No other compound-related histologic changes were noted. Exposure-related increase in liver weights was observed.

No corneal, iritic, or conjunctival effects were seen in any of the rats examined after 5 or 9 exposure days.

References: DuPont Co. (1979). Unpublished Data, Haskell Laboratory Report No. 253-79.

Kennedy, G. L., Jr. et al. (1983). The Toxicologist, 3:22.

**Type:** **Repeated Exposure Inhalation Study**

Species/Strain: Rats/Crl:CD®

Sex/Number: Male/24/group

Exposure Period: 2 weeks; 84 days of recovery

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| Frequency of Treatment: | 6 hours/day, 5 days/week |
| Exposure Levels: | 0, 1, 8, 80 mg/m$^3$ |
| Method: | Dust atmospheres of ammonium perfluorooctanoate were generated by passing air through a glass generator. For the high concentration (80 mg/m$^3$), chamber atmosphere concentrations were primarily determined by gravimetric analysis. For the intermediate and low concentrations (8 and 1 mg/m$^3$, respectively), chamber atmospheres were determined by a chemical analyses.<br><br>Male rats (age 7-8 weeks, weighing 240-279 g) were exposed head-only to dust atmospheres for 6 hours/day, 5 days/week for 2 weeks (weekends excluded). During exposure, rats were observed and clinical signs were noted. Post-exposure rats were weighed and observed daily for 14 recovery days, then weighed and observed 2 times/week through 84 days of recovery. Five rats/group were sacrificed at 0, 14, 28, 42, and 84 days of recovery, for a total of 96 test days.<br><br>Clinical laboratory examinations were performed on 5 rats from each group at 0, 14, 28, 42, and 84 days post-exposure. After a total of 10 exposures, 5 rats from each group were pathologically evaluated at 0, 14, 28, 42, and 84 days post-exposure. The rats were examined grossly and tissues and organs were saved for microscopic evaluation. In addition, lungs, heart, thymus, spleen, liver, testes, and kidneys were weighed.<br><br>At necropsy, blood was collected for analysis of organofluoride levels in rat blood. Blood samples from the 0 and 80 mg/m$^3$ groups were analyzed at each recovery period. Blood samples from the 1 and 8 mg/m$^3$ groups were analyzed only at 0 and 28 recovery days. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Overall mean concentrations were 1.0 ± 0.5, 7.6 ± 2.5, and 83.9 ± 12.8 mg/m$^3$ for the 1, 8, and 80 mg/m$^3$ groups, respectively.<br><br>Body weight analysis demonstrated no significant differences between controls and 1 mg/m$^3$. Body weights from animals exposed to 8 mg/m$^3$ were significantly higher than controls from test days 17-33. Body weights from animals exposed to 80 mg/m$^3$ were significantly lower than controls from test days 2-16. Observations of clinical signs during exposures showed only slight signs of nasal and ocular discharge. However, at the high concentration, after 3-4 days on test 1 rat died during exposure and 1 rat was sacrificed |

*in extremis*. Both of these rats had severe weight loss. Mortality was probably exposure-related, although pathologic evaluation could not determine the cause of death. Three of 24 rats in the 80 mg/m$^3$ exposure group exhibited lung noise during the 12-day exposure period.

Organ to body weight ratios demonstrated a significant, dose-related increase in lung, liver, and testes weights after 0 recovery days. The liver/body weight ratios were significantly higher in animals exposed to 80 mg/m$^3$ through 28 days of recovery. Mean absolute liver weights were significantly higher in the 8 mg/m$^3$ animals through 28 days of recovery, but this may be an artifact caused by an unexplainable increase in body weight accompanied by a normal increase in liver weights at 8 mg/m$^3$.

Clinical laboratory measurements demonstrated an increase in alkaline phosphatase in all groups exposed to ammonium perfluorooctanoate after 10 exposures, but this finding was significant only at 8 and 80 mg/m$^3$. This increase persisted in the 80 mg/m$^3$ animals through 14 days of recovery. After 28, 42, and 84 days of recovery no differences were found.

Compound-related pathologic findings included heavy livers, panlobular hepatocellular hypertrophy, centrolobular hepatocellular hypertrophy, and hepatocellular necrosis in animals exposed to 8 and 80 mg/m$^3$. These findings showed an exposure-response relationship, but were reversible by 28 days of recovery (8 mg/m$^3$) or 42 days of recovery (80 mg/m$^3$).

Blood organofluoride analysis clearly demonstrated an exposure-related presence in all groups (including the controls, this finding remains unexplained). Blood organofluoride levels decreased with time, but was detectable after 84 days of recovery in both the control and 80 mg/m$^3$ exposure levels.

References:

DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 205-81.

Kennedy, G. L., Jr. et al. (1983). The Toxicologist, 3:22.

Kennedy, G. L., Jr. et al. (1986). Food Chem. Toxicol., 24:1325-1329.

**Type:** **Repeated Dose Dermal Study**
Species/Strain: Rats/ChR-CD
Sex/Number: Male/15/group

EID091555



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| Exposure Period: | 2 weeks; 42 days of recovery |
| Frequency of Treatment: | 6 hours/day, 5 days/week |
| Exposure Levels: | 0, 20, 200, 2000 mg/kg |
| Method: | Male rats (age 8 weeks, weighing 210-245 g) were collared to prevent ingestion of the compound when preening and grooming during the 2-week exposure period. Ammonium perfluorooctanoate, as an aqueous paste, was applied to the backs of each rat that had been shaved free of hair. Daily 6-hour exposures ended when the compound was wiped from the rats' backs with a gauze pad. Collars were removed after exposure day 10. Throughout the test period, food and water were available *ad libitum*. Body weights and clinical signs were recorded.<br><br>After exposure day 10 and on recovery days 14 and 42, blood was taken from 5 rats from each group for hematology measurements. Gross necropsy and histopathological examinations were performed on 5 rats/group after exposure 10, and on recovery days 14 and 42. Mean absolute and relative organ to body weight analyses were performed. After exposure 10 and on recovery days 14 and 42, blood was collected from 5 rats/group for organofluorine determinations. Eye examinations were performed on each rat after exposure 9, and on recovery days 13 and 41. The procedure included gross observation of the eyes using a bright light, and semimicroscopic observation using a hang magnifying lamp and a slit-lamp biomicroscope.<br><br>Data were analyzed by appropriate statistical methods. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Rats treated with 20 mg/kg ammonium perfluorooctanoate showed normal body weights and no unusual clinical signs during the experiment. During the 10-day exposure period, rats treated with either 200 or 2000 mg/kg lost weight, followed by a normal growth after the exposure period. Slight redness of the skin was observed in these 2 groups, along with salivation in the 2000 mg/kg group only.<br><br>Clinical enzyme determinations monitoring liver function (alkaline phosphatase, GPT, and GOT) showed dose-related increases in all treated groups after exposure 10. These values returned to normal at recovery days 14 and 42.<br><br>Liver damage characterized by coagulative necrosis was observed in all treated groups following the 10$^{th}$ dose. The incidence and |

severity of liver damage was dose-related. Recovery was complete in the 20 mg/kg group 14 days following the 10th dose, and was essentially complete in the 200 mg/kg group at the same time. On recovery day 42, reversal of liver damage was essentially complete in the 2000 mg/kg group. Two rats exposed to 2000 mg/kg had coagulative necrosis of the epidermis at the dose site following 10 exposures. Liver weights, both on an absolute and relative to body weight basis, showed a dose-related increase on exposure day 10, with a return to normal weight seen in the 20 mg/kg group at 14 days and in the 200 mg/kg group at 42 days of recovery. The increased liver weight persisted for 42 days in the 2000 mg/kg group, although a trend toward normal was observed. On exposure day 10, the mean kidney weights of the 20 mg/kg group were significantly greater than controls, and the mean absolute spleen and kidney weights from the 2000 mg/kg group were significantly less than controls. The mean relative testicular weights from the 2000 mg/kg group showed a statistically significant increase on exposure day 10. On recovery day 14, there was a statistically significant increase in the mean relative kidney and testes weights of the 200 and 2000 mg/kg groups.

Blood organofluoride levels showed dose-related elevation on exposure day 10, followed by a decrease in the levels at recovery days 14 and 42. These values after the 10th exposure ranged from 52, 81, and 118 ppm (20, 200, and 2000 mg/kg, respectively) to 1, 4, and 8 ppm (20, 200, and 2000 mg/kg, respectively) at recovery day 42. At a blood organofluoride concentration of approximately 10 ppm, there were normal liver weight to body weight ratios and serum enzyme activity, and no clinical signs or body weight differences from controls. One rat showed liver changes at this level. At a blood organofluoride level of approximately 50 ppm, there were marked liver changes and significant increases in the mean absolute and relative liver weights. No other toxic effects were evident at this blood organofluoride concentration.

References:

DuPont Co. (1980). Unpublished Data, Haskell Laboratory Report No. 589-80.

Kennedy, G. L., Jr. (1985). Toxicol. Appl. Pharmacol., 81:348-355.

**Developmental Toxicity**

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| **Type:** | **Developmental Toxicity in Rats** |
| Species/Strain: | Rats/Crl:CD®(SD)BR |
| Sex/Number: | Females/6-15/group |
| Route of Administration: | Inhalation |

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| Exposure Period: | Days 6-15 of Gestation |
| Frequency of Treatment: | 6 hours/day |
| Exposure Levels: | 0, 0.1, 1.0, 10.0, 25.0 mg/m$^3$ |
| Method: | Female rats were mated to males, and mating was verified by detection of spermatozoa in the vaginal lavage each morning following overnight cohabitation. The day that spermatozoa were detected was designated as Day 1 of gestation (Day 1G). |

For Experiment I (females sacrificed before parturition), a total of 24 mated females were to have been assigned to each group (12 females/group/Run). However, due to the degree of maternal toxicity in evidence in the 25.0 mg/m$^3$ group in Run I, this concentration was reduced to 10.0 mg/m$^3$ for Run II, and 15 mated females were assigned to this new group. Furthermore, two more control groups (6 mated females/group) were added to Run II; one was pair-fed to the 25.0 mg/m$^3$ group, and the other was pair fed to the 10 mg/m$^3$ group.

For Experiment II (females allowed to give birth), in Run I 12 mated female rats were distributed to each group. With the addition of the 10.0 mg/m$^3$ group in Run II, 6 mated females were added to both the control and the 10.0 mg/m$^3$ groups.

Rats were exposed whole-body to atmospheres of ammonium perfluorooctanoate in 150 L glass and stainless steel Rochester-type chambers within which the rats were housed individually in wire-mesh modules. Chamber concentrations were determined by gravimetric analysis either each ½ hour (1.0 mg/m$^3$, and 10 or 25 mg/m$^3$) or each hour (0.1 mg/m$^3$), and by a spectrophotometric technique (on each 0.1 mg/m$^3$ sample, and on 5-6 samples per exposure day for the other levels tested).

Dams were weighed and observed for clinical signs. Feed consumption was measured during gestation (2 females/cage). The dams were coded from before sacrifice until all maternal and fetal data were collected, and until all structural alterations noted among the fetuses were classified. After sacrifice, the dams were examined for gross pathologic changes, liver weight was recorded, and reproductive status was determined. The number of corpora lutea and implantation sites were counted, and the number and position of all live, dead, and resorbed fetuses were recorded.

All live and dead fetuses were weighed and sexed externally and

internally, and the live fetuses were examined for external alterations. Approximately ½ of the fetuses of each litter that were alive when removed from the dam were examined for visceral alterations, and all stunted or malformed fetuses were examined similarly. The fetuses that were examined for visceral alterations were also examined for head alterations. Sections, containing the eyes of 3 fetuses from each litter of the 25.0 mg/m$^3$ group and of 2 fetuses from each litter of the control group, were processed histologically for examination. In Run II, one fetal head from each of 4 litters from the 10.0 mg/m$^3$ group and the control group were examined. In addition; the heads from all fetuses in the group pair fed to the 25 mg/m$^3$ group were processed for examination. All fetuses were examined for skeletal alterations.

For Experiment II, the procedures used until Day 21G were the same as for Experiment I, except that the dams were weighed less frequently during gestation, feed consumption was not measured, and the identity of each offspring within litters was not retained. Before expected parturition, each dam was housed in a polycarbonate cage that contained bedding. The date of parturition was noted and was termed Day 1 PP. The dams were weighed and examined for clinical signs. For each test group fertility index and gestation index were calculated, and for each litter viability index and lactation index were calculated. All dams were sacrificed on Day 22 PP.

The pups from each dam were counted, sexed, weighed, and examined for external alterations toward the end of Day 1 PP. Thereafter, each pup was weighed and inspected for adverse clinical signs on Day 4, 7, 14, and 22 PP. The eyes of the pups in all groups of Run I (Experiment II) were examined by an ophthalmologist on Days 15, 16, or 17 PP, shortly after the eyes opened. This examination was conducted with the exposure levels coded. On Day 35 PP, each pup was sacrificed and the eyes were fixed for possible future evaluation.

Data were analyzed by appropriate statistical methods.

GLP: Yes

Test Substance: Ammonium perfluorooctanoate, purity > 95%

Results: The actual mean concentrations achieved were 0 and approximately 0.14, 1.2, 9.9, and 21.0 mg/m$^3$ for the 0, 0.1, 1.0, 10.0, and 25.0 mg/m$^3$ exposure groups.

No effects were observed at 0.1 or 1.0 mg/m$^3$. None of the 21 dams exposed to ammonium perfluorooctanoate at 10.0 mg/m$^3$ died, but they showed similar clinical signs to a lesser degree than

that seen at the 25.0 mg/m$^3$ level. At 25 mg/m$^3$, ammonium perfluorooctanoate was overtly toxic to rats in that 5/24 did not survive to term, most of the survivors had wet abdomens; reddish-brown discoloration around the eyes, nose, and mouth; lethargy; decreased feed consumption and body weight gain during the exposure period; and an unkempt appearance.

Maternal liver weight changes among groups on a relative weight basis indicated that exposure to ammonium perfluorooctanoate at 10.0 mg/m$^3$ or greater resulted in significantly larger livers. This increase in liver weight occurred despite the significant decrease in body weight gain in these groups, which significantly reduced the relative liver weights of the pair-fed control groups.

No differences from control were observed in the mean number of implants, mean number of corpora lutea, or fetal death in any dose level tested.

Developmental toxicity was not demonstrated upon sacrifice of the dams on Day 21 of gestation at any concentration of ammonium perfluorooctanoate tested. Concentration-related embryo-fetal toxicity, expressed as decreased fetal weight, occurred only at 25.0 mg/m$^3$, which was overtly toxic to the dams. This decreased body weight persisted to Day 1 PP, but not to Day 4 PP.

On Days 15, 16, or 17 PP, coded examination of the pups' eyes *in vivo* from Run I did not reveal concentration-related malformations. In view of these negative results, similar *in vivo* examination of the eyes of the pups from Run II was not conducted.

Ammonium perfluorooctanoate did not demonstrate a unique hazard to the conceptus.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 881-81.

Staples, R. E. et al. (1984). Fundam. Appl. Toxicol., 4:429-440.

**Type:** **Developmental Toxicity in Rats**
Species/Strain: Rats/Crl:CD®(SD)BR
Sex/Number: Female/25/group (Experiment I), 12/group (Experiment II)
Route of Administration: Gavage
Exposure Period: Days 6-15 of Gestation

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Exposure Levels: 0, 100 mg/kg

Method: In a pretest, 2 non-pregnant female rats were administered ammonium perfluorooctanoate by gavage at 150 mg/kg/day or 100 mg/kg/day to determine the maximum dose that the dams could tolerate for the planned exposure period of 10 days.

For the definitive study, female rats were mated to males, and the day that spermatozoa were detected in vaginal lavage was designated as Day 1 of gestation (Day 1G).

For Experiment I, body weights, clinical signs, and feed consumption during gestation were recorded. The dams were coded from just before sacrifice until all maternal and fetal data were collected, and until all structural alterations noted among the fetuses were classified.

After sacrifice of the dams on Day 21G, gross pathologic changes were examined, liver weight was recorded, and reproductive status was determined. The number of corpora lutea and implantation sites were counted, and the number and position of all live, dead, and resorbed fetuses were recorded. All live and dead fetuses were weighed and sexed externally and internally, and the live fetuses were examined for external alterations. Approximately ½ of the fetuses of each litter that were alive when removed from the dam were examined for visceral alterations. In addition, all stunted or malformed fetuses were similarly examined. The heads of all fetuses examined for visceral alterations and a sufficient number of the remainder to total 2/3 of each litter were fixed and examined for head alterations. All fetuses, except the heads of those that were fixed, were examined for skeletal alterations.

For Experiment II, the procedures used until Day 21G were the same as for Experiment I, except that body weights were collected on different gestation days, feed consumption was not measured, and the identity of each offspring within litters was not retained. At least two days before expected parturition, each dam was housed in a polycarbonate cage with bedding. The date of parturition was noted, and was termed Day 1 PP. The dams were weighed and examined for clinical signs. For each test group fertility index and gestation index were calculated, and for each litter viability index and lactation index were calculated. All dams were sacrificed on Day 23 PP, without pathological examination.

The pups from each dam were counted, sexed, weighed, and examined for external alterations toward the end of Day 1 PP.

Thereafter, each pup was weighed and inspected for adverse clinical signs on Day 4, 7, 14, and 22 PP. The eyes of the pups in both groups were examined by an ophthalmologist between Days 27 and 31 PP. This examination was conducted with the exposure levels coded. On Day 35 PP, each pup was sacrificed and the eyes were fixed for histologic evaluation.

Data were analyzed by appropriate statistical methods.

GLP: Yes

Test Substance: Ammonium perfluorooctanoate, purity ≥ 95%

Results: In the pretest, one rat (weighing 278 g) dosed at 150 mg/kg showed severe clinical signs of toxicity by the 4th day and was found dead on the morning of the 5th day, by which time it had lost approximately 40 g body weight. Another rat (weighing 260 g) dosed at 150 mg/kg lost approximately 11 g by the 3rd day. After 5 days of dosing at 100 mg/kg, adverse clinical signs were not noted in 1 rat that lost approximately 6 g, and were minimal in the other rat that lost approximately 14 g. On this basis, the 100 mg/kg/day dosage level was judged to be the maximum that the dams could tolerate for the planned exposure period of 10 days.

In Experiments I and II, 5 of the 37 dams given ammonium perfluorooctanoate were found dead and 1 was sacrificed in a moribund state, as compared to 0 of the 37 control animals. During the dosing period, all but 1 of the dams that subsequently died had wet perineal areas and were lethargic. Two also had chromodacryorrhea and chromorhinorrhea. Among the remaining dams, 4 developed alopecia, 1 had lung noise, and 1 had diarrhea. In the control group, the only clinical sign noted was focal alopecia that developed in 1 dam.

From Days 6-15G, the treated group in Experiment I gained approximately 1/3 less than the control group, and during the post-treatment period (Days 16-21G), the body weight gain of the ammonium perfluorooctanoate-treated group significantly exceeded that of the control group. In Experiment II, the Day 16G body weight was not taken, therefore, Days 6-15 body weight gains were not calculated.

Feed consumption was measured only for Experiment I. During the dosing period, the treated group consumed significantly less feed than the control group. Feed consumption was similar to the control value in the post-exposure period.

Mean maternal liver weight for the treated group was increased, but the difference was not statistically significant. At sacrifice, 1 of the

dams given ammonium perfluorooctanoate was observed to have several red areas on the visceral surface of the median lobe of the liver.

In Experiment I, the maintenance of pregnancy, the incidence of resorptions, and fetal body weight were not adversely affected by ammonium perfluorooctanoate administration. Similarly, in Experiment II, no adverse effect on reproductive performance or on pup viability or growth was demonstrated.

The only embryo-fetal toxicity finding noted that could be compound-related was an increased incidence of fetuses with ossification sites on the first lumbar vertebrae versus the incidence in the control group. This difference in incidence was statistically significant only if analyzed by a one-tailed test. Its presence was probably a response to generalized stress evoked by the toxic state of the dams. The postpartum viability, growth rate, and development of the offspring from additional dams given ammonium perfluorooctanoate were not affected.

*In vivo* examination of pups' eyes between Days 27 and 31 PP revealed no compound-related alterations in the pups.

Ammonium perfluorooctanoate did not demonstrate a unique hazard to the conceptus.

Reference: DuPont Co. (1982). Unpublished Data, Haskell Laboratory Report No. 1-82.

**Miscellaneous**

**Type:** **Metabolism**

Species/strain: Female and male rats/Crl:CD®

Method: Female albino rats (7-8 weeks old and weighing approximately 200 g); pregnant primigravida females (weighing approximately 200 g); and male rats (approximately 8 weeks old and weighing approximately 250 g) were used. The rats were housed 2/cage and allowed food and water *ad libitum*.

Groups A-E rats were tested to measure blood organofluoride levels in female rats as a function of post-exposure time following oral administration and to investigate multiple versus single doses. Group A (21 female rats) was dosed with 25 mg/kg orally and 3 rats were sacrificed at ¼, ½, 1, 2, 4, 8, and 24 hours after dosing. Groups B and C (12 female rats/group) were orally dosed with 2.5 or 150 mg/kg, respectively, and 3 rats were sacrificed at ½, 2, 8, and 24 hours after dosing. Group D (21 female rats) was dosed

11 days with 25 mg/kg orally each day and 3 rats were sacrificed at ¼, ½, 2, 4, 8, 24, and 168 hours after the 11th dose. Group E (12 male rats) was dosed at 25 mg/kg orally and 3 rats were sacrificed at ½, 8, 24, and 168 hours after dosing.

Groups F-I rats were tested to measure blood organofluoride levels in female rats following inhalation exposure. Group F (24 female rats) was exposed to a single 6-hour exposure of 10 $mg/m^3$ and 3 rats were sacrificed at ¼, ½, 1, 2, 4, 8, 24, and 168 hours after exposure. Groups G and H (12 female rats/group) were exposed to a single 6-hour exposure of 1 $mg/m^3$ or 0.1 $mg/m^3$, respectively, and 3 rats were sacrificed at ½, 2, 8, and 24 hours after exposure. Group I (12 male rats) was exposed to a single 6-hour exposure of 10 $mg/m^3$ and 3 rats were sacrificed at ½, 2, 8, and 24 hours after exposure.

Groups J-N were tested to compare blood organofluoride levels in pregnant versus non-pregnant rats and to compare oral exposure versus inhalation exposure. Group J (12 pregnant female rats) was administered 25 mg/kg orally on gestation day 15 and 3 rats were sacrificed at ½, 2, 8, and 24 hours after dosing. Group K (6 pregnant female rats) was administered 25 mg/kg orally on gestation days 6-11 and 3 rats were sacrificed at ½ and 2 hours after the 6th dose. Group L (12 pregnant female rats) was administered 25 mg/kg orally on gestation days 6-15 and 3 rats were sacrificed at ½, 2, 8, and 24 hours after the 10th dose. Group M (6 pregnant female rats) was exposed via a single 6-hour inhalation exposure to 10 $mg/m^3$ on gestation day 15 and 3 rats were sacrificed at ½ and 2 hours after exposure. Group N (3 pregnant female rats) was exposed via inhalation, 6 hours/day, to 10 $mg/m^3$ on gestation days 6-15 and 3 rats were sacrificed at ½ hour after the 10th exposure.

Blood samples were obtained from each rat.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: The uptake and clearance of ammonium perfluorooctanoate from the blood of female rats following a single oral dose was rapid with the peak reached 1-2 hours post-treatment and with virtual total clearance by 24 hours. A dose-response was demonstrated with no apparent changes in blood organofluoride levels following multiple oral dosing. A slower clearance rate in male rats was demonstrated following a single oral dose.

A single 6-hour inhalation exposure resulted in peak blood levels within 1 hour after cessation of exposure. The test substance

rapidly cleared from the blood, and the number of exposures did not affect blood levels. Male rats cleared the compound much more slowly.

Pregnant and non-pregnant rats showed similar organofluoride blood levels following either oral or inhalation exposures.

The amount of ammonium perfluorooctanoate present as the straight chain isomer increases relative to the non-straight chain isomers as the time following ammonium perfluorooctanoate administration increases. This suggested that the non-straight chain isomers are cleared from the blood more rapidly than the straight chain isomer, or that a metabolite is present.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 593-81.

**Type:** **Metabolism**

Species/strain: Male rats/Crl:CD®
Male mice

Method: $^{14}$C-Ammonium perfluorooctanoate (0.5 µCi/mg) as an aqueous solution was administered by intragastric intubation to 8 young adult male rats and 6 adult male mice at a dose of 10 mg/kg. One control group (2 rats and 2 mice) was sacrificed at 24 hours and 1 control group (3 rats and 2 mice) was sacrificed at 96 hours. The treated group (3 rats and 2 mice) was dosed with cholestyramine (1000 mg/kg) 24 hours after dosing with the test substance and then sacrificed at 96 hours. After dosing, rats and mice were placed in individual glass metabolism chambers. Exhaled $^{14}$C was sampled at 24, 48, 72, and 96 hours. Feces and urine were collected. At sacrifice time, 1-2 mL of blood was removed from the mice and approximately 8 mL of blood was removed from the rats. Livers were removed, homogenized, weighed, and 0.5 g samples were oxidized and counted.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity approximately 100%

Results: Although a previous study (DuPont Report No. 828-81 – described below) indicated that cholestyramine resin could reduce the acute lethal effects of ammonium perfluorooctanoate in this study, there was no sign of enhanced elimination of $^{14}$C-ammonium perfluorooctanoate via feces, urine, or exhaled air. The differences between the 2 studies are most likely because of differences in absorption of the test substance. In the previous study, much of the dose was probably still in the gastrointestinal tract when cholestyramine was administered. The non-absorbed ammonium perfluorooctanoate then associated with cholestyramine, was removed before absorption could occur, and thus prevented the

acute lethal effects of ammonium perfluorooctanoate.

For both rats and mice, the primary route of excretion was urinary, followed by fecal and expired air.

After 96 hours, fecal elimination was nearly the same for both species; however, the mouse expired significantly more $^{14}C$ in the air than the rat. Most notably, the mouse excreted less in the urine than the rat. This difference in urinary excretion was reflected as a greater concentration in mouse liver.

Reference: DuPont Co. (1982). Unpublished Data, Haskell Laboratory Report No. 405-82.

Pastoor, T. P. et al. (1983). The Toxicologist, 3:82.

**Type:** **Metabolism**

Species/strain: Male and female rats
Male and female mice
Male and female hamsters
Male and female rabbits

Method: A male and female of each species received a single 10 mg/kg dose of $^{14}C$-ammonium perfluorooctanoate via intragastric intubation. The rats, mice, and hamsters were housed individually in glass metabolism units immediately after dosing. Expired $CO_2$, urine, and feces were collected at 12, 24, 48, 72, 96, and 120 hours after dosing. The male and female rabbits were individually housed in stainless steel metabolism cages immediately after dosing. Urine and feces were collected 24, 48, 72, 96, 120, 144, and 168 hours after dosing. Expired $CO_2$ was not collected. Blood was drawn from the rabbits 168 hours after dosing.

The rats, mice, and hamsters were all sacrificed 120 hours after dosing and blood was drawn. All animals were dissected with following tissues excised, weighed, and frozen: heart, lungs, liver, kidneys, spleen, testes or ovaries, brain, G. I. tract, and muscle, skin, and fat samples. The carcasses were then weighed and frozen. Metabolism units were washed and the cage washes were collected and refrigerated.

Urine, $CO_2$ samples, and cage washes were analyzed directly for radioactivity using a liquid scintillation counter. Blood, feces, tissues, organs, and homogenized carcass samples were analyzed for $^{14}C$ content by tissue oxidation using a tissue oxidizer and liquid scintillation counter. Rabbit carcasses were not analyzed for $^{14}C$ content.

GLP: No

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Test Substance: Ammonium perfluorooctanoate, purity 70%

Results: Substantial sex differences in rats and hamsters were observed in the excretion of $^{14}C$ activity following a single oral dose. The female rat and the male hamster excreted over 99% of the original $^{14}C$ activity by 120 hours after dosing, conversely the male rat and the female hamster excreted 39 and 60% of the original $^{14}C$ activity, respectively, by 120 hours post-dosing. Both sexes of rabbits excreted the $^{14}C$ activity as rapidly and completely as the female rat and the male hamster. The male and female mice excreted only 21% of the original $^{14}C$ activity by 120 hours post-dosing. The rapid excretors (female rat, male hamster, and male and female rabbits) contained negligible amounts of $^{14}C$ in organs and tissues at sacrifice. The slow excretors exhibited the highest $^{14}C$ concentrations in the blood and liver with substantial levels in the kidneys, lungs, and skin.

Reference: DuPont Co. (1982). Unpublished Data, Haskell Laboratory Report No. 62-82.

**Type:** **Placental Transfer**

Species/strain: Pregnant rats/strain not specified

Sex/Number: Females/6

Method: $^{14}C$-labeled ammonium perfluorooctanoate was labeled on the carbonyl position and had a specific activity of 0.5 μCi/mg. Water was used as the dosing vehicle. The pregnant female rats received a single 10 mg/kg dose of $^{14}C$-labeled ammonium perfluorooctanoate by gavage on the 19$^{th}$ day of gestation. The rats were individually placed in glass metabolism units immediately after dosing.

Two rats were sacrificed at each time interval of 2, 4, and 8 hours after dosing. Blood was drawn at sacrifice and refrigerated. The placentas, umbilical cords, and fetuses were then removed, dissected from each other, and weighed. The umbilical cords and placentas were individually placed into paper combustion cones and fetuses were frozen. Organs and tissues were also excised, weighed, and frozen. The carcasses were also weighed and frozen. Urine and fecal samples excreted between dosing and sacrifice were collected and frozen. The metabolism units were washed successively with dilute detergent, water, and acetone. The washes were collected and stored.

The placentas, umbilical cords, and fetuses were oxidized in their entirety. Samples of the maternal tissues, carcass, and feces were also oxidized and analyzed for $^{14}C$ activity by liquid scintillation counting. The urine and cage washes were analyzed directly by liquid scintillation counting.

| | |
|---|---|
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 70% |
| Results: | Placental transfer of $^{14}C$-labeled ammonium perfluorooctanoate was shown to occur after administration of a single oral dose of $^{14}C$-labeled ammonium perfluorooctanoate. The comparison of fetal levels of ammonium perfluorooctanoate at 2 and 4 hours relative to the concentrations observed in the maternal blood, placenta, and other organs revealed evidence of resistance to placental transfer of the test compound. However, by 4 hours, the fetal concentrations of ammonium perfluorooctanoate increased substantially more than all other organs and tissues examined. In contrast to other tissues examined, ammonium perfluorooctanoate in the fetuses did not decrease between 4-8 hours. The peak fetal ammonium perfluorooctanoate concentrations were similar in magnitude to the levels observed in the spleen, heart, lungs, and fat. Significant quantities of ammonium perfluorooctanoate can, therefore, be transferred from the placenta to the fetus with the placental barrier offering minimal resistance to transfer. |
| Reference: | DuPont Co. (1982). Unpublished Data, Haskell Laboratory Report No. 61-82. |
| **Type:** | **Effects of Dowex® Ion Exchange on the Toxicity of Ammonium Perfluorooctanoate in Rats** |
| Species/strain: | Male rats/Crl:CD® |
| Method: | A range-finding study was conducted prior to the test to find the tolerated dose of Dowex® 1-X2-C1 (cholestyramine). In that study, rats were dosed from 200-1000 mg/kg, 1 rat/dose level, and 6 rats dosed at 1000 mg/kg. No clinical signs of toxicity were noted in this study.<br><br>Ammonium perfluorooctanoate, as a suspension in corn oil, was administered by intragastric intubation to young adult male rats. Four groups, 6 rats/group, were used in the study. Group I was dosed with 500 mg/kg of ammonium perfluorooctanoate, Group II was dosed with 500 mg/kg of ammonium perfluorooctanoate and immediately dosed with 1000 mg/kg of Dowex® 1-X2-C1, Group III was dosed with 1000 mg/kg Dowex® 1-X2-C1 and 2 hours later dosed with 500 mg/kg of ammonium perfluorooctanoate, and Group IV was dosed with 500 mg/kg of ammonium perfluorooctanoate and 2 hours later dosed with 1000 mg/kg of Dowex® 1-X2-C1. All rats were weighed and observed over a 14 day recovery period and then sacrificed. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity approximately 100% |
| Results: | Pre-dosing or post-dosing with Dowex® 1-X2-C1 Ion Exchange Resin at 1000 mg/kg changes the toxic effects of ammonium |

perfluorooctanoate in rats. All rats dosed with the Dowex® 1-X2-C1, either before or after the ammomium perfluooctanoate had reduced mortalities compared to the rats dosed with ammomium perfluooctanoate alone. Mortality ratios were 5/6, 0/6, 1/6, and 0/6 for Groups I, II, III, and IV, respectively.

A follow-up study (DuPont Report No. 405-82 – described above) was performed to determine whether cholestyramine would enhance the elimination of absorbed ammonium perfluorooctanoate from the body.

Reference: DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 828-81.

**Type:** **Respirator Evaluation**

Method: The purpose was to evaluate M.S.A. Combination Type GMA-H respirator cartridges against ammonium perfluorooctanoate dust for breakthrough. The cartridge pairs were tested against an average concentration of 0.56 $mg/m^3$ ammonium perfluorooctanoate generated in a dust generator, and introduced into an air blow of 60 L/min maintained at 50% relative humidity and ambient room temperature. Both upstream and downstream airborne concentrations were monitored for ammonium perfluorooctanoate dust by filter paper cassettes backed up with an impinger sampling train to absorb possible ammonium perfluorooctanoate vapors. Aliquots of the aqueous extracts of the filter paper and aliquots of the aqueous impinger samples were analyzed colorimetrically for ammonium perfluorooctanoate.

GLP: No

Test Substance: Ammonium perfluorooctanoate, purity not specified

Results: After 40 hours of continuous exposure, no detectable amount of ammonium perfluorooctanoate dust or vapor was found downstream on the breathing side of the cartridge pairs tested. The minimum detectable limit was 1.5 µg of ammonium perfluorooctanoate. Based on a 420 L air sample, this would calculate to be 0.004 $mg/m^3$. The mean upstream airborne concentrations were $0.67 \pm 0.33$ and $0.48 \pm 0.15$ $mg/m^3$ at 40 and 54 hours, respectively. The results demonstrated that the M.S.S. combination type GMA-H cartridges effectively filter ammonium perfluorooctanoate dust and vapor concentrations at the test conditions for a minimum of 40 hours.

Reference: DuPont Co. (1980). Unpublished Data, Haskell Laboratory Report No. 664-80.

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| | |
|---|---|
| **Type:** | **Glove Permeation Testing** |
| Method: | Five types of gloves (neoprene; neo-synthetic latex rubber, floc lined; latex; natural latex; neoprene rubber) were evaluated for their permeation resistance to a 30% aqueous solution of ammonium perfluorooctanoate and ammonium perfluorooctanoate dry powder. Samples of the gloves were tested in duplicate in 10 mL glass permeation cells with water used as a collection medium on the inside surface of the gloves. A 10 mL aliquot of the collecting medium was analyzed for ammonium perfluorooctanoate by spectrophotometry after an 8-hour exposure. Breakthrough was determined when a detectable amount of ammonium perfluorooctanoate was found in the collection medium. The minimum detection limit for ammonium perfluorooctanoate was 1 µg/mL in the collection medium. |
| GLP: | No |
| Test Substance: | Ammonium perfluorooctanoate, purity not specified |
| Results: | Results of the evaluation show that 3 of the glove samples (neoprene; neo-synthetic later rubber, floc lined; and natural latex) do have a measurable breakthrough time and permeation rate after 8 hours of continuous exposure to a 30% solution of ammonium perfluorooctanoate. However, the permeation rates are very low indicating that the materials do offer some resistance to the test substance.<br><br>Results of the tests performed with ammonium perfluorooctanoate powder show that in 4 of the samples (neoprene; neo-synthetic latex rubber, floc lined; latex; and neoprene rubber) no breakthrough was observed after 8 hours of continuous exposure, and only a very small amount permeated the latex sample. |
| Reference: | DuPont Co. (1981). Unpublished Data, Haskell Laboratory Report No. 612-81. |
| **Type:** | **Biopersistence Screening Study** |
| | Summarized in the PFOS section, see DuPont (2000). Haskell Laboratory Report No. 2922. |

EID091570



000736

## SUMMARY OF STUDIES CONDUCTED WITH AMMONIUM PERFLUORONONANOATE AT DUPONT

### Mammalian Toxicity

#### Acute Toxicity

| | |
|---|---|
| **Type:** | **Oral ALD** |
| Species/strain: | Male rats/ChR-CD |
| Value: | 187 mg/kg |
| Method: | The test substance, as a solution in water, was administered by intragastric intubation to young adult male rats (1/group) in single doses. Concentrations tested were 1.0, 1.5, 2.3, 3.4, 5.1, 7.7, 12, 17, 26, 40, 60, 90, 130, 187, 300, 450, 670, 1000, 1500, and 2250 mg/kg. Survivors were sacrificed 14 days later, and body weights and liver weights were recorded. |
| GLP: | No |
| Test Substance: | Ammonium perfluorononanoate, purity not specified |
| Results: | Mortality was observed at concentrations of 187 mg/kg and above. Deaths occurred within 6 days after dosing. Slight initial weight losses occurred at 26 and 40 mg/kg. Weight loss occurred for 9, 12, and 15 days at 60, 90, and 130 mg/kg, respectively. No clinical signs were observed below 26 mg/kg. Clinical signs observed at 26 mg/kg and above included pallor, salivation, polyuria, and chewing-motions. Additional clinical signs observed at 187 mg/kg and above included belly-crawling, half-closed eyes, incoordination, ruffled fur, diarrhea, and emanciation. Increased liver weights and increased liver/body weight ratios occurred at 3.4 mg/kg and above. |
| Reference: | DuPont Co. (1968). Unpublished Data, Haskell Laboratory Report No. 129-68. |

| | |
|---|---|
| **Type:** | **Inhalation ALC** |
| Species/strain: | Male rats/Crl:CD®(SD)BR |
| Value: | 590 $mg/m^3$ |
| Method: | Male rats (6/group), 8 weeks old and weighing between 234 and 298 g, were exposed via nose-only inhalation for a single, 4-hour period to a dust atmosphere of ammonium perfluorononanoate in air. Concentrations tested were 620, 910, 1600, and 4600 $mg/m^3$. Dust atmospheres were generated with a bin feeder regulated with a volumetric feed controller. The bin feeder metered test substance into a glass transfer tube. Air introduced at the tube swept the test substance through a size-reducing cyclone and into the exposure chamber. For the lowest exposure concentration, the atmosphere was generated by passing pressurized air through a glass generator. A flask at the bottom of the generator served as a dust reservoir. A |

cyclone elutriator was inserted above the reservoir. A motorized stirring rod agitated dust in the generator. Air introduced at the bottom of the reservoir and at the cyclone elutriator swept dust particles into the exposure chamber. The atmospheric concentrations was determined at approximately 30 minute intervals by drawing calibrated volumes of chamber atmosphere through filters. The atmospheric concentration of particulate was determined from the filter weight differential before and after sampling. Chamber temperature, relative humidity, and chamber oxygen content were measured. Except during exposure, food and water were available *ad libitum*. Body weights and clinical signs were recorded. Survivors were sacrificed 14 days later.

To monitor the effects of ammonium perfluorononanoate on the liver, 2 groups of 10 rats, 8 weeks old and weighing between 237 and 277 g, were exposed to 67 and 590 mg/m$^3$, respectively. Two groups of 10 rats, 8 weeks old and weighing 231 and 267 g, were exposed to air only. Each control group was exposed concurrently with one of the test groups. Five rats/group were sacrificed 5 or 12 days after exposure for pathologic examination of the liver.

GLP: Yes

Test Substance: Ammonium perfluorononanoate, purity >99%

Results: Chamber temperatures ranged between 23-27°C, relative humidities ranged from 19-45%, and chamber oxygen contents were 21%.

One of the rats in the 590 mg/m$^3$ died on the 12$^{th}$ day of exposure. Mortality ratios of 0/6, 4/6, 6/6, and 6/6 were observed in the 620, 910, 1600, and 4600 mg/m$^3$. Clinical signs observed during or immediately post-exposure included red or brown facial discharge (67-1600 mg/m$^3$), test substance on the head (620, 1600, and 4600 mg/m$^3$), labored breathing (4600 mg/m$^3$), profuse clear nasal and oral discharges (4600 mg/m$^3$), and diminished startle response (4600 mg/m$^3$). No adverse clinical signs were observed in rats exposed to 67 mg/m$^3$ throughout the recovery period. Common clinical signs at higher concentrations included hunched posture, ruffled or discolored fur, red or brown facial discharges, wet or stained perineum, pallor, lung noise or labored breathing, lethargy, limpness, and hair loss.

Rats exposed to 67 mg/m$^3$ lost 1-9% of initial body weight 1 day post-exposure, followed by normal weight gain. At concentrations greater than 67 mg/m$^3$, rats lost approximately 6-15% of initial body weight 1 day post-exposure, and continued to lose weight either throughout the recovery period or until they died. Most

surviving rats exposed to 590, 620, or 910 mg/m$^3$ weighed only 54-71% of initial body weight when they were sacrificed 12 days post-exposure or at the end of the recovery period.

Rats exposed to 67 mg/m$^3$ had significantly elevated mean liver weights and liver-to-body weight ratios on the 5$^{th}$ and 12$^{th}$ days after exposure. Rats exposed to 590 mg/m$^3$ had significantly elevated liver-to-body weight ratios on the 12$^{th}$ day after exposure. Liver weights for these rats were not significantly different from the controls on the 5$^{th}$ day of recovery, and mean liver weights were significantly depressed on the 12$^{th}$ day of recovery. However, these seemingly inconsistent changes were due to severe body weight loss in rats exposed to 590 mg/m$^3$. Gross pathologic examination of rats exposed to 590 mg/m$^3$ revealed discolored livers with prominent lobular patterns in 4/5 rats sacrificed on the 5$^{th}$ day of recovery, and similar gross liver lesions in 2/5 rats sacrificed on the 12$^{th}$ day of recovery.

References: DuPont Co. (1985). Unpublished Data, Haskell Laboratory Report No. 293-85.

Kinney, L. A. et al. (1989). Food Chem. Toxicol., 27:465-468.

**Repeat Dose Toxicity**

| | |
|---|---|
| **Type:** | **Repeated Dose Oral Toxicity Study** |
| Species/Strain: | Mice/Crl:CD®-1(ICR)BR |
| Sex/Number: | Male and female/5/group |
| Exposure Period: | 14 days |
| Frequency of Treatment: | *Ad libitum* for 14 days |
| Exposure Levels: | 0, 1, 3, 10, 30, 100, 300, 1000, 3000, 10,000 mg/kg |
| Method: | Male and female mice (5-6 weeks old) were fed diets containing ammonium perfluorononanoate for 14 consecutive days. Verification of the concentration of the test substance in the prepared diets were performed. Body weights and clinical signs were recorded throughout the test. After sacrifice, liver weights were recorded. Body weights and liver weights were analyzed with appropriate statistical methods. Sacrifices occurred on test day 6 at concentrations of 1000 ppm and greater, on test day 8 at concentrations of 100 and 300 ppm, and on test day 14 for the remaining test groups. |
| GLP: | No |
| Test Substance: | Ammonium perfluorononanoate, purity 99% |

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000[illegible]

Results: Mortality ratios of 0/5, 0/5, 0/5, 3/5, and 1/5 were observed for the male mice fed 100, 300, 1000, 3000, and 10,000 ppm, respectively. Mortality ratios of 0/5, 3/5, 4/5, 5/5, and 3/5 were observed for the female mice fed 100, 300, 1000, 3000, and 10,000 ppm, respectively. Mice fed concentrations of 100 ppm or less exhibited slight to severe sporadic weight loss. Mice fed concentrations of 300 ppm and higher exhibited severe weight loss, ruffled fur, lethargy, low posture, and limpness.

Male and female mice fed diets of 3 ppm or higher had significantly increased mean absolute and mean relative liver weights. The mean relative liver weights of male mice fed the 1 ppm diet were also significantly heavier than the controls. Comparisons of mean body and liver weights of mice fed diets above 30 ppm were not possible because there were no concurrent control groups.

Reference: DuPont Co. (1985). Unpublished Data, Haskell Laboratory Report No. 401-85.

**Anthony J Playtis**
06/09/2000 02:59 PM

To: Paul J Bossert/AE/DuPont@DuPont, Robert L Ritchey/CL/DuPont@DuPont, Dawn D Jackson/CL/DuPont@DuPont, Oscar T Garza/AE/DuPont@DuPont

cc:

Subject: C-8 TLV

I checked the ACGIH TLV listing for ammonium perfluorooctanoate, and they do indeed give it an A3 notation, which means:

"*Confirmed Animal Carcinogen with Unknown Relevance to Humans:* The agent is carcinogenic in animals at a relatively high dose, by route(s) of administration, at site(s), of histologic type(s), or by mechanism(s) that may not be relevant to worker exposure. Available epidemiologic studies do not confirm an increased risk of cancer in exposed humans. Available evidence does not suggest that the agent is likely to cause cancer in humans except under uncommon or unlikely routes or levels of exposure."

RLR002852

000745

H David Ramsey
06/13/2000 02:48 PM

To: Dawn D Jackson/CL/DuPont@DuPont, Lynwood K Ireland/CL/DuPont@DuPont, Paul J Bossert/AE/DuPont@DuPont, Robert L Ritchey/CL/DuPont@DuPont, John S Sieg/EUR/DuPont@DuPont
cc:
Subject: Mr. Graham of Dry Run

---------- Forwarded by H David Ramsey/AE/DuPont on 06/13/2000 02:48 PM ----------

From: Bernard J Reilly on 06/13/2000 01:51 PM

To: H David Ramsey/AE/DuPont@DuPont, Ralph G Stahl_Jr/AE/DuPont@DuPont, Isidoros J Zanikos/AE/DuPont@DuPont, John R Bowman/AE/DuPont@DuPont@DuPont, Andrea V Malinowski/AE/DuPont@DuPont, Paula Durst-Gillis/STB/DUP@DUP, M. Ann Bradley/STB/DUP@DUP
cc:
Subject: Mr. Graham of Dry Run

---------- Forwarded by Bernard J Reilly/AE/DuPont on 06/13/2000 01:48 PM ----------

"Dr. Perry Habecker" <habecker@vet.upenn.edu> on 06/13/2000 12:58:06 PM

To: "Bob Poppenga" <poppenga, "Bob Munson" <munson, "GREG P. SYKES 6-6070" <SYKESGP cc: REILL2BJ
Subject: Mr. Graham of Dry Run

---

I just talked to Cyril Graham, the Jersey cow dairyman from the Dry Run area, about his concerns for potential toxicities. In short, he has already contracted a lab to analyze his cow□s milk. He said he was testing for cadmium, arsenic and several other items. When I said □heavy metal screen□ , he affirmed this. He prefers to wait to see what results this lab generates before contacting us. I offered him Bob Poppenga□s (sorry Bob) and my NBC phone numbers if he needs clarification or thinks he needs more testing. During a previous phone conversation, I had suggested that testing milk was probably not the most expeditious route to finding trouble. So, for the moment, we don□t need to talk with him.
Perry

- att1.htm

---------- Forwarded by H David Ramsey/AE/DuPont on 06/13/2000 02:48 PM ----------

From: Bernard J Reilly on 06/13/2000 02:04 PM

To: H David Ramsey/AE/DuPont@DuPont, Ralph G Stahl_Jr/AE/DuPont@DuPont, Isidoros J Zanikos/AE/DuPont@DuPont, John R Bowman/AE/DuPont@DuPont@DuPont, M. Ann Bradley/STB/DUP@DUP, Paula Durst-Gillis/STB/DUP@DUP, Greg P Sykes/RND/Pharma@DPC
cc:
Subject: RE: Mr. Graham of Dry Run

---------- Forwarded by Bernard J Reilly/AE/DuPont on 06/13/2000 02:00 PM ----------

"Dr. Perry Habecker" <habecker@vet.upenn.edu> on 06/13/2000 02:02:11 PM

To: REILL2BJ
Subject: RE: Mr. Graham of Dry Run

---

I should tell you that I cc'd that e-mail message to Sarah Caspar of the EPA regional office. I don't think that this will make for complications, but the cattle team had agreed a long time ago that we would notify her of relevant consultations with the WV locals.

-----Original Message-----
From: Bernard J Reilly [mailto:Bernard.J.Reilly@USA.dupont.com]
Sent: Tuesday, June 13, 2000 1:52 PM
To: Dr. Perry Habecker
Cc: GREG P. SYKES; Ralph G Stahl_Jr
Subject: Re: Mr. Graham of Dry Run

Perry,

Thank you for following up.

--Bernie

DDJ001517

EID086147

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June 23, 2000

Dr. Charles M. Auer, Director
U.S. Environmental Protection Agency
Office of Pollution Prevention and Toxics
Chemical Control Division
401 M Street NW, Room 403
Washington, D.C. 20460

Dear Dr. Auer:

As you requested in your April 19, 2000 letter and during our subsequent meeting on May 1, 2000, attached is a summary of DuPont's U.S. uses of Ammonium Perfluorooctanoate (APFO, CAS# 3825-26-1) as a fluoropolymer reaction aid including releases from DuPont site and the fate of APFO in fluoropolymer dispersion products, a summary of industrial hygiene data collected at our U.S. fluoropolymer manufacturing site, and a summary of the employee blood data from a site in the U.S. A summary of the toxicology available to DuPont was sent under separate cover on May 26, 2000.

It is important to emphasize the following points:

- DuPont does not manufacture APFO. All APFO used in our processes as a reaction aid is purchased from an outside supplier.

- Most of the APFO used is removed from the fluoropolymer products before they are sold to outside customers. A relatively small amount of APFO (worldwide, in the U.S.) leaves DuPont facilities in fluoropolymer dispersion products.

- Of the APFO in the products sold, most (>97%) is destroyed during customer processing to a non-carboxylated hydrofluorocarbon.

- All of the U.S. DuPont operations that use APFO with significant exposure potential are concentrated at one location; Washington Works in Washington, WV. Therefore, most of the industrial hygiene data and blood serum data presented in this document are from that location.

- Extensive industrial hygiene data collected on workers potentially exposed to APFO show airborne exposures to be significantly below the ACGIH TLV of 0.01mg/m$^3$ 8 hr. TWA. Exposure levels of plant workers have dropped significantly since the conversion to an APFO solution from a dry powder.

- As part of the ongoing surveillance of workers potentially exposed to APFO, in March and April of this year a series of blood samples were taken from workers in the U.S., The Netherlands and Japan to be analyzed for serum APFO concentration. DuPont has not received the results from our contract laboratory at this time. DuPont will submit a summary of the results when they become available.

The format of the information in the attached is a modified UEIP format. If you wish to discuss the information contained in the attachment, please contact Robert F. Pinchot at (302) 999-4074 or e-mail at Robert.F.Pinchot@usa.dupont.com or me at (302) 366-5259.

Very truly yours,

Gerald L. Kennedy
Director, Applied Toxicology
and Health

# Voluntary Use and Exposure Information Profile
# Ammonium Perfluorooctanoate (APFO)

## I. CHEMICAL IDENTIFICATION

Chemical Name: Ammonium Perfluorooctanoate
CAS Number: 3825-26-1

## II. COMPANY IDENTIFICATION

**Company Name:** E. I. du Pont de Nemours and Company

**Site Locations:**

Site where APFO is used as a reaction aid:

Washington Works
Route 892
Washington, WV 26181

Sites where APFO containing products made at Washington Works are processed:

Parlin Plant
Cheesequake Road
Parlin, NJ 08859

Spruance Plant
5401 Jefferson Davis Hwy.
Richmond, VA 23234

Site which disposes of waste containing APFO:

Chambers Works
Rte. 130
Deepwater, NJ 08023

**Technical Contact:** Robert F. Pinchot
(302) 999-4074
DuPont Fluoroproducts
Chestnut Run Plaza
Bldg. 711/2210
Centre Boulevard
Wilmington, DE 19805-0711

## III. DUPONT AND CUSTOMER ACTIVITIES

### Narrative Description of APFO Use

The block diagram on the back page titled "DuPont US APFO Balance" describes the processes discussed below.

DuPont uses APFO as a reaction aid in the production of polytetrafluoroethylene (PTFE) and tetrafluoroethylene (TFE) co-polymers. The process utilized at DuPont's Washington Works for making PTFE and co-polymers consists of polymerizing TFE (and other co-monomers if desired) in an aqueous media with a small amount of APFO to aid in the reaction.

Following the polymerization step, the polymer dispersion is either dried to remove water and APFO or concentrated (removing some of the APFO), stabilized and sold as an aqueous dispersion. The dried polymer contains very little, if any, APFO.

The APFO removed from the polymer is recovered for recycle, captured and destroyed off site in an incinerator, captured and sent to an offsite industrial landfill, and/or emitted to air or water at the Washington Works.

The stabilized polymer dispersions are sold by DuPont to industrial customers (both in the US and outside the US) for a variety of uses, internally transferred to the DuPont Spruance Plant for the production of Teflon® fibers and PTFE coated synthetic fibers, or internally transferred to the DuPont Parlin Plant for the production of Teflon® Finishes.

A small amount of non-hazardous waste polymer, water, APFO and other additives generated at Washington Works is treated in a wastewater treatment facility at DuPont's Chambers Works. This material is either emitted in the Chambers Works water discharge or captured on carbon and landfilled in a secure landfill.

The internal process at the DuPont Spurance Plant to produce Teflon® fibers involves, for most of the product, a "sintering" step in which the APFO contained in the product is destroyed by the following reaction:[1]

$$CF_3(CF_2)_6COO^-NH_4^+ \rightarrow CF_3(CF_2)_5CF_2H + CO_2 + NH_3$$

This reaction goes to completion at 350°C and 0.2s residence time. A small amount of product processed at DuPont's Spruance plant does not get sintered and thus contains a small amount of residual APFO. These products are used for industrial pump, valve and compressor packing materials.

