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October 18, 1992

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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)

8EHQ-92-13156
INIT
88920010959

SECTION 2 M10-52

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.

8E CAP

For Regulatee,

Mark H. Christman
Counsel
Legal D-7158
1007 Market Street
Wilmington, DE 19898
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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 criteri. 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*.

TEST TYPE	1978 POLICY CRITERIA EXIST?	New 1991 GUIDE CRITERIA EXIST?
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
Reprodcutive	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: Not available

Chem: Trifluorochloroethylene

Title: Possible toxicity of Tetrafluoroethylene and
Trifluorochloroethylene; Possible toxicity of Tetrafluoroethylene
+ Trifluorochloroethylene

Date: 10/7/46

Summary of Effects: damage to meninges and brain

Personal and Confidential

October 7, 1946

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[REDACTED]

[REDACTED]

36-46

HL-0036-46

Medical Research Project No. MR-127

Possible Toxicity of Tetrafluoroethylene
and Trifluorochloroethylene

Possible Toxicity of Tetrafluoroethylene (F-114)
and Trifluorochloroethylene (F-113) Monomers

Haskell Laboratory of
Industrial Toxicology

Wilmington, Delaware

Medical Research Project No. MR-127

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10/7/46

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Medical Research Project No. MR-127

Possible Toxicity of Tetrafluoroethylene (F-1114)
and Trifluorochloroethylene (F-1113)

Summary and Conclusions

The toxicity of F-1114 monomer and polymer ("Teflon") and F-1113 monomer has been investigated in this project.

F-1114

Monomeric F-1114 in the pure state is not highly toxic to dogs when inhaled five days a week for six hours daily in concentrations around 1000 p.p.m. over a period of several weeks. Dogs that had been thus exposed were able to tolerate a concentration estimated to be 4000 p.p.m. in the atmosphere for four hours on one day and six hours on another. Dogs that received only an occasional exposure to 1000 p.p.m. or higher showed a sharp drop in systolic and diastolic pressure as a result of exposure.

F-1113

Rats exposed to high concentrations of F-1113 in the atmosphere for short periods (five minutes) tolerated 68 exposures quite well. Dogs exposed for a longer period (up to four hours) to various concentrations showed drastic effects when the concentration was around 500 p.p.m. Death resulted and at autopsy the animals showed severe damage to the meninges and brain. Concentrations of 100 p.p.m. produce definite physiological effects.

Teflon

A hazard exists when polymerized F-1114 ("Teflon") is heated to a point where there is a simultaneous evolution of HF and a fine sublimate from the polymer. This combination gives rise to the attacks of "shakes" noted in workers handling this product. These attacks resemble very closely the syndrome known as "brass founder's ague" caused by the inhalation of zinc vapor. Skin contact with an alcohol slurry of the polymer may be followed by nausea, vomiting, chills, and fever.

It is recommended that whenever the polymer is heated above 200°C, adequate ventilation be provided to remove any fumes or dust that may be formed. Cases accidentally gassed with fumes from "Teflon" should be treated following the procedure recommended for nitrous fumes (3) including the inhalation of oxygen under a pressure of 4 to 6 cm. of water.

HASKELL LABORATORY OF
INDUSTRIAL TOXICOLOGY

John H. Foulger, M. D.
Director

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BY: Allan J. Fleming, M. D.
Assistant Director

AJF:gfd
10/3/46

Medical Research Project No. MR-127

Possible Toxicity of Tetrafluroethylene (F-1114)
and Triflurochloroethylene (F-1113) Monomers

This project was undertaken to obtain more information about the possible toxicity of the above two compounds. Workers exposed to one or the other of these compounds were reported to have exhibited peculiar symptoms characterized by shivering spells, a feeling of tightness in the chest and difficulty in breathing. Fever and marked fatigue were also among the complaints.

