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Part XI

Environmental Protection Agency

Assessment of Chloroprene as a Potentially Toxic Air Pollutant; Notice

ENVIRONMENTAL PROTECTION AGENCY

[ADL-FRL-2859-3]

Assessment of Chlorcprene as a Potentially Toxic Air Pollutant

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice of intent not to regulate and solicitation of information.

SUMMARY: This notice describes the results of EPA's assessment of chloroprene as a potentially toxic air pollutant. The EPA is announcing its intent not to specifically regulate chloroprene as an air pollutant under any section of the Clean Air Act (CAA). Given that there are uncertainties in the health and exposure information incorporated in this notice and that there has been limited opportunity for public review, the Agency is also soliciting comment on this notice. A further notice will be published, however, only if the public comment indicates a need to reconsider the conclusions presented here. This determination has no effect on the regulation of chloroprene as a volatile organic compound in order to attain the national ambient air quality standards (NAAQS) for ozone. In addition, this determination does not preclude any State or local air pollution control agency from specifically regulating emission sources of chloroprene.

DATES: Written comments pertaining to this notice must be received on or before November 26, 1985.

ADDRESSES: Submit written materials (duplicate copies are preferred) to: Central Docket Section (A–130), Environmental Protection Agency, Attn: Docket No. A–85–11, 401 M Street, SW, Washington, DC. Docket A–85–11, which contains information relevant to this decision, is located in the Central Docket Section of the U.S. Environmental Protection Agency, West Tower Lobby Gallery I, 401 M Street, SW, Washington, DC. The docket may be inspected between 8:00 a.m. and 4:30 p.m. on weekdays, and a reasonable fee may be charged for copying.

FOR FURTHER INFORMATION CONTACT: Robert M. Schell, Pollutant Assessment Branch (MD–12), Strategies and Air Standards Division, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711 (Telephone: 919–541–5645 commercial/629–5645 FTS).

SUPPLEMENTARY INFORMATION: The EPA initiated this assessment because of the potential for human exposure to chloroprene as a result of emissions from industrial sources, because of reports of adverse health effects, and because of its chemical structural similarity to vinyl chloride, which is a carcinogen. As a first step in this process, a Health Assessment Summary (HAS) for chloroprene was drafted, summarizing properties, sources, emissions, and health effects. Because of the lack of available data, a full health assessment document was not written nor was the HAS reviewed by the Science Advisory Board (SAB), a group of independent scientists. The HAS was reviewed both internally and externally for technical quality and conclusions.

Other Federal Activities

Other EPA review activities involving gathering information on chloroprene are being conducted under section 4 of the Toxic Substances Control Act (TSCA). The EPA is committed to further analysis of new data as it becomes available.

Introduction

Chloroprene (2-chloro-1,3-butadiene), a monomer used in the manufacture of synthetic rubber, is a volatile and highly reactive chemical with an estimated residence time in the atmosphere of 4.8 hours. Chloroprene's only known use is in the manufacture of polychloroprene synthetic rubber. Solid polychloroprene, also known as neoprene synthetic rubber, is used in the automotive industry for tubing, belts, and gaskets, in the construction industry, and in the manufacture of wire and cable jackets, and consumer goods. Liquid or latex polychloroprene is used in adhesives and as fabric coatings.

Health Effects

Carcinogenicity/Mutagenicity— Epidemiological studies conducted in the Soviet Union have reported conflicting results (IARC, 1979; HAS). Results from studies of chloroprene workers in this country are suggestive of a slightly increased cancer risk (Infante, 1977; Pell, 1978; HAS). Serious limitations in data regarding the likelihood of chloroprene being carcinogenic for humans preclude the development of conclusions (Infante, 1977; IARC, 1979, HAS).

