AR226-1327

RECEIVED
OPPT CBIC
03 JUN 20 PM 2:52

8EHQ-0603-00373

8EHQ-00-373

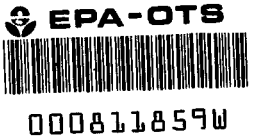
23 pp

Andrea V. Malinowski
Corporate Counsel
DuPont Legal D-7078
1007 Market Street
Wilmington, Delaware 19898
Phone: 302-774-6443
Fax: 302-774-4812
e-mail: **Andrea.V.Malinowski@usa.dupont.com**

Mr. Richard H. Hefter, Chief
High Production Volume Chemicals Branch
U.S. Environmental Protection Agency
Office of Pollution Prevention and Toxics
1200 Pennsylvania Avenue, NW
Washington, DC 20460-0001

Document Processing Center (7407M)
EPA East – Room 6428 Attn: Section 8(e)
U.S. Environmental Protection Agency
Office of Pollution Prevention and Toxics
1200 Pennsylvania Avenue, NW
Washington, DC 20460-0001

VIA HAND DELIVERY



June 20, 2003

CONTAIN NO CBI

Dear Mr. Hefter:

TSCA Section 8(e) PFOA Reporting Requirements

This letter is submitted in response to your May 22, 2003 letter to DuPont concerning perfluorooctanoic acid (PFOA) and to address certain unwarranted allegations made in an April 11, 2003 letter to Administrator Whitman concerning PFOA, which you reference in your letter. The April 11th letter claims that information contained in a one-page 1981 DuPont document listing PFOA blood sampling results and pregnancy status for eight employees should have been reported to the Environmental Protection Agency (EPA) as information supporting a conclusion of "substantial risk" under Section 8(e) of the Toxic Substances Control Act (TSCA), and makes the same unfounded claim for some drinking water sampling data on PFOA that DuPont collected in the mid-1980s. In your May 22nd letter, you asked that DuPont provide the company's perspective on the § 8(e)-reportability of this information.

As set forth in more detail below, the information reflected in the one-page 1981 document and the detection of minute traces (around 1-2 ppb) of PFOA in drinking water near the plant did not trigger reporting obligations under TSCA § 8(e). The information in the 1981 document does not in any way even suggest that PFOA is the cause of any adverse effect. Similarly, the data on water also does not in any way suggest PFOA is the cause of any adverse effect, as supported by the fact that the levels found are more than an order of magnitude below recently established drinking water safety standards. Presence alone, at the levels found for this substance, does not indicate substantial risk and therefore does not trigger reporting obligations under TSCA § 8(e).

RECEIVED
OPPT NCIC
2003 JUN 25 PM 2:14

267690

The 1981 One-page Document

- ***Background***

DuPont uses ammonium perfluorooctanoate (APFO), which converts to PFOA in solution, as a processing aid at its facility in Parkersburg, West Virginia. In over 50 years of use by DuPont and others, DuPont is not aware of any adverse human health effects that have been shown to be caused by PFOA.

In March 1981, the 3M Company (3M), which was the manufacturer of APFO (tradenamed FC-143) and DuPont's supplier, notified DuPont (and EPA) that in an oral rangefinder study in rats, designed to determine the maximum dosage rate that pregnant females could tolerate, and run in preparation for a full-scale teratology study, researchers observed what appeared to be treatment-related damage to the eye lenses of some rat pups.[1] Within a few months, however, the testing laboratory, 3M, and DuPont, as well as reviewers from The National Institute of Neurological Diseases and Blindness and the National Institutes of Health, all concluded that PFOA did not cause any developmental lens abnormalities in the fetal rat. This conclusion was based primarily on a determination that the lens damage reported in the 3M study was the result of artifacts from fixation and tissue sectioning for dissecting microscope observations, and was not treatment-related. EPA reviewers subsequently concurred with the conclusion that the lens effect was not caused by PFOA.[2] Shortly after the initial rangefinder test, four full-scale teratology studies using proper analysis technique found no evidence that PFOA created any teratogenic effects in rats or rabbits.

When DuPont first received word of the eye lens damage in 3M's preliminary study, as a precautionary measure while the chemical's potential was being studied, DuPont temporarily reassigned women of childbearing potential from the fluoropolymers unit to other locations where APFO exposure would be lower. DuPont permitted the women to return to the fluoropolymers unit shortly after the full-scale teratology studies showed no teratogenic effects.

