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Andrea V. Malinowski
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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).

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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

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[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

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[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

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[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.

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[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.

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[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).

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[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

AR226_1328

Du Pont Reassigns 50 Women Workers In Chemical Case

* * *

Substance, Produced by 3M, Was Linked During Tests To Birth Defects in Rats

By a WALL STREET JOURNAL Staff Reporter

WILMINGTON—Du Pont Co. said it reassigned about 50 women at its Parkersburg, W.Va., plant after learning that a chemical they have been working with might cause birth defects. The women all are of childbearing age.

The substance, known as FC-143, is a compound used in making resins, film products and coatings. Its manufacturer, Minnesota Mining & Manufacturing Co., warned Du Pont in late March that tests had shown the compound causes birth defects in rats.

"We don't know the significance of this study, but it was felt this was the prudent course of action," said Du Pont's corporate medical director, Dr. Bruce W. Karrh.

The chemical, ammonium perfluorooctanoate, is used at other Du Pont plants. But Dr. Karrh said exposure was considered high enough to warrant removing women workers only at the West Virginia facility and at a plant in Circleville, Ohio, where one woman was reassigned.

Dr. Karrh said FC-143 has been used by the company "for some period of time."

Attachment 1

AR226_1329

THE NEW YORK TIMES, WEDNESDAY, APRIL 8, 1981

50 Women Workers Shifted By Du Pont to Avoid Peril

PARKERSBURG, W. Va., April 7 (AP) — About 50 women were transferred to new jobs at a Du Pont Company plant after a preliminary study indicated that a chemical they were working with might cause birth defects, a company official said.

The chemical, known as FC-143, has been used in plastics for many years, and most has been supplied by the Minnesota Mining and Manufacturing Company, officials said.

"We were advised that FC-143 caused birth defects in the unborn when fed by stomach tube to female rats in a laboratory experiment," said Howard Todd, manager of the plant owned by E. I. du Pont de Nemours & Company.

"This was a preliminary, range-finding study designed to determine dosage limits prior to a full-scale study on FC-143's potential to cause birth defects in rats," he added.

John Bowers, superintendent of employee relations at the company's Washington Works plant near Parkersburg, said that none of the women would lose their jobs or current pay.

Additional laboratory tests are planned by 3M and Du Pont, Mr. Todd said.

Attachment 2

AR226-1330

DUPONT
ESTABLISHED 1802
E. I. DU PONT DE NEMOURS & COMPANY
INCORPORATED
P. O. Box 1217
PARKERSBURG, W. VA. 26101

POLYMER PRODUCTS DEPARTMENT

CC: Jack J. Schra. Regional Adm.,
EPA, Region III
Permit Programs Monitoring Unit,
3EN43MI
6th and Walnut Streets
Philadelphia, Pennsylvania 19106

C. Ronald Sandy, Supervisor
W. Va. Div. of Water Resources
6321 Emerson Avenue
Parkersburg, WV 26101

June 9, 1981

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

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

Dear Sir:

This letter is to inform you of toxicity information we have received from our supplier of the surfactant ammonium perfluorooctanoate, also known as FC-143, which is present in our outfall 005 (permit (WV0001279) in a concentration of about 0.1 mg/L. The 3M Company has advised us that this material has been found to cause defects in the unborn when fed by stomach tubes to female rats in a preliminary laboratory experiment. Du Pont uses FC-143 in the manufacture of fluoropolymer resins.

Much more testing must be conducted to determine the significance of the 3M experiment. As part of the ongoing program to determine the safety of our materials, both Du Pont's Haskell Laboratory and 3M are now planning more detailed experiments. However, we have taken the precaution of reassigning female personnel of childbearing capability to areas outside those in which fluoropolymer resins are manufactured or FC-143 is handled.

At this time, we do not know the significance, if any, of the preliminary animal experiment. FC-143 has been in use for decades without apparent adverse affects in humans.

If you need additional information, please let me know.

Very truly yours,

A. C. Huston
Environmental Control Consultant
Washington Works

ACH:hcw
1306A

Attachment 3

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

POLYMER PRODUCTS DEPARTMENT

CC: Timothy T. Laraway
Hazardous Waste/Ground Water Branch
WV Division of Water Resources
1201 Greenbrier Street
Charleston, WV 25311

June 5, 1985

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Mr. Stephen R. Wassersug, Director
Hazardous Waste Management Division
U.S. Environmental Protection Agency, Region III
6th and Walnut Streets
Philadelphia, Pennsylvania 19106

Re: EPA I.D. No. WVD 04 587 5291

Dear Mr. Wassersug:

U.S. EPA SOLID WASTE MANAGEMENT UNIT SURVEY

The following comments apply to this survey:

The solid waste management units included are those on this site or contiguous to it. This is our understanding based on questions reviewed with Mr. John Potosnak.

