

ENVIRONMENTAL PROTECTION AGENCY

[OPTS-42045 TSH-FRL 2465-1]

Halogenated Alkyl Epoxides: Response to the Interagency Testing Committee**AGENCY:** Environmental Protection Agency (EPA).**ACTION:** Notice.

SUMMARY: The Interagency Testing Committee recommended that EPA consider requiring health effects testing of halogenated alkyl epoxides under section 4 of the Toxic Substances Control Act (TSCA). In a separate notice in this issue of the Federal Register, EPA is proposing testing of hexafluoropropylene oxide, one member of the category of halogenated alkyl epoxides. This notice describes EPA's decision not to initiate rulemaking at this time to require testing of other members of the category of halogenated alkyl epoxides.

FOR FURTHER INFORMATION CONTACT: Jack P. McCarthy, Director, TSCA Assistance Office (TS-799), Office of Toxic Substances, Environmental Protection Agency, Rm. E-543, 401 M. St., SW., Washington, D.C. 20460, Toll Free: (800-424-9065), In Washington, D.C.: (554-1404), Outside the USA: (Operator-202-554-1404).

SUPPLEMENTARY INFORMATION:**I. Background**

Section 4(e) of TSCA (Pub. L. 94-469, 90 Stat. 2003 *et seq.*, 15 U.S.C. 2601 *et seq.*) established an Interagency Testing Committee (ITC) to recommend to EPA a list of chemicals to be considered for testing under section 4(a) of the Act.

The ITC designated halogenated alkyl epoxides for priority consideration in its Second Report, published in the Federal Register of April 19, 1978 (43 FR 18684). The ITC defined the category of halogenated alkyl epoxides as "halogenated noncyclic aliphatic hydrocarbons with one or more epoxy functional groups." Seven specific compounds in this category were discussed in this ITC's report: 1-chloro-2,3-epoxypropane (epichlorohydrin); 1,1,1-trichloro-2,3-epoxypropane (TCPO); 1-bromo-2,3-epoxybutane (epibromohydrin or EBH); 1,4-dichloro-2,3-epoxybutane (DCBO); 1,1,1-trichloro-3,4-epoxybutane (TCBO); tetrafluoroethylene oxide (TFEO); hexafluoropropylene oxide.

The ITC recommended that halogenated alkyl epoxides be considered for testing for carcinogenicity, mutagenicity, teratogenicity, and other chronic effects.

The ITC also recommended that epidemiology studies be considered. The ITC's recommendations for this chemical category were based on high production levels for one member of this chemical category (500 million pounds annually for epichlorohydrin), (2) a National Institute for Occupational Safety and Health (NIOSH) estimate that between 50,000 and 140,000 workers are exposed to epichlorohydrin annually, (3) expected increases in the use of other halogenated alkyl epoxides, and (4) limited studies on oncogenic, mutagenic, teratogenic, and other chronic effects of halogenated alkyl epoxides.

In a separate notice in today's Federal Register, EPA is proposing testing of hexafluoropropylene oxide, one member of the category of halogenated alkyl epoxides. This notice describes EPA's decision not to initiate rulemaking to require testing of other members of the category of halogenated alkyl epoxides at this time. In combination, these two notices provide EPA's response to the ITC's designation of halogenated alkyl epoxides for testing consideration as required by TSCA section 4(e).