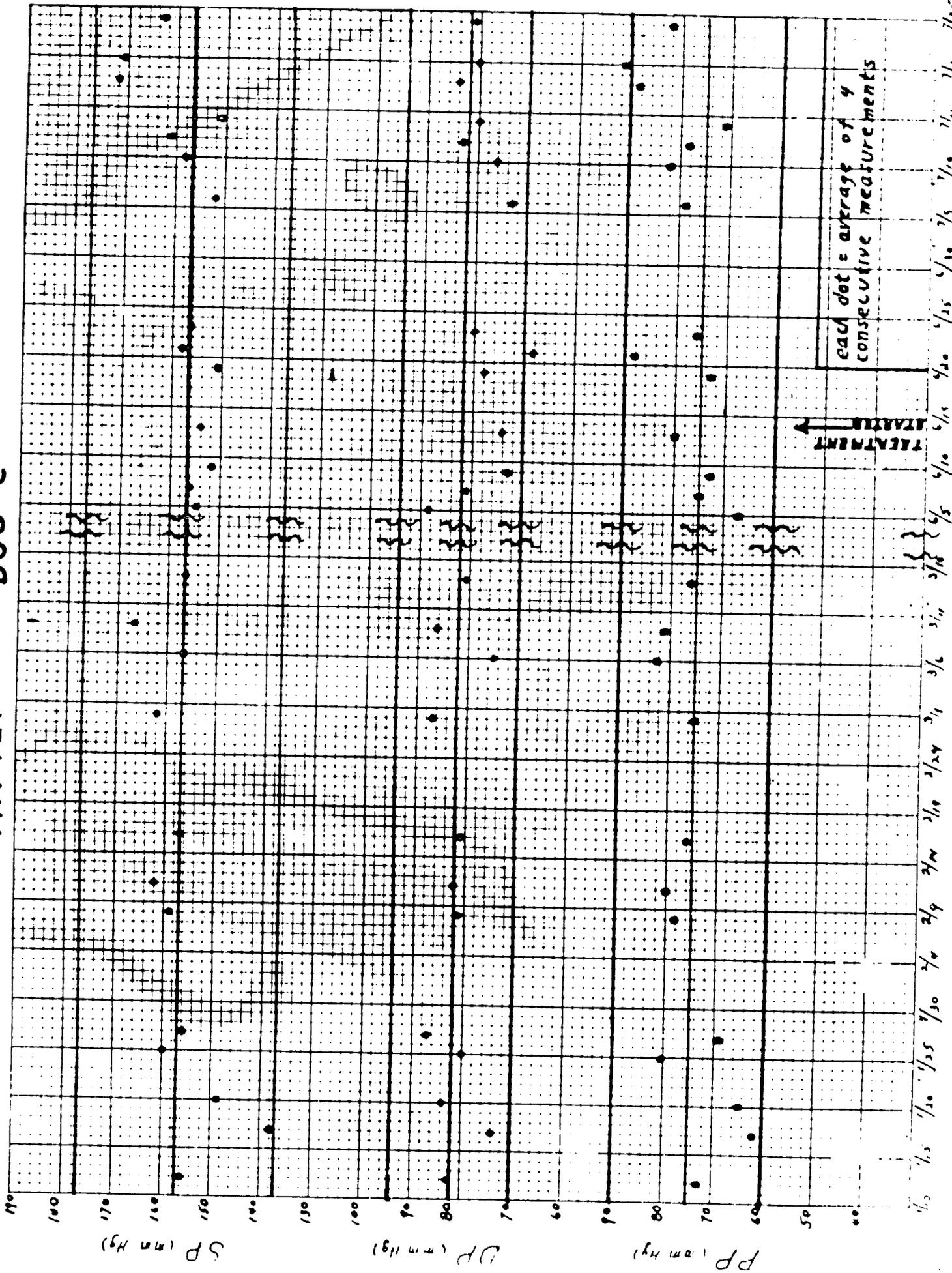
Certain fluorinated hydrocarbons are known to be pulmonary irritants. Consequently, our investigation at first was centered around the effect of these compounds on the respiratory system.

Due to the inertness of these compounds chemically, it was not possible to devise a suitable method of air analysis and the concentrations in the atmosphere were obtained by liberating a known volume of gas in an exposure chamber of known capacity. The concentrations mentioned hereafter, are the maximum theoretical concentration obtainable, and it is probable that the maximum is only attained during the third and fourth hours of exposure.

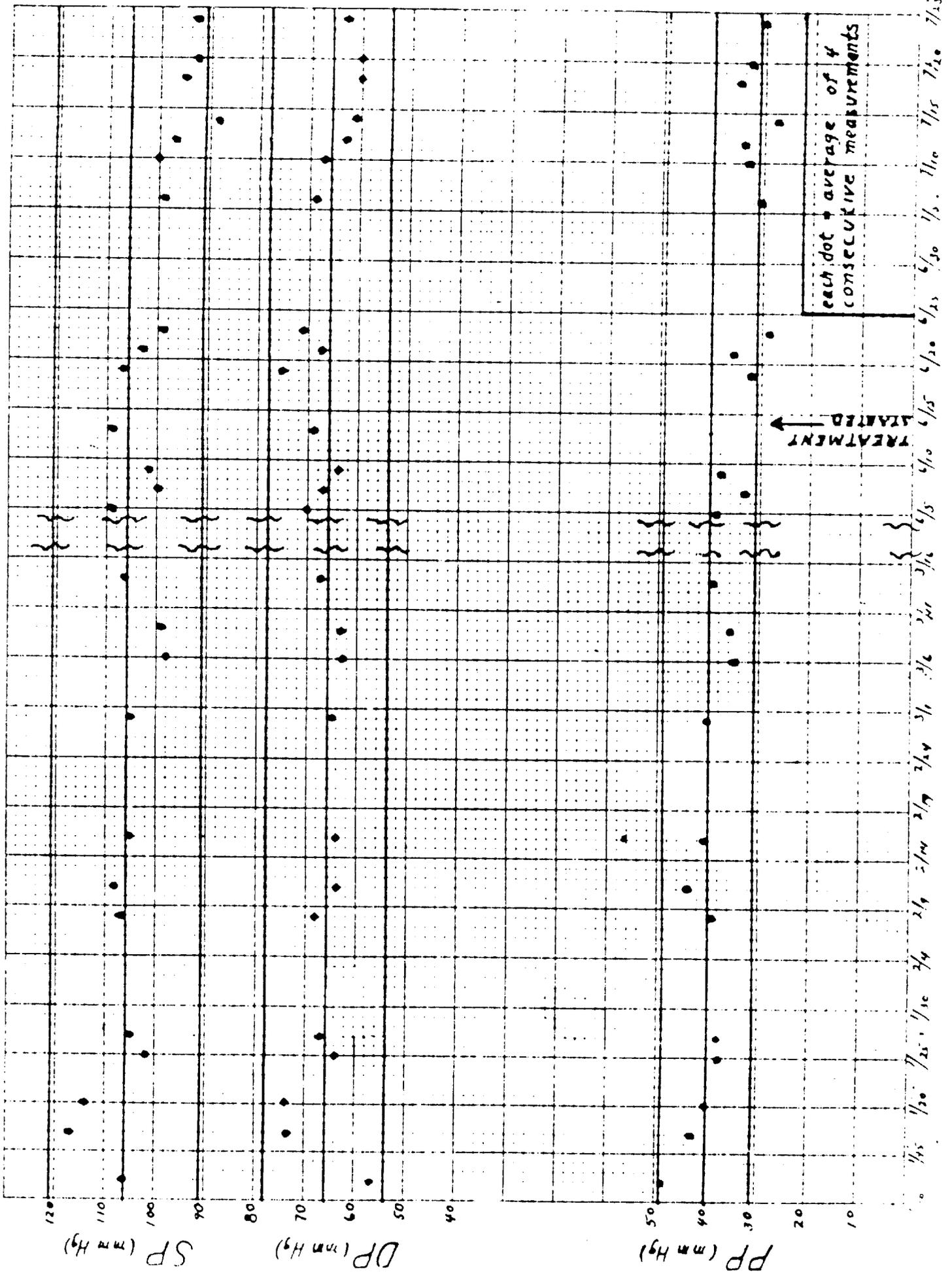
I. Chronic Exposure of Dogs to Tetrafluroethylene Monomer.

