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CHEMICAL MANUFACTURERS ASSOCIATION

December 28, 1989

Dear Vinyl Chloride Panel:

Enclosed, for your information, is a copy of the summary and conclusions of the BUA Report on Vinyl Chloride (Beratergremium fur Umweltrelevante Altstoffe, Federal Republic of Germany, 1989). The BUA is recommending no additional ecology or toxicology testing in the FRG.

Please call me at 202/887-1293 if you need additional information.

Sincerely,

Susan R. Howe
Manager
Vinyl Chloride Panel

2501 M Street, NW, Washington, DC 20037 202-887-1100 Telex 89617 (CMA WSH)

For Distribution by CMA
CHEMSTAR DIVISION
From: Susan B Hawe
Re: No. VC Panel
Date: 12/26/89

BUA Report on Vinyl Chloride

Summary and Conclusions

ECOLOGICAL ASPECTS

Production, entry into, occurrence and distribution in the environment

In the Federal Republic of Germany, the vinyl chloride production sums up to nearly 1.5 million tons in 1987. About 99 % are used for subsequent polymerization to polyvinyl chloride and copolymers. The remainder (about 9,500 - 12,700 tons) is required for the synthesis of 1,1,2-trichloroethane.

For the region of the Federal Republic of Germany, the vinyl chloride discharge into the atmosphere can be estimated to about 330 tons per annum, 9 % of which are due to the monomer production, 91 % are due to the production of polyvinyl chloride. About 4 tons per year are released into the air when processing PVC, owing to its residual monomer content.

The entry of vinyl chloride into waste water can be estimated at 25 - 30 tons in 1987. About 7 % of the amount discharged can be attributed to the production of the monomer itself, whereas about 93 % is associated with the production of polyvinyl chloride. The waste water is treated in biological sewage treatment plants in which vinyl chloride evaporates as a result of its physical properties. Generally, vinyl chloride is not detectable in the effluent.

In surface waters, vinyl chloride can be traced in individual cases only. For the Rhine, maximum concentrations of 1 $\mu g\ l^{-1}$ have been reported before 1978; in tributaries of the Rhine, maximum concentrations of 5 $\mu g\ l^{-1}$ of vinyl chloride have been detected.

In the Federal Republic of Germany, vinyl chloride generally cannot be detected in potable waters. Concentrations of up to 1.7 µg l^{-1} recorded in isolated cases seem to be due to prolonged exposure times in PVC pipes with an elevated background level of non-polymerized vinyl chloride.

Vinyl chloride forms as an intermediate during the biological breakdown of tetrachloroethane and trichloroethane. In these cases, it can be detected in groundwater and at dumping sites in seeping water and as a gas component.

Recent investigations have revealed vinyl chloride in the atmosphere in concentration ranges from "not detectable" (< 0.3 µg m^{-3}) to 15 µg m^{-3}. 99 percentile values of up to 69 µg m^{-3} have been reported.

Due to its physico-chemical properties, vinyl chloride will mainly evaporate into the atmosphere.

Degradation

The abiotic degradation in the atmosphere via photochemical-oxidative reaction most probably forms the main degradation pathway. For this process, the half-life can be calculated at 2.2 to 2.7 days.

Based on available experimental data the half-life of the hydrolytic degradation of vinyl chloride dissolved in water can be estimated to several years.

Biodegradation of vinyl chloride takes place by means of specialized, adapted bacteria.

Bioaccumulation

The bioaccumulation factor ranges from < 10 for fish to 15 - 30 for invertebrates and 40 for algae.

These results indicate a low potential for bioaccumulation and biomagnification.

Ecotoxic effects

Gaseous vinyl chloride displays mutagenic effects towards plant cells, the threshold value is 194 mg m^{-3}.

During anaerobic sewage treatment, a first retardation of gas production can be seen between 5 and > 64 mg l^{-1} vinyl chloride.

The 96 h LC_{50}-values for fish lie between 1.1 g l^{-1} and 1.2 g l^{-1}.

The present data on the ecotoxicological properties of VC are limited to the results of laboratory studies. Data obtained by studies in the outdoor environment are not available.