II. Decision Not to Initiate Rulemaking A. Epichlorohydrin

EPA has decided not to initiate rulemaking at this time to require health effects testing of epichlorohydrin under section 4 of TSCA for the reasons outlined below. (1) Recent bioassays demonstrate that epichlorohydrin is oncogenic in rats, and provide sufficient data to assess its oncogenic hazard. On the basis of these data, EPA has initiated an evaluation of the need to undertake regulatory activities to further control exposures to epichlorohydrin. (2) Existing data suggest that epichlorohydrin may cause mutagenic, neurotoxic and reproductive effects as well as certain other chronic effects (e.g., liver and kidney effects). These data further suggest that such effects are likely to be induced only at or above exposure levels (or doses) that would cause oncogenic effects. (3) EPA believes that defining an exposure level that is adequate for control of any unreasonable oncogenic risk should adequately protect against any unreasonable risks of the other health effects for which the ITC recommended testing. Thus, EPA believes that no information essential to potential regulatory activities would be gained through additional health effects testing at this time. (4) Available retrospective epidemiology studies on epichlorohydrin are generally flawed by multiple chemical exposures, short latency

periods among the selected cohorts, or inappropriate selection of the exposure group. Because of difficulties in identifying a suitable cohort for epidemiology studies of epichlorohydrin, EPA believes that similar limitations would be encountered in any newly initiated studies. Additionally, EPA believes that the animal studies on epichlorohydrin are sufficient for any needed regulatory action, and that epidemiology studies are not likely to be necessary to support regulatory decisions on this particular chemical substance.

If, however, EPA finds at any time that the health effects data on epichlorohydrin are not sufficient for any needed regulatory action, or if it appears that epidemiology studies are necessary to support regulatory decisions on this chemical substance and a suitable cohort can be identified at that time, EPA will reconsider this decision not to initiate rulemaking to require health effects testing or epidemiology studies of epichlorohydrin.

1. *Release and exposure.* An estimated 400 million pounds of epichlorohydrin are produced annually (Ref. 45). Actual production figures have been claimed confidentially by the manufacturers. The volume of epichlorohydrin imported in 1981 was 3.0 million pounds (Ref. 6). In 1981, approximately 55 percent of the U.S. supply of epichlorohydrin was used as an intermediate in the production of epoxy resins, 25 percent in the production of synthetic glycerin, 10 percent in miscellaneous uses, and 10 percent was exported (Ref. 45). Miscellaneous uses of epichlorohydrin include use in the manufacture of elastomers, glycidyl ethers, modified epoxy resins, paper sizing agents, inks, textile additives, ion exchange resins, and surface active agents. Epichlorohydrin is also used as a stabilizer in materials that contain chlorine such as chlorinated rubbers (Refs. 32, 35, and 83).

Human exposure to epichlorohydrin appears to be principally in the workplace. NIOSH most recently estimated that 50,000 workers are potentially exposed to epichlorohydrin (Ref. 49). This estimate is an extrapolation based on a 1972-1974 survey of workers and industries using epichlorohydrin.

The current Occupational Safety and Health Administration (OSHA) standard for occupational exposure to epichlorohydrin is 5 ppm (19 mg/m³) as an 8-hour time weighted average (TWA) (29 CFR 1910.1000). In 1976, NIOSH recommended an occupational exposure

limit of 0.5 ppm (2 mg/m³) as a 10-hour TWA for a 40-hour week and a ceiling limit of 5 ppm (Ref. 49). NIOSH arrived at this recommendation after concluding that risks from exposure to epichlorohydrin may include carcinogenesis, mutagenesis, and sterility. In 1980, the American Conference of Government Industrial Hygienists (ACGIH) lowered their recommended threshold limit value (TLV) for epichlorohydrin from 5 ppm to 2 ppm (8 mg/m³, with a 5 ppm short term exposure limit (STEL) (Ref. 3).

Personal exposure monitoring of workers from an epoxy resin producing unit, a glycerin producing unit, and an epichlorohydrin producing unit show concentrations of epichlorohydrin as high as 15 ppm (grab sample), 4.89 ppm (7-hour TWA), and 2.1 ppm (8-hour TWA), respectively (Refs. 49 and 55). Concentrations of epichlorohydrin in each unit averaged less than 1 ppm (Refs. 49 and 55).