Dogs 127B and C were exposed daily to T.F.E. monomer in the atmosphere for a period of four hours. The capacity of the chamber was

MR 127 DOG C



MR 127 DOG B



10 cu. meters. Each day's exposure was as follows:

8:55 a.m.	Dogs placed in exposure chamber.
9:00 " "	20 liters T.F.E. liberated in the chamber.
10:00 a.m.	10 liters T.F.E. liberated in the chamber.
11:00 " "	10 liters T.F.E. liberated in the chamber.
12:00 Noon	10 liters T.F.E. liberated in the chamber.
1:00 p.m.	Dogs removed from the chamber.

The results of the blood pressure studies are summarized in the charts for Dogs 127B and 127C. Each dot represents the average of four consecutive blood pressure measurements.

Dog 127B showed a gradual drop in systolic and diastolic pressure sufficient to produce pulse pressure readings outside the normal range (values outside the limits set by the mean \pm twice the standard deviation).

Dog 127C did not show any significant trend during the course of the experiment. Both dogs gained weight satisfactorily and did not show other evidence of having been adversely affected. Special studies of the heart sounds did not reveal any abnormal shift in the frequency of the sounds as a result of exposure to F-1114.

Acute Exposure to Tetrafluoroethylene Monomer
Superimposed on a Chronic Exposure.

Dogs 127B and C were exposed to approximately 4,000 p.p.m., F-1114 monomer for four hours on one day and six hours the following day, after having been exposed for 6 weeks (25 four-hour exposures) to approximately 1,000 p.p.m. Both dogs tolerated these high concentrations quite well and did not show drastic changes in the circulation.

Acute Exposures to Tetrafluoroethylene Monomer.

Dog 127D was exposed to T.F.E. on several occasions at intervals two to three weeks apart. Exposure to concentrations of approximately 500 p.p.m. for four hours did not affect the blood pressure. Exposures to 1,000 p.p.m. or higher gave rise to a fairly marked drop in systolic and diastolic pressures. Otherwise, the dog showed no untoward sign.

Dog 127A received two exposures each of an hour's duration to an estimated concentration of 2,000 p.p.m. There was not a great change in blood pressure after the first exposure. Following the second exposure, there was a fairly sharp drop in systolic and diastolic pressure noted about 4 hours after the dog was removed from the exposure chamber.

Autopsy Examination.

Dogs 127 A and D were killed a month after the last exposure. There was no gross pathology noted, and no microscopic pathology of the heart, lungs, spleen, liver, kidneys or adrenals.

Dogs 127B and C both died as a result of an acute exposure to F-1113, and the results of their autopsies will be reported under this compound.

Acute and Semi-Acute Experiments with Trifluorochloroethylene (F-1113)

A. Experiments with Rats

Four rats were exposed twice daily for 5 minutes to an atmosphere containing about 2,000 p.p.m. F-1113, for 68 exposures, and an additional 10 exposures to approximately 4,000 p.p.m. When compared with four control rats, the rats exposed to F-1113 did not gain weight as rapidly.

A slight irritation to the eyes and nose was noted with each exposure; otherwise, the rats behaved normally. Blood counts done before and after exposure did not show any sign of the hematopoietic system having been affected. At autopsy, there was no significant pathology noted in the lungs, liver or kidney. The brain was not examined.

B. Experiments with Dogs

On the basis of the experiments on rats, dogs were exposed to a fairly high concentration of F-1113 in the atmosphere over several hours, since it was believed the monomer was not too highly toxic. This proved not to be the case.

Dog 127C, at the termination of the F-1114 experiment, was given a single exposure to an estimated maximum concentration of 4,000 p.p.m. of F-1113, for a period of 4 hours. The dog vomited repeatedly after removal from the exposure chamber. It became maniacal two hours after removal and began to have severe convulsions. It died two hours and forty minutes after removal from the chamber.