At the time of the initial report from 3M, DuPont also provided its Parkersburg facility employees, and the public, with information on the situation.[3] DuPont offered blood testing for

---

[1] *See* Gortner, EG (1981) Oral Rangefinder Study of T-2998 CoC in Pregnant Rats. Riker Laboratories, Inc. Experiment No. 0680RR0018, February 1981. EPA has a copy of this study.

[2] See EPA Preliminary Risk Assessment of the Developmental Toxicity Associated with Exposure to Perfluorooctanoic Acid and its Salts (April 10, 2003), page 28 ("... the fetal lens finding ... was later determined to be an artifact of the free-hand sectioning technique and therefore was not considered to be treatment-related.")

[3] April 8, 1981 Wall Street Journal; April 8, 1981 New York Times (copies included as Attachment 1 and Attachment 2).

PFOA (referred to as "C8" in the one-page 1981 document) to employees at the plant site.[4] Those who volunteered for blood testing included eight women who worked at the plant and who either were pregnant or had given birth recently. DuPont staff also inquired about the outcomes of the employees' pregnancies. This informal collection of information, reflected in a half-page table and some handwritten margin notes, is the "study" that the April 11th letter alleges DuPont should have reported to EPA. In fact, this document does not contain information obtained as the result of any designed or controlled scientific study.

The table in the 1981 document indicates that five of the women gave birth to normal children. One woman was on pregnancy leave at the time the document was prepared. Although it is not noted on the document, this sixth child also was born normal. With respect to the pregnancies of the remaining two women, one had a child listed as being four months old and having one nostril and eye defect and the other had a child listed as being over two years old and having an "unconfirmed eye and tear duct defect." C-8 blood concentration for the four-month old child is listed as 0.012 ppm; there is no blood data listed for the two-year old child.

- ***8(e)-Reportability of Information in the 1981 One-page Document***

The information in the document concerning the child having a nostril and eye defect was not reportable under TSCA § 8(e), per the statutory language and guidance issued by EPA. TSCA § 8(e) states:

> "Any person who manufactures, processes, or distributes in commerce a chemical substance ... and who obtains information which reasonably supports the conclusion that such substance ... presents a substantial risk of injury to health or the environment shall immediately inform the Administrator of such information unless such person has actual knowledge that the Administrator has been adequately informed of such information."

15 U.S.C. § 2607(e). Thus, to trigger reporting obligations under TSCA section 8(e), an item of information must reasonably support the conclusion that the substance does present a substantial risk to health.

EPA states in the Agency's 1978 TSCA Section 8(e) Statement of Interpretation and Enforcement Policy (hereafter EPA's 1978 Policy Statement) that to "reasonably support" a conclusion of substantial risk, the information must do more than simply

---

[4] In the one-page document, blood values reported as ppm C-8 are actually ppm fluorine. PFOA was measured, but the value was converted to ppm fluorine for comparison with the results of total organic fluorine (another method that had been used to measure for PFOA). Estimated uncertainty in the measurement is plus or minus 10% standard deviation. The level of detection is 0.004 ppm; concentrations in that range cannot be well quantitated and were reported as less than 0.007 ppm F. PFOA concentration would be determined by dividing the ppm F value (listed as ppm C-8 in the document) by 0.668.

"suggest" that a chemical might be causing a substantial risk of some adverse effect; to the contrary, the information must "...reliably ascribe the effect to the chemical."[5] In short, to trigger a reporting obligation, there must be evidence of an adverse effect to health and the effect must be reliably ascribed to the substance in issue.

Underscoring this point, in response to a comment submitted on a 1977 draft of the policy statement, the EPA's 1978 Policy Statement provides the following, clarifying that a single incident of a birth defect is not reportable unless a chemical is "strongly implicated":

> "*Comment 12*: The reporting of 'any instance' of cancer, birth defects, etc., in humans is too broad and such information will be of little use; chemical workers, like the general population, develop cancers and other ailments of uncertain etiology.
>
> [EPA] *Response*: This [1978] policy statement clarifies that the reporting of single occurrences of human cancer or other serious effects will depend upon evidence strongly implicating one (or a few) chemicals."[6]

Nothing in the 1981 document suggests any link -- let alone "reliably ascribes" -- the child's defect, or any other adverse effect, to the presence of PFOA in the mother's blood. The mother and child's blood concentration samples were taken four months after the child was born. As such, the data do not provide any reliable information about the presence, level, or absence of PFOA in the blood during the pregnancy. Even if PFOA was present in the blood during pregnancy, presence of a substance alone does not support the conclusion that the substance caused or likely caused an adverse human health effect. As the Centers for Disease Control and Prevention (CDC) stated in its Second National Report on Human Exposure to Environmental Chemicals:

> "The measurement of an environmental chemical in a person's blood or urine does not by itself mean that the chemical causes disease. Advances in analytical methods allow us to measure low levels of environmental chemicals in people, but studies of varying exposure levels and health effects are needed to determine which blood or urine levels result in disease."

