



TECHNICAL SUPPORT DOCUMENT

**PROPOSED IDENTIFICATION OF
VINYL CHLORIDE
AS A TOXIC AIR CONTAMINANT**

**SCIENTIFIC REVIEW PANEL VERSION
PART B**

AUGUST 1990

**State of California
Air Resources Board
Stationary Source Division**

SPI-05579

HEALTH EFFECTS OF AIRBORNE VINYL CHLORIDE

CALIFORNIA DEPARTMENT OF HEALTH SERVICES

August 1990

SPI-05580

1.0 EXECUTIVE SUMMARY

Vinyl chloride is a short-chain halogenated hydrocarbon used predominantly in the manufacture of polyvinyl chloride and various packaging and construction products. Vinyl chloride has a very low degree of acute toxicity, with two-hour inhalation LD₅₀ values ranging from 27,419 ppm in mice to 236,215 ppm in rabbits and guinea pigs. Exposure to high concentrations can lead to narcosis, cardiovascular and respiratory irregularity, convulsions, cyanosis and death. Several human deaths have been attributed to occupational exposure to very high levels of vinyl chloride. Autopsies of these patients revealed congestion of the liver, spleen and kidneys. Acute toxicity symptoms are thought to occur above 100 ppm.

Chronic exposure of workers to vinyl chloride has been shown to lead to "vinyl chloride disease", characterized by occupational acro-osteolysis, vasospasm of the hands similar to Raynaud's syndrome, dermatitis, circulatory and central nervous system alterations, thrombocytopenia, splenomegaly and changes in liver function. Eight symptoms commonly reported by workers exposed to vinyl chloride (including dizziness, headaches and nausea) were observed even at dose levels below 50 ppm.

Vinyl chloride has been shown to induce cancer in animals in utero, but has not been shown to cause any other reproductive or developmental effects in rats, mice and rabbits. Epidemiologic studies of families of vinyl chloride workers or communities having vinyl chloride processing facilities

Although pharmacokinetic studies in humans exposed to vinyl chloride are rare, limited evidence indicates that, following inhalation of low levels of vinyl chloride (3 to 24 ppm), up to 71% (with a mean value of 42%) of the given dose may be absorbed. Vinyl chloride absorption appears to depend on its metabolism, which is a dose-dependent, saturable process. Due to saturation of the enzyme systems responsible for the metabolism of vinyl chloride (cytochrome P-450 and alcohol dehydrogenase), exposure to concentrations above approximately 250 ppm would not necessarily be expected to lead to a perceptibly increasing incidence of tumor development. Metabolism of vinyl chloride leads to formation of chloroethylene oxide and chloroacetaldehyde, two reactive intermediates which undergo covalent binding to cellular macromolecules and are thought to be responsible for the toxic effects of vinyl chloride. These and other metabolites may be further metabolized and excreted in the urine. Unmetabolized vinyl chloride is eliminated primarily in exhaled air.

Vinyl chloride is mutagenic in both prokaryotic and eukaryotic test systems, with significantly greater genotoxicity seen after metabolic activation. DHS staff have found no evidence of a carcinogenic threshold level and the staff recommends that vinyl chloride be considered as not having a threshold for carcinogenicity.

Several studies of carcinogenicity of vinyl chloride in animals and in occupationally exposed workers have been analyzed for risk assessment purposes. The lowest lifetime equivalent concentration associated with an increased incidence of tumors in laboratory animals is 0.06 ppm or 6 to 60-fold above potential human exposure concentrations. Although measurements

The model predicts that the 95% upper confidence limit on cancers due to lifetime exposure of 131,000 residents to 1 ppb would be in the range of 3 to 36. Based on the finding of vinyl chloride-induced carcinogenicity and the results of the risk assessment, DHS staff finds that vinyl chloride is an air pollutant which may cause or contribute to an increase in mortality or an increase in serious illness, or which may pose a present or potential hazard to human health.

1.1 Vinyl Chloride Highlights

I. National and International Evaluation (Other Agencies' Evaluation)

A. International Agency for Research on Cancer (IARC)

1. Short-Term Tests: Sufficient evidence of mutagenic activity exists, both with and without an exogenous metabolic activation system.
2. Animal carcinogenicity bioassays: Sufficient evidence of animal carcinogenicity by oral administration or inhalation exists.
3. Human evidence: Sufficient evidence of carcinogenicity to humans exists. Occupational exposure to vinyl chloride has been linked with development of angiosarcoma of the liver, and has been associated with tumors of the brain and lung and of the hematopoietic and lymphatic

II. Exposure Sources

A. Air Levels

1. Throughout 1987 the South Coast Air Quality Maintenance District monitored near two landfill sites in the Los Angeles area. The highest annual average obtained at any of three stations near the BKK site was 2.6 ppb, and the highest annual average at any of three stations near the OII site was 2.0 ppb.

III. Quantitative Risk Assessment

A. Range of Extrapolation: Animal to human exposures in air for calculated lifetime daily exposure.

1. Experimental to ambient: Vinyl chloride has not been detected in ambient air, except at "hot spots".
2. Experimental to "hot spots": The lowest exposures in the animal studies are approximately 10- to 20-fold higher than the highest residential exposures.

B. Range of Risks:

The human risks associated with the equivalent of a continuous, lifetime exposure to vinyl chloride have been estimated using the linearized multistage model from both animal



A Division of The Society of The Plastics Industry, Inc.

November 27, 1990

Mr. Ladd Smith
Occidental Chemical Co.
360 Rainbow Boulevard South
Niagara Falls, NY 14302

Dear Ladd:

As follow up to our conversation this morning on the upcoming hearings of the California Air Resources Board on the listing of vinyl chloride as an air contaminant, enclosed is the material forwarded on November 5 to members of the VI Health, Safety & Environment Committee. If you would also like the background documents, let me know.

If after reviewing this information you believe that the Vinyl Institute should plan on submitting comments, let me know. The time is growing short for preparing comments and I would need some assistance in their preparation. Since I spoke with you earlier, Frank Borrelli (Chairman of the VI Health, Safety & Environment Committee) and I have spoken and are in the process of making additional follow up calls on my November 5 memo.

It was nice talking with you again.

Sincerely,

Meredith N. Scheck
Assistant Director

CC: J. Coburn
F. Borrelli, Georgia Gulf

SPI-05585



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