Dog 127E was similarly exposed to 2,000 p.p.m. and appeared to remain normal during the three hours following exposure. However, it died some time during the night.

The chief finding at autopsy in both dogs was an intense engorgement and congestion of the brain with some softening. Grossly, the other organs in the body did not appear abnormal. Microscopically, the chief findings were an acute injury to the liver cells and marked congestion of the brain with degeneration of the ganglion cells. Edema of the heart muscle was also noted.

A third dog (127F) was exposed on three occasions for four hours to a maximum estimated concentration of 500 p.p.m. of F-1113. The protocol of this experiment is given in detail as the reaction noted is quite typical of what occurs with exposure to this compound when the concentration is below that which is lethal in a short time.

Dog 127F Exposed to F-1113 Gas in a Chamber of 10 Cubic Meters Capacity

<u>Date</u>	<u>Time</u>	<u>Blood Pressure</u>	<u>Pulse</u>	<u>Respiration</u>	<u>Rectal Temp.</u>
10/12/44	9:10 AM	132/84	96	20	100.2 Control reading.
	9:15	Exposure started. Five liters F-1113 passed into chamber.			
	10:15	Five liters of F-1113 passed into chamber.			
	11:15	Five liters of F-1113 passed into chamber.			
	12:15 PM	Five liters of F-1113 passed into chamber.			
	1:15	Dog removed from chamber.			
	1:20	112/75	92	24	99.6
	4:25	122/64	88	26	100.4
10/13/44	10:00 AM	156/84	68	18	100.8
	4:15 PM	106/56	92	24	102.0
10/14/44	10:20 AM	156/78	80	20	101.4

Observations were made daily from 10/15 to 10/27 during which time the systolic and diastolic pressure occasionally dropped markedly. The temperature fluctuated in the normal range.

<u>Date</u>	<u>Time</u>	<u>Blood Pressure</u>	<u>Pulse</u>	<u>Respira- tion</u>	<u>Rectal Temp.</u>
10/30/44	8:30 AM	106/84	88	16	100.4
	9:00 AM to 1:00 PM	Dog exposed four hours as on 10/12/44.			
	1:10 PM	120/64	80	18	100.6
	4:15 PM	110/78	68	22	100.0
10/31/44	8:30 AM	124/66	92	18	100.8
	9:00 AM to 1:00 PM	Dog exposed four hours as on 10/12/44.			
	1:20 PM	110/60	110	20	99.8 Dog very thirsty when removed from the cham- ber.

2:30 PM Dog became maniacal. Barked incessantly for half an hour and jumped around inside its cage. About 3:00 PM, it went into a series of convulsions. Amyl nitrite inhalations were given for a few seconds, but before any further treatment could be attempted, the dog went into a severe convulsion and remained rigid until death. The heart continued to beat slowly for several minutes after respirations had ceased. The rectal temperature immediately after death was 108° F.

Autopsy was performed an hour after death, at which time rigor mortis was complete and extreme. The eyes were bloodshot. The heart was greatly dilated. The lungs were grossly normal as was the spleen and pancreas. There was nothing grossly

abnormal with the liver, stomach or intestines. The medullary portion of the kidneys was congested. The adrenals appeared normal. The brain was slightly congested.

Microscopically, the heart muscle fibers were frequently separated by edema fluid. The lung, pancreatic and adrenal tissue was normal. The splenic tissue was anemic. The perihepatic cells of the liver were slightly enlarged and stained less intense than the periportal cells. Congestion of the blood vessels of the medullary portion of the kidneys was noted. The brain was edematous and showed meningeal congestion.

Dog 127G was exposed for four hours to the same concentration of F-1113 used for Dog 127F (about 500 p.p.m.) on two occasions, one week apart. It died during the night following the second exposure. The chief finding at autopsy was the acute congestion of the brain and meninges.

Dog 127I received four 3-hour exposures to 400 p.p.m. over a period of 10 days. It survived and did not show any gross or microscopic pathology when sacrificed six days after the last exposure.

Dog 127H received ten exposures of three hours each to approximately 100 p.p.m. F-1113 in the atmosphere over a period of sixteen days. It survived all exposures and was sacrificed four days after the last exposure. There was no gross

or microscopic pathology noted except a hemorrhage at the ileo-cecal valve with enlargement of the regional lymph nodes. One tapeworm was found in the small intestine. The hemorrhage noted above was probably coincidental and not due to any action of the F-1113.

It appears from the foregoing that trifluorochloroethylene has a very pronounced effect on the brain and meninges, and that concentrations around 500 p.p.m. if inhaled for a period of time may be highly dangerous. Concentrations around 100 p.p.m. will produce a sharp drop in systolic and diastolic pressure if inhaled over a period of four hours.

Experiments with Polymerized F-1114 ("Teflon")

Since neither F-1114 or F-1113 in the monomeric form gave rise to symptoms analogous to those noted in workers exposed to "Teflon", further tests were carried out on rats to determine if the dust or gas given off from heated polymer might be responsible for the attacks of "shakes" noted from time to time in workers handling "Teflon" products. The various samples of polymer that have been tested are listed in Appendix A. The details of these tests are voluminous and only the pertinent observations and conclusions are herein recorded.

