

1



LEGAL
Wilmington, Delaware 19898

Contains No CBI

15

88 9200 10570

No CBI

BEHQ-92-12361

October 16, 1992

**Certified Mail
Return Receipt Requested**

Document Processing Center (TS-790)
Office of Pollution Prevention and Toxics
Environmental Protection Agency
401 M Street., S.W.
Washington, D.C. 20460
Attn: Section 8(e) Coordinator (CAP Agreement)

Dear Coordinator:

8ECAP-0025

On behalf of the Regulatee and pursuant to Unit II B.1.b. and Unit II C of the 6/28/91 CAP Agreement, E.I. Du Pont de Nemours and Co. hereby submits (*in triplicate*) the attached studies. Submission of this information is voluntary and is occasioned by unilateral changes in EPA's standard as to what EPA now considers as reportable information. Regulatee's submission of information is made solely in response to the new EPA §8(e) reporting standards and is not an admission: (1) of TSCA violation or liability; (2) that Regulatee's activities with the study compounds reasonably support a conclusion of substantial health or environmental risk or (3) that the studies themselves reasonably support a conclusion of substantial health or environmental risk.

The "Reporting Guide" creates new TSCA 8(e) reporting criteria which were not previously announced by EPA in its 1978 Statement of Interpretation and Enforcement Policy, 43 Fed Reg 11110 (March 16, 1978). The "Reporting Guide states criteria which expands upon and conflicts with the 1978 Statement of Interpretation. Absent amendment of the Statement of Interpretation, the informal issuance of the "Reporting Guide" raises significant due processes issues and clouds the appropriate reporting standard by which regulated persons can assure TSCA Section 8(e) compliance.

For Regulatee,

Mark H. Christman
Counsel
Legal D-7158
1007 Market Street
Wilmington, DE 19898
(302) 774-6443

ATTACHMENT 1

Submission of information is made under the 6/28/91 CAP Agreement, Unit II. This submission is made voluntarily and is occasioned by recent changes in EPA's TSCA §8(e) reporting standard; such changes made, for the first time in 1991 and 1992 without prior notice and in violation of Regulatee's constitutional due process rights. Regulatee's submission of information under this changed standard is not a waiver of its due process rights; an admission of TSCA violation or liability, or an admission that Regulatee's activities with the study compounds reasonably support a conclusion of substantial risk to health or to the environment. Regulatee has historically relied in good faith upon the 1978 Statement of Interpretation and Enforcement Policy criteria for determining whether study information is reportable under TSCA §8(e), 43 Fed Reg 11110 (March 16, 1978). EPA has not, to date, amended this Statement of Interpretation.

After CAP registration, EPA provided the Regulatee the June 1, 1991 "TSCA Section 8(e) Reporting Guide". This "Guide" has been further amended by EPA, EPA letter, April 10, 1992. EPA has not indicated that the "Reporting Guide" or the April 1992 amendment supersedes the 1978 Statement of Interpretation. The "Reporting Guide" and April 1992 amendment substantively lowers the Statement of Interpretation's TSCA §8(e) reporting standard². This is particularly troublesome as the "Reporting Guide" states criteria, applied retroactively, which expands upon and conflicts with the Statement of Interpretation.³ Absent amendment of the Statement of Interpretation, the informal issuance of the "Reporting Guide" and the April 1992 amendment clouds the appropriate standard by which regulated persons must assess information for purposes of TSCA §8(e).

²In sharp contrast to the Agency's 1977 and 1978 actions to soliciting public comment on the proposed and final §8(e) Policy, EPA has unilaterally pronounced §8(e) substantive reporting criteria in the 1991 Section 8(e) Guide without public notice and comment, See 42 Fed Reg 45362 (9/9/77), "Notification of Substantial Risk under Section 8(e): Proposed Guidance".

³A comparison of the 1978 Statement of Interpretation and the 1992 "Reporting Guide" is appended.