(CDC Report at p.2)[7] Further, although DuPont cannot discuss the details of confidential employee medical records, the child's eye defect did not involve lens damage, which is the only

---

[5] Statement of Interpretation and Enforcement Policy; Notification of Substantial Risk, 43 Fed. Reg. 11,110 (March 16, 1978) (emphasis added).

[6] 43 Fed. Reg. 11,114

[7] The CDC report can be viewed at www.cdc.gov/nceh/dls/report.

type of teratogenic effect ever even suggested (although as stated above, later proven to be incorrect) to have been caused by pre-natal exposure to PFOA. Since 1981 there have been additional full-scale teratology studies on PFOA, none of which produced any birth defects (structural or functional abnormalities) in the offspring of test animals.[8] Thus, in 1981 there was no data that would reliably ascribe the child's defects to PFOA, or strongly implicate PFOA as the cause, and no study run in the interim has created any such implication.

As noted previously, the 1981 one-page document also refers to a child born over two years previously as having unconfirmed "eye and tear duct" defects. Assuming that the defects did exist (and we have not been able to obtain any additional information on the unconfirmed defects), again the information contained in the 1981 document does not provide evidence that any such defects could have been reliably ascribed to PFOA exposure, either then or now. The mother's blood samples were taken more than two years after the pregnancy. The document indicates that the employee had worked in the fluoropolymer area for only one month before her pregnancy. Thus, here again, the data in the 1981 document -- listing blood levels more than two years after the pregnancy -- do not provide any reliable information about the presence, levels, or absence of PFOA in the employee's blood during her pregnancy. As such, from the information in this document, it cannot be reasonably concluded -- even assuming PFOA exposure during pregnancy -- that PFOA is "strongly implicated" as the cause of the unconfirmed defects.

The 1981 one-page document also indicates that one of the women, who gave birth a few weeks after the initial blood tests, permitted DuPont to test for PFOA concentration in the blood of the umbilical cord. PFOA was found to be present at a concentration level lower than that found in the mother's blood. In the course of investigating the basis for the information contained in this one-page document, DuPont recently found that the umbilical cord blood of the child of the fourth employee on the list was tested as well. The level reported was 0.43 ppm, again a lower level than that reported in the blood of the employee.

Nothing about this detection of the presence of PFOA in the umbilical cord blood at lower levels than in the mother's blood is unexpected or would reasonably support a conclusion of substantial risk. Indeed, teratology studies (such as were being run in 1981 on PFOA) are run on the assumption that the chemical in question will cross the placenta and will be present in the umbilical cord and come in contact with the developing fetus. The levels that DuPont detected in the umbilical cords simply confirm that there was no unexpected accumulation of PFOA at levels above those in the mother's blood. As explained above, and as supported by the CDC, presence alone does not indicate substantial risk of harm. Both children who had PFOA in their umbilical cords were born normal.

---

[8] Gortner, EG. (1981) Oral teratology study of T-2998CoC in rats. Safety Evaluation Laboratory and Riker Laboratories, Inc. Experiment Number: 0681TR0110, December 1981; Gortner, EG. (1982) Oral teratology study of T-3141CoC in rabbits. Safety Evaluation Laboratory and Riker Laboratories, Inc. Experiment number: 0681TB0398, February 1982; Staples, RE; Burgess, BA; Kerns, WD. (1984) The embryo-fetal toxicity and teratogenic potential of ammonium perfluorooctanoate (APFO) in the rat. Fundam. Appl. Toxicol. 4:429-440 (two studies -- inhalation and oral dose administration).

In summary, nothing reported in this 1981 document -- and no data generated to date -- even suggests, much less reasonably supports, a conclusion that PFOA presents a substantial risk of injury to human health. Therefore, the information did not and does not trigger any reporting obligations under TSCA § 8(e).