The attacks referred to above resemble "brass founder's ague" caused by the inhalation of zinc vapor. Similar symptoms may be caused by the inhalation of the vapors of iron, nickel, copper, tin, and cadmium. The activity of these metals seems to depend on their being inhaled in an extremely fine state of division. The symptoms and signs resulting from inhaling metallic vapors may not develop until several hours after exposure, and consist of fatigue to the point of exhaustion, aching pains in the limbs, chills, elevated temperature, roughness of the throat, and sometimes bronchitis.

The symptoms from "Teflon" are similar in nature and may be accompanied by signs of pulmonary edema.

It has been known for some time that small amount of hydrofluoric acid is liberated when "Teflon" is baked at 360° to 390°C. This was investigated by Dr. Lewis at Arlington and the results of his investigation reported in a letter of October 22, 1945. He showed that during the first hour of baking (with dry air passing over the polymer at 0.12 liters per minute) the rate of evolution of HF from 100 grams of polymer was around 4 mg. per hour. During the second hour the rate fell to around 2 mg. per hour, and then remained constant at a rate of 1.2 to 1.3 mg. per hour. The rate of evolution of HF increased with increasing humidity of the air passing over the polymer. When 0.83 cc. of water per minute was vaporized over the polymer, the rate of evolution of HF increased tenfold.

In our experiments evolution of gas or sublimate producing a loss in weight of 30 mg. from 20 grams of polymer over a period of two hours caused deaths in rats from pulmonary edema. The maximum concentration of HF in these experiments would be of the order of 0.05 mg./liter.

Ronzani (1) reported deaths in guinea pigs after inhaling hydrofluoric acid in a concentration of 0.03 mg./liter for 24 hours. Machle and Kitzmiller (2) reported that exposure of rabbits, guinea pigs and monkeys to concentrations around 0.01 mg./liter for six to seven hours for 50 days produced degenerative changes in the lungs and liver.

From our observations on experimental animals (rats), two factors seem to be necessary before drastic effects are produced in the lungs by the gases from "Teflon".

1. The polymer must be heated until the evolution of HF takes place. This usually occurs when the temperature of the polymer is around 180° to 200° C., there being considerable variation in different samples of polymer.

2. A fine sublimate formed during the heating must be inhaled by the rats. The activity of the baking gases has been reduced considerably by reducing the volume (and speed) of the air flowing over the heated polymer and by placing an electrostatic precipitator in the line to remove dust. Bubbling the gases through three tubes each containing 10 cc. of distilled water did not reduce the toxicity.

Somewhat crucial experiments have been carried out to evaluate the effect of this sublimate. The polymer was heated to 380° - 400° C., and a current of air blown through the heating tube at five liters a minute. This air current was alternated every few minutes between two bell jars each containing two rats. The current of air passed directly to one bell jar from the heating tube, but had to pass through an electrostatic precipitator to reach

the other bell jar. The precipitator is designed to remove dust or fine particles from the atmosphere, but subsequent examinations of the rat lungs showed that the removal was not too efficient. Each pair of rats was given a two-hour exposure at the end of which time one rat from each pair was killed. The one from the unprotected bell jar had severe pulmonary edema. The other rat showed only slight congestion of the lungs. Frozen sections were made at once of the lungs, and after being stained by a special technique, they were examined under a polarizing microscope. It was possible to count the number of particles of polymer in the lung alveoli by counting a large number of fields. It was found that there were 30 to 40 per cent more particles in the lung of the rat with pulmonary edema than in the other lung (obtained from the rat in the chamber protected by the electrostatic precipitator). There was also a gross difference between the lungs of the other two rats killed two and one-half hours after removal from the bell jar, but no particle counts were made on the lungs.

Inhalation of Teflon dust from the original powdered polymer supplied in December, 1944 or dusts produced by grinding polymer in a micronizer and under alcohol in a Waring Blender (to avoid heat)

gave rise to some microscopic changes in the lungs. Dust from the original powdered polymer sample and dust from the Waring Blender treated polymer gave rise to a slight swelling in the alveolar walls of the lung. Dust from the micronizer gave a slightly more severe reaction with definite edema in one rat. The amount of dust to which the rats were exposed was high (1.5 to 16 million particles per cubic foot). With each sample the majority of the particles were under 4 microns in size.

It appears that dust alone can produce some irritation in the lung tissue, but it is not highly active unless it arises from heated polymer.

Effect of Temperature to which Polymer is Heated.

Severe pulmonary edema has been noted in rats exposed for two hours to gases coming from 20 grams of "Teflon" heated to 280° C. Slight changes were noted in the lungs of rats when exposed for two hours to the gases from polymer heated to 260° C., and similar changes noted when rats were exposed for six hours to gases from polymer heated to 240° C. No changes were noted in rats exposed to gases from the polymer heated to 220° C. for six hours. One case of "shakes" occurred in a workman who was feeding scrap "Teflon" through a mill for several hours. It is possible that the polymer

temperature was in excess of 200°C. during the short intervals it was between the rollers of the mill.

Influence of other Factors on the Toxicity of F-1114 Polymer.

Toxic reactions were observed in rats exposed to regular polymer of best, average, and poor quality as measured by tensile and hardness tests, and in rats exposed to polymer that had been pre-baked for various intervals. Toxic reactions were also observed in rats exposed to polymer that had been extracted with alcohol or nitric acid, or to polymer made without borax. Any of these samples when heated to 360° or higher gave rise to pulmonary irritation.