Throughout the CAP, EPA has mischaracterized the 1991 guidance as reflecting "longstanding" EPA policy concerning the standards by which toxicity information should be reviewed for purposes of §8(e) compliance. Regulatee recognizes that experience with the 1978 Statement of Interpretation may cause a review of its criteria. Regulatee supports and has no objection to the Agency's amending reporting criteria *provided that* such amendment is not applied to the regulated community in an unfair way. However, with the unilateral announcement of the CAP under the auspices of an OCM enforcement proceeding, EPA has wrought a terrific unfairness since much of the criteria EPA has espoused in the June 1991 Reporting Guide and in the Agency's April 2, 1992 amendment is new criteria which does not exist in the 1978 Statement of Interpretation and Enforcement Policy.

The following examples of new criteria contained in the "Reporting Guide" that is not contained in the Statement of Interpretation follow:

- o even though EPA expressly disclaims each "status report" as being preliminary evaluations that should not be regarded as final EPA policy or intent⁴, the "Reporting Guide" gives the "status reports" great weight as "sound and adequate basis" from which to determine mandatory reporting obligations. ("Guide" at page 20).
- o the "Reporting Guide" contains a matrix that establishes new numerical reporting "cutoff" concentrations for acute lethality information ("Guide" at p. 31). Neither this matrix nor the cutoff values therein are contained in the Statement of Interpretation. The regulated community was not made aware of these cutoff values prior to issuance of the "Reporting Guide" in June, 1991.
- o the "Reporting Guide" states new specific definitional criteria with which the Agency, for the first time, defines as 'distinguishable neurotoxicological effects'; such criteria/guidance not expressed in the 1978 Statement of Interpretation.⁵

othe "Reporting Guide" provides new review/ reporting criteria for irritation and sensitization studies; such criteria not previously found in the 1978 Statement of Interpretation/Enforcement Policy.

othe "Reporting Guide" publicizes certain EPA Q/A criteria issued to the Monsanto Co. in 1989 which are not in the Statement of Interpretation; have never been published in the Federal Register or distributed by the EPA to the Regulatee. Such Q/A establishes new reporting criteria not previously found in the 1978 Statement of Interpretation/Enforcement Policy.

⁴The 'status reports' address the significance, if any, of particular information reported to the Agency, rather than stating EPA's interpretation of §8(e) reporting criteria. In the infrequent instances in which the status reports contain discussion of reportability, the analysis is invariably quite limited, without substantial supporting scientific or legal rationale.

⁵ See, e.g., 10/2/91 letter from Du Pont to EPA regarding the definition of 'serious and prolonged effects' as this term may relate to transient anesthetic effects observed at lethal levels; 10/1/91 letter from the American Petroleum Institute to EPA regarding clarification of the Reporting Guide criteria.

In discharging its responsibilities, an administrative agency must give the regulated community fair and adequate warning to as what constitutes noncompliance for which penalties may be assessed.

Among the myriad applications of the due process clause is the fundamental principle that statutes and regulations which purport to govern conduct must give an adequate warning of what they command or forbid.... Even a regulation which governs purely economic or commercial activities, if its violation can engender penalties, must be so framed as to provide a constitutionally adequate warning to those whose activities are governed.

Diebold, Inc. v. Marshall, 585 F.2d 1327, 1335-36 (D.C. Cir. 1978). See also, Rollins Environmental Services (NJ) Inc. v. U.S. Environmental Protection Agency, 937 F. 2d 649 (D.C. Cir. 1991).

While neither the are rules, This principle has been applied to hold that agency 'clarification', such as the Statement of Interpretation, the "Reporting Guide" nor the April 1992 amendments will not applied retroactively.

...a federal court will not retroactively apply an unforeseeable interpretation of an administrative regulation to the detriment of a regulated party on the theory that the post hoc interpretation asserted by the Agency is generally consistent with the policies underlying the Agency's regulatory program, when the semantic meaning of the regulations, as previously drafted and construed by the appropriate agency, does not support the interpretation which that agency urges upon the court.