[1] P.J. Krusic, D.C. Roe, "Thermal decomposition of C8 fluorinated surfactants and related materials studied by high temperature gas-phase $^{19}F$ NMR. A new Alternative to thermal gravimetric analysis, DuPont Internal Report.

The process for making Teflon® finishes at the DuPont Parlin Plant involves a blending operation of fluoropolymer dispersions with other additives including solvents, binders, and pigments. The small amount of APFO emissions to water from this facility is due to waste generated during product changeovers. Some of the fluoropolymer dispersion is processed at contract facilities where the material is dried at temperatures >350°C thus destroying the APFO according to the reaction above. This dried material is then incorporated into finishes products.

The final product produced is then sold to applicators that apply the product to a substrate (such as cookware) via automated spraying or rollercoating. Emissions of APFO from these operations consist of overspray that is either captured on filters and landfilled or absorbed into water resulting in a water emission. Product that is applied to the substrate is then typically "sintered" at temperatures approaching 800°F resulting in the removal of the APFO from the substrate and subsequent destruction according to the reaction above.

Customers of dispersion products use the material for a variety of applications. However, most applications involve a "sintering" step where the APFO is destroyed. There are a small number of applications where the customer heats the dispersion products to temperatures that allow the APFO to sublime resulting in air emissions. There are also a small number of applications where the customer's product is not heated resulting in the APFO staying with the product. These applications include industrial packings, and industrial filter fabrics.

## IV. SITE RELEASE AND TRANSFER INFORMATION FOR TRI CHEMICALS

Not applicable- APFO is not listed on the TRI

## V. SITE RELEASE AND TRANSFER INFORMATION FOR NON-TRI CHEMICALS

A. On-site Air Releases

| | Estimated Total Annual Releases ( lbs.1999) | | | |
|---|---|---|---|---|
| | Washington Works | Parlin | Spruance | Chambers Works |
| Fugitive | Negligible | 0 | 0 | 0 |
| Stack (Point Source) | 24000 | 0 | 0 | 0 |

Comments

Air emissions are estimated using engineering calculations and judgements and limited measurements of specific point sources conducted in the past.

B. On-site Water Releases

| | Estimated Total Annual Releases ( lbs.1999) | | | |
|---|---|---|---|---|
| | Washington Works | Parlin | Spruance | Chambers Works |
| Point Source | 55000 | 300 | 150 | 9500 |

Comments

Water emissions are estimated using engineering calculations and judgements and limited measurements of specific sources conducted in the past.

Washington Works emissions occur for approximately 350 days/yr while the other sites' emissions occur for 10-100 days/yr. Releases of APFO to the Ohio River from the DuPont Washington Works Plant were modeled using the Probabilistic Dilution Model (PDM Beta Version 4.0 Beta June 11, 1999, US EPA Office of Pollution Prevention and Toxics) and a constructed Microsoft® Excel spreadsheet model. APFO release data for 1996 were used in both modeling exercises.[2] The PDM indicated that APFO concentrations of 1.0 ug C-8/L would be exceeded about 50% of the time during the year. APFO concentrations of in the river would exceed 0.1 µg APFO/L 90% of the time during the year and 10 µg APFO/L about 2.2% of the time during the year.

Average annual APFO concentrations in the Ohio River calculated by using a Microsoft® Excel spreadsheet was 0.423 µg APFO/L. Modeled AFPO concentrations in the river ranged from a low of 0.199 µg APFO/L in March to a high of 0.965 µg APFOC-8/L in September, which correspond to high and low river flows, respectively. Average Ohio River flows and volume data calculated from the US Geological Survey was collected at the Belleville Dam and used in the spreadsheet model. The Belleville Dam is on the Ohio River 13 miles downstream of the Washington Works Plant. This river flow data is the closest location downstream from the plant where this type of information is available.

In 1999 a drinking water sample obtained from GE plastics, Washington WV, immediately downstream on the Ohio River from DuPont Washington Works showed 0.552µg/l APFO.

In addition samples obtained in January 2000 from three different wells at the Lubeck Public Service District, downstream of Washington Works on the Ohio River, showed 0.8µg/l, 0.44µg/l and 0.313 µg/l. APFO.

[2] W.R.Berti, Modeling releases of ammonium perfluorooctanoate into the Ohio River, DuPont Internal Report EMSE-054-00.

C. On-Site Land Releases

Chambers Works treats APFO containing waste in a wastewater treatment system. Engineering calculations and measurements indicate that approximately 30% of the APFO in the wastewater treated is absorbed on to a carbon media that is landfilled on site. These land releases are estimated to be 3900lb in 1999.

Prior operations have resulted in measurable APFO concentrations in three landfills operated by the Washington Works in West Virginia. At Letart[3] landfill surface water measurements in 1999 and 2000ytd range from 2.23µg/l to 3240µg/l with an average of 1392ug/l. Groundwater measurements taken during the same time period at Letart landfill range from 60.3µg/l to 17400µg/l with an average of 2537µg/l. At the "local landfill" the groundwater concentrations range from 0.046µg/l to 39µg/l with an average of 8.83µg/l. Surface water samples at the "local landfill" range from 0.54µg/l to 87µg/l with an average of 18.5µg/l. At Dry Run landfill there are limited measurements of groundwater and surface water with maximum concentrations in groundwater of 15µg/l and the maximum concentration in the permitted outfall has been 33µg/l.

In 1999 a RCRA Facility Investigation was completed for Washington Works and was submitted to EPA Region III in June 1999[4]. The report contains data on groundwater concentrations of APFO at Washington Works.

D. Transfers to Off-site Locations

Washington Works:

| | Estimated Total Annual Releases or Transfers (lb. 1999) |
|---|---|
| Incineration | 16000 |
| Wastewater treatment | 13400[5] |
| Underground Injection | 0 |
| Hazardous Waste Landfill | 2600 |
| Other landfill | 0 |
| Recycle or recovery | |

## IV. ON-SITE WORKPLACE EXPOSURE

A. Information on the Number of Employees Potentially Exposed

[3] Maps of the landfill locations and specific monitoring locations and results are available upon request.
[4] Report was submitted to Martin. T. Kotsch, Remedial Program Manager, EPA Region III, Philadelphia.
[5] This is the same material that was described above in paragraph 1 of section V.D.

The tables below describe the number of workers that may be exposed to APFO during their normal work activities for each of the three sites where APFO is used or APFO containing product is processed.

Washington Works

| Hours/Day | Days/yr | | | |
|---|---|---|---|---|
| | <10 | 10-100 | 100-250 | >250 |
| <0.25 | | | | |
| 0.25-1 | | 242 | | |
| 1-8 | | | | |
| >8 | | | | |

Routine worker activities that have potential for exposure:

- Handling raw material APFO
- Handling raw dispersions containing APFO
- Maintenance of polymerization reaction systems
- Polymer dryer operation and maintenance
- Packout of PTFE and co-polymer dispersion products
- Operation and maintenance of APFO recovery systems

Parlin Plant

| Hours/Day | Days/yr | | | |
|---|---|---|---|---|
| | <10 | 10-100 | 100-250 | >250 |
| <0.25 | | | | |
| 0.25-1 | | | 18 | |
| 1-8 | | | | |
| >8 | | | | |

Routine worker activities that have potential for exposure:

- Handling of PTFE and Co-polymer dispersion products
- Operation and maintenance of blending facilities
- Packout of finished product

Note that at no time is the material handled at the Parlin Plant at an elevated temperature where the APFO could sublime. Therefore there is little potential for exposure to airborne APFO at this facility. All exposure potential is through skin contact during handling of the polymer dispersion materials all of which contain <1% APFO with most containing <0.25% APFO.

Spruance Plant

| Hours/Day | Days/yr | | | |
|---|---|---|---|---|
| | <10 | 10-100 | 100-250 | >250 |
| <0.25 | | | | |
| 0.25-1 | | <10 | | |
| 1-8 | | | | |
| >8 | | | | |

Routine worker activities that have potential for exposure:

- Handling of PTFE and Co-polymer dispersion products
- Operation and maintenance of fiber coating facilities
- Operation and maintenance of sintering rolls
- Packaging of non-sintered product.

Note that the PTFE and co-polymer dispersion products used at the Spruance site contain <0.9% APFO with most containing approximately 0.3% APFO.

B. Information on the Exposure Levels of Washington Works Employees

Since most of the processing done in the US with APFO and APFO containing intermediates and products is done at Washington Works, DuPont's airborne industrial hygiene data is concentrated at that site. The limited measurements of airborne APFO concentrations at the other sites where APFO containing products are used have shown much lower levels (mostly non-detectable) levels of APFO. The data in the table below

reflect monitoring done over the last 5 years at Washington Works. The sample results are a combination of chemical operator and maintenance worker personal samples.

| Year | Sample Type | # of Samples | Minimum Concentration (mpb[6]) | Maximum Concentration (mpb) | Mean (mpb) | Standard Deviation |
|---|---|---|---|---|---|---|
| 1999 | Partial Shift (mostly 6-8 hours) | 100 | <0.01 | 0.58 | 0.061 | 0.151 |
| 1998 | | 83 | .001 | 0.78 | 0.103 | 0.145 |
| 1997 | | 100 | <0.01 | 2.4 | 0.146 | 0.378 |
| 1996 | | 73 | N/D | 0.29 | 0.055 | 0.069 |
| 1995 | | 32 | N/D | 0.16 | 0.067 | 0.063 |

Partial shift air samples are taken at the rate of 200 mL/min using a Tenax collection tube that has been pretreated with sodium hydroxide/ethylene glycol/methanol. The APFO is desorbed from the tubes using methanolic hydrogen chloride, which also serves as a derivatizing reagent, converting the APFO to its methyl ester. After workup, the methyl ester is quantified using a gas chromatograph equipped with an electron capture detector. The methyl ester of perfluorodecanoic acid is used as an internal standard, and at least three calibration samples are prepared to cover the concentration range of interest. Precision is estimated to be +/- 10% relative.

The data above show averages consistently below the AGCHI TLV of 0.01mg/m$^3$ with only a very few samples above the TLV. Where results are above or near to the TLV, the event is investigated and corrective action (additional personal protective equipment or engineering controls) to reduce the exposure levels is undertaken. Older data from the 1980's show higher levels of exposure. In the early 1990's Washington Works switched from receiving the APFO as a powder to receiving it as an aqueous solution. This change was done to reduce the potential for exposure during handling of the dry powder. It should be noted that in the 1997 time period, the site was starting up new APFO recovery facilities. Operating and maintenance difficulties associated with the start-up of these facilities may have contributed to the higher levels of APFO in the personal samples during that year.

Task specific monitoring data and wipe monitoring data exist. However these data are not indicative of employee exposure and are not presented here. These samples are taken to identify areas where additional exposure controls may be necessary.

Engineering controls to reduce exposure consist of the following:

- Reaction systems are closed systems with continuous ambient monitoring for monomer concentrations
- Ventilation systems are installed where airborne concentrations are significant
- The polymer dryers operate under negative pressure to contain APFO and other materials.
- Recovery systems are in place to reduce airborne emissions.

[6] mpb= moles per billion. 0.56mpb is equivalent to the ACGIH TLV of 0.01mg/m$^3$

Personal protective equipment that workers regularly wear consist of the following:

- Safety shoes and side-shield safety glasses in all areas.
- Impervious gloves when handling APFO solutions or aqueous dispersion products.
- Chemical protective coveralls and goggles or face shields when the possibility of splashes of APFO containing solutions is present.
- Airline respirators or cartridge respirators where monitoring has shown to have high exposure potential.

At Washington Works, blood serum levels of APFO have been measured since 1981. Prior measurements of blood fluoride levels have been taken prior to 1981 but are of limited value in assessing exposure to APFO. A summary of results of employees with identified APFO exposure potential the 1995, 1989-90, 1985,and 1984 volunteer sampling events is in the table below. Due to significant job assignment movement during this period of time, analysis of trends of data are difficult. The data in the table below prior to 1995 are for employees included in the 1995 sampling data so that comparisons of relative levels of APFO in blood serum can be compared. The entire data set of blood concentrations is available upon request.

| Year | # of Samples | Minimum Concentration (ppm) | Maximum Concentration (ppm) | Mean Concentration (ppm) |
|---|---|---|---|---|
| 1995 | 73 | 0.12 | 4.5 | 1.57 |
| 1989-90 | 23 | 0.4 | 8.5 | 3.13 |
| 1985 | 21 | 0.06[7] | 18[8] | 2.44 |
| 1984 | 19 | 0.07[7] | 24[8] | 3.82 |

[7] This individual was working in a job that has APFO exposure potential at the time of the sample.
[8] This individual consistently has had the highest blood concentration of APFO since APFO specific samples were taken. This employee left an APFO exposure potential assignment in 1991. In 1995 this employee's blood serum level was 4.4ppm.

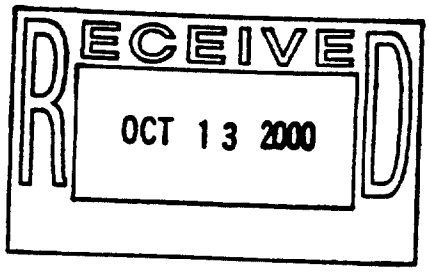
092000

8/15/00 LPSD Water Sample Analytical Results

(Letter, R. L. Ritchey to Mr. James C. Cox, LPSD dated 10/4/00)

BCC: A. V. Malinowski, Legal, D7078
M. A. Bradley, Spilman, Thomas, & Battle
P. J. Bossert /H. D. Ramsey/D. D. Jackson
I. J. Zanikos/A. S. Hartten
R. F. Pinchot
R. Banerjee/R. J. Zipfel

dsw / 2:817.2




EID090036

DuPont
Washington Works
P. O. Box 1217
Parkersburg, WV 26102-1217

October 4, 2000

Mr. James C. Cox
Lubeck Public Service District
P.O. Box 700
Washington, WV 26181

RE: LPSD Water Sample Analytical Results

Dear Mr. Cox:

Attached are copies of the analytical results for water samples taken on August 15, 2000 from three LPSD wells and the LPSD Building 1 main water line.

If you have any questions or need additional information, please contact me at 304-863-4271.

Sincerely,

R. L. Ritchey
Sr. Environmental Control Consultant
Washington Works

RLR/gw:dsw
Attachments
cc: William J. Gibbs
Lubeck Public Service District
P.O. Box 700
Washington, WV 26181

CI:009525

Lancaster Laboratories
Where quality is a science.



Lancaster Laboratories Sample No. WW 3439739

Collected:08/15/2000 13:40 by BJD

Account Number: 07032

Submitted: 08/16/2000 09:10
Reported: 09/12/00 at 09:28 PM
Discard: 10/13/00
WWK-G-LPWD-WELL_A Unspiked Water Sample
LUBECK WELLS 3Q00

CRG-E.I.DuPont de Nemours & Co
Barley Mill Plaza, Bldg. 27
Routes 141 & 48
Wilmington DE 19805

WELLA SDG#: XXX01-01BKG

| CAT No. | Analysis Name | CAS Number | As Received Result | As Received Method Detection Limit | Units | Dilution Factor |
|---|---|---|---|---|---|---|
| 02324 | FC-143 | 3825-26-1 | 0.535 | 0.029 | ug/l | 1 |

The FC-143 recovery is outside the QC limits for the LCS, MS, and MSD.
The sample was re-extracted past holding time with acceptable LCS recovery.
Original results were reported.

The 11H-PFUA and PFDDA surrogate recoveries are outside the QC limits.

## Laboratory Chronicle

| CAT No. | Analysis Name | Method | Trial# | Analysis Date and Time | Analyst | Dilut Fact |
|---|---|---|---|---|---|---|
| 02324 | FC-143 | FC-143 by GC/ECD 10/91 | 1 | 08/31/2000 21:05 | Rick Shober | 1 |
| 02325 | Pesticide Alternate Water Extr | FC-143 10/91 | 1 | 08/24/2000 08:00 | Deborah M. Zimmerman | 1 |

EID090038

000703

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425

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Where quality is a science.



Lancaster Laboratories Sample No. WW 3439740

Collected:08/15/2000 13:25 by BJD

Account Number: 07032

Submitted: 08/16/2000 09:10
Reported: 09/12/00 at 09:28 PM
Discard: 10/13/00
WWK-G-LPWD-WELL_B Water Sample
LUBECK WELLS 3Q00

CRG-E.I.DuPont de Nemours & Co
Barley Mill Plaza, Bldg. 27
Routes 141 & 48
Wilmington DE 19805

WELLB

| CAT No. | Analysis Name | CAS Number | As Received Result | As Received Method Detection Limit | Units | Dilution Factor |
|---|---|---|---|---|---|---|
| 02324 | FC-143 | 3825-26-1 | 0.159 | 0.029 | ug/l | 1 |

The FC-143 recovery is outside the QC limits for the LCS, MS, and MSD.
The sample was re-extracted past holding time to confirm original results.
The LCS recovery for the second extraction was within acceptance limits.
Similar results were obtained in both extracts. Original results were reported.
The 11H-PFUA and PFDDA surrogate recoveries are outside the QC limits.

## Laboratory Chronicle

| CAT No. | Analysis Name | Method | Trial# | Analysis Date and Time | Analyst | Dilu Fact |
|---|---|---|---|---|---|---|
| 02324 | FC-143 | FC-143 by GC/ECD 10/91 | 1 | 08/31/2000 22:37 | Rick Shober | 1 |
| 02325 | Pesticide Alternate Water Extr | FC-143 10/91 | 1 | 08/24/2000 08:00 | Deborah M. Zimmerman | 1 |

EID090039

000764

Lancaster Laboratories
Where quality is a science.



Lancaster Laboratories Sample No. WW 3439741

Collected:08/15/2000 13:15 by BJD

Account Number: 07032

Submitted: 08/16/2000 09:10
Reported: 09/12/00 at 09:28 PM
Discard: 10/13/00
WWK-G-LPWD-WELL_F Water Sample
LUBECK WELLS 3Q00

CRG-E.I.DuPont de Nemours & Co
Barley Mill Plaza, Bldg. 27
Routes 141 & 48
Wilmington DE 19805

WELLF

| CAT No. | Analysis Name | CAS Number | As Received Result | As Received Method Detection Limit | Units | Dilution Factor |
|---|---|---|---|---|---|---|
| 02324 | FC-143 | 3825-26-1 | 0.073 J | 0.029 | ug/l | 1 |

The FC-143 recovery is outside the QC limits for the LCS, MS, and MSD.
The sample was re-extracted past holding time to confirm original results.
The LCS recovery for the second extraction was within acceptance limits.
Similar results were obtained for both extracts. Original results were reported.

The 11H-PFUA and PFDDA surrogate recoveries are outside the QC limits.

## Laboratory Chronicle

| CAT No. | Analysis Name | Method | Trial# | Analysis Date and Time | Analyst | Dilu Fac |
|---|---|---|---|---|---|---|
| 02324 | FC-143 | FC-143 by GC/ECD 10/91 | 1 | 08/31/2000 23:08 | Rick Shober | 1 |
| 02325 | Pesticide Alternate Water Extr | FC-143 10/91 | 1 | 08/24/2000 08:00 | Deborah M. Zimmerman | 1 |

EID090040

000785



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Where quality is a science.



Lancaster Laboratories Sample No. WW 3439738

Collected:08/15/2000 12:45 by BJD
through 08/15/2000 12:45
Submitted: 08/16/2000 09:10
Reported: 09/14/00 at 08:54 AM
Discard: 10/15/00
WWK-D-BUILDING-1_MAIN Water Sample
LUBECK WELLS 3Q00

Account Number: 07032

CRG-E.I.DuPont de Nemours & Co
Barley Mill Plaza, Bldg. 27
Routes 141 & 48
Wilmington DE 19805

BBAAA

| CAT No. | Analysis Name | CAS Number | As Received Result | As Received Method Detection Limit | Units | Dilution Factor |
|---|---|---|---|---|---|---|
| 02324 | FC-143 | 3825-26-1 | 0.589 | 0.029 | ug/l | 1 |

The FC-143 recovery is outside the QC limits for the LCS, MS, and MSD. Since there was insufficient sample amount for a reextraction, the results were reported.

The 11H-PFUA and PFDDA surrogate recoveries are outside the QC limits.

## Laboratory Chronicle

| CAT No. | Analysis Name | Method | Trial# | Analysis Date and Time | Analyst | Dilu Fac |
|---|---|---|---|---|---|---|
| 02324 | FC-143 | FC-143 by GC/ECD 10/91 | 1 | 08/31/2000 20:34 | Rick Shober | 1 |
| 02325 | Pesticide Alternate Water Extr | FC-143 10/91 | 1 | 08/24/2000 08:00 | Deborah M. Zimmerman | 1 |

EID090041

000784

000767

CHEMICALS

- PFOS - or "Scotchgard"

# 3M'S BIG CLEANUP

NOTICE:
THIS MATERIAL MAY BE PROTECTED BY COPYRIGHT LAW (TITLE 17 U.S. CODE)

## Why it decided to pull the plug on its best-selling stain repellant

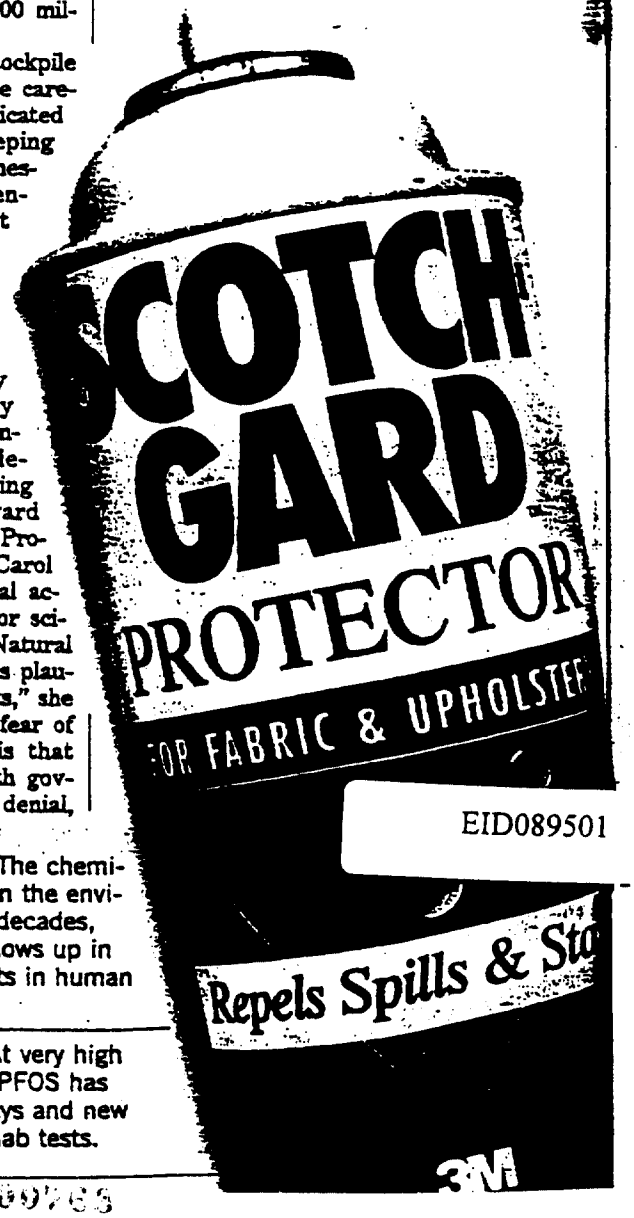
At first, Dr. Larry R. Zobel and other researchers at 3M Co. figured the findings had to be a mistake. Investigators from Cornell University, using a powerful new technique to scan the blood of 3M's factory workers, were testing some serum they bought from commercial blood banks to establish base lines for the machines. The highly sensitive devices kept turning up the same odd result: Tiny amounts of a chemical 3M had made for nearly 40 years were showing up in blood drawn from people living all across the country, even in places far from 3M factories. "It took months before all the chemists were convinced that it was there," recalls Zobel, 3M's medical director. "There was disbelief."

For 3M, the late 1997 test results were troubling. If they held up, it meant that virtually all Americans—and folks far beyond the U.S.—may be carrying some minuscule amount of a 3M chemical, called perfluorooctane sulfonate (PFOS), in their systems. How PFOS got there, whether it could pose a health risk and, more important, what should be done about it, were questions that 3M executives felt driven to ask. Although they have yet to come up with definitive answers—and they insist that there's no evidence of danger to humans—the Minnesota company's research led it to a drastic decision. On May 16, 3M decided to phase out PFOS and products containing related chemicals, first and foremost among them its ubiquitous Scotchgard fabric protector. Since there is no replacement chemical as yet, that means a potential loss of $500 million in annual sales, out of total corporate revenues of $16 billion, and one-time restructuring charges of up to $200 million.

The news caused a rush to stockpile Scotchgard by the clean and the careless. Heloise, author of the syndicated "Hints from Heloise" housekeeping column, says everyone from clothes-conscious TV hosts to flight attendants have fretted to her about the loss of Scotchgard. Even she, an expert at stain-removal, admits: "When my husband gets a new tie I spray it on right away."

**"GUTS."** Scotchgard's popularity makes its removal particularly noteworthy, given that 3M was under no mandate to act. "3M deserves great credit for identifying this problem and coming forward voluntarily," says Environmental Protection Agency Administrator Carol M. Browner. Even environmental activists like Linda E. Greer, senior scientist at the Washington-based Natural Resources Defense Council, gives plaudits to the company. "It took guts," she says, even if they did it out of fear of government action. "The fact is that most companies, when faced with government nudging, go into anger, denial, and the rest of that stuff. What we are accustomed to seeing is decades-long arguments about whether a chemical is really toxic."

The long and tortuous trail that led 3M to its decision highlights the growing concern within the chemical industry over persistent chemicals, a relatively recent environmental worry. There are scores of these chemicals, woven into the very fabric of modern life, that resist natural processes of decay and can linger in the environment for decades. Some have already been banned—most prominently DDT, PCBs, and CFCs—but no one is quite certain what damage, if any, the rest might be causing. The




### THE PROBLEM WITH SCOTCHGARD

*It contains POSF, an organic fluorine that repels water and oil. It can turn into a second fluorine, PFOS, when it gets into mammalian cells.*

▶ **DANGER** The chemicals persist in the environment for decades, and PFOS shows up in trace amounts in human blood..

▶ **EFFECT** At very high daily doses, PFOS has killed monkeys and new born rats in lab tests.

LAYNE KENNEDY

EPA is pressing the chemical industry to reduce the manufacture of these substances, however, and is currently negotiating international conventions with Canada aimed at reducing their presence in the Great Lakes.

PFOS is not on environmentalists' list of the most worrisome persistent chemicals. But 3M, which is responsible for most of the world's supply of the substance, decided not to wait until all the scientific cards fell into place. It took its costly step based on scientific detective work that has built up incrementally over the past 30 years—and far more damningly in the past two years. Its choice threatens the jobs of some 1,500 3M workers in Alabama, Minnesota, even Belgium. It also discomfits scores of industrial customers, chiefly papermakers and textile mills, who apply the 3M chemicals to goods as far-ranging as pet-food bags, candy wrappers, and carpeting.

**BLOOD TESTS.** The first big twist in the tale took place in 1968, with a physician at the University of Rochester. Dr. Donald R. Taves was studying the effects of water fluoridation when he found tiny quantities of an unusual form of fluorine—a kind that didn't come from fluoridated water—in human blood. Just how it got there and what its presence meant were unclear. But scientists familiar with the organic fluorines, known as POSF, took note. The finding drew still more attention when Taves, working with colleagues at the University of Florida, confirmed the results in a 1976 study. Chillingly, Taves even found the fluorine in his own blood, though he lived far from any potential factory source.

Researchers at 3M, which says it is vigilant about chemical exposure of its workers, were paying particular attention. They quickly launched programs to test employees at plants in Cottage Grove (Minn.), Decatur (Ala.), and later Antwerp (Belgium), to see if fluorine exposures were high. While they found that fluorines were registering at higher levels in workers' blood than in the general population, the medical evidence suggested that they posed no problem. Researchers found no unusual ailments in the living workers and, after scanning death records, nothing unusual in the deaths of former workers. "Physicians who were seeing the employees generally found no significant health problems, no health problems that would be unexpected in a typical population like this," says Zobel.

For 3M veterans, the cheerful medical results were not surprising. Although unusually hardy, organic fluorines for decades were thought to be inert. PFOS, a 3M product used in fire-fighting foams and industrial acid-suppression products, is also produced in animals and humans when certain precursor chemicals get into the cells. These precursors are part of the chemical makeup of Scotchgard fabric protector and are valued precisely for their hardiness. They repel water and oil like nothing else, making them potent stain-resisters. Indeed, 3M has long revered the in-house scientists who developed the man-made chemicals, after a lab staffer in the early 1950s chanced on them. The staffer had spilled one of the chemicals on her sneaker, found it impossible to wash out with either water or solvents, and started investigating its repelling powers.

While routine employee monitoring continued throughout the 1980s, the company had little reason to worry. Even when blood tests grew more sophisticated in the early 1990s—and 3M could screen for specific fluorines such as PFOS or related chemicals—the lack of any damning health effects kept concerns to a minimum. Repeated medical reviews seemed to clear the chemicals of any problems.

Then, in 1997, powerful new detection techniques changed everything. The new tests turned up evidence of the chemicals in levels as low as 0.5 parts per million in human blood. "That is not much," says Zobel drily. "It's like 50 seconds in 32 years." It was by using the new techniques that the Cornell University lab, working for 3M, found the PFOS in blood from the scattered blood banks. 3M promptly launched an international testing program, screening blood from 18 U.S. blood banks along with samples from Europe and Asia. It even scanned old, stored blood samples from Korean War veterans. The result: tiny amounts of contamination in the U.S. and Europe, except in the veterans' blood, which predated the Scotchgard chemical.

3M regularly updated the EPA on all of its research. For their part, EPA officials were particularly concerned by the persistence of PFOS. It is so hardy that no one knows when, if ever, it will break down. Worse, it accumulates in human and animal tissues. "With things that are persistent, the only way for

## THE PATH TO A PAINFUL CHOICE

**1968** A researcher at the University of Rochester finds organic fluorine in human blood samples from the general population. Publishes finding in *Nature*.

**1976** Academic researchers refine the earlier find, and the Rochester investigator, D.R. Taves, discovers the chemical in his own blood.

**1978** 3M reviews 30 years of death records among factory workers exposed to organic fluorine on the job but finds nothing unusual.

**EARLY 1990s** 3M uses enhanced mass spectrometry to scan workers' blood. Reliably detects contamination down to the level of 0.5 parts per million.

**1993** Researchers at University of Minnesota report finding no increased mortality in workers exposed to an organic fluorine. Elsewhere, it is linked to cancer in rodents and changes in reproductive hormones in humans.

**1996** University of Minnesota researchers publish study finding no toxicity to the liver in 115 3M workers from PFOS exposures.

**1997** Researchers at Michigan State University and 3M report that organic fluorine chemicals are appearing in water, air, and soil.

**MAY, 1998** 3M advises EPA that it found organic fluorine in blood-bank samples in tiny amounts. Company decides to move away from this chemistry.

**SEPTEMBER, 1998** Company tells EPA of disturbing animal-test findings. The offspring of rats heavily dosed with organic fluorine die within days.

**SEPTEMBER, 1999** 3M researchers find no adverse health effects from on-the-job exposure to PFOS.

**FEBRUARY, 2000** Researchers alert 3M that PFOS is found in tissue of birds from the Pacific Ocean to the Baltic.

**MARCH, 2000** 3M and EPA discuss latest findings, including the deaths of heavily dosed monkeys.

**MAY 16, 2000** 3M and EPA announce that the company will voluntarily phase out the organic fluorines.

DATA: 3M, EPA, BUSINESS WEEK

# "3M deserves great credit for identifying this problem and coming forward voluntarily," says the EPA's Browner

the concentrations to go is up—in our bodies and in wildlife," says the Natural Resources Defense Council's Greer. "And pretty much every chemical in the world is toxic at some dose."

Because PFOS is both enduring and widespread, 3M made some crucial choices in mid-1998. Company executives decided they would—eventually—abandon such formulations and find replacements for the troublesome fluorines. "We began to realize then, and came to realize more later, that this would be a chemical that would constantly be involved in environmental debate," says Charles Reich, a chemist in charge of Scotchgard as the company's executive vice-president for specialty-material markets.

**NO SURE THING.** Still, 3M's top executives argued over just how long they could continue. Could they wait until replacements for the chemicals were found? That might take seven years or more, and was not a sure thing. And what about the effect on corporate customers, who valued the 3M products for their ability to repel water or oil? Shouldn't 3M keep the supplies coming, since there was no hint of human danger. "All the data we had on health...pointed toward no health effect," says William E. Coyne, senior vice-president for research and development.

But then 3M took a couple of crucial steps that sped up its decision to chuck the product: It ordered up studies on rats, monkeys, and other animals to see what heavy doses of PFOS might do. And it commissioned more research, forking over $800,000 for an investigator at Michigan State University to test wildlife samples to see just how pervasive the chemicals were. The results of both sets of tests, though preliminary, proved fatal for the chemicals.

The wildlife tests confirmed the initial fears of the 3M executives. Michigan State University researcher John P. Giesy, a zoologist and a faculty member of the school's National Food Safety & Toxicology Center, found PFOS in some very odd places. Scanning samples of animal tissue from all across the globe, which he keeps on hand in lab freezers, Giesy found the chemical in "various animals from various places." While he won't list the animals or locations—for fear of preempting scientific publication—3M executives say PFOS turned up in flesh-eating birds in the Pacific Ocean and the Baltic regions. Giesy is now halfway through tests on 2,000 tissue samples drawn from as far away as the Arctic and Antarctic.

**REICH AND COYNE:** *3M data showed no adverse health effects*

When Giesy started presenting his initial results to 3M officials in February, the concern level at the company rose sharply. He was summoned back to repeat his presentation for Chief Executive Officer Livio D. DeSimone and his most senior executives. "This was very important to 3M," recalls the researcher.

Since neither Giesy nor in-house scientists could say just how PFOS got into the far-flung animal tissues, the 3M scientists now plan a global research effort aimed at tracking the chemical's sources and destinations. Says Katherine E. Reed, a chemist who is 3M's executive director of environmental technology: "We believe that our responsibility for materials continues...into disposal. It's a concept we call life-cycle management."

The most damning evidence against PFOS began emerging in September, 1998. That's when 3M got the results of animal tests in which heavy doses of PFOS were administered to rats—10,000 to 100,000 times what humans would ever likely be exposed to. The investigators found that the rats' offspring were dying within days of birth. Then, this past March, they reported that two monkeys that were also dosed heavily died, after suffering severe gastrointestinal problems and convulsions. The company promptly notified the EPA of the results.

Those animal tests got the agency's attention. Although 3M had kept the EPA informed of all its PFOS studies, the animal mortality data—and especially the rat fatalities—sparked a steady series of "no lunch, no refreshments...roll-up-your-sleeves" meetings in March at EPA headquarters in Washington, one top EPA official recalls. Says the official: "The seriousness of the issues grew over time."

When the 3M executives finally decided to pull the plug on PFOS and related products, they didn't need the heavy hammer of an EPA ruling—but that possibility hung heavily in the background. "They could see the writing on the wall," argues the senior EPA official. "They could see we were going to continue our assessment of this and it would get more detailed and at the end of the day we would make some kind of decision...3M decided that the better course of action was to get out of it early." For their part, 3M officials say they would have made the same decision whether the EPA pushed them or not.

Just how quickly 3M can adapt to its far-reaching decision isn't clear. It intends to be out of production on most, if not all, PFOS-related products by yearend. With a few products, such as fire-fighting foams, it may take longer to adjust. With over $1 billion pouring in each year from all sorts of new products that don't use the chemicals, the company feels sure it can come up with substitutes—ideally, nonpersistent ones. And it expects it will find jobs elsewhere or will pay separation benefits to affected workers. Meanwhile, the worries about PFOS are not yet over. For years to come, 3M will be keeping track of the stuff it has already put out there.

*By Joseph Weber in St. Paul, Minn.*

LAYNE KENNEDY

000771

# TEFLON® NON-SALABLE PROCESS WASTE DISPOSAL.

## BACKGROUND

Waste disposal at the DuPont Letart Landfill will cease effective January 1, 1996. This landfill had been the disposal point for TEFLON®'s process waste (excluding RCRA hazardous waste). The process waste that TEFLON® is sending to Letart can generally be classified as C-8 contaminated waste and non-C-8 waste. Since the Letart Landfill is closing, alternate disposal sites will have to be used for TEFLON®'s process waste. A portion of TEFLON®'s process waste is currently sold as scrap, however, not all of the process waste is salable.

For the TEFLON® Division at Washington Works, the C-8 contaminated process waste will be sent to a hazardous waste landfill in Emelle, Alabama. It is anticipated that the majority of TEFLON®'s C-8 contaminated waste that is sent to to Emelle will be shipped in 55-gallon steel drums. The estimated cost to send process waste to Emelle is $145 per drum.

Process waste that does not contain C-8 will be sent to DuPont's Dry Run Landfill for disposal. The Dry Run Landfill is not permitted for disposal of waste in steel or plastic drums; therefore, process waste sent to Dry Run will have to be shipped in fiber containers. It is estimated that the disposal cost at Dry Run will be $15-$25 per drum.

**THIS PROCEDURE DOES NOT CHANGE THE DISPOSAL PROCESS FOR RCRA HAZARDOUS WASTE.** Consult the TEFLON® Waste Disposal Manual (#30) for disposal of RCRA hazardous waste.

## PROCEDURE

The process waste has been tabulated in the attached tables. Some of the material in the tables might currently be sold as scrap. Areas should continue to sell scrap where possible. However, if the scrap cannot be sold, it should be properly disposed. If there are questions as to how a particular waste should be disposed, contact the area shift supervisor on duty. If additional assistance is still needed, contact our environmental coordinator. (Today, this is Keith Coffman, X4370). **IT IS EXTREMELY IMPORTANT THAT NO C-8 CONTAMINATED WASTE BE SENT TO DRY RUN LANDFILL.**

### Packaging of C-8 Contaminated Process Waste - Emelle Landfill

1. Waste will be packaged in a red 55-gallon steel drum. Drums will be available at the TEFLON® Pad northwest of B-164 (see attached B-164 West Pad diagram). Contact Polly Shackelford if additional drums are needed. **EXCEPT IN THE CASE OF NO AVAILABLE SUPPLY, TEFLON® WILL NO LONGER USE BLACK STEEL DRUMS FOR WASTE DISPOSAL.**



* Du Pont's registered trademark

CONTROLLED DOCUMENT IF RED

ATTACHMENT 122

is TSCA CBI.
DCN 6301000014
No Sanitized Version
was provided.

## PART 801—LABELING

1. The authority citation for 21 CFR part 801 continues to read as follows:

Authority: 21 U.S.C. 321, 331, 351, 352, 360i, 360j, 371, 374.

2. Section 801.430 is amended by revising the table in paragraph (e)(1) to read as follows:

**§ 801.430 User labeling for menstrual tampons.**

* * * * *

(e) * * *

(1) * * *

| Ranges of absorbency in grams[1] | Corresponding term of absorbency |
|---|---|
| 6 and under | Light absorbency. |
| 6 to 9 | Regular absorbency. |
| 9 to 12 | Super absorbency. |
| 12 to 15 | Super plus absorbency. |
| 15 to 18 | Ultra absorbency. |
| Above 18 | No term. |

[1] These ranges are defined, respectively, as follows: Less than or equal to 6 grams (g); greater than 6 g up to and including 9 g; greater than g up to and including 12 g; greater than 12 g up to and including 15 g; greater than 15 g up to and including 18 g; and greater than 18 g.

* * * * *

Dated: October 2, 2000.

**Margaret M. Dotzel,**

*Associate Commissioner for Policy.*

[FR Doc. 00–26249 Filed 10–17–00; 8:45 am]

BILLING CODE 4160–01–F

## ENVIRONMENTAL PROTECTION AGENCY

### 40 CFR Parts 52 and 81

**[MO 114–1114; FRL–6885–7]**

### Approval and Promulgation of Implementation Plans; State of Missouri; Designation of Areas for Air Quality Planning Purposes; Dent Township

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Proposed rule.

**SUMMARY:** EPA proposes to approve a State Implementation Plan (SIP) revision submitted by the state of Missouri and Missouri's request to redesignate the lead nonattainment area in western Iron County, Missouri, to attainment of the National Ambient Air Quality Standards (NAAQS). EPA proposes to approve the maintenance plan for this area including a consent order which was submitted with the redesignation request, and also proposes to approve the revision to Missouri's Restriction of Emissions of Lead From Specific Lead Smelter-Refinery Installations rule which ensures the permanent and enforceable emission reductions by clarifying the emissions limits for the Doe Run Resource Recycling Facility, and removes the text which could have allowed this facility to resume operation as a primary smelter.

In the final rules section of today's **Federal Register**, EPA is approving the state's SIP revision and redesignation request as a direct final rule without prior proposal because the Agency views this as a noncontroversial action and anticipates no relevant adverse comments to this action. A detailed rationale for the approval is set forth in the direct final rule. If no relevant adverse comments are received in response to this action, no further activity is contemplated in relation to this action. If EPA receives relevant adverse comments, the direct final rule will be withdrawn and all public comments received will be addressed in a subsequent final rule based on this proposed action. EPA will not institute a second comment period on this action. Any parties interested in commenting on this action should do so at this time.

**DATES:** Comments on this proposed action must be received in writing by November 17, 2000.

**ADDRESSES:** Comments may be mailed to Kim Johnson, Environmental Protection Agency, Air Planning and Development Branch, 901 North 5th Street, Kansas City, Kansas 66101.

**FOR FURTHER INFORMATION CONTACT:** Kim Johnson at (913) 551–7975.

**SUPPLEMENTARY INFORMATION:** See the information provided in the direct final rule which is located in the rules section of today's **Federal Register**.

Dated: September 27, 2000.

**Dennis Grams,**

*Regional Administrator, Region 7.*

[FR Doc. 00–26502 Filed 10–17–00; 8:45 am]

BILLING CODE 6560–50–P

## ENVIRONMENTAL PROTECTION AGENCY

### 40 CFR Part 721

**OPPTS–50639; FRL–6745–5**

**RIN 2070–AD43**

### Perfluorooctyl Sulfonates; Proposed Significant New Use Rule

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Proposed rule.

**SUMMARY:** EPA is proposing a significant new use rule (SNUR) under section 5(a)(2) of the Toxic Substances Control Act (TSCA) for the following chemical substances: Perfluorooctanesulfonic acid (PFOSA) and certain of its salts (PFOSS), perfluorooctanesulfonyl fluoride (PFOSF), certain higher and lower homologues of PFOSA and PFOSF, and certain other chemical substances, including polymers, that contain PFOSA and its homologues as substructures. All of these chemical substances are referred to collectively in this proposed rule as perfluorooctyl sulfonates, or PFOS. This proposed rule would require manufacturers and importers to notify EPA at least 90 days before commencing the manufacture or import of these chemical substances for the significant new uses described in this document. EPA believes that this action is necessary because the chemical substances included in this proposed rule may be hazardous to human health and the environment. The required notice would provide EPA with the opportunity to evaluate an intended new use and associated activities and, if necessary, to prohibit or limit that activity before it occurs.

**DATES:** Comments, identified by the docket number OPPTS–50639, are due November 17, 2000.

**ADDRESSES:** Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**. To ensure proper receipt by EPA, it is imperative that you identify docket control number OPPTS–50639 in the subject line on the first page of your response.

**FOR FURTHER INFORMATION CONTACT:** *For general information contact*: Barbara Cunningham, Director, Office of Program Management and Evaluation, Office of Pollution Prevention and Toxics (7401), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (202) 554–1404; e-mail address: TSCA-Hotline@epa.gov.

*For technical information contact*: Mary Dominiak, Chemical Control Division (7405), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (202) 260–7768; fax number: (202) 260–1096; e-mail address: dominiak.mary@epa.gov.

**SUPPLEMENTARY INFORMATION:**

## I. General Information

### *A. Does this Notice Apply to Me?*

You may be affected by this action if you manufacture (defined by statute to include import) any of the chemical substances that are listed in Table 2 or Table 3 of this unit. Persons who intend to import any chemical substance governed by a final SNUR are subject to the TSCA section 13 (15 U.S.C. 2612) import certification requirements, and to the regulations codified at 19 CFR 12.118 through 12.127 and 12.728. Those persons must certify that they are in compliance with the SNUR requirements. The EPA policy in support of import certification appears at 40 CFR part 707, subpart B. In addition, any persons who export or intend to export any of the chemical substances listed in Table 2 or Table 3 of this unit are subject to the export notification provisions of TSCA section 12(b) (15 U.S.C. 2611(b)), and must comply with the export notification requirements in 40 CFR 721.20 and 40 CFR part 707, subpart D. Entities potentially affected by the SNUR requirements in this proposed rule may include, but are not limited to:

TABLE 1.—ENTITIES POTENTIALLY AFFECTED BY THE SNUR REQUIREMENTS

| Categories | NAICS codes | Examples of potentially affected entities |
|---|---|---|
| Chemical manufacturers or importers | 325 | Persons who manufacture (defined by statute to include import) one or more of the subject chemical substances |
| Chemical exporters | 325 | Persons who export, or intend to export, one or more of the subject chemical substances |

This listing is not intended to be exhaustive. Instead, it provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in Table 1 of this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist in determinations of whether this action might apply to certain entities. To determine if you or your business is affected by this action, you should carefully examine the applicability provisions at 40 CFR 721.5 for SNUR-related obligations. Also, consult Unit III. Note that because this proposed rule would designate certain manufacturing and importing activities as significant new uses, persons that solely process the chemical substances that would be covered by this action would not be subject to the rule. If you have any questions regarding the applicability of this action to a particular entity, consult the technical person listed under **FOR FURTHER INFORMATION CONTACT**.

TABLE 2.—CHEMICALS REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2001

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| 383–07–3 | 2-Propenoic acid, 2-[butyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester. |
| 423–82–5 | 2-Propenoic acid, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester. |
| 2250–98–8 | 1-Octanesulfonamide, N,N',N''-[phosphinylidynetris(oxy-2,1-ethanediyl)]tris[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-. |
| 14650–24–9 | 2-Propenoic acid, 2-methyl-, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester. |
| 30381–98–7 | 1-Octanesulfonamide, N,N'-[phosphinicobis(oxy-2,1-ethanediyl)]bis[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt. |
| 55120–77–9 | 1-Hexanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-, lithium salt. |
| 57589–85–2 | Benzoic acid, 2,3,4,5-tetrachloro-6-[[[3-[[(heptadecafluorooctyl)sulfonyl]oxy]phenyl]amino]carbonyl]-, monopotassium salt. |
| 61660–12–6 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-[3-(trimethoxysilyl)propyl]-. |
| 67969–69–1 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-[2-(phosphonooxy)ethyl]-, diammonium salt. |
| 68156–01–4 | Cyclohexanesulfonic acid, nonafluorobis(trifluoromethyl)-, potassium salt. |
| 68329–56–6 | 2-Propenoic acid, eicosyl ester, polymer with 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, hexadecyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and octadecyl 2-propenoate. |
| 68555–91–9 | 2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, polymer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-methyl-2-propenoate. |
| 68555–92–0 | 2-Propenoic acid, 2-methyl-, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, polymer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-methyl-2-propenoate. |

TABLE 2.—CHEMICALS REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2001—Continued

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| 68608–14–0 | Sulfonamides, C4-8-alkane, perfluoro, N-ethyl-N-(hydroxyethyl), reaction products with 1,1'-methylenebis[4-isocyanatobenzene]. |
| 68909–15–9 | 2-Propenoic acid, eicosyl ester, polymers with branched octyl acrylate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl acrylate, 2-[methyl[(nonafluorobutyl) sulfonyl]amino]ethyl acrylate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(undecafluoropentyl) sulfonyl]amino]ethyl acrylate, polyethylene glycol acrylate Me ether and stearyl acrylate. |
| 70776–36–2 | 2-Propenoic acid, 2-methyl-, octadecyl ester, polymer with 1,1-dichloroethene, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, N-(hydroxymethyl)-2-propenamide, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate and 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate. |
| 73772–32–4 | 1-Propanesulfonic acid, 3-[[3-(dimethylamino)propyl][(tridecafluorohexyl)sulfonyl]amino]-2-hydroxy-, monosodium salt. |
| 81180–38–7 | 1-Propanaminium, N-(2-hydroxyethyl)-3-[(2-hydroxy-3-sulfopropyl) [(tridecafluorohexyl)sulfonyl]amino]-N,N-dimethyl-, hydroxide, monosodium salt. |
| 94133–90–1 | 1-Propanesulfonic acid, 3-[[3-(dimethylamino)propyl][(heptadecafluorooctyl)sulfonyl]amino]-2-hydroxy-, monosodium salt. |
| 117806–54–9 | 1-Heptanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-, lithium salt. |
| 127133–66–8 | 2-Propenoic acid, 2-methyl-, polymers with Bu methacrylate, lauryl methacrylate and 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl methacrylate. |
| 129813–71–4 | Sulfonamides, C4-8-alkane, perfluoro, N-methyl-N-(oxiranylmethyl). |
| 148240–78–2 | Fatty acids, C18-unsatd., trimers, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl esters. |
| 148240–79–3 | Fatty acids, C18-unsatd., trimers, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl esters. |
| 148240–80–6 | Fatty acids, C18-unsatd., trimers, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl esters. |
| 148240–81–7 | Fatty acids, C18-unsatd., trimers, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl esters. |
| 148240–82–8 | Fatty acids, C18-unsatd., trimers, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl esters. |
| 148684–79–1 | Sulfonamides, C4-8-alkane, perfluoro, N-(hydroxyethyl)-N-methyl, reaction products with 1,6-diisocyanatohexane homopolymer and ethylene glycol. |
| 178535–22–3 | Sulfonamides, C4-8-alkane, perfluoro, N-ethyl-N-(hydroxyethyl)-, polymers with 1,1'-methylenebis[4-isocyanatobenzene] and polymethylenepolyphenylene isocyanate, 2-ethylhexyl esters, Me Et ketone oxime-blocked. |
| P-83–1102 | Fatty acids, linseed-oil, dimers, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl esters. |
| P-84–1163 | Propanoic acid, 3-hydroxy-2-(hydroxymethyl)-2-methyl-, polymer with 2-ethyl-2-(hydroxymethyl)-1,3-propanediol and N,N',2-tris(6-isocyanatohexyl)imidodicarbonic diamide, reaction products with N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-1-octanesulfonamide and N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-1-heptanesulfonamide, compds. with triethylamine. |
| P-84–1171 | Propanoic acid, 3-hydroxy-2-(hydroxymethyl)-2-methyl-, polymer with 1,1'-methylenebis[4-isocyanatobenzene] and 1,2,3-propanetriol, reaction products with N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-1-octanesulfonamide and N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-1-heptanesulfonamide, compds. with morpholine. |
| P-86–0301 | Sulfonamides, C4-8-alkane, perfluoro, N-(hydroxyethyl)-N-methyl, reaction products with 12-hydroxystearic acid and 2,4-TDI, ammonium salts. |
| P-89–0799 | Sulfonamides, C4-8-alkane, perfluoro, N-ethyl-N-(hydroxyethyl), reaction products with 2-ethyl-1-hexanol and polymethylenepolyphenylene isocyanate. |
| P-94–0545 | 1-Hexadecanaminium, N,N-dimethyl-N-[2-[(2-methyl-1-oxo-2-propenyl)oxy]ethyl]-, bromide, polymers with Bu acrylate, Bu methacrylate and 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate. |
| P-94–0927 | 2-Propenoic acid, 2-methyl-, 2-methylpropyl ester, polymer with 2,4-diisocyanato-1-methylbenzene, 2-ethyl-2-(hydroxymethyl)-1,3-propanediol and 2-propenoic acid, N-ethyl-N-(hydroxyethyl)perfluoro-C4-8-alkanesulfon amides-blocked. |
| P-94–2205 | Polymethylenepolyphenylene isocyanate and bis(4-NCO-phenyl)methane reaction products with 2-ethyl-1-hexanol, 2-butanone, oxime, N-ethyl-N-(2- hydroxyethyl)-1-C4-C8 perfluoroalkanesulfonamide. |
| P-94–2206 | Siloxanes and Silicones, di-Me, mono[3-[(2-methyl-1-oxo-2-propenyl)oxy]propylgroup]-terminated, polymers with 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate and stearyl methacrylate. |
| P-96–1645 | Fatty acids, C18-unsatd., dimers, 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl esters. |
| P-97–0790 | 1-Decanaminium, N-decyl-N,N-dimethyl-, salt with 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-1-octanesulfonic acid (1:1). |
| P-98–0251 | 2-Propenoic acid, butyl ester, polymers with acrylamide, 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate and vinylidene chloride. |
| P-98–1272 | 2-Propenoic acid, 2-methyl-, 3-(trimethoxysilyl)propyl ester, polymers with acrylic acid, 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate and propylene glycol monoacrylate, hydrolyzed, compds. with 2,2'-(methylimino)bis[ethanol]. |
| P-99–0188 | Hexane, 1,6-diisocyanato-, homopolymer, N-(hydroxyethyl)-N-methyl perfluoro C4-8-alkane sulfonamide- and stearyl alc-blocked. |
| P-99–0319 | Poly(oxy-1,2-ethanediyl), alpha.-[2-(methylamino)ethyl]-.omega.-[(1,1,3,3-tetramethylbutyl)phenoxy]-, N-[(perfluoro-C4-8-alkyl)sulfonyl] derivs. |

TABLE 3.—CHEMICALS SUBJECT TO VOLUME CAP RESTRICTIONS ON OR AFTER JANUARY 1, 2001 AND REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2003

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| 307–35–7 | 1-Octanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- |
| 307–51–7 | 1-Decanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heneicosafluoro- |
| 376–14–7 | 2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester |

TABLE 3.—CHEMICALS SUBJECT TO VOLUME CAP RESTRICTIONS ON OR AFTER JANUARY 1, 2001 AND REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2003—Continued

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| 423–50–7 | 1-Hexanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro- |
| 754–91–6 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- |
| 1652–63–7 | 1-Propanaminium, 3-[[(heptadecafluorooctyl)sulfonyl]amino]-N,N,N-trimethyl-, iodide |
| 1691–99–2 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)- |
| 1763–23–1 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- |
| 2795–39–3 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, potassium salt |
| 2991–51–7 | Glycine, N-ethyl-N-[(heptadecafluorooctyl)sulfonyl]-, potassium salt |
| 4151–50–2 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- |
| 17202–41–4 | 1-Nonanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,9-nonadecafluoro-, ammonium salt |
| 24448–09–7 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-N-methyl- |
| 25268–77–3 | 2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester |
| 29081–56–9 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt |
| 29117–08–6 | Poly(oxy-1,2-ethanediyl), .alpha.-[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl]-.omega.-hydroxy- |
| 29457–72–5 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, lithium salt |
| 31506–32–8 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-methyl- |
| 38006–74–5 | 1-Propanaminium, 3-[[(heptadecafluorooctyl)sulfonyl]amino]-N,N,N-trimethyl-, chloride |
| 38850–58–7 | 1-Propanaminium, N-(2-hydroxyethyl)-N,N-dimethyl-3-[(3-sulfopropyl)[(tridecafluorohexyl)sulfonyl]amino]-, inner salt |
| 67584–42–3 | Cyclohexanesulfonic acid, decafluoro(pentafluoroethyl)-, potassium salt |
| 67906–42–7 | 1-Decanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heneicosafluoro-, ammonium salt |
| 68298–62–4 | 2-Propenoic acid, 2-[butyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, telomer with 2-[butyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, methyloxirane polymer with oxirane di-2-propenoate, methyloxirane polymer with oxirane mono-2-propenoate and 1-octanethiol |
| 68541–80–0 | 2-Propenoic acid, polymer with 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-propenoate |
| 68555–90–8 | 2-Propenoic acid, butyl ester, polymer with 2-[[(heptadecafluorooctyl)sulfonyl] methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate and 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate |
| 68586–14–1 | 2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, telomer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, alpha.-(2-methyl-1-oxo-2-propenyl)-.omega.-hydroxypoly(oxy-1,2-ethanediyl), .alpha.-(2-methyl-1-oxo-2-propenyl)-.omega.-[(2-methyl-1-oxo-2-propenyl)oxy]poly(oxy-1,2-ethanediyl), 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 1-octanethiol |
| 68649–26–3 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-, reaction products with N-ethyl-1,1,2,2,3,3,4,4,4-nonafluoro-N-(2-hydroxyethyl)-1-butanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-1-heptanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-(2-hydroxyethyl)-1-hexanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-(2-hydroxyethyl)-1-pentanesulfonamide, polymethylenepolyphenylene isocyanate and stearyl alc. |
| 68867–60–7 | 2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, polymer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and .alpha.-(1-oxo-2-propenyl)-.omega.-methoxypoly(oxy-1,2-ethanediyl) |
| 68867–62–9 | 2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, telomer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 1-octanethiol and .alpha.-(1-oxo-2-propenyl)-.omega.-methoxypoly(oxy-1,2-ethanediyl) |
| 68891–96–3 | Chromium, diaquatetrachloro[.mu.-[N-ethyl-N-[(heptadecafluorooctyl)sulfonyl]glycinato-.kappa.O:.kappa.O']]-.mu.-hydroxybis(2-methylpropanol)di- |
| 68958–61–2 | Poly(oxy-1,2-ethanediyl), .alpha.-[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl]-.omega.-methoxy- |
| 70225–14–8 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, compd. with 2,2'-iminobis[ethanol] (1:1) |
| 71487–20–2 | 2-Propenoic acid, 2-methyl-, methyl ester, polymer with ethenylbenzene, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 2-propenoic acid |
| 91081–99–1 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-N-methyl-, polymer with(chloromethyl)oxirane, 1,1,2,2,3,3,4,4,4-nonafluoro-N-(2-hydroxyethyl)-N-methyl-1-butanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-N-methyl-1-heptanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-(2-hydroxyethyl)-N-methyl-1-hexanesulfonamide and 1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-(2-hydroxyethyl)-N-methyl-1-pentanesulfonamide, hexanedioate (ester) |
| 98999–57–6 | Sulfonamides, C7-8-alkane, perfluoro, N-methyl-N-[2-[(1-oxo-2-propenyl) oxy]ethyl], polymers with 2-ethoxyethyl acrylate, glycidyl methacrylate and N,N,N-trimethyl-2-[(2-methyl-1-oxo-2-propenyl)oxy]ethanaminiumchloride |
| 182700–90–9 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-methyl-, reaction products with benzene-chlorine-sulfur chloride (S2Cl2) reaction products chlorides |
| L-92–0151 | 2-Propenoic acid, 2-methyl-, butyl ester, polymer with 2-methyl-, 2-[ethyl [(heptadecafluorooctyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, and 2-propenoic acid |
| P-80–0183 | Sulfonamides, C4-8-alkane, perfluoro, N-[3-(dimethylamino)propyl], reaction products with acrylic acid |

TABLE 3.—CHEMICALS SUBJECT TO VOLUME CAP RESTRICTIONS ON OR AFTER JANUARY 1, 2001 AND REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2003—Continued

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| P-86-0958 | 2-Propenoic acid, 2-methyl-, dodecyl ester, polymers with 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate and vinylidene chloride |
| P-90-0111 | Sulfonamides, C4-8-alkane, perfluoro, N-methyl-N-[(3-octadecyl-2-oxo-5-oxazolidinyl)methyl] |
| P-91-1419 | Poly(oxy-1,2-ethanediyl), alpha -hydro- omega,-hydroxy-, polymer with 1,6-diisocyanatonexane, N-(2-hydroxyethyl)-N-methyl perfluoro C4-8-alkane sulfonamide-blocked |
| P-93-1444 | 2-Propenoic acid, 2-methyl-, dodecyl ester, polymers with N-(hydroxymethyl)-2-propenamide, 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl methacrylate, stearyl methacrylate and vinylidene chloride |
| P-95-0120 | Sulfonamides, C4-8-alkane, perfluoro, N,N'-[1,6-hexanediylbis[[2-oxo-3,5-oxazolidinediyl)methylene]]bis[N-methyl- |
| P-96-1262 | Sulfonic acids, C6-8-alkane, perfluoro, compds. with polyethylene-polypropylene glycol bis(2-aminopropyl) ether |
| P-96-1424 | 2-Propenoic acid, 2-methyl-, 2-(dimethylamino)ethyl ester, telomers with 2-[ethyl[(perfluoro-C4-8-alkylsulfonyl]amino]ethyl methacrylate and 1-octanethiol, N-oxides |
| P-96-1433 | Sulfonamides, C4-8-alkane, perfluoro, N-[3-(dimethyloxidoamino)propyl], potassium salts |

*B. How Can I get Additional Information, Including Copies of this Document or Other Related Documents?*

1. *Electronically.* You may obtain electronic copies of this document and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select "Law and Regulations," "Regulations and Proposed Rules," then look up the entry for this document under "**Federal Register**—Environmental Documents." You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/. To access the OPPTS Harmonized Guidelines referenced in this document, go directly to the guidelines at http://www.epa.gov/opptsfrs/home/guidelin.htm. In addition, you may access other information about the Office of Prevention, Pesticides and Toxic Substances (OPPTS) and related programs at http://www.epa.gov/internet/oppts/.

