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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)

October 15, 1992

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

mm
2/2/95

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;
- o the "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.
- o the "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 CRITERIA EXIST?</u>	<u>New 1991 GUIDE CRITERIA EXIST?</u>
ACUTE LETHALITY		
Oral	N}	Y}
Dermal	N}	Y}
Inhalation (Vapors)	N} ⁶	Y} 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 *in vitro* discussed; discussion of "Ames test".

¹⁹Guide at pp-23.

²⁰43 Fed Reg at 11112; 11115 at Comment 16.

CAS # 122-60-1; 538-43-2; 122-60-1; 122-60-1;

Chem: (1) 1,2-epoxy-3-phenoxypropane as received from Shell

(2) 3-phenoxy-1,2-propanediol;

(3) 1,2-epoxy-3-phenoxypropane, purified Shell material;

(4) 1,2-epoxy-3-phenoxypropane

Title: No Title

Date: 8/1/72

Summary of Effects: D/C

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Copies to: C. I. Handy
J. S. Ramsey (1)

E. I. du Pont de Nemours and Company
Haskell Laboratory for Toxicology and Industrial Medicine

HASKELL LABORATORY REPORT NO. 7-72 MR NOS. 1453 AND 1524

Materials Tested	Haskell Nos.	Other Codes	Material's Submitted By
1) 1,2-Epoxy-3-phenoxypropane, as received from Shell	1) 7107	None	J. S. Ramsey
2) 3-Phenoxy-1,2-propanediol	2) 7109		Textile Fibers Department
3) 1,2-Epoxy-3-phenoxypropane, purified Shell material	3) 7255		Kinston Plant
4) 1,2-Epoxy-3-phenoxypropane, 99.5% pure Du Pont material	4) 7374		

Introduction:

A stabilized, low carboxyl-type polyester fiber is made by addition of 0.5% 1,2-epoxy-3-phenoxypropane also known as phenylglycidylether (PGE), to the polymer just prior to extrusion. Calculations have shown that 0.5% should be consumed in the stabilizing reaction so a small amount of unreacted EPP passes through the system. The hold-up time from the point of injection to the spinneret is approximately thirty minutes and the temperature varies from 250° to 305°C. Unreacted EPP, its impurities and decomposition products volatilize at the spinneret producing a strong, slightly sickening odor. It is surmised that the odor originates from decomposing EPP (EPP at room temperature has a pleasant odor).

Several years ago, Haskell Laboratory carried out extensive testing on EPP and the literature pertaining to EPP toxicity is extensive. However, for several reasons (discussed below), additional tests were conducted. A known impurity of technical grade EPP is 3-phenoxy-1,2-propanediol, reported in the literature to be a muscle relaxant and central nervous system depressant. It is also reported that liver homogenate will quantitatively convert 2-cresylglycidyl ether to the corresponding diol. Since EPP may respond in similar fashion, both EPP and the corresponding diol were tested for central nervous system effect.

Diglycidyl ether (DGE), reported to be the most toxic of the glycidyl ethers tested, has been found by Textile Fibers Department personnel as an impurity in the Shell produced EPP at levels up to 2.4%. Since it is also a possible pyrolysis product from EPP and since no studies have been reported on the toxicity of EPP heated to temperatures well above its boiling point, inhalation tests were conducted under conditions simulating those of actual manufacture. This program incorporated the testing of the commercial Shell material, highly purified Shell material, and a 99.5% pure Organic Chemicals Department-produced EPP representing anticipated manufacturing quality.

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- 2 -

Procedure:

Central Nervous System Effect

To compare the effects of H-7107 and H-7109, groups of six male albino CBR-CD rats were given intraperitoneal injections of the test materials as 10% suspensions in a 5% gel of gum acacia in isotonic saline. The dose levels ranged from 200 to 800 mg/kg. Observations were made of the clinical signs, number losing righting reflex and deaths per group, and the recovery time.