Drinking Water Monitoring Data – 8(e) Reportability

The April 11th letter and a related website report that your letter mentions also claim that DuPont should have reported to EPA under TSCA § 8(e) the presence of approximately 1 to 2 parts per billion (ppb) PFOA in drinking water from two communities in the area of the Parkersburg facility.[9] As noted above, however, TSCA § 8(e) requires reporting only if the information received reasonably supports the conclusion that the substance presents a substantial risk of injury to health or the environment. There is no evidence that the presence of those ppb levels of PFOA in drinking water, or any levels subsequently found in drinking water in that area, presents any risk of injury, let alone a substantial risk, which would be necessary to trigger reporting obligations. The detected levels fall far below levels that the governing regulatory authorities subsequently have set as screening levels for presence in drinking water. Further, DuPont alerted EPA and other state and local agencies to the presence of PFOA traces in groundwater and drinking water near the plant many years ago in reports filed under other regulatory programs.

EPA's recently published clarifications to the TSCA § 8(e) reporting guidance reflect the Agency's longstanding position that mere detection of the presence of a chemical in environmental media such as drinking water does not trigger reporting obligations; rather, reporting obligations are triggered only by a finding of levels that are high enough to support a conclusion of substantial risk.[10] To illustrate that point, EPA's clarification states:

> "From time to time EPA establishes concentrations of various substances in different media that trigger a regulatory response or establish levels that are presumed to present no risk to human health or the environment. For example, EPA establishes Maximum Contaminant Levels (MCLs) in drinking water, Ambient Water Quality Criteria for receiving bodies of water, and Reference Doses (RfDs) or Concentrations (RfCs). For the purposes of section 8(e), information about contamination found at or below these kinds of benchmarks would not be reportable. Conversely, information about contamination found at or above benchmarks that trigger regulatory requirements . . . is to be

---

[9] In Little Hocking, Ohio, PFOA was detected at the level of detection (0.6 ppb) in March 1984. Subsequent sampling in June 1984, March 1987 and May 1988 did not detect the presence of PFOA. In Lubeck, West Virginia, PFOA was detected in the range of 0.7 to 2.2 ppb from 1984 through 1989. DuPont reported the presence of PFOA in the Lubeck drinking water at ppb levels to EPA Region III in 1990 (see Attachment 5).

[10] 68 Fed. Reg. 33129 (June 3, 2003)

> considered for possible reporting, based on potential exposure to humans and/or non-human organisms and other relevant factors."[11]

In other words, to trigger reporting obligations, detected levels in drinking water must be sufficiently high to support a finding of substantial risk. Detection of levels below safe levels set by EPA are not reportable.

In all of DuPont's water monitoring tests, the levels measured in drinking water have never even remotely approached the 150 ppb screening level (the level requiring a response) set by the C8 Assessment of Toxicity Team (CATT), whose members include toxicologists from EPA Region III and EPA Headquarters and from the Agency for Toxic Disease Registry, as well as representatives from the West Virginia Departments of Environmental Protection ("WVDEP") and Health and Human Resources ("WVDHHR"). The highest levels DuPont has detected have been more than an order of magnitude lower than this 150 ppb screening level. In fact, the highest levels DuPont has detected do not even approach the far more conservative interim screening level of 14 ppb proposed by ENVIRON International Corporation and used by EPA Region III in a Safe Drinking Water Act consent order executed with DuPont, which governs the same geographic areas and the same drinking water supplies.[12] It is recognized that these screening levels have only recently been established, but they are indicative of what a team of experts believes is a safe level for PFOA in drinking water. Therefore, the screening levels set by these expert teams strongly support the conclusion that the levels DuPont found previously -- which are a tiny fraction of the level that the agencies have declared to be safe -- never were reportable under TSCA § 8(e), because they could not reasonably support any conclusion of a substantial risk of harm to human health.

Although the drinking water data did not trigger reporting requirements under TSCA § 8(e), on a number of occasions as far back as 1981, DuPont apprised EPA of the presence of PFOA in water (surface water, ground water and drinking water) in the area in question in reports filed pursuant to Clean Water Act or Resource Conservation and Recovery Act requirements. As examples, the following submissions are noted:

- DuPont's June 9, 1981 letter to West Virginia Division of Water Resources (WVDWR), with copy to EPA Region III, stating that PFOA (referred to by the 3M trade name "FC-143" in the letter) is present in an outfall that discharges to the Ohio River. Information on the toxicology of PFOA, including reference to the above-discussed 3M study, also was provided. This letter is enclosed as Attachment 3.