Records are not available for the early days of plant operation so we have relied on discussions with longer service employees and on aerial photographs. Earliest aerial photos are for 1960 although plant operation started in 1948.

One difficult aspect of the survey is providing engineering prints on the solid waste management units. As a result of process and equipment changes made over the years, drawings do not always accurately reflect the units in place today. The prints provided--although limited in accuracy--are the most appropriate available.

The topographical prints provided are planimetrics which are computer-prepared based on a 1983 aerial photo.

In our operations many plastics are reused or reworked under conditions which could be interpreted as "recycling." The number of accumulation or storage areas for these plastics on this site is considerable. We consider this reuse as an integral part of the process and have not, as a result, included these locations as solid waste management units in this survey. None of these plastics would be hazardous under RCRA regulations.

I certify under penalty of law that this document and all attachments were prepared under my direction or supervision in accordance with a system designed to assure that qualified personnel properly gather and evaluate the information submitted. Based on my inquiry of the person or persons who manage the system, or those persons directly responsible for gathering the information, the information submitted is, to be the best of my knowledge and belief, true, accurate, and complete. I am aware that there are significant penalties for submitting false information, including the possibility of fine and imprisonment for knowing violations.

Very truly yours,

H. V. Bradley

H. V. Bradley
Works Manager

HVB:hcw
Attachment
0290H

B-4 ANAEROBIC DIGESTION PONDS (CONT'D.)

Redacted

Releases, Spills, etc.

Prior to 1969 the lower river bank level on which the containment basins are located would flood periodically with inundation of the ponds. This has not occurred since installation of the Belleville Dam. When the third basin was constructed, leakage of surfactant was detected to the river which led to relining of the units. The FC-143 surfactant has also been detected in the aquifer at part per billion levels - just above the analytical detection limit.

E-47 REV. 6-82

DUPONT

ESTABLISHED 1802

E. I. DU PONT DE NEMOURS & COMPANY

INCORPORATED

P.O. Box 1217

PARKERSBURG, W. VA. 26102

POLYMER PRODUCTS DEPARTMENT

cc: B. Douglas Steele, Ph.D.,
Chief
West Virginia Department
of Natural Resources
1260 Greenbrier Street
Charleston, WV 25311

Mr. G. Dale Farley, Director
West Virginia Air Pollution
Control Commission
1558 Washington Street, East
Charleston, WV 25311

February 9, 1990

CERTIFIED MAIL -
RETURN RECEIPT REQUESTED

Mr. Stephen R. Wassersug, Director
RCRA Programs Branch (3HW50)
U. S. Environmental Protection Agency
Region III
841 Chestnut Building
Philadelphia, PA 19107

Dear Mr. Wassersug:

RE: Permit WVD 04 587 5291

Attached are three copies of the submittals required within 60 days of the effective date of the referenced permit. Attachment I is the Verification Investigation Work Plan required for the six Solid Waste Management Units listed in Part II, Section A. Attachment II is the information for existing ground-water monitoring wells required in Part II, Section B, 4. Attachment III is the detailed waste management plan on the hazardous waste incinerator required by Part III, 3.

If you have any questions , please feel free to contact me or our environmental coordinator, A. C. Huston, on 304-863-4271. Thank you.

Very truly yours,

H. V. Bradley

H. V. Bradley
Works Manager

— Redacted —

Releases have occurred in the past. Flooding occasionally inundated the impoundments prior to construction of the Belleville Dam in 1964. No flooding has occurred since the construction of the dam. The impoundments were re-lined with a 6 to 12-inch layer of bentonite to reduce the potential of infiltration to the groundwater in 1973-74. The Lubeck public supply wells have detectable levels (ppb) of ammonium perfluorooctanoate (also called C-8). Washington Works is in the process of purchasing these wells from Lubeck Water supply.

— Redacted —

AR226_1333

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAY 15 1992

ASSISTANT ADMINISTRATOR
FOR ENFORCEMENT AND
COMPLIANCE ASSURANCE

Certified Mail
Return Receipt Requested

Andrea Malinowski (D-8078-1)
E.I. du Pont de Nemours and Co.
CAP ID#: 8ECAP-0025
1007 Market Street
Wilmington, DE 19898

Dear CAP Participant:

On September 30, 1991, EPA announced in the Federal Register an extension of the TSCA Section 8(e) CAP reporting deadline for submission of information regarding release of chemical substances to and detection of chemical substances in environmental media. This announcement established a Phase Two of the CAP for section 8(e) information on the release of chemical substances to and the detection of chemical substances in environmental media and environmental toxicity data for plant effluents. All TSCA Section 8(e) CAP submissions under Phase 2 were to be delivered to EPA no later than six months after EPA publishes final revised environmental guidance ("guidance"). The exact date would appear in the Federal Register notice announcing the revised guidance.

