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February 3, 2003

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Re: Notification Of Human Health Threat Arising From Releases Of PFOA/APFO/C-8 In West Virginia And Ohio

Ladies and Gentlemen:

Our law firm serves as class counsel for all persons whose drinking water is or has been contaminated with ammonium perfluorooctanoate (PFOA/APFO/C-8) attributable to releases from E.I. duPont de Nemours and Company's ("DuPont's") Washington Works Plant in Wood County, West Virginia. Based upon data collected to date by both DuPont and the State of West Virginia's Department for Environmental Protection ("WVDEP"), we understand that there are currently tens of thousands of individuals living in both Ohio and West Virginia whose drinking water is contaminated with C-8 from DuPont's Washington Works Plant. We write on behalf of that Class to bring to your attention what we believe may be an imminent and substantial threat to the health of the Class members arising from their past, current, and on-going exposure to such C-8 in their air, drinking water, and possibly other media.

We understand that USEPA requested in September of 2002 a priority review of the human health risks associated with C-8 exposure, after reviewing the results of recent C-8 toxicology studies and human blood data from industry. More specifically, we understand that, upon review of blood sampling data indicating the presence of PFOA in blood of the general U.S. population at an average level of approximately 3 parts per billion and the results of a 6-month primate study and 2-generation rat reproduction study recently completed by industry, USEPA concluded that "toxicological studies in rodents and primates have shown that exposure to PFOA can result in a variety of effects including developmental/reproductive toxicity, liver toxicity and cancer," that "blood monitoring data suggested widespread exposure to the general population, albeit at low levels," and that "human blood monitoring data, coupled with what is currently understood about the hazards of PFOA is sufficient from EPA's perspective to generate concern about potential PFOA risks." In response, USEPA's OPPT ordered a "priority review . . . to determine the significance of the risks presented by PFOA and its salts" because of the "possibility that PFOA might meet the criteria for action under Section 4(f) of the Toxic Substances Control Act." (*Id.*) Section 4(f) of TSCA is the section that authorizes USEPA to undertake a risk review when the Agency receives information "which indicates . . . that there may be a reasonable basis to conclude that a chemical or substance or mixture presents or will present a significant risk of serious or widespread harm to human beings from cancer, gene mutations, or birth defects." 15 U.S.C. 2603(f). In addition, we understand that the Science Advisory Board is to perform a peer review of a draft risk assessment for PFOA prepared but not yet released by USEPA. Thus, we understand that USEPA's interpretation of the existing human exposure and toxicological data for PFOA was sufficient to raise concerns with respect to the potential health risks posed to the general U.S. population that may have PFOA in their blood at an average level of approximately 3 parts per billion.

With these health risk issues in mind, we believe that we have a responsibility to bring to your attention information we reviewed from DuPont relating to the level of PFOA exposure in the communities surrounding DuPont's Washington Works Plant that, according to DuPont's

data, may be approximately *a thousand times higher* than the general population exposure levels that initially triggered USEPA's concerns.

More specifically, we understand that DuPont performed internal PFOA kinetics/pharmicokinetics research that resulted in the development of a model during the Summer of 2000 to relate C-8 exposure levels to human blood levels. (*See* Exhibit A (DuPont Abstract # 19) [1/] DuPont continued working with that data to generate by October of 2001, a draft, simple "compartmental model" to relate C-8 exposures through air and drinking water to estimates of perfluorooctanoate (PFO) concentrations in human blood. (*See* Exhibit B) The creation of that model enabled DuPont to develop a table from which estimated PFO levels in human blood can be determined from known C-8 exposure levels in air and drinking water. (*See id.* (Table 1))

Sampling and modeling performed to date by both DuPont and WVDEP confirm levels of C-8 over 4 ppb in the drinking water provided to at least one of the communities (with approximately 12,000 water customers) adjacent to DuPont's Washington Works Plant, and confirm annual average levels of C-8 in community air (based on DuPont's Year 2000 emissions) as high as 1 ug/m3. (Exhibits C and D) The Ohio Environmental Protection Agency recently confirmed that its own air modeling and analysis revealed predicted eight-hour ambient concentration of C-8 "well above the ACGIH health based work place standard [of 10 ug/m3] at multiple receptors in Ohio" that "raise our level of concern with regards to short term exposures to workers and sensitive populations." (Exhibit E) According to DuPont's PFOA blood model and related exposure table, such exposure levels would be expected to result in concentrations of PFO in the blood of those exposed at several *thousand* parts per billion. In fact, we understand that, according to DuPont's own PFOA blood model, some of the communities receiving the highest levels of C-8 in their drinking water and air would be expected to have levels of C-8 in their blood in concentrations that are approximately *double* the average levels of C-8 found in the DuPont employees working directly with C-8 at the Washington Works Plant (reported by DuPont to be an average of 1530 ppb in 2000). (*See* Exhibit F) As indicated in the attached report from David Gray, Ph.D., of Sciences International, such levels of exposure are of particular concern, given that the C-8 exposure in these communities is estimated by DuPont's model to result in C-8 blood concentrations well above the C-8 blood levels that caused renal and developmental toxicity in female rats. (*See* Exhibit I) Moreover, these affected local

---

[1/] As a defendant in a class action lawsuit pending against DuPont in a West Virginia State Court, DuPont has the right to mark documents produced in that case as "confidential" under the terms of a Stipulated Protective Order that allows parties to limit distribution of documents, if they qualify as documents for which "confidential business information" (CBI), protection would be available under, among other provisions, 40 C.F.R. Part 2, Subpart B. DuPont did not mark the attached documents from its files as "confidential" under the terms of the Stipulated Protective Order.

communities include numerous sensitive sub-populations, such as the elderly, pregnant women, and infants that are not found among the DuPont "healthy," predominantly adult male workers.

To put this exposure data into perspective, it should be noted that, as far back as 1987, DuPont told its own employees at the Washington Works plant that an "acceptable level" of C-8 in blood was 500 ppb and that employees with more than 50% of this "acceptable level" (250 ppb) should be removed from further exposure. (See Exhibits G and H) Although DuPont's modeling indicates that the local community is likely to have C-8 in their blood at levels more than *8-16 times higher* than DuPont's 250 ppb "acceptable" level, we are unaware of any steps having been taken by DuPont to remove any members of the community from their exposure to the Washington Work's ongoing C-8 emissions. Moreover, although DuPont's Washington Works employees exposed to C-8 are provided special personal protective equipment, bottled water at the Plant, and routine medical surveillance, DuPont does not provide such benefits to the communities surrounding its Washington Works Plant. In addition, DuPont continues to discharge C-8 into the environment from its Washington Works Plant. As of today's date, we are unaware of any steps having been taken by WVDEP to even acknowledge excessive exposures among the local communities, let alone address such exposures.

In summary, DuPont's own modeling data, including kinetic models prepared by DuPont several years ago, indicate that residents living in the communities surrounding DuPont's Washington Works Plant in Wood County, West Virginia have been and are continuing to be exposed to levels of PFOA that are possibly *a thousand times higher* than the low levels of exposure among the general U.S. population that triggered USEPA's recent "priority" review of the human health risks associated with such exposures. There is no indication that WVDEP intends to address this issue. We request, therefore, that USEPA and Ohio EPA take those steps necessary to respond to this situation and that ATSDR consider the exposure of the specific communities actually impacted by DuPont's C-8 emissions (as opposed to broad County-wide statistics) in connection with any investigation of the impact of C-8 exposures in the communities surrounding DuPont's Washington Works Plant. Thank you.

Very truly yours,

Robert A. Bilott

RAB/mdm
Enclosures (Exhibits A-I)
cc: R. Edison Hill, Esq. (w/o encls.)
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Gerald J. Rapien, Esq. (w/o encls.)
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Mary Dominiak, (USEPA, OPPT) (for inclusion in AR-226) (w/ encls.)
Michael Baker (Ohio EPA, DDAGW) (w/ encls.)

From: Paul M Hinderliter on 08/29/2000 10:17 AM

To: Gary W Jepson/AE/DuPont@DuPont
cc:
Subject:


HInderliter SOT 2001.d


Jepson SOT 2001.do

EID277489

DEVELOPMENT OF A BIOLOGICALLY BASED MODEL TO DESCRIBE PERFLUOROOCTANOIC ACID (PFOA) KINETICS. P M Hinderliter, and G W Jepson, Haskell Laboratory, DuPont, Newark, DE

Perfluorooctanoic acid is a fluoroorganic surfactant used in the production of a variety of fluoropolymers. Questions about general fluororganic surfactant biodistribution and persistence have been generated as a result of a recent decision to remove a widely used fluoroorganic fabric treatment from the marketplace. In order to better understand the biodistribution and biopersistence potential of PFOA in humans, a biologically based kinetic (BBK) human model was developed. A physiologically-based pharmacokinetic model (PBPK) using Advanced Continuous Simulation Language (ACSL) was first developed and validated using available rodent and primate data. This model was then scaled up to humans. Both dermal and oral routes of exposure were included in the model as these routes account for nearly all human exposure to PFOA. The resulting BBK model for PFOA had explicit descriptions of liver, gut, lung, fat, kidney, and skin as well as general descriptions for compartments with kinetically similar behavior. Binding equations were included to account for the interaction between PFOA and tissue and plasma proteins. The resulting model successfully described the plasma and tissue kinetics of PFOA in rats exposed via repeated oral dosing. Additionally, comparison of human model simulations with human PFOA blood data demonstrated the utility of the BBK model in assessing the behavior of PFOA in humans exposed to PFOA.

COMPARISON OF PERFLUOROOCTANOIC ACID (PFOA) AND PERFLUOROOCTANYL SULFONATE (PFOS) BLOOD KINETICS IN RATS FOLLOWING REPEATED ORAL DOSING. G W Jepson, P M Hinderliter, J Stadler, C Finlay, Haskell Laboratory, DuPont, Newark, DE

Perfluorooctanoic acid is a fluoroorganic surfactant that has been used for decades to produce a variety of fluoropolymers. Considerable focus has been placed on determining the biopersistence potential PFOA because of a recent decision to remove another fluoroorganic compound, perfluorooctanyl sulfonate, from the marketplace. In order to compare the biopersistence potential of PFOA and PFOS, male rats were dosed once per day for 10 days and followed for 94 days. Blood was drawn and analyzed for total fluoride on days 1, 5, 10, 13, 24, 52 and 94. Noncompartmental analysis of the blood data was conducted using a WinNonlin mathematical software package. There was considerable difference in PFOA and PFOS blood kinetics as determined by peak blood concentrations and total exposure as determined by area under the curve (AUC) for doses normalized to 0.1 mmoles/Kg body weight. The maximum concentration (Cmax) in blood was 518 and 990 uM equivalents for PFOA and PFOS, respectively. The PFOS AUC in blood was 8 times higher than the PFOA AUC in blood at a normalized dose of 0.1 mmoles/Kg and the terminal half-life in blood was 40.5 days for PFOS as compared to 8.3 days for PFOA. While PFOA and PFOS are both fluoroorganic materials, comparison of PFOA and PFOS blood kinetics following repeated oral dosing illustrates that PFOA and PFOS are kinetically different in the rat. In the male rat, PFOS attains higher concentrations in the blood and persists in the blood longer than does PFOA.

B

A Simple, Conservative Compartmental Model to Relate Ammonium Perfluorooctanoate (APFO) Exposure to Estimates of Perfluorooctanoate (PFO) Blood Levels in Humans

Paul M. Hinderliter, Ph.D.

Gary W. Jepson, Ph.D.

