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vinyl chloride:

An Information Resource

U.S. Department of Health, Education, and Welfare

Public Health Service

National Institutes of Health

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vinyl chloride:

An Information Resource

Prevention Branch

Division of Cancer Control and Rehabilitation

National Cancer Institute

Bethesda, Maryland

U.S. Department of Health, Education, and Welfare

Public Health Service

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Thomas H. Milby, M.D.
Editor

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Chapter I

INTRODUCTION AND REGULATORY HISTORY

Vinyl chloride monomer is a widely used chemical, the parent compound of polyvinyl chloride (PVC), a plastic resin used in innumerable consumer and industrial products--containers, wrapping film, electrical insulation, pipelines, credit cards, and many other items. At the same time, however, it can be said that overwhelming scientific evidence shows vinyl chloride to be a human carcinogen. Moreover, medical evidence suggests a strong association between exposure to vinyl chloride and the occurrence of a number of toxic, nonmalignant illnesses involving skin, bones, liver, lungs, and blood. PVC and other vinyl chloride polymers are not, at the time of this writing, considered to represent a carcinogenic risk to man.

Early Evidence of Problems

First produced commercially in the United States in 1927, vinyl chloride gas was for many years regarded by toxicologists as having only moderate liver toxicity. It was once considered for use as a general anesthetic, but its use for this purpose was abandoned in the early 1930s after experiments in animals demonstrated that its anesthetic effects were often accompanied by cardiac irregularities. In 1961, a threshold limit value (TLV) of 500 parts per million (ppm) for vinyl chloride was suggested by the American Conference of Governmental Industrial Hygienists (ACGIH), a standard apparently based upon its narcotic properties.

Acroosteolysis

In 1967, a hitherto unrecognized occupational disease was reported to be associated with exposure to vinyl chloride. This disease appeared among workers engaged in the process by which vinyl chloride was reacted (polymerized) to PVC. The disease was termed occupational acroosteolysis (AOL). Its symptoms included tenderness of the fingertips, sometimes accompanied by a gradual destruction of the bony integrity of the fingers. Workers suffering from this condition exhibited a form of vascular disease known as Raynaud's phenomenon. Only workers involved in the manual cleaning of PVC reactor vessels developed AOL.

In 1972, the ACGIH lowered its TLV for vinyl chloride to 200 ppm, following appearance of the evidence linking exposure to vinyl chloride with development of AOL, as well as new evidence of liver dysfunction among vinyl chloride workers.

Liver Cancer and Development of Stringent Regulatory Controls

Then, on January 22, 1974, the Occupational Safety and Health Administration (OSHA) was informed by the National Institute for Occupational Safety and Health (NIOSH) that the B. F. Goodrich Chemical Company had reported the deaths of several employees from a rare liver cancer (angiosarcoma). Shortly thereafter, a fact-finding hearing was held by the U.S. Department of Labor.

As a result, on April 5, 1974, the Department of Labor promulgated an emergency temporary exposure standard. This standard reduced the permissible exposure level to 50 ppm and established other safety requirements. On May 10, OSHA proposed a permanent standard consisting of a downward revision of the temporary standard to a "nondetectable level," as measured by a sampling and analytical method sensitive to 1 ppm. This proposed standard also contained occupational health requirements in addition to limitations on airborne levels.

A hearing on the proposed permanent standard was conducted in late June and early July of 1974, and a permanent standard was established. This standard set an exposure limit of 1 ppm averaged over any 8-hour period, with a ceiling of 5 ppm averaged over any period not exceeding 15 minutes. Health and safety requirements, including medical surveillance, were also established. In addition, the standard provided for an "action level" of 0.5 ppm time-weighted average. When monitoring demonstrates that no employee is exposed in excess of this action level, the employer may be exempted from some provisions of the standard. The purpose of this action level is to minimize the impact of the standard on employers who have attained exposure levels well below the permissible limit.

