The Vinyl Institute

TECHNICAL INFORMATION

COMMUNITY HEALTH EFFECTS OF VINYL CHLORIDE

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I. SUMMARY

This document reviews the toxicity and human health effects of ambient exposure to vinyl chloride (VC), the raw material used in the production of polyvinyl chloride (PVC). It summarizes the extensive and rigorous federal regulation of the VC/PVC industry, and compares quantitative risk assessments with actual health observations of individuals in non-occupational settings.

A review of the world scientific literature shows no community health impacts associated with exposure to VC emissions from VC/PVC manufacturing facilities.

II. INTRODUCTION

Vinyl chloride is the basic building block for producing the most versatile plastic yet developed -- polyvinyl chloride and its copolymers with other monomers. Most of the seven billion pounds of VC produced annually in the United States is converted into PVC used in thousands of products in the home and in industry -- products such as wallcoverings, upholstery, flooring, house siding, water pipes, sewer pipes, luggage, clothing, automotive parts, medical devices, food wrap, windows, doors, wire insulation, garden hoses, and phonograph records.

PVC is a polymer produced from VC through a chemical reaction called polymerization. VC is converted into PVC by suspension, emulsion, bulk or solution polymerization methods. PVC resins can be extruded, molded or calendared into diverse shapes, sizes, and colors. Mechanical characteristics can be controlled to produce forms that are rigid, flexible, or in a liquid form such as latexes, pastes, and adhesives.

Vinyl chloride became of industrial importance approximately fifty years ago when Semon (1933) discovered that the polymer could be converted into useful articles by plasticization with phthalate esters. Commercial development began first in Europe and then in the United States in the late 1930's. It was not until the early 1950's that widespread consumer applications developed. PVC is now a mature product, and its growth rate falls in step with the Gross National Product.

III. HEALTH HISTORY

Acute Toxicity

Vinyl chloride is a strong anesthetic at 8-12% in animals and humans. Death follows rapidly after unconsciousness sets in if exposure is not reduced quickly (Patty et al, 1930). No major histological changes were reported after 100 days at exposures of 50,000 ppm (Kuebler, 1964). Reversible liver effects at 100-500 ppm led to a recommendation of a 50 ppm TWA exposure limit (Torkelson, Oyen, and Rowe, 1961), but the American Conference of Governmental Industrial Hygenists adopted instead a recommendation by Yale scientists of 500 ppm. This is the value later accepted by OSHA and it served until 1974. Lehman and Flury (1943) termed vinyl chloride to be "one of the least dangerous of the chlorinated hydrocarbons".

There are no other known acute human physiological effects from vinyl chloride exposure. The odor threshold is about 1,000 ppm. The high heat of vaporization causes a substantial part of a large spill to liquify and presents the danger of frostbite. Vinyl chloride is flammable over the range of 3.6-33% in air, and extreme care must be taken to avoid spills and leaks for that reason. Most measurement and warning systems were designed to hold plant atmospheres below the flammable limits. Retrospective estimates of typical time-weighted average personal exposures for polymerization workers in England have been estimated (Barnes, 1980) as follows:

1945 to 1955	-	1,000 ppm (or above)
1955 to 1960	-	400 to 500
1960 to 1970	-	300 to 400
Mid 1973	-	150
1975	-	5

In some jobs, particularly the cleaning of polymerization reactors, exposures in the thousands of ppm range were experienced for short periods. (See Purchase, et al, 1985 and Barr, 1986 for reviews of the toxicity of VC).

Chronic Health Effects

The first clear indication of chronic health problems associated with VC came in the 1960's in men who entered VC polymerization reactors to remove build-up of polymer from the walls. Some of these men developed acro-osteolysis, a disease resulting in softening of bones in the fingers (Suciu, et al, 1963; Harris and Adams, 1967; Cook, et al, 1981). Modification of working practices has led to the elimination of this disease in workers in PVC plants. In the late 1960's, Professor P.L. Viola of the Solvay Company tried to reproduce acro-osteolysis in rats by exposing them to high concentrations of VC for long periods. He failed to produce acro-osteolysis, but he reported an increase in incidence of a variety of tumors at various sites. For the first time, it had been suggested that VC was an animal carcinogen. (Viola, 1969, 1970; Viola, Bigotti and Caputo, 1971).

As a direct result of the Viola work, four West European VC/PVC manufacturing companies in Italy, France, Belgium, and England supported a comprehensive study of the animal toxicology of VC by Professor C. Maltoni, Director of the Institute of Oncology at Bologna. Maltoni's work which extended over eight years has proved to be the most comprehensive study of VC toxicology (Maltoni et al, 1984). By the end of 1972, Maltoni had found a rare tumor, angiosarcoma of the liver (ASL), in some of the exposed rats and confirmed that VC is indeed an animal carcinogen. These early findings were reported at an international symposium in 1973 (Maltoni, 1977). Maltoni recommended epidemiological investigations and medical controls of exposed workers and early in 1974, a U.S. company announced that they had found three ASL cases in employees at one of their PVC polymerization plants. This finding led to the conclusion that VC was a human carcinogen because it gave rise to a rare tumor whose only other known etiological agents in man were thorium dioxide, arsenic and possibly anabolic steroids.