Inhalation Toxicity

The test material, as a liquid, was metered by infusion pump into a stainless steel tube which was heated to 25°C. It was carried as a vapor by nitrogen (2.4 L/min) through a tube furnace heated to 310°C. The delivery tube with a dilution port for oxygen (0.6 L/min) was also heated from the furnace to the exposure chamber to prevent condensation. When necessary, the chamber was cooled with ice packs to keep the temperature below 30°C. In the subacute test, an equilibrating flask was added before the chamber so that exposure was to saturated atmosphere and not droplets.

Atmospheric samples were taken by drawing a known volume of air from the chamber through a midjet impinger containing redistilled water as the solvent. The impinger was chilled in an ice bath; the inlet arm was horizontally positioned and rinsed with water into the impinger after each sampling period. The samples were then diluted to a known volume and the optical density read at 268 m on the Beckman DU spectrophotometer. Concentrations were calculated from a standard curve prepared from a tested compound.

In all exposures, groups consisted of six male albino CBR-CD rats having initial body weights of 250-300 grams. The animals were observed and weighed daily during the test and two week recovery period.

Approximate Lethal Concentrations (ALC) Test

Groups of rats were given single four-hour exposures to the 310°C pyrolysate of the test material in the atmosphere. Histopathologic examination of tissues was done on selected animals that died during exposure or were sacrificed one, two, seven and fourteen days post-exposure.

Procedure: (Continued)

Subacute Test

A group of six rats received ten four-hour exposures to the 310°C pyrolysate of the test material during a two-week period. A similar group was exposed to house-line air for the same time periods and served as a control. Following the final exposure, half of each group was sacrificed for histopathologic examination. The remaining animals were sacrificed after a two-week recovery period.

Results:

<u>Haskell No.</u>	<u>Dose (mg/kg)</u>	<u>Central Nervous System Effect</u>		<u>Clinical Signs</u>
		<u>Loss of Righting Reflex</u>	<u>Mortality</u>	
7107	200	0/6	0/6	6/6 incoordination at all doses above 200 mg/kg. Recovery within 30 minutes up to 500 mg/kg, minutes or more at higher levels.
	300	1/6	0/6	
	400	1/6	0/6	
	500	1/6	0/6	
	600	5/6	0/6	
	700	5/6	0/6	
7109	800	6/6	0/6	6/6 incoordination at all doses. Recovery in 90 minutes or more at all levels. Death in 5-10 minutes at lethal level.
	200	0/6	0/6	
	400	4/6	0/6	
	600	5/6	0/6	
	700	6/6	5/6	

Inhalation Toxicity - ALC

<u>Haskell No.</u>	<u>Exposure No.</u>	<u>Concentration (mg/L)</u>		<u>Mortality</u>
		<u>Pre</u>	<u>Post</u>	
7107	1	0.10	16	0
	2	0.18	29	0
	3	0.91	145	0
	4	1.88	301	0
	5	4.02	643	3/5

Sublethal Concentrations: Irritability, hyperemia, wet fur, moderate weight loss up to 3 days post-exposure.

Lethal Concentrations: Same as above plus lacrimation, nasal discharge, incoordination, swelling of scrotum, marked weight loss and 3/6 deaths one day post-exposure.

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Results: (Continued)

Inhalation Toxicity - ALC (Continued)

Faskell No.	Exposure No.	Concentration*		Mortality	Clinical Signs
		(mg/L)	(ppm)		
7255	1	1.18	189	0/6	Sublethal Concentrations: Swelling of scrotum within 24 hours, irritable, moderate weight loss 1-3 days.
	2	1.27	203	0/6	
	3	1.60	256	0/6	
	4	2.02	323	3/6	

Lethal Concentrations: Inactivity, wet fur, hyperemia, nasal discharge, lacrimation, swelling of scrotum within 24 hours, irritability, marked weight loss and 3/6 deaths 1-6 days post-exposure.