Repeated heating of one sample of polymer over two hour periods gave rise to deaths in rats exposed to the fumes even after four such periods. Fresh rats were exposed during each two hour interval. The polymer after repeated heating continues to give off the toxic gas or sublimate. Whenever the loss in weight of a 20 gram sample of compressed polymer exceeded 30 mg. over a two hour period the rats usually died or showed severe lung damage.

1. Ronzani, E.; Arch. Hyg., 70: 217
(1909), quoted in J. of Ind. Hyg. & Tox., 19:
129 (March) 1937.

2. Machle, Willara, and Kitzmiller,
Karl; J. Ind. Hyg. & Tox., 17: 223, No. 5 (Sept.)
1935.

3. Fleming, A. J.; A Method for Handling
Cases Gassed with Nitrous Fumes. Ind. Med., 12:
127-132, No. 3, (March) 1943.

AJF:efd
10/3/46

Triage of 8(e) Submissions

Date sent to triage: 2/5/96

NON-CAP

CAP

Submission number: 13156A

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

S~~B~~TOX

SEN

w/~~NEUR~~

Group 3 - Elizabeth Margosches (1 copy each)

w/neur
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 <u>2</u> pages	<u>1, 1st tab</u> pages <u>[REDACTED]</u>
Notes:	
Contractor reviewer: <u>LPS</u>	Date: <u>12/6/95</u> <u>[Signature]</u>

8E Number and Chemical Name	Rank	Reason or Brief Description
-13156 Trifluorochloro- ethylene, CAS 74- 38-9, Tetrafluoro- ethylene (Teflon), CAS 116-14-3	Low	<p>In 1992 a chemical company submitted a medical research report completed in 1946 that anecdotally discusses health effect symptoms in humans exposed to the chemicals as well as toxicologic testing in dogs and rats. Industrial hygienists had reported workers exhibiting shivering spells, chest tightness, labored breathing, fever and marked fatigue. The chemicals have been studied more completely since this early report.</p>

CECATS TRIAGE TRACKING DRASE ENTRY FORM

CECATS DATA: Submission # SEHO-1192-13156 SEQ. A
 TYPE: INT. SUPP FLWP
 SUBMITTER NAME: F. 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:
 0505 REFER TO CHEMICAL SCREENING
 0506 CAP NOTICE

VOLUNTARY ACTIONS:
 0601 NO ACTION PERMITTED
 0602 STUDIES PLANNED/IN PROGRESS
 0603 NOTIFICATION TO WORKERS REQUIRED
 0604 LABELING/MSDS (YIANGHS)
 0605 PROCESSING/IMP. (YIANGHS)
 0606 APP. USE DISCONTINUED
 0607 PRODUCTION DISCONTINUED
 0608 CONFIDENTIAL

SUB. DATE: 10/18/92 OTS DATE: 11/02/92 CSRAD DATE: 03/22/95

CHEMICAL NAME: F-1113 (Teflon Monomers)
F-1114 (Teflon Monomers)
 CASE: 79-38-9
116-14-3

INFORMATION TYPE	P.F.C.	INFORMATION TYPE	P.F.C.	INFORMATION TYPE	P.F.C.
0201 ONCO (HUMAN)	01 02 04	0216 EPICLIN	01 02 04	0241 IMBUNO (ANIMAL)	01 02 04
0202 ONCO (ANIMAL)	01 02 04	0217 HUMAN EXPOS (PROD. CONTAM)	01 02 04	0242 IMBUNO (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 BONAQUA TOX	01 02 04	0245 CLASTO (ANIMAL)	01 02 04
0206 REPRO/TERATO (HUMAN)	01 02 04	0221 ENV. OCCUREL/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	0248 PRODUSE/PROC	01 02 04
0209 NEURO (ANIMAL)	01 02 04	0224 PRODCOMP/CHEM ID	01 02 04	0251 MSDS	01 02 04
0210 ACUTE TOX. (HUMAN)	01 02 04	0225 REPORTING RATIONALE	01 02 04	0259 OTHER	01 02 04
0211 CHR. TOX. (HUMAN)	01 02 04	CONFIDENTIAL	01 02 04		
0212 ACUTE TOX. (ANIMAL)	01 02 04	ALLERG (HUMAN)	01 02 04		
0213 SUB ACUTE TOX (ANIMAL)	01 02 04	ALLERG (ANIMAL)	01 02 04		
0214 SUB CHRONIC TOX (ANIMAL)	01 02 04	METAPHARMACO (ANIMAL)	01 02 04		
0215 CHRONIC TOX (ANIMAL)	01 02 04	METAPHARMACO (HUMAN)	01 02 04		

Studies have been reviewed, several times, before PRODUCTION.

TRANSMISSION: YES NO
 NON-CBI INVENTORY: YES NO (CONTINUE)
 ONGOING REVIEW: YES (DROP/REFER) NO (CONTINUE)
 SPECIES: DOG RAT
 TOXICOLOGICAL CONCERN: LOW MED HIGH

1946 (Shadoks)

USING Rats and dogs were exposed to trichloroethylene, tetrafluoroethylene, tetrafluoroethane, at a variety of concentrations. Dogs exposed to the chemicals (during the 70s specified) at a concentration around 500 ppm produced deaths and at a top of 1000 ppm, severe damage to the meninges and brain.