2. *In person.* The Agency has established an official record for this action under docket control number OPPTS-50639. The official record consists of the documents referenced in this action, any public comments received during the comment period, and other information related to this rulemaking, including information claimed as Confidential Business Information (CBI). This official record includes the documents that are physically located in the docket, as well as all documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed paper versions of any electronic comments that may be submitted during an applicable comment period, is available for inspection in the TSCA Nonconfidential Information Center, Room NE B-607, 401 M St., SW., Washington, DC. The Center is open from noon to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number of the Center is (202) 260-7099.

*C. How and to Whom Do I Submit Comments?*

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, your comments must identify docket control number OPPTS-50639 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Document Control Office (7407), Office of Pollution Prevention and Toxics (OPPT), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: OPPT's Document Control Office (DCO), East Tower Room G-099, Waterside Mall, 401 M St., SW., Washington, DC. The DCO is open from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the DCO is (202) 260-7093.

3. *Electronically.* You may submit your comments electronically by e-mail to: oppt.ncic@epa.gov, or mail or deliver your computer disk to the addresses identified in Unit I.C.1. or I.C.2. Do not submit any information electronically that you consider to be CBI. E-mailed comments must be submitted as an ASCII file, avoiding the use of special characters or any form of encryption. Comments will also be accepted on standard computer disks in WordPerfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number OPPTS-50639. Electronic comments may also be filed online at many Federal Depository Libraries.

*D. How Should I Handle CBI Information that I Want to Submit to the Agency?*

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comments that include any information claimed as CBI, a sanitized copy of the comments which does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record by EPA without prior notice. If you have any questions about CBI or the procedures for claiming CBI, consult the technical person listed under **FOR FURTHER INFORMATION CONTACT**.

*E. What Should I Consider as I Prepare My Comments for EPA?*

We invite you to provide your views on the various options we propose, new approaches we have not considered, the potential impacts of the various options (including possible unintended consequences), and any data or information that you would like the Agency to consider during the development of the final SNUR. You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

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4. If you estimate potential burden or costs, explain how you arrived at the estimate.

5. Provide specific examples to illustrate your concerns.

6. Offer alternative ways to improve the proposed rule or data collection activity.

7. Make sure to submit your comments by the deadline specified in this document.

8. At the beginning of your comments, be sure to properly identify the document you are commenting on. To ensure proper receipt by EPA, your comments must identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the title, date, and **Federal Register** citation.

## II. Background

### *A. What Action is the Agency Taking?*

This proposal would require persons to notify EPA at least 90 days before commencing the manufacture or import of the chemical substances identified in Table 2 or Table 3 of Unit I.A., for the significant new uses described in this document. The chemical substances identified in Table 2 and Table 3 of Unit I.A. include PFOSA, PFOSS, PFOSF, certain higher and lower homologues of PFOSA and PFOSF, and certain other chemical substances, including polymers, that contain PFOSA and its homologues as substructures. These chemical substances are collectively referred to throughout this proposed rule as PFOS.

The significant new uses described by this notice are:

1. The manufacture or import for any use of any of the chemicals listed in Table 2 of Unit I.A. on or after January 1, 2001.

2. The manufacture or import for any use of any one or more of the chemicals listed in Table 3 of Unit I.A. in excess of an aggregate volume of 1,100,000 pounds per person per calendar year on or after January 1, 2001 and before January 1, 2003.

3. The manufacture or import for any use of any of the chemicals listed in Table 3 of Unit I.A. on or after January 1, 2003.

### *B. What is the Agency's Authority for Taking this Action?*

Section 5(a)(2) of TSCA (15 U.S.C. 2604(a)(2)) authorizes EPA to determine that a use of a chemical substance is a "significant new use." The Agency makes this determination by rule after considering all relevant factors, including those listed in TSCA section 5(a)(2). These factors include the volume of a chemical substance's production; the extent to which a use changes the type, form, magnitude, or duration of exposure to the substance; and the reasonably anticipated manner of producing or otherwise managing the substance. Once EPA makes this determination and promulgates a SNUR, TSCA section 5(a)(1)(B) requires persons to submit a significant new use notice (SNUN) to EPA at least 90 days before they manufacture, import, or process the chemical substance for that significant new use (15 U.S.C. 2604 (a)(1)(B)).

### *C. Which General Provisions Apply?*

General provisions for SNURs are published under 40 CFR part 721, subpart A. These provisions describe persons subject to the rule, recordkeeping requirements, exemptions to reporting requirements, and applicability of the rule to uses occurring before the effective date of the final rule. Note that because this proposed rule would designate certain manufacturing and importing activities as significant new uses, persons that solely process the chemical substances that would be covered by this action would not be subject to the rule. Provisions relating to user fees appear at 40 CFR part 700. Persons subject to this proposed SNUR would be required to comply with the same notice requirements and EPA regulatory procedures as submitters of Premanufacture Notices (PMNs) under TSCA section 5(a)(1)(A). In particular, these requirements include: the information submission requirements of TSCA section 5(b) and 5(d)(1); the exemptions authorized by TSCA section 5 (h)(1), (2), (3), and (5); the export notification provisions of TSCA section 12(b); and the export notification requirements in 40 CFR part 707, subpart D. Once EPA receives a SNUN, EPA may take regulatory action under TSCA sections 5(e), 5(f), 6, or 7, if appropriate, to control the activities on which it has received the SNUN. If EPA does not take action, EPA is required under TSCA section 5(g) to explain in the **Federal Register** its reasons for not taking action.

## III. Summary of this Proposed Rule

The chemical substances subject to this proposed SNUR are listed in Table 2 and Table 3 of Unit I.A. These chemical substances include PFOSA, PFOSS, PFOSF, certain higher and lower homologues of PFOSA and PFOSF, and certain other chemical substances, including polymers, that contain PFOSA and its homologues as substructures. All of these chemical substances are referred to collectively in this proposed rule as perfluorooctyl sulfonates, or PFOS. All of these chemical substances have the potential to degrade back to PFOSA in the environment, and PFOSA does not degrade further. PFOSA is highly persistent in the environment and has a strong tendency to bioaccumulate. Studies have found PFOS in very small quantities in the blood of the general human population as well as in wildlife, indicating that exposure to the chemicals is widespread, and recent tests have raised concerns about their potential developmental, reproductive, and systemic toxicity (Refs. 1, 2, and 3). These factors, taken together, raise concerns for long term potential adverse effects in people and wildlife over time if PFOS should continue to be produced, released, and built up in the environment.

EPA believes that the chemical substances listed in Tables 2 and 3 of Unit I.A. are manufactured and imported in the United States only by the Minnesota Mining and Manufacturing Company (3M) (Refs. 4 and 5). 3M has committed to phase out these chemicals voluntarily by discontinuing the manufacture of certain of these chemical substances on a global basis for their most widespread uses by the end of December 2000, by steadily reducing their production volume on the remaining chemicals through 2001 and 2002, and by entirely discontinuing the manufacture of all of these PFOS chemicals by December 31, 2002 (Ref. 6). The chemicals listed in Table 2 of Unit I.A. are those which 3M has committed to cease manufacturing by December 31, 2000. The chemicals listed in Table 3 of Unit I.A. are those which 3M has committed first to reduce, and then to cease manufacturing by December 31, 2002. EPA believes that any manufacture or import of these PFOS chemicals occurring after 3M's global phase-out dates would increase the magnitude and duration of exposure to these chemicals. Therefore, EPA is proposing to designate the following as significant new uses:

1. Any manufacture or import for any use of the chemicals listed in Table 2 of Unit I.A. on or after January 1, 2001.

2. Any manufacture or import for any use of the chemicals listed in Table 3 of Unit I.A. in excess of an aggregate annual manufacture and import volume cap for all of these chemicals of 1,100,000 pounds per person per calendar year on or after January 1, 2001 and before January 1, 2003.

3. Any manufacture or import for any use of any of the chemicals listed in

Table 3 of Unit I.A. on or after January 1, 2003.

Given that no companies other than 3M are currently producing the chemicals listed on Table 3 of Unit I.A., and given the negative commercial and regulatory environment associated with these chemicals, EPA believes it is unlikely that companies would incur the costs associated with establishing new manufacturing capacity for these chemicals in order to enter this market.

This proposed rule, when finalized, would require persons who intend to manufacture or import the PFOS chemicals listed in this proposed rule to notify EPA, through the submission of a SNUN, at least 90 days before commencing the manufacture or importation of any of these chemicals for any use designated by this proposed SNUR as a significant new use. The required notice would provide EPA with the opportunity to evaluate the intended use, and, if necessary, to prohibit or limit that use before it occurs. These proposed requirements are summarized in the following Table 4:

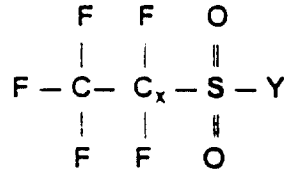
TABLE 4.—SUMMARY OF PROPOSED SNUR REQUIREMENTS

| You must file a significant new use notice (SNUN) if you: | | |
|---|---|---|
| Manufacture or import: | When? | How much? |
| Chemical substances listed in Table 2 of Unit I.A. | After December 31, 2000 | Any amount |
| Chemical substances listed in Table 3 of Unit I.A. | January 1, 2001 through December 31, 2002 | Aggregate amount exceeding 1,100,000 lbs per person per calendar year |
| Chemical substances listed in Table 3 of Unit I.A. | After December 31, 2002 | Any amount |

## IV. Chemical Compound History

### A. *Defining PFOS*

This proposed rule applies to a large group of fully fluorinated alkyl sulfonate-containing substances, none of which occur naturally. The Ninth Collective Index chemical names and CAS Registry Numbers (CAS No.) (when available) provided in Table 2 and Table 3 of Unit I.A. are for the specific chemical substances that are subject to the provisions contained in this proposed SNUR (for example, entry #8 on Table 3 of Unit I.A. lists CAS No.1763–23–1 for the compound named 1-octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, commonly referred to as PFOSA). All of the chemical substances listed in Table 2 and Table 3 of Unit I.A. have a common chemical structure consisting of a PFOS moiety, as illustrated here, somewhere in the molecule.



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The number of carbon atoms present in the moiety varies from 4 to 10 (x = 3–9) among the listed chemicals. In addition, there are many examples of different chemical functionality (free acids (Y = OH), metal salts (Y = $O^- M^+$), sulfonyl halides (Y = X), sulfonamides (Y= $NH^2$), and other derivatives). The listed chemical substances also include polymers.

The class of chemical substances including the perfluoroalkyl sulfonyl moiety described by the structure shown in this unit contains more chemical substances than are specified in the lists in Table 2 and Table 3 of Unit I.A. Only the listed chemical substances, which are manufactured or imported exclusively by 3M and which 3M has voluntarily committed to cease producing, are subject to this SNUR. EPA is evaluating further this overall structural class of chemical substances and may take additional regulatory action as appropriate.

### B. *Environmental Fate*

The basic building block of all of the PFOS chemicals is PFOSF, which is used as an intermediate in the production of the PFOS chemicals. PFOSA results from the chemical or enzymatic hydrolysis of PFOSF. Current information strongly supports that PFOSA is an extremely stable substance which resists breakdown by chemical or biological processes. Therefore PFOSA is the ultimate degradation product from PFOS chemicals and will persist in that form (Refs. 1 and 2).

EPA cannot currently conduct a definitive assessment of the environmental transport and partitioning of PFOS. The available data are limited and their accuracy uncertain. Also, the accuracy of the estimation models is limited by the quality of data input into them. Depending on what data are consulted and utilized, the environmental fate and transport of PFOS vary. Biological sampling recently discovered the presence of certain perfluoroalkyl compounds in fish and in fish-eating birds across the United States and in locations in Canada, Sweden, and the South Pacific (Ref. 1). The wide distribution of the chemicals in high trophic levels is strongly suggestive of the potential for bioaccumulation/bioconcentration. The widespread presence of PFOS suggests the possibility of transport in air as well as water, but the multimedia equilibrium criterion model (EQC) suggests otherwise (Ref. 7). Using data provided by 3M as inputs, the model indicates that PFOS would fall out of air and partition almost equally in water and soil. The Henry's Law values calculated utilizing the vapor pressure of 3.31 E-4 Pa@20 C and water solubility values of 370, 570, 5, and 25 milligram/Liter (mg/L) in fresh water, pure water, unfiltered seawater, and filtered seawater, respectively yielded Henry's Law values of 4.7 E-9 , 7.2 E-9, 6.4 E-11, and 3.2 E-10 atm.m3/mole (atmospheres per meter cubed per mole), respectively. The vapor pressure and water solubility values were obtained from Table 4, p.16 of the March 1, 2000, white paper by 3M, *Sulfonated Perfluorochemicals in the Environment: Sources, Dispersion, Fate and Effects* (Ref. 1). These Henry's Law values suggest that volatilization from water to air is not very likely. According to 3M, testing is planned and/or underway for the environmental properties, fate, and transport of PFOS (Ref. 1). With more complete data, EPA would be able to make more definitive assessments. With the present data, the Agency can only speculate on environmental transport and partitioning of PFOS, although current information suggests strongly that it is persistent and may bioaccumulate.

### C. *Health Effects*

The Agency's hazard analysis for PFOS is a review of health hazard and biomonitoring data (Ref. 8). Toxicology studies show that PFOS is well absorbed

orally and distributes primarily in the serum and liver. PFOS can also be formed as a metabolite of other perfluorinated sulfonates. It does not appear to be further metabolized. Elimination from the body is slow and occurs via both urine and feces. Serum PFOS levels in three retired male 3M chemical workers have been followed for 5½ years and suggest a mean elimination half-life ($t_{½}$) of 1,428 days (approximately 4 years). Based on the pharmacokinetic data obtained from a 28-day oral study in male and female monkeys, a volume of distribution (Vd) of 0.19 L/kilogram (kg) was reported; no sex differences in the pharmacokinetic parameters were noted.

PFOS has shown moderate acute toxicity by the oral route with a rat $LD_{50}$ of 251 mg/kg. A 1-hour $LC_{50}$ of 5.2 mg/L in rats has been reported. PFOS was found to be mildly irritating to the eyes and non-irritating to the skin of rabbits. PFOS was negative in mutagenicity studies in five strains of salmonella and did not induce micronuclei in an *in vivo* mouse bone marrow micronucleus assay.

Numerous repeat-dose oral toxicity studies on PFOS have been conducted in rats and primates. Adverse signs of toxicity observed in rat studies included increases in liver enzymes, hepatic vacuolization and hepatocellular hypertrophy, gastrointestinal effects, hematological abnormalities, weight loss, convulsions, and death. These effects were reported at doses of 2 mg/kg/day and above. Adverse signs of toxicity observed in Rhesus monkey studies included anorexia, emesis, diarrhea, hypoactivity, prostration, convulsions, atrophy of the salivary glands and the pancreas, marked decreases in serum cholesterol, and lipid depletion in the adrenals. The dose range for these effects was reported between 1.5–300 mg/kg/day. No monkeys survived beyond 3 weeks into treatment at 10 mg/kg/day, or beyond 7 weeks into treatment at doses as low as 4.5 mg/kg/day. At doses as low as 0.75 mg/kg/day, Cynomolgus monkeys exhibited low food consumption, excessive salivation, labored breathing, hypoactivity, ataxia, hepatic vacuolization and hepatocellular hypertrophy, significant reductions in serum cholesterol levels, and death.

Postnatal deaths and other developmental effects were reported at low doses in offspring in a 2-generation reproductive toxicity study in rats. At the two highest doses of 1.6 and 3.2 mg/kg/day, pup survival in the first generation was significantly decreased. All first generation offspring (F1 pups) at the highest dose died within a day after birth while close to 30% of the F1 pups in the 1.6 mg/kg/day dose group died within 4 days after birth. As a result of the pup mortality in the two top dose groups, only the two lowest dose groups, 0.1 and 0.4 mg/kg/day, were continued into the second generation. The no observed adverse effect level (NOAEL) and lowest observed adverse effect level (LOAEL) for the second generation offspring (F2 pups) were 0.1 mg/kg/day and 0.4 mg/kg/day, respectively, based on reductions in pup body weight. Reversible delays in reflex and physical development were also observed in this study, raising concerns about the potential for developmental neurotoxicity following exposure to PFOS.

Developmental effects were also reported in prenatal developmental toxicity studies in the rat and rabbit, although at slightly higher dose levels. Signs of developmental toxicity were evident at doses of 5 mg/kg/day and above in rats administered PFOS during gestation. Significant decreases in fetal body weight and significant increases in external and visceral anomalies, delayed ossification, and skeletal variations were observed. Abnormalities of the lens of the eye were also reported at doses as low as 1 mg/kg/day in one rat prenatal developmental study, but could not be repeated in a second study of similar design. At doses of 2.5 mg/kg/day and above, significant reductions in fetal-body weight and significant increases in delayed ossification were observed in rabbits administered PFOS during gestation.

In human blood samples, PFOS has been detected in the serum of occupational and general populations in the parts per million (ppm) to parts per billion (ppb) range. In the United States, recent blood serum levels of PFOS in manufacturing employees have been as high as 12.83 ppm, while in the general population, serum collected from blood banks and commercial sources have indicated mean PFOS levels of 30–44 ppb. Levels in a very small sample of children yielded even higher results, with a mean level of 54 ppb.

Sampling of several wildlife species from a variety of sites across the United States has shown widespread distribution of PFOS. In recent analyses, PFOS was detected in the ppb range in the plasma of several species of eagles, wild birds, and fish. Endogenous levels of PFOS have also been detected in the ppb range in the livers of unexposed rats used in toxicity studies, presumably through a dietary source (fishmeal).

Although the PFOS levels detected in the blood of the general population are low, this widespread presence, combined with the persistence, the bioaccumulative potential, and the reproductive and subchronic toxicity of the chemical, raises concerns for potential adverse effects on people and wildlife over time should the chemical substances continue to be produced, released, and accumulated in the environment.

*D. Exposure Data*

As indicated in Unit IV.C., PFOS has been detected at low levels in the blood of humans and wildlife throughout the United States, providing clear evidence of widespread exposure to the chemical. PFOS has been in commercial use since the 1950's, predominantly in soil and stain-resistant coating products on fabrics, carpets, and leather, and in grease and oil resistant coatings on paper products, including food contact papers. Other uses leading to environmental releases include fire fighting foams. The various surface treatment uses constitute the largest volume of PFOS production and are believed to present the greatest potential for widespread human and environmental exposure to PFOS. Studies are underway to determine the routes of exposure which have led to the detection of PFOS in human and animal blood. There are several potential pathways that may account for the widespread exposure to PFOS including: Dietary intake from the consumption of food wrapped in paper containing PFOS derivatives; inhalation from aerosol applications of PFOS-containing consumer products; and inhalation, dietary, or dermal exposures resulting from manufacturing, as well as industrial, commercial, and consumer use and disposal of PFOS-derived chemicals and products.

*E. Use Data*

PFOS and related sulfonyl-based fluorochemicals are used in a variety of products, which can be divided into three main categories of use: Surface treatments, paper protectors, and performance chemicals (Ref. 4). The various surface treatment and paper protection uses constitute the largest volume of PFOS production and are believed to present the greatest potential for widespread human and environmental exposure to PFOS.

PFOS chemicals produced for surface treatment applications provide soil, oil, and water resistance to personal apparel and home furnishings. Specific applications in this use category include protection of apparel and leather, fabric/upholstery, and carpet. These applications are undertaken in

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industrial settings by customers such as textile mills, leather tanneries, finishers, fiber producers, and carpet manufacturers. PFOS chemicals are also used in aftermarket treatment of apparel and leather, upholstery, carpet, and automobile interiors by the general public or professional applicators (Ref. 4). In 2000, the domestic production volume of PFOS chemicals for this use category is estimated to be approximately 2.4 million pounds (Ref. 6).

PFOS chemicals produced for paper protection applications provide grease, oil, and water resistance to paper and paperboard as part of a sizing agent formulation. Specific applications in this use category include food contact applications (plates, food containers, bags, and wraps) regulated by the Food and Drug Administration (FDA) under 21 CFR 176.170, as well as non-food contact applications (folding cartons, containers, carbonless forms, and masking papers). The application of sizing agents is undertaken mainly by paper mills and, to some extent, converters who manufacture bags, wraps, and other products from paper and paperboard (Ref. 4). In 2000, the domestic production volume of PFOS chemicals for this use category is estimated to be approximately 2.7 million pounds (Ref. 6).

PFOS chemicals in the performance chemicals category are used in a wide variety of specialized industrial, commercial, and consumer applications. Specific applications include fire fighting foams, mining and oil well surfactants, acid mist suppressants for metal plating and electronic etching baths, alkaline cleaners, floor polishes, photographic film, denture cleaners, shampoos, chemical intermediates, coating additives, carpet spot cleaners, and as an insecticide in bait stations for ants (Ref. 4). In 2000, the domestic production volume of PFOS chemicals for this use category is estimated to be approximately 1.5 million pounds (Ref. 6).

On May 16, 2000, following discussions with the Agency, 3M issued a press release announcing that it would discontinue the production of perfluorooctanyl chemicals used to produce some of its repellent and surfactant products. In its statement, 3M committed to "substantially phase out production" by the end of calendar year 2000 (Ref. 9). In subsequent correspondence with the Agency, 3M provided a schedule documenting its complete plan for discontinuing all manufacture of specific PFOS and related chemicals for most surface treatment and paper protection uses (including food contact uses regulated by the FDA) by the end of 2000, and discontinuing all manufacture for any uses by the end of 2002 (Ref. 6). This schedule, and 3M's anticipated production volumes, are summarized in Table 5.

TABLE 5.—ANTICIPATED ANNUAL U.S. PRODUCTION VOLUME (POUNDS) FOR PFOS USE CATEGORIES

| Use category | 2000 | 2001 | 2002 | 2003 |
|---|---|---|---|---|
| Surface treatment | 2,356,700 | 0 | 0 | 0 |
| Paper protection | 2,670,700 | 0 | 0 | 0 |
| Performance chemicals | 1,462,500 | 1,011,900 | 443,700 | 0 |
| Total | 6,489,900 | 1,011,900 | 443,700 | 0 |

According to the information currently available to EPA, 3M is the sole manufacturer of PFOS chemicals affected by this proposed SNUR (Ref. 5). 3M plans to discontinue the manufacture of the chemicals identified in Table 2 of Unit I.A. (in general, those associated with surface treatment and paper protection uses) by the end of 2000 and to discontinue the chemicals identified in Table 3 of Unit I.A. (in general, those associated with performance chemical applications) by the end of 2002.

## V. Objectives and Rationale for this Proposed Rule

In determining what would constitute a significant new use for the chemical substances that are the subjects of this proposed SNUR, EPA considered relevant information on the toxicity of the substances, likely exposures associated with potential uses, information provided by industry sources, and the four factors listed in TSCA section 5(a)(2) and Unit II.B.

Based on these considerations, EPA wants to achieve the following objectives with regard to the significant new uses that are designated in this proposed rule. EPA wants to ensure that:

1. EPA would receive notice of any person's intent to manufacture or import PFOS chemicals for a designated significant new use before that activity begins.

2. EPA would have an opportunity to review and evaluate data submitted in a SNUN before the notice submitter begins manufacturing or importing the subject chemical substances for a significant new use.

3. EPA would be able to regulate prospective manufacturers and importers of the subject chemical substances before a significant new use occurs, provided such regulation is warranted pursuant to TSCA section 5(e) or (f).

EPA has concerns regarding the toxicity, persistence, and bioaccumulative potential of the chemical substances that are included in this proposed SNUR. 3M, the sole manufacturer of these chemicals in the United States, has chosen voluntarily to discontinue their manufacture and sale for all uses by December 31, 2002, and to substantially reduce their manufacture for their most widespread uses by December 31, 2000. With 3M's exit from the market, EPA believes that all manufacture of these chemicals likely will cease. However, EPA is concerned that manufacture could be reinitiated in the future, and wants the opportunity to evaluate and control, if appropriate, exposures associated with that activity. The notice that would be required by the SNUR would provide EPA with the opportunity to evaluate activities associated with a significant new use as proposed herein and an opportunity to protect against unreasonable risks, if any, from exposure to the substances which could result.

Given that no companies other than 3M are currently producing the chemicals listed on Table 3 of Unit I.A., and given the negative commercial and regulatory environment associated with these chemicals, EPA believes it is unlikely that companies would incur the costs associated with establishing new manufacturing capacity for these chemicals in order to enter this market. EPA will use information submitted pursuant to the Inventory Update Rule (40 CFR part 710) to track the production volumes of these chemicals. In the event that the phase-out of these chemicals does not progress as

described in this proposed rule. EPA may pursue additional regulatory action as appropriate under TSCA sections 4, 6, and 8.

## VI. Alternatives

Before proposing this SNUR, EPA considered the following alternative regulatory actions for the chemical substances listed in Tables 2 and 3 of Unit I.A. In addition, EPA determined that these chemical substances are currently not subject to Federal notification requirements.

1. Promulgate a chemical-specific TSCA section 8(a) reporting rule for the chemical substances listed in Tables 2 and 3 of Unit I.A. Under a TSCA section 8(a) rule, EPA could require any person to report information to the Agency when they intend to manufacture or import the substances listed in Tables 2 and 3 of Unit I.A. for the significant new uses listed in this proposed rule (15 U.S.C. 2607). However, the use of TSCA section 8(a) rather than SNUR authority would not provide the opportunity for EPA to review human and environmental hazards and exposures associated with the new uses of these substances and, if necessary, to take immediate regulatory action under TSCA section 5(e) or section 5(f) to prohibit or limit the activity before it begins. In addition, EPA may not receive important information from small businesses, because those firms generally are exempt from TSCA section 8(a) reporting requirements. In view of EPA's concerns about these chemical substances and its interest in having the opportunity to regulate these substances further as needed, pending the development of exposure and/or hazard information should a significant new use be initiated, the Agency believes that a TSCA section 8(a) rule for those chemical substances would not meet all of EPA's regulatory objectives.

2. Regulate the chemical substances listed in Tables 2 and 3 of Unit I.A. under TSCA section 6. EPA must regulate under TSCA section 6 if there is a reasonable basis to conclude that the manufacture, import, processing, distribution in commerce, use, or disposal of a chemical substance or mixture "presents or will present" an unreasonable risk of injury to human health or the environment. Given the decision by the sole manufacturer 3M to discontinue manufacturing these chemicals, and thus to remove the bulk of the existing potential risk which they present, EPA concluded that risk management action under TSCA section 6 is probably not necessary at this time. This proposed SNUR will allow the Agency to address the potential risks associated with any intended significant new use of these substances. If the phase-out of these chemicals does not occur as anticipated, EPA may reconsider this decision and pursue additional regulatory action as appropriate.

## VII. Applicability of Proposed Rule to Uses Occurring Before the Effective Date of the Final Rule

EPA believes that the intent of TSCA section 5(a)(1)(B) is best served by designating a use as a significant new use as of the proposal date of the SNUR, rather than as of the effective date of the final rule. If uses begun after publication of the proposed SNUR were considered to be ongoing, rather than new, it would be difficult for EPA to establish SNUR notice requirements, because any person could defeat the SNUR by initiating the proposed significant new use before the rule became final, and then argue that the use was ongoing.

Persons who begin commercial manufacture or import of PFOS for the significant new uses listed in this proposed SNUR after the proposal has been published must stop that activity before the effective date of the final rule. Persons who ceased those activities will have to meet all SNUR notice requirements and wait until the end of the notice review period, including all extensions, before engaging in any activities designated as significant new uses. If, however, persons who begin commercial manufacture or import of these chemical substances between the proposal and the effective date of the SNUR meet the conditions of advance compliance as codified at 40 CFR 721.45(h), those persons will be considered to have met the final SNUR requirements for those activities.

## VIII. Test Data and Other Information

EPA recognizes that under TSCA section 5, persons are not required to develop any particular test data before submitting a SNUN. Rather, persons are required only to submit test data in their possession or control and to describe any other data known to, or reasonably ascertainable by them (15 U.S.C. 2604(d); 40 CFR 721.25).

However, in view of the potential health and environmental risks posed by the significant new uses of the chemical substances listed in Table 2 and Table 3 of Unit I.A., EPA requests that potential SNUN submitters include data that would permit a reasoned evaluation of risks posed by these chemical substances when used for an intended significant new use. EPA currently believes that the known or reasonable ascertainable results of the following tests could help adequately characterize possible health effects of these chemical substances: Reproductive and developmental toxicity studies, mutagenicity, gene mutation, immunotoxicity, neurotoxicity, carcinogenicity, and acute, subchronic, and chronic toxicity studies, as well as pharmacokinetics and mechanistic studies. Because of the specific concerns that EPA has for the persistence and bioaccumulation potential of these chemicals, EPA also encourages SNUN submitters to provide information on environmental fate and transport, specifically including measured values for the octanol/water partition coefficient (log P), log of the soil/sediment adsorption coefficient (log Koc), bioconcentration factor (BCF), melting and/or boiling point, vapor pressure, Henry's Law constant, biodegradation, atmospheric oxidation and the fugacity-based multimedia equilibrium criterion level III (EQC III) model (Ref. 10). However, completion of those studies may not be the only means of identifying potential risks. For example, analyses of potential exposure may demonstrate that associated risks would be of low concern. A SNUN submitted without accompanying test data may increase the likelihood that EPA will take action under TSCA section 5(e).

EPA encourages persons to consult with the Agency before submitting a SNUN for any of the PFOS substances listed in Table 2 or Table 3 of Unit I.A. As part of this optional pre-notice consultation, EPA will discuss specific test data it believes are necessary to evaluate a significant new use of the chemical substances and advise the submitter on the selection of test protocols. The Agency requests that all test data be developed according to the TSCA Good Laboratory Practice Standards in 40 CFR part 792. Failure to do so may result in EPA's finding that submitted data are insufficient to reasonably evaluate the health effects and public health implications of these chemical substances.

EPA urges SNUN submitters to provide detailed information on human and environmental exposures that would result or could reasonably be anticipated to result from the significant new uses of the chemical substances listed in Table 2 and Table 3 of Unit I.A. and at § 721.9582 of the proposed regulation. In addition, EPA encourages persons to submit information on potential benefits of these chemical substances and information on risks posed by these chemical substances compared to risks posed by possible substitutes.

## IX. Economic Considerations

EPA has evaluated the potential costs of establishing a SNUR for PFOS listed in Table 2 and Table 3 of Unit I.A. These potential costs are related to the submission of SNUNs, the export notification requirements of TSCA section 12(b), and the development of test data. EPA notes that, with the possible exception of the export notification requirements, these costs will not be incurred by any company unless that company decides to pursue a significant new use as defined in this SNUR.

### A. SNUNs

Because of uncertainties related to predicting the number of SNUNs that will be submitted as a result of this SNUR, EPA is unable to calculate the total annual cost of compliance with the final rule. However, EPA estimates that the cost for preparation and submission of a SNUN ranges from approximately $8,500 to $9,800, which includes a $2,500 user fee (Ref. 11). EPA notes that small businesses with annual sales of less than $40 million are subject to a reduced user fee of $100.

Based on past experience with SNURs and the low number of SNUNs which are submitted on an annual basis, EPA believes that there would be few, if any, SNUNs submitted as a result of this SNUR. Furthermore, no company is required to submit a SNUN for the chemicals listed in this SNUR unless that company decides to begin manufacture or importation those chemicals. As a result, EPA expects that companies would be able to determine if the burden of submitting a SNUN would be likely to create significant adverse economics impacts for the company prior to incurring SNUN-related costs.

### B. Export Notification

As noted in Unit II.A., persons who intend to export a chemical substance identified in a proposed or final SNUR are subject to the export notification provisions of TSCA section 12(b) (15 U.S.C. 2611 (b)). These provisions require that a company notify EPA of the first shipment to a particular country of an affected chemical. EPA believes that most companies comply with these provisions by compiling a list of products that are subject to TSCA section 12(b) reporting. Outgoing orders are checked to see if the chemical or product is on the list, and whether it is the first shipment to the importing country or the first shipment of the calendar year to that country. If so, a form letter is sent to EPA. In most cases, the entire process is computerized. The estimated cost of the TSCA section 12(b)(1) export notification, which would be required for the first export to a particular country of a chemical subject to the rule, is estimated to be $83.38 for the first time that an exporter must comply with TSCA section 12(b)(1) export notification requirements, and $19.08 for each subsequent export notification submitted by that exporter (Ref. 12).

EPA is unable to estimate the total number of TSCA section 12(b) notifications that will be received as a result of this SNUR, or the total number of companies that will file these notices. However, EPA expects that the total cost of complying with the export notification provisions of TSCA section 12(b) will be limited based on historical experience with TSCA section 12(b) notifications, the relatively few companies with fluorocarbon production capabilities, and the limited number of chemicals listed in this SNUR. If companies were to manufacture any of the chemicals covered by this SNUR for export only, these companies would incur costs associated with export notification even if these companies decided to forgo any domestic significant new use. EPA is not aware of any companies in this situation, and expects that any potential impact would be limited to the small burden of export notification.

### C. Testing

In Unit VIII., EPA has identified certain tests that SNUN submitters may choose to conduct to assist EPA in evaluating the risks posed by these chemical substances when used for an intended significant new use. The estimated cost of these tests ranges from $1,450 for the acute oral toxicity test using the up-or-down method to $2.24 million for the 2-species carcinogenicity test by the inhalation route (Ref. 13).

As noted in Unit VIII., development of any particular test data would be at the discretion of the submitter of the SNUN. EPA is not able to predict which specific tests will be conducted for chemicals that are the subject of SNUNs. However, EPA notes that companies would be able to determine if the burden of developing test data would be likely to create significant adverse economic impacts for the company prior to incurring these testing costs.

## X. References

These references have been placed in the official record that was established under docket control number OPPTS-50639 for this rulemaking as indicated in Unit I.B.2. Reference documents identified with an administrative record number (AR) are cross-indexed to non-regulatory, publicly accessible information files maintained in the TSCA Nonconfidential Information Center. Copies of these documents can be obtained as described in Unit I.B.2.

1. (AR226-0620) Sulfonated Perfluorochemicals in the Environment: Sources, Dispersion, Fate, and Effects. 3M. St. Paul, MN. March 1, 2000.

2. (AR226-0547) The Science of Organic Fluorochemistry. 3M. St. Paul, MN. February 5, 1999.

3. (AR226-0548) Perfluorooctane Sulfonate: Current Summary of Human Sera, Health and Toxicology Data. 3M. St. Paul, MN. January 21, 1999.

4. (AR226-0550) Fluorochemical Use, Distribution, and Release Overview. 3M. St. Paul, MN. May 26, 1999.

5. Rice, Cody. Domestic Manufacturers or Importers of PFOS Chemicals Other Than 3M. USEPA/OPPT/EETD. Washington, DC. August 31, 2000.

6. (AR226-0600) Weppner, William A. Phase-out Plan for POSF-Based Products. 3M. St. Paul, MN. July 7, 2000.

7. MacKay, D., DiGuardo, A., Paterson, S., and Cowan, C.E. Evaluating the Environmental Fate of a Variety of Types of Chemicals Using the EQC Model. Environmental Toxicology and Chemistry. SETAC Press. Houston, TX. 1996. Vol. 15, No. 9, pp.1627-1637.

8. Seed, Jennifer. Hazard Assessment and Biomonitoring Data on Perfluorooctane Sulfonate—PFOS. USEPA/EPA/RAD. Washington, DC. August 31, 2000.

9. 3M Phasing Out Some of its Specialty Materials. 3M News. 3M. St. Paul, MN. May 16, 2000.

10. Guidelines for the requested fate and transport data can be found in OPPTS Harmonized Test Guidelines, Series 835. Fate, Transport And Transformation Test Guidelines. These guidelines, both Public Drafts and Finals, are available electronically in PDF (portable document format) on the EPA World Wide Web site, see Unit I.B.1., or in paper by contacting the OPP Public Docket at (703) 305-5805 or by e-mail at: opp-docket@epa.gov. Final guidelines, only, are available from the U.S. Government Printing Office Bookstore, 810 North Capitol St., NW., Washington, DC or by calling (202) 512-1800 and ordering ASCII disks or paper copies. The EQC model is available for download from the Trent University web site at http://www.trentu.ca/envmodel.

11. (AR 204-001) TSCA Section 5(a)(2) Significant New Use Rules for

Existing Chemicals. ICR #1188.06, OMB No. 2070–0038 (Undated).

12. (AR 205–001) TSCA Section 12(b) Notification of Chemical Exports. ICR #0795.10, OMB No. 2070–0030 (Undated).

13. Rice, Cody. Estimated Costs of Testing Recommended for PFOS SNUR. USEPA/OPPT/EETD. Washington, DC. July 25, 2000.

## XI. Regulatory Assessment Requirements

Under Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993), the Office of Management and Budget (OMB) has determined that SNURs are not a "significant regulatory action" subject to review by OMB, because SNURs do not meet the criteria in section 3(f) of the Executive Order.

Based on EPA's experience with past SNURs, State, local, and tribal governments have not been impacted by these rulemakings, and EPA does not have any reasons to believe that any State, local, or tribal government will be impacted by this rulemaking. As such, EPA has determined that this regulatory action does not impose any enforceable duty, contain any unfunded mandate, or otherwise have any affect on small governments subject to the requirements of sections 202, 203, 204, or 205 of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104–4).

Similarly, this action is not subject to the requirement for prior consultation with Indian tribal governments as specified in Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19,1998). Nor will this action have a substantial direct effect on States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132, entitled *Federalism* (64 FR 43255, August 10, 1999).

In issuing this proposed rule, EPA has taken the necessary steps to eliminate drafting errors and ambiguity, minimize potential litigation, and provide a clear legal standard for affected conduct, as required by section 3 of Executive Order 12988, entitled *Civil Justice Reform* (61 FR 4729, February 7, 1996).

EPA has complied with Executive Order 12630, entitled *Governmental Actions and Interference with Constitutionally Protected Property Rights* (53 FR 8859, March 15, 1988), by examining the takings implications of this proposed rule in accordance with the "Attorney General's Supplemental Guidelines for the Evaluation of Risk and Avoidance of Unanticipated Takings" issued under the Executive Order.

This action does not involve special considerations of environmental justice related issues as required by Executive Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994).

This action is not subject to Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997), because this is not an economically significant regulatory action as defined by Executive Order 12866, and this action does not address environmental health or safety risks disproportionately affecting children.

In addition, since this action does not involve any technical standards, section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note), does not apply to this action.

Pursuant to section 605(b) of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*), the Agency hereby certifies that promulgation of this SNUR will not have a significant adverse economic impact on a substantial number of small entities. A SNUR applies to any person (including small or large entities) who intends to engage in any activity described in the rule as a "significant new use." By definition of the word "new," and based on all information currently available to EPA, it appears that no small or large entities currently engage in such activity. Since a SNUR requires merely that any person who intends to engage in such activity in the future must first notify EPA (by submitting a SNUN), no economic impact will even occur until someone decides to engage in those activities. As a voluntary action, it is reasonable to presume that this decision would be based on a determination by the person submitting the SNUN that the potential benefits would outweigh the costs. Although some small entities may decide to conduct such activities in the future, EPA cannot presently determine how many, if any, there may be. EPA's experience to date is that, in response to the promulgation of over 530 SNURs, the Agency has received fewer than 15 SNUNs. Of those SNUNs submitted, none appear to be from small entities. In fact, EPA expects to receive few, if any, SNUNs from either large or small entities in response to any SNUR. Therefore, EPA believes that, the economic impact of complying with a SNUR is not expected to be significant or adversely impact a substantial number of small entities. This rationale has been provided to the Chief Counsel for Advocacy of the Small Business Administration.

According to the Paperwork Reduction Act (PRA), 44 USC 3501 *et seq.*, an agency may not conduct or sponsor, and a person is not required to respond to a collection of information that requires OMB approval under the PRA, unless it has been approved by OMB and displays a currently valid OMB control number. The OMB control numbers for EPA's regulations, after initial display in the **Federal Register** and in addition to its display on any related collection instrument, are listed in 40 CFR part 9.

The information collection requirements related to this action have already been approved by OMB pursuant to the PRA under OMB control number 2070–0038 (EPA ICR No. 1188.06). This action does not impose any burden requiring additional OMB approval. If an entity were to submit a SNUN to the Agency, the annual burden is estimated to average between 98.96 and 118.92 hours per response at an estimated reporting cost of between $5,957 and $7,192 per SNUN. This burden estimate includes the time needed to review instructions, search existing data sources, gather and maintain the data needed, and complete, review and submit the required significant new use notice, and maintain the required records. This burden estimate does not include 1 hour of technical time at $64.30 per hour estimated to be required for customer notification of SNUR requirements, or the $2,500 user fee for submission of a SNUN ($100 for businesses with less than $40 million in annual sales).

Send any comments about the accuracy of the burden estimate, and any suggested methods for minimizing respondent burden, including through the use of automated collection techniques, as instructed in Unit I.C. or to the Director, Collection Strategies Division, Office of Environmental Information,. Environmental Protection Agency (2822), 1200 Pennsylvania Ave., NW., Washington, DC 20460. Please remember to include the OMB control number in any correspondence, but do not submit any completed forms to this address.

## List of Subjects in 40 CFR Part 721

Environmental Protection, Chemicals, Hazardous materials, Recordkeeping and reporting requirements.

Dated: October 12, 2000.

**William H. Sanders, III**

*Director, Office of Pollution Prevention and Toxics.*

Therefore, it is proposed that 40 CFR chapter I be amended as follows:

## PART 721—[AMENDED]

1. The authority citation for part 721 would continue to read as follows:

**Authority:** 15 U.S.C. 2604, 2607 and 2625(e).

2. By adding new § 721.9582 to subpart E to read as follows:

**§ 721.9582 Certain perfluorooctyl sulfonates.**

(a) *Chemical substances and significant new uses subject to reporting.* (1) The chemical substances listed in Tables 1 and 2 of this paragraph are subject to reporting under this section for the significant new uses described in paragraph (a)(2) of this section.