Biochemical Toxicology
DuPont Haskell Laboratory for Health and Environmental Sciences

10 October, 2001

10

Abstract

A simple and conservative compartmental model was developed to relate ammonium perfluorooctanoate (APFO) exposures to estimates of perfluorooctanoate (PFO) concentrations in human blood. The model was based on kinetic principles, but it did not include mechanistic or physiological descriptions. Further, the model was not intended to replace the need for more robust models that include mechanistic and appropriate physiological descriptions. The model included zero-order mathematical descriptions of oral and inhalation input and a first order elimination description. Standard estimates of the volumes of daily water consumption and air breathed were used to relate daily intake of APFO to concentrations of APFO in air and drinking water. The model was exercised under a variety of exposure conditions and used to create a table relating APFO intake via drinking water and/or air to PFO blood concentrations. The simplicity and utility of this model provide decision-makers with an easily applied tool to relate APFO exposures to estimates of resulting PFO concentrations in human blood.

Introduction

A simple compartmental model was developed and used to estimate the concentration of perfluorooctanoate (PFO) in blood following inhalation or ingestion of ammonium perfluorooctanoate (APFO). The model presented is intended to complement various consequence analysis and planning activities and is not intended to be a substitute for a robust, mechanism based physiological model. It order to realize both the strengths and limitations of the model, it is important to carefully consider the assumptions and caveats relevant to the model development and application.

Approach

Model Development:

The model developed for this application was a two-compartment open model with one compartment defined as the blood compartment and the other as the body compartment. While the model is constructed as a two-compartment model, transfer of PFO is confined to only one compartment (blood compartment) in order to provide a conservative estimate of PFO concentrations in blood following APFO exposure. Functionally, this reduces to a one-compartment open model with two zero-order-input processes and one first-order elimination process. In other words, PFO is confined to the blood compartment and the PFO concentration in blood cannot be reduced by the distribution of PFO into other body tissues. In order to contribute to the conservative estimates produced by this model, any APFO that is ingested or inhaled is not subject to diffusional resistance and is assumed to be completely and instantly absorbed into the blood compartment. Since PFO is not metabolized, elimination from the blood is via renal excretion. In this model the elimination is described as a pseudo first-order process. A schematic of the model is shown in Figure 1.

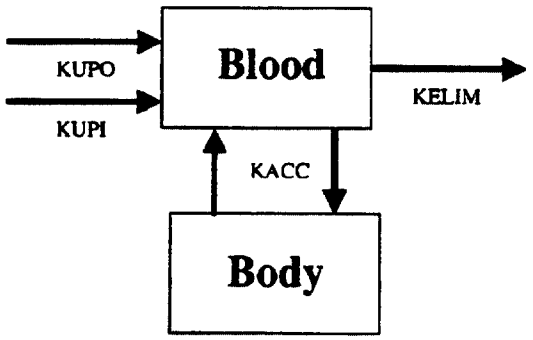


Figure 1. Schematic of PFO Compartmental Model.

In Figure 1, KACC is the distribution coefficient for transfer of PFO between the blood and body compartments. It has the units of day^{-1}, but as discussed earlier, it is set to zero

in order to create a conservative one-compartment model. KUPO is a zero-order term to describe PFO input into the blood compartment (ug/day) via the oral route. KUPI is a zero-order term to describe PFO input into the blood compartment (ug/day) via tne inhalation route. KELIM is a pseudo first-order elimination coefficient (day^{-1}) that describes removal of PFO from the blood compartment via renal excretion. Differential rate equations were developed from the schematic in Figure 1 and the equations were solved using Advanced Continuous Simulation Language (ACSL, Aegis Corp.). The mathematical equations used to describe the concentration of PFO in the blood compartment (CBLOOD) are shown in the series of equations below.

$$\frac{dAB}{dt} = KUPO + KUPI - KELIM * CBLOOD * VOL - RAF \quad (1)$$

$$dAB = (KUPO + KUPI - KELIM * CBLOOD * VOL - RAF)dt \quad (2)$$

$$\int_{AB=0}^{AB} dAB = \int_{t=0}^{t} (KUPO + KUPI - KELIM * CBLOOD * VOL - RAF)dt \quad (3)$$

$$AB = \int_{t=0}^{t} (KUPO + KUPI - KELIM * CBLOOD * VOL - RAF)dt \quad (4)$$

$$CBLOOD = AB / VOL \quad (5)$$

In the equations above, AB is the amount (ug) of PFO in blood, t is time (days), VOL is the volume (ml) of the blood compartment and RAF (ug/day) is the rate of PFO movement between the blood and body compartments (RAF=0 in this model). The ACSL coding of the above equations is given immediately below and in Appendix 1. The corresponding ACSL command file is provided in Appendix 2.

```
RA=KUPO + KUPI - KELIM*CBLOOD*VOL - RAF          (6)
CBLOOD=INTEG(RA,0.)/VOL                          (7)
```

Model Input Assumptions/Descriptions:

Blood Compartment Volume: The blood volume of 3.5 L used in the model was that of a 50-Kg human (average human female weight). The female weight was selected to maintain the conservative approach desired for this model. Obviously, blood volume is a function of body weight so larger body weights will equate to larger blood volumes. PFO concentrations in blood will therefore decrease for a given APFO exposure as body weights increase.

Elimination Rate Constant: The elimination rate constant, KELIM, was assigned a value of 0.0019/day. This was derived assuming a PFO half-life ($t_{1/2}$) in humans of 365 days and that first order kinetics apply. While current human half-life estimates are placed in the 200-300 day range, the 365-day half-life is a conservative value for initial model conditions. The actual value for KELIM was derived using the relationship between the half-life and the elimination rate constant where first order kinetics are obeyed.

$$KELIM = \frac{\ln 2}{t_{1/2}} \quad (8)$$

Input of APFO via Drinking Water: Drinking water concentrations of APFO were converted to micrograms (ug) of APFO ingested per day using the assumption that approximately 2L of the water are consumed per day. An example follows where drinking water containing 1 part per billion (ppb) APFO was consumed:

$$1\ ppb = \frac{1ug}{L} \quad so \quad \frac{1ug}{L} \times \frac{2L}{day} = \frac{2ug}{day} \tag{9}$$

Input of APFO via Inhalation: Inhaled concentrations of APFO were converted to micrograms of APFO absorbed into the blood using the assumption that approximately 20 m^3 of air are breathed per day. An example follows where air containing 1 ug/m^3 APFO was inhaled.

$$\frac{1ug}{m^3} \times \frac{20m^3}{day} = \frac{20ug}{day} \tag{10}$$

General Assumptions:

The simple model described here is designed to be conservative and is not intended to be a substitute for a more robust, mechanism based physiological model. Consistent with the design of this model, several general assumptions have been made.

(1) The PFO is distributed only in the human blood compartment.

(2) There is no metabolism of PFO.

(3) No binding or mechanistic descriptions are included in the model.

(4) Elimination occurs by a single first-order pathway. It is likely that elimination actually displays biphasic elimination with an initial rapid elimination phase followed by a slower or terminal phase elimination. In order to be consistent with the conservative nature of the model, only the slow (terminal) phase elimination is included in the model.

(5) All APF● inhaled or ingested in drinking water is instantly and completely absorbed into the blood compartment.

(6) APFO exposures occur every day throughout the exposure period modeled.

Results

The simulated PFO levels in human blood resulting from repeated ingestion of 6 ug/day APFO are shown in Figure 2. As would be expected based on the estimated half-life of PFO in the human body, the simulation illustrates that steady-state PFO blood levels are reached only after repeated exposure for over 6 years. Figure 3 is a simulation of the elimination of PFO from the blood once PFO levels are at steady state and PFO exposure is terminated.

Figure 2. Simulated PFO Concentration in Human Blood Following Continuous Intake of 6 ug/day

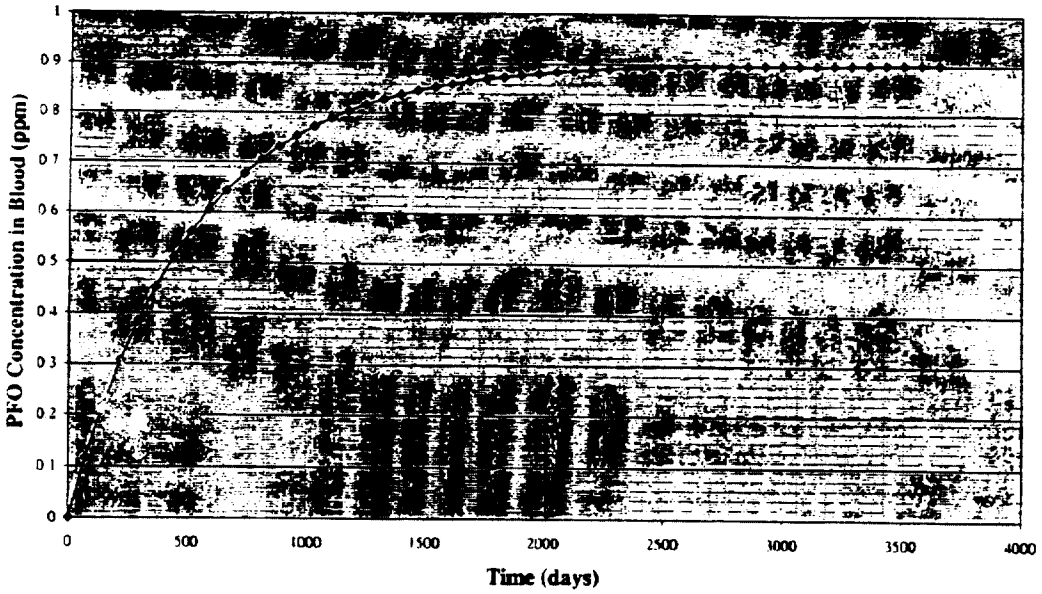
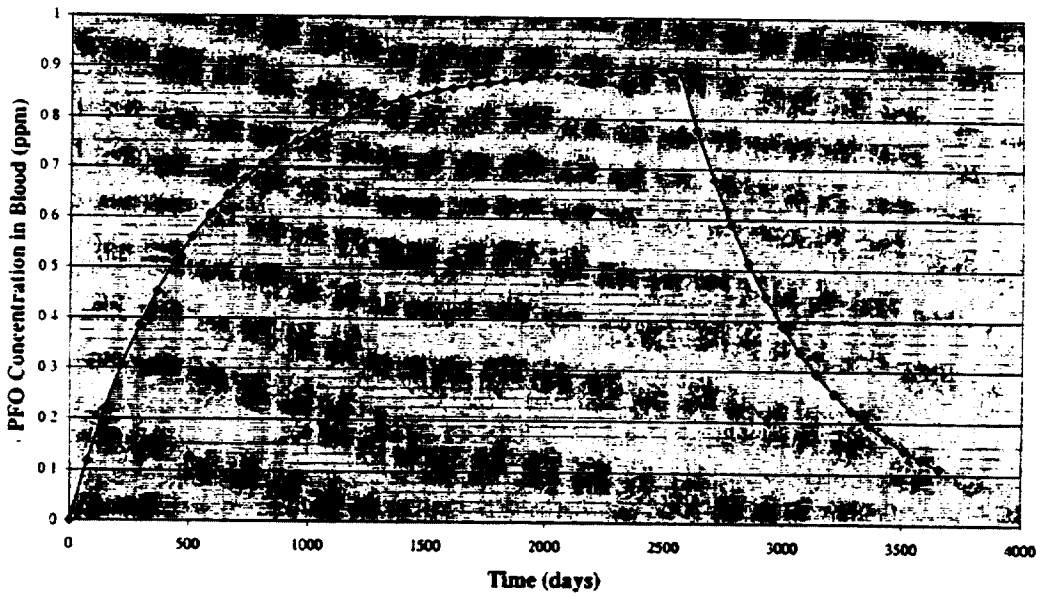


Figure 3. Simulated PFO Concentration in Human Blood During and After 2600 Days of Exposure to 6 ug/day APFO



A series of model simulations were run to estimate the steady-state human PFO blood levels resulting from drinking water containing APFO, breathing air containing APFO or combinations of the two. The resulting estimates of PFO concentrations in human blood are shown in Table 1. Table 1 can be used under the conditions described in the text, to assign a PFO blood concentration to a particular exposure. Example 1: If drinking water containing 1 ppb APFO was consumed and no APFO was present in the inhaled air, the resulting steady-state PFO concentration estimate in human blood would be 0.30 ppm. Example 2: If no APFO was present in the drinking water and 0.05 ug/m^3 APFO was in the inhaled air, the resulting steady-state PFO concentration estimate in human blood would be 0.15 ppm. Example 3: If APFO was present in the drinking water at 1ppb and in the air at 0.3 ug/m^3, the resulting steady-state PFO concentration estimate in human blood would be 1.20 ppm.