Standard Oil Co. v. Federal Energy Administration, 453 F. Supp. 203, 240 (N.D. Ohio 1978), aff'd sub nom. Standard Oil Co. v. Department of Energy, 596 F.2d 1029 (Em. App. 1978):

The 1978 Statement of Interpretation does not provide adequate notice of, and indeed conflicts with, the Agency's current position at §8(e) requires reporting of all 'positive' toxicological findings without regard to an assessment of their relevance to human health. In accordance with the statute, EPA's 1978 Statement of Interpretation requires the regulated community to use scientific judgment to evaluate the significance of toxicological findings and to determining whether they reasonably support a conclusion of a substantial risk. Part V of the Statement of Interpretation urges persons to consider "the fact or probability" of an effect's occurrence. Similarly, the 1978 Statement of Interpretation stresses that an animal study is reportable only when "it contains reliable evidence ascribing the effect to the chemical." 43 Fed Reg. at 11112. Moreover, EPA's Statement of Interpretation defines the substantiality of risk as a function of both the seriousness of the effect and the probability of its occurrence. 43 Fed Reg 11110 (1978). Earlier Agency interpretation also emphasized the "substantial" nature of a §8(e) determination. See 42 Fed Reg 45362, 45363

(1977). [Section 8(e) findings require "extraordinary exposure to a chemical substance...which critically imperil human health or the environment"].

The recently issued "Reporting Guide" and April 1992 Amendment guidance requires reporting beyond and inconsistent with that required by the Statement of Interpretation. Given the statute and the Statement of Interpretation's explicit focus on substantial human or environmental risk, whether a substance poses a "substantial risk" of injury requires the application of scientific judgment to the available data on a case-by-case basis.

If an overall weight-of-evidence analysis indicates that this classification is unwarranted, reporting should be unnecessary under §8(e) because the available data will not "reasonably support the conclusion" that the chemical presents a substantial risk of serious adverse consequences to human health.

Neither the legislative history of §8(e) nor the plain meaning of the statute support EPA's recent lowering of the reporting threshold that TSCA §8(e) was intended to be a sweeping information gathering mechanism. In introducing the new version of the toxic substances legislation, Representative Eckhart included for the record discussion of the specific changes from the version of H. R. 10318 reported by the Consumer Protection and Finance Subcommittee in December 1975. One of these changes was to modify the standard for reporting under §8(e). The standard in the House version was changed from "causes or contributes to an unreasonable risk" to "causes or significantly contributes to a substantial risk". This particular change was one of several made in TSCA §8 to avoid placing an undue burden on the regulated community. The final changes to focus the scope of Section 8(e) were made in the version reported by the Conference Committee.

The word "substantial" means "considerable in importance, value, degree, amount or extent". Therefore, as generally understood, a "substantial risk" is one which will affect a considerable number of people or portion of the environment, will cause serious injury and is based on reasonably sound scientific analysis or data. Support for the interpretation can be found in a similar provision in the Consumer Product Safety Act. Section 15 of the CPSA defines a "substantial product hazard" to be:

"a product defect which because of the pattern of defect, the number of defective products distributed in commerce, the severity of the risk, or otherwise, creates a substantial risk of injury to the public."

Similarly, EPA has interpreted the word 'substantial' as a quantitative measurement. Thus, a 'substantial risk' is a risk that can be quantified, See, 56 Fed Reg 32292, 32297 (7/15/91). Finally, since information pertinent to the exposure of humans or the environment to chemical substances or mixtures may be obtained by EPA through Sections 8(a) and 8(d) regardless of the degree of potential risk, §8(e) has specialized function. Consequently, information subject to §8(e) reporting should be of a type which would lead a reasonable man to conclude that some type action was required immediately to prevent injury to health or the environment.