Beverages, Foods, and Other Consumer Products

Meanwhile, other concerns over the potential adverse effects of vinyl chloride had been voiced. In 1973, it was discovered by the Food and Drug Administration (FDA) that vinyl chloride could migrate from PVC containers into alcoholic beverages stored in them. Accordingly, the use of PVC bottles to contain distilled spirits was prohibited by the U.S. Treasury Department.

Discovery that vinyl chloride could also migrate from PVC containers to nonalcoholic beverages and to foods eventually led the FDA to propose, in September of 1975, more general regulations regarding vinyl chloride polymers in contact with food. At the time of this writing, the proposed FDA regulations state that any food containing detectable levels of vinyl chloride would be deemed adulterated. Hence, all uses of vinyl chloride polymers in rigid and semi-rigid food contact materials, such as bottles and sheets, would be prohibited because, in those forms, entrapped vinyl chloride may reasonably be expected to become a component of the food. The proposal would permit, however, the continued use of the "film" type of vinyl chloride polymers in food packaging and in other food contact

materials such as bottle cap linings, can coatings, and gaskets, since in these forms the migration of vinyl chloride is reduced to extremely low levels.

The proposal includes an interim food additive regulation allowing the use of water pipe made from vinyl chloride polymers. This interim regulation would remain in effect pending development of additional data to determine if vinyl chloride may be reasonably expected to exist in potable water that is drawn from systems using such water pipe. Preliminary data from an Environmental Protection Agency (EPA) survey of community drinking water supplies indicate that, although vinyl chloride can occasionally be found in small concentrations, the actual source of it is yet unknown.*

The use of vinyl chloride as a propellant in aerosol cans designated for household use was banned in 1974 through separate actions of the FDA, EPA, and Consumer Product Safety Commission (CPSC). A recall order on aerosol cans containing vinyl chloride was finalized in September of 1975 by the CPSC.

In order to protect consumers from the possible migration of vinyl chloride from PVC articles, the FDA is, at the time of this writing, working on proposals to regulate the use of PVC as a container for drugs and cosmetics, and in medical devices. Although these proposals will likely be patterned after the FDA's proposed regulations on food contact materials, they must necessarily utilize slightly different approaches because of the difference in the agency's statutory authority over these areas.

The General Environment

On October 21, 1976, the Environmental Protection Administration (EPA) promulgated regulations requiring the installation of the best available control technology to reduce vinyl chloride emissions to the air from all point and fugitive emission sources in plants that manufacture ethylene dichloride (by the oxychlorination process), vinyl chloride, or PVC. In addition, vinyl chloride emissions are limited to 0.02 g/kg of ethylene dichloride product. These regulations apply to all existing and new ethylene dichloride plants, vinyl chloride plants, and PVC plants in the U.S. The EPA estimates that these regulations will result in reduction of vinyl chloride emissions to the ambient air from hitherto uncontrolled sources by more than 90%. These regulations resulted in part from an EPA study of ambient concentrations of vinyl chloride in residential areas located in the vicinity of vinyl chloride or PVC plants. This study showed that although the ambient levels of vinyl chloride exceeded 1 ppm less than 10% of the time, the new regulations

*The question of whether this issue falls within the jurisdiction of the FDA or the EPA is being debated by the two agencies at the time of this writing.

could have a significant impact on the total vinyl chloride exposure of the approximately 4.6 million people living within a 5-mile radius of these plants.

Although the disposal of vinyl chloride into ocean waters has been banned, there are currently no regulations on vinyl chloride emissions into other water systems. However, the EPA air emissions standard indirectly affects water emissions, since it establishes a 10 ppm limitation on in-process waste water. This restriction, in conjunction with the tendency of vinyl chloride to escape from water, makes significant water-pollution problems unlikely.

The disposal of solid waste containing vinyl chloride is not currently seen as being a major cause for concern. The EPA, howver, is drawing up guidelines that would ensure the safe disposal of such waste.