Based on emission factors reported by manufacturers of epichlorohydrin, Scientific Applications Inc. (SAI), a private contractor, estimated that 146,600 pounds of epichlorohydrin were released into the atmosphere from production facilities in 1978 (Ref. 60). In 1974, one manufacturer of epichlorohydrin estimated that release of epichlorohydrin from production facilities was less than 18,000 pounds per year (Ref. 68). Information submitted under section 8(a) of TSCA on releases of epichlorohydrin in 1981 has been claimed confidential by the manufacturers. Total atmospheric emissions from all production and uses of epichlorohydrin in 1978 were estimated by SAI to be 480,000 pounds (Ref. 60). Using U.S. Census Bureau files, SAI estimated that 10.6 million people live within 12.5 miles of epichlorohydrin production and use facilities (Ref. 60). Based on estimated emission rates, chemical properties, and dispersion modelling, SAI estimated that these persons may be exposed to epichlorohydrin concentrations ranging from 0.0001 ug/m³ (0.000026 ppb) to 50 ug/m³ (13 ppb) due to these atmospheric releases (Ref. 60).

The manufacturers of epichlorohydrin reported that the heavy ends (liquid wastes) from production facilities are recovered or thermally degraded, and that no epichlorohydrin is discharged (Ref. 68). Epichlorohydrin is regulated under the Resource Conservation and Recovery Act (RCRA) as a hazardous waste on the basis of its potential hazard to human health (40 CFR 261.10).

Epichlorohydrin was reported twice in effluent from an unspecified chemical plant in Louisville, Kentucky (Ref. 82)

and was detected at 3 ppm in the effluent from the American Aniline and Extract Co., Calvert City, Kentucky (Ref. 21). No reports on epichlorohydrin were found in STORET, a data base that contains water quality information for the majority of the streams in the United States.

On the basis of available data, and because of the reactivity of the epoxide ring, epichlorohydrin is unlikely to be sufficiently persistent in the environment to result in significant general population or environmental exposure. The available data indicate that: (a) Epichlorohydrin is volatile and, under simulated atmospheric smog conditions, reportedly decomposes with a half-life of 16 hours (Ref. 15); (b) it is water soluble, with dissolved epichlorohydrin having a hydrolytic half-life of 2-3 days (Refs. 8 and 59); (c) an estimated log P of 0.26 suggests that epichlorohydrin has a low potential for uptake and bioconcentration from the aquatic environment (Ref. 32); (d) modelling by Falco et al. (Ref. 23) indicates a low potential for adsorption of epichlorohydrin onto soil and sediments; and (e) studies by Castro and Bartnicki (Ref. 11) indicate that epichlorohydrins are generally susceptible to microbially-mediated hydrolysis (or biodegradation) in the soil.

The potential exists for consumer exposure to epichlorohydrin vapors and leachates when epoxy resins are used as adhesives, coatings, and plastics. Although epichlorohydrin theoretically is not present after an epoxy resin is formed, it is possible that some epichlorohydrin remains as a contaminant. No U.S. studies have been found that present data on epichlorohydrin exposures resulting from the use of epoxy resins. However, several foreign studies report vapor concentrations of 0.23-12 mg/m³ (0.06-3 ppm) epichlorohydrin from epoxy-based adhesives and plastics (Refs. 20, 38 and 57). Epoxy resins also are used as coatings for articles intended for use in packaging food. This use is regulated by the Food and Drug Administration (21 CFR 175, 178, 177, 178).