ASL is a very rare tumor. Less than 20 cases per year from all these causes occur in this country. A review (Popper, et al, 1978) of all cases reported in the United States for the period 1964-1974 revealed 167 cases, of which 19 were ascribed at that time to occupational VC exposure, 26 to thorium dioxide given medically, and 9 to arsenic in Fowler's solution, also used medically. The remainder were of unknown etiology, with no connection to VC. The high level of interest in this specific tumor is such that any subsequent cases associated with environmental exposure to VC would most certainly have been reported, and none have. For a time, NIOSH published a summary of VC-related cases (Falk, et al, 1981), but this task was taken over first by John Stafford of ICI, England (Foreman, et al, 1985) and later by Brian Bennett also of ICI. The 1986 update of VC-related ASL cases shows a total of 38 cases in the United States and 120 worldwide. All of these cases involve high occupational exposures to VC.

The average ASL latency period (years from first exposure to diagnosis) in the United States has been 25 years, but with a median of about 22 years. The latency period in Europe, particularly in Germany, has been somewhat shorter, approximately 19 years. All the U.S. occupational cases, and almost all such cases in the rest of the world are closely associated with the job of reactor cleaning, which was once done manually at the end of the polymerization cycle. There is clustering of cases in relatively few plants and the majority of plants have had no cases. Differing work programs and job progressions may have had some effect on reducing rates at various plants.

An industry-sponsored epidemiological survey of workers in the VC/PVC industry covered 8,384 men with at least one year of exposure before 1973 (Tabershaw and Gaffey, 1974). The expected excess of ASL was found. There were also suggestions of an excess of cancers at other sites. This study was expanded to 10,173 workers (Cooper, 1981), where suggested excess of brain and respiratory cancers continued to be seen without, however, an association between the brain cancer and exposure. In addition, most of the lung cancer cases come from the same facility, with many plants having no cases. A follow-up study of this expanded cohort to determine the status of the workers as of the end of 1980 is underway.

Several studies have been made of the general population using ASL as the marker disease in an effort to detect an association with possible environmental exposure to VC. There was no association with living near a plant manufacturing or using VC in the general U.S. survey conducted by the Center For Disease Control (Popper et al, 1978; Falk, et al, 1981). Brady et al, (1977) surveyed 26 ASL deaths in New York State between 1970 and 1975, and found five who lived nearer plants handling VC than did their matched controls, but could not establish a direct connection with the disease to exposure. Ten cases of ASL in Wisconsin were examined for possible connection with VC exposure, and none was found (Fiechtner et al, 1976). Baxter et al, (1977) found no relationship between distance of residence from VC emitters and the 47 cases of ASL in the general population of Great Britain reported in 1963-1973. A later update (Baxter et al, 1980) found one case where the person had lived the last six years of his life near a PVC plant and three cases where the men had worked in the plastics fabricating industry but for whom there were no records to indicate exposure to VC.

The lack of relationship between residence near vinyl chloride operations and cases of unknown etiology was confirmed. Saric et al, (1976) studied the deaths during the years 1968-1971 in an area surrounding a PVC plant that had been in operation since 1949 and in which three workers had died of ASL. No relationship was found for liver or for lung or bronchial cancer and place of residence for the general population. A similar study for communities near a Swedish plant that had operated since 1945 and had found four ASL cases showed (Elinder and Pershagen, 1978) no unexpected elevation of fetal mortality, deaths from all cancers, or cancer of the liver or lungs during the years 1961-1974. Pancreatic cancer in males was elevated in the age group over 60. All ASL cases in Holland since 1950 (27 cases) were studied, and none had any traceable contact with VC (Dalderup et al, 1976). Iturra (1976) observed an excess of cancer deaths in a city in Canada with a PVC plant compared to a similar nearby city. This difference was principally found in males aged 20 to 64, which is not indicative of a general pollution effect. The author drew no conclusion as to why the condition existed.

Representatives of the Environmental Protection Agency have stated that it has been unable to establish a link between living near VC manufacturing and using plants and ASL.

There are about 20 cases of ASL per year in the United States that cannot be ascribed to one of the known causes of the disease. There are also about 5 in Europe each year. Accordingly, there will be one case of ASL among the 5 million - 5 mile neighbors of VC/PVC facilities about every two years by chance alone. This has been seen in the studies in New York by Brady, et al, (1970), and in Connecticut (Heath and Landrigan, 1974). These states have cancer registries, which are of great value. In one case, a jury award was made to the estate of an individual who died of ASL, and who had lived the last four years of his life near a PVC plant. Inasmuch as that person also had occupational exposure to VC and exposure to other ASL causative agents, it cannot be concluded that ambient VC exposure caused his ASL (In re Grasso, Civil Action No. 78-1562, D.N.J.).