Attachment

Comparison:

Reporting triggers found in the 1978 "Statement of Interpretation/ Enforcement Policy", 43 Fed Reg 11110 (3/16/78) and the June 1991 *Section 8(e) Guide*.

<u>TEST TYPE</u>	<u>1978 POLICY</u> <u>CRITERIA EXIST?</u>	<u>New 1991 GUIDE</u> <u>CRITERIA EXIST?</u>
ACUTE LETHALITY		
Oral	N}	Y}
Dermal	N}	Y}
Inhalation (Vapors)	} ⁶	} ⁷
aerosol	N}	Y}
dusts/ particles	N}	Y}
SKIN IRRITATION	N	Y ⁸
SKIN SENSITIZATION (ANIMALS)	N	Y ⁹
EYE IRRITATION	N	Y ¹⁰
SUBCHRONIC (ORAL/DERMAL/INHALATION)	N	Y ¹¹
REPRODUCTION STUDY	N	Y ¹²
DEVELOPMENTAL TOX	Y ¹³	Y ¹⁴

⁶43 Fed Reg at 11114, comment 14:

"This policy statements directs the reporting of specific effects when unknown to the Administrator. Many routine tests are based on a knowledge of toxicity associated with a chemical. Unknown effects occurring during such a range test may have to be reported if they are those of concern to the Agency and if the information meets the criteria set forth in Parts V and VII."

⁷Guide at pp.22, 29-31.

⁸Guide at pp-34-36.

⁹Guide at pp-34-36.

¹⁰Guide at pp-34-36.

¹¹Guide at pp-22; 36-37.

¹²Guide at pp-22

¹³43 Fed Reg at 11112

"Birth Defects" listed.

¹⁴Guide at pp-22

NEUROTOXICITY	N	Y ¹⁵
CARCINOGENICITY	Y ¹⁶	Y ¹⁷
MUTAGENICITY		
<i>In Vitro</i>	Y ¹⁸	Y ¹⁹
<i>In Vivo</i>	Y}	Y}
ENVIRONMENTAL		
Bioaccumulation	Y}	N
Bioconcentration	Y} ²⁰	N
Oct/water Part. Coeff.	Y}	N
Acute Fish	N	N
Acute Daphnia	N	N
Subchronic Fish	N	N
Subchronic Daphnia	N	N
Chronic Fish	N	N
AVIAN		
Acute	N	N
Reproductive	N	N
Reproductive	N	N

¹⁵Guide at pp-23; 33-34.

¹⁶43 Fed Reg at 11112
"Cancer" listed

¹⁷Guide at pp-21.

¹⁸43 Fed Reg at 11112; 11115 at Comment 15

"Mutagenicity" listed/ *in vivo* vs *invitro* discussed; discussion of "Ames test".

¹⁹Guide at pp-23.

²⁰43 Fed Reg at 11112; 11115 at Comment 16.

CAS # 13098-39-0

Chem: Hexafluoroacetone

Title: Inhalation Studies on Hexafluoroacetone

Date: 1-25-65

Summary of Effects: Testicular effects

INHALATION STUDIES ON HEXAFLUOROACETONE

Part II

A. The Lethality of Short-Term (< 1 hr.) Exposures

B. The Persistence of Tissue Effects

Medical Research Project No. 627

Report No. 6-65

INTRODUCTION

Previous work at Haskell Laboratory (Report No. 46-62) showed the Approximate Lethal Concentration of hexafluoroacetone gas (HFA) for rats to be 300 ppm for a single exposure of four hours. Repeated exposures at a nominal concentration of 60 ppm were not lethal. Clinically, they produced inactivity, deep respiration, small conjunctival hemorrhage and progressive weight loss. Pathological examination showed marked cumulative injury to the testes. Other organs were affected, but the action of the compound on the testes was pre-dominant, if not specific. The work presented here was designed to evaluate further two aspects of the toxicity. Part I was designed to simulate possible short exposures experienced by plant personnel and Part II was designed to follow the postexposure course of the injury to the rat testes. Part II was run at a level which earlier experiments had shown to be a testicular effect level.