Table 1. Estimated human steady-state PFO blood levels (ppm) following exposure to APFO via air and/or drinking water.*

Parts per billion APFO in drinking water

µg/m^3 APFO in air	0	1	2	3	4	5	6	7	8	9	10	15	30	40
0.00	0.00	0.30	0.60	0.90	1.20	1.50	1.80	2.10	2.40	2.70	3.01	4.51	9.02	12.02
0.05	0.15	0.45	0.75	1.05	1.35	1.65	1.95	2.25	2.55	2.85	3.16	4.66	9.17	12.17
0.10	0.30	0.60	0.90	1.20	1.50	1.80	2.10	2.40	2.70	3.00	3.31	4.81	9.32	12.32
0.15	0.45	0.75	1.05	1.35	1.65	1.95	2.25	2.55	2.85	3.16	3.46	4.96	9.47	12.47
0.20	0.60	0.90	1.20	1.50	1.80	2.10	2.40	2.70	3.00	3.31	3.61	5.11	9.62	12.62
0.30	0.90	1.20	1.50	1.80	2.10	2.40	2.70	3.00	3.31	3.61	3.91	5.41	9.92	12.92
0.40	1.20	1.50	1.80	2.10	2.40	2.70	3.00	3.31	3.61	3.91	4.21	5.71	10.22	13.22
0.50	1.50	1.80	2.10	2.40	2.70	3.00	3.31	3.61	3.91	4.21	4.51	6.01	10.52	13.52
1.00	3.00	3.31	3.61	3.91	4.21	4.51	4.81	5.11	5.41	5.71	6.01	7.51	12.02	15.02
2.00	6.01	6.31	6.61	6.91	7.21	7.51	7.81	8.11	8.41	8.71	9.01	10.52	15.02	18.03
3.00	9.01	9.31	9.61	9.91	10.22	10.52	10.82	11.12	11.42	11.72	12.02	13.52	18.03	21.03
4.00	12.02	12.32	12.62	12.92	13.22	13.52	13.82	14.12	14.42	14.72	15.02	16.53	21.03	24.04

PFO Blood levels less than or equal to 5 ppm
PFO Blood levels greater than 5 ppm but less than or equal to 10 ppm

* Use of this table requires careful consideration of assumptions and limitations described in the text.

Discussion

A relatively simple and conservative compartmental model was developed and exercised to create an estimate of the PFO concentration in human blood following exposure to APFO in drinking water and/or inhaled air. The model was then used to create a table relating APFO exposures to estimates of steady-state PFO blood concentrations. Within the constraints of the assumptions and descriptions provided in this report, a variety of

exposure combinations could be evaluated using the model. Given a specific PFO concentration in blood, the model could also be used to create a plausible exposure scenario that could produce the observed PFO blood level. For example, if one had a hypothetical steady-state PFO concentration of 5 ppb in blood, the corresponding APFO exposure estimate using the model would be approximately 16 parts per trillion (ppt).

The model and approach presented in this report may be valuable for consequence analysis or planning activities, however, it should not serve as a substitute for more robust mechanistic, physiologically based models as they become available. The model presented here is based on sound compartmental analysis principles and is exclusive of mechanistic or physiological descriptions. As discussed earlier, this model is based on conservative assumptions and therefore is likely to provide high estimates of PFO concentrations in blood following ingestion or inhalation of PFO. Nevertheless, the simplicity and utility of this model provide decision-makers an easily applied tool to relate APFO exposures to estimates of resulting PFO concentrations in human blood.

Appendix 1: ACSL Model Code

```
PROGRAM
!MODEL TO SIMULATE PFO BLOOD LEVELS FOLLOWING ORAL AND
!INHALATION OF APFO
VARIABLE TIME

INITIAL

!CONSTANTS CAN BE GIVEN VALUES TO SIMULATE EXPOSURE AND
!SYSTEM OF INTEREST

CONSTANT KUPI      = 0.    !ZERO ORDER INHALATION UPTAKE (ug/day)
CONSTANT KUPO      = 0.    !ZERO ORDER ORAL UPTAKE (ug/day)
CONSTANT KELIM     = 0.    !FIRST-ORDER ELIMINATION (/day)
CONSTANT KACC      = 0.    !FIRST-ORDER DISTRIBUTION TO BODY (/day)
CONSTANT VOL       = 1.    !BLOOD VOLUME (ml)
CONSTANT VF        = 1.    !BODY VOLUME (ml)

!TIMING COMMANDS

CONSTANT TSTOP     =3650.      !LENGTH OF EXPOSURE (days)
CONSTANT POINTS    =3650.      !NO. OF POINTS IN PLOT
CONSTANT TOFF      =3650.      !END OF EXPOSURE TIME (DAYS)

CINT=TSTOP/POINTS              !COMMUNICATION INTERVAL
END                            !END INITIAL

DYNAMIC

ALGORITHM IALG=2

DERIVATIVE
IF (TIME .GT. TOFF) THEN
KUPI = 0.
KUPO=0.

END

IF TERMT(TIME.GE.TSTOP)

!CONCENTRATION OF PFO IN THE BLOOD COMPARTMENT (ug/day)
RA=KUPO + KUPI - KELIM*CBLOOD*VOL - RAF
CBLOOD=INTEG(RA,0.)/VOL

!CONCENTRATION OF PFO IN THE BODY
RAF = KACC*(CBLOOD*VOL-CF*VF)
CF = INTEG(RAF,0.0)/VF

END    !END DERIVATIVE
END    !END DYNAMIC
END
```

Appendix 2: ACSL Command File for Assigning Appropriate Parameter Values

```
TSTOP=10*365;
POINTS=50;
TOFF=TSTOP+1;
VOL=3500;

KACC=0.;
KELIM=0.0019;
KUPO=2;
KUPI=6;

keyboard
figure;
!!START
line(_time, _cblood, @linestyle="+");
_cblood(POINTS)

xlabel('Time (Days)');
ylabel('Conc. in blood (ug/mL)');
title('BLOOD CONCENTRATION');
```

C

DuPont Engineering
Barley Mill Plaza - Bldg. 27
Lancaster Pike & Rte. 141
Wilmington, DE 19805

O.E.P.A.

DuPont Engineering

November 20, 2002

David P. Watkins
West Virginia Department of Environmental Protection
Division of Water Resources
1201 Greenbrier Street
Charleston, West Virginia 25311-1088

3Q02 and 4Q02 Public Water Supply Results, West Virginia and Ohio
DuPont Washington Works, Washington, WV

Dear Mr. Watkins:

Sampling of public water supplies along the Ohio River to determine C-8 concentrations began in December 2001 as required by the Consent Order (Order No. GWR-2001-019). Public water supplies located as far as three and a half miles upstream of the DuPont Washington Works facility and 53 miles downstream have been included in the sampling events. Based on the very low C-8 concentrations measured at the various public water supplies, the number and frequency of sampling events at the public water supplies was reduced by the Groundwater Investigation Steering Team in May 2002. Currently, three public water supplies, Lubeck (West Virginia), Tuppers Plains (Ohio) and Little Hocking (Ohio), are sampled on a quarterly basis.

The finalized C-8 results for the 3Q02 and 4Q02 sampling events conducted at Lubeck (West Virginia), Tuppers Plains (Ohio) and Little Hocking (Ohio) are presented in Table 1. For the public water supplies that have been sampled more than once, results are presented with the most recent sampling event listed last. Figure 1 shows the locations of all the public water supplies sampled along the Ohio River.

The next sampling event at these Public Water Supply Wells is scheduled for 1Q2003. Please contact me at (302) 992-6820 if you have any questions.

Sincerely,

Andrew S. Hartten
Project Director

cc: GIST Members

Attachments

OEPA 2548

Table 1.
Summary of C-8 in Groundwater
Public Water Supplies, West Virginia and Ohio
Dupont Washington Works, Washington WV

Location	Sample ID	Sample Date	C-8 ug/l	Comments
Parkersburg, WV	PPSDPT	3/6/2002	NQ (<0.050)	Before Treatment Sample
	PPSDAT	3/6/2002	NQ (<0.050)	After Treatment Sample
	PPSDAT	4/25/2002	NQ (<0.050)	After Treatment Sample
	PPSDRANY1	3/6/2002	0.0693	Production Well
	PPSDRANY1	3/6/2002	0.0686	duplicate
	PPSDRANY1	4/25/2002	0.0746	Production Well
	PPSDRANY2	3/6/2002	ND (<0.010)	Production Well
	PPSDRANY3	3/6/2002	ND (<0.010)	Production Well
	PPSDRANY4	3/6/2002	ND (<0.010)	Production Well
	PPSDRANY5	3/6/2002	ND (<0.010)	Production Well
Belpre, OH	BELPSDAT	2/7/2002	0.0818	After Treatment Sample
	BELPSDAT	3/25/2002	0.113	After Treatment Sample
	BELPSDAT	4/23/2002	0.12	After Treatment Sample
	BELPSDPW1	2/7/2002	0.0995	Production Well
	BELPSDPW1	3/25/2002	0.13	Production Well
	BELPSDPW2	2/7/2002	NQ (<0.050)	Production Well
	BELPSDPW2	2/7/2002	NQ (<0.050)	duplicate
	BELPSDPW2	3/25/2002	NQ (<0.050)	Production Well
	BELPSDPW3	3/25/2002	0.141	Production Well
	BELPSDPW3	4/23/2002	0.12	Production Well
	BELPSDPW4	2/7/2002	0.101	Production Well
	BELPSDPW4	3/25/2002	0.133	Production Well
	BELPSDPW4	4/23/2002	0.114	Production Well
	BELPSDPW5	2/7/2002	0.107	Production Well
	BELPSDPW5	3/25/2002	0.103	Production Well
	BELPSDPW5	4/23/2002	0.107	Production Well
	BELPSDPW5	4/23/2002	0.111	duplicate
Blennerhassett Island, WV	BLENI TW0	2/21/2002	ND (<0.010)	Test well
	BLENI TW7	2/21/2002	NQ (<0.050)	Test well
	BLENI W19A	2/21/2002	0.316	Test well
	BLENISLEPS1	1/30/2002	0.165	Drinking Supply Well
Little Hocking, OH	LHPSD1	12/20/2001	1.82	Production Well
	LHPSD1	1/21/2002	1.72	Production Well
	LHPSD1	2/22/2002	2.37	Production Well
	LHPSD1	3/26/2002	2.99	Production Well
	LHPSD1	4/23/2002	2.02	Production Well
	LHPSD1	8/21/2002	3.65	Production Well
	LHPSD1	10/16/2002	3.41	Production Well
	LHPSD2	12/20/2001	3.72	Production Well
	LHPSD2	12/20/2001	3.52	duplicate
	LHPSD2	1/21/2002	2.97	Production Well
	LHPSD2	2/22/2002	2.03	Production Well
	LHPSD2	2/22/2002	2.07	Production Well
	LHPSD2	3/26/2002	3.31	Production Well
	LHPSD2	4/23/2002	3.4	Production Well
	LHPSD2	8/21/2002	4.26	Production Well
	LHPSD2	10/16/2002	3.98	Production Well
	LHPSD3	12/20/2001	0.844	Production Well
	LHPSD3	1/21/2002	0.744	Production Well
	LHPSD3	2/22/2002	0.42	Production Well
	LHPSD3	3/26/2002	0.827	Production Well
	LHPSD3	4/23/2002	0.783	Production Well