TABLE 1.—CHEMICALS REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2001

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| 383-07-3 | 2-Propenoic acid, 2-[butyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester. |
| 423-82-5 | 2-Propenoic acid, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester. |
| 2250-98-8 | 1-Octanesulfonamide, N,N',N''-[phosphinylidynetris(oxy-2,1-ethanediyl)]tris[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-. |
| 14650-24-9 | 2-Propenoic acid, 2-methyl-, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester. |
| 30381-98-7 | 1-Octanesulfonamide, N,N'-[phosphinicobis(oxy-2,1-ethanediyl)]bis[N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt. |
| 55120-77-9 | 1-Hexanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-, lithium salt. |
| 57589-85-2 | Benzoic acid, 2,3,4,5-tetrachloro-6-[[[3-[[(heptadecafluorooctyl)sulfonyl]oxy]phenyl]amino]carbonyl]-, monopotassium salt. |
| 61660-12-6 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-[3-(trimethoxysilyl)propyl]-. |
| 67969-69-1 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-[2-(phosphonooxy)ethyl]-, diammonium salt. |
| 68156-01-4 | Cyclohexanesulfonic acid, nonafluorobis(trifluoromethyl)-, potassium salt. |
| 68329-56-6 | 2-Propenoic acid, eicosyl ester, polymer with 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, hexadecyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and octadecyl 2-propenoate. |
| 68555-91-9 | 2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, polymer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-methyl-2-propenoate. |
| 68555-92-0 | 2-Propenoic acid, 2-methyl-, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, polymer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-methyl-2-propenoate. |
| 68608-14-0 | Sulfonamides, C4-8-alkane, perfluoro, N-ethyl-N-(hydroxyethyl), reaction products with 1,1'-methylenebis[4-isocyanatobenzene]. |
| 68909-15-9 | 2-Propenoic acid, eicosyl ester, polymers with branched octyl acrylate, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl acrylate, 2-[methyl[(nonafluorobutyl) sulfonyl]amino]ethyl acrylate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl acrylate, 2-[methyl[(undecafluoropentyl) sulfonyl]amino]ethyl acrylate, polyethylene glycol acrylate Me ether and stearyl acrylate. |
| 70776-36-2 | 2-Propenoic acid, 2-methyl-, octadecyl ester, polymer with 1,1-dichloroethene, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, N-(hydroxymethyl)-2-propenamide, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate and 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate. |
| 73772-32-4 | 1-Propanesulfonic acid, 3-[[3-(dimethylamino)propyl][(tridecafluorohexyl)sulfonyl]amino]-2-hydroxy-, monosodium salt. |
| 81190-38-7 | 1-Propanaminium, N-(2-hydroxyethyl)-3-[(2-hydroxy-3-sulfopropyl) [(tridecafluorohexyl)sulfonyl]amino]-N,N-dimethyl-, hydroxide, monosodium salt. |
| 94133-90-1 | 1-Propanesulfonic acid, 3-[[3-(dimethylamino)propyl][(heptadecafluorooctyl)sulfonyl]amino]-2-hydroxy-, monosodium salt. |
| 117806-54-9 | 1-Heptanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-, lithium salt. |
| 127133-66-8 | 2-Propenoic acid, 2-methyl-, polymers with Bu methacrylate, lauryl methacrylate and 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl methacrylate. |
| 129813-71-4 | Sulfonamides, C4-8-alkane, perfluoro, N-methyl-N-(oxiranylmethyl). |
| 148240-78-2 | Fatty acids, C18-unsatd., trimers, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl esters. |
| 148240-79-3 | Fatty acids, C18-unsatd., trimers, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl esters. |
| 148240-80-6 | Fatty acids, C18-unsatd., trimers, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl esters. |
| 148240-81-7 | Fatty acids, C18-unsatd., trimers, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl esters. |
| 148240-82-8 | Fatty acids, C18-unsatd., trimers, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl esters. |
| 148684-79-1 | Sulfonamides, C4-8-alkane, perfluoro, N-(hydroxyethyl)-N-methyl, reaction products with 1,6-diisocyanatohexane homopolymer and ethylene glycol. |
| 178535-22-3 | Sulfonamides, C4-8-alkane, perfluoro, N-ethyl-N-(hydroxyethyl)-, polymers with 1,1'-methylenebis[4-isocyanatobenzene] and polymethylenepolyphenylene isocyanate, 2-ethylhexyl esters, Me Et ketone oxime-blocked. |
| P-83-1102 | Fatty acids, linseed-oil, dimers, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl esters. |
| P-84-1163 | Propanoic acid, 3-hydroxy-2-(hydroxymethyl)-2-methyl-, polymer with 2-ethyl-2-(hydroxymethyl)-1,3-propanediol and N,N',2-tris(6-isocyanatohexyl)imidodicarbonic diamide, reaction products with N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-1-octanesulfonamide and N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-1-heptanesulfonamide, compds. with triethylamine. |

TABLE 1.—CHEMICALS REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2001—Continued

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| P-84–1171 | Propanoic acid, 3-hydroxy-2-(hydroxymethyl)-2-methyl-, polymer with 1,1'-methylenebis[4-isocyanatobenzene] and 1,2,3-propanetriol, reaction products with N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-1-octanesulfonamide and N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-1-heptanesulfonamide, compds. with morpholine. |
| P-86–0301 | Sulfonamides, C4-8-alkane, perfluoro, N-(hydroxyethyl)-N-methyl, reaction products with 12-hydroxysteanc acid and 2,4-TDI, ammonium salts. |
| P-89–0799 | Sulfonamides, C4-8-alkane, perfluoro, N-ethyl-N-(hydroxyethyl), reaction products with 2-ethyl-1-hexanol and polymethylenepolyphenylene isocyanate. |
| P-94–0545 | 1-Hexadecanaminium, N,N-dimethyl-N-[2-[(2-methyl-1-oxo-2-propenyl)oxy]ethyl]-, bromide, polymers with Bu acrylate, Bu methacrylate and 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate. |
| P-94–0927 | 2-Propenoic acid, 2-methyl-, 2-methylpropyl ester, polymer with 2,4-diisocyanato-1-methylbenzene, 2-ethyl-2-(hydroxymethyl)-1,3-propanediol and 2-propenoic acid, N-ethyl-N-(hydroxyethyl)perfluoro-C4-8-alkanesulfon amides-blocked. |
| P-94–2205 | Polymethylenepolyphenylene isocyanate and bis(4-NCO-phenyl)methane reaction products with 2-ethyl-1-hexanol, 2-butanone, oxime, N-ethyl-N-(2- hydroxyethyl)-1-C4-C8 perfluoroalkanesulfonamide. |
| P-94–2206 | Siloxanes and Silicones, di-Me, mono[3-[(2-methyl-1-oxo-2-propenyl)oxy]propylgroup]-terminated, polymers with 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate and stearyl methacrylate. |
| P-96–1645 | Fatty acids, C18-unsatd., dimers, 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl esters. |
| P-97–0790 | 1-Decanaminium, N-decyl-N,N-dimethyl-, salt with 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-1-octanesulfonic acid (1:1). |
| P-98–0251 | 2-Propenoic acid, butyl ester, polymers with acrylamide, 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate and vinylidene chloride. |
| P-98–1272 | 2-Propenoic acid, 2-methyl-, 3-(trimethoxysilyl)propyl ester, polymers with acrylic acid, 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate and propylene glycol monoacrylate, hydrolyzed, compds. with 2,2'-(methylimino)bis[ethanol]. |
| P-99–0188 | Hexane, 1,6-diisocyanato-, homopolymer, N-(hydroxyethyl)-N-methyl perfluoro C4-8-alkane sulfonamide- and stearyl alc.-blocked. |
| P-99–0319 | Poly(oxy-1,2-ethanediyl), .alpha.-[2-(methylamino)ethyl]-.omega.-[(1,1,3,3-tetramethylbutyl)phenoxy]-, N-[(perfluoro-C4-8-alkyl)sulfonyl] derivs.. |

TABLE 2.—CHEMICALS SUBJECT TO VOLUME CAP RESTRICTIONS ON OR AFTER JANUARY 1, 2001 AND REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2003

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| 307–35–7 | 1-Octanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- |
| 307–51–7 | 1-Decanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heneicosafluoro- |
| 376–14–7 | 2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester |
| 423–50–7 | 1-Hexanesulfonyl fluoride, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro- |
| 754–91–6 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- |
| 1652–63–7 | 1-Propanaminium, 3-[[(heptadecafluorooctyl)sulfonyl]amino]-N,N,N-trimethyl-, iodide |
| 1691–99–2 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)- |
| 1763–23–1 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- |
| 2795–39–3 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, potassium salt |
| 2991–51–7 | Glycine, N-ethyl-N-[(heptadecafluorooctyl)sulfonyl]-, potassium salt |
| 4151–50–2 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro- |
| 17202–41–4 | 1-Nonanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,9-nonadecafluoro-, ammonium salt |
| 24448–09–7 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-N-methyl- |
| 25268–77–3 | 2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester |
| 29081–56–9 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, ammonium salt |
| 29117–08–6 | Poly(oxy-1,2-ethanediyl), .alpha.-[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl]-.omega.-hydroxy- |
| 29457–72–5 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, lithium salt |
| 31506–32–8 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-methyl- |
| 38006–74–5 | 1-Propanaminium, 3-[[(heptadecafluorooctyl)sulfonyl]amino]-N,N,N-trimethyl-, chloride |
| 38850–58–7 | 1-Propanaminium, N-(2-hydroxyethyl)-N,N-dimethyl-3-[(3-sulfopropyl)[(tridecafluorohexyl)sulfonyl]amino]-, inner salt |
| 67584–42–3 | Cyclohexanesulfonic acid, decafluoro(pentafluoroethyl)-, potassium salt |
| 67906–42–7 | 1-Decanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,9,9,10,10,10-heneicosafluoro-, ammonium salt |
| 68298–62–4 | 2-Propenoic acid, 2-[butyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, telomer with 2-[butyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, methyloxirane polymer with oxirane di-2-propenoate, methyloxirane polymer with oxirane mono-2-propenoate and 1-octanethiol |
| 68541–80–0 | 2-Propenoic acid, polymer with 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate and octadecyl 2-propenoate |
| 68555–90–8 | 2-Propenoic acid, butyl ester, polymer with 2-[[(heptadecafluorooctyl)sulfonyl] methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate and 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate |
| 68586–14–1 | 2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, telomer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, .alpha.-(2-methyl-1-oxo-2-propenyl)-.omega.-hydroxypoly(oxy-1,2-ethanediyl), .alpha.-(2-methyl-1-oxo-2-propenyl)-.omega.-[(2-methyl-1-oxo-2-propenyl)oxy]poly(oxy-1,2-ethanediyl), 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 1-octanethiol |

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TABLE 2.—CHEMICALS SUBJECT TO VOLUME CAP RESTRICTIONS ON OR AFTER JANUARY 1, 2001 AND REQUIRING A SIGNIFICANT NEW USE NOTICE ON OR AFTER JANUARY 1, 2003—Continued

| CAS No./PMN | Ninth Collective Index chemical name |
|---|---|
| 68649–26–3 | 1-Octanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-, reaction products with N-ethyl-1,1,2,2,3,3,4,4,4-nonafluoro-N-(2-hydroxyethyl)-1-butanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-1-heptanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-(2-hydroxyethyl)-1-hexanesulfonamide, N-ethyl-1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-(2-hydroxyethyl)-1-pentanesulfonamide, polymethylenepolyphenylene isocyanate and stearyl alc. |
| 68867–60–7 | 2-Propenoic acid, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl ester, polymer with 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and .alpha.-(1-oxo-2-propenyl)-.omega.-methoxypoly(oxy-1,2-ethanediyl) |
| 68867–62–9 | 2-Propenoic acid, 2-methyl-, 2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl ester, telomer with 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 1-octanethiol and .alpha.-(1-oxo-2-propenyl)-.omega.-methoxypoly(oxy-1,2-ethanediyl) |
| 68891–96–3 | Chromium, diaquatetrachloro[.mu.-[N-ethyl-N-[(heptadecafluorooctyl)sulfonyl]glycinato-.kappa.O:.kappa.O']]-.mu.-hydroxybis(2-methylpropanol)di- |
| 68958–61–2 | Poly(oxy-1,2-ethanediyl), .alpha.-[2-[ethyl[(heptadecafluorooctyl)sulfonyl]amino]ethyl]-.omega.-methoxy- |
| 70225–14–8 | 1-Octanesulfonic acid, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-, compd. with 2,2'-iminobis(ethanol) (1:1) |
| 71487–20–2 | 2-Propenoic acid, 2-methyl-, methyl ester, polymer with ethenylbenzene, 2-[[(heptadecafluorooctyl)sulfonyl]methylamino]ethyl 2-propenoate, 2-[methyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-propenoate, 2-[methyl[(undecafluoropentyl)sulfonyl]amino]ethyl 2-propenoate and 2-propenoic acid |
| 91081–99–1 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-(2-hydroxyethyl)-N-methyl-, polymer with(chloromethyl)oxirane, 1,1,2,2,3,3,4,4,4-nonafluoro-N-(2-hydroxyethyl)-N-methyl-1-butanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,7-pentadecafluoro-N-(2-hydroxyethyl)-N-methyl-1-heptanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,6-tridecafluoro-N-(2-hydroxyethyl)-N-methyl-1-hexanesulfonamide and 1,1,2,2,3,3,4,4,5,5,5-undecafluoro-N-(2-hydroxyethyl)-N-methyl-1-pentanesulfonamide, hexanedioate (ester) |
| 98999–57–6 | Sulfonamides, C7-8-alkane, -perfluoro, N-methyl-N-[2-[(1-oxo-2-propenyl) oxy]ethyl], polymers with 2-ethoxyethyl acrylate, glycidyl methacrylate and N,N,N-trimethyl-2-[(2-methyl-1-oxo-2-propenyl)oxy]ethanaminiumchloride |
| 182700–90–9 | 1-Octanesulfonamide, 1,1,2,2,3,3,4,4,5,5,6,6,7,7,8,8,8-heptadecafluoro-N-methyl-, reaction products with benzene-chlorine-sulfur chloride (S2Cl2) reaction products chlorides |
| L-92–0151 | 2-Propenoic acid, 2-methyl-, butyl ester, polymer with 2-methyl-, 2-[ethyl [(heptadecafluorooctyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(nonafluorobutyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(pentadecafluoroheptyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, 2-[ethyl[(tridecafluorohexyl)sulfonyl]amino]ethyl 2-methyl-2-propenoate, and 2-propenoic acid |
| P-80–0183 | Sulfonamides, C4-8-alkane, perfluoro, N-[3-(dimethylamino)propyl], reaction products with acrylic acid |
| P-86–0958 | 2-Propenoic acid, 2-methyl-, dodecyl ester, polymers with 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl acrylate and vinylidene chloride |
| P-90–0111 | Sulfonamides, C4-8-alkane, perfluoro, N-methyl-N-[(3-octadecyl-2-oxo-5-oxazolidinyl)methyl] |
| P-91–1419 | Poly(oxy-1,2-ethanediyl), .alpha.-hydro-.omega.-hydroxy-, polymer with 1,6-diisocyanatohexane, N-(2-hydroxyethyl)-N-methyl perfluoro C4-8-alkane sulfonamide-blocked |
| P-93–1444 | 2-Propenoic acid, 2-methyl-, dodecyl ester, polymers with N-(hydroxymethyl)-2-propenamide, 2-[methyl[(perfluoro-C4-8-alkyl)sulfonyl]amino]ethyl methacrylate, stearyl methacrylate and vinylidene chloride |
| P-95–0120 | Sulfonamides, C4-8-alkane, perfluoro, N,N'-[1,6-hexanediylbis[[2-oxo-3,5-oxazolidinediyl)methylene]]bis[N-methyl- |
| P-96–1262 | Sulfonic acids, C6-8-alkane, perfluoro, compds. with polyethylene-polypropylene glycol bis(2-aminopropyl) ether |
| P-96–1424 | 2-Propenoic acid, 2-methyl-, 2-(dimethylamino)ethyl ester, telomers with 2-[ethyl[(perfluoro-C4-8-alkylsulfonyl]amino]ethyl methacrylate and 1-octanethiol, N-oxides |
| P-96–1433 | Sulfonamides, C4-8-alkane, perfluoro, N-[3-(dimethyloxidoamino)propyl], potassium salts |

(2) The significant new uses are:

(i) Any manufacture or import for any use of any chemical listed in Table 1 of paragraph (a)(1) of this section on or after January 1, 2001.

(ii) Any manufacture or import for any use of any one or more of the chemicals listed in Table 2 of paragraph (a)(1) of this section in excess of an aggregate volume for all of these chemicals of 1,100,000 pounds per person per calendar year on or after January 1, 2001 and before January 1, 2003.

(iii) Any manufacture or import for any use of any of the chemicals listed in Table 2 of paragraph (a)(1) of this section on or after January 1, 2003.

(b) [Reserved].

[FR Doc. 00–26751 Filed 10–17–00; 8:45 am]
BILLING CODE 6560–50–S

# LUBECK

## Public Service District

P.O. Box 700, Washington, West Virginia 26181-0700

James M. Cox, Manager

Telephone (304) 863-3341
Facsimile (304) 863-3791

October 31, 2000

**COMMISSIONERS:**
James E. Smith, Chairman
David D. Steele, Secretary
Lee "Ox" Johnson, Treasurer

Dear Lubeck PSD Customers:

In compliance with the Safe Drinking Water Act Amendments and the West Virginia Bureau for Public Health, Lubeck Public Service District (LPSD) must provide our customers with an annual water quality report, which was done in an advertisement in The Parkersburg News and The Parkersburg Sentinel on July 1, 2000.

In order to insure that tap water is safe to drink, the U. S. Environmental Protection Agency (EPA) prescribes regulations which limit the amount of certain contaminants in water provided by public water systems. The presence of those contaminants does not necessarily indicate that water poses a health risk. LPSD routinely monitors your drinking water according to Federal and State laws. The July 1, 2000 advertisement provided the results of monitoring for regulated contaminants.

An unregulated chemical for which LPSD has monitoring data is ammonium perfluorooctanoate (also known as PFOA, APFO, FC-143, and C-8), which is used at DuPont Washington Works. Attention has recently been focused on PFOA because it is one of several products being phased out by its manufacturer, the 3M Company.

PFOA is a persistent chemical that is slow to be eliminated from the blood stream of people who have been exposed to it. DuPont reports that it has toxicological and epidemiological data to support confidence that exposure guidelines established by DuPont are protective of human health.

Although PFOA is unregulated for drinking water purposes (that is, it has no limit established by a regulatory agency), the DuPont Company has established its own drinking water guideline of 1 part per billion. The DuPont Company has advised the District that low concentrations have been found in drinking water at Washington Works (0.2 parts per billion, or ppb, as of August 2000) and in the LPSD wells (0.2, 0.5, and 0.1 ppb, also as of August 2000). These levels are below the DuPont guideline and DuPont has advised the District that it is confident these levels are safe.

LPSD is committed to providing safe water to our customers. If you have any questions, you can reach the District at 863-3341 or Dawn Jackson at DuPont at 863-2513.

Sincerely,

James M. Cox
Manager

6329-230
6329.231
5 pgs

Re-faxing
5/19

**GROUP: APFO TOX WORKING GROUP**

**Basics**

| | |
|---|---|
| Group name: | APFO TOX WORKING GROUP |
| Group type: | Multi-purpose |
| Description: | CONFERENCE CALL LIST |
| Members: | peter.thomford@covance.com 608.242.2736<br>Paul Lieder<br>David Farrar-ICI-(Fax 011-44-1928-515-146<br>CElcombe-Dundee-(011-44-1382-669-993)<br>R Jung-Hoechst(011-49-6190-807-354<br>M Mistrorigo -Miteni(011-39-0445-963-847)<br>G Costa - Verona (011-39-0445-963-847)<br>S Shin-ya - Asahi (011-81332-11-7671)<br>G Kennedy - Dupont (302-366-5207)<br>G Lin - Daikin (914-365-9515)<br>J O'Connor - Dupont (302-366-5503) |

**Administration**

| | |
|---|---|
| Owners: | Rosamaria Maldonado/US-Corporate/3M/US |
| Administrators: | Rosamaria Maldonado/US-Corporate/3M/US |
| Foreign directory sync allowed: | Yes |

**1ST CLASS MAIL**
SD P. Thomford
ST D. Aldridge
SC P. Meier-Pesik

**ADDL. DIST.**
L. Biegel

**TOX FILE**

CLI 0304

Covance Laboratories
Madison, WI
MAY 19 1999
Received # 15

May 19, 1999

To: APFO Sub-committee: APME
From: Paul H. Lieder

Attached is the preliminary analytical results for the animals at the end of the dosing phase of the study. One animal in the low dose group died during the dosing phase of the study as did one high dose animal.

In summary, the low dose and mid dose animals had similar serum and liver values. Three high dose animals became very sick during dosing and consequently dosing was terminated mid-study. The serum and liver PFOA levels from these animals appear to be much lower than the high dose animals still receiving compound, indicating a relatively (vs. humans) short half-life(?)

The results of the histopathology and hormone analysis will be sent by Peter Thomford shortly.

Regards,
PHL

6 Month Capsule Toxicity Study wit APFO in Cynomolgus Monkeys
Sera and Liver Analysis at Study Termination
Preliminary Results: 18/5/99

Part 1: Serum Analysis

Control Group
0 mg/kg/da
I.D. Conc (ug/mL)

5709 0.393
5714 0.229
5715 0.099
5718 0.215
5719 0.322
5725 0.142
**Average=0.233**

---

Low Dose
3 mg/kg/da
Conc (ug/mL)

5702 49.7
5706 66.9
5716 45.7
5721 47.7
5723 62.2
**Average=54.5**

---

Mid-high Dose
10 mg/kg/da
I.D. Conc (ug/mL)

5707 87.0
5708 33.3
5709 49.4
5712 104
5717 81.0
5718 67.6
**Average= 70.5**

---

High Dose
20 to 30 mg/kg/da
On dosing (receiving compound at end of 6 months)
I.D. Conc (ug/mL)

5703 60.8
5713 192
**Average= 125**

High Dose—Moribund-sacrificed mid-study
I.D. Conc (ug/mL)

**5724 403**

High Dose-Recovery-removed from dosing mid-study due to toxicity
I.D. Conc (ug/mL)

5703 7.76
5711 0.909
5722 6.90
**Average=5.19**

Part 2: Liver Analysis

Control Group
0 mg/kg/da
I.D. Conc (ug/g)

5709 <MDL
5714 <MDL
5715 0.0474
5725 <MDL
**Average=0.0474**

---

Low Dose
0 mg/kg/da
I.D. Conc (ug/g)

5702 5.77

5706 6.81
5716 4.12
5723 6.80
**Average=5.88**

---

Mid-high Dose
0 mg/kg/da
I.D. Conc (ug/mL)

5707 8.7
5708 3.61
5709 5.64
5719 5.50
**Average=5.86**

High Dose
30—20mg/kg/da

On Compound—receiving compound at end of study
I.D. Conc (ug/mL)

5704 8.03
5713 28.3
**Average= 17.2**

High Dose-Recovery-removed from dosing mid-study due to toxicity
I.D. Conc (ug/mL)

5703 1.05
5711 0.0278
5722 1.218
Average=0.766

5724 30.3 Moribund-sacrificed mid-study

000788

**mTitle:**
**Toxicity of Ammmonium Perfluorooctanoate (APFO) in Cynomolgus Monkeys after Twenty-Six Weeks of Oral Dosing**

Authors in sequence to be determined

EID101453

**Abstract:**

The purpose of this study was to identify the earliest clinically measurable biological response from repeated daily exposure to ammonium perfluorooctanoate (APFO) and to correlate this response to serum concentrations for purposes of risk assessment and medical monitoring of exposed populations. Groups of male Cynomolgus monkeys received daily doses of 0 (six per sex), 3 (four per sex), 10 (six per sex), or 30/20 (six per sex) mg/kg/day ammonium perfluorooctanoate for 26 weeks by gelatin capsule via gastric intubation. The high-dose group received 30 mg/kg for 11 days, after which dosing was discontinued for ten days (through study dosing day 21) due to signs of few feces, low food consumption and weight loss. Beginning on study dosing day 22, the high-dose group was given 20 mg/kg and remained on this dose through termination of the dosing phase with the exception of three animals for which dosing was discontinued between days 43 and 81 due to emaciated appearance, weight loss, few or no feces and low or no food consumption. Two males in the control and mid-dose (10 mg/kg) group were followed for ninety days after cessation of dosing and observed for reversibility, persistence, or delayed occurrence of toxic effects. One high-dose male was sacrificed in moribund condition on day 29 after exhibiting hypoactivity, body cold to the touch, few feces, low or no food consumption and weight loss. One low-dose male was sacrificed in moribund condition on day 137 after exhibiting limited use and paralysis of the hind limbs, ataxic and hypoactive behavior, few feces and no food consumption. There were no effects on estrone, estradiol, estriol, testosterone, cholecystokinin, thyroid stimulating hormone, or thyroxin. Triiodothyronine tremded down in three high-dose monkeys who were taken off compound for the remainder of the study. T3 values for these monkeys trended up after cessation of dosing.. At all dose groups, significant increases in mean absolute liver weight and mean liver-to-body weight percentages were observed. The liver weight increase in the low dose animals occurred at a mean serum concentration of approximately 50 ppm. Mean liver-to-brain weight ratios were increased in the mid-dose group. Organ weight and body weight effects were not evident at the end of the recovery period. No test material related macroscopic or microscopic changes were observed in any organs, including liver, adrenal, spleen, pancreas and testis. The liver weight increase at the low dose occurred at serum concentrations which overlap those which have been observed in some workers with high exposure; therfore, the liver enlargement was considered a significant effect and the low dose of 3 mg/kg/day is characterized as the lowest observed effect level (LOEL).

UK00254

## INTRODUCTION

Ammonium perfluorooctanoate ($C_7F_{15}COO^-NH_4^+$; C.A.S. Registry number 3825-26-1; ammonium perfluorocaprylate, ammonium pentadecafluorooctanoate; FLUORAD ™ Brand FC-143 Fluorochemical Surfactant; C-8; APFO[1]; PFOA[1]; Mol. Wgt. 431.1 g) is used as a surfactant in the aqueous polymerization of fluorinated monomers such as tetrafluoroethylene.

The assumed presence of PFOA in general population blood and in exposed worker blood, coupled with the observed persistence in workers, has led to extensive research into the potential health risk of exposure to APFO (Guy, 1972; Venkateswarlu, 1975; Guy and Taves, 1976; Guy et al, 1976; Belisle and Hagen, 1978; Belisle and Hagen, 1980; Singer, 1979; Paez, 1980; Ubel, 1980; Belisle, 1981; Yamamoto, 1989).

APFO has been produced since the early 1950's. Medical monitoring of employees involved in PFOA production began in 1976, by measuring serum levels of organic fluorine (OF) and performing medical assessments. Serum PFOA levels in production workers have been measured as high as 114 ppm. Eighty percent of the workers had serum PFOA levels less than 10 ppm. Average serum concentrations have been approximately 2 ppm and individual values have range from 0 to 114 ppm.

Past studies have demonstrated that the primary target organ for APFO-induced toxicity is the liver (refs). The hepatotoxicity manifests as increased liver weights, hepatocellular hypertrophy, liver degeneration, increases in liver enzymes, necrosis of the liver, and proliferation of endoplasmic reticulum, microsomes, and peroxisomes (rats and mice only) yet does not result in hypolipidemia (Pastoor et al, 1987). Many of these effects were demonstrated to be reversible when animals were provided with a recovery period.

APFO has been demonstrated to be a peroxisome proliferator in the rat. APFO exposure in the rat was found to be associated with tumors in the liver, Leydig cell, and pancreatic acinar cell. Peroxisome proliferation is associated with: increases in number and volume of peroxisomes; an increase in DNA synthesis and liver growth; and liver, Leydig cell, and pancreatic acinar cell tumors. Several known peroxisome proliferators (clofibrate, HCFC-123, methylclofenapate, and Wyeth-14,643) are reported to induce this triad of tumors in rats. The phenomenon of peroxisome proliferation is not uniform across all species. While rats and mice are particularly sensitive to this phenomenon, guinea pigs, cats, dogs and primates (including man), are predominantly non-responsive.

As noted above, chronic dietary studies in rats have produced hepatocellular adenoma, pancreatic acinar cell adenoma and Leydig cell adenomas (refs). The mechanisms of tumor formation have been studied. Hepatocellular tumors in the rat may arise from the proliferation of peroxisomes (refs). In investigating the role of increased cholecystokinin (CCK) or $CCK_A$ receptor binding as possible mechanism for pancreatic tumors, APFO

failed to bind directly to the $CCK_A$ receptor and it failed to inhibit trypsin, a common mechanism for increasing plasma CCK levels (ref). *In vivo* studies with Wyeth-14, 643, a model peroxisome proliferator, suggest that these peroxisome proliferators produce pancreatic tumors by cholestasis, which may be responsible for the decrease in bile acid output which contributes to the increase in plasma CCK levels (Obourn et al, 1997). Therefore, the pancreatic tumors produced by APFO may be secondary to hepatic cholestasis. The Leydig-cell tumors appear to arise from an induction of aromatase with a consequent increased conversion of testoterone to estradiol an

A retrospective cohort mortality study of workers engaged in APFO production found no significantly increased cause-specific standardized mortality ratio, although prostate showed a non-significant excess (4 cases observed, 2.1 expected) (Gilliland and Mandel, 1993). Hepatic toxicity and hypolipidemia have not been associated with the PFOA levels measured in APFO production workers (Gilliland and Mandel, 1993; Gilliland, 1992; Gilliland and Mandel, Am J Ind Med 129:560-568; Olsen et al, 2000). Based on toxicological findings that suggested PFOA may increase estradiol due to an induction of hepatic aromatase activity, Gilliland examined and found a positive association between serum total organic fluorine levels, used as an approximate measurement for PFOA, and estradiol (Gilliland, 1992). Gilliland and Mandel also observed that serum total organic fluorine negatively modulated the effect alcohol has on high-density lipoprotein (HDL) levels and exacerbated the effect that obesity has on hepatic enzyme tests among these workers (Gilliland and Mandel, Am J Ind Med 129:560-568). However, an analysis of two subsequent medical surveillance examinations on this APFO production workforce, which specifically assayed for PFOA, did not confirm these associations (Olsen et al, 1998 and Olsen et al, 2000). Neither did Olsen et al find an association between plasma cholecystokinin and measured serum PFOA levels (Olsen et al, 2000).

The occupational exposure limit in current use is the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV®) of 0.01 $mg/m^3$, as an 8-hour, time-weighted average with a notation for skin exposure and an A3 designation for animal carcinogen with questionable relevance to humans [13, 45]

The purpose of this study was to assess the effect of APFO on critical enzyme levels, hormones, and other selected biochemical parameters when administered daily by oral capsule to cynomolgus monkeys for at least 26 weeks and to assess reversibility, persistence, or delayed occurrence of toxic effects for 13 weeks after the 26-week treatment period.

## MATERIALS AND METHODS

EID101456

***Animals and husbandry.*** Young adult to adult male Cynomolgus monkeys were obtained from Covance Research Products Inc. (Denver, PA) on August 25, 1998, and

weighed 3.2 to 4.5 Kg at initiation of treatment. Animals were acclimated for 35 days before initiation of treatment. Each animal was assigned a permanent number upon arrival and identified with a collar tag. The animals were housed individually in suspended stainless-steel cages. The animal room was environmentally controlled to maintain 18 to 29°C, a relative humidity of 30 to 70%, and a 12-hr light/dark cycle.

Certified primate diet, (#8726C,Harlan Teklad) was provided once or twice daily along with fruits or additional supplements. Water was provided *ad libitum*. Samples of the water were analyzed for specific microorganisms and contaminants. During the study, several animals experiencing low food consumption in the high-dose group were offered supplements to rehydrate and stimulate food consumption.

Animals were assigned to treatment groups using a computerized blocking procedure designed to achieve body weight balance within each treatment group.

***Materials, dose preparation and treatment.*** Ammonium perfluoroocatnoate (APFO, lot 332) was obtained from 3M (Saint Paul MN; purity xxx). Gelatin capsules (Torpac, Inc., Fairfield, NJ, Size No. 2, Lot No. 122932) containing the appropriate dose of APFO were used for dose administration. Control animals received empty gelatin capsules. Capsules were prepared at least weekly. Individual daily doses were calculated based on the most recently recorded body weights. The dose preparations were stored at room temperature. Since the APFO was added directly to capsules as supplied without the use of a vehicle, no dose analysis was performed.

Dose levels were 0 (control group, n = 6), 3 mg/kg (low-dose group, n = 4), 10 mg/kg (mid-dose group, n = 6) or 30 reduced to 20 (30/20) mg/kg (high-dose group, n = 6). The capsules were administered orally once a day, seven days per week, for at least 26 weeks (183 days) except as noted below. One female in the low-dose group needed to be replaced for non-treatment-related issues. The replacement animal (I05721) received his initial dose on day 17. High-dose animals were initiated on 30 mg/kg; however, due to toxicity, dosing was discontinued from day 12 through 21 and recommenced on day 22 at 20 mg/kg. Also due to toxicity, dose administration was also discontinued for three high-dose animals on either day 43 (I05711), day 66 (I05722) or day 81 (I05703). Two animals in both the control and mid-dose groups were designated as recovery group animals and were observed for reversibility, persistence, or delayed occurrence of toxic effects for 13 weeks after the 26-week treatment period.

***Observations.*** The animals were observed twice daily for general health, behavior and qualitative food consumption. Ophthalmic examinations were conducted on each animal before treatment and on weeks 27 and 40 (recovery animals). Body weights were recorded weekly before initiation of treatment, on the day before initiation of treatment for dose calculation, on the first day of treatment, and weekly thereafter.

***Determination of blood hormones.*** Blood was collected from the femoral vein of non-fasted animals 18, 8, and 4 days prior to initiation of treatment and on days 35, 66, 94 and 183 of treatment. Blood collection from recovery animals occurred on days 220, 248 and 276 after initiation of treatment prior to collection. Blood samples were split into serum and plasma samples for further analysis for various hormones.

Plasma samples were analyzed for cholecystokinin and testosterone (with the exception of recovery animals in which case serum samples were used for testosterone).

Serum was analyzed for estradiol, estrone, estriol, thyroid stimulating hormone, total and free triiodothyronine, total and free thyroxin.

***Clinical pathology observations.*** A clinical laboratory evaluation was conducted before initiation of treatment, on days 31, 63, 91 and 182 of treatment and during recovery on days 217, 245, and 275 after initiation of dosing. At each sampling time, blood and urine samples were collected from animals that had been fasted overnight. Urine was collected overnight on wet ice before blood sampling; water was provided ad libitum. Blood was collected from the femoral vein. Sodium citrate was used as the anticoagulant for coagulation tests, and potassium EDTA was the anticoagulant used for hematology tests. Anticoagulants were not used for the clinical chemistry test sample collection. Blood was collected from animals with unscheduled sacrifices. Otherwise, animals with scheduled collections were bled in random order.

The following hematological parameters were measured or calculated: erythrocyte (RBC) count, leukocytes (WBC) and platelets (PLAT); hemoglobin concentration (Hb) and hematocrit (Hct); mean corpuscular volume (MCV);mean corpuscular hemoglobin (MCHh); mean corpuscular hemoglobin concentration (MCHC); differential blood cell count of segmented neutrophils (N-SEG), lymphocytes (), eosinophils () basophils (); and blood cell morphology and reticulocyte count.

Blood cell counts, hemoglobin concentration hematocrit MCV, MCH, MCHV, were determined on a Abbott Cell-Dyn 35000 hematology analyzer. Differential cell counts were determined on an Abbott Cell-Dyn 3500 and reticulocyte counts were obtained using a Miller Disk microscopy technique. Bone marrow smears were prepared at euthanization.

Coagulation parameters measured were prothrombin time, activated partial thromboplastin time, and fibrinogen.

Clinical chemistry measurements were made for glucose, urea nitrogen, creatinine, total protein, albumin, globulin, total bilirubin, cholesterol, triglycerides, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, gamma glutamyl transferase, sorbitol dehydrogenase, creatine kinase, calcium, inorganic phosphorus, sodium, potassium, chloride, bile acids, amylase, lipase, and pancreatic-specific amylase. Clinical chemical analytes were measured on a Hitachi Model 704 Chemistry Analyzer. Serum globulin concentration was calculated from the total protein and albumin concentrations.

Urine was analyzed for volume (approximately 16 hours overnight), specific gravity, pH, protein, glucose, ketones, bilirubin, blood, urobilinogen, microscopic examination of sediment, and general appearance.

***Determination of serum and liver PFOA content.*** Approximately 2 ml of whole blood (for serum) were collected from the femoral vein of non-fasted animals during week 2 and every two weeks thereafter.

A section of liver was collected at sacrifice from each animal, weighed, flash frozen in liquid nitrogen, and stored at –60 to –80 degrees C until shipped on dry ice to 3M for analysis.

Sera and liver samples were anlalyzed for PFOA content by 3M methodology (refs).

Concurrent with blood collections for PFOA determination, at least 2 ml of urine was collected on wet ice and at least 5 g of feces (overnight) were collected from each animal. Results of urine and fecal analysis will be discussed in a future paper.

***Anatomic pathology.*** After 26 weeks of treatment, four animals in the control, low and mid-dose groups, and the five surviving animals in the high-dose group were fasted overnight, anesthetized with ketamine and xylazine, weighed, exsanguinated, necropsied. Similarily the two animals in the control and mid-dose recovery groups were sacrified 13 weeks following the last dose. Necropsies were also performed on the low-dose and high-dose animals that had unscheduled sacrifices on treatment days 137 and 29, respectively. The adrenals, brain, epididymides, kidneys, liver, pancreas, testes and thyroids with parathyroid were collected and weighed. Organ to body weight and organ to brain weight ratios were calculated.

The following tissues were collected for microscopic evaluation: adrenal; aorta; brain; cecum; colon; duodenum; epididymus; esophagus; eyes; femur; gall bladder; heart; ileum; jejunum; kidney; lesions; lung; mammary gland; mesenteric lymph node; pancreas; pituitary; prostate; rectum; salivary gland (mandibular); sciatic nerve; seminal

vesicles; skeletal muscle (thigh); skin; spinal cord; spleen; sternum with bone marrow; stomach; testes; and thyroid; trachea; and, urinary bladder. Tissues were embedded in paraffin, stained with hematoxylin and eosin and examined by light microscopy.

***Palmitoyl CoA oxidase determinations.***
Samples of the right lateral lobe of the livers from each animal at scheduled and unscheduled sacrifices were flash-frozen in liquid nitrogen at necropsy and analyzed for palmitoyl CoA oxidase activity (need Cliff's method here).

***Cell proliferation determinations.***
Representative samples of the left lateral lobe of the liver, left and right testes and pancreas were collected from each animal. After fixation (formalin or zinc formalin), samples were embedded in paraffin,and sections stained with hemotoxylin and eosin were prepared. Previously described methods were used to stain tissues for PCNA (Eldridge *et al.*, 1993).
***Bile acid determination.*** Bile (up to 5 ml) was collected at sacrifice of each animal, flash frozen in liquid nitrogen and analyzed for specific bile acids (insert Cliff's methods)

***Statistics.*** Levene's test (Levene, 1960) was used for variance homogeneity. In the case of heterogeneity of variance at $p \leq 0.05$, transformations were used to stabilize the variance.

One-way analysis of variance (ANOVA, Winer 1971a) was used to analyze initial body weights, body weight changes, continuous clinical pathology values, and organ weight data. ANOVA was done on the homogeneous or transformed data. If the ANOVA was significant, Dunnett's t-test (Dunnett, 1964) was used for control versus treated group comparisons.

One-way analysis of variance (ANCOVA, Winer, 1971b) was used to analyze body weights, with initial body weights as the covariate. Although Levene's test for variance homogeneity was done, no transformations were used because covariance adjustment removed extraneous heterogeneity. If the ANCOVA was significant, covariate-adjusted means were used for control versus treated group comparisons.

Dosed groups were compared to the controls at the 5% two-tailed probability level. Blood hormone levels were compared by ANCOVA with significance at the p=0.05 level.

For CCK, all data were analyzed using Jonckheere's test for trend ($p < 0.05$).

## RESULTS

***Clinical observations.*** During the first week of dosing, all animals in the 30 mg/kg dose group had qualitatively low food consumption and and lost from 3.1 to 7.5 percent of

body weight. Four of the six animals also had few or no feces. Dosing was suspended on Day 12 and recommenced at 20 mg/kg/day on day 22.

Only two animals were continuously treated at the 20 mg/kg/day dose for the remainder of the dosing period. Dosing of the other three high-dose animals was discontinued on Days 43, 66 and 81. These animals exhibited low or no food consumption, few or no feces, and dramatic body-weight losses prior to being suspended from dosing. Body-weight losses were 17.5, 23.1 and 18.7 percent prior to suspension of dosing on days 43, 66 and 81, respectively. The remaining high-dose animal was sacrified in moribund condition on day 29. This animal had lost 12.5 percent of body weight, was hypoactive, cold to the touch, exhibited low or no food consumption, and few or no feces.

There was a lack of opthalmic effects in all animals.

Overall, body-weight losses in the 30/20 mg/kg dose group were significantly lower than controls (14.3 percent over the 27 week period). Dosing at 30 mg/kg during the first two weeks caused considerable weight loss. After commencing the 20 mg/kg/day dosing on day 22, weight loss was significant compared to controls during weeks 7, 9 and 24 (Table 1).

Clinical signs and body-weights were normal in monkeys receiving 10 mg/kg and all but one monkey receiving 3 mg/kgc. However, one low-dose animal was sacrificed in mroibund condition on day 137. In this one animal, paralysis of the hind limbs and ataxia occurred as well as few feces and low or no food consumption and loss of 9.5 percent of body weight in the week prior to sacrifice.

***Blood homone determinations.*** No effects on estrone, estradiol, estriol or testosterone were noted. Mean group estradiol values in all groups including the controls tended to be considerably lower dutring the treatment period than the corresponding pretreament values. In the high-dose animals these values appeared to be lower than the other groups. The estradiol values in the recovery animals tended to be similar to the predose values. No significant changes in testosterone values were seen during the treatment period. In the last recovery sampling, mean values (9 and 15 ng/ml) tended to be higher than means during the rest of the study (1-8 ng/ml).

Taken collectively, values for the thyroid hormones in APFO-treated monkeys appeared to be unaffected. With total and free T3, mean values from test day –4 through to the end of the study remained relatively constant; although, monkeys in the high-dose group exhibited generally lower group mean values. It should be pointed out that values on pretest days –8 and –18 were considerably higher than those during the rest of the study. Group mean total T3 values are shown in Figure 1, page 56). During the treatment period, the three high-dose animals that were eventually taken off compound for toxicity tended to have decreasing T3 values prior to removal from treatment. These values tended to increase toward pre-study values after cessation of treatment. Figures 2 (page

57) and 3 (page 60) represent the individual trends in Total T3 for the control and high-dose groups, respectively.

No statistically significant or biologically significant alterations in plasma CCK concentrations were observed.

***Clinical pathology.*** Administration of APFO at 3 or 10 mg/kg produced no apparent effects on measured clinical parameters including hematology, coagulation, clinical chemistry, and urinanalysis. The 3 mg/kg dose group animal that was sacrifced in moribund condition on day 137 showed marked hyperfibrogenemia, moderate lymphopenia, moderate hypoalbuminemia and mild hypocholesterolemia.

Findings in the 30/20 mg/kg dose group were complicated by the unscheduled sacrifice of one animal on day 29 and the cessation of treatment for three others by day 80. Only two animals remained on treatment during clinical pathology tesing intervals on days 91 and 182. In this group, mild increases in triglycerides and mild to marked decreases in absolute neutrophile count, total protein concentration, and albumin concentration occurred. The differences in triglycerides were statistically significant on days 31 and 91. The other differences were not statistically significant but were consistent over time. Absolute neutrophile count and albumin concentration were mildly decreased prior to cessation of treatment for the three animals for which treatment was stopped early. One monkey at this dose had moderate increases in serum activities of aspartate aminotransferase, alanine aminotransferase and creatine kinase when dosing was stopped on day 66 and moderate increases in serum bile acids on day 63. The high-dose dose group animal which was sacrificed in moribund condition on day 29 had numerous non-specific clinical pathology findings commonly observed in animals that are very ill along with marked serum activities of aspartate aminotransferase , alanine aminotransferase, sorbitol dehydrogenase and creatine kinase as well as unusually low cholesterol (14 mg/dL).

Eleven days prior to initiation of treatment (day –11), the animal in the low-dose group which was sacrificed in moribund condition had a high hematocrit (49.7%) and albumin concentration (5.6 g/dL), possibly indicative of mild dehydration, and it had the lowest neutrophil count (1,600/μL) of all animals.

***PFOA levels in Serum and Liver.***
Steady-state values for PFOA in the serum were attained within 4 to 6 weeks of dosing. In the 3 mg/kg group, this value was approximately 110ppm and in the 10 mg/kg group it was 100ppm. Although difficult to determine because of discontinuous treatment, peak values were highly variable and averages of around 150 to 1000ppm were seen in animals receiving 30/20 mg/kg. It is apparent that the relationship between APFO administered and APFO in the serum is not linear (Table X)

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Amounts of APFO in monkey liver averaged 14 to 16 ppm in the 2 lower dose groups (Table Y).In the higher group the values for the 2 monkeys which continued through the entire treatment period were 17 and 87 ppm. In animals from this group that were removed from dosing, liver concentration of from 0.2 to 1.5ppm were obtained. In the 2 mid-dose recovery monkeys, APFO concentrations in the liver went from a mean of 14 ppm to 0.1 and 0.3 ppm respectively.

Preliminary data from urine analysis for PFOA suggests that most PFOA is excreted in the urine. This will be presented in a future manuscript.

***Palmitoyl CoA determinations.*** There were increases over control in palmitoyl CoA oxidase activity.

***Cell proliferation results.*** There were no significant differences in cell proliferation observed in liver, pancreas or testes as measured by PCNA.

***Anatomic Pathology.*** Increases occurred in mean absolute liver weights and mean liver-to-body weight percentages in all dose groups at terminal sacrifice after 26 weeks of dosing (Table Z). Liver weight increases showed a positive dose-response trend. The low-dose and mid-dose animals group mean absolute and relative liver weights were similar and significantly elevated over control values. The two high-dose animals that were treated until the end of the dosing period had absolute and relative liver weights which were significantly elevated over the other treatment groups and control.

No APFO-related macroscopic or microscopic changes were observed in any organs at the terminal sacrifice. This includes key organs such as the liver, adrenal, spleen, pancreas and testis. At the recovery sacrifice of the 10 mg/kg animals, there were no effects on terminal body weight, there were no macroscopic or microscopic findings, and liver weights were normal.

Table Z: Absolute and relative liver and body weights at scheduled dosing termination.

| Dose group, mg/kg (n) | Body weight (g) | Liver weight (g) | % Control | L wgt/B wgt (%) | % Control |
|---|---|---|---|---|---|
| 0 Control (4) | 3947 ± 591 | 60.16 ± 6.93 | 100 | 1.53 ± 0.07 | 100 |
| 3 (3) | 4486 ± 30 | 81.79 ± 2.81 | 135 | 1.82 ± 0.05 | 119 |
| 10 (4) | 4447 ± 498 | 83.17 ± 9.66 | 138 | 1.87 ± 0.06 | 122 |
| 30/20 (2) | 3925 ± 583 | 90.39 ± 4.22 | 150 | 2.40 ± 0.51 | 157 |

Two animals were found in moribund condition and sacrificed at unscheduled times during the dosing period. A 30/20 mg/kg animal was sacrificed on day 29 and found to have edema and inflammation of the esophagus and stomach indicative of dosing injury. This animal also had liver lesions, including mid-zonal and centrilobular hepatocellular

degeneration and necrosis, diffuse hepatocellular vacuolation, and hepatocyte basophilia in centrilobular areas indicative of liver regeneration. Involution of the thymus, a common stress response, and degeneration and necrosis of the heart, probably agonal changes, were also observed in this animal. Although the esophageal and gastric lesions are complicating factors, the contribution of APFO to this animals moribund condition cannot be dismissed due to findings in the liver, which were likely due to APFO exposure.

A 3 mg/kg animal was found in moribund condition with hind-limb paralysis and ataxia and sacrificed on day 137. The blood supply to this animals hind limbs was severely compromised, as indicated by the cool temperature of the hind limbs on final medical examination and the fact that ketamine injected into the thigh muscle failed to reach the systemic circulation. The microscopic and macroscopic findings did not reveal evidence of spinal cord injury or impaired blood circulation.

## DISCUSSION

- Quality of the study (GK)
- Dose-level selection (PL)
- High-dose unscheduled sacrifice (JB)
- High-dose cessation of dosing (JB)
- Liver findings (JB)
- Sex hormones (GK)
- Thyroid hormones (JB)
- Recovery story (PL)
- Mechanistic endpoints (PcoAO, PCNA, CCK, bile acids) (GK)
- Pharmacokinetics (PL)
- LOEL / NOEL (GK)
- Low-dose death (GK)
- Relate to rhesus study (JB)
- Relate to rodent story (PL)
- Relate to human story (JB)

## CONCLUSIONS

GK002557

EID101464

Resource material for discussion follows:

These findings were non-specific and, with the exception of low albumin concentration, were not consistent with the findings observed for animals in the high-dose group that were withdrawn from treatment. Therefore, the clinical pathology results for this low-dose animal were not assumed to be due to APFO exposure.

There are no known metabolic processes that degrade PFOA. The urinary elimination of PFOA in rats appears to be hormonally regulated (refs). Female rats and castrated male rats rapidly clear PFOA in the urine; whereas, male rats have a significantly longer urinary clearance half-life (refs). Sex differences in urinary clearance have not been found in [species] (ref). PFOA is excreted in the bile and enters enterohepatic circulation (Johnson, J.D. et al. 1980; Johnson, J.D., et al., 1984). Follow-up on blood levels in retired workers indicated that, although the concentrations detected were in the few ppm range, the chemical was present in some individuals well after the possibility of exposure had ceased, suggesting an elimination half-life on the order of 18 months (Ubel, 1980). A recent study of 3M retired chemical plant workers suggests that the mean serum elimination half life is approximately 300 days (Burris et al, 2000).

**Liver tumors**

The finding of liver enlargement in male cynomolgous monkeys at all doses in this study is significant in ligt of prior information available from a 90-day oral gavage study in male and female rhesus monkeys. The rhesus study was conducted with APFO (FC-143, Lot 340) in male and female rhesus monkeys at doses of 0 (control), 3, 10, 30 and 100 mg/kg/day in 0.5% Methocel.. Unfortunately, all animals in the 100 mg/kg/d group and 3 of 4 animals in the 30 mg/kg/d group died. No treatment related gross pathological lesions or alterations in urinalysis values were seen. Significant effects (and LOELs in mg/kg/day) included: weight loss (30); anorexia (10); emesis (30); black stools (10); pale, swollen face (10), swollen eyes(30); decreased activity (30); prostration and trembling (100); anemia (30), slightly increased prothrombin and activated partial thromboplastin times (30), decreased serum alkaline phosphatase activity (30); increased serum aspartate aminotransferase (SGOT) activity (30); increased serum creatinine phosphokinase (CPK) activity (30); diffuse lipid depletion of the adrenals (30), bone marrow hypocellularity (30), splenic and lymph node follicular atrophy (30). The NOAEL for this study was 3 mg/kg/d (ref). Subsequent analysis of serum and liver samples showed dose-related FC-143 concentrations. There was no apparent difference in serum or liver FC-143 levels between males and females (ref).

Testosterone inhibits urinary elimination of PFOA in male rats. It has been suggested that the gender difference in elimination may be due to a testosterone inducible serum or tissue protein which binds PFOA [48].

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No changes in liver weight to body weight ratios were seen in female rats given single oral doses of up to 200 mg PFOA/kg. In male rats, single oral doses of either 100 or 200 mg PFOA/kg produced an increase in relative liver weight. Castration reduced the magnitude of the liver weight increase [13].

Estradiol treatment of castrated or intact male rats produced PFOA excretion rates similar to those in females. Castration without estradiol treatment also enhanced PFOA excretion but not as effectively as estradiol treatment [51].

PFOA induced hepatomegaly, peroxisomal β-oxidation, microsomal 1-acylglycerophosphocoline acyltransferase and cytosolic long-chain acyl CoA hydrolase in male rats to a much greater extent than in female rats. Testosterone was shown to be necessary for these effects and estradiol blocked these effects in male rats [53].

Activities of liver microsomal 1-acylglycerophosphocholine acyltransferase (AGA), microsomal stearoyl-CoA desaturase (SD) and peroxisomal β-oxidation and the acyl composition of liver microsomal phosphatidylcholine (PC) were measured in rats fed a diet containing 0.01 % PFOA for 2, 22 or 26 weeks. Females showed only slight changes in these parameters. In males, activities for all three enzymes were elevated by two weeks. Activities of AGA and β-oxidation remained unchanged throughout 26 weeks. The activity of SD declined but was still higher than controls at 26 weeks. The acyl composition of microsomal PC was significantly altered in males. All of these parameters returned to control levels within 4 weeks after PFOA was withdrawn from the diet [72].

PFOA in the diet of mice at a concentration of 0.02-0.05% for 5-10 days caused increased hepatic peroxisomal fatty acid β-oxidation, decreased hepatic mitochondrial size and increases in the following hepatic enzyme activities: ω-hydroxylation of lauric acid and cytosolic epoxide hydrolase, glutathione transferase and DT-diaphorase. There were no significant differences between males and females [85].

PFOA has been shown to induce hepatic peroxisomal proliferation in rats and mice There was a marked sex difference in rats [38, 53, 54, 58, 83]. PFOA-induced hepatic peroxisomal proliferation in male rats is most prevalent in the centrilobular region and is associated with increased peroxisomal palmitoyl-CoA oxidation, marked hypolipidemia, an almost 10-fold increase in hepatic free acid soluble CoA, and altered peroxisomal polypeptides [83].

Perfluorooctane, $C_8F_{18}$, did not induce peroxisomal proliferation in rats, indicating the importance of the carboxylic acid group in causing this effect [54].

PFOA caused peroxisomal proliferation in primary cultures of rat hepatocytes. PFOA-mediated increases in the peroxisomal enzyme fatty acyl-CoA β-oxidase (FACO) were inhibited by cycloheximide and correlated with elevations in FACO mRNA levels. Thus,

PFOA appears to induce peroxisomal proliferation through the regulation of gene transcriptional and translational processes [69].

PFOA-induced hepatomegaly is due predominantly to cell hypertrophy resulting from proliferation of smooth endoplasmic reticulum, mitochondria and peroxisomes [38]. PFOA-induced DNA synthesis (hyperplasia) has also been shown to occur in rat liver, primarily in the periportal region. Increased DNA synthesis occurred in the absence of necrosis, suggesting that the cell proliferative activity is not a regenerative response to hepatocellular injury. Peroxisome proliferation occurred primarily in the centrilobular region suggesting that PFOA-induced hyperplasia and peroxisomal proliferation are independent processes[73].

PFOA in the diet of male mice at concentrations of 0.02 to 0.1 % for 2-10 days caused decreased body weight and the following hepatic effects: hepatomegaly, decreased mitochondrial size, increased microsomal P450, P450 reductase and epoxide hydrolase, increased cytosolic epoxide hydrolase and DT-diaphorase, and decreased mitochondrial lipid peroxidation. Hepatic microsomal,$b_5$ and cytosolic glutathione transferase, glutathione peroxidase and superoxide dismutase were not significantly affected [84].

Hepatomegaly, but not peroxisome proliferation, caused by PFOA in rats was shown to be dependent on corticosterone, the primary adrenal glucocorticoid in rodents [73].

Unlike many hepatocarcinogenic peroxisome proliferators, PFOA did not cause hypolipidemia. Rather, PFOA caused a marked increase in hepatic lipid synthesis [38].

Dietary treatment of male rats with PFOA (0.02% for 7-14 days) caused decreased serum cholesterol and triacylglycerols and increased liver free cholesterol. Hepatocytes isolated from treated rats showed reduced synthesis of cholesterol from acetate, pyruvate and hydroxymethylglutarate but not from mevalonate, increased oxidation of palmitate and reduced fatty acid synthesis. Activities of liver hydroxymethyl glutaric acid-CoA reductase and acyl-CoA:cholesterol acyltransferase were reduced [82].

PFOA in the diet of mice for up to 10 days caused greater increases in liver weight, hepatic mitochondrial protein, hepatic peroxisomal palmitoyl-CoA oxidation, lauroyl CoA oxidase activity and catalase activity than did perfluoroacetic, -butyric or decanoic acids. PFOA increased peroxisomal fatty acid β-oxidation (which produces $H_20_2$) to a much greater extent than catalase (which reduces $H_20_2$ to water and molecular oxygen). Dietary levels of PFOA as low as 0.001 % caused significant changes[61].

PFOA caused enhanced affinity and reduced maximal binding capacity of hepatic inositol triphosphate (ITP) receptors in male rats. ITP is a second messenger involved in the mobilization of calcium from intracellular stores and may be involved in the process of peroxisomal proliferation [74].

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Valproate (2-propylpentanoic acid), a non-fluorinated analog of PFOA, is a hepatotoxin which impairs mitochondrial function and fatty acid metabolism [97].

Induction of peroxisomes by treatment with PFOA did not increase rates of hydrogen peroxide production in rat or mouse liver [12]. Oxidative stress resulting from the increased production of hydrogen peroxide has been postulated as a mechanism by which peroxisome proliferators cause liver damage and cancer.

PFOA induced hepatic microsomal carboxylesterase isozymes in male rats [106].

PFOA displaced a fluorescently labeled fatty acid from liver fatty acid binding protein (L-FABP) and bovine serum albumin *in-vitro* [109]. To test the hypothesis that fluorocompounds act as peroxisome proliferators by competitive binding to the fatty acid binding site of L-FABP, displacement of the fluorescently labeled fatty acid analogue 11-(5-dimethylaminonapthalenesulfphonyl)-undecanoic acid (DAUDA) from L-FABP was measured *in vitro* as a percent loss of initial fluorescence. 10 µM PFOA caused a 42% reduction of initial fluorescence when added to solutions containing 1 µM L-FABP and 1µM DAUDA. The LOEL for PFOA in this assay was 0.5 - 1.0 µM. By comparison, 10 µM of the known or suspected peroxisome proliferators: perfluorooctane sulfonic acid (PFOS), Wyeth, N-Ethyl perfluorooctanesulfonamide (FX-12) and N-Ethylperfluorooctane sulfonamide ethanol (N-EtFOSE) caused 66%, 48%, 45% and 39% reduction of initial fluorescence, respectively. Results for BSA were very similar to those seen with L-FABP [109, 122].