CECATS/TRIAGE TRACKING DBASE ENTRY FORM

CECATS DATA: Submission # BEHO-1192-13156 SEQ. A

TYPE: INT. SUPP FLWP
 SUBMITTER NAME: F.I. Dupont de Nemours and Company

SUB. DATE: 10/18/92 OTS DATE: 11/02/92 CSRAD DATE: 03/22/95

CHEMICAL NAME: F-1113
F-1114

CASE # 79-38-9
116-14-3

INFORMATION REQUESTED: FLWP DATE:
 0201 NO INFO REQUESTED
 0302 INFO REQUESTED (TECH)
 0303 INFO REQUESTED (VOL. ACTIONS)
 0304 INFO REQUESTED (REPORTING RATIONALE)
 DISPOSITION:
 0209 REFER TO CHEMICAL SCREENING
 0210 CAP NOTICE

VOLUNTARY ACTIONS:
 0401 NO ACTION REPORTED
 0402 STUDY'S PLANNED/IN PROGRESS
 0403 NOTIFICATION OF WORKING RATIONALE
 0404 LABELING/STUDY/TESTING
 0405 PROFESSIONAL/INDUSTRY/AGENCY
 0406 APP/USE DISCONTINUED
 0407 PRODUCTION DISCONTINUED
 0408 CONFIDENTIAL

INFORMATION TYPE	P.F.C.	INFORMATION TYPE	P.F.C.	INFORMATION TYPE	P.F.C.
ONCO (HUMAN)	01 02 04	EPICLIN	01 02 04	IMMUNO (ANIMAL)	01 02 04
ONCO (ANIMAL)	01 02 04	HUMAN EXPOS (PROD CONTAM)	01 02 04	IMMUNO (HUMAN)	01 02 04
CELL TRANS (IN VITRO)	01 02 04	HUMAN EXPOS (ACCIDENTAL)	01 02 04	CHEMPHYS PROP	01 02 04
MUTA (IN VITRO)	01 02 04	HUMAN EXPOS (MONITORING)	01 02 04	CLASTO (IN VITRO)	01 02 04
MUTA (IN VIVO)	01 02 04	BIO/AQUA TOX	01 02 04	CLASTO (ANIMAL)	01 02 04
REPRO/TERATO (HUMAN)	01 02 04	ENV. OCCURENCE/FATE	01 02 04	CLASTO (HUMAN)	01 02 04
REPRO/TERATO (ANIMAL)	01 02 04	EMER INCI OF ENV CONTAM	01 02 04	DNA DAMAGE/REPAIR	01 02 04
NEURO (HUMAN)	01 02 04	RESPONSE REQ/ST DELAY	01 02 04	PROD/USE/PROC	01 02 04
NEURO (ANIMAL)	01 02 04	PROD/COMP/CHIEM ID	01 02 04	MSDS	01 02 04
ACUTE TOX. (HUMAN)	01 02 04	REPORTING RATIONALE	01 02 04	OTHER	01 02 04
ACUTE TOX. (ANIMAL)	01 02 04	CONFIDENTIAL	01 02 04		
CHR. TOX. (HUMAN)	01 02 04	ALLERG (HUMAN)	01 02 04		
CHR. TOX. (ANIMAL)	01 02 04	ALLERG (ANIMAL)	01 02 04		
SUB ACUTE TOX (ANIMAL)	01 02 04	METAB/PHARMACO (ANIMAL)	01 02 04		
SUB CHRONIC TOX (ANIMAL)	01 02 04	METAB/PHARMACO (HUMAN)	01 02 04		
CHRONIC TOX (ANIMAL)	01 02 04				

TRACKING: NON-CBI INVENTORY

ONGOING REVIEW: YES (DROP/REFER) NO (CONTINUE) REPT

SPECIES: DOG RAT

TOXICOLOGICAL CONCERN: LOW MED HIGH

CAS SR: YES NO

IN PLANNING:

PRODUCTION:

11/11/92

CECATS TRIAGE TRACKING DBASE ENTRY FORM

CECATS DATA: Submission # BEHO-1192-13156 SEQ. A

INFORMATION REQUESTED: FLWP DATE: _____

- 6501 NO INFO REQUESTED
- 6502 INFO REQUESTED (TECH)
- 6503 INFO REQUESTED (VOL. ACTIONS)
- 6504 INFO REQUESTED (REPORTING RATIONALE)

VOLUNTARY ACTIONS:

- 6401 NO ACTION REQUIRED
- 6402 STUDY'S PLANNING IN PROGRESS
- 6403 IDENTIFICATION IN WORK PROGRESS
- 6404 LABELING IN PROGRESS
- 6405 PROCESSING IN PROGRESS
- 6406 APPROUSE DISCONTINUED
- 6407 PRODUCTION DISCONTINUED
- 6408 CONFIDENTIAL