2. Health effects—*a. Carcinogenicity.* Epichlorohydrin induced squamous cell carcinomas of the nasal cavity in Sprague-Dawley rats inhaling either 100 ppm, 6 hours/day for 30 days followed by lifetime observation or 30 ppm, 6 hours/day, 5 days/week for life (Ref. 41); no nasal carcinomas were produced by 6 hours/day, 5 days/week lifetime exposure to 10 ppm epichlorohydrin. Epichlorohydrin induced squamous cell hyperplasia, papillomas, and carcinomas in the forestomach in Wistar rats when administered at 750 or 1,500

ppm in drinking water for 81 weeks (Ref. 34); the total amount of epichlorohydrin ingested was 8.9 g and 15.1 g, respectively. In the 1,500 ppm dosage group, squamous cell carcinomas of the oral cavity were also reported. Hyperplasia was found at the 375 ppm epichlorohydrin dose level (5.0 g total ingested epichlorohydrin). Another study reported a dose-related incidence of squamous cell carcinomas in the forestomach in Wistar rats after lifetime exposure to epichlorohydrin by gavage at 2 or 10 mg/kg/day, 5 days/week (Ref. 72). In mice, epichlorohydrin was a weak initiator in a two-stage skin carcinogenesis assay; induced local sarcomas and one malignant tumor, other than those at the injection site, following subcutaneous injections of 1.0 mg/week for life; and showed no carcinogenic effect with the mouse skin painting technique or by intraperitoneal injection (Ref. 70). EPA has concluded that the data from these studies are sufficient to characterize the oncogenic potential of epichlorohydrin and, therefore, further oncogenicity testing is not necessary.

b. Mutagenicity. There are extensive data on the mutagenicity of epichlorohydrin. The data show that epichlorohydrin induces gene mutations and chromosomal aberrations *in vitro* and *in vivo*. Epichlorohydrin has been shown to be a direct-acting mutagen in several bacteria and yeast (*E. coli*, *S. typhimurium*, *K. pneumoniae*, *S. cerevisiae*), primarily inducing base-pair substitutions (Refs. 7, 42, 65, 69 and 75). Epichlorohydrin induced reverse mutations in a purple adenineless mutant of *Neurospora crassa* (Ref. 37), eceriferum mutants in barley (Ref. 43), and sex-linked recessive lethal mutations in *Drosophila* (Refs. 36 and 58).

Epichlorohydrin induced chromosome aberrations in human lymphocytes *in vitro* (Refs. 10, 39 and 76) and mouse bone marrow cells *in vivo* (Ref. 65). An increase in chromosome breaks and exchanges was observed in lymphocytes from epichlorohydrin-exposed workers in several studies (Refs. 40, 56, 67); no differences in the incidence of chromosomal aberrations in exposed and unexposed workers were reported in another study (Ref. 17). In dominant lethal assays in mice, epichlorohydrin was negative in two studies (Refs. 27 and 65) and positive in another (Ref. 66).

Epichlorohydrin was one of the substances tested in the International Collaborative Program on Short-term Tests for Carcinogenicity. Epichlorohydrin was almost universally found to be mutagenic in the *in vitro* test

systems. This included forward and reverse mutations in bacteria, the Rec and Pol assays in bacteria, mutation and recombinational end-points in yeast, unscheduled DNA synthesis in cultured fibroblasts, and gene mutations, sister-chromatid exchanges, and chromosome aberrations in mammalian cells (Ref. 13).

EPA has concluded that the data from these studies are sufficient to indicate that epichlorohydrin is mutagenic to somatic cells. The results of the dominant lethal assay in mice raise concern that epichlorohydrin also has the potential to induce mutations in germ line cells.

c. Teratogenicity. Two studies have addressed the teratogenic potential of epichlorohydrin. In rats and rabbits exposed by inhalation to 0, 2.5 or 25 ppm epichlorohydrin for 7 hours/day on days 6 through 15 of gestation for rats and on days 6 through 18 gestation for rabbits, no embryotoxic or teratogenic effects were demonstrated (Ref. 18). Slight maternal toxicity was seen at the 25 ppm dose level. In a second study, epichlorohydrin administered by gastric intubation to rats at 40, 80, or 160 mg/kg/day and to mice at 80, 120, or 160 mg/kg/day on days 6 through 15 of gestation did not demonstrate teratogenic effects (Ref. 46). The 160 mg/kg/day dose in the rat was maternally toxic but not fetotoxic. The 120 and 160 mg/kg/day levels in the mice were maternally toxic and fetotoxic. EPA has concluded that these studies are sufficient to characterize the teratogenic potential of epichlorohydrin and, therefore, additional teratogenicity testing is not necessary.