Tumorigenic effects of chloroprene have been studied in mice following skin application and in rats by oral, subcutaneous, and intratracheal administration. No conclusive tumorigenic effects were found. However, the compound was reported to increase the rate of tumor growth of transplanted tumor cells possibly due to immunosuppression. None of these studies are adequate for evaluating the carcinogenicity of chloroprene in experimental animals, as they lacked adequate durations of exposure and the experimental details reported are not adequate (HAS). Based on both the International Agency for Research on Cancer (IARC, 1979) and EPA's proposed weight-of-evidence criteria (FR 49 46294–46301; November 23, 1984), the available data are inadequate to evaluate the carcinogenic potential for chloroprene (IARC Group 3, EPA Group D).

Chloroprene has been reported to be mutagenic in bacteria. Poor quality Russian studies have reported mutagenesis in other systems (HAS).

Acute Toxicity (less than 24 hours)— The HAS reports human exposure to chloroprene at 970 parts per million (ppm) for less than 15 minutes causes giddiness and nausea (Nystrom, 1948; HAS). The threshold limit value (TLV), recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) is 10 ppm averaged over 8 hours.

Based on various species and exposure paradigms Von Oettingen et al. (1936) concluded that several thousand ppm for several hours should be considered lethal, 280 ppm should be considered dangerous and 80 ppm may cause less severe toxic effects. Recent better controlled studies indicate lethal acute inhalation exposures of rats at 2280 ppm for 4 hours (Clary et al., 1978, HAS). A single inhalation exposure of 225 ppm for 4 hours has been reported to result in liver damage in rats. This effect was not seen after exposure to 150 ppm for 4 hours (Plugge & Jaeger, 1979).

Subchronic Toxicity (less than 3 months but greater than 24 hours)— Exposures of humans to 55 to 333 ppm for a minimum of one week have been reported to produce fatigue, pressure and chest pain (electrocardiograms showed no abnormalities), dermatitis and hair loss in a substantial number of workers (Nystrom, 1948; HAS).

Exposure of rats and hamsters to chloroprene (39 ppm for 6 hours per day, 5 days per week for 4 weeks) resulted in slight growth depression, behavioral effects, eye and skin irritation. At higher concentrations (625 and 160 ppm) tissue damage, especially to lung and livers, and mortality were observed (Clary et al., 1978; HAS). Exposure of both male and pregnant female rats to 25 ppm for 4 hours per day for 12 to 22 days resulted in no obvious toxic effects for parents or embryos, including teratogenic effects (Culik et al., 1978; HAS).

Chronic Toxicity (Noncarcinogenic)— There are no epidemiological studies with reported exposure levels. In one

study, biochemical and hematological evaluation of workers exposed to chloroprene showed no significant differences compared to controls (Gooch and Hawn, 1981; HAS). However, another study suggested that exposure to chloroprene may contribute to liver function abnormalities (Ward et al., 1981; HAS). Exposure concentrations were not reported in either study.

Reproductive Toxicity—A number of papers, chiefly from the Soviet Union, have consistently reported reproductive toxicity in the range of 1–10 ppm. These studies are poorly reported and consequently are inadequate for risk assessment purposes. Attempts have been made to validate these studies but these attempts have largely been unsuccessful (NIOSH, 1977). One animal study performed in this country has not substantiated these effects (Culik et al., 1978; HAS). Based primarily on these Soviet studies and the lack of additional information, the National Institute of Occupational Safety and Health has recommended a 1 ppm, 15-minute ceiling for workplace chloroprene exposure.

Overall, the data available on potential reproductive hazard, carcinogenicity or other toxicity subsequent to chronic chloroprene exposure is inadequate to support a decision to regulate under the Clean Air Act.