Transportation of Vinyl Chloride

Although some vinyl chloride is transported by barge and truck, most of it is shipped by rail tank car. In a three-year period from 1971-1974, there were 16 reported rail accidents involving vinyl chloride tank cars.

The Department of Transportation (DOT) has regulatory responsibility over the transportation and handling of vinyl chloride as a "hazardous material." Regulations by DOT include requirements for transportation preparations such as construction of containers, packaging, volume, and marking. Until recently, DOT regulations were directed almost exclusively to the fire and explosion hazard of this very flammable gas. In April 1975, however, the Coast Guard (a branch of DOT) promulgated new regulations for the transport of vinyl chloride by barge. These regulations would require the implementation of control measures similar to those specified in the OSHA regulations.

Extent of Potential for Exposure

As indicated previously, the potential for human contact with vinyl chloride is great. There is, first of all, production of the compound itself (vinyl chloride monomer); in the United States, some 2.5 billion kilograms (5.6 billion pounds) per year are produced at 15 plants employing some 900 workers. Also, vinyl chloride is polymerized to produce PVC at 39 plants employing over 5,500 workers, and tens of thousands of workers fabricate PVC into the many products that reach consumer and industrial users. Finally, EPA has estimated that nearly 5 million people reside sufficiently close to the nation's vinyl chloride facilities to be within range of detectable airborne concentrations of the gas.

In the chapters that follow, the production, uses, and dispersion of this hazardous compound are discussed, evidence of its toxic and carcinogenic effects is summarized, and control strategies and programs are set forth.

Table 1

PHYSICAL PROPERTIES OF VINYL CHLORIDE*

Formula	CH_2:CHCl
Molecular weight	62.50
Melting point	-153.7
Boiling point, 1 atm	-13.9°C (7.0°F)
Specific gravity, gas, 15°C, 1 atm (air = 1)	2.15
Flash point	-78°C (-108°F)
Self-ignition temperature	472°C (881.6°F)
Explosive limits in air, % by vol	4-20
Viscosity, liquid, eP	
-40°C	0.3339
-30°C	0.3026
-20°C	0.2780
-10°C	0.2563

Vapor pressure

°C	atm	°C	atm
-30	0.50	20	3.33
-20	0.77	30	4.51
-10	1.17	40	5.94
0	1.70	50	7.80
10	2.43	60	9.93

Solubility in water, 24°C, 1 atm	0.11g/100g water
Conversion factors, 25°C, 1 atm	
1 ppm	2.56 mg/m^3
1 mg/liter	391 ppm

*Compiled from: Kirk-Othmer Encyclopedia of Chemical Technology, Second edition, Vol. 5 (Interscience Publishers, John Wiley & Sons, Inc., New York, 1964), p. 172; and from U.S. Environmental Protection Agency, Office of Research and Development, "Scientific and Technical Assessment Report on Vinyl Chloride and Polyvinyl Chloride," Report No. EPA-600/6-75-004 (Washington, D.C., U.S. Govt. Printing Office, 1975), p. 6.

	1961	1963	1971	1972	1973	1974	1975	1976
American Conference of Governmental Industrial Hygienists*	Established a threshold limit value (TLV) of 500 ppm	Modified 500 ppm TLV exposure limit to 500 ppm maximum concentration		Revised 500 ppm maximum concentration limit downward to 200 ppm				
Coast Guard (Department of Transportation)							Promulgated new regulations for transportation vinyl chloride by barge (measures similar to OSHA regulations)†	
Consumer Product Safety Commission						Banned the use of vinyl chloride as a propellant in *aerosols*	Finalized (in Sept.) a recall on aerosol cans containing *vinyl chloride*	
Environmental Protection Agency						Banned the use of vinyl chloride as a propellant in aerosols	(Dec.)--proposed regulations requiring installation of best available technology to reduce emissions to air and a 10 ppm limit for in-process waste water.	
Food and Drug Administration						Banned the use of vinyl chloride propellant in aerosols	Proposed regulations prohibiting any detectable level in food, thereby prohibiting use of rigid and semi-rigid vinyl chloride polymers as food containers	Working on proposals to regulate use of PVC as container material for drugs, cosmetics, and medical devices
Occupational Safety and Health Administration			Published a list of TLV's for *pollutants* in the work place--vinyl chloride limit set at 500 ppm			April 5--Set Emergency Temporary Standard at 50 ppm; Oct. 1--50 ppm made permanent through Dec. 31 then dropped to 1 ppm over 8-hr period and 5 ppm for 15-min. periods		
Treasury Department					Prohibited use of PVC bottles to contain distilled spirits			