d. Reproductive effects. Reproductive studies demonstrate that epichlorohydrin produces infertility in male rats. Oral administration of 15 mg/kg/day epichlorohydrin produced infertility in male Sprague-Dawley rats within 7 days; the effect was found to be reversible (Ref. 29). Reduced fertility was reported in male Wistar rats given 5 daily oral doses of 20 mg/kg or 50 mg/kg; a single dose of 100 mg/kg produced permanent sterility (Ref. 12). Histologic examination revealed retention cysts in the ductuli efferentes and proximal caput of the testis in the sterile animals.

In another study, male rabbits and male and female rats were exposed to 0, 5, 25 or 50 ppm epichlorohydrin vapor for 6 hr/day for 10 weeks (Ref. 31). Transient infertility was seen in treated male rats at 50 ppm and pre-implantation loss was observed in nontreated female rats mated with males treated at 25 ppm; no effects were noted at 5 ppm. Reversal of these effects was observed 14 days post-exposure. No

reproductive effects were observed in the treated male rabbits or treated female rats. EPA has concluded that these data are adequate to indicate that epichlorohydrin causes reproductive effects in mammals. The number of data points from these studies is limited, however, and will only allow a rough quantitative risk assessment. EPA has concluded, however, that more testing to define more precisely the quantitative aspects of the reproductive effects risk is not necessary at this time because the existing data suggest that adverse reproductive effects are likely to be induced only at or above exposure levels that would cause oncogenic effects, that any regulatory action taken to control for unreasonable oncogenic risk will adequately protect against any unreasonable risk of adverse reproductive effects, and, thus, that no information essential to potential regulatory activities would be gained through additional reproductive effects testing at this time.

e. Other chronic effects. Several subchronic and chronic studies of epichlorohydrin have been reported. Rats and mice exposed to concentrations of 0, 5, 25, or 50 ppm epichlorohydrin for 6 hours/day, 5 days/week for a 90-day period exhibited toxic effects in the nasal turbinates, kidneys, adrenal glands, and epididymides of the testis at the 25 and 50 ppm concentrations when compared with controls (Ref. 18). No observed effects relative to controls were noted at 5 ppm. During testing for carcinogenicity, renal lesions were noted in rats inhaling 10 or 30 ppm epichlorohydrin for 6 hours/day, 5 days/week over their lifetimes or 100 ppm, 6 hours/day for 30 days (Ref. 41). Abnormalities of the lungs, blood vessels, kidney and brain were observed in rats exposed to 5.3 ppm epichlorohydrin for 24 hours/day for 98 days (Refs. 24 and 25). At 0.5 ppm, white blood cell counts were elevated; no adverse effects were observed at 0.05 ppm (Refs. 24 and 25). EPA concludes that the data are adequate to characterize the chronic toxicity, excluding neurotoxicity of epichlorohydrin.

With respect to neurotoxicity, most of the studies of epichlorohydrin do not note any substantive neurotoxic effects, although a single Russian study (Ref. 25) reports behavioral and neuropathological effects in rats after exposure to concentrations of 5.3 ppm, 24 hours/day for 98 days; the author further reported that the greatest toxic changes were found in the lungs and kidneys. Because the details of this study are scant, EPA believes these data can only be considered weakly

suggestive and that the weight of the available evidence is not sufficient to indicate a neurotoxic hazard for epichlorohydrin, especially in light of other effects of concern for epichlorohydrin.

f. Epidemiology. Several epidemiological studies have been performed on workers exposed to epichlorohydrin in the U.S. Two mortality studies (Refs. 19 and 64) provide no evidence for a carcinogenic effect in humans from epichlorohydrin. However, one of the studies was complicated by multiple chemical exposures and in both studies, because of the fairly young ages of the cohorts (mean ages less than 40 years and 48 years), the latency periods may not have been long enough for carcinogenic effects to have developed.