MATERIAL

The HFA used for these studies was supplied in a cylinder by D. G. Coe under the Jackson Laboratory code FPS-310. It was given the Haskell Laboratory No. 3447.

PROCEDURE

Part I. Short Exposures

Hexafluoroacetone gas was metered at a known rate by a motor-driven calibrated syringe into a measured air stream. The air stream with its load of gas was passed through a bell jar containing four male rats of ChR-CD strain having initial body weight of 235-327 grams. Exposure times were 30 minutes at concentration X or, in one exposure, 15 minutes at concentration 2X. An approximation known as "Haber's Law", states that, within reasonable time limits, the product of concentration and time of exposure is a constant with regard to a particular biological effect. Although this rule does not always apply, it provided a basis for setting exposure conditions in the present study.

Part II. Postexposure Course of Testicular Effect

The gas was metered into a dry measured air stream, as previously described. The gaseous mixture, at a nominal concentration of 200 ppm, was passed into a 32-liter exposure chamber for four hours. Twelve male rats of ChR-CD strain were placed within the chamber for the exposure. The animals were compartmented by means of a wire rack. The average weight of the test group was

TABLE I

<u>Nominal Concentration</u> (ppm)	<u>Ct*</u> (ppm-hrs)	<u>Air Flow</u> (lit./min.)	<u>Period of Exposure</u> (hrs:min.)	<u>Mortality</u> Ratio	<u>Fate**</u>
6000	3000	2.64	0:30	4/4	All found dead 1-4 days after exposure.
4800	2400	3.30	0:30	3/4	Three found dead 4-6 days after exposure; survivor killed 16 days later.
9600	2400	4.09	0:15	3/4	Three found dead 1-2 days after exposure survivor killed 14 days later
3600	1800	2.25	0:30	0/4	All killed 14 days after exposure.
2400	1200	3.37	0:30	0/4	All killed 14 days after exposure.

* The Ct of the 4-hour ALC is 1200 ppm-hrs.

** All rats examined for pathological signs after death.

U.S. GOVERNMENT PRINTING OFFICE: 1964 O 454-000

257 grams (245-274 grams). For control purposes, a similar set-up was used with air being passed through the chamber at the same flow rate as the test group. The average weight of the control group was 256 grams (245-274 grams). For pathological examination, three animals each from the control and test groups were selected at random for sacrifice at each of 7, 14, 28, and 57 days following exposure. Histopathology was done on the testes, brain, lung, liver, kidney, and thymus of the test group and on the testes of the control animals.

EXPERIMENTAL RESULTS

Part I. Short Exposures

A. Clinical

The conditions and concentrations of exposure with the mortality rates produced are given in Table I. At all concentrations, the animals showed lacrimation, salivation, fluid around the nose, intermittent gasping, and inactivity during exposure. Animals exposed to lethal concentrations showed, in addition, cyanosis of the limbs and deep respiration. Following lethal exposures, the rats exhibited conjunctival hemorrhage and/or pigmented secretion around the eyes for 1-5 days and weakness of body and leg muscles for 1-6 days. One rat from the 4800 ppm exposure and one at the 9600 ppm level developed temporary corneal opacity two and nine days after exposure, respectively.

At sublethal concentrations respiration was slightly deep during exposure. Following exposures, the rats showed weakness of body and leg muscles for 1-3 days, the characteristic pigmented secretion around the eyes was evident in only one animal (3600 ppm). Following exposures at all concentrations, the rats lost weight. Those animals receiving nonlethal concentrations lost weight for only 2-3 days. Exposure to lethal concentrations produced weight loss which was progressive until death. Each of the two survivors of lethal concentrations lost weight for seven and 14 days, respectively.