Table 1.
Summary of C-8 in Groundwater
Public Water Supplies, West Virginia and Ohio
Dupont Washington Works, Washington WV

Location	Sample ID	Sample Date	C-8 ug/l	Comments
Little Hocking, OH (cont.)	LHPSD3	8/21/2002	0.952	Production Well
	LHPSD3	10/16/2002	0.495	Production Well
	LHPSD3	10/16/2002	0.434	duplicate
	LHPSD5	12/20/2001	7.66	Production Well
	LHPSD5	1/21/2002	6.22	Production Well
	LHPSD5	1/21/2002	6.14	duplicate
	LHPSD5	2/22/2002	5.69	Production Well
	LHPSD5	3/26/2002	6.57	Production Well
	LHPSD5	4/23/2002	6.11	Production Well
	LHPSD5	8/21/2002	8.09	Production Well
	LHPSD5	10/16/2002	8.58	Production Well
	LHPSDEP001	1/22/2002	1.69	Water System Point
	LHPSDEP001	3/26/2002	2.62	Water System Point
	LHPSDEP001	4/23/2002	1.93	Water System Point
	LHPSDEP001	10/16/2002	4.29	Water System Point
	LHPSDTW1	1/22/2002	2.16	Test well
	LHPSDTW1	8/21/2002	0.81	Test well
	LHPSDTW10	1/21/2002	1.9	Test well
	LHPSDTW10	8/21/2002	1.1	Test well
	LHPSDTW11	1/21/2002	1.41	Test well
	LHPSDTW11	8/21/2002	1.73	Test well
	LHPSDTW12	1/21/2002	0.758	Test well
	LHPSDTW12	8/21/2002	0.824	Test well
	LHPSDTW2	1/22/2002	0.103	Test well
	LHPSDTW2	8/21/2002	0.081	Test well
	LHPSDTW3	1/22/2002	4.48	Test well
	LHPSDTW3	8/21/2002	4.17	Test well
	LHPSDTW4	1/22/2002	37.1	Test well
	LHPSDTW4	3/26/2002	33.3	Test well
	LHPSDTW4	4/23/2002	28.7	Test well
	LHPSDTW4	8/21/2002	12.3	Test well
	LHPSDTW4	10/16/2002	14.5	Test well
	LHPSDTW5	8/21/2002	6.26	Test well
	LHPSDTW6	1/22/2002	1.79	Test well
	LHPSDTW6	8/21/2002	1.15	Test well
	LHPSDTW6	8/21/2002	1.23	duplicate
	LHPSDTW9	1/22/2002	0.364	Test well
	LHPSDTW9	8/21/2002	0.812	Test well
	LHTORCHBS	1/22/2002	1.85	Water System Point
	BARTLETTCC	1/22/2002	1.94	Water System Point
	339B STA	1/22/2002	1.81	Water System Point
General Electric, WV	GE WELL 3	1/3/2002	1.78	Production Well
	GE WELL 3	1/3/2002	1.87	duplicate
	GE WELL 3	2/21/2002	1.75	Production Well
	GE WELL 3	4/26/2002	1.84	Production Well

Table 1.
Summary of C-8 in Groundwater
Public Water Supplies, West Virginia and Ohio
Dupont Washington Works, Washington WV

Location	Sample ID	Sample Date	C-8 ug/l	Comments
Lubeck, WV	LPSDAT	3/28/2002	0.69	After Treatment Sample
	LPSDAT	4/26/2002	0.652	After Treatment Sample
	LPSDAT	6/24/2002	0.6	After Treatment Sample
	LPSDAT	10/15/2002	0.653	After Treatment Sample
	LPSD WELL A	1/3/2002	0.764	Production Well
	LPSD WELL A	2/21/2002	0.683	Production Well
	LPSD WELL A	3/28/2002	0.796	Production Well
	LPSD WELL A	4/26/2002	0.938	Production Well
	LPSD WELL A	7/24/2002	0.753	Production Well
	LPSD WELL A	10/15/2002	0.856	Production Well
	LPSD WELL B	2/21/2002	0.61	Production Well
	LPSD WELL B	3/28/2002	0.551	Production Well
	LPSD WELL B	4/26/2002	0.532	Production Well
	LPSD WELL B	7/24/2002	0.443	Production Well
	LPSD WELL B	10/15/2002	0.537	Production Well
	LPSD WELL C	1/3/2002	0.592	Production Well
	LPSD WELL C	2/21/2002	0.479	Production Well
	LPSD WELL C	3/28/2002	0.491	Production Well
	LPSD WELL C	4/26/2002	0.471	Production Well
	LPSD WELL C	7/24/2002	0.398	Production Well
	LPSD WELL C	10/15/2002	0.504	Production Well
	LPSD WELL D	1/3/2002	0.758	Production Well
	LPSD WELL D	2/21/2002	0.725	Production Well
	LPSD WELL D	3/28/2002	0.692	Production Well
	LPSD WELL D	4/26/2002	0.506	Production Well
	LPSD WELL D	7/24/2002	0.444	Production Well
	LPSD WELL D	10/15/2002	0.517	Production Well
	LPSD WELL E	1/3/2002	0.332	Production Well
	LPSD WELL E	2/21/2002	1	Production Well
	LPSD WELL E	3/28/2002	1.09	Production Well
	LPSD WELL E	4/26/2002	1.11	Production Well
	LPSD WELL E	7/24/2002	1.02	Production Well
	LPSD WELL E	10/15/2002	1.21	Production Well
	LPSD WELL F	1/3/2002	1.04	Production Well
	LPSD WELL F	2/21/2002	0.313	Production Well
	LPSD WELL F	3/28/2002	0.358	Production Well
	LPSD WELL F	3/28/2002	0.352	duplicate
	LPSD WELL F	4/26/2002	0.332	Production Well
	LPSD WELL F	7/24/2002	0.284	Production Well
	LPSD WELL F	7/24/2002	0.283	duplicate
	LPSD WELL F	10/15/2002	0.355	Production Well
Belleville Hydra Plant, WV	BELLEVILLELD	1/29/2002	NQ (<0.050)	Miscellaneous Use
Tuppers Plains PSD, OH	TPPSDPT	2/6/2002	0.372	Before Treatment Sample
	TPPSDPT	3/25/2002	0.347	Before Treatment Sample
	TPPSDPT	7/23/2002	0.24	Before Treatment Sample
	TPPSDPT	10/15/2002	0.226	Before Treatment Sample
	TPPSDAT	2/6/2002	0.361	After Treatment Sample
	TPPSDAT	3/25/2002	0.358	After Treatment Sample
	TPPSDAT	4/24/2002	0.363	After Treatment Sample
	TPPSDAT	7/23/2002	0.246	After Treatment Sample
	TPPSDAT	10/15/2002	0.268	After Treatment Sample
	TPPSDPW1	2/6/2002	0.726	Production Well
	TPPSDPW1	3/25/2002	0.705	Production Well

Table 1.
Summary of C-8 in Groundwater
Public Water Supplies, West Virginia and Ohio
Dupont Washington Works, Washington WV

Location	Sample ID	Sample Date	C-8 ug/l	Comments
Tuppers Plains PSD, OH (cont.)	TPPSDPW1	4/24/2002	0.702	Production Well
	TPPSDPW1	7/23/2002	0.588	Production Well
	TPPSDPW1	10/15/2002	0.486	Production Well
	TPPSDPW2	2/6/2002	0.417	Production Well
	TPPSDPW2	3/25/2002	0.327	Production Well
	TPPSDPW2	4/24/2002	0.371	Production Well
	TPPSDPW2	7/23/2002	0.235	Production Well
	TPPSDPW2	10/15/2002	0.255	Production Well
	TPPSDPW3	2/6/2002	NQ (<0.050)	Production Well
	TPPSDPW3	3/25/2002	NQ (<0.050)	Production Well
	TPPSDPW3	7/23/2002	ND (<0.010)	Production Well
	TPPSDPW3	10/15/2002	ND (<0.010)	Production Well
	TPPSDPW4	2/6/2002	0.0734	Production Well
	TPPSDPW4	3/25/2002	0.07	Production Well
	TPPSDPW4	7/23/2002	0.052	Production Well
	TPPSDPW4	10/15/2002	0.076	Production Well
	TPPSDPW5	2/6/2002	0.201	Production Well
	TPPSDPW5	3/25/2002	0.201	Production Well
	TPPSDPW5	7/23/2002	0.216	Production Well
	TPPSDPW5	10/15/2002	0.229	Production Well
	TPPSDPW6	2/6/2002	0.649	Production Well
	TPPSDPW6	3/25/2002	0.634	Production Well
	TPPSDPW6	4/24/2002	0.62	Production Well
	TPPSDPW6	7/23/2002	0.62	Production Well
	TPPSDPW6	10/15/2002	0.433	Production Well
Ravenswood Municipal, WV	COR ASIN	3/27/2002	ND (<0.010)	Air Stripper In
	COR ASOUT	3/27/2002	NQ (<0.050)	Air Stripper Out
	COR BLEND AT	3/27/2002	ND (<0.010)	Blend After Treatment
	COR WELL1	3/27/2002	ND (<0.010)	Production Well
	COR WELL 2	3/27/2002	ND (<0.010)	Production Well
	COR WELL3	3/27/2002	ND (<0.010)	Production Well
	COR WELL 4	3/27/2002	ND (<0.010)	Production Well
	COR WELL 5	3/27/2002	ND (<0.010)	Production Well
Mason County PSD, WV	MASONCPSD1	1/29/2002	NQ (<0.050)	Production Well
	MASONCPSD1	3/27/2002	NQ (<0.050)	Production Well
	MASONCPSD1	4/25/2002	NQ (<0.050)	Production Well
	MASONCPSD2	1/29/2002	0.0618	Production Well
	MASONCPSD2	3/27/2002	0.0838	Production Well
	MASONCPSD2	4/25/2002	0.0714	Production Well
	MASONCPSD3	1/29/2002	0.0707	Production Well
	MASONCPSD3	3/27/2002	0.102	Production Well
	MASONCPSD3	4/25/2002	0.063	Production Well
Racine Locks and Dam, WV	RACINELD	1/4/2002	0.518	Miscellaneous Use
Village of Racine, OH	VORAT	3/26/2002	ND (<0.010)	After Treatment Sample
	VORRV3	3/26/2002	ND (<0.010)	Production Well
	VORWELL1	3/26/2002	ND (<0.010)	Production Well
	VORWELL2	3/26/2002	ND (<0.010)	Production Well
New Haven Water Dept., WV	NHPSDAT	4/10/2002	ND (<0.010)	After Treatment Sample
	NHPSDPW1	4/10/2002	NQ (<0.050)	Production Well
	NHPSDPW1	4/10/2002	NQ (<0.050)	duplicate

OEPA 2552

Table 1.
Summary of C-8 in Groundwater
Public Water Supplies, West Virginia and Ohio
Dupont Washington Works, Washington WV

Location	Sample ID	Sample Date	C-8 ug/l	Comments
Village of Syracuse, OH	VOSAT	3/29/2002	NQ (<0.050)	After Treatment Sample
	VOSAT	4/24/2002	ND (<0.010)	After Treatment Sample
	VOS NORTH 2	3/29/2002	NQ (<0.050)	Production Well
	VOS NORTH 2	4/24/2002	0.491	Production Well
	VOS SOUTH R3	3/26/2002	0.208	Production Well
	VOS SOUTH R3	4/24/2002	ND (<0.010)	Production Well
Village of Pomeroy, OH	VOPAT	3/26/2002	0.0659	After Treatment Sample
	VOPAT	4/24/2002	0.0628	After Treatment Sample
	VOPWELL1	4/24/2002	ND (<0.010)	Production Well
	VOPWELL2	3/26/2002	0.0689	Production Well
	VOPWELL2	4/24/2002	ND (<0.010)	Production Well
	VOPWELL4	3/26/2002	0.0851	Production Well
	VOPWELL4	4/24/2002	0.0712	Production Well

ND = Not Detected at or above the limit of detection (LOD).
The listed LOD is approximate and varies by instrument and over time.
NQ = Not Quantifiable. Detected at a level above the LOD and below the limit of quantification (LOQ).
All C-8 results are reported in ug/l.
Misc. = Miscellaneous water use is not used for drinking.