*Advisory only.

†Department of Transportation regulations have required all along that vinyl chloride be transported and handled as a "hazardous material."

FIGURE 1 REGULATION OF VINYL CHLORIDE AS A HEALTH HAZARD IN THE U.S.

Chapter II

PRODUCTION, USE, EMISSIONS, AND HUMAN EXPOSURE

A major part of any carcinogen control program is quite direct in concept--reducing human exposure to a carcinogenic agent. Effective reduction of exposure, in turn, depends upon understanding at what points in the exposure pathways control measures can be implemented successfully. In particular, it is important to understand which sources of exposure are dominating, so that limited control resources can be applied most judiciously.

Exposures to vinyl chloride occur in the workplace, through general air and water pollution, and, to a limited extent, from the use of fabricated products. Although large quantities of vinyl chloride are produced each year in the United States, it is not in the production of the chemical itself that the greatest potential for harmful exposure exists. Rather, the greatest hazard is when the chemical is polymerized to form other materials, nearly all of which are polyvinyl chloride (PVC) resins, during which process vinyl chloride escapes into the air. Inhalation of this vinyl chloride is by far the most important exposure pathway.

This chapter examines the production and use of vinyl chloride in the United States and emissions of the chemical into the environment, all of which are factors in human exposure. From this information, a conceptual model, which will be found at the end of this chapter, has been made to illustrate the flow of vinyl chloride from its sources to the populations at risk.

Production and Consumption[1,2]

Some 2.5 billion kilograms (kg) of vinyl chloride are produced in the United States each year. About 188 million kg of the material were exported in 1975; imports of vinyl chloride are negligible. (See Table 2.) Virtually all (96%) of the more than 2 billion kg consumed in the United States are for production of PVC (2.2 billion kg in 1974, 1.7 billion in 1975, and an estimated 2.2 billion in 1976).

After being compounded with various other materials, PVC is converted to a variety of end products. Categories of end use for PVC products and the estimated proportion of total production for each are (1975):

Construction (primarily pipe and conduit)	48%
Consumer goods	16
Electrical	8.5
Packaging	8.5

Transportation	6
Home furnishings	6
Miscellaneous	7

(coatings, adhesives, and inks; medical tubing and bloodbags; credit cards, carpet backcoatings; traffic cones; novelties, etc.)

The 4% of vinyl chloride not used in producing PVC is used for the most part to produce methyl chloroform, which is used primarily as a solvent. Vinyl chloride was once used in the production of Dynel® acrylic fibers, but that production was discontinued in 1974. As noted in the previous chapter, use of vinyl chloride as a propellant in aerosol cans was banned in 1974. It has been reported that vinyl chloride has found use in the production of chloroacetaldehyde, an intermediate in the synthesis of sulfa drugs, but no evidence was found in this study that it is currently being used for this purpose. Also, it has been reported that vinyl chloride found use as a refrigerant and as an extraction solvent for heat-sensitive materials; however, no evidence was found that it is currently being used for these purposes.

Table 2

PRODUCTION, EXPORTS, AND APPARENT CONSUMPTION OF VINYL CHLORIDE, UNITED STATES, 1970-1975[a]
(Millions of kg)

Year	Production	Exports	Apparent U.S. Consumption
1970	1834.2	302	1532
1971	1968.5	281	1688
1972	2310.4	282	2028
1973	2429.4	191	2238
1974	2551.9	187	2365
1975	1905.0[b]	188	1717
1976	2604.0[c]		

[a]Imports are negligible.