The serum concentration at which this liver enlargement occurred (approx. 50 ppm) is within the range experienced by workers with higher exposures. Studies in exposed workers have not indicated liver toxicity. However, the enlargement at the low dose in the cynomolgus monkey was not associated with any clinical manisfestations of liver toxicity; therefore, it is not possible to know if workers with similar body burdens, as represented by serum concentration, experience similar liver enlargement.

**Pancreatic tumors**

The absence of an increase in cholecystokinin is also also significant in light of Cook et al.'s findings in rats (ref). This may indicate a decreased risk of pancreatic tumors in primates. It is also important to point out that $CCK_A$ receptors are distributed differently in the rodent as oppsed to humans and primates (ref). [comment on $CCK_A$ receptor agonism] PFOA has been asssociated with elevated CCK levels and pancreatic tumors. PFOA did not bind directly to the $CCK_A$ receptor in a competition binding assay, and did not inhibit trypsin *in vitro*, thus two possible contributing mechanisms for pancreatic carcinogeneis were ruled out [111].

**Leydig cell tumors**

In the male rat, estradiol is elevated and testoterone decreased on repeated exposure. The absence of this effect in the present study and in studies of workers suggests that the primate is not responsive to the hormonal changes seen in the rat. Since these changes are thought to occur in the rat via induction of aromatase and are related to the formation of Leydig cell tumors in the rat, the risk of testicular tumors in primates from exposure to APFO is assumed to be much lower than that in the rat. In fact, the spontaneous incidence of Leydig cell tumors in rats is orders of magnitude higher than in the humans (insert detail and refs).

The historical incidence of Leydig cell tumors in Sprague-Dawley rats ranges from 1.4 to 10% with a mean of 4.7% [60]. The incidence of Leydig cell tumors in man is estimated to be less than 0.0003%. A number of chemical agents, nutritional and hormonal factors have been shown to cause Leydig cell tumors in rodents. However, where there have been studies on the same chemicals in man, there appears to be no evidence of Leydig cell tumor formation in man [50].

Elevated estrogen levels have been associated with Leydig cell tumors in mice, rats and humans [91,95,96]. Serum estradiol levels were significantly elevated in rats treated with PFOA at doses of more than 10 mg/kg/d and accessory sex organ relative weights were significantly reduced in rats fed 50 mg/kg/d PFOA for 14 days. In addition, serum testosterone levels showed a dose-dependent decrease which was, however, not statistically significant. This decrease in testosterone levels was shown, by human chorionic gonadotropin (cHG) challenge, to be associated with inhibition of steroidogenesis in Leydig cells [10]

The increase in serum estrogen caused by PFOA appears to be due to induction of the enzyme aromatase (Cytochrome P450 XIX) which metabolizes testosterone to estradiol. A two-fold increase in serum estradiol showed a linear correlation with a 16-fold increase in total hepatic aromatase activity [57, 118].

PFOA caused a dose-dependent decrease in hCG stimulated testosterone secretion and also reduced the basal release of estradiol by rat Leydig cells exposed *in vitro,* suggesting that PFOA may have a direct effect on the ability of Leydig cells to produce testosterone and estradiol [55,119]. In contrast, *ex-vivo* studies demonstrate an increase in testosterone production in hCG-stimulated Leydig cells from rats treated with 25 mg/kg/day APFO for 14 days, indicating that the direct inhibition of testosterone synthesis seen *in-vitro* is reversible *in-vivo*. Furthermore, in the *in vivo* studies serum estradiol and testicular interstitial fluid estradiol and transforming growth factor alpha (TGF alpha) levels were also increased, consistent with the hypothesis that estradiol may modulate growth factor expression within the testis. APFO produced a 4.5 fold increase in hepatic aromatase *in-vivo*, suggested as the primary cause of increased serum estradiol [120].

PFOA at 300 ppm in the diet for 1 year in male rats caused increased serum estradiol levels, increased hepatic β-oxidation (peroxisome proliferation) and increased relative

liver weights. PFOA had no effect on hepatic or Leydig cell proliferation, Leydig cell β-oxidation, or serum testosterone, FSH, prolactin or luteinizing hormone concentrations. Thus, PFOA did not induce peroxisome proliferation in Leydig cells at dietary levels which did induce hepatic peroxisome proliferation [56].

The lack of peroxisome proliferation seen in this study in not unexpected. Primates are less responsive to peroxisome proliferation from xenobiotic compounds than rodents, partially due to the fact that primates have much fewer PPARα receptors than rodents. Since the hepatocelluar tumors observed in rats with APFO are thought to derive from peroxisome proliferation and the pleiomorphic response from activation of PPARα, the risk of these tumors occuring in primates is considered low.

Waxman et al (1999) have studied the effects of PFOA on transactivation of PPARα and PPARγ in COS-1 cells transfected with mouse and human PPARα and PPARγ receptors. Human PPARα was less sensitive to PFOA than mouse PPARα. Human and mouse PPARγ were non-responsive to PFOA.

**Thyroid hormones**

All T4 values were within the range published for Rhesus monkeys of 0.8 – 6.6 ug/mL for males, and 1.3 .-7.6 ug/mL for females (DePaolo and Masaro. 1989). Taken together, the data suggest that the changes in T4 values observed appear to be systemic trends that are not compound related and not biologically significant.

All T3 values were within the reference range for male and female Macaques (54 –115 ng/100ml and 65- 295 ng/100 ml for males and females, respectively (DePaolo and Masaro 1989).

**Cell proliferation discussion**

**Membrane Effects:** At sub-lethal concentrations, PFOA did not alter the expression or secretion of IgM from cultured human or mouse B cells. At lethal concentrations (> 0.8 mM), PFOA caused a detergent-like solubilization of membrane proteins. The authors have suggested that some of the biochemical changes and target organ effects observed *in vivo* with PFOA may be secondary to cell lysis [92].

PFOA caused lipid peroxidation *in vitro* [80].

PFOA and other perfluorinated surfactants cause alterations in the fatty acid composition, organization and fluidity of cell membranes [62,92,93,94].

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At sub-lethal concentrations, PFOA did not affect migration or capping of cell surface immunoglobulins after antigen recognition in lymphoblastoid cells *in vitro*. However, PFOA (0.9 mM for 15 min) did cause changes in membrane lipid architecture. It was suggested that direct, physical disruption of cell surface membranes is the likely mechanism of acute PFOA toxicity [93].

Sodium perfluorooctanoate had a much higher affinity for dipalmatoylphosphatidylcholine vesicle membranes than did the corresponding non-fluorinated compound, sodium octanoate. This difference was attributed to the greater hydrophobicity of the fluorinated acid [compound [94].

PFOA did not inhibit cell-to-cell communication in cultures of freshly isolated or established (ARL) rat liver cells as did PFDA [76].

PFOA uncoupled ($ED_{50}$ = 0.18 mM) and at higher concentrations inhibited rat mitochondrial oxidative phosphorylation *in vitro*. The authors hypothesized that uncoupling of mitochondrial oxidative phosphorylation by PFOA *in vivo* would lead to reduced cellular ATP levels and elevated intracellular unsaturated fatty acids which would activate protein kinase c leading to increased cell turnover which is believed to be involved in the carcinogenic process for nongenotoxic carcinogens [67].

PFOA (100 μM) exhibited a general detergent-like effect on mitochondrial membranes as reelected by a decrease in respiratory control without uncoupling oxidative phosphorylation *in-vitro*. FC-143 slightly increased the activity of enzymes of oxidative phosphorylation and respiratory chain, the most probable mechanism being the fluidisation of the inner mitochondrial membrane [121].

PFOA at concentrations up to 500 μg/ml did not impair clone-forming ability in L5178Y mouse lymphoma cells *in vitro* [77].

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## CONCLUSIONS

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## REFERENCES


1) Abdellatif, A.G., V. Préat, H.S. Taper, and M. Roberfroid. The modulation of rat liver carcinogenesis by perfluorooctanoic acid, a peroxisome proliferator. *Toxicology & Applied Pharmacology* 111 (1991) 530-537 [*Submitted May 25, 2000*].

2) Abdellatif, A.G., V. Préat, J. Vamecq, R. Nilsson, and M. Roberfroid. Peroxisome proliferation and modulation of rat liver carcinogenesis by 2,3-dichlorophenoxyacetic acid, 2,4,5-trichlorophenoxyacetic acid, perfluorooctanoic acid and nafenopin. *Carcinogenesis* 11 (1990) 1899-1902 [*Submitted May 25, 2000*].

3) Belisle, J., and D. Hagen. A method for determination of perfluorooctanoic acid in blood and other biological samples. *Analytical Biochemistry* 101 (1980) 369-376 [*Submitted May 25, 2000*].

4) Biegel, L.B., M.E. Hurtt, and J.C. Cook. Effects of ammonium perfluorooctanoate (C8) on Leydig cell function: In vivo, ex vivo, and in vitro studies. *The Toxicologist* 13[1] (1993) 399 [*Submitted May 25, 2000*].

5) Biegel, L.B., R.C.M. Liu, M.E. Hurtt, and J.C. Cook. Effects of ammonium perfluorooctanoate on Leydig cell function: In vivo, ex vivo, and in vitro studies. *Toxicology & Applied Pharmacology* 134 (1995) 18-25 [*Submitted May 25, 2000*].

6) Borjes, H.K. and R.G. Thurman. Peroxisomal proliferators inhibit Acyl CoA synthesis and stimulate protein kinase C in vivo. *Toxicology & Applied Pharmacology* 126 (1994) 233-239 [*Submitted May 25, 2000*].

7) Clapperton, R.M., B.T. Ingram, R.H. Ottewill, and A.R. Rennie. NMR spectroscopic and neutron scattering studies on ammonium decanoate-ammonium perfluorooctanoate mixtures. In: *Mixed Surfactant Systems. ACS Symposium Series 501,* Eds.: P.M. Holland and D.N. Rubingh, American Chemical Society: Washington, DC, 1992, 268-277 [*Submitted May 25, 2000*].

8) Clegg, E.D., J.C. Cook, R.E. Chapin, P. Foster, and G.P. Daston. Leydig cell hyperplasia and adenoma formation: Mechanisms and relevance to humans (abstract). *Reproductive Toxicology* 11(1) (1997) 107-121 [*Submitted May 25, 2000*].


EID101473

9) Cook, J.C., S.M. Murray, S.R- Frame, and M.E. Hurtt. Induction of Leydig cell adenomas by ammonium perfluorooctanoate: A possible endocrine related mechanism. *Toxicology & Applied Pharmacology* 113 (1991) 209-217 [*Submitted May 25, 2000*].

10) Cook, J.C., M.E. Hurtt, S.R. Frame, L.B. Biegel. Effects of Wyeth-14,643 (WY) and ammonium perfluorooctanoate (C8) in Crl:CD BR (CD) rats. *The Toxicologist* 13[1] (1993) 399 [*Submitted May 25, 2000*].

11) Cook, J.C., M.E. Hurtt, S.R. Frame, L.B. Biegel. Mechanisms of extrahepatic tumor induction by peroxisome proliferators in Crl:CD BR (CD) rats. *The Toxicologist* 14[1] (1994) 301 [*Submitted May 25, 2000*].

12) Feller, D.R. and U. Intrasuksri. Mechanism of peroxisome proliferation by perfluorinated fatty acids in primary cultures of rat hepatocytes (abstract). *Toxicologist* 13 (1993) 395 [*Submitted May 25, 2000*].

13) Gilliland, F.D. Fluorocarbons and human health: Studies in an occupational cohort. Unpublished Ph.D. Dissertation, University of Minnesota, 1992 [*Submitted May 25, 2000*].

14) Gilliland, F.D. and J.S. Mandel. Mortality among employees of a perfluorooctanoic acid production plant. JOM 35[9] (September 1993) 950-954 [*Submitted May 25, 2000*].

15) Gilliland, F.D. and J.S. Mandel. Serum perfluorooctanoic acid and hepatic enzymes, lipoproteins, and cholesterol: a study of occupationally exposed men. *American Journal of Industrial Medicine* 29 (1996) 560-568 [*Submitted May 25, 2000*].

16) Griffith, F.D., and J.E. Long. Animal toxicity studies with ammonium perfluorooctanoate. *American Industrial Hygiene Association Journal* 41 (1980) 576-583 [*Submitted May 25, 2000*].

17) Handler, J.A., C.B. Seed, B.U. Bradford, and R.G. Thurmond. Induction of peroxisomes by treatment with perfluorooctanoate does not increase rates of $H_20_2$ production in intact liver. *Toxicology Letters* 60 (1990) 61-68 [*Submitted May 25, 2000*].

18) Hanhijärvi, H., M. Ylinen, A. Kojo, and V.M. Kosma. Elimination and toxicity of perfluorooctanoic acid during subchronic administration in the Wistar rat. *Pharmacology & Toxicology* 61 (1987) 66-68 [*Submitted May 25, 2000*].

EID101474

GK002567

19) Hanhijärvi, H., R.H. Ophaug, and L. Singer. The sex-related difference in perfluorooctanoate excretion in the rat. *Proceedings of the Society for Experimental Biology & Medicine* 171 (1982) 50-55 [*Submitted May 25, 2000*].

20) Haughom, B., and O. Spydevold. The Mechanism Underlying the Hypolipemic Effect of Perfluorooctanoic Acid (PFOA), Perfluorooctane Sulphonic Acid (PFOSA) and Clofibric Acid. *Biochim. Biophys. Acta.* 1128[l] (1992) 65-72 [*Submitted May 25, 2000*].

21) Hosokawa, M., and T. Satoh. Differences in the induction of carboxylesterase isozymes in rat liver microsomes by perfluorinated fatty acids. *Xenobiorica* 23 (1993) 1125-1133 [*Submitted May 25, 2000*].

22) Hurtt, M.E., S.M. Murray, S.R. Frame, and J.C. Cook. Investigation of a hormonally-mediated mechanism for ammonium perfluorooctanoate (C8)-induced Leydig cell adenomas. *Toxicologist* 10 (1990) 192 [*Submitted May 25, 2000*].

23) Ikeda, T., K. Aiba, K. Fukuda, and M. Tanaka. The induction of peroxisome proliferation in rat liver by perfluorinated fatty acids, metabolically inert derivatives of fatty acids. J. *Biochem.* 98 (1985) 475-482 [*Submitted May 25, 2000*].

24) Inoue, T., T. Iwanaga, K. Fukushima, and R. Shimozawa. Effects of sodium octanoate and sodium perfluorooctanoate on the gel-to-liquid-crystalline phase transition of dipalmitoylphosphatidylcholine vesile membrane. *Chem. Physics Lipids* 46 (1988) 25-30 [*Submitted May 25, 2000*].

25) Johnson, J.D., S.J. Gibson, and R.E. Ober. Cholestyramine-enhanced fecal elimination of Carbon-14 in rats after administration of ammonium perfluorooctanoate or potassium perfluorooctanesulfonate. *Fundamental & Applied Toxicology* 4 (1984) 972-976 [*Submitted May 25, 2000 & April 21, 2000*]

26) Just, W.W., K. Gorgas, F. Hartl, P. Heinemann, M. Salzer, and H. Schimassek. Biochemical effects and zonal heterogeneity of peroxisome proliferation induced by perfluorocarboxylic acids in rat liver. *Hepatology* 9 (1989) 570-581 [*Submitted May 25, 2000*].

27) Kawashima, Y., T. Matsunaga, N. Uy-Yu, and H. Kozuka. Induction by perfluorooctanoic acid of microsomal I-acylglycerophosphocholine acyltransferase in rat kidney. Sex related difference. *Biochemical Pharmacology* 42 (1991) 1921-1926 [*Submitted May 25, 2000*].

EID101475

28) Kawashima, Y., S. Suzuki, H. Kozuka, J. Sato, and Y. Suzuki. Effects of prolonged administration of perfluorooctanoic acid on hepatic activities of enzymes which detoxify peroxide and xenobiotic in the rat. *Toxicology* 93 (1994) 85-87 [*Submitted May 25, 2000*].

29) Kawashima, Y., N. Uy-Yu, and H. Kozuka. Sex-related difference in the inductions by perfluorooctanoic acid of peroxisomal β-oxidation, microsomal 1-acyglycerophocholin acyltransferase and cytosolic long-chain acyl-CoA hydrolase in rat liver. *Biochem J.* 261 (1989) 595-600 [*Submitted May 25, 2000*].

30) Keller, B.J., D.S. Marsman, J.A. Popp, and R.G. Thurman. Several nongenotoxic carcinogens uncouple mitochondrial oxidative phosphorylation. *Biochimica et Biophysica Acta.* 1102 (1992) 237-244 [*Submitted May 25, 2000*].

31) Kennedy, G.L. Dermal toxicity of ammonium perfluorooctanoate. *Toxicology & Applied Pharmacology* 81 (1985) 348-355 [*Submitted May 25, 2000*].

32) Kennedy, G.L. Increase in Mouse Liver Weight Following Feeding of Ammonium Perfluorooctanoate and Related Fluorochemicals. *Toxicology Letters* 39 (1987) 295-300 [*Submitted May 25, 2000*].

33) Kennedy, G.L., G.T. Hall, M.R. Brettelli, J.R. Barnes, and H.C. Chen. Inhalation toxicity of ammonium perfluorooctanoate. *Fd. Chem. Toxic.* 24 (1986) 1325-1329 [*Submitted May 25, 2000*].

34) Kinney, L.A., N.C. Chromey, G.L. Kennedy. Acute inhalation toxicity of ammonium perflurooctanoate (abstract). *Food Chem. Toxicol.* 21 (1989) 465-468 [*Submitted May 25, 2000*].

35) Kojo, A., H. Hanhijärvi, M. Ylinen, and V.-M. Kosma. Toxicity and kinetics of perfluoro-octanoic acid in the Wistar rat. *Archives of Toxicology (Toxic Interfaces of Neurones)* Supplement 9 (1986) 465-468 [*Submitted May 25, 2000*].

36) Kun, E. Fluorocarboxylic acids as enzymatic and metabolic probes. In: *Biochemistry Involving Carbon-Fluorine Bonds.* Ed: R. Filler. ACS Symposium Series Number 28, American Chemical Society, Washington 1976, 1-22. (QDl Am3s #28) [*Submitted May 25, 2000*].

37) Kuslikis, B.I., J.P. Vanden Heuvel, and R.E. Peterson. Lack of evidence for perfluorodecanoyl-or perfluorooctanoyl-coenzyme A formation in male and female rats. J. *Biochem. Toxicology* 7 (1992) 25-36 [*Submitted May 25, 2000*].

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EID101476

38) Levitt, D., and A. Liss. Perfluorinated fatty acids alter merocyanine 540 dye binding to plasma membranes. *Journal of Toxicology & Environmental Health* 20 (1987) 303-316 [*Submitted May 25, 2000*].

39) Levitt, D., and A. Liss. Toxicity of perfluorinated fatty acids for human and murine B cell lines. *Toxicology & Applied Pharmacology* 86 (1986) 1-11 [*Submitted May 25, 2000*].

40) Liu, M.S., and D.M. Long. Biological disposition of perfluoroctylbromide: Tracheal administration in alveolography and bronchography. *Investigative Radiology* 11 (1976) 479-485 [*Submitted May 25, 2000*].

41) Liu, R.C.; M.E. Hurtt, J.C. Cook, L.B. Biegel. Effect of ammonium perfluorooctanoate (C8) on aromatase activity in tissues of male Crl:CD BR (CD) rats. *Toxicologist* 14[1] (1994) 300 [*Submitted May 25, 2000*].

42) Liu, R.C.M., C. Hahn, and M.E. Hurtt. The direct effect of hepatic peroxisome proliferators on rate Leydig cell function in vitro (abstract). *Fundamental & Applied Toxicology* 30 (1996) 102-108 [*Submitted May 25, 2000*].

43) Liu, R.C., M.E. Hurtt, J.C. Cook, and L.B. Biegel. Effect of peroxisome proliferator, Ammonium Perfluorooctanoate (C8), on hepatic aromatase activity in adult male Crl:CD BR (CD) rats. *Fundamental & Applied Toxicology* 30 (1996) 220-228 [*Submitted May 25, 2000*].

44) Longstaff, E., M. Robinson, C. Bradbrook, J.A., and Styles, F.H. Purchase. Genotoxicity and carcinogenicity of fluorocarbons: Assessment by short-term in vitro tests and chronic exposure in rats (abstract). *Toxicol. Appl. Pharmacol.* 72 (1984) 15-31 [*Submitted May 25, 2000*].

45) Nabbefeld, DJ. An Investigation of the Effects of Fluorochemicals on Liver Fatty Acid-Binding Protein. A thesis submitted to the Graduate School of the University of Minnesota, April 1998 [*Submitted May 25, 2000*].

46) Nabbefeld, et al., Displacement of a Fluorescently Labeled Fatty Acid Analogue from Fatty Acid Carrier Proteins by Wyeth – 14,643, Ammonium Perfluorooctanoate, Potassium Perfluorooctane Sulfonate and Other Known Peroxisome Proliferators, Abstract, Society of Toxicology, 1998 Annual Meeting [*Submitted May 25, 2000*].

47) Nilsson, R., B. Beije, V. Préat, K. Erixon, and C. Ramel. On the mechanism of the Hepatocarcinogenicity of peroxisome proliferators. *Chemical-Biological Interactions* 78 (1991) 235-250 [*Submitted May 25, 2000*].

GK002570

EID101477

48) Nordby, G.L., and J.M. Luck. Perfluorooctanoic acid interactions with human serum albumin. *Journal of Biological Chemistry* 219 (1956) 399-404 [*Submitted May 25, 2000*].

49) Obourn, J.D., S.R. Frame, R.H. Bell, Jr., D.S. Longnecker, G.S. Elliott, and J. C. Cook. Mechanisms for the pancreatic oncogenic effects of the peroxisome proliferators Wyeth-14,634 (abstract). *Toxicology & Applied Pharmacology* 145(2) (1997) 425-436 [*Submitted May 25, 2000*].

50) Odell, W.D., R.S. Swerdloff, J. Bain, F. Wollensen, and P.K. Grover. The effect of sexual maturation on testicular response to LH stimulation of testosterone secretion in the intact rat. *Endocrinology* 95 (1974) 1380-1384 [*Submitted May 25, 2000*].

51) Ohya, et al. Determination of Perfluorinated Carboxylic Acids in Biological Samples by High-Performance Liquid Chromatography. *J. Chromatography*, 720 (1998) 1-7 [*Submitted May 25, 2000*].

52) Olsen, G.W., F.D. Gilliland, M.M. Burlew, J.M. Burris, J.S. Mandel and J.H. Mandel. An epidemiologic investigation of reproductive hormones in men with occupational exposure to perfluorooctanoic acid. JOEM 40[7] (July 1998) 614-622 [*Submitted May 25, 2000*].

53) Olsen, Geary W [et al], An Epidemiologic Investigation of Plasma Cholecystokinin and Hepatic Function in Perfluorooctanoic Acid Production Workers, 3M Final Report EPI-0003, (1997) [*Submitted May 25, 2000*].

54) Olson, C.T., and M.E. Anderson. The acute toxicity of perfluorooctanoic and perfluorodecanoic acids in mate rats and effects on tissue acids. *Toxicology & Applied Pharmacology* 70 (1983) 362-372 [*Submitted May 25, 2000*].

55) Olsson, U., C. Sundberg, K. Andersson, and J.W. DePierre. Further studies on the involvement of selenium in peroxisome proliferation in rat liver: Comparison of effects with clofibric acid and perfluorooctanoic acid and the pharmacokinetics of {14C} clofibrate. *Biochemical Pharmacology* 46 (1993) 1805-1810 [*Submitted May 25, 2000*].

56) Ophaug, R.H., and L. Singer. Metabolic handling of perfluorooctanoic acid in rats. *Proc. Soc. Exp. Biol. Med.* 163 (1980) 19-23 [*Submitted May 25, 2000*].

57) Palepu, R., and V.C. Reinsborough. Solution inclusion complexes of cyclodextrins with sodium perfluorooctanoate. *Canadian Journal of Chemistry* 67 (1989) 1550-1553 [*Submitted May 25, 2000*].

GK002571

EID101478

58) Pastoor, T.P., K.P. Lee, M.A. Perri, and P.J. Gillies. Biochemical and morphological studies of ammonium perfluorooctanoate-induced hepatomegaly and peroxisome proliferation. *Experimental and Molecular Pathology* 47 (1987) 98-109 [*Submitted May 25, 2000*].

59) Perkins, R. Investigation of ammonium perfluorooctanoate effect on hormone levels and peroxysome proliferation in the rat. *Toxicologist* 12 (1992) 38 [*Submitted May 25, 2000*].

60) Permadi, H., B. Lundgren, K. Andersson, and J.W. DePierre. Effects of perfluoro fatty acids on xenobiotic-metabolizing enzymes, enzymes which detoxify reactive forms of oxygen and lipid peroxidation in mouse liver. *Biochemical Pharmacology* 44 (1992) 1183-1191 [*Submitted May 25, 2000*].

61) Permadi, H., B. Lundgren, K. Andersson, C. Sundberg, and J.W. Depierre. Effects of perfluorofatty acids on peroxisome proliferation and mitochondrial size in mouse liver - dose and time factors and effect of chain length. *Xenobiotica* 23 (1993) 761 [*Submitted May 25, 2000*].

62) Peterson, R.E., B.I. Kuslikis, and J.P. Vanden Heuvel. Protein acylation by the peroxisome proliferators perfluorodecanoic acid (PFDA) and perfluorooctanoic acid (PFOA) in rats. *Toxicologist* 11 (1991) 185 [*Submitted May 25, 2000*].

63) Reich, I.L., H.J. Reich, L.A. Menahan, and R-E. Peterson. Synthesis of $^{14}$C-labeled perfluorooctanoic and perfluorodecanoic acids: purification of perfluorodecanoic acid. *Journal of Labeled Compounds & Radiopharmaceuticals* 24 (1987) 1235-1244 [*Submitted May 25, 2000*].

64) Rozner, M.A. The effect of perfluorooctanoic acid upon microsomal membrane peroxidation. *Pharmacology* 41 (1981) 2997-3013 [*Submitted May 25, 2000*].

65) Simister, E.A., E.M. Lee, J.R. Lu, R.K. Thomas, R.H. Ottewill, A.R. Rennie, and J. Penfold. Adsorption of ammonium perfluorooctanoate and ammonium decanoate at the air/solution interface. *Journal of the Chemical Society* 88 (1992) 3033-3041 [*Submitted May 25, 2000*].

66) Sohlenius, A.K., A.M. Eriksson, C. Hogstrom, M. Kimland, J.W. Depierre. Perfluorooctane sulfonic acid is a potent inducer of peroxisomal fatty acid B-oxidation and other activities known to be affected by peroxisome proliferators in mouse liver. *Pharmacology & Toxicology* 72 (1993) 90 [*Submitted May 25, 2000*].

EID101479

GK002572

67) Sohlenius, A.K., K. Andersson, and J. DePierre. The effects of perfluoro-octanoic acid on hepatic peroxisome proliferation and related parameters show no sex-related differences in mice. *Biochem. J.* 285 (1992) 779-783 [*Submitted May 25, 2000*].

68) Staples, R.E., B.A. Burgess, and W.D. Kerns. The embryo-fetal toxicity and teratogenic potential of ammonium perfluorooctanoate (APFO) in the rat. *Fundamental & Applied* Toxicology 4 (1984) 429-440 [*Submitted May 25, 2000*].

69) Staples, R.E. Improper interpretation of data concerning teratogenicity: A case report. *Prevention of Physical and Mental Congenital Defects, Part C: Basic and Medical Science, Education and Future Strategies* (1985) 161-163 [*Submitted May 25, 2000*].

70) Takagi, A., K. Sai, T. Umemura, R. Hasegawa, and Y. Kurokawa. Short-term exposure to the peroxisome proliferators, perfluorooctanoic acid and perfluorodecanoic acid, causes significant increases of 8-hydroxydeoxyguanosine in liver DNA of rats. *Cancer Letters* 57 (1991) 55-60 [*Submitted May 25, 2000*].

71) Thottassery, J., L. Wingerg, J. Youssef, M. Cunningham, and M. Badr. Regulation of perfluorooctanoic acid-induced peroxisomal enzyme activities and hepatocellular growth by adrenal hormones. *Hepatology* 15 (1992) 316-322 [*Submitted May 25, 2000*].

72) Treiner, C., and A.K. Chattopadhyay. The partition coefficient of various alcohols between water and alkaliperfluorooctanoate micelles. *Journal of Colloid & Interface Science* 92 (1984) 447-458 [*Submitted May 25, 2000*].

73) Trosko, James E. Oxidative Stress, Signal Transduction, Cell-Cell Communication, U.S. Dept. of Commerce, NTIS # F42960-97-0022 (February 28, 1999) [*Submitted May 25, 2000*].

74) Ubel, F.A., et al. Health Status of Plant Workers Exposed to Fluorochemicals- A Preliminary Report, *Am. Ind. Hyg. Assoc. J.* 41 (August 1980) 584-589 [*Submitted May 25, 2000*].

75) Uy-yu, N., Y. Kawashima, S. Horii, and H. Kozuka. Effects of Chronic Administration of Perfluorooctanoic Acid on Fatty Acid Metabolism in Rat Liver: Relationship among Stearoyl-Coenzyme A Desaturase, 1-Acylglycerophosphocholine Acyltransferase, and Acyl Composition of Microsomal Phosphatidylcholine. *J. Pharmacobio-Dyn.* 13 (1990) 581-5 [*Submitted May 25, 2000*].

GK002573

EID101480

76) Vanden Heuvel, J.P., J.W. Davis, R. Sommers, and R.E. Peterson. Renal excretion of perfluorooctanoic acid in male rats: Inhibitory effect of testosterone. *Journal of Biochemical Toxicology* 7 (1992) 31-36 [*Submitted May 25, 2000*].

77) Vanden Heuvel, J.P., B.I. Kuslikis, and R.E. Peterson. Covalent binding of perfluorinated fatty acids to proteins in the plasma, liver and testes of rats. *Chem.-Biol. Interactions* 82 (1992) 317-328 [*Submitted May 25, 2000*].

78) Vanden Heuvel, J.P., B.I. Kuslikis, M.J. Van Rafelghem, and R.E. Peterson. Tissue distribution, metabolism and elimination of perfluorooctanoic acid in male and female rats. *Journal of Biochemical Toxicology* 6 (1991) 83-92 [*Submitted May 25, 2000*].

79) Vanden Heuvel, J.P., M.J. Van Rafelghem, L.A. Menahan, and R.E. Peterson. Isolation and purification of perfluorodecanoic and perfluorooctanoic acids from rat tissues. *Lipids* 23 (1989) 526-531 [*Submitted May 25, 2000*].

80) Witzmann, F.A., C. Fultz, and J. Lipscomb. Toxicant-induced Alterations in Two-dinensional Electrophoretic Patterns of Hepatic and Renal Stress Proteins. *Electrophoresis* 17 (1996) 198-202 [*Submitted May 25, 2000*].

81) Witzmann, F.A., B.M. Jarnot, D.N. Parker, J.W. Clack. Modification of Hepatic Immunoglobulin Heavy Chain Binding Protein (BIP/Grp 78) Following Exposure to Structurally Diverse Peroxisome Proliferators. *Fundamental & Applied Toxicology* 23 (1994) 1-8 [*Submitted May 25, 2000*].

82) Witzmann, F.A., D. Parker, and B. Jarnot. Induction of Enoyl-CoA Hydratase by LD50 Exposure to Perfluorocarboxylic Acids Detected by Two-dimensional Electrophoresis. *Toxicology Letters* 71 (1994) 271-277 [*Submitted May 25, 2000*].

83) Ylinen, M., A. Kojo, H. Hanhijdrvi, and P. Peura. Disposition of perfluorooctanoic acid in the rat after single and subchronic administration. *Bulletin of Environmental Contamination & Toxicology* 44 (1990) 46-53 [*Submitted May 25, 2000*].

84) Ylinen, M., H. Hanhijdrvi, I. Jaakonaho, and P. Peura. Stimulation by oestradiol of the urinary excretion of perfluorooctanoic acid in the male rat. *Pharmacology & Toxicology* 65 (1989) 274-277 [*Submitted May 25, 2000*].

85) Ylinen, M., H. Hanhijärvi, P. Peura, and O. Räm . Quantitative gas chromatographic determination of PFOA as the benzyl ester in plasma and urine. *Archives of Environmental Contamination & Toxicology* 14 (1985) 713-717 [*Submitted May 25, 2000*].

EID101481

GK002574

86) Youssef, J. and M. Badr. Extraperoxisomal Targets of Peroxisome Proliferators: Mitochondrial, Microsomal, and Cytosolic Effects. Implications for Health and Disease. *Critical Reviews in Toxicology* 28[1] (1998) 1-33 [*Submitted May 25, 2000*].

1. 3M Company. 1992. FC-143 Fluorad™ Brand Fluorochemical Surfactant. MSDS No. 10-3808-2, 3M Industrial Chemical Products Division, St. Paul, MN.

2. 3M Company. 1981. Fluorad TM Brand Fluorochemical Surfactant FC-143. Product Environmental Data, 3M Environmental Engineering and Pollution Control, St. Paul., MN.

3. ACGIH. 1991. Ammonium perfluorooctanoate. In: Documentation of the Threshold Limit Values and Biological Exposure Indices. 6th ed. , Vol. 1. American Conference of Governmental Industrial Hygienists, Inc., Cincinnati, OH, pp. 61-2.

4. Behr, F.E., Johnson, J.D. and Ober, R.E. 1979. Synthesis and Characterization of FC-143-$^{14}$C. 3M Commercial Chemicals Division and Riker Laboratories, Inc., St. Paul, MN.

5. Belisle, J. 1978. Memo to J. Pendergrass regarding IBT 8532-10654 and IBT 8532-10655.

6. Belisle, J. 1978. FC-143 rat toxicity study. Request No. A67821, 3M Central Analytical Laboratory, St. Paul, MN.

7. Belisle, J. 1978. FC-143 analysis of IRDC 137-090 samples. Request No. A68616, 3M Central Analytical Laboratory, St. Paul, MN.

8. Belisle, J. and Bunnelie, V. 1979. FC-143 inhalation study - analysis for $F^-$ and RF. Report No. 7166, 3M Central Research Laboratory, St. Paul, MN.

9. Christopher, B., Marias, A.J., Amold, D.W., Kinoshita, F.K., and Bonahoom, Y. 1977. 28-Day oral toxicity study with FC-143 in albino mice. IBT No. 8532-10655, Industrial Bio-Test Laboratoies, Inc., Northbrook, IL

10. Cook, J.C., Murray, S.M., Frame, S.R. and Hurtt, M.E. 1992. Induction of Leydig cell adenomas by ammonium perfluorooctanoate: a possible endocrine-related mechanism. *Toxicol. Appl. Pharmacol.* 113, 209-17.

11. Dean, W.P., Jessup, D.C., Thompson, G., Romig, G. and Powell, D. 1978. Fluorad™ Fluorochemical FC-143 acute oral toxicity (LD50) study in rats. Report No. 137-091, International Research and Development Corp., Mattawan, MI.

GK002575

12. Handler, J.A., Seed, C.B., Bradford, B.U. and Thurman, R.G. 1992. Induction of peroxisomes by treatment with perfluorooctanoate does not increase rates of $H_2O_2$ production in intact liver. *Toxicol. Lett.* 60, 61 -M.

13. DuPont. 1988. Ammonium perfluorooctanoate. Toxicity Hazard Information for MSDS.

14. Gabriel, K.L. 1976. Primary eye irritation study - rabbits; T-1395. Biosearch, Inc., Philadelphia, PA.

15. Gabriel, K.L. 1976. Primary skin irritation study - rabbits; T-1395. Biosearch, Inc., Philadelphia, PA.

16. Gabriel, K.L. 1976. Primary eye irritation [washout] study - rabbits; T-1395. Biosearch, Inc., Philadelphia, PA.

17. Gabriel, K.L. 1976. Acute oral toxicity - rats [LD50 with T-1395]. Biosearch, Inc., Philadelphia, PA.

18. Gabriel, K.L. 1976. Acute oral toxicity - rat [limit test with T-1385 (sic)]. Biosearch, Inc., Philadelphia, PA.

19. Garry, V.F. and Nelson, R.L. 1981. An assay of cell transformation and cytotoxicity in C3H 10T1/2 clonal cell line for the test chemical T-2942CoC. Stone Research Laboratories, Minneapolis, MN.

20. Goldenthal, E.I., Jessup, D.C., Geil, R.G., Jefferson, N.D. and Arceo, R.J. 1978. Fluorad(R) Fluorochemical FC-143 ninety day subacute rat toxicity study. Report No. 137-089, International Research and Development Corp., Mattawan, MI.

21. Goldenthal, E.I., Jessup, D.C., Geil, R.G. and Mehring, J.S. 1978. Fluorad(R) Fluorochemical FC-143 ninety day subacute rhesus monkey toxicity study. Report No. 137-090, International Research and Development Corp., Mattawan, MI.

22. Gortner, E.G., Lamprecht, E.G. and Case, M.T. 1981. Oral rangefinder study of T-2998CoC in pregnant rats. Expt. No. 0680RR0018, Riker Laboratories, Inc., St. Paul, MN.

23. Gortner, E.G., Lamprecht, E.G. and Case, M.T. 1981. Oral rangefinder study of T-3141CoC in pregnant rabbits. Expt. No. 0681RB0331, Riker Laboratories, Inc., St. Paul, MN.

24. Gortner, E.G., Lamprecht, E.G. and Case, M.T. 1981. Oral teratology study of T-2998CoC in rats. Expt. No. 0681TR0110, Riker Laboratories, Inc., St. Paul, MN.

GK002576

25. Gortner, E.G., Lamprecht, E.G. and Case, M.T. 1982. Oral teratology study of T-3141CoC in rabbits. Expt. No. 0681TBO398, Riker Laboratories, Inc., St. Paul, MN.

26. Griffith, F.D. and Long, J.E. 1980.,Animal toxicity studies with ammonium perfluorooctanoate. *Am. Ind. Hyg. Assoc. J.* 41, 576-583.

27. Jagannath, D.R. and Brusick, D. 1978. Mutagenicity evaluation of T-2015CoC in the Ames Salmonella/microsome plate test. Project No. 20838, Litton Bionetics, Inc., Kensington, MD.

28. Johnson, J.D. and Conard, G.J. 1983. Extent and route of excretion of total carbon-14 pregnant rats after a single oral dose of ammonium $^{14}$C-perfluorooctanoate. Riker Laboratories, Inc., St. Paul, MN.

29. Johnson, J.D. and Ober, R.E. 1979. Absorption of FC-143-$^{14}$C in rats after a single oral
dose. Riker Laboratories, Inc., St. Paul, MN.

30. Johnson, J.D. and Ober, R.E. 1980. Extent and route of excretion and tissue distribution of total carbon-14 in male and female rats after a single IV dose of FC-143-$^{14}$C. Riker Laboratories, Inc., St. Paul, MN.

31. Johnson, J.D. and Ober, R.E. 1980. Enhanced elimination of FC-95-$^{14}$C and FC-143-$^{14}$C in rats with cholestyramine treatment. Riker Laboratories, Inc., St. Paul, MN.

32. Kennedy, G.L., Jr. 1985. Dermal toxicity of ammonium perfluorooctanoate. *Toxicol. Appl. Pharmacol.* 81, 348-355.

33. Kennedy, G.L., Jr., Hall, G.T., Brittelli, M.R., Barnes, J.R. and Chen, H.C. 1986. Inhalation toxicity of ammonium perfluorooctanoate. *Fd. Chem. Toxicol.* 24, 1325-1329.

34. Kennedy, G.L. and Keplinger, M.L. 1976. Reverse mutation studies with T-1485 in five *Salmonella* strains and one *Saccharomyces* strain. IBT No. 854G-09238, Industrial Bio-Test Laboratories, Inc., Northbrook, IL.

35. Metrick, M., Marias, A.J., Arnold, D.W., Kinoshita, F.K., Phillips, S.M. and Bonahoom, Y. 1977. 28-Day oral toxicity study with FC-143 in albino-rats. IBT No. 8532-10654, Industrial Bio-Test Laboratories, Inc., Northbrook, IL.

GK002577

EID101484

36. O'Malley, K.D. and Ebbens, K.L. 1981. Repeat application 28 day percutaneous absorption study with T-2618CoC in albino rabbits. Expt. No. 09790AB0485, Riker Laboratories, Inc., St. Paul, MN.

37. Palazzolo, M.J. 1991. 13-Week toxicity study with T-5180, ammonium perfluorooctanoate (CAS No. 3825-25-1) in male rats. Draft Report No. HWI 6329-100, Hazleton Wisconsin, Madison, WI.

38. Pastoor, T.P., Lee, K.P., Perri, M.A. and Gilles, P.J. 1987. Biochemical and morphological studies of ammonium perfluorooctanoate-induced hepatomegaly and peroxisome proliferation. *Exp. Mol. Pathol.* 47, 98-109.

39. Rusch, G.M. and Rinehart, W.E. 1979. An acute inhalation toxicity study of T-2305CoC in the rat. Project No. 78-7184, Bio/dynamics, Inc.

40. Sibinski, L.J., Allen, J.L. and Erickson, E.E. 1983. Two year oral (diet) toxicity/carcinogenicity study of fluorochemical FC-143 in rats. Expt. No. 0281CR0012, Riker Laboratories, Inc., St. Paul, MN.

41. Staples, R.E., Aftosmis, J.G. and Barba, C.M. 1982. C-8 gavage: embryo-fetal toxicity and teratogenicity study in the rat. Report No. 881-82, DuPont Haskell Laboratory, Newark, DE.

42. Staples, R.E., Aftosmis, J.G. and Barba, C.M. 1982. C-8 inhalation: embryo-fetal toxicity and teratogenicity study in the rat. Report No. 881-81, DuPont Haskell Laboratory, Newark, DE.

43. Staples, R.E., Burgess. B.A. and Kerns, W.D. 1984. The embryo-fetal toxicity and teratogenic potential of ammonium perfluorooctanoate (APFO) in the rat. *Fund. Appl. Toxicol.* 4, 429-440.

44. Ubel, F.A., Sorenson, S.D. and Roach, D.E. 1980. Health status of plant workers exposed to fluorochemicals, a preliminary report. *Am. Ind. Hyg. Assoc. J.* 41, 584-589.

45. ACGIH. 1993. Ammonium perfluorooctanoate (draft). In: Documentation of the Threshold Limit Values and Biological Exposure Indices. American Conference of Governmental Industrial Hygienists, Inc., Cincinnati, OH.

46. Hanhijarvi, H., Ophuag, R.H. and Singer, L. 1982. The sex-related difference in perfluorooctanoate excretion in the rat. *Proc. Soc. Exp. Biol. Med.* 171, 51-55.

47. Perkins, R.G. 1988. Summary of the review of the FC-143 two-year feeding study report (reviewed by the consultants). 3M Internal Correspondence to F.D. Griffith. February 3, 1988.

GK002578

48. Vanden Heuvel, J.P., Davis, J.W., Sommers, R. and Peterson, R.E. 1992. Renal excretion of perfluorooctanoic acid in male rats: inhibitory effect of testosterone. *J. Biochem. Toxicol.* 7, 316.

49. Cook, J.C. 1993. DuPont Internal Correspondence provided to J.L. Butenhoff. September 23, 1993.

50. PAFT. 1993. Safety implications of Leydig cell tumors associated with HFA-134a in rats. Program for Alternative Fluorocarbon Toxicity Testing Consortium.

51. Ylinen, M., Hanhijarvi, H., Jaakonaho, J. and Peura, P. 1989. Stimulation by oestradiol of the urinary excretion of perfluorooctanoic acid in the male rat. *Pharmacol. Toxicol.* 65, 274-277.

52. Hanhijarvi, H., Ylinen, M., Haaranen, T. and Nevalainen, T. 1988. A proposed species difference in the renal excretion of perfluorooctanoic acid in the beagle dog and rat. In *New Developments in Biosciences: Their implications for Laboratory Animal Science,* A.C. Beynen and H.A. Solleveld, Eds., pp. 409-412, Martinus Nijhoff, Dordrecht.

53. Kawashima, Y., Uy-Yu, N. and Kozuka, H. 1989. Sex-related difference in the inductions by perfluoro-octanoic acid of peroxisomal -oxidation, microsomal 1-acylglycerophosphocholine acyltransferase and cytosolic long-chain acyl-CoA hydrolase in rat liver. *Biochem. J.* 261, 595-600.

54. Ikeda, T., Aiba, K., Fukuda, K. and Tanaka, M. 1985. The induction of peroxisome proliferation in rat liver by perfluorinated fatty acids, metabolically inert derivatives of fatty acids. *J. Biochem.* 98, 475-4822.

55. Biegel, L.B., Hurtt, M.E., Frame, S.R. and Cook, J.C. 1993. Effects of ammonium perfluorooctanoate (C8) on leydig cell function: in vivo, ex vivo and in vitro studies. *Toxicologist,* 13, 399.

56. Cook, J.C., Hurtt, M.E., Frame, S.R. and Biegel, L.B. 1993. Effects of Wyeth-14,643 (WY) and ammonium perfluorooctanoate (C8) in Crl:CD®BR(CD) Rats. *Toxicologist* 13, 399.

57. Liu, R.C.M., Hurtt, M.E., Cook, J.C. and Biegel, L.B. 1994. Effect of ammonium perfluorooctanoate (C8) on aromatase activity in tissues of male Crl:CD®BR(CD) Rats. *Toxicologist* 14, 300.

58. Uy-Yu, N., Kawashima, Y. and Kozuka, H. 1990. Comparative studies on sex-related difference in biological responses of livers to perfluorooctanoic acid between rats and mice. *Biochem. Pharmacol* 39, 1492-1495.

GK002579

59. Abdellatif, A.G., Preat, V., Taper, H.S. and Roberfroid, M. 1991. The modulation of rat liver carcinogenesis by perfluorooctanoic acid, a peroxisome proliferator. *Toxicol. Appl. Pharmacol.* 111, 530-537.

60. Long, P.L. 1992. Spontaneous Neoplastic Lesions and Selected Non-Neoplastic Lesions in the Crl:-.CD®BR Rat. Charles River, Inc., Kingston, NY.

61. Permadi, H., Lundgren, B., Andersson, K., Sundberg, C. and DePierre, J.W. 1993. Effects of perfluoro fatty acids on peroxisome proliferation and mitochondrial size in mouse liver: dose and time factors and effect on chain length. *Xenobiotica* 23, 761-70.

62. Olson, C.T. and Anderson, M.E. 1983. The acute toxicity of perfluorooctanoate and perfluorodecanoic acids in male rats and effects on tissue fatty acids. *Toxicol Appl. Pharmacol.* 70, 362-72.

63. Kennedy, G.L., Jr. 1987. Increase in mouse liver weight following feeding of ammonium perfluorooctanoate and related fluorochemicals- *Toxicol. Lett.* 39, 295-300.

64. Longstaff, E., Robinson, M., Bradbrook, C., Styles, J.A. and Purchase, I. 1984. Genotoxicity and carcinogenicity of fluorocarbons: assessment by short-term in vitro tests and chronic exposure in rats. *Toxicol. Appl. Pharmacol.* 72, 15-31.

65. Lundgren, B., Meijer, J., Birberg, W., Piotti, A. and DePierre, J.W. 1988. Induction of cytosolic and microsomal epoxide hydrolases in mouse liver by peroxisome proliferators, with special emphasis on structural analogues of 2-ethylhexanoic acid. *Chem. Biol. Interact.* 68, 219-240.

66. Revici, E. 1986. Treatment of symptoms of neoplastic diseases. U.S. Patent No. 4624851, 11/25/86 (Avram, Elena).

67. Keller, B.J., Marsman, D.S., Popp, J.A. and Thurman, R.G. 1992. Several nongenotoxic carcinogens uncouple mitochondrial oxidative phosphorylation. *Biochim.* Biophys. Acta 1102, 237-244.

68. Perkins, R.G. 1992. Investigation of ammonium perfluorooctanoate effect on hormone levels and peroxisomal proliferation in the rat. *Toxicologist* 12, 38.

69. Fuller, D.R. and Intrasuksri, U. 1993. Mechanism of peroxisome proliferation by perfluorinated fatty acids in primary cultures of rat hepatocytes. *Toxicologist* 13, 395.

GK002580

EID101487

70. Gilliland, F.D. and Mandel, J.S. 1996 Serum perfluorooctanoic acid and hepatic enzymes, lipoproteins, cholesterol and triglycerides: a study of occupationally exposed men. *Am. J. Ind. Med.* 29. 560-568.

71. Gilliland, F.D. and Mandel, J.S. Mortality among employees of a perfluorooctanoic acid production plant. 1993. *J. Occup. Med.* 35, 950 -954.

72. Uy-Yu, N., Kawashima, Y., Horii, S. and Kozuka, H. 1990. Effects of chronic administration of perfluorooctanoic acid on fatty acid metabolism in rat liver: relationship among stearoyl-coenzyme A desaturase, 1-acylglycerophosphocholine acyltransferase, and acyl composition of microsomal phosphatidylcholine. *J. Pharmacobio.-Dyn.* 13, 581-590.

73. Thoftassery, J., Winberg, L., Youssef, J., Cunningham, M. and Badr, M. 1992. Regulation of perfluorooctanoic acid-induced peroxisomal enzyme activities and hepatocellular growth by adrenal hormones. *Hepatology* 15, 316-22.

74. Youssef, J., lgwe, O., Cunningham, M. and Badr, M. 1992. Regulation of hepatic inositol trisphosphate receptors by peroxisome proliferators. *Toxicologist* 12, 37.

75. Gilliland, F.D. 1992. Fluorocarbons and human health: studies in an occupational cohort. Ph.D. Dissertation, U. of Minnesota, Minneapolis, MN.

76. Rogers-Back, A.M., and Clarke, J.J. 1986. Effect of perfluorinated carboxylic acids and polychlorinated biphenyls on intracellular communication processes. *Toxicologist* 6, 39.

77. Andersen, M,E., Rogers, A.M., George, M.E. and Back, K.E. 1983. The toxicity of perfluoro-n-decanoic acid and 2,3,7,8-tetrachlorodibenzo-p-dioxin in L5178Y mouse lymphoma cells. Report No. AFAMRL-TH-82-50, Air Force Aerospace Medical Research Laboratory, Wright-Patterson AFB, Ohio.

78. Kubinski, H., Gutzke, G.E. and Kubinski, Z.O. 1981. DNA-cell-binding (DCB) assay for suspected carcinogens and mutagens. *Mutat. Res.* 89, 95-136.

79. Guy, W.S., Taves, D.R. and Brey, W.S. 1976. Organic fluorocompounds in human plasma: prevalence and characterization. In: R. Filler, ed., *Biochemistry Involving Carbon-Fluorine Bonds.* ACS Symposium Series No. 28. Washington, DC, American Chemical Society, Vol. 7, p. 117.

80. Rozner, M.A. and Taves, D.R. 1980. Comparison of lipid peroxidation by perfluorooctanoic acid or $CCl_4$. Presented at Society of Toxicology Meeting, Washington, D.C.

EID101488

GK002581

81. Abdellatif, A., Preat, V., Vamecq, J., Nilsson, R. and Roberfroid, M. 1990. Peroxisome proliferation and modulation of rat liver carcinogenesis by 2,3-dichlorophenoxyacetic acid, 2,4,5-trichlorophenoxyacetic acid, perfluorooctanoic acid and nafenopin. *Carcinogenesis* 11, 1899-1902.

82. Haughom, B. and Spydevold, O. 1992. The mechanism underlying the hypolipemic effect of perfluorooctanoic add (PFOA), perfluorooctane sulfonic acid (PFOSA) and clofibric acid. *Biochim. Biophys. Acta* 1128, 65-72.

83. Just, W., Gorgas, K., Hartl, F., Heinemann, P., Salazar, M. and Schimassek, H. 1989. Biochemical effects and zonal heterogeneity of peroxisome proliferation induced by perfluorocarboxylic acids in rat liver. *Hepatology* 9, 570-81.

84. Permadi, H., Lundgren, B., Andersson, K. and DePierre, J.W. 1992. Effects of perfluoro fatty acids on xenobiotic-metabolizing enzymes, enzymes which detoxify reactive forms of oxygen and lipid peroxidation in mouse liver. *Biochem. Pharmacol.* 44, 1183-91.

85. Sohlenius, K., Andersson, K. and DePierre, J. 1992. The effects of perfluoro-octanoic acid on hepatic peroxisome proliferation and related parameters show no sex-related differences in mice. *Biochem. Toxicol.* 285, 779-83.

86. Takagi, A., Sai, K., Umemura, T., Hasegawa, R. and Kurokawa, Y. 1991. Short-term exposure to the peroxisome proliferators, perfluorooctanoic acid and perfluorodecanoic acid, causes significant increases of 8-hydroxydeoxy-guanosine in liver DNA of rats. *Cancer Lett.* 57, 55-60.

87. Ylinen, M., Kojo, A., Hanhijarvi, H. and Peura, P. 1990. Disposition of perfluorooctanoic acid in the rat after single and subchronic administration. *Bull. Environ. Contam. Toxicol.* 44, 46-53.

88. Hanhijarvi, H., Ylinen, M., Kojo, A. and Kosma, U. 1987. Elimination and toxicity of perfluorooctanoic acid during subchronic administration in Wistar rats. *Pharmacol. Toxicol.* 61, 66-8.

89. Ophaug, R. and Singer, L. 1980. Metabolic handling of perfluorooctanoic acid in rats. Proc. *Soc. Exp. Biol. Med.* 163, 19-23.

90. Vanden Heuval, J., Kuslikis, B., Van Refelghem, M. and Peterson, R. 1991. Tissue distribution, metabolism and elimination of perfluorooctanoic acid. *J. Biochem. Toxicol.* 6, 83-92.

91. Bonser, G.M. and Robson, J.M. 1940. The effects of prolonged oestrogen administration upon male mice of various strains: development of testicular tumors in the strong A strain. *J. Pathol. Bacteriol.* 51, 9-22.