OEPA 2553

D

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June 13, 2002

Mr. Armando Benincasa
Dr. Dee Ann Staats
West Virginia Department of Environmental Protection
1356 Hansford Street
Charleston, WV 25301

Dear Dr. Staats and Mr. Benincasa:

Enclosed is the final report of the air dispersion modeling for year 2000 actual emissions. Please contact me if you have any questions regarding this matter.

Very truly yours,

M. Ann Bradley

Enclosure

cc: Christopher Negley, Esq.
Mr. Michael Baker, Ohio EPA
Mr. John Benedict, DAQ
Mr. Jesse Hanshaw, DAQ

ISC Modeling Methodology and Results

Emission Source Information

The ISC3 model was used to calculate ambient ground-level air concentrations and deposition rates for year 2000 actual C8 emissions from the Washington Works site. Table 1 shows the stack parameters used in the model for each emission point. Table 2 shows the emission rates used. The stack parameters and emission rates used are those that were submitted pursuant to Consent Order GWR-2001-019. In addition, two additional emission points have been added to the model beyond what was submitted pursuant to the Consent Order. Since the C8 emissions are partitioned between the vapor and particle phases, deposition runs were completed by modeling each phase separately. (Modeling runs to determine ground-level concentrations were based on the total emissions.)

Deposition modeling requires particle size distribution information and scavenging coefficients for each phase of emissions (vapor and particle). The size distribution information used in the modeling for the particle phase was obtained from testing at the Washington Works site. The scavenging coefficients used for the particle phase were obtained from Figure 1-11 of the EPA ISC3 User's Guide. The vapor phase scavenging coefficients used were based on calculations by DuPont which were submitted under the Consent Order. This data shows the calculated vapor scavenging coefficient based on rain intensity. Since only one value of the scavenging coefficient can be entered into the ISC3 model, the largest scavenging coefficient was chosen to ensure that the model predictions were conservative. Table 3 shows the gas and particle data used in the model and, additionally, shows the basis for the vapor scavenging coefficient used in the model.

Modeling Methodology

Dispersion and deposition modeling was performed using the Industrial Source Complex 3 Model (ISC3), version 00101, provided by Lakes Environmental. All modeling was done in accordance with the procedures in EPA's Guideline on Air Quality Models (40 CFR Part 51, Appendix W). The EPA regulatory default options and rural dispersion coefficients were used in the model.

The C8 emission sources were evaluated for downwash effects from surrounding buildings. The Lakes Environmental BPIP View model was used to provide wind direction specific building parameters. All buildings on the site were evaluated to determine if they could potentially impact the stack by causing building downwash effects. A plot plan showing the location of buildings included in the model is shown in Figure 1. (The buildings included in the model are identical to the list submitted under Consent Order GWR-2001-019).

A 100-meter grid extending out 4,000 meters from the source was used. In addition, discrete receptors with 100-meter spacing were placed on the plant property line. Terrain elevations were imported from electronic files obtained from the U.S. Geological Survey using the "highest" method to assign an elevation to each receptor.

An additional receptor grid was used to determine deposition to the watershed for the Little Hocking Water Association well field. A USGS topographical map was used to identify the general area of the watershed (Figure 2), and a receptor grid with 100 meter spacing was placed within this watershed (Figure 3).

One year of on-site meteorological data (1996) was analyzed. The data was processed by Trinity consultants, using Wilmington, Ohio for the upper air data. The precipitation data used is from the Parkersburg, West Virginia airport. Missing data and measured wind speeds of less than 1 m/s were treated consistent with the recommendations made in EPA's On-site Meteorological Program Guidance for Regulatory Modeling. An anemometer height of 10 meters was used for the modeling.

Modeling Results

An averaging time of one year was used to determine the annual average vapor concentrations and annual deposition rates over the entire receptor grid. A contour plot of the annual average vapor concentrations is shown in Figure 4. Contour plots of the total deposition rates for the particle and vapor phases are shown in Figures 5 and 6. The maximum off-site values predicted by the model were:

Maximum Annual Average Ground-Level Concentration = 2.675 $\mu g/m^3$

Particle Phase: Maximum Dry Deposition Rate = 0.1347 $g/m^2/yr$
Maximum Wet Deposition Rate = 0.0456 $g/m^2/yr$
Maximum Total Deposition Rate = 0.1803 $g/m^2/yr$

Vapor Phase: Maximum Wet Deposition Rate = 0.0085 $g/m^2/yr$

The maximum ground-level concentration and all of the maximum deposition rates were predicted to occur at the same receptor (442135.47E, 4346899N), which is located on the plant fenceline north of the plant. The maximum annual ground-level concentration predicted to occur in areas where people may reside in the community is approximately 0.8 $\mu g/m^3$.

Additionally, a smaller receptor grid was used to determine the annual deposition rate to the Little Hocking well watershed. The model was run to calculate vapor and particle phase deposition rates for each receptor, which rates were then imported into a spreadsheet. An average deposition rate was calculated for all of the receptors and multiplied by the receptor grid area (2.57 km^2) to get a total deposition per year over the entire watershed. The deposition amounts calculated were:

Particle Phase: Total Dry Deposition = 6,732 g/yr (14.8 lb/yr)
Total Wet Deposition = 12,608 g/yr (27.8 lb/yr)
Total Deposition = 19,340 g/yr (42.6 lb/yr)
Vapor Phase: Total Wet Deposition = 1,644 g/yr (3.6 lb/yr)

Table 1

Stack Parameters

Permit Number	Permit Point ID	Reg 29 Point ID	Zone 17 UTM E	UTM N	Stack Height (ft)	Stack Diameter (ft)	Stack Flow (acfm)	Stack Velocity (ft/s)	Stack Temperature (F)
1823A	T7IME	662	442025	4346847	150	1.33	3,349	40.2	172
815D	T6IFCE	644	442084	4346835	59	1.5	18,000	169.8	111
815D	T6IZCE	699	442091	4346836	63	18 -1 ft	18000[a]	21.2[b]	111
1353A	164-5E	652	441920	4346767	70	1.96	9,800	54.1	200
Pre-Existing	164-2E	658	441923	4346756	68	1.63	2,800	22.4	300
614A	163-E-26	231	441952	4346776	93	0.67	500	23.6	130
614A	163-E-11	232	441953	4346766	81	0.67	600	28.4	130
781	163-E-33	216	441960	4346788	60	1.3	2,750	34.5	158
1953	242	242	441954	4346741	114.5	0.5	1,250	106.1	200
2365A	C1FSE	274	441787	4346744	110	0.69	1,000	44.6	255
2365A	C1FKE	268	441774	4346753	72.5	0.27	100	29.1	110
2365A	C1CAE	205	442310	4346800	6.66	0.5	1,000	84.9	70
Semiworks Application	RO22EEF6		442086	4346624	47	2.5	8836	30.0	80
Semiworks Application	RO22EEF86		442069	4346627	49	2.0	7540	40.0	80
Semiworks Application	RO22EEF87		442058	4346634	49	2.0	1885	10.0	80
Semiworks Application	RO22EEF89		442063	4346635	49	2.0	3770	20.0	80

[a]Vent ID T61ZCE consists of 18 one-foot diameter vents. The flow rate given is the total for all 18 vents.

[b]The velocity listed is the velocity calculated for one individual vent.

Table 2
Emission Information

Permit or ID	Reg 29 Number	Particle Mass Fraction	Vapor Mass Fraction	Year 2000 Actual C8 Emissions (lb/yr)	Year 2000 Actual C8 Emissions (g/s)	2000 Actual Particle Phase Emission Rate (g/s)	2000 Actual Vapor Phase Emission Rate (g/s)
T7IME	662	0	1	0	0	0	0
T6IFCE	644	0.54	0.46	13,977	0.2010	0.1086	0.0925
T6IZCE	699	0.9	0.1	0	0	0	0
164-5E	652	0.9	0.1	33	0.0005	4.27E-04	4.75E-05
164-2E	658	0.9	0.1	79	0.0011	0.00102	1.14E-04
163-E-26	231	0.11	0.89	3,541	0.0509	0.00560	0.0453
163-E-11	232	0.09	0.91	4,680	0.0673	0.00606	0.0613
163-E-33	216	0	1	0	0	0	0
242	242	0.9	0.1	3,510	0.0505	0.0454	0.00505
C1FSE	274	0.03	0.97	5,414	0.0779	0.00234	0.0755
C1FKE	268	1	0	107	0.0015	0.0015	0
C1CAE	205	1	0	0	0	0	0
RO22EEF6		1	0	12	1.73E-04	1.73E-04	0
RO22EEF86		1	0	0.3	4.32E-06	4.32E-06	0
RO22EEF87		1	0	3	4.32E-05	4.32E-05	0
RO22EEF89		1	0	0.6	8.63E-06	8.63E-06	0

Table 3
Gas & Particle Data

Particle Phase:

Particle Diameter (microns)	Mass Fraction	Particle Density (g/cm^3)	Scavenging Coefficients Liquid Precipitation ($s^{-1}/mm\text{-}h^{-1}$)	Scavenging Coefficients Frozen Precipitation ($s^{-1}/mm\text{-}h^{-1}$)
0.2	0.538	2.2	$1.2x10^{-4}$	$4x10^{-5}$
0.4	0.267	2.2	$5x10^{-5}$	$1.67x10^{-5}$
0.75	0.035	2.2	$4x10^{-5}$	$1.33x10^{-5}$
2.0	0.127	2.2	$1.3x10^{-4}$	$4.33x10^{-5}$
4.0	0.033	2.2	$2.8x10^{-4}$	$9.33x10^{-5}$

Vapor Phase:

Liquid Scavenging Coefficient ($s^{-1}/mm\text{-}h^{-1}$) = $6.4x10^{-6}$
Frozen Scavenging Coefficient ($s^{-1}/mm\text{-}h^{-1}$) = $6.4x10^{-6}$

Calculations of Vapor Scavenging Coefficient:
- vapor scavenging coefficients are presented in the consent order submittal as a list of values for different rainfall intensities
- the vapor scavenging coefficient that is entered into the ISC model is in units of $s^{-1}/mm\text{-}h^{-1}$, therefore the scavenging coefficients shown in the consent order must be adjusted to the proper units and then divided by the rainfall intensity
- to ensure that model predictions would be conservative, the scavenging coefficient based on a 1 mm/hr rain intensity was used, as this gives the largest value for input into the model

$$2.311x10^{-2}\frac{1}{hr}\times\frac{hr}{3600s}\times\frac{hr}{1mm}=6.4x10^{-6}\frac{hr}{s\cdot mm}=6.4x10^{-6}\frac{s^{-1}}{mm\cdot hr^{-1}}$$