B. Histology

The most striking effect of lethal and nonlethal exposures was a marked degeneration and necrosis of the germinal cells in the testis. The resultant atrophy of the testis was noted grossly as a significant reduction of weight of this organ. In addition, lung and thymus changes were still evident in the animals killed 14 days after exposure to the lowest nonlethal dose, i.e., 2400 ppm. Hemorrhage and depletion of blood-forming cells in the bone marrow of one rat suggest that hexafluoroacetone may affect the hemopoietic system as did hexafluoroisopropanol (Haskell Laboratory Report No. 2-65).

Part II. Postexposure Course of Testicular Effect

A. Clinical

During exposure the animals of the test group showed slightly deep respiration, lacrimation, salivation, and redness of the ears. Following exposure five of these animals showed slight to moderate pigmented secretion around the eyes for 1-7 days. All members of the test group exhibited weight losses for 1-3 days following exposure and one animal continued to lose weight for

TABLE II

	Days Following Exposure							
	7		14		28		57	
	Test	Control	Test	Control	Test	Control	Test	Control
Average Final Body Weight (gm)	223	304	321	352	392	434	504	532
Average Testes Weight (gm)	1.79	2.91	1.81	3.09	1.78	3.38	2.17	3.68
Average Ratio of Testes Weight to Body Weight (x 100)	0.789	0.959	0.569	0.877	0.456	0.782	0.430	0.694
% Decrease in Ratio From Previous Sacrifice Value	-	-	28	8.3	20	11	5.7	11

seven days. Weight gain thereafter was normal, but at sacrifice the average weight of the test group remained 20-40 grams below that of the controls. There were no clinical signs of toxicity after seven days postexposure.

Table II gives the average testes weights and their ratio to average body weight as obtained at necropsy.

The effect on the testes may have undergone some remission over the course of the 57-day recovery period, as is shown in the last line of Table II. There was a large decrease in the ratio of testicular weight to body weight at the 14-day and 28-day sacrifice periods in the test animals. However, the decrease was slightly less at the 28-day sacrifice. At the 57-day sacrifice, the per cent decrease in the ratio was less than that for the controls, suggesting that there might be some recovery in the testes.

B. Histology

The apparent improvement in the testes shown grossly during the hold period was borne out by microscopic examination of the testes. Microscopic examination revealed regeneration of some spermatogenic tubules by 57 days after exposure. However, even at 57 days postexposure, there were still some tubules which contained no germinal cells. This suggests that the more seriously affected tubules did not regenerate.

As with hexafluoroisopropanol, testicular interstitial hyperplasia and thymus effects were also observed.

SUMMARY AND CONCLUSIONS

Two additional inhalation experiments have been carried out with hexafluoroacetone. In one, short-term high-level exposures such as might occur in a plant were simulated. In another experiment, the postexposure course of the rat testicular change was followed for 57 days. These experiments have confirmed the high toxicity of this compound by inhalation even for periods of 1/4 - 1/2 hour. However, a slightly higher Ct was required for lethality in exposures of less than one hour than was required for exposures of four hours. Nevertheless, Ct values of 1200 to ca. 2000 ppm-hrs caused fatalities in rats for all exposure times used. These exposures also showed that hexafluoroacetone gas can cause corneal opacity in rats. They also indicate that the testicular effect caused by inhalation of hexafluoroacetone is slow to heal and that some of the testicular tubules may be permanently damaged.

RECOMMENDATIONS

In view of these findings and the potential for eye opacity and skin irritation already reported for the sesquihydrate (Haskell Laboratory Report No. 54-63), the following precautions are recommended in handling hexafluoroacetone gas:

1. It should be handled only by properly instructed personnel and in well ventilated areas. Inhalation should be avoided and respiratory protection should be available in case of leaks.