GK002582



000425

92. Levitt, D. and Liss, A. 1986. Toxicity of perfluorinated fatty acids for human and murine B cell lines. *Toxicol. Appl. Pharmacol.* 86, 1-11.

93. Levitt, D. and Liss, A. 1987. Perfluorinated fatty acids alter merocyanine 540 dye binding to plasma membranes. *J. Toxicol. Environ. Health.* 20, 303-316.

94. Inoue, T., lwanaga, T., Fukushima, K. and Shimozawa, R. 1988. Effects of sodium octanoate and sodium perfluorooctanoate on the gel-to-liquid crystalline phase transition of dipalmitoylphosphatidylcholine vesicle formation. *Chem. Physics Lipids* 46, 25-30.

95. Due, W., Dieckmann, K., Loy, V. and Stein, H. 1989. lmmunohistological determination of oestrogen receptor. progesterone receptor and intermediate filaments in Leydig cell tumors, Leydig cell hyperplasia and normal Leydig cells of the human testis. *J. Pathol.* 157, 225-234.

96. Castle, W. and Richardson, J. 1986. Leydig cell tumors and metachronous Leydig cell
hyperplasia: a case associated with gynecomastia and elevated urinary estrogens. *J. Urol.* 136, 1307-1308.

97. Rail, T. and Schleifer, L. 1990. Drugs effective in the therapies of epilepsies. In: A. Gilman, et al., eds. *The Pharmacological Basis of Therapeutics.* New York, Pergamon, pp. 450-1.

98. Nordby, G.L. and Luck, J.M. 1956. Perfluorooctanoic acid interactions with human serum albumin. *J. Biol. Chem.* 219, 399-404.

99. Belisle, J. and Hagen, D.F. 1978. Method for the determination of the total fluorine content of whole blood, serum/plasma, and other biological samples. *Anal. Biochem.* 87, 545-55.

100. Belisle, J. and Hagen, D.F. 1980. A method for the determination of perfluorooctanoic acid in blood and other biological samples. *Anal. Biochem.* 101, 369-376.

101. Venkateswarlu, P. 1975. Determination of total fluorine in serum and other biological materials by oxygen bomb and reverse extraction techniques. *Anal. Biochem.* 68, 512-521.

102. Belisle, J. 1981. Organic fluorine in human serum: natural versus industrial sources. *Science*, 212, 1509-10.

EID101490

GK002583

103. Venkateswarlu, P. 1982. Sodium biphenyl method for determination of covalently bound fluorine in organic compounds and biological materials. *Anal. Chem.* 54,1132-37.

104. Johnson, J.D., Gibson, S.J. and Ober, R.E. 1984. Cholestramine-enhanced fecal elimination of carbon-14 in rats after administration of ammonium [$^{14}$C]perfluorooctanoate or potassium [$^{14}$C]perfluorooctanesulfonate. *Fund. Appl. Toxicol.* 4, 972-976.

105. Kuslikis, B.I., Vanden Heuvel, J.P. and Peterson, R.E. 1992. Lack of evidence for perfluorodecanoyl- or perfluorooctanoyl-coenzyme A formation in male and female rats. J. *Biochem. Toxicol* 7, 25-29,

106. Hosokawa, M. and Satoh, T. 1993. Differences in the induction of carboxylesterase isozymes in rat liver microsomes by perfluorinated fatty acids. *Xenobiotica* 23, 1125-33.

107. Vanden Heuvel, J.P., Kuslikis, B.I. and Peterson, R.E. 1992. Covalent binding of perfluorinated fatty acids to proteins in the plasma, liver and testes of rats. *Chem. Biol. Interact.* 82, 317-328.

108. Glaza M.S. Single dose dermal absorption/toxicity study of T-6075 in rabbits. 1995. Laboratory Project number HWI 6329-153. Hazelton Wisconsin Inc. Madison WI.

109. Nabbefeld D., Butenhoff J., Bass N. and Seacat A. 1998. Displacement of a fluorescently labeled fatty acid analogue from fatty acid carrier proteins by wyeth-14,643, ammonium perfluorooctanoate, potassium perfluorooctane sulfonate and other known peroxisome proliferators. Abstract. Accepted, *Toxicologist* 1998).

110. Olsen G.W., Gilliland FD, Burlew M.M., Burris J.M., Mandel J.S., and Mandel J.H. An epidemiological investigation of reproductive hormones in men with occupational exposure to perfluorooctanoic acid. Abstract, Final Report, Manuscript submitted for publication 9/97.

111. Obourn J.D., Frame S.R., Elliot G.S., and Cook J.C. 1997. Pancreatic oncogenic effects of Wyeth 14,643. (Abstract 1181) *Toxicologist.* 36, 232-233.

112. Johnson J. D. Final Report- Analytical study. Single-dose dermal absorption/toxicity study of T-6075 in rabbits. 1995 (in vivo reference number: HWI#6329-153, Study number: AMDT-011795.1) 3M Environmental Laboratory

113. Murli H. Mutagenicity test on T-6358 measuring chromosomal aberrations in Chinese hamster ovary (CHO) cells. 1996. CHV Study No.: 17388-0-437. Corning Hazelton Inc. Vienna, VA.

GK002584

114. Murli H. Mouse micronucleus test on T-6358 in an in-vivo mouse micronucleus assay. 1996. CHV Study No.:17388-0-455. Corning Hazelton Inc. Vienna, VA.

115. Müller W. Perfluorooctanoic acid ammonium salt, solution 30% Chromosome abberations *in vitro* in V79 Chinese hamster cells. 1996. Study number 96.0003. Hoechst Aktiengesellschaft Pharma Development Corporate Toxicology. Frankfurt am Main.

116. Müller W. Perfluorooctansäureammoniusazlösung (30% ig) Study of the mutagenic potential in strains of Salmonella typhimurium (AMES TEST). 1996. Study number 96.0002. Hoechst Aktiengesellschaft Pharma Development Corporate Toxicology. Frankfurt am Main.

117. Murli H. Mutagenicity test on T-6342 measuring chromosome aberrations in human lymphocytes with a confirmatory assay with multiple harvests. 1996. CHV study No.: 17073-0-449CO. Corning Hazelton Inc. (CHV), Vienna ,VA.

118. Liu, R.C.M., Hurtt M.E., Cook J.C. and Biegel L.B. 1996. Effects of the peroxisome proliferator, ammonium perfluorooctanoate (C8) on hepatic aromatase activity in adult male Crl:CD BR (CD) rats. *Fund. Appl. Toxicol.* 30, 220-228

119. Liu R.C.M., Hahn C., and Hurtt M.E. 1996. The direct effect of hepatic peroxisome proliferators on rat Leydig cell function *in vitro*. *Fund. Appl. Toxicol.* 30,102-108.

120. Biegel L.B., Liu R.C.M., Hurtt M.E., and Cook J.C. Effects of ammonium perfluorooctanoate on leydig cell function: *In-vitro*, *in-vivo*, and *ex-vivo* studies. 1995. *Toxicol. Appl. Pharmacol.* 134, 18-25.

121. Wallace K. B. and Starkov A. 1998. The effect of perfluorinated arylalkylsulfonamides on bioenergetics of rat liver mitochondria. Dept of Biochemistry and Molecular Biology, University of MN School of Medicine. Duluth, MN 55812, USA. Supported by a grant from 3M Company.

122. Nabbefeld D. 1998 An Investigation of the Effects of Fluorochemicals on Liver Fatty Acid-Binding Protein. Masters Thesis, University of Minnesota. Thesis research performed at and supported by 3M.

/ams 1/26/98, replaces: /scg 9/14/94

GK002585

EID101492

GK002586

**References:**

Adams, M.R., Kaplan, J.R., Clarkson T.B. and Koritnik D.R. 1985. Ovariectomy, social status and atherosclerosis in Cynomolgus monkeys. Arteriosclerosis 5:192-200.

Baselet R.C. and Carvey, R.H. 1995. Disposition of f Toxic Drugs and Chemicals in man, Fourth edition. Chemical Toxicology Institute, Foster City, CA.

Cattley RC, DeLuca J, Elcombe C, Fenner-Crisp P, Lake BG, Marsman DS, Pastoor TA, Popp JA, Robinson DE, Schwetz B, Tugwood J, Wahli W. Do peroxisome proliferating compounds pose a hepatocarcinogenic hazard to humans? Regul Toxicol Pharmacol. 1998 Feb;27(1 Pt 1):47-60.

Colvin, P.L., Wagner J.D., Adams, M.R., and Sorci-Thomas, M.G. 1998. Sex steroids increase cholesterol 7 α-Hydroxylase mRNA in nonhuman primates. Metabolism: Clinical and Experimental. 47:391-395.

Cook JC, Murray SM, Frame SR, Hurtt ME. 1992. Induction of Leydig cell adenomas by ammonium perfluorooctanoate: a possible endocrine-related mechanism. Toxicol Appl Pharmacol. Apr;113(2):209-17.

DePaolo L.V. and Masaro E.J. 1989 Endocrine Hormones In Laboratory Animals. In *The Clinical Chemistry of Laboratory Animals*. Eds W. Loeb and F. Quimby. New York :Parmagon Press. (Note: These are secondary references.

Eldridge, S.R., Butterworth, B.E., and Goldsworthy, T.L. Proliferating cell nuclear antigen: a marker for hepatocellular proliferation in rodents. Environ. Health Perspect. 101: 211-218, 1993.

Eldridge, S.R. February 25 2000. 26-week Capsule Toxicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOA;T-6295) in Cynomolgus Monkeys. Draft Cell Proliferation report. Client Number 6329-223, 3M T-6295.7. Pathology Associates International, Frederick, MD.

Goldenthal, E.I., Jessup, D.C., Geil, R.G., Jefferson, N.D. and Arceo, R.J. 1978a. Ninety-day subacute rat study. Study No. 137-085, International Research and Development Corporation, Mattawan, MI.

GK002587

EID101494

Goldenthal, E.I., Jessup, D.C., Geil, R.G. and Mehring, J.S. 1978b. Ninety-day subacute rhesus monkey toxicity study. Study No. 137-092, International Research and Development Corporation, Mattawan, MI.

Guy, WS, Taves, and DR, Brey, Jr., WS (1976). Organic fluorocompounds in human plasma: prevalence and characterization. (In) Biochemistry Involving Carbon-Fluorine Bond, pages 117-134.

Haughom, B. and Spydevold, O. 1992. The mechanism underlying the hypolipemic effect of perfluorooctanoic acid (PFOA), perfluorooctane sulphonic acid (PFOAA) and clofibric acid. *Biochim. Biophys. Acta.* 1128, 65-72.

Johnson, J. D. and Ober, R.F . 1 979. Absorption of FC-95-14C in rats after a single oral dose. Project No. 8900310200, Riker Laboratories, Inc., St. Paul, MN.

Johnson, J.D. and Behr, F.E- 1979. Synthesis and Characterization of FC-95-$^{14}$C. Project No. 8900310200, Riker Laboratories, Inc., St. Paul, MN.

Johnson, J.D., Gibson, S.J. and Ober, R.E. 1979. Extent and route of excretion and tissue. distribution of total carbon-14 in rats after a single i.v. dose of FC-95-$^{14}$C. Project No. 8900310200, Riker Laboratories, Inc., St. Paul, MN.

Johnson, J.D., Gibson, S.J. and Ober, R.E. 1980. Enhanced elimination of FC.-95-$^{14}$C and FC-143-$^{14}$C in rats with cholestyramine treatment. Project No. 8900310200, Riker Laboratories, Inc., St. Paul, MN.

Johnson, J.D., Gibson, S.J. and Ober, R.E. 1984. Cholestramine-enhanced fecal elimination of carbon-14 in rats after administration of ammonium [$^{14}$C]perfluorooctanoate or potassium [$^{14}$C]perfluorooctanesulfonate. *Fund. Appl. Toxicol.* 4, 972-976.

Just W. W., Gorgas K., Hartl F-U., Heinemann P., Salzer M. and Schimassek H. 1989. Biochemical effects and zonal heterogeneity of peroxisome proliferation induced by perfluorocarboxylic acids in rat liver. Hepatology 9:570-581 et al. 1989

Kennedy G.L. Jr., Keller D. A., Biegel L.B. and Brock W.J. 1998. Repeated dose toxicityu of fluorinated acids in rats. *Toxicologist* Volume 42. Abstract No. 1829 p. 371.

Larson P.R. and Ingbar S.H. 1992 The Thyroid Gland. In *Williams Textbook of Endocrinology*, 8th ed. Eds. J. D. Wilson and D. W. Foster, pp 357-487, Philadelphia: W.B. Saunders Co).

Nabbefeld D., Butenhoff J., Bass N. and Seacat A. 1998. Displacement of a fluorescently labeled fatty acid analogue from fatty acid carrier proteins by wyeth-14,643, ammonium

perfluorooctanoate, potassium perfluorooctane sulfonate and other known peroxisome proliferators. *Toxicologist* 41. 1998.

Nold J.B. July 13 1999. 26-week Capsule Toxicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOA;T-6295) in Cynomologus Monkeys - Draft Pathological Report (Ancilliary Study) Electron Microscopic Evaluation of Liver in Cynomologus Monkeys. Client Number 6329-223 PAI Study Number EM 99.76. Pathology Associates International, Durham NC.

Nold, J.B. May 30, 2000. 26-week Capsule Toxicity Study with Perfluorooctane Sulfonic Acid Potassium Salt (PFOA;T-6295) in Cynomologus Monkeys - Draft Pathological Report (Ancilliary Study) Electron Microscopic Evaluation of Liver in Cynomologus Monkeys (Recover Animals). Client Number 6329-223 PAI Study Number EM 99.76. Pathology Associates International, Durham NC.

O'Conner J.C., Frame, S.C., Biegel L.B. Cook J.C. and Davis L.G. Sensitivity of a Tier 1 screening battery compared to an *in-utero* exposure for detecting the estrogen receptor agonist 17 β-estradiol. Tox. Sci. 44, 169 – 184, 1998.

Olsen G.W., Burris J.M., Mandel J.H., Zobel L.R. Serum perfluorooctane sulfonate and hepatic and lipid clinical chemistry tests in fluorochemical production employees. JOEM 41:799-805.

Reimers J.R. 1999. Hormones. In: *The Clinical Chemistry of Laboratory Animals. Second edition.* Eds.: Loeb W.F and Quimby F. W. pp 455-499, Philadelphia PA. Taylor and Frances.

Smith, D.R. 1998. Animal models: Nutrition and lipoprotein metabolism. *Current Opinion in Lipidology.* 9:3-6.

Turley, S.D., Spady D.K., and Dietschy J.M. 1995. Role of liver in the synthesis of cholesterol and the clearance of low density lipoproteins in the Cynomolgus monkey Journal of Lipid Research. 36:67-79.

Wagner, J.D., Zhang, L., and Adams, M.R. 1997. Effect of estrogens on arterial LDL metabolism. In *Hormonal, metabolic, and cellular influences on cardiovascular disease in women.* Ed. T.L. Forte, pp 153-174. New York: Futura Publishing Co.

Wallace K. B. and Starkov A. 1998. The effect of perfluorinated arylalkylsulfonamides on bioenergetics of rat liver mitochondria. Dept of Biochemistry and Molecular Biology, University of MN School of Medicine. Duluth, MN 55812, USA. Supported by a grant from 3M Company.

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September 29, 2000

Dr. Peter J. Thomford, Ph.D.
Covance Laboratories Inc.
3301 Kinsman Blvd.
Madison WI 53704

Dear Dr.Thomford,

Enclosed please find a copy of the Draft Cell Proliferation Report for the Study identified below:

**26-Week Capsule Toxicity Study with Ammonium Perfluorooctanoate (APFO) in Cynomolgus Monkeys**

**Covance Study Number 6329-231**

Sincerely,

Kim Claggett

Kim Claggett
Research Assistant
Cell Kinetics Division
301-624-2917

Enclosure

Covance Laboratories
Madison, WI

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# DRAFT
# CELL PROLIFERATION REPORT

## 26-WEEK CAPSULE TOXICITY STUDY WITH AMMONIUM PERFLUOROOCTANOATE (APFO) IN CYNOMOLGUS MONKEYS
## COVANCE STUDY NUMBER 6329-231

**PREPARED FOR:**

**3M**
**TOXICOLOGY SERVICES**
**BUILDING 220-2E-02, 3M CENTER**
**ST. PAUL, MN 55144-1000**

**PREPARED BY:**

**PATHOLOGY ASSOCIATES INTERNATIONAL**
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**SEPTEMBER 26, 2000**

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# CELL PROLIFERATION REPORT FOR:

# 26-WEEK CAPSULE TOXICITY STUDY WITH AMMONIUM PERFLUOROOCTANOATE (APFO) IN CYNOMOLGUS MONKEYS COVANCE STUDY NUMBER 6329-231

## INTRODUCTION

The purpose of the study was to assess the effect of the test material on critical enzyme levels, hormones, and other selected biochemical parameters when administered daily by capsule to cynomolgus monkeys for at least 26 weeks.

This report, submitted by Pathology Associates International (PAI) to the study Sponsor, 3M Toxicology Services, represents the cell proliferation findings and interpretation for Covance Study Number 6329-231 entitled "26-Week Capsule Toxicity Study with Ammonium Perfluorooctanoate In Cynomolgus Monkeys".

## REGULATORY COMPLIANCE

All aspects of the tasks associated with PAI's portion of this study were conducted in compliance with the Environmental Protection Agency Good Laboratory Practice (GLP) Regulations as set forth in Title 40 of the US Code of Federal Regulations, Part 792, issued November 29, 1983(effective December 29, 1983), and with any applicable amendments.

## MATERIALS AND METHODS

### Experimental Design

| Treatment Group | Dose Level (mg/kg/day) | Number of Animals Main Study | Recovery |
|---|---|---|---|
| 1 (Control) | 0 | 4 | 2 |
| 2 (Low-dose) | 3 | 4 | 0 |
| 3 (Mid-dose) | 10 | 4 | 2 |
| 4 (High-dose) | 30 | 4 | 2 |

All animals (main study and recovery) were treated for up to 26 weeks. Two animals in groups 1, 3 and 4 were designated as recovery animals. Treatment in these animals was discontinued and the Sponsor determined the recovery period.

**Tissue Collection for Cell Proliferation**

Representative samples of the left lateral lobe of the liver, left and right testes, and pancreas from each of the 22 animals was fixed and processed to paraffin block by Covance per protocol specifications. Tissue blocks were shipped to PAI for sectioning and staining. From each block, slides were prepared for H&E evaluation and immunohistochemical detection of proliferating cell nuclear antigen (PCNA), a marker of cell proliferation.

**Immunohistochemistry for Cell Proliferation**

Sections of paraffin-embedded tissues were cut ≅ 5 µm and placed on positively charged slides (Superfrost Plus, Fisher Scientific, Pittsburgh, PA) to ensure adhesion during processing for PCNA. Standard immunohistochemical methods for PCNA (Eldridge, et al., 1993) were used to stain tissues. Briefly, tissue sections were incubated with a monoclonal antibody to PCNA ( Lot # 107 Exp. 11/00 DAKO, Carpinteria, CA) and reagents required for the avidin-biotin peroxidase (MslgG Kit Lot# PK6102 Exp. 4/22/00, Vector, Burlingame, CA) method for the detection of the antigen-antibody complex. PCNA expression in cells in all phases of the cell cycle (G1, S, G2 and M) was localized by the chromagen 3,3'-diaminobenzidine (DAB Lot# 108H8210 Exp. 8/20/01; Sigma Chemical Co., St. Louis, MO). Tissue sections were counterstained with hematoxylin.

**Cell Proliferation Measurements**

Positive staining for PCNA was categorized based on cellular distribution and intensity of the brown to black reaction product that correlated with the different phases of the cell cycle (Eldridge et al., 1993). Uniform, dark brown to black nuclear staining was judged to be positive for S phase cells. A cell in the $G_1$ phase of the cell cycle had diffuse, stippled nuclear staining that was lighter than a cell in S phase. Cells in the $G_2$ phase exhibited distinct, diffuse cytoplasmic stippling with or without nuclear staining. Mitotic figures were conspicuous with diffuse cytoplasmic stippling. A negative control slide was included in the staining run and consisted of study tissue (animal no. I05709) that was not incubated with the primary antibody.

For cell proliferation evaluations, slides were first perused at low magnification (100X) to judge quality of staining, processing and sectioning, pattern of cell labeling (e.g., centrilobular or panlobular, extrahepatocellular proliferation, such as Kupffer cells, bile duct epithelium, endothelium), and histomorphologic changes. The later was further assessed by evaluating the serial H&E slide for each animal evaluated for cell proliferation. Cell proliferation was then quantified at higher magnification (200X) as described above.

For cell proliferation measurements in the liver, the percentage of dark, nuclear-stained hepatocytes that represent cells in S-phase was determined by scoring at least 3000 hepatocytes in 10 randomly selected fields per animal. Cell proliferation within islet and exocrine cells of the pancreas was scored subjectively with 3 = exocrine stained greater than islets, and 4 = islets and exocrine stained heavily as determined by positive PCNA staining. The entire tissue section

represented on the slide was perused at 100X and 400X. For measuring cell proliferation in the testes, the section farthest from the slide label was scored. The percentage of PCNA-labeled Leydig cells was determined by scoring at least 100 Leydig cells in the entire section that was examined.

### Statistical Analysis

Due to the small samples sizes within main study and recovery groups, and because not all animals within a treatment group were sacrificed on the same day, statistical analysis was not performed on cell proliferation data.

## RESULTS

### Cell Proliferation

Individual animal cell proliferation data are presented in Section II (Table II-1) and graphically in Figures III-1 - 3. Clearly, cell proliferation was not increased by treatment in the pancreas or testes of monkeys as determined by examining the scatter plots in Section II, Figures III-2 and III-3. In the liver, however, there did appear to be an enhancement of cell proliferation in the highest dose tested (30 mg/kg/day) as revealed in Section II, Figure (III -1). Data points 6, 8, 9 and 10 (30 mg/kg/day; animals I05704, I05713, I05711, I05722, and I05703, respectively) were greater than or equal to the highest control value (data point 1; animal I05714). The lowest dose tested (data point 3; 3 mg/kg/day), however, had one animal (animal I05706) with a labeling index that exceeded all animals examined. Thus, an apparent increase in cell proliferation was not necessarily dose-related.

### Histopathology

Sections from the same tissue blocks used for preparation of PCNA-stained slides were stained with hematoxylin and eosin (H&E) for histopathologic evaluation to facilitate the interpretation of the immunostained slides. Individual animal findings are presented in Section IV.

Representative sections of liver, pancreas and testes from 22 monkeys were evaluated histologically. Each tissue section was stained with hematoxylin and eosin. The purpose of this evaluation was to determine whether or not morphologic changes were occurring in these tissues that may alter or confound the specificity of the PCNA staining observed in these animals.

The results showed that no significant changes that would alter the interpretation of the PCNA staining in this study were observed in the liver, pancreas or testes from male monkeys. Although chronic inflammation was reported for several animals, it was minimal in severity and occurred across all groups. Furthermore, the PCNA labeling index did not correlate with the observation of inflammation in the liver. For example, inflammation was not observed in the animal with the highest liver labeling index (animal I05706). Numerous findings were reported for animal I05724 that included hypertrophy, inflammation, fat infiltration and possibly bacterial

colonization. Based on the substantial non-specific PCNA staining observed in the liver of this animal, clearly these lesions confounded the ability to measure hepatocellular proliferation; therefore, no attempt was made to evaluate the PCNA labeling index in animal I05724.

## DISCUSSION

In the present study, cell proliferation was measured within the liver, pancreas and testes of male monkeys from control and treated groups after up to 26 weeks on study, as well as a recovery period. An increase in cell proliferation, as determined by the PCNA labeling index, was not observed in the pancreas or testes of male monkeys. Cell proliferation in the liver, however, was equivocal.

## SUMMARY

After up to 26 weeks on study, enhanced cell proliferation was not evident in the pancreas or testes of male monkeys, whereas in the liver the findings were equivocal.

## LITERATURE CITED

Eldridge, S.R., Butterworth, B.E., Goldsworthy, T.L. (1993). Proliferating cell nuclear antigen: a marker for hepatocellular proliferation in rodents. *Environ. Health Perspec.* 101: 211-218.

## II. TABLES

TABLE II-1. CELL PROLIFERATION IN MONKEYS

| Dose Group | Day of Death | Animal Number | Labeling Index Liver | Labeling Index Pancreas | Labeling Index Testes |
|---|---|---|---|---|---|
| 0 mg/kg/day (Control) | 26 Weeks | IO5709 | 0.025% | 3 | 22.86% |
| 0 mg/kg/day (Control) | 26 Weeks | IO5714 | 0.075% | 3 | 11.00% |
| 0 mg/kg/day (Control) | 26 Weeks | IO5715 | 0.000% | 3 | 14.91% |
| 0 mg/kg/day (Control) | 26 Weeks | IO5725 | 0.074% | 4 | 27.00% |
| 0 mg/kg/day (Control) | Recovery animal | IO5718 | 0.025% | 3 | 41.51% |
| 0 mg/kg/day (Control) | Recovery animal | IO5720 | 0.000% | 3 | 23.42% |
| 3 mg/kg/day (Low dose) | 26 Weeks | IO5702 | 0.000% | 4 | 10.58% |
| 3 mg/kg/day (Low dose) | 26 Weeks | IO5706 | 0.170% | 3 | 21.78% |
| 3 mg/kg/day (Low dose) | 26 Weeks | IO5717 | 0.099% | 3 | 22.77% |
| 3 mg/kg/day (Low dose) | Sacrificed week 20 | IO5721 | 0.000% | NP | 13.00% |
| 10 mg/kg/day (Mid dose) | 26 Weeks | IO5707 | 0.075% | 3 | 15.53% |
| 10 mg/kg/day (Mid dose) | 26 Weeks | IO5708 | 0.050% | 3 | 17.14% |
| 10 mg/kg/day (Mid dose) | 26 Weeks | IO5710 | 0.024% | 3 | 17.92% |
| 10 mg/kg/day (Mid dose) | 26 Weeks | IO5719 | 0.025% | 3 | 35.17% |
| 10 mg/kg/day (Mid dose) | Recovery animal | IO5712 | 0.073% | 3 | 18.81% |
| 10 mg/kg/day (Mid dose) | Recovery animal | IO5716 | 0.000% | 4 | 10.89% |
| 30 mg/kg/day (High dose) | 26 Weeks | IO5704 | 0.100% | 3 | 10.78% |
| 30 mg/kg/day (High dose) | 26 Weeks | IO5713 | 0.075% | 4 | 15.84% |
| 30 mg/kg/day (High dose) | Sacrificed day 29 | IO5724 | ND | 3 | 16.35% |
| 30 mg/kg/day (High dose) | Stopped dosing week 7; Sacrificed week 27 | IO5711 | 0.126% | 4 | 21.70% |
| 30 mg/kg/day (High dose) | Stopped dosing week 10; Sacrificed week 27 | IO5722 | 0.149% | 3 | 14.29% |
| 30 mg/kg/day (High dose) | Stopped dosing week 12; Sacrificed week 27 | IO5703 | 0.075% | 3 | 20.19% |

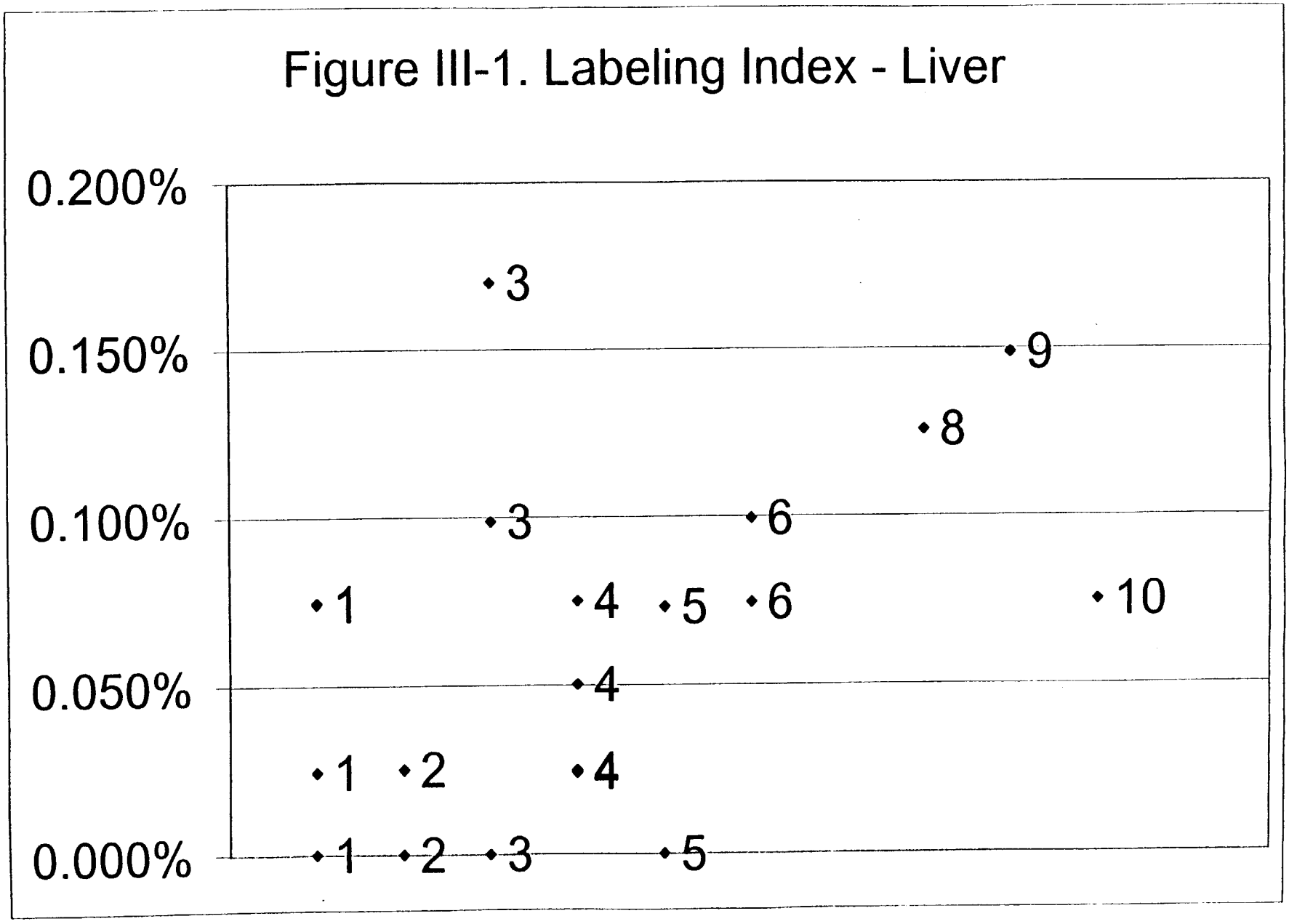
Skip slide=negative control; animal no. IO5709
ND, not determined due to too much non-specific staining
NP, tissue not present on slide
Liver: Only dark nuclear stained hepatocytes scored as S-phase; 200X
Pancreas: Scored subjectively with 4=islets and exocrine stained heavily (>50% labeled); 3=exocrine stained greater than islets; perused at 100X and 400X
Testes: Section farthest from slide label scored; Leydig cells scored at 400X

## III. FIGURES

**Legend for data points on Figures III-1 - 3**

| Group | Treatment | Day of Death |
|---|---|---|
| 1 | 0 mg/kg/day | 26 weeks |
| 2 | 0 mg/kg/day | Recovery animal |
| 3 | 3 mg/kg/day | 26 weeks |
| 4 | 10 mg/kg/day | 26 weeks |
| 5 | 10 mg/kg/day | Recovery animal |
| 6 | 30 mg/kg/day | 26 weeks |
| 7 | 30 mg/kg/day | Sacrificed day 29 |
| 8 | 30 mg/kg/day | Stopped dosing day 43 |
| 9 | 30 mg/kg/day | Stopped dosing day 66 |
| 10 | 30 mg/kg/day | Stopped dosing day 81 |

# Figure III-1. Labeling Index - Liver



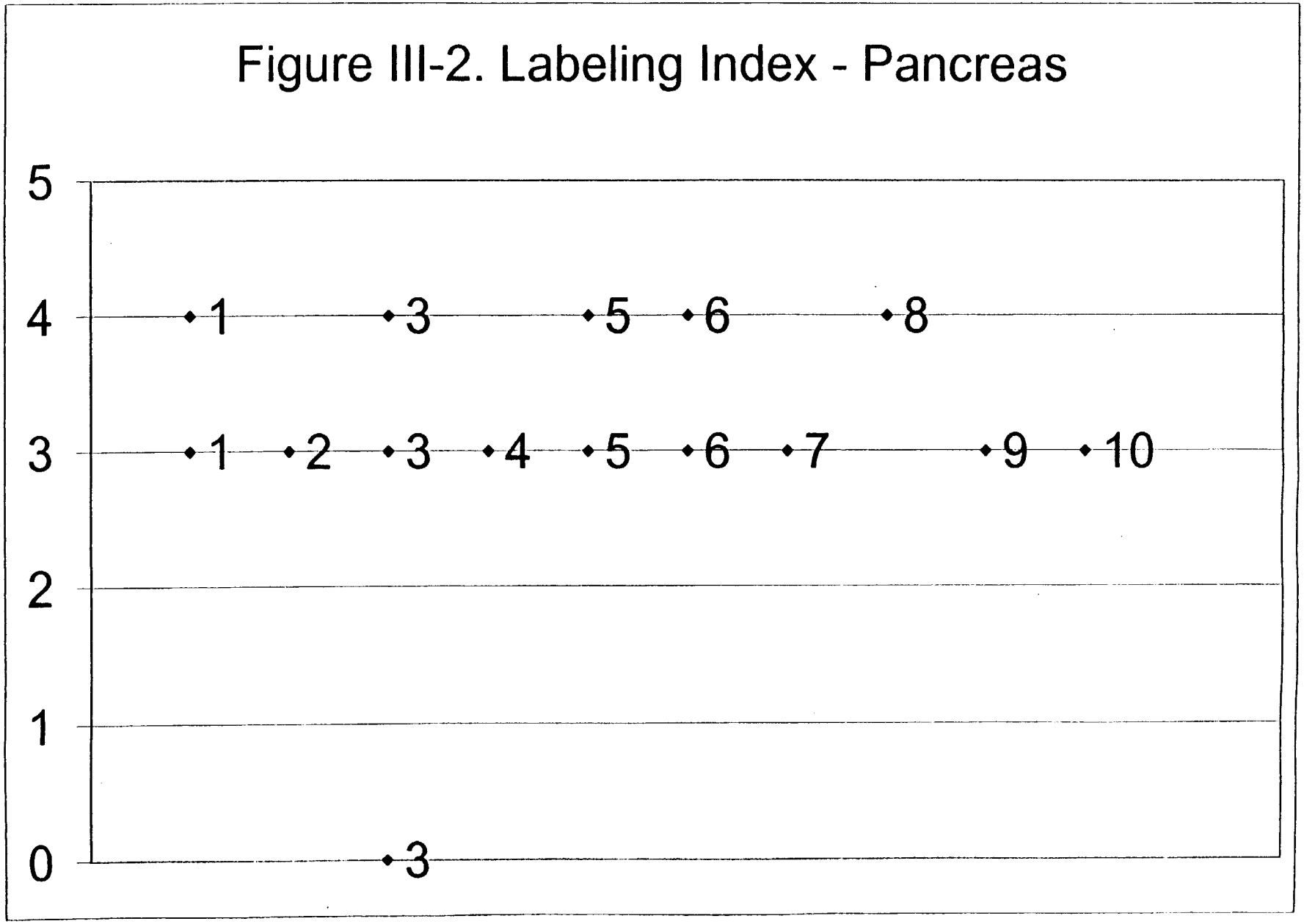


Figure III-2. Labeling Index - Pancreas
5
4
3
2
1
0
1
3
5
6
8
1
2
3
4
5
6
7
9
10
3

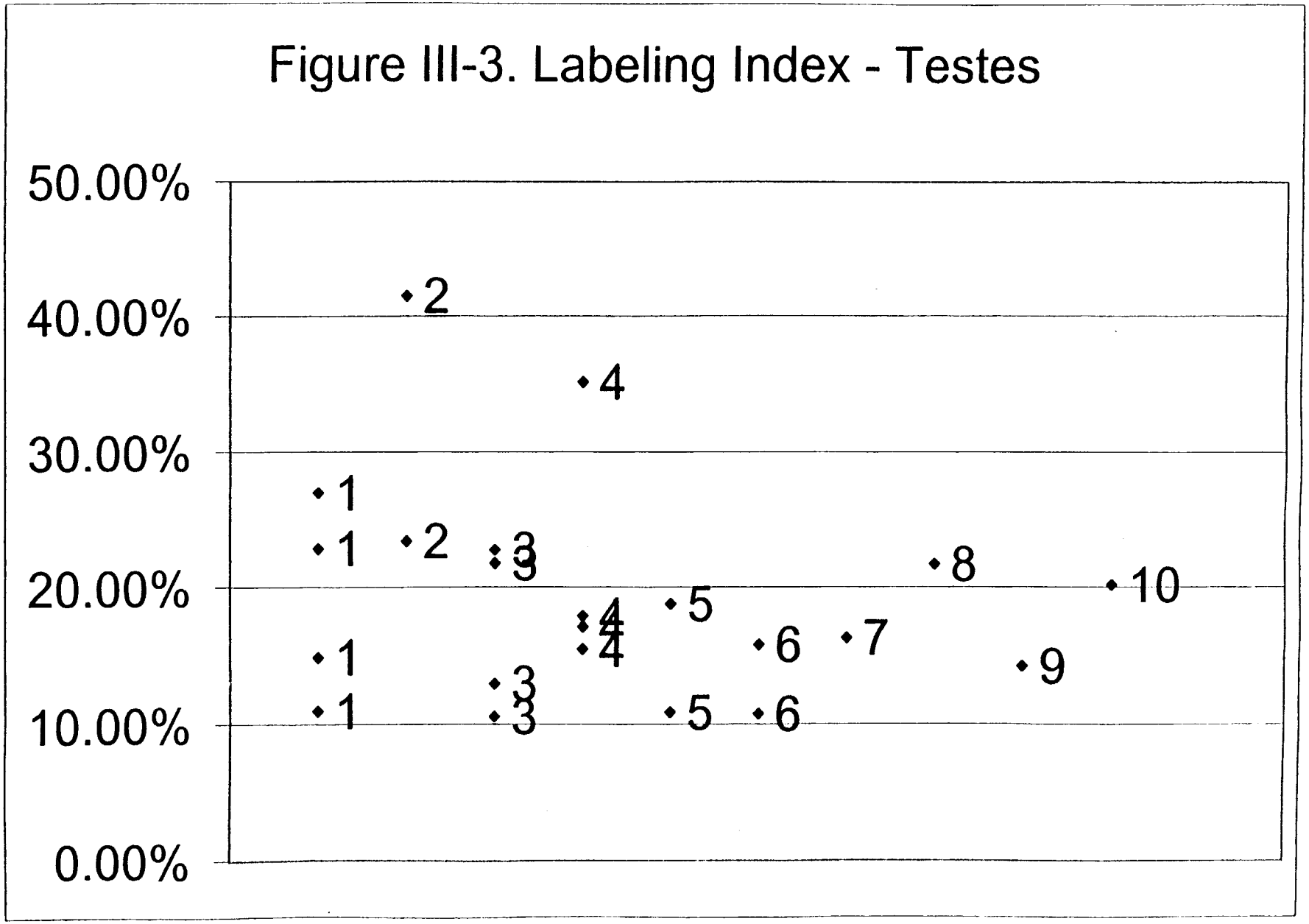


Figure III-3. Labeling Index - Testes
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
1
2
3
4
5
6
7
8
9
10

# IV. INDIVIDUAL ANIMAL HISTOPATHOLOGY FINDINGS

## Individual Animal Findings

# 26-WEEK CAPSULE TOXICITY STUDY WITH AMMONIUM PERFLUOROOCTANOATE (APFO) IN CYNOMOLGUS MONKEYS

| ANIMAL NUMBER | SEX | DOSE GROUP | HISTOLOGIC FINDINGS |
|---|---|---|---|
| I05709 | M | 1 | Liver - chronic inflammation, minimal<br>Pancreas - autolysis, focal<br>Testes - crush artifact<br>Spleen - NSF |
| I05714 | M | 1 | Liver - chronic inflammation, minimal<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05715 | M | 1 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05725 | M | 1 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05718 | M | 1 | Liver - NSF<br>Pancreas - NSF<br>Testes - crush artifactS<br>Spleen - NSF |
| I05720 | M | 1 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05702 | M | 2 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - hemosiderin deposition, mild |
| I05706 | M | 2 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05717 | M | 2 | Liver - NSF<br>Pancreas - NSF<br>Testes - crush artifact<br>Spleen - NSF |
| I05721 | M | 2 | Liver - chronic inflammation, minimal<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |

**Legend:** NSF = no significant findings

## Individual Animal Findings

## 26-WEEK CAPSULE TOXICITY STUDY WITH AMMONIUM PERFLUOROOCTANOATE (APFO) IN CYNOMOLGUS MONKEYS

| ANIMAL NUMBER | SEX | DOSE GROUP | HISTOLOGIC FINDINGS |
|---|---|---|---|
| I05707 | M | 3 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - hemosiderin deposition, minimal |
| I05708 | M | 3 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05710 | M | 3 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05712 | M | 3 | Liver - chronic inflammation, minimal<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05716 | M | 3 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05719 | M | 3 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05703 | M | 4 | Liver - NSF<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05704 | M | 4 | Liver - chronic inflammation, minimal<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - hemosiderin, minimal |
| I05711 | M | 4 | Liver - chronic inflammation, minimal<br>- hemosiderin, minimal<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05713 | M | 4 | Liver - chronic inflammation, minimal<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |



**Legend:** NSF = no significant findings

## Individual Animal Findings

# 26-WEEK CAPSULE TOXICITY STUDY WITH AMMONIUM PERFLUOROOCTANOATE (APFO) IN CYNOMOLGUS MONKEYS

| ANIMAL NUMBER | SEX | DOSE GROUP | HISTOLOGIC FINDINGS |
|---|---|---|---|
| I05722 | M | 4 | Liver - hemosiderin, minimal<br>Pancreas - NSF<br>Testes - NSF<br>Spleen - NSF |
| I05724 | M | 4 | Liver - periportal , fat infiltration, severe<br>periportal, cellular hypertrophy, severe<br>periportal, chronic inflammation,moderate<br>midzonal, necrosis, moderate to severe<br>centrilobular, fat infiltration, severe<br>centrilobular, cellular hypertrophy, severe<br>random distribution, bacterial colonies???<br>Pancreas - islets, fat infiltration, mild to moderate<br>Testes - NSF<br>Spleen - NSF |



**Legend:** NSF = no significant findings

## V. SIGNATURE PAGE

Submitted by:

Project Manager:

Sandra R. Eldridge, Ph.D. _____ Date

Project Pathologist:

Carolyn Moyer, D.V.M., Diplomate, A.C.V.P. _____ Date

# VI. QUALITY ASSURANCE STATEMENT

# Cell Proliferation Report

## 26-Week Capsule Toxicity Study With Ammonium Perfluorooctanoate (APFO) in Cynomolgus Monkeys

Covance Study Number: 6329-228

## QUALITY ASSURANCE STATEMENT

This cell proliferation project has been inspected and audited by the PAI Quality Assurance Unit (QAU) as required by the Good Laboratory Practice (GLP) regulations promulgated by the U.S. Environmental Protection Agency. The cell proliferation report is an accurate reflection of the recorded data. The following table is a record of the inspections/audits performed and reported by the QAU.

| Date of Inspection | Phase Inspected | Date Findings Reported to PAI Management/Project Manager |
|---|---|---|
| 10/25/99 | PCNA Dilutions/Staining | 10/25/99 |
| 09/06,07/00 | Study Data and Supporting Documentation | 09/07/00 |
| 09/06,07,25/00 | Draft Cell Proliferation Report | 09/07/00 |

Doraine Dundee
Quality Assurance Auditor

September 29, 2000
Date

# APPENDIX I

## PROTOCOL DEVIATION REPORT

| | |
|---|---|
| Date: | September 28, 2000 |
| Study Number: | Covance Study No. 6329-231 |
| Title: | 26-Week Capsule Toxicity Study with Ammonium Perfluorooctanoate In Cynomolgus Monkeys |
| Protocol Deviation #1: | Representative samples of liver, testes and pancreas were to be collected for cell proliferation evaluation, which included preparation of an H&E slide for correlative morphologic interpretation of the cell proliferation findings. Samples of spleen were inadvertently submitted to PAI and the H&E slide examined for this tissue. |
| Reason: | Spleen was inadvertently submitted. The H&E slide was inadvertently evaluated microscopically and the histologic findings reported. This deviation had no impact on the study. |
| Submitted by: | Sandra R. Eldridge, Ph.D.<br>PAI Project Manager |

Sandra R. Eldridge
PAI Project Manager

9-28-00
Date

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| Local LF | 1996 | 1997 | 1998 |
|---|---|---|---|
| MW-04 | 39 | 14 | 26 |
| MW-06 | .97 | 11 | 9 |
| MW-09 | .14 | ND | ND |

| DRY RUN LF | 1996 | 1997 | 1998 | 1999 |
|---|---|---|---|---|
| OGI OUTLET | | 33 | 17 | |
| LEACHATE | | 62 | 56 | |
| STREAM I | 7.2 | 11 | 1.0 | |
| PROPERTY BOUNDARY | 9.9 | 2.0 | .88 | |
| MW-13 | | 7.0 | 9.2 | |
| MW-6 | 15 | 1.0 | NA | |

| OTHER OFFSITE | 6/91 |
|---|---|
| NEW LUBECK WELL | 2.4 |

| SITE DRINKING TAPS | |
|---|---|
| BLDG 3 | L.6 |
| BLDG 212 | L.6 |

ASI1010156

EID103018

00067

# WASHINGTON WORKS

## 1997/1998 C-8 SAMPLING RESULTS ug/L

### LETART LANDFILL

| | 1998 | 1997 |
|---|---|---|
| LOWER POND | 1100 | 1600 |
| UPPER POND | 480 | 100 |
| MW-6 | 30 | |
| MW-2A | 990 | 460 |
| MW-4 | 260 | 53 |
| MW-1 | 24000 | 5100 |
| MW-3 | 2700 | 2000 |
| STREAM 5-B | ND | 2.0 |
| | NA | 480 |

### [illegible] LANDFILL

| | 1998 | 1997 |
|---|---|---|
| TW-25 (PLANT [illegible]) | [illegible] | |
| MW-04 | 26 | 14 |
| MW-06 | 9 | 11 |
| MW-09 | ND | ND |
| MW-10 | .22 | |

### PLANT SUPPLY

| | 98 | 97 |
|---|---|---|
| WELL 331 | 1.0 | .55 |
| WELL 336 | .4 | .79 |
| WEST WELL FIELD | NA | 7.9 |

*SEE 1997 DATA FOR PLANT OUTFALLS

### [illegible] LANDFILL

| | 1998 | 1997 |
|---|---|---|
| MW-11 | NL | ND |
| MW-12B | ND | |
| STREAM I | 1.0 | 11 |
| STREAM II | 4.6 | |
| OCI OUTLET | 17 | 33 |
| LEACHATE | 56 | 62 |
| MW-13 | 9.2 | 7.0 |
| MW-12 | ND | ND |
| MW-12A | ND | ND |
| MW-04 | .52 | |
| MW-13A | 8.7 | 15 |
| MW-6 | ND | 1.0 |
| OCI BOTTLE | NL | 23 |

| | 1997 |
|---|---|
| PROPERTY BOUNDARY | 2.0 |
| 1000 FT D.S. | 3.5 |

ASH010160

EID103019

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## C-8 OFF SITE SAMPLING

| | | C-8 PPB |
|---|---|---|
| 3/13/87 | LUBECK BUSINESS TAP (2) | 1.9, 1.9 |
| 5/12/88 | LPSD HOME TAP -P | 2.2 |
| 11/ 2/88 | LPSD HOME TAP -P | 1.4 |
| 5/ 7/89 | LPSD HOME TAP -P | 0.7 |
| 5/23/91 | LPSD HOME TAP -M | 3.8 |
| 5/29/91 | LPSD HOME TAP -C | 3.8 |
| 8/ 8/91 | LPSD HOME TAP -M | 3.9 |
| 3/13/87 | VIENNA HOME TAP -M | <0.6 |
| 3/13/87 | LITTLE HOCKING BUSINESS TAP | <0.6 |
| 5/12/88 | LITTLE HOCKING HOME TAP -R | <0.6 |
| 11/28/90 | LUBECK PRIVATE WELLS (2) | <0.6, <0.6 |
| 8/ 9/91 | LUBECK PRIVATE WELLS (2) | <1.0, <1.0 |
| 6/23/91 | NEW LUBECK WELL | 2.4 (*) |

(*) CH2MHILL CONFIRMED "PRESENCE" OF C-8

ASI010161

EID103020

000429

## C-8 HUMAN EXPOSURE

| LIMITS | uG/M3 |
|---|---|
| TLV (3M) | 100 |
| AEL (DUPONT) | 10 |
| CEG (AIR, WATER) | |

HASKELL ESTABLISHED: 8 uG C-8 PER 24 HOURS

80% BY AIR ..... 6.4 uG/ 20M3 = 0.32 OR 0.3 uG/M3

20% BY WATER ..... 1.6 uG/ 2 L = 0.80 OR 1 PPB

OUTSIDE CONTRACT LAB: CH2MHILL

$23M AUTH TO PROVIDE 0.1 PPB C-8 IN WATER ANALYSIS

ASI1010162

EID103021

-Pcm

## C-8 SAMPLING (MARCH - JUNE 1984)

| LOCATION | DISTANCE (MILES) | C-8 PPB (0.6 LIMIT) |
|---|---|---|
| PKSBG-HOME TAP | 7.5 UPSTREAM | < |
| WW-DRINK FTN | --- | < |
| DIST. CTR-WELL | 0.25 DOWN | < |
| WASHINGTON-STORE TAP | 0.25 DOWN | 1.2,1.0 |
| LUBECK-STORE TAP | 0.25 DOWN | 1.5 |
| L. HOCKING-STORE TAP | 3 DOWN | 0.8,0.6 |
| BELLEVILLE-PRIVATE WELL | 12 DOWN | < |
| REEDSVILLE-STORE TAP | 14 DOWN | < |
| RAVENSWOOD-STORE TAP | 29 DOWN | < |
| RACINE-STORE TAP | 50 DOWN | < |
| POINT PLEASANT-STORE TAP | 74 DOWN | < |
| GALLIPOLIS-STORE TAP(*) | 79 DOWN | < |

(*) NEAREST COMMUNITY TO TAKE WATER DIRECTLY FROM OHIO RIVER.

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EID103022

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# C-8 ON SITE SAMPLING

| TEST WELL #27 | C-8 PPB | |
|---|---|---|
| 6/ 4/87 | 2.0 | |
| 5/11/88 | 1.5 | |
| 11/ 4/88 | 1.3 | |
| 5/ 4/89 | <0.6 | |
| 8/ 1/89 | 1.3 | |
| 10/24/89 | 1.5 | |
| 2/27/90 | 1.5 | |
| 4/20/90 | 1.5 | |
| 7/13/90 | 1.6 | |
| 8/ 9/90 | <10 | (3.0) |
| 10/19/90 | <10 | (3.0) |
| **REVISED TEST** | | |
| 1/15/91 | 2.9 | |
| 4/18/91 | 3.0 | |
| 7/24/91 | 5.9 | |
| 8/ 2/91 | 5.0 | |
| **ADJACENT WELL: MW-4** | | |
| 5/13/91 | 1.5 | |
| 8/ 1/91 | 1.4 | |

| MW DRINKING WATER | | |
|---|---|---|
| 3/13/87 | BLDG 3 | <0.6 |
| 11/ 2/88 | BLDG 212 | <0.6 |
| 5/12/89 | BLDG 212 | <0.6 |
| 5/ 8/90 | BLDG 212 | <0.6 |

ASH010164

EID103023

**Robert L Ritchey**
09/14/2000 03:52 PM

To: H David Ramsey/AE/DuPont@DuPont
cc: George Woytowich/CL/DuPont@DuPont, Andrew S Hartten/AE/DuPont@DuPont (bcc: Robert L Ritchey/CL/DuPont)
Subject: GE sampling results

Spoke with Dale Van de Velde at GE this afternoon. He got his FC-143 results back a few days ago from their August 15, 2000 sampling of drinking water wells. Well #3 analyzed at .707 ug/l. Well #4 analyzed at .275 ug/l.
Well #3 is closest to us. He estimated #4 to be 600-700 feet further downriver. He believes they pump about 50% from each well, which would be consistent with the earlier composite result at a site tap. He asked if we had seen numbers as high as the .707 ug/l and I pointed out to him that in the TSCA material we sent to EPA, which he has a copy of, we discuss a .8 ug/l result on a Lubeck well and a .965 ug/l modeled result in the river. We briefly discussed influences such as river flow, distance downriver, and test accuracy.
He indicated that they plan to continue to sample periodically.

RLR003302

EID103379

ATTACHMENT 130

is TSCA CBI.

DCN 63D1000014

No sanitized version

was provided.