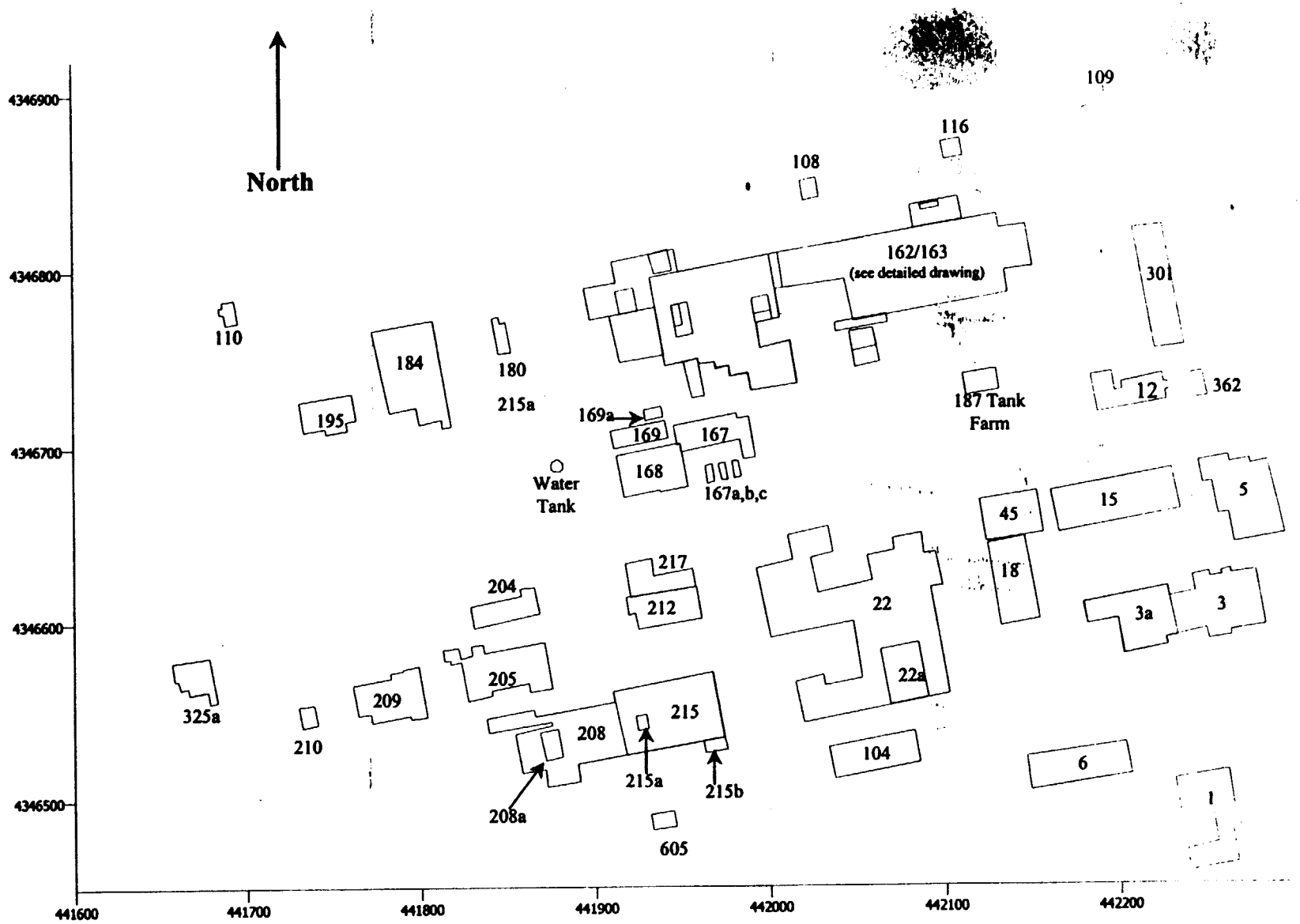

Figure 1 – Building Plot Plan

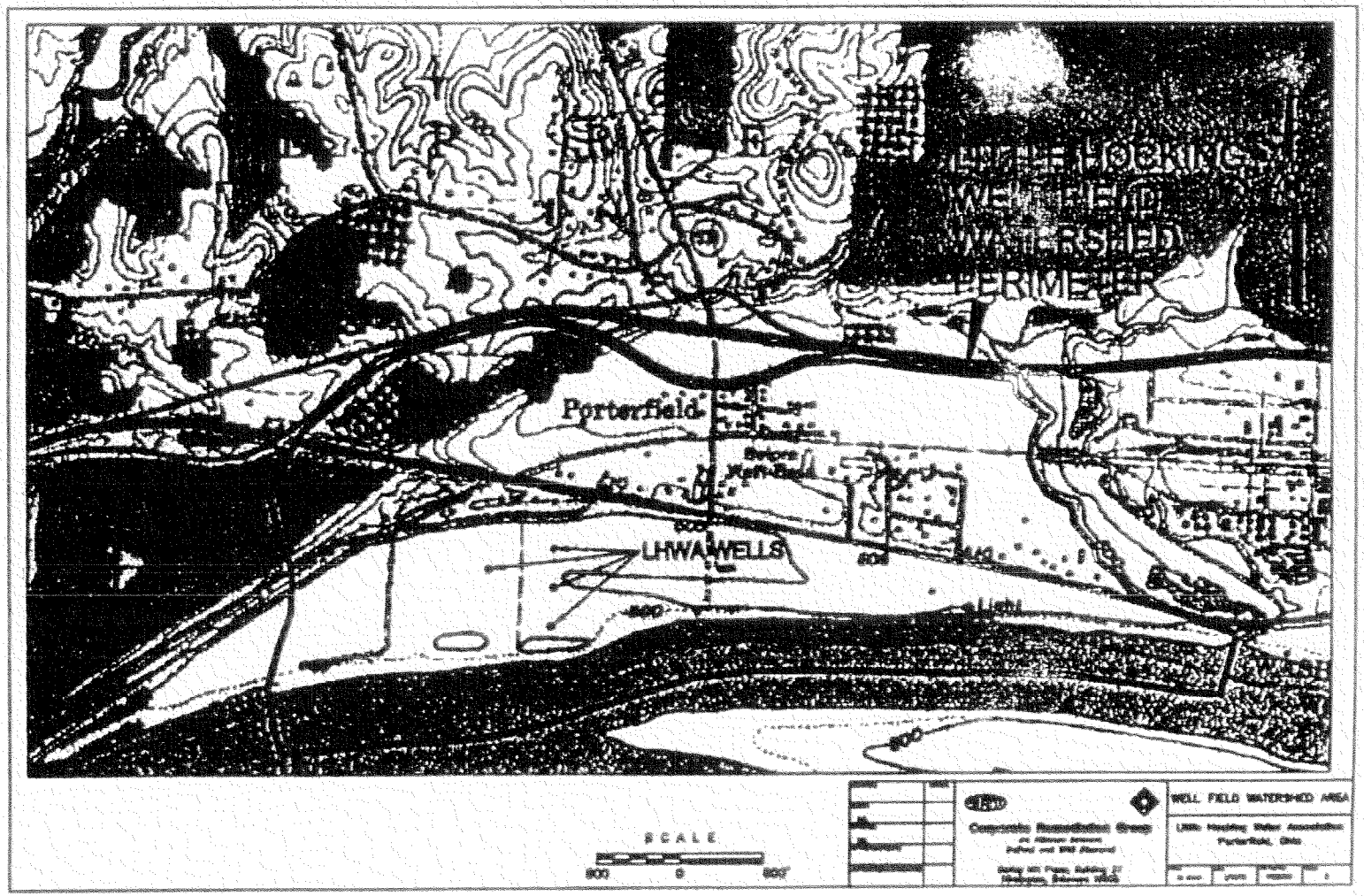

Figure 2

35

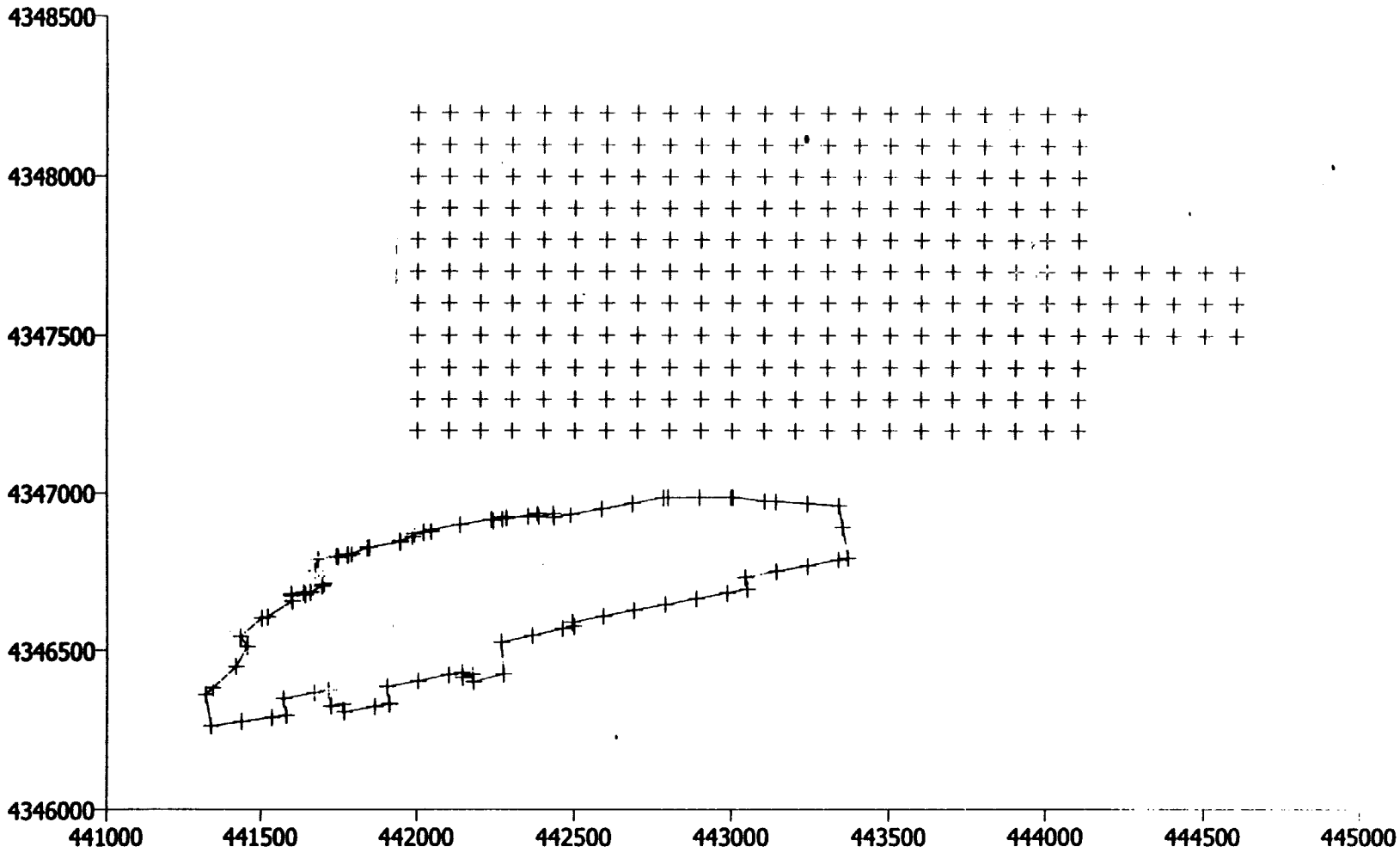

Figure 3
Little Hocking Well Watershed Receptors Modeled

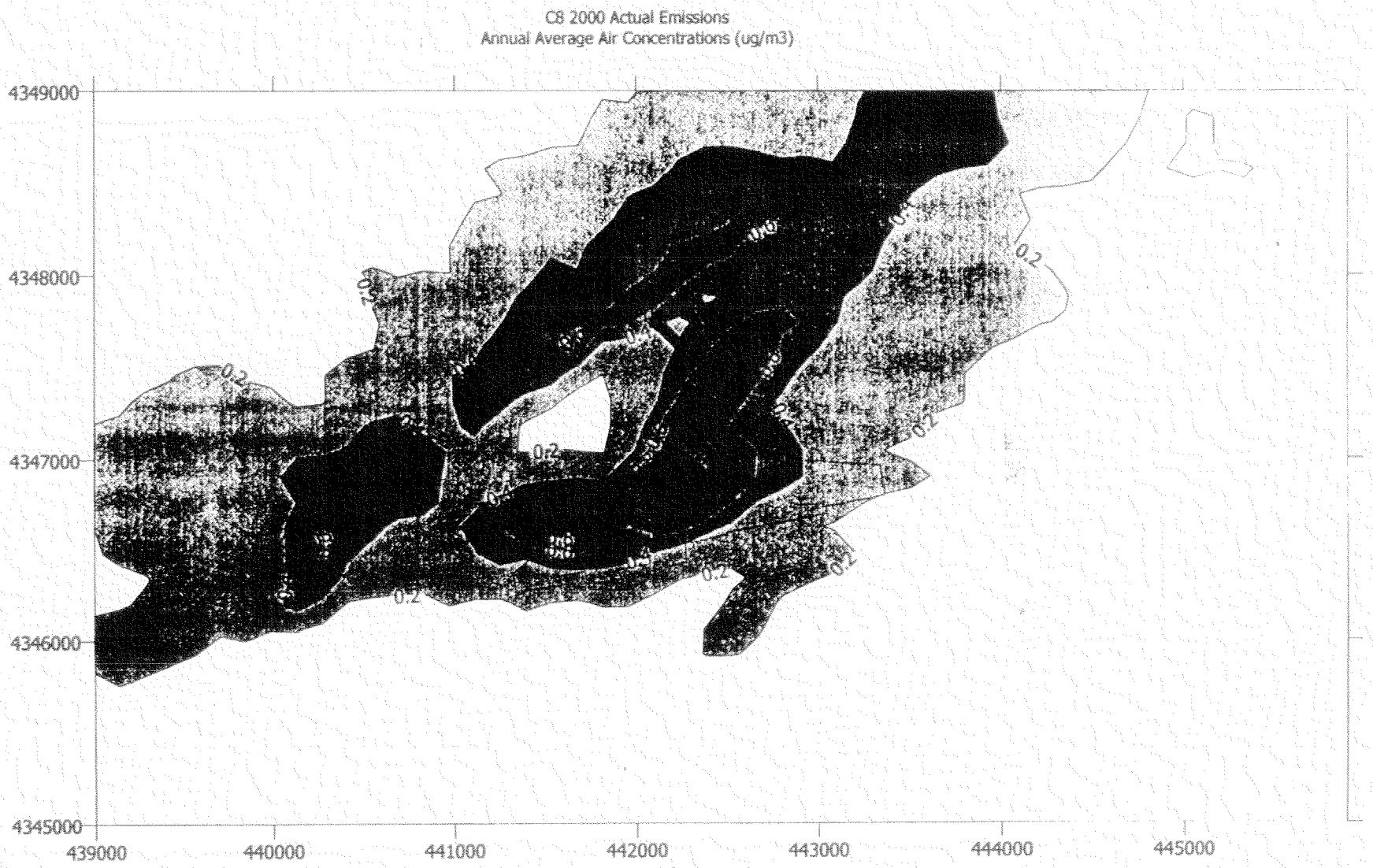

Figure 4
Maximum Ground-Level Concentrations

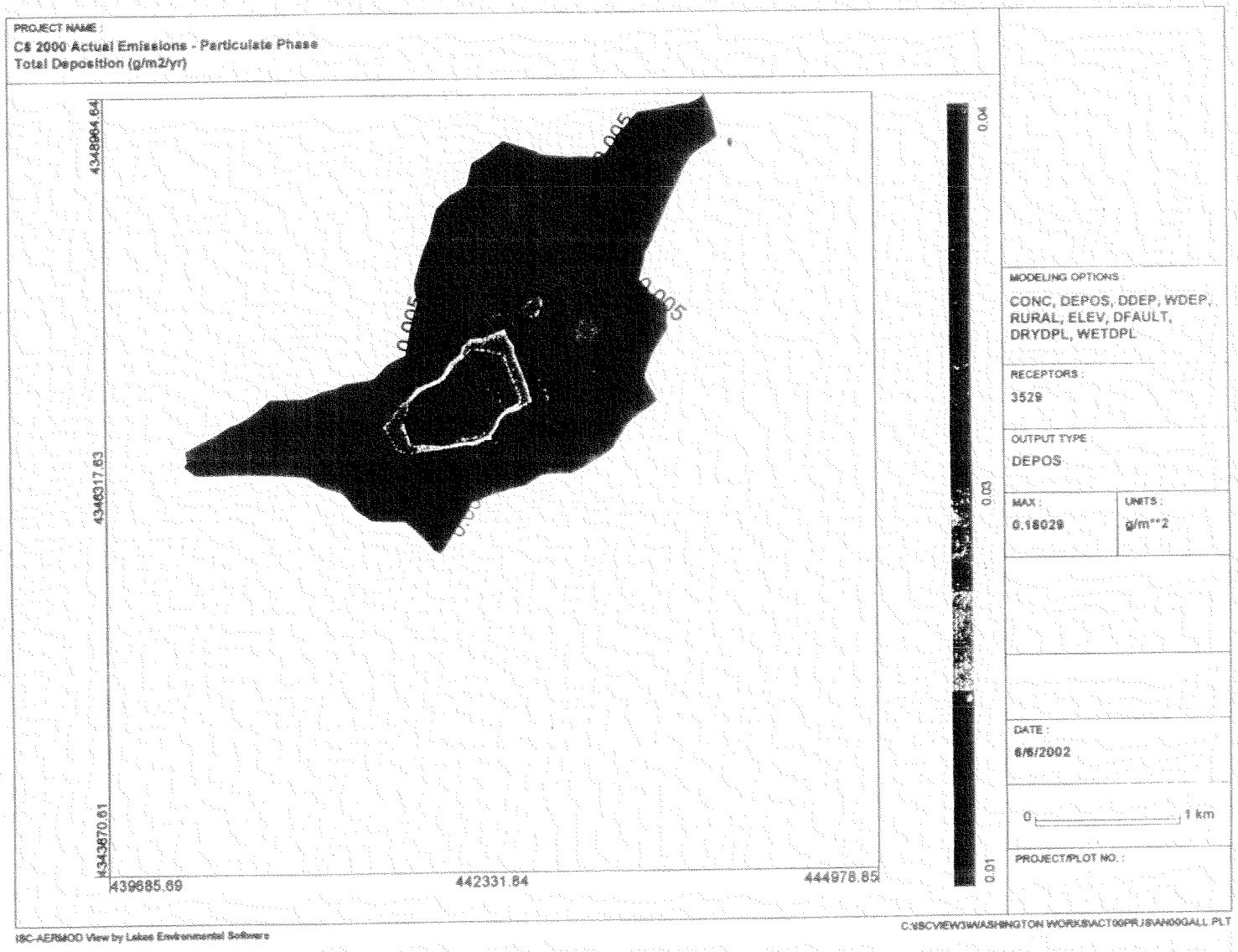

Figure 5 – Particle Phase Total Deposition Rates

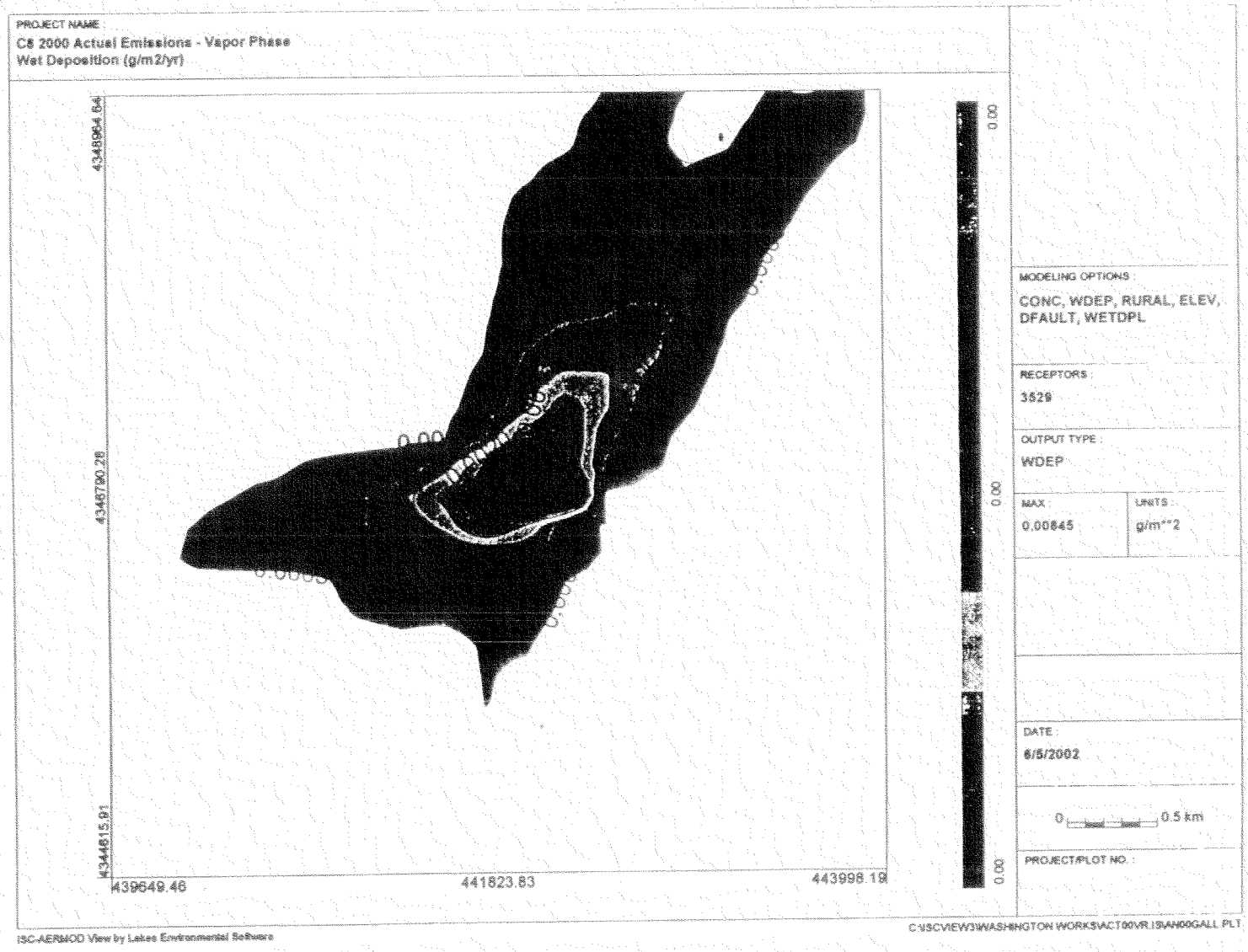

Figure 6 – Vapor Phase Wet Deposition Rates

E

OhioEPA
State of Ohio Environmental Protection Agency

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Columbus, OH 43215-1099

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January 7, 2003

Post-it® Fax Note 7671 | Date | # of pages ▶ 2
To JOHN BEDFORD | From BILL SPIRES
Co./Dept. | Co.
Phone # | Phone #
Fax # | Fax #

Mr. Paul Bossert
E.I. Du Pont de Nemours and Company
Washington Works
P.O. Box 1217
Parkersburg, West Virginia 26102

Re: Ammonium Perfluorooctanoate (C8) Impacts in Little Hocking, Ohio

Dear Mr. Bossert:

I am writing to express Ohio EPA's concerns with respect to air impacts in Ohio due to emissions of C8 from your Washington Works facility. We have been following the progress on the evaluation and review of ambient impacts due to this facility, as well as the evaluation of ambient screening levels for both the air and water pathways developed by the C8 Assessment of Toxicity Team (CATT). We are also aware of additional evaluations in progress by U.S. EPA and the Science Advisory board for reassessing the toxicity and possible carcinogenic effects of C8.

In addition, we have been contacted by a citizens group expressing concern over ambient levels of C8, as predicted by air quality modeling of year 2000 actual emissions. Short term levels used to assess workplace exposures were identified as health based levels not fully considered by the CAT team in the development of an air screening level for C8.

My staff has assessed the modeled ambient impacts resulting from the year 2000 emission levels. Among the averaging times evaluated, my staff has identified predicted eight-hour concentrations well above the ACGIH health based work place standard at multiple receptors in Ohio. While these ambient concentrations may not be directly comparable to workplace exposures, these predicted concentrations raise our level of concern with regards to short term exposures to workers and sensitive populations.