Inhalation Toxicity - Subacute

7374	1-10	0.18±	29±	0/6	Initial labored respiration, hyperemia of ears, inactivity, yellow fur, depressed rate of weight gain during exposure period.
		0.024*	3.99*		

* As Epp

* Standard deviation

Pathology:

H-7107: Single exposure at the ALC concentration of H-7107 caused severe irritation of the respiratory tract, acute pulmonary edema and acute bronchopneumonia. At levels below the ALC level, no histopathologic changes were observed.

H-7374: Microscopic examination revealed depletion of hepatic glycogen, atrophy of hepatocytes, subacute and chronic inflammation of the mucous membranes of the trachea and possible oligospermia.

Discussion:

The initial exposure set-up and sampling technique varied somewhat from that described in the above procedure. The test material was maintained slightly above the boiling point throughout the exposures. Vapors of the material were carried through the tube furnace by nitrogen; air and/or oxygen were added before or at the point of entry into the chamber. All delivery lines were kept heated to above the boiling point to avoid condensation and blockage. Samples were taken as described in the procedure, but the sampling point was vertically positioned and the impinger inlet tube was not rinsed after each sample.

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Discussion: (Continued)

During these exposures, the sample of the test material in the heated flask changed markedly in color from yellow-tinged to deep amber, suggesting decomposition of the compound or selective evaporation. In industrial use, the material undergoes only short-term heating. To more closely simulate use conditions and more accurately determine chamber concentration, the apparatus was changed to the technique described above. Results of these earlier exposures are not included in this report.

Summary:

The test for central nervous system effect indicated that both H-7107 and H-7109 caused loss of righting reflex. The data suggest that H-7109 produced more severe results causing a greater number and more prolonged paralysis at a lower dose than did H-7107. H-7109 also appeared to be the more toxic, causing death in 5/6 animals at 7 mg/kg while H-7107 caused no deaths at 800 mg/kg. Lack of material precluded further testing with H-7109.

The approximate Lethal Concentration of H-7107 was 4.02 mg/L (643 ppm); that of H-7255 was 2.00 mg/L (320 ppm). Clinical signs of toxicity were similar in both cases.

The concentration for the subacute test on H-7374 was selected on the basis of the ALC for H-7109. At a concentration of 0.18 mg/L (29 ppm), the atmospheric saturation point, the compound caused mild clinical effects throughout the exposure period and none during the recovery period. Histopathology revealed prominent subacute and chronic catarrhal tracheitis, atrophy of vital organs and depletion of hepatic glycogen.

Conclusion: On the basis of these tests, the previously recommended concentration limit of 0.75 ppm should be maintained for industrial use.

§ Letter to J. S. Ramsey from F. D. Griffith dated December 16, 1971.

† Hine, et al. [J. Pharmacol. Expt'l. Therap., 27:411-419 (1949)] in a study on related glycerol ethers called this effect paralysis. Contemporary neuropharmacologists call it loss of righting reflex, which is a descriptive term, without assigning a cause to it, if only this simple test is used. Further work would be needed to determine whether the loss of righting ability was due to a nervous system depression or was a true paralysis.

Report by: Constance H. Selig
Constance W. Eddy

Raymond W. Norris
Raymond W. Norris

Approve by: John A. Zapp
John A. Zapp, Jr.
Director

MB 927

Case: 5M: pgh

Date: August 1, 1972



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

MAY 08 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" .