130

EXTRACTABLE ORGANICS ANALYSIS DATA SHEET

CLIENT SAMPLE NO

TP02

Lab Name: PACIFIC ANALYTICAL, INC.
Matrix: WATER
Sample wt/vol: 1000.000 ml
Concentrated Extract Volume: 1000 ul
Injection Volume: 1 ul

Lab Sample ID: 08702
Lab File ID: 712614
Date Sampled: 08/17/00
Date Extracted: 08/22/00
Date Analyzed: 08/26/00
Time Analyzed: 1827

| Compound | Sample Conc ug/L | MDL ug/L | Surrogate Recovery |
|---|---|---|---|
| Perfluorooctanoic Acid Este | 0.1727 | 0.1187 | 84% |

U = Undetected  J = Estimated Concentration  B = Found in Blank
D = Dilution Results  E = Result Exceeds Calibration Curve
na = Compound has no corresponding labeled reference
PQL = Practical Quanitation Limit

EXTRACTABLE ORGANICS ANALYSIS DATA SHEET

CLIENT SAMPLE NO

| TR04 |
|---|

| | | | |
|---|---|---|---|
| Lab Name: PACIFIC ANALYTICAL, INC. | | Lab Sample ID | 98704 |
| Matrix: WATER | | Lab File ID | 713b1s |
| Sample Wt/Vol: 1000.000 ml | | Date Sampled | 08/17/00 |
| Concentrated Extract Volume: | 1000 ul | Date Extracted | 09/22/00 |
| Injection Volume: 1 ul | | Date Analyzed | 09/26/00 |
| | | Time Analyzed | 1940 |

| Compound | Sample Conc ug/L | MDL ug/L | Surrogate Recovery |
|---|---|---|---|
| Perfluorooctanoic Acid Este | 0.1205 | 0.1187 | 83% |

U = Undetected J = Estimated Concentration B = Found in Blank
D = Dilution Results E = Result Exceeds Calibration Curve
na = Compound has no corresponding labeled reference
PQL = Practical Quanitation Limit

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for faster computers.

* Carborundum Co., Niagara Falls, N.Y., opened a plant in Phoenix last year to develop ceramic materials and products for microelectronic compenents. The company makes silicon carbide, aluminum nitride and boron nitride, which are all used as insulators and heat dissipaters for semiconductor chips.

* W.L. Gore & Associates Inc., based in Delaware, has teams in Flagstaff and Phoenix still finding uses for a substance patented in 1937: Teflon. Gore researchers in Flagstaff work on new medical products, such as liners for casts, synthetic blood vessels and artificial ligaments. Researchers there and in Phoenix work on electronic products such as cables to detect leaks in buried fuel tanks.

* Hexcel Corp., San Francisco, has been making lightweight structural materials since 1986 in Casa Grande. Its composite, honeycomb-textured materials lighten many aircraft and cushioned the first manned landing on the moon.

GRAPHIC: Photo

LANGUAGE: ENGLISH

UMI-ACC-NO: 9021645

LOAD-DATE: June 10, 1994

LEXIS·NEXIS LEXIS·NEXIS LEXIS·NEXIS

321ST STORY of Level 1 printed in FULL format.

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January, 1986

SECTION: Sec 1; pg 24

LENGTH: 1749 words

HEADLINE: Grand Isle's One Manufacturer a One-of-a-Kind

BYLINE: Michele Patenaude

DATELINE: South Hero; VT; US

BODY:

There is only one manufacturer in the entire county of Grand Isle -- Phoenix Wire Inc. Not only is the firm unique in its county, but it is also one-of-a-kind in the world, according to company officials.

"We do only one thing," says Horace Corbin, the firm's president, "we insulate wire with Teflon." The company's claim to fame is not in insulating wire with Teflon. Many companies do that. Phoenix insulates the tiniest wires in the world.

Called micro miniature wires, some of them are several times finer than a human hair. Single strands can be virtually invisible.

They were to Tom Parizo, Phoenix's vice president and product engineer, until he started wearing glasses six months ago. "Product quality has improved tremendously since I got them," he jokes.

These tiny wires may end up in such extraordinary places as the inside of a human brain or an instrument panel on the space shuttle. The combination of small size and Teflon coating makes them ideal in applications involving medicine and electronics.

In most cases the wire makes a long and complicated journey from its origins at Phoenix to its final home in a piece of equipment, so Corbin is not sure where it all ends up. But NASA is a regular customer and so are medical establishments famous around the world. He does know for sure that it is used in cardiac catheters (tubes), pacemakers, probes and sutures.

Teflon, a DuPont trade name for polytetrafluoethelene or PTFE, has some unique properties that make it especially appropriate for use inside the body. Chemically, it is inert; it doesn't react to other substances such as body tissues and fluids, which makes it one of the safest materials available for implantation in the body.

Also, because Teflon can be applied to wire in extremely thin layers, it is



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ideal for use in miniaturized electronics. And it's one of the best of all materials for electrical insulation.

The most obvious property of Teflon -- one known to anyone familiar with a modern kitchen -- is that nothing sticks to it. For that reason some of Phoenix's product is used in such "low tech" operations as cookie cutting, chocolate cutting and the cutting of plastic bags. Phoenix Wire is a small company. Only five full-time and two part-time employees work on the South Hero side street in a building as small and unassuming as its products. Except for listings in Thomas' Register and the Yellow Pages, advertising is a word-of-mouth affair.

Ohmer Corbin, Horace's cousin and the firm's sales manager, says a recent phone call he received is indicative of how that works. An Irish doctor called to place an order for some wire he had been shown by a Spanish doctor at a conference in Isreal. The wire had been coated at Phoenix.

Customers call from as far away as France, India and Japan, and as nearby as the University of Vermont 20 miles south.

All the work is done on a custom basis. The customer ships the wire to Phoenix, where it is coated to the specified thickness and color, and then shipped back to the customer. Horace and Ohmer Corbin, as well as Tom Parizo, live within five miles of the business. That, probably more than any other factor, is the reason for the plant's location on Station Road in South Hero.

Because they all love living near the water and the boating and fishing it affords, Grand Isle's Lake Champlain environs offer them a perfect place to live and work. And much to their delight they have discovered that being off the beaten path helps keep salespeople from beating a path to the company door. The firm maintains a profile so low they'd have to camouflage the building to make it any harder to find. Conspicuously absent is any kind of an identifying sign.

The location, while convenient for the principals, is not a critical concern for the business. Production requires little space, few resources and shipping is done through United Parcel Service.

"Our product is a lightweight, high-value product," says Horace Corbin, "We could be anywhere we wanted." That fact has led Parizo to suggest they put the whole operation into a large trailer truck and drive south to Florida as the winter approaches and the lake begins to freeze over.

Eight miles wide and more than 30 gently rolling miles long, Grand Isle County is the state's smallest. Located in the northwestern corner of Vermont and comprised mostly of islands, it is known for its beauty, its shoreline, its farms and apple orchards. Tourists and cows form the bedrock of its economy and undoubtedly will for a long time to come.

Parizo, a native of the county, would like to see more light industry there to broaden the tax base. Present development in the county is primarily residential. Parizo explains that houses, and the families they bring to the community, consume more in services than the taxes they put into the coffers. "To have a good economic system you have to have a balance between residential, commercial and industrial.

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Rebecca Kaiser agrees with him. As the executive director of the Grand Isle County Development Corporation (GICDC), she would also like to see more light industry in the county. GICDC is a two-year-old, nonprofit organization, one of 12 Vermont development corporations, and is supported by public and private funds. Its board of directors are resident volunteers and its mission is to provide more jobs for the community.

Presently the GICDC is developing a couple of sites as potential locations for manufacturing concerns. A recently completed countywide survey indicates residents are opposed to heavy industry being located there, but are more favorable to light industry.

Kaiser says the survey results show "residents generally support an expansion of the tax base and the generation of more local jobs."

Ironically, for a county surrounded by water, water is the major obstacle to industrial development -- both getting it and getting rid of it. The area's clay soil is a poor one for septic leaching. And although water can be pumped in from the lake to shoreside sites, away from shore water must be drilled for, and even then it is often found to be contaminated with sulfur.

Water is not a probelm for Phoenix Wire because so little is used in the production process. In that process, called "dispersion coating," wire is drawn through a milky looking liquid which is actually a Teflon and water suspension. The wire then travels through an oven which bakes the coating on. The process is repeated over and over until the required thickness of insulation is reached.

Most other types of wire are insulated with polyvinyl chloride or polyethylene using an extrusion process. Unlike Teflon dispersion coating, this process does not ensure uniform thickness of insulation around the wire. It is also more prone to pinholes because the coating is a single layer rather than the multi-layers of the Teflon process. The only major disadvantage of Teflon as a wire insulator is that it is soft and subject to abrasion. Sometimes a layer of a harder material is applied over the Teflon to solve this problem.

Over the years, Phoenix has developed its methods and equipment to perform the operation efficiently. The company owns six complex machines, each of which very slowly feeds the wire from the spools through the Teflon suspension into an over and then back on the spools again. Production continues around the clock from Monday morning until Thursday afternoon.

Three-day weekends and long summer vacations are the rule at Phoenix Wire. (Nobody here is overworked," says Horace Corbin.) It's for benefits such as these that the company policy is to stay small. Corbin says the demand for small wires is growing and he intends for the company to keep pace and grow to meet the demand. But expanding into other areas is definitely not in the picture. The headaches and problems that go with a large business are something he has every intention of avoiding, after experiencing them himself.

At one time the business employed 350. That was before a fire destroyed it in January of 1969. At that time the company was known as Thermal Wire, and besides coating wire of all types, the company was also a sub-contractor for electronic sub-assemblies. IBM was a major customer.

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It all began in 1956 when Horace Corbin got out of the service and decided to start his own business. His initial inclination was toward hardware or boating supplies, but a friend convinced him of the profits to be found in wire. Parizo and cousin Ohmer came in on it almost from the beginning. The business was indeed profitable and it grew. It was housed in a large modern plant only a short distance away from the present Phoenix Wire location.

Then came the fire. At about the same time IBM phased out the product for which Thermal was making the subassemblies.

In December after the fire the company resumed operation in an unscathed Thermal warehouse which is the present site. The business was renamed Phoenix after the mythological bird that burned itself on a furneral pyre and rose up anew from the ashes.

It was at this time a deliberate decision was made to keep the business small. Teflon dispersion wire coating, only a small fraction of what had been Thermal's business, was chosen to be the sole business of Phoenix because it was both profitable and required few employees.

"With 350 people you've got 350 problems," Corbin says of the decision. "We're much happier doing it this way."

It was a good choice for many reasons, not just the personal preferences of the principals. The large wire companies that had been coating miniature wires began to drop out to concentrate on other aspects of the wire business that, for them, were more profitable and did not require extensive employee training. (Corbin says it takes a minimum of two years to properly train an operator.)

Eventually Phoenix was the only one left. And so customers from all over the world who need the tiny Teflon insulated wires must do business with Grand Isle's one and only manufacturer.

"We'll do anything we can to help a customer develop a product," says Horace Corbin, "however we don't have the philosophy that the customer is always right. We've been in this business long enough to know our wire is good wire, we know what can and can not be done. We're independent Vermonters. If they push us too far we'll tell them to go someplace else."

The question is -- where?

LANGUAGE: ENGLISH

UMI-ACC-NO: 8602610

LOAD-DATE: October 9, 1995



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IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF WEST VIRGINIA
PARKERSBURG DIVISION

COPY-The original was filed in the Clerk's Office at Charleston on
OCT 21 1999
SAMUEL L. KAY, CLERK
U.S. District & Bankruptcy Courts
Southern District of West Virginia

WILBUR EARL TENNANT, ERWIN JACKSON TENNANT, JAMES DAVID TENNANT, DELLA MARIE TENNANT, and SANDRA K. TENNANT,

Plaintiffs,

v.

CIVIL ACTION NO. 6:99-0488

E.I. DUPONT DE NEMOURS & CO., INC.,

Defendant.

## SECOND AMENDED COMPLAINT

Now come plaintiffs Wilbur Earl Tennant, Erwin Jackson Tennant, James David Tennant, Della Marie Tennant and Sandra K. Tennant ("Plaintiffs") and state as follows for their Second Amended Complaint against defendant E.I. DuPont de Nemours & Co., Inc. ("DuPont") herein:

## NATURE OF ACTION

1. This is a civil action for declaratory, injunctive, and equitable relief, monetary damages, and response costs incurred and to be incurred by Plaintiffs for bodily injury and property damage to Plaintiffs and their property arising from the acts and/or omissions of DuPont during the ownership and/or operation of DuPont's Dry Run Landfill in Wood County, West Virginia, defined herein as the "Site" under the authority of: (i) the Comprehensive Environmental Response, Compensation and Liability Act of 1980, as amended ("CERCLA"), 42 U.S.C. ¶¶ 9601 *et seq.*; (ii) the Federal Declaratory Judgment Act, 28 U.S.C. §§ 2201 *et seq.*; (iii) the Resource Conservation and Recovery Act of 1976, as amended ("RCRA"), 42 U.S.C. §§ 6901 *et seq.*; the Clean Water Act, as amended ("CWA"), 33 U.S.C. §§ 1251 *et seq.*; (v) the West Virginia Hazardous Waste

Management Act (the "Waste Act"), W.Va. Code §§ 22-18-1 *et seq.*; and (vi) principles of common law.

## JURISDICTION AND VENUE

2. This Court has jurisdiction over this action pursuant to: 28 U.S.C. §§ 1331, 1332, 1367, 2201, and 2202; Sections 107 and 113 of CERCLA, 42 U.S.C. §§ 9607 and 9613; Section 7002(a) of RCRA, 42 U.S.C. § 6972(a); and Section 505(a) of CWA, 33 U.S.C. § 1365(a).

3. This Court has pendant and ancillary/supplemental jurisdiction over Plaintiffs' claims arising under state statutory and federal and state common law.

4. Venue is proper in this District pursuant to 28 U.S.C. § 1391(b); Sections 107 and 113 of CERCLA, 42 U.S.C. §§ 9607 and 9613; Section 7002(a) of RCRA, 42 U.S.C. § 6972(a); and Section 505(c) of CWA, 33 U.S.C. § 1365(c), because the discharge, disposal and/or release of solid, toxic and/or hazardous wastes, substances, pollutants, and/or contaminants that gives rise to these claims occurred and/or is occurring in this District, because the real property that is the subject of this action is located within this District, and because a substantial part of the events or omissions giving rise to these claims occurred and continues to occur within this District.

## PARTIES

5. Plaintiff Wilbur Earl Tennant is an individual residing at Route 3, Box 17, Washington, Wood County, West Virginia 26181.

6. Plaintiff Erwin Jackson Tennant is an individual residing at Route 3, Box 17A, Washington, Wood County, West Virginia 26181.

7. Plaintiff James David Tennant is an individual residing at Route 3, Box 372, Parkersburg, Wood County, West Virginia 26101.



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8. Plaintiff Della Marie Tennant is an individual residing at Route 3, Box 372, Parkersburg, Wood County, West Virginia 26101.

9. Plaintiff Sandra K. Tennant is an individual residing at Route 3, Box 17, Washington, Wood County, West Virginia 26181.

10. Defendant E.I. DuPont de Nemours ("DuPont") is a Delaware corporation authorized to conduct business in the State of West Virginia and has a principal place of business at 1007 Market Street, Wilmington, Delaware 19898.

## GENERAL ALLEGATIONS

11. Since approximately 1982, DuPont has owned and/or operated a facility known as the E.I. DuPont Dry Run Landfill located adjacent to Route No. 68 in the Harris District of Wood County, West Virginia, near the town of Lubeck, which facility is referred to herein as the "Site" and identified generally for illustrative purposes only as the "Site" on Exhibit A to Plaintiffs' First Amended Complaint.

12. During DuPont's ownership and/or operation of the Site, DuPont has disposed of one or more solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants at the Site, including but limited to, asbestos, fly ash, bottom ash, boiler ash, coal ash, filter cake sludges, polyamides, acrylics, polyacetal, polyvinyl butyral, polyethylene terephthalate, fluropolymers, paraffin wax, sludges from DuPont's Washington Works plant, filter aids, construction dirt, paper, cardboard, glass, scrap piping and metals, and railroad ties.

13. During DuPont's ownership and/or operation of the Site, DuPont has failed to compact wastes appropriately at the Site.



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14. During its ownership and/or operation of the Site, DuPont has failed to adequately control fugitive dust at the Site.

15. During its ownership and/or operation of the Site, DuPont has failed to maintain adequate slopes of its landfill surface areas at the Site.

16. During its ownership and/or operation of the Site, DuPont has failed to properly control stormwater runoff at the Site.

17. During its ownership and/or operation at the Site, DuPont has failed to adequately control landfill leachate and runoff at the Site.

18. During its ownership and/or operation of the Site, DuPont has failed to install and or maintain an appropriate leachate collection and/or treatment system at the Site.

19. During its ownership and/or operation of the Site, DuPont has failed to install and or maintain appropriate catch basins at the Site.

20. During its ownership and/or operation of the Site, DuPont has failed to install and or maintain appropriately lined sedimentation/settling ponds at the Site.

21. During its ownership and/or operation of the Site, DuPont has failed to install and or maintain appropriate runoff/leachate diversion ditches at the Site.

22. During its ownership and/or operation of the Site, DuPont has failed to appropriately close and/or retrofit surface impoundments at the Site.

23. During its ownership and/or operation of the Site, DuPont has failed to submit timely notice of its violation of applicable surface and/or groundwater protection standards at the Site

24. During its ownership and/or operation of the Site, DuPont has failed to notify appropriate government officials that it changed the nature of the waste DuPont disposed at the Site



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25. During its ownership and/or operation of the Site, DuPont has disposed of wastes, including certain sludges, without first obtaining appropriate government approvals.

26. During its ownership and/or operation at the Site, DuPont failed to close a drain valve for one of its leachate/sedimentation ponds resulting in the discharge of pollutants into the environment, including the nearby Dry Run stream.

27. As a result of DuPont's ownership and/or operation of the Site, one or more solid, toxic and/or hazardous wastes, substances, pollutants, and/or contaminants have been and continue to be disposed, discharged, and/or otherwise released into environmental media at the Site (the "Contaminants"), including surface and groundwaters at the Site, including the nearby Dry Run stream.

28. At all times relevant hereto, Plaintiffs collectively have owned, leased, and/or otherwise possessed several hundred acres of real property adjacent and/or contiguous to the Site which properties are collectively referred to herein as "Plaintiffs' Properties" and are depicted generally for illustrative purposes only as "Plaintiffs' Properties" on Exhibit A to Plaintiffs' First Amended Complaint.

29. At all times relevant hereto, one or more of the Contaminants originating from the Site have entered and continue to enter one or more portions of Plaintiffs' Properties through various pathways, including but not limited to the Dry Run stream, resulting in the contamination of environmental media, including but not limited to soils and surface and groundwaters, at Plaintiffs' Properties (the "Contamination").

30. Several hundred of Plaintiffs' cattle have been and continue to be killed, injured, or otherwise adversely affected by the Contamination.



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31. The Contamination has adversely impacted and continues to adversely impact flora and fauna at Plaintiffs' Properties, and has impacted and continues to otherwise adversely impact the value of Plaintiffs' Properties.

32. The Contamination has made and continues to make Plaintiffs physically ill or otherwise physically damaged, and has caused and continues to cause associated emotional and/or mental stress, anxiety, and/or fear of current and/or future illnesses.

## FIRST COUNT
## (CERCLA)

33. Plaintiffs incorporate herein the allegations contained in Paragraphs 1 through 32 of this Complaint as if fully restated herein.

34. Plaintiffs and DuPont are each "persons" within the meaning of Section 101(21) of CERCLA, 42 U.S.C. § 9601(21).

35. The Site is and was at all times relevant hereto, a "facility" within the meaning of Section 101(9) of CERCLA, 42 U.S.C. § 9601(9).

36. At all times relevant hereto, and at the time of disposal and/or release and/or threatened release at the Site of one or more "hazardous substances," as that term is defined in Section 101(14) of CERCLA, 42 U.S.C. § 9601(14), DuPont owned and/or possessed hazardous substances and/or materials containing hazardous substances and by contract, agreement, or otherwise arranged for the treatment and/or disposal of such hazardous substances and/or materials containing hazardous substances at the Site, and is, therefore, a liable party within the meaning of Section 107(a)(3) of CERCLA, 42 U.S.C. § 9607(a)(3).



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37. At all times relevant hereto, and at the time of disposal and/or release and or threatened release at the Site of one or more "hazardous substances," as that term is defined in Section 101(14) of CERCLA, 42 U.S.C. § 9601(14), DuPont is and/or was at the time of disposal of such hazardous substances, an owner and/or operator of the Site and is, therefore, a liable party within the meaning of Section 107(a)(1) and/or (2) of CERCLA, 42 U.S.C. § 9607(a)(1) and/or (2)

38. At all times relevant hereto, and at the time of disposal and/or release and or threatened release of one or more "hazardous substances," as that term is defined in Section 101(14) of CERCLA, 42 U.S.C. § 9601(14), DuPont is and/or was a person who accepts or accepted one or more such hazardous substances for transport to the Site and is, therefore, a liable party within the meaning of Section 107(a)(4) of CERCLA, 42 U.S.C. § 9607(a)(4).

39. As a result of the actions of DuPont as referenced in Paragraphs 1 through 38 above, a "release" and/or "threatened release," as those terms are defined in Section 101(22) of CERCLA, 42 U.S.C. § 9601(22), of one or more hazardous substances has occurred, is continuing to occur, and all relevant times hereto was occurring at, under, and/or from the Site.

40. Plaintiffs have incurred and will continue to incur costs to investigate and/or otherwise respond to the release and/or threatened release of hazardous substances at, under, and/or from the Site, within the meaning of Section 101(25) of CERCLA, 42 U.S.C. § 9601(25).

41. Plaintiffs' actions taken in response to the release and/or threatened release of one or more hazardous substances at, under, and/or from the Site and the costs incurred incident thereto were necessary and undertaken in a manner consistent with the National Contingency Plan set forth at 40 C.F.R. Part 300.



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42. Plaintiffs have satisfied any and all conditions precedent to the undertaking of response actions, the incurrence of response costs, and to the recovery of such costs from DuPont under CERCLA.

43. Under Section 107(a) of CERCLA, 42 U.S.C. § 9607(a), DuPont is strictly liable to Plaintiffs, jointly and severally, for all response costs, including prejudgment interest, incurred and to be incurred by Plaintiffs in response to hazardous substances at, under, and/or from the Site

44. Under Section 113(f) of CERCLA, 42 U.S.C. § 9613(f), Plaintiffs are entitled to contribution from DuPont for DuPont's equitable share of all liability, response costs, and damages incurred and to be incurred by Plaintiffs in response to hazardous substances at, under, and/or from the Site.

## SECOND COUNT
## (RCRA)

45. Plaintiffs incorporate herein the allegations set forth in Paragraphs 1 through 44 of this Complaint as if fully restated herein.

46. Plaintiffs and DuPont are each "persons" within the meaning of Section 1004(15) of RCRA, 42 U.S.C. § 6903(15).

47. DuPont is a past or present generator, past or present transporter, and/or past or present owner and/or operator of a treatment, storage, and/or disposal facility, who has contributed and/or is contributing to the past and/or present handling, storage, treatment, transportation, and/or disposal of "solid waste" within the meaning of Section 1004(27) of RCRA, 42 U.S.C. § 6903(27), and/or "hazardous waste" within the meaning of Section 1004(5) of RCRA, 42 U.S.C. § 6903(5), at, under, and/or from the Site, which may present an imminent and substantial endangerment to health



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or the environment, within the meaning of Section 7002(a)(1)(B) of RCRA, 42 U.S.C. § 6972(a)(1)(B).

48. As a direct and proximate result of DuPont's acts and/or omissions contributing to the past and/or present handling, storage, treatment, transportation, and/or disposal of solid and or hazardous waste at, under, and/or from the Site, which may present an imminent and substantial endangerment to health or the environment, DuPont is liable for such endangerment under Section 7002(a)(1)(B) of RCRA, 42 U.S.C. § 6972(a)(1)(B).

49. DuPont does not dispute that Plaintiffs have complied with the notice requirements set forth in Section 7002(b)(2) of RCRA, 42 U.S.C. § 6972(b)(2), and at least ninety (90) days have past between the date of such notice and the date of filing this Second Amended Complaint.

50. Plaintiffs have complied with all conditions precedent to bringing their claim against DuPont under Section 7002(a)(1)(B) of RCRA, 42 U.S.C. § 6972(a)(1)(B), and no action has been taken which could prevent the filing of this RCRA claim under Section 7002(b)(2) of RCRA, 42 U.S.C. § 6972(b)(2).

51. Plaintiffs are entitled to an injunction against DuPont ordering DuPont to undertake and/or pay for any and all investigations, studies, monitoring, response, removal, and remedial actions and/or any other activities and/or actions necessary and/or required to respond to, abate, remediate and/or otherwise address any environmental contamination at, under, and/or from the Site and/or Plaintiffs' Properties that may present an imminent and substantial endangerment to health or the environment, and ordering DuPont to take all such other actions as this Court may deem necessary pursuant to Section 7002(a)(1)(B) of RCRA, 42 U.S.C. § 6972(a)(1)(B).



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## THIRD COUNT
## (DECLARATORY JUDGMENT)

52. Plaintiffs incorporate herein the allegations set forth in Paragraphs 1 through 51 of this Complaint as if fully restated herein.

53. An actual, substantial and justiciable controversy exists between Plaintiffs and DuPont regarding their respective rights and obligations for the response costs and/or damages that have been and will be incurred in connection with the release and/or threatened release of one or more hazardous substances at, under, and/or from the Site.

54. Plaintiffs seek a declaratory judgment against DuPont under Section 113(g)(2) of CERCLA, 42 U.S.C. §9613(g)(2), and the Federal Declaratory Judgment Act, 28 U.S.C. §§ 2201, 2202, holding DuPont strictly liable, jointly and severally, for all and/or DuPont's equitable share of the response costs incurred and to be incurred by Plaintiffs that will be binding in any subsequent action to recover further response costs or damages.

55. Pursuant to Section 113(g)(2) of CERCLA, 42 U.S.C. § 9613(g)(2), and 28 U.S.C. §§ 2201, 2202, Plaintiffs are entitled to a declaration from this Court that DuPont is strictly liable, jointly and severally, to Plaintiffs for all and/or DuPont's equitable share of the response costs and damages incurred or to be incurred by Plaintiffs.

## FOURTH COUNT
## (CLEAN WATER ACT)

56. Plaintiffs incorporate herein the allegations contained in Paragraphs 1 through 55 of this Complaint as of fully restated herein.

57. Plaintiffs are each "citizens" within the meaning of Section 505(g) of CWA 33, U.S.C § 1365(g).



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58. DuPont is a "person" within the meaning of Section 502(5) of CWA, 33 U.S.C. § 1362(5).

59. On April 14, 1998, the West Virginia Division of Environmental Protection ("WVDEP") renewed and reissued a National Pollutant Discharge Elimination System permit (Permit No. WV0076244) (the "Permit"), to DuPont for water discharges from the Site through authority of Section 1342 of CWA, 33 U.S.C. § 1342.

60. Monthly monitoring reports submitted to WVDEP by DuPont for at least the months of May, July, and August, 1998 confirm that DuPont has discharged and continues to discharge waste from Outlet 001 at the Site in amounts and concentrations that exceed applicable limits established under the Permit for at least iron, manganese, aluminum, and total suspended solids, thereby violating at least conditions A.1, B.1, C.1, C.3, C.12, D.1, F.2, F.3, F.4, G.7, and G.17 of the Permit.

61. Monthly monitoring reports submitted to WVDEP by DuPont for at least the months of May, July, and November of 1998 confirm that DuPont has discharged and continues to discharge waste from the Site into the nearby Dry Run stream in amounts and concentrations that exceed applicable limits established under the Permit for at least aluminum and iron, thereby violating at least conditions C.1, C.3, C.12, D.1, F.2, F.3, F.4, G.7, G.8, and G.17 of the Permit.

62. Monthly monitoring reports submitted to WVDEP by DuPont for at least the months of May, July, August, and November of 1998 confirm that DuPont is currently in violation of at least Paragraphs 1 and 3 of the Compliance Order set forth on Page 4 of the April 14, 1998, Findings and Order issued by WVDEP to DuPont as a result of violations of the CWA and Permit standards and limitations referenced above (Findings and Order No. 4015).



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63 DuPont's continuing and on-going violations referenced above constitute continuing and on-going violations of at least Sections 301(a) and 405(a) of the CWA, 33 U.S.C. §§ 1311(a), 1345(a), and Sections 22-11-6, 22-11-8, 22-11-16, and 22-11-18 of the West Virginia Water Pollution Control Act, W.Va. Code §§ 22-11-1 *et seq.* (collectively the "Law").

64. The Permit limits, Law, and compliance order provisions referenced above constitute "effluent standards or limitations" within the meaning of Section 505(f) of the CWA, 33 U.S.C. § 1365(f).

65. By virtue of DuPont's violations of the effluent standards or limitations referenced above, DuPont is in violation of "(A) an effluent standard or limitation under this chapter or (B) an order issued by the Administrator or a State with respect to such a standard or limitation" within the meaning of Section 505(a)(1) of the CWA, 33 U.S.C. § 1365(a)(1).

66. DuPont does not dispute that Plaintiffs have complied with the notice requirements set forth in Section 505(b)(1)(A) of the CWA, 33, U.S.C. § 1365(b)(1)(A), and at least sixty (60) days have passed between the date of such notice and the date of filing this Second Amended Complaint.

67. Plaintiffs have complied with all conditions precedent to bringing their claim against DuPont under Section 1365(a)(1) of the CWA, 33 U.S.C. § 1365(a)(1), and no action has been taken which could prevent the filing of this CWA claim under Section 505(b)(1)(B) of the CWA, 33 U.S.C. § 1365(b)(1)(B).

68. Plaintiffs are entitled to an injunction against DuPont ordering DuPont to comply with the effluent standards or limitations and compliance order referenced above, to pay appropriate civil penalties under Section 309(d) of the CWA, 33 U.S.C. § 1319(d), to pay all costs of litigation



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(including reasonable attorney and expert witness fees) and to take all such other action as the Court may deem necessary, pursuant to Section 505(a)(1) of the CWA, 33 U.S.C. § 1365(a)(1).

## FIFTH COUNT
## (WEST VIRGINIA HAZARDOUS WASTE MANAGEMENT ACT)

69. Plaintiffs incorporate herein the allegations contained in Paragraphs 1 through 67 of this Complaint as if fully restated herein.

70. Plaintiffs and DuPont are each "persons" within the meaning of Section 22-18-3(11) of the Waste Act, W.Va. Code §22-18-3(11).

71. As a result of the acts and/or omissions of DuPont as referenced in Paragraphs 1 through 70 above, DuPont has and continues to operate the Site in violation of applicable permit requirements set forth in Section 22-18-8 of the Waste Act, W.Va. Code §22-18-8.

72. As a result of the acts and/or omissions of DuPont as described in Paragraphs 1 through 71 above, Plaintiffs are entitled to bring a citizens suit against DuPont to obtain DuPont's compliance with applicable law, pursuant to Section 22-18-19(a) of the Waste Act, W.Va. Code §22-18-19(a).

73. Dupont does not dispute that Plaintiffs have complied with the notice requirements set forth in Section 22-18-19(a) of the Waste Act, W.Va. Code §22-18-19(a), and at least sixty (60) days have passed between the date of such notice and the date of filing this Second Amended Complaint.

74. Plaintiffs have complied with all conditions precedent to bringing their claim against DuPont under Section 22-18-19(a) of the Waste Act, W.Va. Code §22-18-19(a), and no action has



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been taken which could prevent the filing of this Waste Act claim under Section 22-18-19(a) of the Waste Act, W.Va. Code §22-18-19(a).

75. Plaintiffs are entitled to an injunction against DuPont, ordering DuPont to comply with the requirements set forth in Section 22-18-8 of the Waste Act, W.Va. Code §22-18-8, and ordering DuPont to take all such other action as this Court may deemed necessary, pursuant to Section 22-18-19(a) of the Waste Act, W.Va. Code §22-18-19(a).

## SIXTH COUNT
## (CONTRIBUTION)

76. Plaintiffs incorporate herein the allegations contained in Paragraphs 1 through 75 of this Complaint as if fully restated herein.

77. To the extent Plaintiffs have incurred damages and response costs and will continue to incur damages and response costs in the future in response to the release and/or threatened release of one or more solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants at, under, and/or from Site, DuPont is liable, jointly and severally, to Plaintiffs for contribution pursuant to Plaintiffs' right of contribution arising under Federal and State common law and applicable statutes

## SEVENTH COUNT
## (NEGLIGENCE)

78. Plaintiffs incorporate the allegations contained in Paragraphs 1 through 77 of this Complaint as if fully restated herein.

79. In connection with its ownership and/or operation of the Site, DuPont has had and continues to have a duty of ordinary and reasonable care to operate and manage the Site in such a way as to not create a nuisance or situation causing any injury or damage to human health or the environment.



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80. DuPont breached its duty of ordinary and reasonable care by negligently operating and/or managing the Site and/or conducting other operations and/or activities at the Site in such a manner as to cause, permit, and/or allow the release and/or threatened release of solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants at, under, and/or from the Site.

81. DuPont's negligent acts and/or omissions proximately caused and continue to proximately cause damage to Plaintiffs in the form of property damage and bodily injury, in addition to creating a situation harmful to human health and the environment, for which DuPont is liable.

## EIGHTH COUNT
## (NEGLIGENCE PER SE)

82. Plaintiffs incorporate herein the allegations contained in Paragraphs 1 through 81 of this Complaint as if fully restated herein.

83. By its acts and/or omissions resulting in the release and/or threatened release of one or more solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants at, under, and/or from the Site, DuPont violated and continues to violate one or more applicable State and/or Federal statutes, including but not limited to Sections 22-11-6, 22-11-8, 22-11-16, 22-11-18, 22-15-10, and 22-18-8 of the West Virginia Code, constituting negligence *per se*.

84. DuPont's violation of law proximately caused and continues to proximately cause damage to Plaintiffs in the form of property damage and bodily injury for which DuPont is liable.

## NINTH COUNT
## (NUISANCE)

85. Plaintiffs incorporate herein the allegations contained in Paragraphs 1 through 84 of this Complaint as if fully restated herein.



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86. DuPont's acts and/or omissions in operating and/or otherwise managing the Site caused and continue to cause a substantial and unreasonable interference with Plaintiffs' use and enjoyment of Plaintiffs' Properties and has materially diminished and continues to diminish the value of Plaintiffs' Properties.

87. DuPont's substantial and unreasonable interference with the use and enjoyment of Plaintiffs' Properties and continuing substantial and unreasonable interference with such use and enjoyment of Plaintiffs' Properties constitutes a continuing private nuisance.

88. DuPont's creation and continuing creation of a continuing private nuisance proximately caused and continues to proximately cause damage to Plaintiffs in the form of property damage and bodily injury, including diminution in the value of Plaintiffs' Properties, for which DuPont is liable.

## TENTH COUNT
## (TRESPASS)

89. Plaintiffs incorporate herein the allegations contained in Paragraphs 1 through 88 of this Complaint as if fully restated herein.

90. DuPont's acts and/or omissions resulting in the release and/or threatened release of one or more solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants at, under, and/or from the Site have resulted and continue to result in the release and/or threatened release of one or more such solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants at, under, onto, and/or into Plaintiffs' Properties.

91. The solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants present on Plaintiffs' Properties originating from the Site were at all relevant times hereto, and continue to be, the property of DuPont.



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92. The invasion and presence of DuPont's solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants at, under, onto, and/or into Plaintiffs' Properties was and continues to be without permission or authority from Plaintiffs or anyone who could grant such permission or authority.

93. The presence and continuing presence of DuPont's solid, toxic, and/or hazardous wastes, substances, pollutants, and/or contaminants at Plaintiffs' Properties constitutes a continuing trespass.

94. DuPont's past and continuing trespass upon Plaintiffs' Properties proximately caused and continues to proximately cause damage to Plaintiffs in the form of property damage and bodily injury, including diminution in the value of Plaintiffs' Properties, for which DuPont is liable.

## ELEVENTH COUNT
## (PUNITIVE DAMAGES)

95. Plaintiffs incorporate herein the allegations contained in paragraphs 1 through 94 of this Complaint as if fully restated herein.

96. DuPont's acts and/or omissions as described above were conducted with such malicious, wanton, willful, and/or reckless indifference to Plaintiffs' rights and/or flagrant disregard for the safety and/or property of Plaintiffs that DuPont is liable for punitive damages.

**WHEREFORE**, Plaintiffs hereby demand that this Court:

A. Enter a judgment against DuPont, pursuant to Section 107 of CERCLA, 42 U.S.C. § 9607, that DuPont is strictly liable, jointly and severally, to Plaintiffs for all response costs and damages that Plaintiffs have incurred and will incur in connection with the release and/or threatened



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release of hazardous substances at, under, and/or from the Site in an amount according to the proof at trial, plus attorneys' fees (to the extent recoverable), costs, and prejudgment interest;

B In the alternative, enter a judgment against DuPont, pursuant to Section 113(f) of CERCLA, 42 U.S.C. § 9613(f), that DuPont is strictly liable to Plaintiffs for DuPont's fair, equitable, and proportionate contribution for all response costs and damages that Plaintiffs have incurred and will incur in connection with the release and/or threatened release of hazardous substances at, under, and/or from the Site in an amount according to the proof at trial, plus attorneys' fees (to the extent recoverable) costs, and prejudgment interest;

C. Enter a declaratory judgment against DuPont, pursuant to Sections 107 and 113(g) of CERCLA, 42 U.S.C. § 9607, 9613(g), and/or 28 U.S.C. §§ 2201, 2202 that DuPont is strictly liable, jointly and severally, to Plaintiffs for all response costs and damages that Plaintiffs have incurred and will incur in connection with the release and/or threatened release of hazardous substances at, under, and/or from the Site in an amount according to the proof at trial, plus attorneys' fees (to the extent recoverable) costs, and prejudgment interest;

D. In the alternative, enter a declaratory judgment against DuPont, pursuant to Section 113(f) and (g) of CERCLA, 42 U.S.C. § 9613(f), (g), and/or 28 U.S.C. §§ 2201, 2202 that DuPont is strictly liable to Plaintiffs for DuPont's fair, equitable, and proportionate contribution for all response costs and damages that Plaintiffs have incurred and will incur in connection with the release and/or threatened release of hazardous substances at, under, or from the Site in an amount according to the proof at trial, plus attorneys' fees (to the extent recoverable), costs, and prejudgment interest;

E. Enter an injunction against DuPont, pursuant to Section 7002(a)(1)(B) of RCRA, 42 U.S.C. § 6972(a)(1)(B), ordering DuPont to undertake and/or pay for any and all investigations,



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studies, monitoring, response, removal, remedial, and/or any other actions necessary and/or otherwise required to respond to, abate, remediate, and/or otherwise address any environmental contamination at, under, and/or from the Site and/or Plaintiffs' Properties that may present an imminent and substantial endangerment to health or the environment, and ordering DuPont to pay all costs of litigation (including reasonable attorney and expert witness fees) and prejudgment interest.

F. Enter an injunction against DuPont, pursuant to Section 505(a)(1) of the CWA, 33 U.S.C. § 1365(a)(1), ordering DuPont to comply with the effluent standards or limitations and compliance orders that DuPont is violating, to pay appropriate civil penalties under Section 309(d) of the CWA, 33 U.S.C. § 1319(d), and ordering DuPont to pay all costs of litigation (including reasonable attorney and expert witness fees) and prejudgment interest;

G. Enter an injunction against DuPont, pursuant to Section 22-18-19(a) of the Waste Act, W.Va. Code § 22-18-19(a), ordering DuPont to comply with applicable provisions of Section 22-18-8 of the Waste Act, W.Va. Code § 22-18-8, and ordering DuPont to pay all costs of litigation (including reasonable attorney and expert witness fees) and prejudgment interest;

H. Enter a judgment against DuPont under Federal and/or State common law that DuPont is liable to Plaintiffs in contribution for DuPont's fair, equitable, and proportionate share of all liability, response costs, and damages incurred and to be incurred in connection with any environmental contamination at the Site;

I. Enter a judgment against DuPont that DuPont is liable to Plaintiffs for negligence in an amount to be determined at trial;

J. Enter a judgment against DuPont that DuPont is liable to Plaintiffs for negligence per se in an amount to be determined at trial;



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K. Enter a judgment against DuPont that DuPont is liable to Plaintiffs for nuisance in an amount to be determined at trial;

L. Enter a judgment against DuPont that DuPont is liable to Plaintiffs for trespass in an amount to be determined at trial;

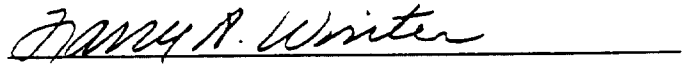
M. Enter a judgment against DuPont that DuPont is liable to Plaintiffs for punitive damages in an amount to be determined at trial; and

N. Enter a judgment against DuPont for Plaintiffs' costs, attorneys' fees, and interest and all such other and further relief as this Court may deem just and appropriate.

**JURY DEMAND**

Plaintiffs hereby demand trial by jury on all issues so triable.

WILBUR EARL TENNANT, ERWIN JACKSON TENNANT, JAMES DAVID TENNANT, DELLA MARIE TENNANT, and SANDRA K. TENNANT



Larry A. Winter
Carey, Hill, Scott, Winter & Johnson PLLC
P.O. Box 3884
Charleston, WV 25338
(304) 345-1234

Robert A. Bilott
J. Steven Justice
Taft, Stettinius & Hollister LLP
1800 Firstar Tower
425 Walnut Street
Cincinnati, OH 45202-3957
(513) 381-2838



000505

## CERTIFICATE OF SERVICE

I hereby certify that a filed, date-stamped copy of the foregoing **Second Amended Complaint**, which was filed with the Court on October 21, 1999, was served upon the following parties by regular U.S. mail, postage prepaid, this 21st day of October, 1999:

The Honorable Janet Reno
Attorney General of the United States
5111 Main Street Building
10th Street and Constitution Ave., N.W.
Washington, D.C. 20530

Carol Browner, Administrator
United States Environmental
Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

W. Michael McCabe, Regional Administrator
United States Environmental
Protection Agency, Region III
1650 Arch Street
Philadelphia, PA 19103-2029

Michael Miano, Director
WV Division of Environmental Protection
10 McJunkin Road
Nitro, WV 25143

Eric M. James, Esq.
Paula Durst Gillis, Esq.
Spilman, Thomas & Battle
P.O. Box 273
Charleston, WV 25321

Larry A. Winter

CAREY, HILL, SCOTT, WINTER & JOHNSON, LLC
1701 BANK ONE CENTER
707 VIRGINIA STREET, EAST
P.O. BOX 3884
CHARLESTON, WEST VIRGINIA 25338

MICHAEL W. CAREY
NANCY C. HILL
PHILLIP B. SCOTT (1963-1995)
LARRY A. WINTER
DAVID D. JOHNSON, III
PAMELA C. DEEM

GEORGE M. SCOTT
OF COUNSEL

PHONE (304) 345-1234
FAX (304) 342-1105
Email: chswj@chswj.com
Website: www.chswj.com
lwinter@chswj.com

Received - IS&H
Date OCT 25 99

October 21, 1999

Samuel L. Kay, Clerk
United States District Court
P.O. Box 1526
Parkersburg, WV 26101

Re: *Wilbur E. Tennant, et al. v. E.I. DuPont de Nemours & Co., Inc.*
Civil Action No. 6:99-0488

Dear Mr. Kay:

I enclose herewith a **Second Amended Complaint** for filing in the above-referenced civil action.

Thank you very much for your assistance in this matter. If you have any questions, please do not hesitate to contact me.

Very truly yours,

Larry A. Winter

Larry A. Winter

LAW:sms

Enclosure

cc: The Honorable Janet Reno
Carol Browner
W. Michael McCabe
Michael Miano
Eric M. James, Esq.
Paula Durst Gillis, Esq.

000507

TAFT, STETTINIUS & HOLLISTER LLP

1800 FIRSTAR TOWER
425 WALNUT STREET
CINCINNATI, OHIO 45202-3957
513-381-2838
FAX: 513-381-0205
www.taftlaw.com

COLUMBUS, OHIO OFFICE
TWELFTH FLOOR
21 EAST STATE STREET
COLUMBUS, OHIO 43215-4221
614-221-2838
FAX: 614-221-2007

NORTHERN KENTUCKY OFFICE
THOMAS MORE CENTRE
2670 CHANCELLOR DRIVE
COVINGTON, KENTUCKY 41017-3431
606-331-2838
513-381-2838
FAX 513-381-6613

ROBERT A. BILOTT
(513) 357-9638
bilott@taftlaw.com

CLEVELAND, OHIO OFFICE
SIXTH FLOOR
BOND COURT BUILDING
1300 EAST NINTH STREET
CLEVELAND, OHIO 44114-1503
216-241-2838
FAX 216-241-2837

August 18, 1999

**REGISTERED MAIL NO. R410009615**
**RETURN RECEIPT REQUESTED**

Statutory Agent
E.I DuPont de Nemours & Co., Inc.
1007 Market Street
Wilmington, DE 19898

Re: Notice Of Intent To Amend Complaint To Bring Citizens Suit Under Federal Clean Water Act In Connection With DuPont's Dry Run Landfill In Wood County, West Virginia: *Tennant, et al. v. E.I. DuPont de Nemours & Co., Inc.* (S.D. W.Va. Civil Action No. 6:99-0488

Dear Statutory Agent:

On behalf of our law firm's clients, Wilbur Earl Tennant (Route 3, Box 17, Washington, West Virginia 26181), Erwin Jackson Tennant (Route 3, Box 17A, Washington, West Virginia 26181), James David Tennant (Route 3, Box 372, Parkersburg, West Virginia 26101), Della Marie Tennant (Route 3, Box 372, Parkersburg, West Virginia 26101), and Sandra K. Tennant (Route 3, Box 17, Washington, West Virginia 26181) (collectively the "Tennants"), E.I. DuPont de Nemours & Co., Inc. ("DuPont") is hereby notified that the Tennants intend to amend their Complaint currently pending against DuPont in the referenced action in Federal Court in the Southern District of West Virginia to bring a claim against Dupont, pursuant to Section 505 of the Federal Clean Water Act, as amended ("CWA"), 33 U.S.C. §1365.

Section 505(a)(1) of the CWA permits the Tennants to commence a civil action against "any person . . . who is alleged to be in violation of (A) an effluent standard or limitation under this chapter or (B) an order issued by the Administrator or State with respect to such a standard or limitation." 33 U.S.C. §1365(a)(1). "Effluent standard or limitation" is defined under the CWA to include, among other things, "a permit or condition thereof issued under Section 1342 of this title, which is in effect under this chapter (including requirement applicable by reason of section 1323 of this title)." Id. at § 1365(f).

Based upon a review of the information provided to date by the State of West Virginia's Division of Environmental Protection ("WVDEP") in response to a Freedom of Information Act request, the Tennants have determined that DuPont is currently in violation of at least the following provisions of the NPDES permit reissued to DuPont by WVDEP on April 14, 1998, under authority of Section 1342 of the CWA (Permit No. WV0076244) with respect to discharges from Dupont's Dry Run Landfill in Wood County, West Virginia:

1. Monthly monitoring reports submitted to WVDEP by DuPont for at least the months of May, July, and August, 1998, confirm that DuPont has discharged and continues to discharge waste from Outlet 001 at the DuPont Dry Run Landfill in amounts and concentrations that exceed applicable permit limits for at least iron, manganese, aluminum, and total suspended solids, thereby violating at least conditions A.1, B.1, C.1, C.3, C.12, D.1, F.2, F.3, F.4, G.7, and G.17 of Permit No. WV0076244.

2. Monthly monitoring reports submitted to WVDEP by DuPont for at least the months of May, July, and November of 1998, confirm that DuPont has discharged and continues to discharge waste from the DuPont Dry Run Landfill into the nearby Dry Run stream in amounts and concentrations that exceed applicable permit limits for at least aluminum and iron, thereby violating at least conditions C.1, C.3, C.12, D.1, F.2, F.3, F.4, G.7, G.8, and G.17 of Permit No. WV0076244.

In addition, the Tennants have determined from the information produced to date by WVDEP that DuPont is currently in violation of at least Paragraphs 1 and 3 of the Compliance Order set forth on Page 4 of the April 14, 1998, Findings and Order issued by WVDEP to DuPont as a result of violation of the CWA standards or limitations referenced above (Findings and Order No. 4015). The continuing and on-going violations referenced herein also constitute continuing violations of at least Sections 301(a) and 405(a) of the CWA, 33 U.S.C. §§ 1311(a), 1345(a), and Sections 22-11-6, 22-11-8, 22-11-16, and 22-11-18 of the West Virginia Water Pollution Control Act, W.Va. Code §§ 22-11-1 et seq.

As indicated above, the continuing and on-going violations referenced in this notice are based upon the information that has actually been produced to date by WVDEP. We did not find within WVDEP's publicly-available files many of the monthly, quarterly, and annual monitoring reports that DuPont is required to submit to WVDEP under the terms of Permit No. WV0076244. Thus, the

Tennants reserve their right to allege and assert additional violations of the CWA, if additional information is obtained from WVDEP, U.S. EPA, or any other source indicating additional CWA violations. Nevertheless, DuPont is hereby notified that the Tennants intend to amend their Complaint currently pending against DuPont in the above-referenced litigation to include a citizens suit claim against DuPont, pursuant to Section 505(a)(1) of the CWA, within sixty (60) days after issuance of this notice to obtain relief at least for DuPont's violations of the CWA referenced above.

The following are receiving copies of this notice.

1. Carol Browner
Administrator
United States Environmental
Protection Agency
401 M Street, S.W.
Washington, DC 20460

**REGISTERED MAIL NO. R410009616**
**RETURN RECEIPT REQUESTED**

2. W. Michael McCabe
Regional Administrator
United States Environmental
Protection Agency, Region III
1650 Arch Street
Philadelphia, PA 19103-2029

**REGISTERED MAIL NO. R410009617**
**RETURN RECEIPT REQUESTED**

3. Michael Miano
Director
West Virginia Division of
Environmental Protection
10 McJunkin Road
Nitro, WV 25143

**REGISTERED MAIL NO. R410009620**
**RETURN RECEIPT REQUESTED**

4. Registered Agent for
E.I. Dupont de Nemours
& Co., Inc.
CT Corporation System
707 Virginia Street, East
Charleston, WV 25301

**REGISTERED MAIL NO. R410009621**
**RETURN RECEIPT REQUESTED**

000515

5. Paula Durst Gillis, Esq. **REGISTERED MAIL NO. R410009622**
Spilman Thomas & Battle, PLLC **RETURN RECEIPT REQUESTED**
Spilman Center
300 Kanawha Boulevard, East
P.O. Box 273
Charleston, WV 25321-0273

Very truly yours,

Robert A. Bilott

RAB:mdm
cc: Wilbur Earl Tennant
Erwin Jackson Tennant
James David Tennant
Della Marie Tennant
Sandra K. Tennant
Larry A. Winter, Esq.
J. Stephen Justice, Esq.

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2:107

DuPont
Washington Works
P. O. Box 1217
Parkersburg, WV 26102-1217

DUPONT

2 274-926-659

January 31, 2000

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Mr. William D. Brannon, Acting Chief
Office of Water Resources
Division of Environmental Protection
1201 Greenbrier Street
Charleston, WV 25311
Attention: Industrial Branch

PLANT FILE COPY
DO NOT REMOVE

Re: WV/NPDES Permit Number WV0076244

Dear Mr. Brannon:

Enclosed is the 1999 annual report summarizing the operations of our Dry Run industrial landfill.

If you have any questions or need additional information, please call me on 863-4271.

Very truly yours,

R L Ritchey

R. L. Ritchey
Sr. Environmental Control Consultant
Washington Works

Attachments
RLR/GW:vlw

cc: John G. Britvec 6.0
Office of Water Resources
Division of Environmental Protection
1304 Goose Run Road
Fairmont, WV 26554
Attention: Industrial Branch

EID076133

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Printed on Recycled Paper

I certify under penalty of law that this document and all attachments were prepared under my direction or supervision in accordance with a system designed to assure that qualified personnel properly gather and evaluate the information submitted. Based on my inquiry of the person or persons who manage the system, or those persons directly responsible for gathering the information, the information submitted is, to the best of my knowledge and belief, true, accurate, and complete. I am aware that there are significant penalties for submitting false information, including the possibility of fine and imprisonment for knowing violations.

R J Ritchey 1/31/00

R. L. Ritchey
Sr. Environmental Control Consultant
Washington Works

CF008244

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# 1999 DRY RUN LANDFILL OPERATIONAL REPORT

## Facility Users

All the waste disposed of in the Dry Run Landfill originates from E. I. du Pont and Company, Washington Works site and its auxiliaries.

## Landfill Shipments

A summary of the 1999 landfill shipments is attached.

## Surface and Groundwater Monitoring

A summary of the 1999 surface and groundwater monitoring activities is attached.

## Landfill Operation, Construction and Maintenance Activity Summary

Approximately 6.1 acres of leachate collection system were constructed during 1999 at the landfill. Project work included construction of:

- 267,000 square feet (6.1 acres) of leachate collection system. This included subgrade preparation, 1,670 linear feet of anchor trench, 16 ounce/square yard nonwoven geotextile, 60-mil textured HDPE geomembrane, HDPE drainage net, 10 ounce/square yard nonwoven geotextile, and 12-inch soil layer.

- 390 linear feet of 4-inch diameter leachate seep collection drain.
- 360 feet of 8-inch dimeter perforated HDPE pipe leachate collection drain.
- 1,430 linear feet of 4-inch diameter perforated HDPE pipe leachate collection drain.
- 1,090 linear feet of riprap ditch.
- 60 linear feet of 24-inch diameter corrugated metal pipe culvert.
- One gas vent.
- Revegetation of 12 acres of disturbed area.

An existing landslide was also repaired just south of the landfill area. The area was regraded and approximately 350 feet of riprap ditch reconstructed.

A collection system to enable truck transport of leachate generated by the landfill remained in service throughout the entire year. 2.9 million gallons of leachate were transported to Washington Works for treatment in 1999.

An updated topographic map of the landfill and cross section map showing the landfill space filled is attached.