2. Safety glasses with side shields are the minimum eye protection that should be worn when working with this compound.
3. Hexafluoroacetone contacting the skin or eyes should be flushed off immediately with large amounts of water and medical attention obtained immediately.

HASKELL LABORATORY FOR TOXICOLOGY
AND INDUSTRIAL MEDICINE

Report by: Carlton H. Tappan
Carlton H. Tappan

Richard S. Waritz
Richard S. Waritz
Chief, Inhalation Toxicity Section

Approved for Pathology by: E. F. Stula
Edwin F. Stula
Chief, Pathology Section

Approved by: J. Wesley Clayton, Jr.
J. Wesley Clayton, Jr.
Assistant Director

CHT/RSW/mfs
Report No. 6-65
January 25, 1965



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

Mark H. Christman
Counsel
E. I. Du Pont De Nemours and Company
Legal D-7010-1
1007 Market Street
Wilmington, Delaware 19898

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

APR 18 1995

EPA acknowledges the receipt of information submitted by your organization under Section 8(e) of the Toxic Substances Control Act (TSCA). For your reference, copies of the first page(s) of your submission(s) are enclosed and display the TSCA §8(e) Document Control Number (e.g., 8EHQ-00-0000) assigned by EPA to your submission(s). Please cite the assigned 8(e) number when submitting follow-up or supplemental information and refer to the reverse side of this page for "EPA Information Requests".

All TSCA 8(e) submissions are placed in the public files unless confidentiality is claimed according to the procedures outlined in Part X of EPA's TSCA §8(e) policy statement (43 FR 11110, March 16, 1978). Confidential submissions received pursuant to the TSCA §8(e) Compliance Audit Program (CAP) should already contain information supporting confidentiality claims. This information is required and should be submitted if not done so previously. To substantiate claims, submit responses to the questions in the enclosure "Support Information for Confidentiality Claims". This same enclosure is used to support confidentiality claims for non-CAP submissions.

Please address any further correspondence with the Agency related to this TSCA 8(e) submission to:

Document Processing Center (7407)
Attn: TSCA Section 8(e) Coordinator
Office of Pollution Prevention and Toxics
U.S. Environmental Protection Agency
Washington, D.C. 20460-0001

EPA looks forward to continued cooperation with your organization in its ongoing efforts to evaluate and manage potential risks posed by chemicals to health and the environment.

Sincerely,

Terry R. O'Bryan

Terry R. O'Bryan
Risk Analysis Branch

Enclosure

12361A



Recycled/Recyclable
Printed with Soy/Candela Ink on paper that
contains at least 50% recycled fiber

Triage of 8(e) Submissions

Date sent to triage: _____

NON-CAP

CAP

Submission number: 12361A
~~12361A~~

TSCA Inventory: Y N D

Study type (circle appropriate):

Group 1 - Dick Clements (1 copy total)

ECO AQUATO

Group 2 - Ernie Falke (1 copy total)

ATOX SBTOX SEN w/NEUR

Group 3 - Elizabeth Margosches (i copy each)

STOX CTOX EPI RTOX GTOX
STOX/ONCO CTOX/ONCO IMMUNO CYTO NEUR

Other (FATE, EXPO, MET, etc.): _____

Notes:

THIS IS THE ORIGINAL 8(e) SUBMISSION; PLEASE REFILE AFTER TRIAGE DATABASE ENTRY

For Contractor Use Only

entire document: 0 1 2 pages 1, 1st TAB pages 1, TABS

Notes:

Contractor reviewer: PDR Date: 3/21/95

12361

CECATS/TRIAGE TRACKING DBASE ENTRY FORM

CECATS DATA:

Submission # BEHQ- 1092 - ~~XXXXXX~~ SEQ. A

TYPE: INT. SUPP FLWP

SUBMITTER NAME: E. I. Dupont de Nemours and Company

INFORMATION REQUESTED: FLWP DATE:

- 0501 NO INFO REQUESTED
- 0502 INFO REQUESTED (TECH)
- 0503 INFO REQUESTED (VOL ACTIONS)
- 0504 INFO REQUESTED (REPORTING RATIONALE)

DISPOSITION:

- 0639 REFER TO CHEMICAL SCREENING
- 0678 CAP NOTICE

VOLUNTARY ACTIONS:

- 0401 NO ACTION REPORTED
- 0402 STUDIES PLANNED/IN PROGRESS
- 0403 NOTIFICATION OF WORKER RIGHTS
- 0404 LABEL/MSDS CHANGES
- 0405 PROCESS/HANDLING CHANGES
- 0406 APP/USE DISCONTINUED
- 0407 PRODUCTION DISCONTINUED
- 0408 CONFIDENTIAL

SUB. DATE: 10/16/92 OTS DATE: 10/27/92 CSRAD DATE: 02/16/95

CHEMICAL NAME:

Hexa Fluoro acetone

CASE#

13098-39-0

INFORMATION TYPE:	P F C	INFORMATION TYPE:	P F C	INFORMATION TYPE:	P F C
0201 ONCO (HUMAN)	01 02 04	0216 EPI/CLIN	01 02 04	0241 IMMUNO (ANIMAL)	01 02 04
0202 ONCO (ANIMAL)	01 02 04	0217 HUMAN EXPOS (PROD CONTAM)	01 02 04	0242 IMMUNO (HUMAN)	01 02 04
0203 CELL TRANS (IN VITRO)	01 02 04	0218 HUMAN EXPOS (ACCIDENTAL)	01 02 04	0243 CHEM/PHYS PROP	01 02 04
0204 MUTA (IN VITRO)	01 02 04	0219 HUMAN EXPOS (MONITORING)	01 02 04	0244 CLASTO (IN VITRO)	01 02 04
0205 MUTA (IN VIVO)	01 02 04	0220 ECO/AQUA TOX	01 02 04	0245 CLASTO (ANIMAL)	01 02 04
0206 REPRO/TERATO (HUMAN)	01 02 04	0221 ENV. OCC/REL/FATE	01 02 04	0246 CLASTO (HUMAN)	01 02 04
<u>0207</u> REPRO/TERATO (ANIMAL)	<u>01 02 04</u>	0222 EMER INCI OF ENV CONTAM	01 02 04	0247 DNA DAM/REPAIR	01 02 04
0208 NEURO (HUMAN)	01 02 04	0223 RESPONSE REQEST DELAY	01 02 04	0248 PROD/USE/PROC	01 02 04
0209 NEURO (ANIMAL)	01 02 04	0224 PROD/COMP/CHEM ID	01 02 04	0251 MSDS	01 02 04
0210 ACUTE TOX. (HUMAN)	01 02 04	0225 REPORTING RATIONALE	01 02 04	0299 OTHER	01 02 04
0211 CHR. TOX. (HUMAN)	01 02 04	0226 CONFIDENTIAL	01 02 04		
<u>0212</u> ACUTE TOX. (ANIMAL)	<u>01 02 04</u>	0227 ALLERG (HUMAN)	01 02 04		
0213 SUB ACUTE TOX (ANIMAL)	01 02 04	0228 ALLERG (ANIMAL)	01 02 04		
0214 SUB CHRONIC TOX (ANIMAL)	01 02 04	0239 METAB/PHARMACO (ANIMAL)	01 02 04		
0215 CHRONIC TOX (ANIMAL)	01 02 04	0240 METAB/PHARMACO (HUMAN)	01 02 04		

TRIAGE DATA	NON-CBI INVENTORY	ONGOING REVIEW	SPECIES	TOXICOLOGICAL CONCERN:	USE:	PRODUCTION:
YES		YES (DROP/REFER)	<u>RAT</u>	LOW		
CAS SR	NO	NO (CONTINUE)		MED		
	<u>IN TRAINING</u>	REFER		HIGH		

1092-39-0