On January 30, 1992, EPA provided CAP participants with an "Addendum to CAP Agreement" and policy statements that formally established the Two Phases to the CAP, and permitted the submission of the following information during Phase Two:

- o information on the release of chemical substances to and detection of chemical substances in environmental media, and

- o environmental toxicity testing performed on plant effluents.

The deadline for reporting all other information under the TSCA section 8(e) Compliance Audit Program remained unchanged at February 28, 1992 unless otherwise extended. The Addendum was to be executed by the Regulatee and returned to EPA for ratification and entry.

Since ratification of the Addendum, EPA has twice issued, for notice and comment, revised draft reporting guidance. After review of extensive comments, EPA has 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 detection of chemical substances in environmental media; or environmental toxicity data on plant effluents that predate the effective date of the guidance will not be the subject of an EPA TSCA Section 8(e) enforcement action. We are aware that some CAP participants may have submitted this data under Phase 1 of the CAP program. Accordingly, penalties will not be assessed for any Phase 2 type studies or reports submitted under the TSCA Section 8(e) CAP as TSCA Section 8(e) data. To effectuate this decision it is necessary to revise the previously ratified Addendum, and modify the Registration and Agreement for TSCA Section 8(e) Compliance Audit Program. The attached Revised Addendum to the CAP Agreement supersedes the previous Addendum and specifies the following:

- The Regulatee no longer is required to conduct a file search for information on the release of chemical substances to and detection of chemical substances in environmental media, or for environmental toxicity data on plant effluents.

- A second Final Report is no longer necessary. Therefore, the first Final Report becomes the controlling document described in Unit II.A.8. of the CAP Agreement.

Please return the signed Revised Addendum to the CAP Agreement within thirty (30) calendar days of your receipt of this letter to:

Brian T. Dyer
ATTN.: TSCA 8(e) CAP
U.S. Environmental Protection Agency
Toxics and Pesticides Enforcement Division, 2245A
401 M Street, S.W.
Washington, D.C. 20460

Upon receipt, EPA will: (1) sign the Revised Addendum: (2) place it in your file; (3) forward a copy to you with the Consent Agreement and Consent Order; and (4) stamp the previously ratified Addendum as null and void.

If you have any questions concerning the CAP or the Revised Addendum for the CAP Agreement, please contact Brian Dyer, of my staff, at (202) 564-4166.

Sincerely,

Jesse Baskerville, Director
Toxics and Pesticides Enforcement Division

Enclosure

Attachment 6

Revised Addendum to the TSCA Section 8(e) CAP Agreement

This Revised Addendum supersedes the "Addendum to the CAP Agreement" and modifies the Registration and Agreement for TSCA Section 8(e) Compliance Audit Program as follows:

I. The TSCA Section 8(e) Compliance Audit Program, which the Regulatee agreed to conduct in the Registration requirement I.A. does not include: information on the release of chemical substances to and detection of chemical substances in environmental media; or environmental toxicity data on plant effluents. The Regulatee, therefore, is no longer required to conduct a file search for this information. Further, footnote 1 of the Agreement pertains solely to chemical release and detection information and therefore, is no longer applicable to the administration of the TSCA Section 8(e) Compliance Audit Program.

II. The first Final Report shall be considered the Final Report and controlling document, as described in Unit II.A.8, for purposes of determining the information listed or submitted under the TSCA Section 8(e) Compliance Audit Program. The first Final Report must have been submitted no later than February 28, 1992, unless an extension had been granted pursuant to Unit I.E. of the CAP Agreement.

III. EPA intends to publish final revised guidance in the Federal Register on reporting information on the release of chemical substances to and detection of chemical substances in environmental media. EPA also intends to publish a question and answer document to illustrate application of the guidance. The final revised guidance will not be effective prior to EPA's publication of the question and answer document.

IV. Impact of the final revised guidance on:

A. Information on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents, that predates the effective date of the final revised guidance will not be the subject of an EPA TSCA section 8(e) penalty enforcement action.

B. Information on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents, that may have been submitted under Phase 1 of the CAP Program will not result in the assessment of penalties for such studies or reports submitted under this TSCA Section 8(e) Compliance Audit Program.

V. Information generated after the effective date of new final revised guidance on the release of chemical substances to and detection of chemical substances in environmental media, or environmental toxicity data for plant effluents, will be submitted prospectively pursuant to TSCA Section 8(e) and the new final revised guidance, not the CAP Agreement. Therefore, no penalty will accrue under the CAP Agreement for the submission of such information.

WE AGREE TO THIS:

For EPA:

Jesse Baskerville, Director
Toxics and Pesticides
Enforcement Division

For Regulatee:

Paul V. Tebo

E. I. du Pont de Nemours and Company
CAP ID: 8ECAP-0025
By: Paul V. Tebo
Title: Vice President
DuPont Safety, Health and
Environment