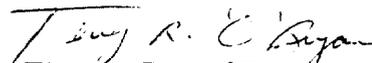
All TSCA 8(e) submissions are placed in the public files unless confidentiality is claimed according to the procedures outlined in Part X of EPA's TSCA §8(e) policy statement (43 FR 11110, March 16, 1978). Confidential submissions received pursuant to the TSCA §8(e) Compliance Audit Program (CAP) should 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
Risk Analysis Branch

Enclosure

12418A



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Triage of 8(e) Submissions

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CAP

Submission number: 12418A

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Group 2 - Ernie Falke (1 copy total)

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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:
0539 REFER TO CHEMICAL SCREENING
0575 CAP NOTICE

VOLUNTARY ACTIONS:
0401 NO ACTION REPORTED
0402 STUDIES PLANNED/IN PROGRESS
0403 NOTIFICATION ON WORK IN PROGRESS
0404 LABEL/MSDS CHANGES
0405 PROCESS/LOADING CHANGES
0406 APP/USE DISCONTINUED
0407 PRODUCTION DISCONTINUED
0408 CONFIDENTIAL

SUB. DATE: 10/15/92 OTS DATE: 10/27/92 CSRAD DATE: 02/02/95

CHEMICAL NAME:

Propane, 1,2-epoxy-3-phenoxy-
1,2-Propanediol, 3-phenoxy-

CASE:

122-60-1
538-43-2

INFORMATION TYPE:

P F C

0201	ONCO (HUMAN)	01 02 04
0202	ONCO (ANIMAL)	01 02 04
0203	CELL TRANS (IN VITRO)	01 02 04
0204	MUTA (IN VITRO)	01 02 04
0205	MUTA (IN VIVO)	01 02 04
0206	REPRO/TERATO (HUMAN)	01 02 04
0207	REPRO/TERATO (ANIMAL)	01 02 04
0208	NEURO (HUMAN)	01 02 04
<u>0209</u>	NEURO (ANIMAL)	01 02 04
0210	ACUTE TOX. (HUMAN)	01 02 04
0211	CHR. TOX. (HUMAN)	01 02 04
<u>0212</u>	ACUTE TOX. (ANIMAL)	01 02 04
<u>0213</u>	SUB ACUTE TOX (ANIMAL)	01 02 04
0214	SUB CHRONIC TOX (ANIMAL)	01 02 04
0215	CHRONIC TOX (ANIMAL)	01 02 04

INFORMATION TYPE:

P F C

0216	EPI/CLIN	01 02 04
0217	HUMAN EXPOS (PROD CONTAM)	01 02 04
0218	HUMAN EXPOS (ACCIDENTAL)	01 02 04
0219	HUMAN EXPOS (MONITORING)	01 02 04
0220	ECO/AQUA TOX	01 02 04
0221	ENV. OCCUR/REL/FATE	01 02 04
0222	EMER INCI OF ENV CONTAM	01 02 04
0223	RESPONSE REQEST DELAY	01 02 04
0224	PROD/COMP/CHEM ID	01 02 04
0225	REPORTING RATIONALE	01 02 04
0226	CONFIDENTIAL	01 02 04
0227	ALLERG (HUMAN)	01 02 04
0228	ALLERG (ANIMAL)	01 02 04
0229	METAB/PHARMACO (ANIMAL)	01 02 04
0230	METAB/PHARMACO (HUMAN)	01 02 04

INFORMATION TYPE:

P F C

0241	IMMUNO (ANIMAL)	01 02 04
0242	IMMUNO (HUMAN)	01 02 04
<u>0243</u>	CHEM/PHYS PROP	01 02 04
0244	CLASTO (IN VITRO)	01 02 04
0245	CLASTO (ANIMAL)	01 02 04
0246	CLASTO (HUMAN)	01 02 04
<u>0247</u>	DNA DAM/REPAIR	01 02 04
<u>0248</u>	PROD/USE/PROC	01 02 04
0251	MSDS	01 02 04
0259	OTHER	01 02 04

TRIAGE DATA:

NON-CBI INVENTORY

ONGOING REVIEW

SPECIES

TOXICOLOGICAL CONCERN:

USE:

PRODUCTION:

YES

YES (DROP/REFER)

RAT

LOW

INDUSTRIAL

CAS SR

NO

NO (CONTINUE)

MED

IN PLANNING

REST-R

HIGH

0209 6 male albino CARC rats were given i.p injection of the chemical at doses of 200 to 800 mg/kg. Clinical observations included incoordination at all doses of 300 mg/kg or more. Recovery occurred within 30 minutes at doses up to 500 mg/kg, and within 90 minutes or more at doses of 500 mg or more.