[b]Sales value of the vinyl chloride produced in 1975 was roughly $438 million (based on a price of 25¢/kg, tank quantities, polymer grade, FOB works, also applied to material used in captive consumption).

[c]Preliminary estimate.

Source: SRI

Long-term demand for PVC in its major markets is expected to remain stable, since its uses are firmly established and because there is no material that can readily replace it. Although investigations by the Environmental Protection Agency and industry of any possible leaching of vinyl chloride into drinking water are continuing, it does not appear now that the continued growth of PVC consumption for water supply pipe will be cut off by any regulatory action, especially since pipe resins are now made with controlled, negligibly low, levels of residual vinyl chloride monomer. And although there is public concern over migration of vinyl chloride from PVC packaging film and bottles into the containers' contents, and the makers of cosmetics and toiletries are abandoning the use of PVC bottles, loss of volume in the packaging industry affects only a minor part of the market for PVC.[2] It is estimated that consumption of PVC in 1980 will be about 3 billion kg.

Ten U.S. companies manufacture vinyl chloride at 15 plants in the nation, including Puerto Rico. (See Table 3.) There are 22 producers of PVC resins at 39 plants in the country. (See Table 4; plant locations, for vinyl chloride as well, are also shown in Figure 2.) Allied Chemical Corporation, Dow Chemical U.S.A., PPG Industries, Inc., and Shell Chemical Company only produce vinyl chloride for sale to others; the other companies also produce PVC resins.

Most large manufacturers of resin also produce PVC resin compounds for sale or for feeding their own end-product manufacturing operations. For example, Continental Oil Company, Conoco Chemicals Division, has a PVC compound production capacity of over 60 million kg per year at Aberdeen, Mississippi, and the Plastics Division of Diamond Shamrock Chemical Company has two compounding plants with a combined capacity of nearly 80 million kg per year. The B. F. Goodrich Chemical Company has long operated several large compounding facilities. Other resin manufacturers that compound resins are Robintech, Inc., Ethyl Corporation, Pantasote Company, Tenneco Chemicals, Inc., and Firestone Plastics Company. Resins are also compounded by an estimated two dozen other firms in the United States, the largest of which is believed to be H. Schulman, Inc., Akron, Ohio.

Production Processes[1]

The processes for manufacturing both vinyl chloride and its polymers, polyvinyl chloride resins, are described briefly in the paragraphs that follow.

Vinyl Chloride

Nearly all of the vinyl chloride produced commercially in the United States is currently made from ethylene by one or the other of two processes (about half by each): oxychlorination of ethylene and direct chlorination of ethylene. A third process, addition of hydrogen chloride to acetylene, accounts for about 4% of the total vinyl chloride produced.

Table 3

U.S. PRODUCERS OF VINYL CHLORIDE

Company and Plant Location	Annual Nameplate[a] Production Capacity Year End, 1976 (million kg) Plant	Total
Shell Oil Company, Shell Chemical Company, Base Chemicals Division		700
Deer Park, Texas	380	
Norco, Louisiana	320	
Dow Chemical U.S.A.		610
Oyster Creek, Texas	320	
Plaquemine, Louisiana	200	
Freeport, Texas	90	
The B. F. Goodrich Company, B. F. Goodrich Chemical Company Division		450
Calvert City, Kentucky		
PPG Industries, Inc.		410
PPG Industries (Caribe), Guayanilla, PR	230	
Industrial Chemicals Division		
Lake Charles, Louisiana	180	
Continental Oil Company, Conoco Chemicals		320
Lake Charles, Louisiana		
Ethyl Corporation, Industrial Chemicals Division		180
Baton Rouge, Louisiana	120	
Pasadena, Texas	60	
Allied Chemical Corporation, Industrial Chemicals Division		140
Baton Rouge, Louisiana		
Borden Inc., Borden Chemical Division, Petrochemicals		140
Geismar, Louisiana		