TYPE: INT. SUPP FLWP
 SUBMITTER NAME: F. I. Dupont de Nemours and Company

DISPOSITION:
 REFER TO CHEMICAL SCREENING
 CAP NOTICE

SUB. DATE: 10/18/92 OTR DATE: 11/02/92 CSRAD DATE: 03/22/95

CHEMICAL NAME: F-1113 CASE: 79-38-9
F-1114 116-14-3

INFORMATION TYPE:	L.F.C.	INFORMATION TYPE:	L.F.C.	INFORMATION TYPE:	L.F.C.
0201 ONCO (HUMAN)	01 02 04	EPICLIR	01 02 04	0201 BUBUNO (ANIMAL)	01 02 04
0202 ONCO (ANIMAL)	01 02 04	HUMAN EXPOS (PROD CONTAM)	01 02 04	0202 BUBUNO (HUMAN)	01 02 04
0203 CELL TRANS (IN VITRO)	01 02 04	HUMAN EXPOS (ACCIDENTAL)	01 02 04	0203 CHEMOPHY'S PROF	01 02 04
0204 MUTA (IN VITRO)	01 02 04	HUMAN EXPOS (MONITORING)	01 02 04	0204 CLASTO (IN VITRO)	01 02 04
0205 MUTA (IN VIVO)	01 02 04	ECOTOXIC TOX	01 02 04	0205 CLASTO (ANIMAL)	01 02 04
0206 REPRO/TERATO (HUMAN)	01 02 04	ENV. COCCUREL FATE	01 02 04	0206 CLASTO (HUMAN)	01 02 04
0207 REPRO/TERATO (ANIMAL)	01 02 04	EMER INCI OF ENV CONTAM	01 02 04	0207 DNA DAMAGE/REPAIR	01 02 04
0208 NEURO (HUMAN)	01 02 04	RESPONSE REQUEST DELAY	01 02 04	0208 PRODUCE/PROC	01 02 04
0209 NEURO (ANIMAL)	01 02 04	PROD/CHEM ID	01 02 04	0209 MSDS	01 02 04
0210 ACUTE TOX. (HUMAN)	01 02 04	REPORTING RATIONALE	01 02 04	OTHER	01 02 04
0211 CHR. TOX. (HUMAN)	01 02 04	CONFIDENTIAL	01 02 04		
0212 ACUTE TOX. (ANIMAL)	01 02 04	ALLERG (HUMAN)	01 02 04		
0213 SUB ACUTE TOX (ANIMAL)	01 02 04	ALLERG (ANIMAL)	01 02 04		
0214 SUB CHRONIC TOX (ANIMAL)	01 02 04	METAPHARMACO (ANIMAL)	01 02 04		
0215 CHRONIC TOX (ANIMAL)	01 02 04	METAPHARMACO (HUMAN)	01 02 04		

NON-CELL INVENTORY: YES NO
 CAS SR: YES NO
 IN FRAMES: _____
 REPORT: _____
 ONGOING REVIEW: YES (DROPPED) NO (CONTINUE)
 SPECIES: DOG RAT
 TOXICOLOGICAL CONCERN: LOW Subacute Inhalation Toxicity (F-1114, F-1113), Acute Inhalation Toxicity (F-1114)
MED Acute Inhalation Toxicity (F-1113)
 HIGH

#13156A

F-1114

L

Subacute inhalation toxicity is of low concern based on no mortality in 2 dogs exposed to 1000 ppm 6 hours/day, 5 days/week for 6 weeks.

L

Acute inhalation toxicity is of low concern based on no mortality in 2 dogs exposed to 4000 ppm for 4 hours and 6 hours, 500 and 1000 ppm for 4 hours in 1 dog, and 2000 ppm for 1 hour in 1 dog. A decrease in systolic and diastolic blood pressures was observed in the dog exposed to 1000 and 2000 ppm.

F-1113

M

Acute inhalation toxicity is of medium concern based on lethality in 1 dog exposed to 4000 ppm for 4 hours. After exposure, observations included repeated vomiting, maniacal behavior and severe convulsions prior to death. A second dog died after exposure to 2000 ppm for 4 hours. At autopsy, both dogs demonstrated intense engorgement and congestion of the brain with some softening. Histological examination revealed liver, brain and heart damage. A third dog was exposed to 500 ppm for 4 hours on 3 occasions (18 days separated first and second exposure; 1 day separated second and third exposures). Observations for the three exposures included: 1) decreased systolic and diastolic blood pressures, 2) nothing reported, and 3) maniacal behavior, convulsions and death (heart dilation, and brain congestion were observed at autopsy). A fourth dog was exposed to 500 ppm for 4 hours on 2 occasions, resulting in death after the second exposure; autopsy revealed brain and meningeal congestion.

L

Subacute inhalation toxicity is of low concern in rats exposed to 2000 ppm twice daily for 5 min, for 68 exposures, and an additional 10 exposures at 4000 ppm. Slight irritation was observed during exposures. Subacute exposures in dogs (4 x 3 hours at 400 ppm over 10 days, and 10 x 3 hours at 100 ppm) did not cause death or signs of toxicity.

8E Number and Chemical Name	Rank	Reason or Brief Description
<p>✓ -13156 Trifluorochloro-ethylene, CAS 74-38-9, Tetrafluoro-ethylene (Teflon), CAS 116-14-3</p>	<p>Low</p>	<p>In 1992 a chemical company submitted a medical research report completed in 1946 that anecdotally discusses health effect symptoms in humans exposed to the chemicals as well as toxicologic testing in dogs and rats. Industrial hygienists had reported workers exhibited shivering spells, chest tightness, labored breathing, fever and marked fatigue. The chemicals have been studied more completely since this early report.</p>
<p>-10445 Pentachloro-phenols, CAS 87-86-5</p>		<p>Submission of missing pages to a previously received epidemiologic study. The original pages are being sought, but they do not constitute -7850 cap set 31 on the same chemical.</p>

Assigned
"to
"beam-up"
set