TOXICOLOGICAL ASPECTS

Vinyl chloride depresses the central nervous system. Due to its cardiac side-effects, it has not been used as an anaesthetic. The acute toxicity of vinyl chloride is low; following a 2-hour inhalation exposure, the LC_{50} values were 294 mg l^{-1} for mice, 390 mg l^{-1} for rats, 595 mg l^{-1} for guinea pigs and 295 mg l^{-1} for rabbits. Liquid vinyl chloride causes hypothermia of the skin, which is similar to frost injuries of the second degree. Furthermore, vinyl chloride can cause temporary injury to the eye.

Vinyl chloride is oxidatively metabolized to chloroethylene oxide (chlorooxirane) catalyzed by cytochrome P-450 dependent monooxygenases. Since this reaction is saturable at relatively high concentrations (2.56 mg/l) it is regarded as the limiting step in the metabolism of vinyl chloride. The alkylating potential of chloroethylene oxide towards DNA suggests this metabolite being responsible for the mutagenic and probably also the carcinogenic effects of vinyl chloride. Metabolically unchanged vinyl chloride is almost quantitatively exhaled.

As additional metabolites chloroacetaldehyde, chloroethanol and chloroacetic acid were also identified.

The metabolic formation of chloroethylene oxide per unit of time is 5 - 10 fold higher in rats and mice than in man. This phenomenon can be explained by the higher activity of the monoxygenase system in rodents compared to man.

Vinyl chloride showed mutagenic effects in laboratory animals and in various short-term assays in vitro. The ultimate mutagenic metabolite is chloroethylene oxide (vide supra).

Following inhalation vinyl chloride induced mainly haemangiosarcomas of the liver (primary target organ) in rats, mice and hamsters, with female rats and mice being clearly more susceptible than males. Furthermore liver tumours, extrahepatic angiosarcomas, lung and mamma tumours were observed in rats and mice. The incidence of hepatocellular carcinomas was higher in male animals.
Zymbal gland tumours, neuroblastomas and nephroblastomas were exclusively observed in rats, whereas forestomach tumours were seen in rats and hamsters. In addition some melanomas were histopathologically identified in hamsters.

Following oral application a similar tumour spectrum was observed.

The induction of tumours appeared over a wide dose range. The minimal tumourigenic concentration after inhalation of vinyl chloride was 0.0256 mg l^{-1} in rats, 0.13 mg l^{-1} in mice and 1.28 mg l^{-1} in hamsters. After oral application (vinyl chloride monomer adsorbed on PVC) a minimal effective dose of 1.7 mg kg^{-1} body weight was observed in rats.

Animal experiments gave no evidence of teratogenic effects of vinyl chloride.

Long term occupational vinyl chloride exposure at high concentrations occurring in some workplaces before the 1970's caused haemangiosarcomas of the liver and damage in other organs. The range of diseases observed in man later was collectively called "vinyl chloride disease". The main effects included periportal fibrosis of the liver, changes in the distal vasculature of the hands, manifested by the development of Raynaud's phenomenon, scleroderma or acro-osteolysis.

Since 1975, when the occupational vinyl chloride concentrations were reduced to some ppm, no new cases of the "vinyl chloride disease" have been reported.

A statistical significant correlation between vinyl chloride exposure in the workplaces and the development of angiosarcomas of the liver, a seldomly found tumour in man, has been established.

Since then, occupational exposure has been largely reduced due to technical safety measures. The "Technische Richtkonzentration (TRK-Wert)" in West Germany is now 5 mg m^{-3} (8 mg m^{-3} in existing VC monomer and PVC production plants).

Since the first appearance of the haemangiosarcomas in man, about 140 cases have been observed worldwide until 1988, including 32 cases in West Germany. Single tumours are expected even in the next decade, due to the long latency period and the high vinyl chloride concentrations in workplaces before 1974.

RECOMMENDATIONS

Ecology

Only few data are available regarding ecotoxicological effects of vinyl chloride. Under this aspect, a comprehensive assessment is not possible.

However, due to the physico-chemical properties (rapid evaporation from aqueous sources) vinyl chloride will not accumulate in water. Negative long term effects are thus not to be expected here.

Out of this reason more experiments regarding the ecotoxicology of vinyl chloride do not seem to be appropriate.

Due to its short half life in the atmosphere no additional experiments with gaseous VC are necessary.

Toxicology

The toxicological knowledge acquired to date is regarded as sufficient and therefore no additional testing is recommended.