A thorough study (Chiazze, et al, (1977), Chiazze, (1980)) of more than 15,000 employees of PVC fabricators found no evidence of VC-related health effects in that group, which was estimated to have been exposued to at least 15 ppm VC for many years.

The disease ASL is often difficult to diagnose (Block, 1974; Heath, Flak and Creech, 1975), is almost invariably fatal within a short time, and presents a variety of symptoms, including portal fibrosis and hypertension with splenomegaly and varices, proliferation of the sinusoidal lining, megalocytosia, and thrombocytopenia (Thomas and Popper, 1975; Gedigk et al, 1975). Metastasis is frequently involved. These symptoms are very similar to those seen in the mouse (Schaffner, 1978) and rat (Feron and Krees, 1979) and the pathology also is similar (Gordon et al, 1975). No really adequate early warning tests have been devised (Whelan et al, 1976; Langbein et al, 1983; Tamburro and Greenberg, 1981), although the gammaglutamyl transpepsidase test is promising, together with ICG clearance and SGOT. Radiographic liver scans and tomography and sonography (Koischwitz et al, 1981) are said to be useful confirmatory tests.

In summary, VC is a classical procarcinogen, and is clearly a human carcinogen, causing ASL in a small percentage of highly exposed workers. There is suggestive evidence that it may be a weak general carcinogen at high concentrations, perhaps through an immunosuppressive mechanism, but more data are required to confirm this suspicion. Several studies of large populations have not shown a connection between general ambient exposure and an increased incidence of cancer.

IV. FEDERAL REGULATION OF VC/PVC INDUSTRY

The primary federal agencies regulating the VC/PVC industry are the Occupational Safety and Health Administration (OSHA), which is part of the U.S. Department of Labor, the U.S. Environmental Protection Agency (EPA), and the Food and Drug Administration (FDA). OSHA regulation focuses on worker health while EPA addresses the control of chemicals outside the workplace. FDA oversees uses of PVC that involve foods, drugs, cosmetics, and medical devices.

A. The Occupational Safety and Health Administration

The allowable occupational exposure for vinyl chloride of 1 ppm on an 8-hour time weighted average (TWA) is set by the OSHA workplace standards at 29 C.F.R. 1910.1017. This was adopted in 1974, after extensive public hearings, and became effective in April 1975. OSHA first set an emergency temporary standard of 50 ppm and proposed a permanent limit of nondetectable exposure by a test sensitive to 1 ppm. OSHA then promulgated a final standard of an 8-hour TWA of 1 ppm, and a 15-minute ceiling of 5 ppm.

In brief, the regulation sets:

1. A level of 0.5 ppm VC below which no action is required. This generally exempts most PVC fabrication plants and laboratories and many monomer plants.

2. A regulated area where exposures are above 0.5 ppm which restricts entry to authorized persons.

3. Medical examination requirements and exposure record retention for specified employees.

4. A list of acceptable respirators.

5. Monitoring and alarm systems for the workplace, and routine measurement of worker exposure.

6. Labeling and signs for regulated areas and containers of vinyl chloride and PVC.

7. Work procedures for hazardous operations.

8. Training programs for employees.

OSHA also has a Hazard Communication Standard (HCS), 29 C.F.R. 1910.1200, which provides labeling requirements complementary to the OSHA Vinyl Chloride Standard. Articles made from PVC are exempt from labeling requirements under the standard.

B. Environmental Protection Agency

EPA regulates the release of vinyl chloride under several statutes, including the Clean Air Act, Clean Water Act, Safe Drinking Water Act, Resource Conservation and Recovery Act (RCRA), Comprehensive Environmental Response, Compensation and Liability Act (CERCLA or Superfund), and the Toxic Substances Control Act (TSCA).

1. Air Standard (40 CFR 61.60)

The EPA standard established in 1976 specified the following conditions:

a. Fugitive emissions controls by leak patrols and design standards for pump and compressor seals, agitators, and loading devices.

b. Work practices for vessel openings and sampling.

c. Stripping requirements for residual monomer in resins and wastewater.

d. Abatement of specified point source emissions to 10 ppm.

e. Prohibition of relief valve discharges, except for emergencies.

f. Extensive monitoring, reporting and recordkeeping requirements.

g. Specific analytical procedures.

EPA estimated that this standard would result in a 95% reduction of VC emissions to the atmosphere from VC/PVC manufacturing plants and reduce the 5 mile annual average VC ambient air concentration from 17 parts per billion (ppb) to less than 1 ppb.