CECATS/TRIAGE TRACKING DBASE ENTRY FORM

CECATS DATA:

Submission # BEHQ-1092-12418 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
- 0878 CAP NOTICE

VOLUNTARY ACTIONS:

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

SUB. DATE: 10/15/92 OTS DATE: 10/27/92 CSRAD DATE: 02/02/95

CHEMICAL NAME:

Propane, 1,2-epoxy-3-phenoxy-
1,2-Propanediol, 3-phenoxy-

CAS#

122-60-1
538-43-2

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. OCCUR/REL/FATE	01 02 04	0246 CLASTO (HUMAN)	01 02 04
0207 REPRO/TERATO (ANIMAL)	01 02 04	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	0251 PROD/USE/PROC	01 02 04
0209 NEURO (ANIMAL)	01 02 04	0224 PROD/COMP/CHEM ID	01 02 04	0299 OTHER	01 02 04
0210 ACUTE TOX. (HUMAN)	01 02 04	0225 REPORTING RATIONALE	01 02 04		
0211 CHR. TOX. (HUMAN)	01 02 04	0226 CONFIDENTIAL	01 02 04		
0212 ACUTE TOX. (ANIMAL)	01 02 04	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:

RAT

LOW Acute i.p. RAT (7107)
Acute inhal RAT (7107)
MED Acute inhal RAT (7255)
HIGH Subacute inhal RAT (7374)

Acute i.p. RAT (7109);

YES

YES (DROP/REFER)

NO

NO (CONTINUE)

IN TRAINING

REFER

CAS SR

1044212

12418A

L

7107: Acute intraperitoneal toxicity in rats is of low concern. Single intraperitoneal injections to male Chr-CD rats (6/dose) at levels of 200, 300, 400, 500, 600, 700, and 800 mg/kg were not lethal. Loss of righting reflex and incoordination occurred at ≥ 300 mg/kg. Recovery occurred within 30 minutes for animals given ≤ 500 mg/kg.

L

7107: Acute inhalation toxicity in rats is of low concern. Single 4-hour inhalation exposures to rats (6/group) at levels of 100, 180, 910, 1880, and 4020 mg/m³ were lethal to 3/6 animals at the high concentration. Clinical signs included inactivity, hyperemia, weight loss, incoordination, and swelling of the scrotum. Pathological examination revealed pulmonary edema and bronchopneumonia at 4020 mg/m³.

L

7109: Acute intraperitoneal toxicity in rats is of low concern. Single intraperitoneal injections to male Chr-CD rats (6/dose) at levels of 200, 400, 600, and 700 mg/kg were lethal to 5/6 animals at the high dose. Loss of righting reflex occurred at ≥ 400 mg/kg, and incoordination occurred at all doses. Recovery occurred within 90 minutes for animals given ≤ 600 mg/kg.

M

7255: Acute inhalation toxicity in rats is of moderate concern. Single 4-hour inhalation exposures to rats (6/group) at levels of 1180, 1270, 1600, and 2020 mg/m³ were lethal to 3/6 animals at the high concentration. Clinical signs included swelling of the scrotum, irritability, inactivity, hyperemia, lacrimation, and weight loss.

M

7374: Subacute inhalation toxicity in rats is of moderate concern. Six rats were subjected to ten 4-hour inhalation exposures at 180 mg/m³. There were no deaths. Animals exhibited initial labored respiration, hyperemia, inactivity, and depressed weight gain. Microscopic examination revealed depletion of hepatic glycogen, atrophy of hepatocytes, inflammation of the tracheal mucous membranes, and possible oligospermia.