Two studies investigating the effects of epichlorohydrin on the human male reproductive system also had shortcomings. The first study has several flaws: (1) The selection of the exposure group was inappropriate (persons with only one day of exposure occurring years in the past were included in the exposed group); (2) a large proportion of the exposed workers were unwilling to participate in the study; and (3) no information about the control population was provided (Ref. 48). In the second study, the timing of exposure relative to sampling was unclear, and the sample size was too small to provide unambiguous results (Ref. 73).

In three cytogenetic studies, an increase in chromosome breaks and exchanges was observed in lymphocytes from epichlorohydrin-exposed workers (Refs. 40, 56, and 67); no differences in the incidence of chromosome breaks and exchanges in exposed and unexposed workers were reported in a fourth cytogenetic study (Ref. 17).

Possible epidemiology followup studies for epichlorohydrin were considered. EPA concluded, however, that additional retrospective epidemiology studies on epichlorohydrin were not likely to provide useful data at this time. EPA believes that limitations among the available retrospective epidemiology studies on epichlorohydrin, including number of years since first exposure, percent participation in studies, number of persons in cohorts, and complications with multiple chemical exposures, would likely be encountered in newly initiated retrospective studies. In addition, EPA believes that the animal studies on epichlorohydrin are sufficient for any needed regulatory action and that epidemiology studies are not necessary to support regulatory

decisions on this particular chemical substance.

B. Other Halogenated Alkyl Epoxides

With the exception of hexafluoropropylene oxide, EPA also has decided not to initiate rulemaking at this time to require testing of other members of this category of halogenated alkyl epoxides for health effects under section 4 of TSCA because there is not sufficient production or exposure to make section 4 findings for these chemical substances. In a separate notice in this issue of the Federal Register, EPA is proposing testing of hexafluoropropylene oxide.

1. **Production.** The sole domestic producer of epibromohydrin in the U.S. produces very small amounts of EBH (0-25 pounds annually) on a custom basis for sale to research chemical suppliers (Ref. 26). The only current use of epibromohydrin appears to be as a laboratory research reagent (Ref. 88). Great Lakes Chemical Corp., listed as a manufacturer of EBH on the 1977 TSCA Inventory, produced their last batch of EBH in 1975 and ceased marketing the chemical in 1980 (Ref. 28). Great Lakes used EBH at one time in the production of a nematocide product (Ref. 32).

1.1.1-Trichloro-2,3-epoxypropane is produced in small amounts for distribution as a laboratory research chemical (Ref. 1). TCPO is a potent, noncompetitive inhibitor of hepatic epoxide hydrases, inducible enzymes that catalyze the inactivation of some carcinogenic and cytotoxic metabolites (Ref. 1). TCPO can also stimulate the binding of benzo(a)pyrene to DNA (Refs. 2 and 61).

1.1.1-Trichloro-3,4-epoxybutane was produced by Olin Chemical Corp. (Ref. 54). In May 1982, Olin ceased production of TCBO and has no plans to resume production. TCBO had been used by Olin as a chemical intermediate. Potential uses of TCBO are as an intermediate in the preparation of urethanes, epoxy resins, esters, phenolic resins, neutralizing agents, insecticides, fungicides, nematocides, glycols, plasticizers, textiles, vulcanized graft polymer rubbers, solvents, and polyurethane and epoxy catalysts (Ref. 54).

No information has been found on the domestic manufacture or importation of 1,4-dichloro-2,3-epoxybutane or tetrafluoroethylene epoxide.

In addition to the seven haloalkyl epoxides identified by the ITC, several other category members have been identified from literature reports (Refs. 35 and 71). However, there is no information which suggests that these chemicals are commercially

manufactured or imported in the U.S. at this time.