2. Water Regulations

Vinyl chloride is listed as a priority pollutant under Section 307(a) of the Clean Water Act, and a Water Quality Criteria Document has been prepared. This subjects VC and PVC manufacturing plants to special considerations when waste water discharge permits are issued pursuant to EPA regulations.

As part of its regulation of carcinogens in drinking water, EPA has published a final Recommended Maximum Contaminant Level (RMCL - a non-binding guideline) for VC in drinking water of zero (see 50FR 46880, Nov. 13, 1985 for this amendment to 40 CFR 141.50.)

However, EPA indicated that a "justifiable" way to determine the absence of vinyl chloride would be by setting a defined, state-of-the-art detection limit sensitive to approximately 1 ppb. (49FR 24,330, 24,347 - June 12, 1984). EPA has also proposed a maximum contaminant level of 1 ppb for vinyl chloride in drinking water. (See 50 FR 46,902 - Nov. 13, 1985).

3. Waste and Spill Regulation

The EPA issued a rule under which certain VC manufacturing distillation residues are listed as hazardous wastes when disposed (49 FR 5308). This rule requires that all such wastes are to be disposed of only by RCRA-approved procedures.

When disposed of, commercial grade VC is classified as a hazardous waste under the Resource Conservation and Recovery Act (RCRA), because of its toxic and ignitable characteristics. Any disposal is subject to regulation under RCRA.

EPA has proposed additional RCRA regulations (51 FR 21648, June 13, 1986) which apply to all wastes containing VC. These proposed regulations define wastes as hazardous when the VC level in the extract by a specified test method exceeds 50 ppb. Congress has specified an interim 1 pound reportable quantity for vinyl chloride. Releases to the environment in excess of 1 pound are regulated under CERCLA.

4. New Product Manufacture

The EPA also administers the Toxic Substances Control Act (TSCA) which establishes health and environmental regulations for both new and existing substances. No one may manufacture or use a substance which is not on the Agency's official inventory, unless the Premanufacturing Notice procedures are followed.

C. Food and Drug Administration

PVC is widely used for food contact applications. In early 1986, the Food and Drug Administration (FDA) confirmed the safety of PVC for all food-contact applications and withdrew an outstanding proposal to limit its use in food packaging. (See 51 FR 4173 - February 3, 1986). An accompanying new proposal would set various residual vinyl chloride levels for different food contact materials. Among other things, FDA found that "vastly improved production technology (since 1975) has made it possible for manufacturers to succeed in reducing the level of residual vinyl chloride monomer in vinyl chloride polymer."

The comment period on the February 3, 1986 FDA proposal closed on June 5, 1986 without any adverse comments on the health or safety of PVC. This FDA proceeding lends further support to the inherent safety of human exposure to PVC. FDA regulates the use of PVC in medical devices and drug packaging on a case-by-case basis.

V. COMMUNITY HEALTH CONCERNS

As was discussed in Section IV, VC is a very strictly regulated substance. The EPA estimated that the 1976 standard would reduce the annual average exposure of the persons living within 5 miles of VC/PVC facilities by 95% (from 17 ppb to about 0.85 ppb.) An EPA report (1985) states that current industry performance has resulted in actual emissions that are significantly less than that predicted amount.

Many authors have attempted to develop quantitative risk assessments for low level exposures to VC. (See Barr, 1982 and Purchase, 1985 for reviews). Some have incorporated human data (Gehring, et al, 1979, Anderson, et al, 1980, Purchase, et al, 1985) and only these predict results which are compatible with the absence of any observed effects on humans from ambient exposures. The remaining estimates all used variations of the EPA upper limit model (Anderson, 1983) and overstate the probability of risk by several orders of magnitude.

There is no confirmed case on record in which a member of the general population has been harmed by exposure to vinyl chloride. That fact sets the upper limit of lifetime risk at less than 0.3 predicted cases of cancer per 1 million for exposure to 1 ppm of VC. Because the data show that industry emissions have been reduced by 99.99% (rather than the 95% estimated by EPA), the actual risk is less than 0.1 case of cancer in the next 70 years among the 5 million presumed to be exposed to VC from living within 5 miles of a VC/PVC facility.

Dr. Richard Wilson of Harvard (1979) has attempted to help people understand this method of stating the risks of every day occurrences. Each of the following activities for example, is predicted to result in one death per million people: smoking 1.4 cigarettes (due to cancer, heart disease); drinking ½ liter of wine (due to cirrhosis of the liver); traveling 6 minutes by canoe, 10 miles by bicyle, 300 miles by car, or 1,000 miles by jet (due to an accident); and having one chest X-ray taken in a good hospital (due to cancer caused by radiation).

We conclude, therefore, that there is no basis for concern by persons living near VC-using or producing facilities for any health effects from exposure to ambient concentrations of VC now being experienced.

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