Sources and Emissions

Limited data are available to determine the occurrence of chloroprene in the environment (SAI, 1982; Radian, 1985). Given the short predicted residence time in the atmosphere (4.8 hours), it is unlikely that detectable amounts would be observed distant from chloroprene emitting sources (Cupitt, 1980; HAS). Only four facilities manufacture or use chloroprene in the United States. Annual production is estimated to be 1.2×10^5 megagrams per year (49 FR 46938, November 29, 1984). Annual emissions are estimated to be 770 megagrams per year (Radian, 1985). Limited measurements have been reported for ambient chloroprene concentrations. Those made in Deer Park, TX were reported as 73.9 and 1111.1 ppt (2 hour sampling periods) (Pellizzari et al., 1979; HAS). Measurements made at an industrial waste treatment facility in Houston, TX ranged from less than 0.02 to 0.40 ppm with approximately 7 hour sampling periods (Timm, 1985).

Exposure Estimates and Risks To Public Health

A preliminary analysis was conducted to examine the potential for short-term concentrations of chloroprene in the ambient air surrounding industrial facilities to approach or exceed those concentrations at which noncarcinogenic health effects have been reported. This is a rough analysis, which uses worst case meteorological conditions in a conservative screening model. This analysis indicated that ambient concentrations resulting from continuous routine emissions would not be expected to approach levels at which systemic toxicity has been reported as a result of acute or subchronic exposures. Approximately 4.7 million people live within 50 kilometers of the four domestic chloroprene producing facilities. A 15-minute concentration of 4.5 ppm, a 4-hour concentration of 2.8 ppm, a 6-hour concentration of 2.6 ppm and an 8-hour concentration of 2.5 ppm were estimated using this short-term exposure model. Given the health effects data, it appears that the potential for systemic toxicity to occur in the general population subsequent to acute or subchronic chloroprene exposures is low (see Table 1) (Cote, 1985). As stated earlier, the health effects data for chronic exposure are inadequate to assess risk.

Discussion

Based on currently available data, EPA has determined that no regulation directed specifically at chloroprene is appropriate at this time under the CAA. Unless additional information becomes available during the public comment period, the effect of this notice is to remove chloroprene from EPA's list of potential air toxics currently under assessment.

In order to improve upon the health effects information base for chloroprene, the National Toxicology Program is testing chloroprene carcinogenicity and reproductive hazard in animal bioassays; however, results are not expected before 1987. Further assessment and review of chloroprene will be initiated upon completion of these studies.

Due to uncertainties in assessing the risk of health effects, the EPA is soliciting health effects and exposure information on chloroprene as well as comments on the analysis and conclusions on which this notice is based. A further notice will be published, however, only if public comments indicate a need to reconsider these conclusions. In addition, if significant new information becomes available, the Agency will reexamine the need to regulate chloroprene.

This notice has no effect on the regulation of chloroprene as a volatile organic compound in order to attain the NAAQS for ozone. In addition, this notice does not preclude any State or local air pollution control agency from specifically regulating emission sources of chloroprene.

Dated: September 17, 1985.

Lee M. Thomas,

Administrator.

TABLE 1.—COMPARISON OF SYSTEMIC TOXICITY, MODELING AND MONITORING DATA

NOEL or TLV[1]	Lowest observed effect level	Modeling	Monitoring
Acute:			
10 ppm (8 hr TLV).	972 ppm × 15 min. (human).	4.5 ppm × 15 min.	1.1 ppb.
150 ppm × 4 hrs (rats).	225 ppm × 4 hrs (rat).	2.8 ppm × 4 hrs.	1.1 ppb.
Subchronic:			
10 ppm (8 hr TLV).	55 ppm × 8 hr (1 wk—humans).	2.5 ppm × 8 hrs[2].	1.1 ppb.
25 ppm × 4 hr (4 wks—rats).	39 ppm × 6 hr (4 wks—rats).	2.6 ppm × 6 hrs[2].	1.1 ppb.

[1] The American Conference of Governmental Industrial Hygienists' recommended threshold limit value (TLV) is presented for comparison in addition to the no observed effect level (NOEL) in animals.

[2] Subchronic exposure estimates were not modeled. The 8-hour and 6-hour acute exposure estimates are used for comparison and are shown here.

Information on health effects subsequent to chronic exposure is inadequate for risk assessment purposes.

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