2. **Health effects.** Health effects information on halogenated alkyl epoxides, other than epichlorohydrin, is limited. The information indicates, however, that other halogenated alkyl epoxides may produce toxic effects similar to epichlorohydrin.

TCPO enhanced the tumor-producing ability of 3-methylcholanthrene and benzo(a)pyrene and decreased the latency period for the development of tumors in mice (Ref. 5). Cis- and trans-1-chloropropene oxide and cis- and trans-1,3-dichloropropene oxide induced squamous carcinomas of the skin following chronic skin application and local tumors, mostly fibrosarcomas, following repeated subcutaneous injection in mice (Ref. 71).

Gene mutation studies in *S. typhimurium*, *N. crassa*, and *E. coli* and host-mediated assays in mice indicate that EBH is mutagenic (Refs. 14, 37, and 75). In a dominant lethal assay in mice, EBH was negative (Ref. 22). TCPO and TCBO have been reported as mutagenic without metabolic activation in gene mutation tests in *S. typhimurium* and *S. cerevisiae* (Refs. 9, 27, 44, 51 and 75). TCPO induced chromosome aberrations and sister-chromatid exchanges in cultured human lymphocytes (Ref. 50). DCBO was slightly mutagenic in *S. typhimurium* (Ref. 4) and produced dose-related increases in recessive lethal mutations in *Drosophila* (Ref. 74).

TCPO caused an increase in the teratogenicity of diphenylhydantoin (DPH) in mice (Refs. 30 and 47). TCPO also caused an increase in the teratogenic activity of styrene and styrene oxide in chick embryos (Ref. 33). The authors also reported that TCPO administered alone did not exhibit significant teratogenic effects in these studies.

TCBO administered orally to rats at dose levels of 10, 30, 100, or 300 mg/kg/day on days 6 through 15 of gestation demonstrated no teratogenic or fetotoxic response at maternally nontoxic dose levels (Ref. 52). At 300 mg/kg/day, maternal and fetal toxicity occurred.

A 90-day rat study in which TCBO was administered by gavage at dosage levels of 30, 100, or 300 mg/kg/day, 7 days/week showed dose-related morphological changes in the liver and kidneys (Ref. 53). Lesions were seen among the livers of male rats receiving 100 and 300 mg/kg/day dose levels; the kidneys of male rats showed nephrosis and necrosis of the tubular epithelial cells at all treatment levels.

Although these data suggest that other halogenated alkyl epoxides may elicit toxic effects similar to those induced by

epichlorohydrin, other identified members of this category, with the exception of hexafluoropropylene oxide, are currently produced only in research quantities or are not produced at all. Therefore, EPA believes that there is no basis at this time for finding that these chemical substances may present an unreasonable risk to health or the environment and, thus, EPA is not initiating rulemaking at this time to require testing of these chemical substances. EPA is, however, considering options for follow-up activities concerning these chemical substances such as reporting of production and importation levels under a TSCA section 8(a) rule or reporting of significant new uses under a TSCA section 5(a)(2) rule.

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IV. Public Record

The EPA has established a public record for this testing decision, docket number [OPTS-42045], which includes:

(1) Federal Register notice containing the designation of halogenated alkyl epoxides to the Priority List and public comments thereon.

(2) Communications (public).

(a) Letters.

(b) Contact reports of telephone conversations.

(c) Meeting summaries. -

(3) Published and unpublished data.

This record, which includes basic information considered by the Agency in developing this decision, is available for inspection in the OPTS Reading Room from 8:00 a.m. to 4:00 p.m. on working days in Rm. E-107, 401 M St. SW., Washington, D.C. 20460.

(Sec. 4, 90 Stat. 2003; [15 U.S.C. 2061])

Dated: December 21, 1983.

William D. Ruckelshaus,

Administrator.

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