---

[11] 68 Fed. Reg. 33,138

[12] EPA Regions III and V Order on Consent Docket Numbers SDWA-03-2002-0019 and SDWA-05-2002-0002 (November 15, 2001)

- DuPont's June 5, 1985 letter to EPA Region III, in which it was reported that FC-143 had been detected in the groundwater aquifer. Excerpts from this letter are enclosed as Attachment 4.

- DuPont's February 9, 1990 letter to EPA Region III, in which it was reported that the Lubeck public supply wells contained ppb levels of C-8. Excerpts from this letter are enclosed as Attachment 5.

- DuPont's groundwater and drinking water monitoring data, filed on a regular basis with WVDEP and WVHHR, with copies to EPA Region III, since the signing of a consent order with WVDEP, effective November 14.

Finally, in assessing reportability of information concerning presence of a substance in the environment, it must be recognized that there was such considerable debate concerning the reportability of that type of information that EPA announced that the Agency was suspending the applicability of the portion of EPA's 1978 Policy Statement which outlined TSCA section 8(e) reportability of data on distribution of substances in environmental media.[13] EPA stated that until the Agency issued additional clarifying guidance, the regulated community was to focus on the statutory language of section 8(e) to determine reportability of information on environmental contamination, both for purposes of ongoing compliance with TSCA 8(e) reporting and for complying with the TSCA section 8(e) Compliance Audit Program (CAP).[14]

This additional clarifying guidance on the standards for reporting this type of information was, as referenced above, only just issued on June 3, 2003. DuPont had always intended to review the data on presence of PFOA in drinking water in light of the new EPA guidance as soon as EPA published it. Until that time, reportability was assessed by focusing on the statutory language of section 8(e). As noted above, EPA's new June 3, 2003 guidance confirms DuPont's conclusion that the drinking water testing information does not trigger any reporting obligation under section 8(e).

---

[13] 56 Fed. Reg. 28,458 (June 20, 1991)

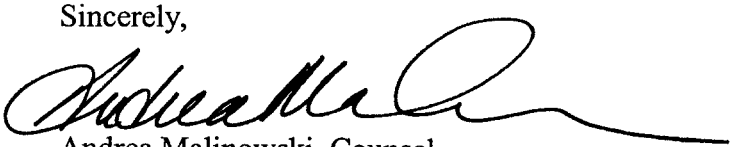
[14] The CAP was an industry-wide TSCA section 8(e) compliance audit program begun because of differing interpretations between EPA and industry regarding TSCA 8(e)-reporting requirements. At the outset, the CAP was meant to cover both health effects reporting (Phase 1) and environmental effects reporting, which was to include information on the release of substances to and detection in environmental media (Phase 2). EPA issued draft reporting guidance on environmental reporting and, after receiving extensive comments, decided that "...it is reasonable and equitable to enforce the final revised reporting guidance on a prospective basis only. Therefore, information on the release of chemical substances to and the detection of chemical substances in environmental media ... that predate the effective date of the guidance will not be the subject of an EPA TSCA Section 8(e) enforcement action." See Attachment 6, letter from EPA Director Toxics and Pesticides Enforcement Division to DuPont (May 15, 1996).

Your letter also requests copies of drinking water sampling data. As noted above, EPA already has received some data on the results of DuPont's drinking water sampling. However, DuPont will compile and forward drinking water data to you under separate cover.

Contact Information

We trust that the information provided in this letter will dispel any concerns about TSCA § 8(e) reporting that may have been triggered by the April 11th letter. Please ask someone from your staff to contact me promptly if EPA has any remaining questions regarding this issue.

Sincerely,



Andrea Malinowski, Counsel
E.I. du Pont de Nemours and Company

Attachments

ATTACHMENTS

Attachment No.	Title	No. of Pages
1	April 8, 1981 Wall Street Journal	1
2	April 8, 1981 New York Times	1
3	June 9, 1981 letter from DuPont to West Virginia Department of Water Resources, with copy to EPA Region III	1
4	Excerpt from June 5, 1985 DuPont letter to EPA Region III	3
5	Excerpt from February 9, 1990 DuPont letter to EPA Region III	2
6	May 15, 1996 letter from EPA to DuPont	5