Du Pont has indicated that future production levels, modifications to a scrubber, a taller stack on a major source, as well as agreements with U.S. EPA to reduce releases to all media will limit future impacts in the area. You presented information that future impacts would be below those associated with year 2000 actual impacts. And, the year 2000 emissions are the basis of your agreement with West Virginia to limit future air emission levels. However, we do not believe that there is enough certainty in the assumptions to insure that future ambient air impacts will be significantly below 2000 levels.

We are requesting that you work with the State of West Virginia to adopt enforceable permit restrictions that will reduce ambient impacts below the workplace exposure limits at all receptors beyond the plant fence line. We believe that this is a prudent interim step until the potential effects of C8 are fully evaluated. Once these evaluations are complete, future acceptable emission levels can be determined.

If you have any questions with respect to our concerns, please feel free to contact Bob Hodanbosi of the Division of Air Pollution Control at 614-644-2270.

Sincerely,

Christopher Jones
Director

cc: Benard J. Reilly Esq., E.I. Du Pont de Nemours and Company, Legal Dept.
John Benedict, WV Department of Environmental Protection

F

FYI-0101-1378

DuPont Haskell Laboratory for Toxicology and Industrial Medicine
Elkton Road P.O. Box 50
Newark DE 19714-0050

DUPONT

DuPont Haskell Laboratory

AR226-1000

FYI-00-001378

45010000003

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
Washington, D.C. 20460

COMPANY SANITIZED

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.

- 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 UEIP. 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)366-5259.

Very truly yours,

Gerald L. Kennedy
Director, Applied Toxicology and Health

Attachments

G

CENTRAL RESEARCH AND DEVELOPMENT DEPARTMENT
HASKELL LABORATORY FOR TOXICOLOGY
AND INDUSTRIAL MEDICINE

cc: J. B. Armitage-
PPD, M-5622
W. L. Sprout

June 25, 1987

TO: H. A. SMITH
PPD
M-5625

FROM: G. L. KENNEDY, JR.

AMMONIUM PERFLUOROOCTANOATE
(Ref.: Letter HAS-GLK, 6/12/87

An acceptable level for ammonium perfluorooctanoate (C-8) in the blood of workers would be 0.5 ppm. This value has been calculated using the average daily C-8 accumulation rate observed in new employees who were exposed to airborne concentrations of 0.008 mg/m^3 (memo, J. G. Loschiavo to R. J. Zipfel, 7/29/82). From this data, a steady-state concentration of 0.5-6 ppm, which represents the dynamics of exposure and elimination, was estimated (Memo, T. P. Pastoor to J. G. Loschiavo, 8/25/82). These estimates appear consistent with most of the reported human data but the data base is not too extensive. In addition, in rat inhalation experiments, no signs of toxicity were detected following exposure to 1 mg/m^3, an atmospheric concentration corresponding to a blood level in the male rat of 12 ppm. Extrapolation of the data relating the concentration of C-8 in the air to blood levels in the rat suggests that inhalation of 0.01 mg/m^3 would result in blood level of approximately 1 ppm (equation is blood level = 12 × air concentration).

An acceptable level for community drinking water would be 5 ppb. This value has been arrived at as follows:

1. The AEL (8-hr TWA) is 0.01 mg/m^3; a worker breathing 10m^3/day would take in 0.1 mg. Assume 100% absorption.

2. Daily ingestion by man of 2 L of water/day: 0.1 mg/2L (assume 100% absorption) = 50 ppb (a concentration in water).

3. However, community populations are not equivalent to worker populations. Therefore, factor in a 10X reduction - 5 ppb (concentration in water).

This doesn't take into account the time factor (worker exposed 8 hours, not-exposed 16 hours, etc. whereas drinking water intake could be anytime during 16 hours, off 8 hours, etc.). However, the long half-life of this chemical in the blood might make this consideration less important.

I hope that these suggested guidelines will be useful. Please call if you have any questions.

GLK:ms

H

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. Stevart

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)

003855-1 - RJZ:kst

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 - ZIPPEL)

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)

003855-2 - RJZ:kst

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)

Attachments
003855-3 - RJZ:kst

PFOA Pharmacokinetics Review

David Gray, Ph.D.
Sciences International Inc.

A considerable amount of data on PFOA distribution and kinetics has been generated in studies on laboratory animals (Biegel, 1997; Goldenthal, 1978a,b; York, 2002; Thomford, 2001). These data reveal a complex relationship between dose, body-burden, and toxic effect.

Serum PFOA concentrations have been determined at various time intervals during repeated oral or inhalation dosing or after a single dose. Urine and fecal PFOA concentrations have also be obtained in some studies. Test species include rats, mice, hamsters, monkeys, and dogs. The data indicate that renal and developmental toxicity occurs in the female rat with PFOA serum levels of approximately 1 part-per-million (ppm). The currently-available compartmental PFOA kinetic model for humans indicates that a PFOA blood level of 1 ppm would be reached in exposed human populations if their only source of PFOA exposure was the consumption of drinking water at something less than 3 parts-per-billion (ppb). If human populations were additionally exposed to PFOA in air, the model indicates that it would take significantly less PFOA in drinking water to reach the 1 ppm blood level that results in toxicity to the female rat. The details of this analysis follow below.

[The data on the partitioning of PFOA between serum and red blood cells indicates that, although there is a difference between serum concentration and blood concentration for PFOA, it is not large since most, but not all, blood PFOA is in the serum fraction. Therefore, serum levels and blood levels will be considered comparable without adjustment in the present report to simplify the presentation.]

1. Gender differences in sensitivity to the effects of PFOA exposure

Much attention has been given to the early observation that the male rat is much less efficient in clearing PFOA than the female rat (Hanhijarvi et al., 1982). Urinary, and to a lesser extent fecal

excretion, are the primary clearance processes for PFOA. PFOA is not thought to be metabolized and is excreted as the intact molecule.

A number of studies have reported that the active secretory mechanism responsible for the rapid urinary excretion of PFOA in the female rat is inhibited by testosterone in the male rat (Ylinen et al., 1989; Vanden Heuvel et al., 1992; Kudo et al., 2002). Consequently, the elimination half-life is much longer in the male rat (9 days) than in the female (4 hours) (Vanden Heuvel et al., 1991). This allows serum PFOA levels in male rats to reach much higher levels than in an equivalently dosed female. Other species also exhibit a considerable gender difference in the elimination of PFOA, e.g. the male hamster is more efficient than the female. Rhesus monkeys did not demonstrate a large sex-linked elimination difference (Goldenthal 1978b).

The greater accumulation of PFOA in a repeatedly dosed male rat results in the male exhibiting much greater toxicity than the female at the same dose level. This difference in apparent sensitivity has led investigators to consider the male rat to be the more susceptible gender and to base PFOA risk analysis on the male rat. However, if one considers differences in body-burden, the female rat is the more sensitive sex.

For most compounds, serum concentration is a reflection of total body-burden once steady-state is reached during a repeated dosing experiment or during chronic exposure to human populations. This is true whether or not a compound binds to serum proteins, such as has been reported for PFOA. In a recent 2 generation reproduction study in rats, York (2002), the high dose parental females exhibited toxicity (absolute and relative kidney weights and F1 pup body weight) at a PFOA serum level of about 1 ppm. The parental males demonstrated more pronounced toxicity, but the average serum level in the equivalent high-dose group was very much higher at 45 ppm.

In an earlier 90-day study by Goldenthal et al. (1978a), serum PFOA levels showed an even greater difference between male and female rats. This study reported liver hypertrophy, pigmentation, and necrosis that was more pronounced in males than females. However, hepatocellular necrosis and diffuse sinusoidal hepatic congestion was reported in females at

doses of 2.5, 18, 25, and 80 mg/kg/d. Serum PFOA levels in the 2.5 mg/kg/d female group averaged 0.15 ppm. Serum PFOA levels in the 2.5 mg/kg/day male rat group average 34 ppm. While the effects in males were more pronounced, their body-burden was over 200 fold greater than that of female rats at the same dose level. Again, these data suggest that female rats may actually be more sensitive to the effects of PFOA than males. A direct comparison of male and female rats at the same or similar serum PFOA levels in the same study is difficult because the range of doses in any of the studies is not great enough to provide an overlap in serum levels.

If one considers nonhuman primates, the same magnitude of gender difference in elimination efficiency has not been reported. Goldenthal et al. (1978b) conducted a PFOA study in Rhesus monkeys. In this study, female monkeys had slightly higher serum PFOA levels than did males from the same dose group, indicating there is not a major gender difference in elimination. This study indicates that the large gender difference in PFOA elimination that has been demonstrated in rats is not present in Rhesus monkeys and may not occur in humans.

In summary, the available distribution and kinetic data for PFOA indicates that the female rat is likely the more sensitive sex when body-burden rather than administered dose is used as a dose metric.

2. The very long PFOA half-life in humans

The current estimate of the elimination half-life in humans is 4 years, much longer than any other species. This long half-life indicates that humans excrete PFOA much more slowly than other species. For long-term human exposures, PFOA would be expected to accumulate to high body-burdens over very long periods (~ 20 years) before leveling out. Low-level environmental exposures to PFOA, accumulated over a long period, could eventually result in significant body-burdens. The inability of humans to efficiently excrete PFOA potentially makes the chronically-exposed human uniquely susceptible to PFOA toxicity. This long period of accumulation to high body-burdens in the human has not been directly factored into any risk estimation to date.

3. Risk estimation based on pharmacokinetic models

Based on available kinetic data, body-burden and the long elimination half-life of PFOA in humans are critical in estimating the risk resulting from human exposure to environmental PFOA concentrations. These considerations can be incorporated into quantitative estimates of human health risk through the development of a physiologically-based pharmacokinetic (PBPK) model. Although efforts are underway to develop such a model for PFOA in the rat and human, no such models have been published.

A simplified approach to obtaining the exposure/blood concentration relationship has been taken in the development of a compartmental model for PFOA based on kinetic principles without mechanistic and limited physiological descriptions (Hinderliter and Jepson, 2001). The stated purpose of the model was to provide decision-makers with an easily applied tool to relate PFOA exposure to concentrations in human blood. The model assumes that all PFOA is absorbed immediately and is eliminated from the body with a half-life of 1 year. The utility of using a model such as this is that takes into account the very long half-life of PFOA in humans.

The model is exercised over an exposure period of 6 years, which purportedly allows steady-state to be achieved and for blood concentrations to level out. Yet recent data indicates the PFOA half-life in humans is over 4 years. Thus, by assuming a half-life of one year, the model will substantially underestimate the steady-state blood level. A higher blood level is reached with the longer half-life because steady-state is not attained until around 24 years of accumulation rather than 6 years. The simplified model also assumes that there is no tissue distribution and that all PFOA is contained in the blood.

The model was exercised with various assumptions regarding input of PFOA via inhalation and/or ingestion of drinking water. The output of these calculations was presented as a matrix of blood levels resulting from combinations of air and water PFOA concentrations. As indicated above, the blood level in a female rat that results in renal and developmental toxicity is about 1 ppm. Using the simplified model, one can easily calculate the drinking water concentration that would result in a human blood level of 1 ppm PFOA. The simple model predicts a blood level of

approximately 1 ppm would result from consuming 3 parts-per-billion (ppb) of PFOA in drinking water over a period of 6 years, assuming no PFOA exposure through air or other media. If the model were adjusted to incorporate the more recent estimate of human half-life of 4 years, a substantially lower level of PFOA in drinking water would be needed to reach the 1 ppm in blood level that produced toxicity in the female rat.

In summary, a simplified compartment model exists to estimate the relationship between PFOA drinking water and air concentrations and blood levels in human populations. The model indicates that a level of PFOA in human blood corresponding to level of PFOA in blood that caused renal and developmental toxicity in the female rat would result from the human consumption of water containing less than 3 ppb PFOA.

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