CF008245

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Dry Run Landfill Remaining Capacity Calculations

Calculation of 1999 fill: Net Waste Fill Volume Method:

Waste volume hauled in 1999:
= 3,857 truck loads x 39.7 yd3/truck load (avg)
= 153,123 ~ 153,000 yd3

Average net waste compaction (daily cover allowance included ) estimated at 80% for 1999:

Total 1999 fill volume = 153,000 x 80% = 122,400 yd3

122,400 ~ 122,000

| | | | |
|---|---|---|---|
| Ravine #1 (existing cell) | Capacity remaining at the beginning of 1999 | = | 810,000 yd3 |
| | Capacity used in 1999 | = | 122,000 yd3 |
| | Capacity remaining at the beginning of 2000 | = | 688,000 yd3 |

Based on 122,000 yd3 per year net fill volume consumption:

688,000 yd3 / 122,000 = 5.6 years remaining life on existing cell

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1999 DRY RUN LANDFILL SHIPMENTS

| Material | Number of Shipments | Total Weight Tons | Total Weight Pounds |
|---|---|---|---|
| Coal Ash | 1294 | 18,604 | 37,207,060 |
| Paper, Cardboard, Wood Glass, Miscellaneous Solids, Metal, Nonasbestos Insulation | 1137 | 40,765 | 81,529,340 |
| Plastic Wastes (Polyamides, Acrylics, Polyacetal, Polyethylene-Terephthalate, Fluoropolymers) | 1326 | 7,615 | 15,230,320 |
| Dirt fill | 100 | 1249 | 2,498,620 |
| Total | 3,857 | 68,233 | 136,465,340 |

CF008247

1999 DRY RUN LANDFILL QUARTERLY LEACHATE ANALYSIS REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 07/01/99 | Second Quarter Leachate Monitoring Report |
| 10/01/99 | Third Quarter Leachate Monitoring Report |
| 1/14/00 | Fourth Quarter Leachate Monitoring Report |

1999 DRY RUN LANDFILL QUARTERLY POND UNDERDRAIN ANALYSIS REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 07/16/99 | Second Quarter Pond Underdrain Monitoring Report |
| 10/01/99 | Third Quarter Pond Underdrain Monitoring Report |
| 1/14/00 | Fourth Quarter Pond Underdrain Monitoring Report |

1999 DRY RUN LANDFILL QUARTERLY WELL MONITORING REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 04/13/99 | First Quarter Monitoring Well Report |
| 07/02/99 | Second Quarter Monitoring Well Report |
| 10/06/99 | Third Quarter Monitoring Well Report |
| 01/07/00 | Fourth Quarter Monitoring Well Report |

1999 DRY RUN LANDFILL SEMI-ANNUAL STORMWATER MONITORING REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| * | ---- |
| 1/21/00 | Second Half Stormwater Monitoring Report |

* No samples obtained for first half stormwater monitoring report because of lack of adequate rainfall.

CF008248

## 1999 DRY RUN LANDFILL MONTHLY DISCHARGE MONITORING REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 02/12/99 | January Discharge Monitoring Report |
| 03/08/99 | February Discharge Monitoring Report |
| 04/15/99 | March Discharge Monitoring Report |
| 05/11/99 | April Discharge Monitoring Report |
| 06/16/99 | May Discharge Monitoring Report |
| 07/15/99 | June Discharge Monitoring Report |
| 08/13/99 | July Discharge Monitoring Report |
| 09/16/99 | August Discharge Monitoring Report |
| 10/20/99 | September Discharge Monitoring Report |
| 11/08/99 | October Discharge Monitoring Report |
| 12/20/99 | November Discharge Monitoring Report |
| 01/20/00 | December Discharge Monitoring Report |

## 1999 DRY RUN LANDFILL ACUTE TOXICITY AND BENTHIC SURVEY REPORTS

(Not otherwise included with the reports listed above)

No benthic surveys performed during the year because of extremely dry weather.

CF008249

DRY RUN LANDFILL

1999

GROUNDWATER/SURFACE WATER MONITORING

SUMMARY OF FC-143* ANALYTICAL RESULTS

Sample Date: 07/22/99

| Sample Location | FC-143 Result (ug/L) |
|---|---|
| Well #6A | 0.096 |
| Well #12 | 0.134 |
| Well #12A | 0.081 |
| Well #12B | 5.4 |
| Well #13 | 3.6 |
| Well #13A | 0.070 |
| Well #14 | 2.5 |
| Well #15 | 0.263 |

Sample Date: 12/29/99

| Sample Location | FC-143 Result (ug/L) |
|---|---|
| Leachate | 34 |
| Outlet 001 | 66 |
| Stream Sampling Station No. 1 | 0.54 |
| Stream Sampling Station No. 2 | 87 |
| Property Line | 39 |

* FC-143 is ammonium perfluorooctanoate ionic surfactant.

All analyses performed by : Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425

EID076140

CF008250

ION LINE

# E.I. DUPONT DE NEMOURS & CO. INC.
# NON HAZARDOUS LANDFILL
# DRY RUN, WOOD COUNTY, W.VA.
# FILL VOLUME AVAILABLE

THIS DRAWING HAS BEEN FURNISHED BY E.I.DUPONT DE NEMOURS & CO. THE INFORMATION AND KNOW-HOW THEREON MAY NOT BE USED NOR THE DRAWING REPRODUCED WITHOUT THE WRITTEN PERMISSION OF DUPONT. ALL REPRODUCTIONS IN WHOLE OR IN PART, INCLUDING VENDOR'S SHOP DRAWINGS, SHALL BEAR OR REFER TO THIS STAMP.

| | | DATE |
|---|---|---|
| SCALE | 1" = 60' | |
| DRAWN BY | J.I. GASTON | 2-8-94 |
| UPDATED BY | JOE GASTON | 1-28-00 |
| CHECKED BY | | |
| APPROVED BY | | |

| ELEC. CODE CLASS | WWJ NO. | FAA NUMBER | PROJ. NO. | WASHINGTON WORKS WW M677 | DF AR |
|---|---|---|---|---|---|
| | | | | | |

EID076141

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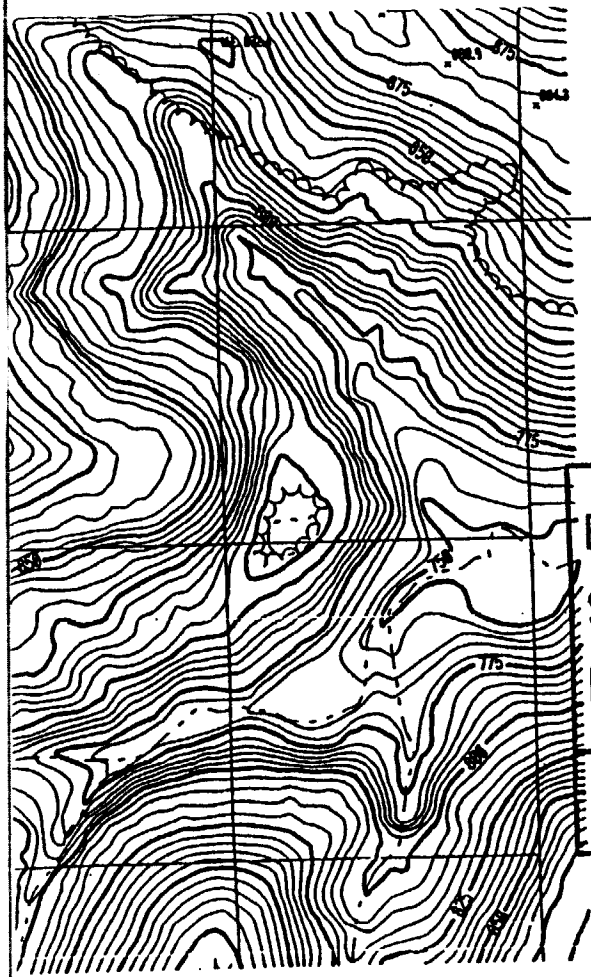



MONITOR WELLS

STREAM SAMPLE POINT

GRID: WEST VIRGINIA STATE PLANE COORDINATE

PHOTO DATE: 4-24-98

CF008252

# E.I. DUPONT DE NEMOURS & CO. INC.

# SOLID WASTE INDUSTRIAL LANDFILL

# DRY RUN, WOOD COUNTY, WV

THIS DRAWING HAS BEEN FURNISHED BY E.I.DUPONT DE NEMOURS & CO. THE INFORMATION AND IDEAS HEREON MAY NOT BE USED NOR THE DRAWING REPRODUCED WITHOUT THE WRITTEN PERMISSION OF DUPONT. ALL REPRODUCTIONS IN WHOLE OR IN PART, INCLUDING VENDOR'S SHOP DRAWINGS, SHALL BEAR OR REFER TO THIS STAMP.

| | | | |
|---|---|---|---|
| SCALE | 1" = 300' | DATE | |
| DRAWN BY | J. L. GASTON | | 10-14-98 |
| UPDATED BY | JOE GASTON | | 1-27-99 |
| CHECKED BY | | | |
| APPROVED BY | | | |

| ELEC. CODE CLASS | W&J NO. | FAA NUMBER | PROJ. NO. | WASHINGTON WORKS | BY |
|---|---|---|---|---|---|
| | | | | WW M760 | AR |

EID076142

1999 ANNUAL LANDFILL REPORT
WV/NPDES PERMIT NUMBER: WV0076244

(Letter, R.L. Ritchey to W.D. Brannon, dated 01/31/00)

BCC: A. V. Malinowski, Legal, D7078
M. A. Bradley, Spilman, Thomas & Battle
A. S. Hartten, BMP-27
M. L. Press, CH Works

In Turn:
J. H. Little
H. D. Ramsey, Jr.
J. E. Auger
W. H. Hopkins
R. Banerjee
D. C. Koelsch
R. L. Ritchey

L. K. Ireland/J. F. Moore/33.05.02.05
G. W. Klesel/J. S. DeBee

In Turn:
D. F. Altman
A. A. Crane
W. E. Lively
J. J. Mentink
M. M Pagels
G. Woytowich

RLR/DFA:vlw

2000 ANNUAL LANDFILL REPORT
WV/NPDES PERMIT NUMBER: WV0076244

(Letter, R. L. Ritchey to A. G. Turner, WV-DEP, OWR dated 1/26/01)

BCC: A. V. Malinowski, Legal, D7078
A. S. Hartten, BMP-27, 2160
M. L. Press, CH Works
M. A. Bradley, Spilman, Thomas, & Battle

In Turn:
P. J. Bossert, Jr.
H. D. Ramsey, Jr.
J. E. Auger
R. Banerjee
D. C. Koelsch
R. L. Ritchey

J. F. Moore/33.05.02.05

In Turn:
G. W. Klesel
J. S. DeBee

In Turn:
D. F. Altman
A. A. Crane
W. E. Lively
J. J. Mentink
M. M Pagels
G. Woytowich

dsw/2:107

EID114142

RECEIVED
JAN 29 2001

MAB

PLANT FILE COPY
DO NOT REMOVE

DuPont
Washington Works
P. O. Box 1217
Parkersburg, WV 26102-1217

DUPONT

January 26, 2001

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Ms. Allyn G. Turner, Chief  Z 403 787 170
Office of Water Resources
Division of Environmental Protection
1201 Greenbrier Street
Charleston, WV 25311
Attention: Industrial Branch

Re: WV/NPDES Permit Number WV0076244

Dear Ms. Turner:

Enclosed is the 2000 annual report summarizing the operations of our Dry Run industrial landfill.

If you have any questions or need additional information, please contact me at (304) 863-4271.

Sincerely,

R. L. Ritchey
Sr. Environmental Control Consultant
Washington Works

RLR/gw:dsw
Attachments
cc: John G. Britvec - 171
Office of Water Resources
Division of Environmental Protection
1304 Goose Run Road
Fairmont, WV 26554
Attention: Industrial Branch

EID114143

I certify under penalty of law that this document and all attachments were prepared under my direction or supervision in accordance with a system designed to assure that qualified personnel properly gather and evaluate the information submitted. Based on my inquiry of the person or persons who manage the system, or those persons directly responsible for gathering the information, the information submitted is, to the best of my knowledge and belief, true, accurate, and complete. I am aware that there are significant penalties for submitting false information, including the possibility of fine and imprisonment for knowing violations.

R L Ritchey 1/26/01

R. L. Ritchey
Sr. Environmental Control Consultant
Washington Works

# 2000 DRY RUN LANDFILL OPERATIONAL REPORT

Facility Users

All the waste disposed of in the Dry Run Landfill originates from E. I. DuPont, Washington Works and its auxiliaries.

Landfill Shipments

A summary of the 2000 landfill shipments is attached.

Surface and Groundwater Monitoring

A summary of the 2000 surface and groundwater monitoring activities is attached.

Landfill Operation, Construction and Maintenance Activity Summary

The leachate collection system constructed during 1999 remained in service throughout the entire year of 2000. Almost 3.3 million gallons of leachate were transported to Washington Works for treatment in 2000.

A cross section map showing the approximate landfill elevation at the end of the year 2000 and a topographic map of the landfill indicating the proposed final grading plan are attached.

Dry Run Landfill Remaining Capacity Calculations

Calculation of 2000 fill: Net Waste Fill Volume Method:

Waste volume hauled in 2000:
= 4,147 truck loads x 38.6 $yd^3$/truck load (avg.)
= 160,074 ~ 160,000 $yd^3$

Average net waste compaction (daily cover allowance included) estimated at 80% for 2000:

Total 2000 fill volume = 160,000 x 80% = 128,000 $yd^3$

| | | | |
|---|---|---|---|
| Ravine #1 (existing cell) | Capacity remaining at the beginning of 2000 | = | 688,000 $yd^3$ |
| | Capacity used in 2000 | = | 128,000 $yd^3$ |
| | Capacity remaining at the beginning of 2001 | = | 560,000$yd^3$ |

Based on 128,000 $yd^3$ per year net fill volume consumption:

560,000 $yd^3$ / 128,000 = 4.4 years remaining life on existing cell

## 2000 DRY RUN LANDFILL SHIPMENTS

| Material | Number of Shipments | Total Weight Tons | Total Weight Pounds |
|---|---|---|---|
| Coal Ash | 1347 | 19767.5 | 39,535,000 |
| Paper, Cardboard, Wood Glass, Miscellaneous Solids, Metal, Non-Asbestos Insulation | 1362 | 6178.78 | 12,357,560 |
| Plastic Wastes (Polyamides, Acrylics, Polyacetal, Polyethylene-Terephthalate, Fluoropolymers) | 1073 | 6339.32 | 12,678,640 |
| Dirt, Construction | 365 | 6502.38 | 13,004,760 |
| Total | 4147 | 38,787.98 | 77,575,960 |

## 2000 DRY RUN LANDFILL QUARTERLY LEACHATE ANALYSIS REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 03/29/00 | First Quarter Leachate Monitoring Report |
| 07/20/00 | Second Quarter Leachate Monitoring Report |
| 10/09/00 | Third Quarter Leachate Monitoring Report |
| 1/16/01 | Fourth Quarter Leachate Monitoring Report |

## 2000 DRY RUN LANDFILL QUARTERLY POND UNDERDRAIN ANALYSIS REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 04/05/00 | First Quarter Pond Underdrain Monitoring Report |
| 07/20/00 | Second Quarter Pond Underdrain Monitoring Report |
| 10/09/00 | Third Quarter Pond Underdrain Monitoring Report |
| 1/16/01 | Fourth Quarter Pond Underdrain Monitoring Report |

## 2000 DRY RUN LANDFILL QUARTERLY WELL MONITORING REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 04/12/00 | First Quarter Monitoring Well Report |
| 07/20/00 | Second Quarter Monitoring Well Report |
| 10/20/00 | Third Quarter Monitoring Well Report |
| 1/17/01 | Fourth Quarter Monitoring Well Report |

## 2000 DRY RUN LANDFILL SEMI-ANNUAL STORMWATER MONITORING REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 7/24/00 | First Half Stormwater Monitoring Report |
| 1/23/01 | Second Half Stormwater Monitoring Report |

## 2000 DRY RUN LANDFILL MONTHLY DISCHARGE MONITORING REPORT SUMMARY

| Date Submitted | Report |
|---|---|
| 02/03/00 | January Discharge Monitoring Report |
| 03/13/00 | February Discharge Monitoring Report |
| 04/14/00 | March Discharge Monitoring Report |
| 05/16/00 | April Discharge Monitoring Report |
| 06/15/00 | May Discharge Monitoring Report |
| 07/17/00 | June Discharge Monitoring Report |
| 08/15/00 | July Discharge Monitoring Report |
| 09/18/00 | August Discharge Monitoring Report |
| 10/09/00 | September Discharge Monitoring Report |
| 11/02/00 | October Discharge Monitoring Report |
| 12/15/00 | November Discharge Monitoring Report |
| 01/16/01 | December Discharge Monitoring Report |

## 2000 DRY RUN LANDFILL BENTHIC SURVEY REPORTS

| | |
|---|---|
| 06/27/00 | Benthic Macroinvertebrate Survey: May |
| 11/28/00 | Benthic Macroinvertebrate Survey: August |

# DRY RUN LANDFILL

## 2000

## GROUNDWATER/SURFACE WATER MONITORING

## SUMMARY OF FC-143* ANALYTICAL RESULTS

**Sample Date: 07/19/00**

| Sample Location | FC-143 Result(ug/L) |
|---|---|
| Well #12 | 0.160 |
| Well #12A | 0.128 |

**Sample Date: 07/20/00**

| | |
|---|---|
| Well #6A | 0.212 |
| Well #12B | <0.029 |
| Well #13 | 9.8 |
| Well #13A | 9.9 |
| Well #14 | 0.115 |
| Well #15 | 0.763 |

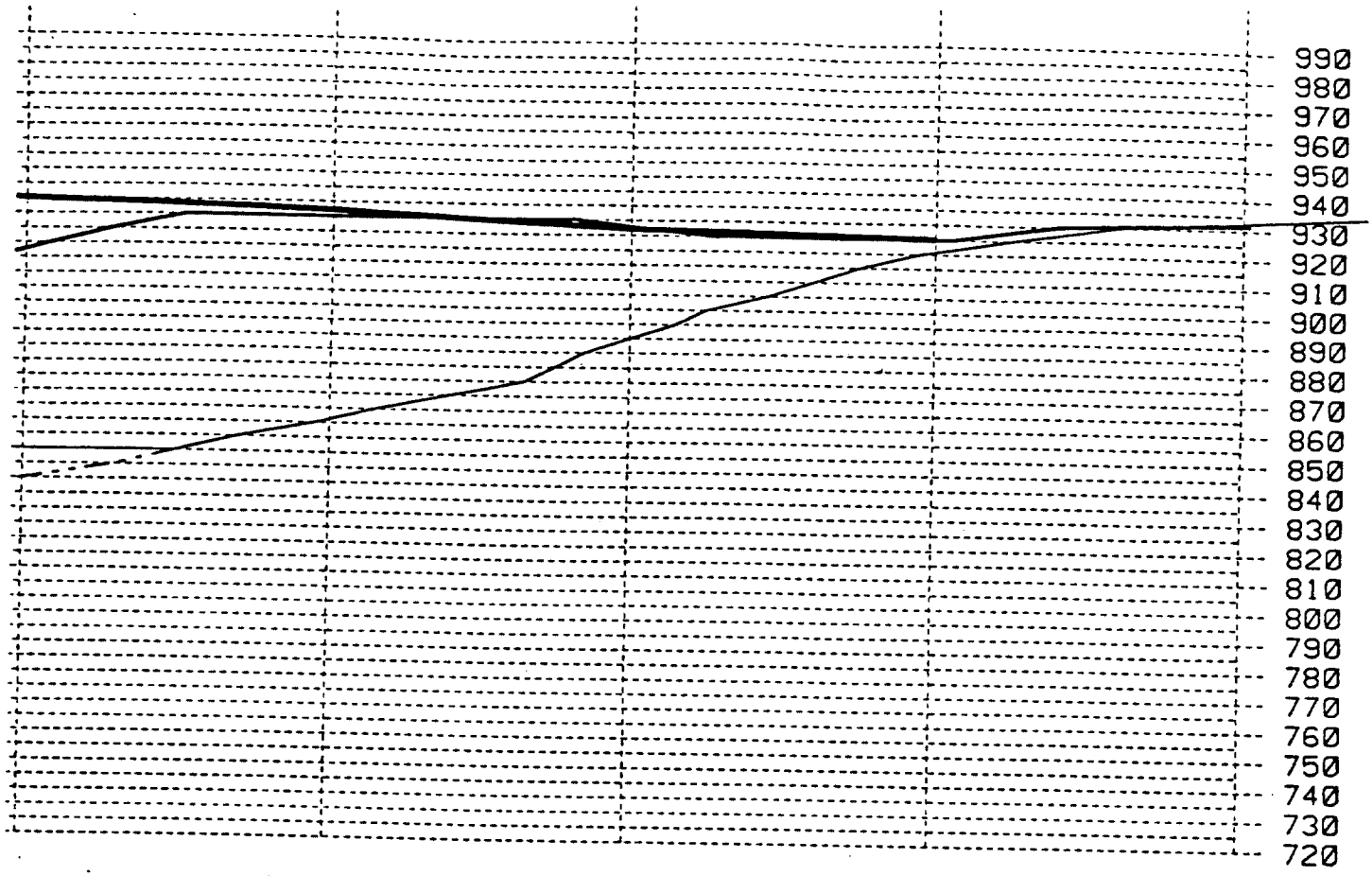
**Sample Date: 10/3/00**

| | |
|---|---|
| Leachate | 27.4 |
| Outlet 001 | 31.5 |
| Stream Sampling Station No. 1 | 0.758 |
| Stream Sampling Station No. 2 | 27.6 |
| Property Line | 10.3 |

*FC-143 is ammonium perfluorooctanoate ionic surfactant

All analyses performed by: Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425

EID114149




EID114150

PONT DE NEMOURS & CO. INC.
AZARDOUS LANDFILL
UN, WOOD COUNTY, W.VA.
VOLUME AVAILABLE

THIS DRAWING HAS BEEN FURNISHED BY E.I.DUPONT DE NEMOURS & CO. THE INFORMATION AND KNOW-HOW THEREON MAY NOT BE USED NOR THE DRAWING REPRODUCED WITHOUT THE WRITTEN PERMISSION OF DUPONT. ALL REPRODUCTIONS IN WHOLE OR IN PART, INCLUDING VENDOR'S SHOP DRAWINGS, SHALL BEAR OR REFER TO THIS STAMP.

| | | DATE |
|---|---|---|
| SCALE | 1" = 60' | |
| DRAWN BY | J. I. GASTON | 2-8-94 |
| UPDATED BY | JOE GASTON | 1-2-01 |
| CHECKED BY | | |
| APPROVED BY | | |

| OE CLASS | WWJ NO. | FAA NUMBER | PROJ. NO. | WASHINGTON WORKS WW M677 | OF AR |
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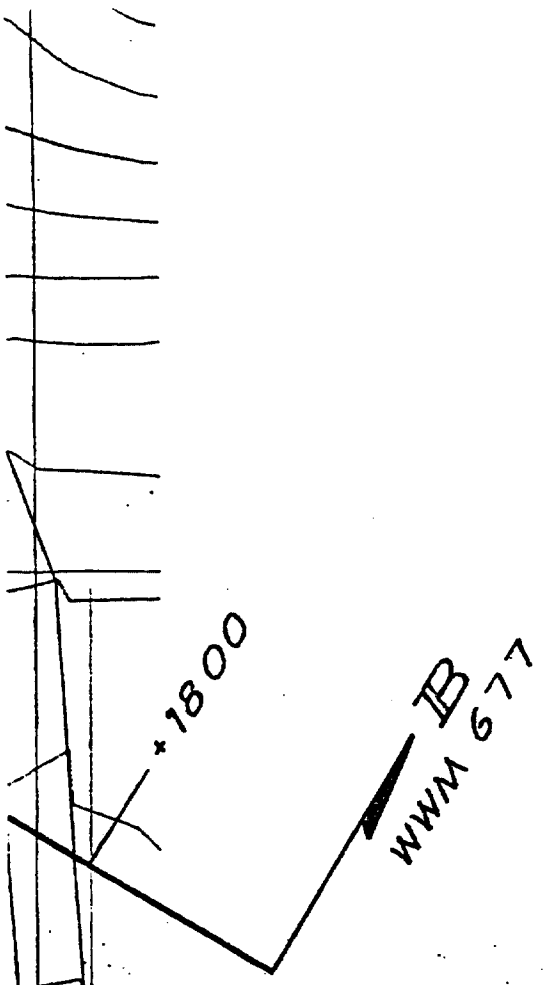


WORK THIS DRAWING
WITH: WWM 760
WWM 677
WBPF 7474

N 255705
E 1381617

WWM 789

JOE GASTON 2-29-00

GRAPHIC SCALE

60 30 0 60 120

1 INCH = 60 FEET

EID114151

PRELIMINARY

PRELIMINARY

Owner
E.I. DuPont de Nemours & Company
DUPONT WASHINGTON WORKS
PARKERSBURG, WEST VIRGINIA

Title
PROPOSED FINAL WASTE GRADING PLAN

1
Drawing No.

000934

Scene

# Lubeck PSD: Chemical in groundwater not a health threat

By ROGER ADKINS
Staff Writer

A little ammonium perfluorooctanoate with your water?

Also called C-8, PFOA, APFO and FC-143, the surfactant chemical found in groundwater consumed by Lubeck Public Service District customers does not pose a health threat, officials said.

Representatives of DuPont Washington Works and the Lubeck PSD said the presence of C-8 is not dangerous at current levels.

The levels of the chemical have been monitored for a long time, Jim Cox, district manager for the Lubeck PSD, said Tuesday.

The chemical has been detected in the drinking water at Washington Works at a level of 0.2 parts per billion and in Lubeck Public Service District wells at levels ranging from 0.1-0.5 parts per billion. These levels are lower than DuPont's own guideline of no greater than 1 part per billion.

Low levels of this chemical have been detected in groundwater wells in the Lubeck area for more than 10 years, Dawn Jackson, external affairs assistant at Washington Works, said its presence at current levels poses no danger to people or the environment.

"We've got many years of monitoring data from the 3M Company, from DuPont and from other labs that have done studies," Jackson said. "There isn't anything in the scientific data to indicate a need for concern. There is nothing to indicate that there is any human health effect."

However, one reason the community is concerned is because C-8 is a persistent chemical, meaning it remains in the human body and the ecosystem for a long time, Jackson said.

"It is a persistent chemical, meaning that it does stick around in the environment and in the human body," Jackson said. "It is not immediately metabolized and eliminated. That does separate it from the way other materials behave, because it does take longer for it to be eliminated from the body."

Washington Works uses C-8 in some of its manufacturing processes, Jackson said.

3-M, the company that manufactures C-8, said in a press release that this persistent compound, like other materials in the environment, can be detected broadly at extremely low levels in the environment and in people. All existing scientific knowledge indicates that the presence of these materials at such low levels does not pose a human health or environmental risk.

of C-8 in drinking water. The PSD, which serves 5,700 customers is given federal and state guidelines on what to monitor. It monitors C-8 levels even though it's not required to.

"If we find something else in the water, such as the C-8, we will monitor for that," Cox added. "So far all of our levels are fine. Certainly we will continue to monitor them to make sure they remain that way."

Jackson said that although there is no federal standard regulating the acceptable level of C-8, Washington Works sets strict guidelines to protect people and the environment.

"There is not a regulatory limit on it," Jackson said. "A lot of times, for what materials we use, we set our own standard."

Jackson said that DuPont's guidelines are so strict that the company will have no trouble adhering to federal or other regulatory standards, if established.

"One of the things we have told our own employees is that if a regulatory agency ever does set a level, we will be able to comply," Jackson said.

Recent attention has been drawn to C-8 because 3M decided to phase out an entire branch of chemistry, called perfluoroctanyl chemistry, and PFOA is one of the minor products of that line.

"They're phasing out a lot of different products," Jackson said. "This is just one of them."

When 3M decided to phase out perfluoroctanyl chemistry, local concerns were raised. Washington Works and the PSD, which continually work together to monitor chemical levels, decided to inform the community of the situation.

"Jim (Cox) got a couple of questions from people in the community last spring," Jackson said. "We started to look at it and find out what people's questions were. We made the decision together that the thing to do was send a letter to the customers of this district."

Although health issues were raised when 3M decided to phase out perfluoroctanyl chemistry, Washington Works officials remain confident in the company's products.

"They're confident that their products are safe," Jackson said. "We've been confident as well, but we've continued to monitor, just as a precaution. We've been using it at the plant for years and we've been monitoring it for 20 years or more."

Moreover, 3M, which has manufactured C-8 and other chemicals like it for approximately 40 years, continually studies its products, according to a statement on the company's Web site.

Jackson said that in the past, DuPont employees who worked with C-8 on a daily basis were exposed to it through skin absorption because it used to be available in powder form.

"Our employees were exposed to it at higher levels, at that time," she said. "The company then started to prepare it for us in a solution form and that reduced it (the exposure level) considerably.

"Through better control measures, better forms that we can purchase it in and better ways to handle it, over the years we've been able to reduce it at the plant," Jackson said. "Of course reducing levels at the plant also would contribute to reducing emissions into the environment."

Jackson said the current acceptable employee exposure limit for C-8 is 0.01 milligrams per cubic meter at an eight hour time-weighted average for skin absorption.

"Somebody could be exposed to that level constantly over an eight-hour work shift," Jackson said. "That's not the same thing as somebody in the community who is not exposed to it for eight hours at a time."

Standards in the workplace are different than they are in the community. Therefore, DuPont came up with an acceptable level for C-8's presence in drinking water.

"Through calculations done about exposure guidelines we've come up with keeping it below one part per billion," Jackson said

Jackson added that these are the guidelines that DuPont has set for the safety of its employees and the general public. However, they are prepared to comply with any regulatory standards that may be set in the future.

"At some point the EPA may come to us or Jim and tell us something different and we will comply with that," Jackson said.

The Parkersburg News

Founded in 1852

February 1, 2001

02/02/01 FRI 10:18 [TX/RX NO 5233]

# Officials: Chemical in groundwater no threat

By ROGER ADKINS
Staff Writer

Representatives of DuPont Washington Works and the Lubeck Public Service District said the presence of a surfactant chemical in groundwater consumed by Lubeck PSD customers does not pose a health threat.

Low levels of the chemical, called ammonium perfluorooctanoate (also called C-8, PFOA, APFO and FC-143), have been monitored by the Lubeck Public Service District for a long time, Jim Cox, district manager for the Lubeck PSD, said Tuesday.

The chemical has been detected in the drinking water at Washington Works at a level of 0.2 parts per billion and in Lubeck Public Service District wells at levels ranging from 0.1-0.5 parts per billion. These levels are lower than DuPont's own guideline of no greater than 1 part per billion.

Low levels of this chemical have been detected in groundwater wells in the Lubeck area for more than 10 years. Dawn Jackson, external affairs assistant at Washington Works, said its presence at current levels poses no danger to people or the environment.

"We've got many years of monitoring data from the 3M Company, from DuPont and from other labs that have done studies," Jackson said. "There isn't anything in the scientific data to indicate a need for concern. There is nothing to indicate that there is any human health effect."

However, one reason the community is concerned is because C-8 is a persistent chemical, meaning it remains in the human body and the ecosystem for a long time, Jackson said.

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Cox said there is no regulatory standard to limit the levels of C-8 in drinking water. The PSD, which serves 3,700 customers is given federal and state guidelines on what to monitor. It monitors C-8 levels even though it's not required to.

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■ See CHEMICAL, Page 5A

## Chemical

■ CONTINUED FROM PAGE 1A

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"At some point the EPA may come to us or Jim and tell us something different and we will comply with that," Jackson said.

*Contact Roger Adkins via e-mail*

DuPont Haskell Laboratory

January 25, 2001

Dr. Charles M. Auer, Director
U.S. Environmental Protection Agency
Office of Pollution Prevention and Toxics
Chemical Control Division
410 M Street NW Room 403
Arlington, D.C. 20460

Dear Dr. Auer

In my June 23, 2000 letter that transmitted DuPont's Voluntary Use and Exposure Information Profile (UEIP) for Ammonium Perfluorooctanoate (APFO, CAS#3825-26-1), I noted that, as part of the ongoing surveillance of workers potentially exposed to APFO, a series of blood samples were taken in year 2000 from workers and that DuPont would voluntarily submit a summary of the results when they became available.

The results of the blood serum tests are now available. A summary of this year's results for workers with identified APFO exposure potential is below.

| Year | # of Samples | Minimum Concentration (ppm) | Maximum Concentration (ppm) | Mean Concentration (ppm) |
|---|---|---|---|---|
| 2000 | 72 | 0.02 | 9.0 | 1.53 |

Note the following concerning the above data:

- Five samples, all from workers in one particular job, tested greater than 5.0 ppm. Among the jobs with potential APFO exposure, this job should have the least exposure potential. We are investigating the cause of these elevated results in this group of workers. Eliminating the five data points from these workers gives a maximum concentration of 4.9 ppm and a mean concentration of 1.16 ppm.
- Some employees not routinely working with APFO provided blood samples. APFO levels in this group of people are consistently less than 0.2 ppm.

RP002069

- Blood serum APFO concentration seems to be a function of length of time in assignments with potential APFO exposure. Due to variances in length of service among workers in assignments with potential APFO exposure, average values may be influenced not only by exposure potential but also by average length of service of the volunteer group.

Additional groundwater and surface water measurements have been reported and some older data has been located. These additional data are reported on pages 5 and 6 of a revised Voluntary [illegible]EIP. Please replace the previous submission with the attached version. Note that there is a public copy and a copy containing Confidential Business Information.

If you wish to discuss the information contained in this document, please contact Robert F. Pinchot at (302)999-4074 or e-mail at Robert.F.Pinchot@usa.dupont.com or me at (302)366-5250.

Very truly yours,

Gerald L. Kennedy

Gerald L. Kennedy
Director, Applied Toxicology and Health

Attachments

PUBLIC COPY

000529

EID114200

NP00207

# Voluntary Use and Exposure Information Profile
# Ammonium Perfluorooctanoate (APFO)
(revised January 2001)

*Note: The information contained in this document is submitted voluntarily and may be subject to future revision and/or modification.*

*CONFIDENTIAL BUSINESS INFORMATION REDACTED*

## I. CHEMICAL IDENTIFICATION

Chemical Name: Ammonium Perfluorooctanoate
CAS Number: 3825-26-1

## II. COMPANY IDENTIFICATION

**Company Name:** E. I. du Pont de Nemours and Company

Site Locations:

Site where APFO is used as a reaction aid:

Washington Works
Route 892
Washington, WV 26181

Sites where APFO containing products made at Washington Works are processed.

Parlin Plant
Cheesequake Road
Parlin, NJ 08859

Spruance Plant
5401 Jefferson Davis Hwy.
Richmond, VA 23234

Site which disposes of waste containing APFO:

Chambers Works
Rte. 130
Deepwater, NJ 08023

EID114201

**Technical Contact:** Robert F. Pinchot
(302) 999-4074
DuPont Fluoroproducts
Chestnut Run Plaza
Bldg. 711/2210
Centre Boulevard
Wilmington, DE 19805-0711

## III. DUPONT AND CUSTOMER ACTIVITIES

### Narrative Description of APFO Use

The block diagram on the back page titled "DuPont US APFO Balance" describes the processes discussed below and the estimated emissions associated with these activities.

DuPont uses APFO as a reaction aid in the production of polytetrafluoroethylene (PTFE) and tetrafluoroethylene (TFE) co-polymers. The process utilized at DuPont's Washington Works for making PTFE and co-polymers consists of polymerizing TFE (and other co-monomers if desired) in an aqueous media with a small amount of APFO to aid in the reaction.

Following the polymerization step, the polymer dispersion is either dried to remove water and APFO or concentrated (removing some of the APFO), stabilized and sold as an aqueous dispersion. The dried polymer contains very little, if any, APFO.

The APFO removed from the polymer is recovered for recycle, captured and destroyed off site in an incinerator, captured and sent to an offsite industrial landfill, and/or emitted to air or water at the Washington Works.

The stabilized polymer dispersions are sold by DuPont to industrial customers (both in the US and outside the US) for a variety of uses, internally transferred to the DuPont Spruance Plant for the production of Teflon® fibers and PTFE coated synthetic fibers, or internally transferred to the DuPont Parlin Plant for the production of Teflon® Finishes.

A small amount of non-hazardous waste polymer, water, APFO and other additives generated at Washington Works is treated in a wastewater treatment facility at DuPont's Chambers Works. This material is either emitted in the Chambers Works water discharge or captured on carbon and landfilled in a secure landfill.

The internal process at the DuPont Spruance Plant to produce Teflon® fibers involves, for most of the product, a "sintering" step in which the APFO contained in the product is destroyed by the following reaction:[1]

[1] P.J. Krusic, D.C. Roe, "Thermal Decomposition of C8 Fluorinated Surfactants and Related Materials Studied by High Temperature Gas-phase $^{19}F$ NMR. A New Alternative to Thermal Gravimetric Analysis", DuPont Internal Report.

$$CF_3(CF_2)_6COO^-NH_4^+ \rightarrow CF_3(CF_2)_5CF_2H + CO_2 + NH_3$$

This reaction goes to completion at 350°C and 0.2s residence time. A small amount of product processed at DuPont's Spruance plant does not get sintered and thus contains a small amount of residual APFO. These products are used for industrial pump, valve and compressor packing materials.

The process for making Teflon® finishes at the DuPont Parlin Plant involves a blending operation of fluoropolymer dispersions with other additives including solvents, binders, and pigments. The small amount of APFO emissions to water from this facility is due to waste generated during product changeovers. Some of the fluoropolymer dispersion is processed at contract facilities where the material is dried at temperatures >350°C thus destroying the APFO according to the reaction above. This dried material is then incorporated into finishes products.

The final product produced is then sold to applicators that apply the product to a substrate (such as cookware) via automated spraying or rollercoating. Emissions of APFO from these operations consist of overspray that is either captured on filters and landfilled or absorbed into water resulting in a water emission. Product that is applied to the substrate is then typically "sintered" at temperatures approaching 800°F resulting in the removal of the APFO from the substrate and subsequent destruction according to the reaction above.

Customers of dispersion products use the material for a variety of applications. However, most applications involve a "sintering" step where the APFO is destroyed. There are a small number of applications [ ] where the customer heats the dispersion products to temperatures that allow the APFO to sublime resulting in air emissions. There are also a small number of applications [ ] where the customer's product is not heated resulting in the APFO staying with the product. These applications include industrial packings, and industrial filter fabrics.

## IV. SITE RELEASE AND TRANSFER INFORMATION FOR TRI CHEMICALS

Not applicable- APFO is not listed on the TRI

## V. SITE RELEASE AND TRANSFER INFORMATION FOR NON-TRI CHEMICALS

A. On-site Air Releases

| | Estimated Total Annual Releases ( lbs.1999) | | | |
|---|---|---|---|---|
| | Washington Works | Parlin | Spruance | Chambers Works |
| Fugitive | Negligible | 0 | 0 | 0 |
| Stack (Point Source) | 24000 | 0 | 0 | 0 |



RP00024

Comments

Air emissions are estimated using engineering calculations and judgements and limited measurements of specific point sources conducted in the past.

B. On-site Water Releases

| | Estimated Total Annual Releases ( lbs.1999) | | | |
|---|---|---|---|---|
| | Washington Works | Parlin | Spruance | Chambers Works |
| Point Source | 55000 | 300 | 150 | 18,500 |

Comments

Water emissions are estimated using engineering calculations and judgements and limited measurements of specific sources conducted in the past.

Washington Works emissions occur for approximately 350 days/yr while the "other sites" emissions occur for 10-100 days/yr. Releases of APFO to the Ohio River from the DuPont Washington Works Plant were modeled using the Probabilistic Dilution Model (PDM Beta Version 4.0 Beta June 11, 1999, US EPA Office of Pollution Prevention and Toxics) and a constructed Microsoft® Excel spreadsheet model. APFO release data for 1996 were used in both modeling exercises.[2] The PDM indicated that APFO concentrations of 1.0 µAPFO/l would be exceeded about 50% of the time during the year. APFO concentrations of in the river would exceed 0.1 µg APFO/l 90% of the time during the year and 10 µg APFO/l about 2.2% of the time during the year.

Average annual APFO concentrations in the Ohio River calculated by using a Microsoft® Excel spreadsheet was 0.423 µg APFO/L. Modeled AFPO concentrations in the river ranged from a low of 0.199 µg APFO/L in March to a high of 0.965 µg APFO/l in September, which correspond to high and low river flows, respectively. Average Ohio River flows and volume data calculated from the US Geological Survey was collected at the Belleville Dam and used in the spreadsheet model. The Belleville Dam is on the Ohio River 13 miles downstream of the Washington Works Plant. This river flow data is the closest location downstream from the plant where this type of information is available.

In 1999, a drinking water sample obtained from GE plastics, Washington WV, immediately downstream on the Ohio River from DuPont Washington Works showed 0.552µg/l APFO.

RP002075

[2] W.R.Berti, "Modeling Releases of Ammonium Perfluorooctanoate into the Ohio River", DuPont Internal Report EMSE-054-00.

In addition, samples obtained in January 2000 from three different wells at the Lubeck Public Service District, downstream of Washington Works on the Ohio River, showed 0.8µg/l, 0.44µg/l and 0.313 µg/l APFO. Subsequent samples in April, May, and August 2000, showed a maximum of 0.59 µg/l and a minimum of 0.07µg/l.

C. On-Site Land Releases

Chambers Works treats APFO containing waste in a wastewater treatment system. Engineering calculations and judgements and limited measurements of specific sources in the past estimate that approximately 30% of the APFO in the wastewater treated is adsorbed on to a carbon media that is landfilled on site. These land releases are estimated to be 8,000 lb in 1999.

Prior operations have resulted in measurable APFO concentrations in three landfills operated by the Washington Works in West Virginia. At Letart[3] landfill, surface water measurements in 1999 and 2000 ytd range from 2.23µg/l to 3240µg/l, with an average of 1392µg/l. Groundwater measurements taken during the same time period at Letart landfill range from 60.3µg/l to 17400µg/l, with an average of 2537µg/l. At the "local landfill", the groundwater concentrations range from 0.046µg/l to 39µg/l with an average of 8.83µg/l. Surface water samples at the "local landfill" range from 0.54µg/l to 87µg/l, with an average of 18.5µg/l. At Dry Run landfill, there are limited measurements of groundwater and surface water, with the maximum concentration in groundwater of 15µg/l and the maximum concentration in the permitted outfall of 200µg/l. In 1990, samples of surface water were taken and showed concentrations as high as 1.6mg/l[4]. In 1992, samples in the "upper" and "lower" ponds near Dry Run landfill measured 220µg/l and 230µg/l, respectively. Samples taken in these locations at Dry Run landfill have since shown lower concentrations.

In 1999, a RCRA Facility Investigation was completed for Washington Works and was submitted to EPA Region III in June 1999[5]. The report contains data on groundwater concentrations of APFO at Washington Works.

[3] Maps of the landfill locations and specific monitoring locations and results are available upon request.
[4] The validity of these 1990 analyses cannot be verified since the method used for analysis, the sample technique, the chain of custody, the sample quality control/quality assurance procedures, and therefore the accuracy of the results are not known.
[5] Report was submitted to Martin. T. Kotsch, Remedial Program Manager, EPA Region III, Philadelphia.

NP000276

D. Transfers to Off-site Locations

Washington Works:

| | Estimated Total Annual Releases or Transfers (lb. 1999) |
|---|---|
| Incineration | 16000 |
| Wastewater treatment | 13400[6] |
| Underground Injection | 0 |
| Hazardous Waste Landfill | 2600 |
| Other landfill | 0 |
| Recycle or recovery | 25000 |

## VI. ON-SITE WORKPLACE EXPOSURE

A. Information on the Number of Employees Potentially Exposed

The tables below describe the number of workers that may be exposed to APFO in year 2000 during their normal work activities for each of the three sites where APFO is used or APFO containing product is processed.

Washington Works

| Hours/Day | Days/yr | | | |
|---|---|---|---|---|
| | <10 | 10-100 | 100-250 | >250 |
| <0.25 | | | | |
| 0.25-1 | | 242 | | |
| 1-8 | | | | |
| >8 | | | | |

Routine worker activities that have potential for exposure:

- Handling raw material APFO
- Handling raw dispersions containing APFO
- Maintenance of polymerization reaction systems
- Polymer dryer operation and maintenance
- Packout of PTFE and co-polymer dispersion products
- Operation and maintenance of APFO recovery systems

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[6] Transferred to Chambers Works facility (see section B and C above)

Parlin Plant

| Hours/Day | Days/yr | | | |
|---|---|---|---|---|
| | <10 | 10-100 | 100-250 | >250 |
| <0.25 | | | | |
| 0.25-1 | | | 18 | |
| 1-8 | | | | |
| >8 | | | | |

Routine worker activities that have potential for exposure:

- Handling of PTFE and Co-polymer dispersion products
- Operation and maintenance of blending facilities
- Packout of finished product

Note that at no time is the material handled at the Parlin Plant at an elevated temperature where the APFO could sublime. Therefore, there is little potential for exposure to airborne APFO at this facility. All exposure potential is through skin contact during handling of the polymer dispersion materials, all of which contain <1% APFO with most containing <0.25% APFO.

Spruance Plant

| Hours/Day | Days/yr | | | |
|---|---|---|---|---|
| | <10 | 10-100 | 100-250 | >250 |
| <0.25 | | | | |
| 0.25-1 | | <10 | | |
| 1-8 | | | | |
| >8 | | | | |

Routine worker activities that have potential for exposure:

- Handling of PTFE and Co-polymer dispersion products
- Operation and maintenance of fiber coating facilities
- Operation and maintenance of sintering rolls
- Packaging of non-sintered product.

Note that the PTFE and co-polymer dispersion products used at the Spruance site contain <0.9% APFO, with most containing approximately 0.3% APFO.

B. Information on the Exposure Levels of Washington Works Employees

Since most of the processing done in the US with APFO and APFO containing intermediates and products is done at Washington Works, DuPont's airborne industrial hygiene data is concentrated at that site. The limited measurements of airborne APFO concentrations at the other sites where APFO containing products are used have shown much lower levels (mostly non-detectable) levels of APFO. The data in the table below

reflect monitoring done over the last 5 years at Washington Works. The sample results are a combination of chemical operator and maintenance worker personal samples.

| Year | Sample Type | # of Samples | Minimum Concentration (mpb[7]) | Maximum Concentration (mpb) | Mean (mpb) | Standard Deviation |
|---|---|---|---|---|---|---|
| 1999 | Partial Shift (mostly 6-8 hours) | 100 | <0.01 | 0.58 | 0.061 | 0.151 |
| 1998 | | 83 | .001 | 0.78 | 0.103 | 0.145 |
| 1997 | | 100 | <0.01 | 2.4 | 0.146 | 0.378 |
| 1996 | | 73 | N/D | 0.29 | 0.055 | 0.069 |
| 1995 | | 32 | N/D | 0.16 | 0.067 | 0.063 |

Partial shift air samples are taken at the rate of 200 mL/min using a Tenax collection tube that has been pretreated with sodium hydroxide/ethylene glycol/methanol. The APFO is desorbed from the tubes using methanolic hydrogen chloride, which also serves as a derivatizing reagent, converting the APFO to its methyl ester. After workup, the methyl ester is quantified using a gas chromatograph equipped with an electron capture detector. The methyl ester of perfluorodecanoic acid is used as an internal standard, and at least three calibration samples are prepared to cover the concentration range of interest. Precision is estimated to be +/- 10% relative.

The data above show averages consistently below the AGCHI TLV of 0.01mg/m$^3$, with only a very few samples above the TLV. Where results are above or near to the TLV, the event is investigated and corrective action (additional personal protective equipment or engineering controls) to reduce the exposure levels is undertaken. Older data from the 1980's show higher levels of exposure. In the early 1990's, Washington Works switched from receiving the APFO as a powder to receiving it as an aqueous solution. This change was done to reduce the potential for exposure during handling of the dry powder. It should be noted that in the 1997 time period, the site was starting up new APFO recovery facilities. Operating and maintenance difficulties associated with the start-up of these facilities may have contributed to the higher levels of APFO in the personal samples during that year.

Task specific monitoring data and wipe monitoring data exist. However, these data are not indicative of employee exposure and are not presented here. These samples are taken to identify areas where additional exposure controls may be necessary.

Engineering controls to reduce exposure consist of the following:

- Reaction systems are closed systems with continuous ambient monitoring for monomer concentrations
- Ventilation systems are installed where airborne concentrations are significant
- The polymer dryers operate under negative pressure to contain APFO and other materials.
- Recovery systems are in place to reduce airborne emissions.

[7] mpb= moles per billion. 0.56mpb is equivalent to the ACGIH TLV of 0.01mg/m$^3$



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Personal protective equipment that workers regularly wear consist of the following:

- Safety shoes and side-shield safety glasses in all areas.
- Impervious gloves when handling APFO solutions or aqueous dispersion products.
- Chemical protective coveralls and goggles or face shields when the possibility of splashes of APFO containing solutions is present.
- Airline respirators or cartridge respirators where monitoring has shown to have high exposure potential.

At Washington Works, blood serum levels of APFO have been measured since 1981. Measurements of blood fluoride levels have been taken prior to 1981, but are of limited value in assessing exposure to APFO. A summary of results of employees with identified APFO exposure potential the 1995, 1989-90, 1985,and 1984 volunteer sampling events is in the table below. Due to significant job assignment movement during this period of time, analysis of trends of data are difficult. The data in the table below prior to 1995 are for employees included in the 1995 sampling data so that comparisons of relative levels of APFO in blood serum can be compared. The entire data set of blood concentrations is available upon request.

| Year | # of Samples | Minimum Concentration (ppm) | Maximum Concentration (ppm) | Mean Concentration (ppm) |
|---|---|---|---|---|
| 1995 | 73 | 0.12 | 4.5 | 1.57 |
| 1989-90 | 23 | 0.4 | 8.5 | 3.13 |
| 1985 | 21 | 0.06[8] | 18[9] | 2.44 |
| 1984 | 19 | 0.07[7] | 24[8] | 3.82 |

[8] This individual was working in a job that has APFO exposure potential at the time of the sample.
[9] This individual consistently has had the highest blood concentration of APFO since APFO specific samples were taken. This employee left an APFO exposure potential assignment in 1991. In 1995 this employee's blood serum level was 4.4ppm.

# DuPont US APFO Balance

1/23/01
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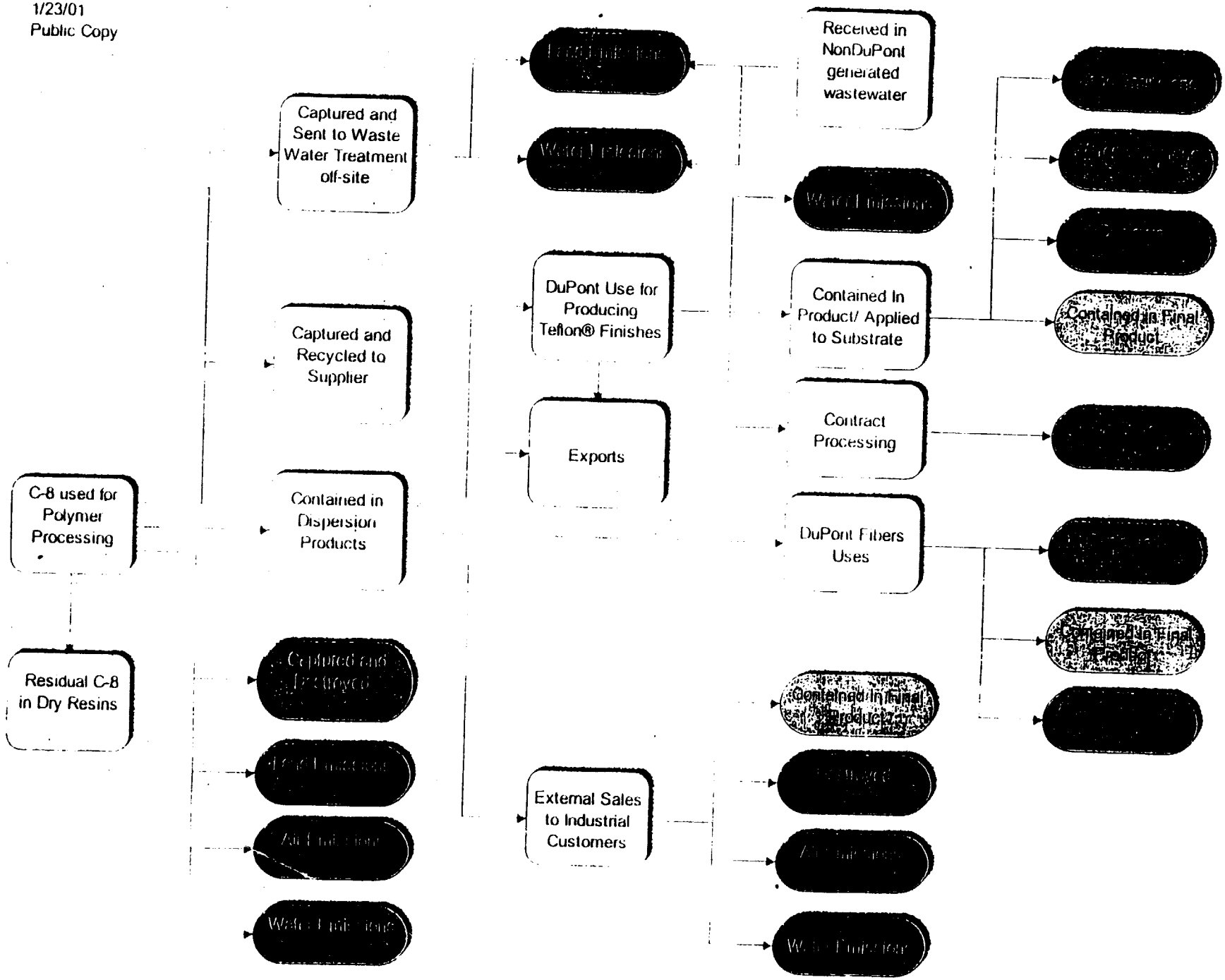


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