Table 3 (Concluded)

Company and Plant Location	Annual Nameplate[a] Production Capacity Year End, 1976 (million kg)	
	Plant	Total
Monochem, Inc. (jointly owned by Borden, Inc. and Uniroyal, Inc.) Geismar, Louisiana		140
Stauffer Chemical Company Carson, California		80
Total		3170

[a]Actual operating capacities are usually somewhat lower. Published capacities are often larger because (1) design capacities presume the continuous production of one type of resin whereas all producers make various types of resins in each plant, and (2) scheduled and unscheduled shutdowns are not taken into account in nameplate capacity figures.

Source: SRI

Table 4

U.S. PRODUCERS OF POLYVINYL CHLORIDE

Company and Plant Location	Annual Nameplate[a] Production Capacity Year End, 1976 (million kg) Plant	Total
The B. F. Goodrich Company, B. F. Goodrich Chemical Company Division		570
Louisville, Kentucky	280	
Pedricktown, New Jersey	80	
Avon Lake, Ohio	70	
Henry, Illinois	70	
Long Beach, California	70	
Diamond Shamrock Corporation, Diamond Shamrock Chemical Company, Subsidiary, Plastics Division		260
Deer Park, Texas	210	
Delaware City, Delaware	50	
Borden, Inc., Borden Chemical Division		250
Illiopolis, Illinois	180	
Leominster, Massachusetts	70	
Tenneco Inc., Tenneco Chemicals, Inc. Organics and Polymers Division		220
Pasadena, Texas	110	
Burlington, New Jersey	70	
Flemington, New Jersey	40	
Tne Firestone Tire & Rubber Company, Firestone Plastics Company, Division		200
Pottstown, Pennsylvania	180	
Perryville, Maryland	20	
Stauffer Chemical Company, Plastics Division		180
Delaware City, Delaware	110	
Long Beach, California	70	
Continental Oil Company, Conoco Chemicals Division		170
Oklahoma City, Oklahoma	90	
Aberdeen, Missouri	80	

Table 4 (Continued)

Company and Plant Location	Annual Nameplate[a] Production Capacity Year End, 1976 (million kg) Plant	Total
Union Carbide Corporation, Chemicals and Plastics Division		160
South Charleston, West Virginia	?	
Texas City, Texas	?	
Certain-Teed Products Corporation		140
Lake Charles, Louisiana		
The Goodyear Tire & Rubber Company, Chemical Division		110
Plaquemine, Louisiana	90	
Niagara Falls, New York	20	
Robintech, Inc.		110
Painesville, Ohio		
Georgia-Pacific Corporation		100
Plaquemine, Louisiana		
Shintech, Inc. (joint venture of Robintech, Inc. and Shin-Etsu Chemical Industry Company, Ltd. of Toyko)		100
Freeport, Texas		
Air Products and Chemicals, Inc., Plastics Division		90
Calvert City, Kentucky	70	
Pensacola, Florida	20	
Occidental Petroleum Corporation, Hooker Chemical Corporation, subsidiary, RUCO, subsidiary		90
Burlington, New Jersey	80	
Hicksville, New York	10	
The General Tire and Rubber Company Chemicals/Plastics Division		90
Ashtabula, Ohio	60	
Point Pleasant, West Virginia	30	
Ethyl Corporation, Industrial Chemicals Division		80
Baton Rouge, Louisiana		

Table 4 (Concluded)

Company and Plant Location	Annual Nameplate[a] Production Capacity Year End, 1976 (million kg)	
	Plant	Total
Rico Chemicals Corporation Guayanilla, Puerto Rico		70
The Pantasote Company of New York, Inc. Eleanora Chemical Division		60
Passaic, New Jersey	30	
Point Pleasant, West Virginia	30	
Great American Chemical Corporation Fitchburg, Massachusetts		30
Atlantic Tubing & Rubber Company Cranston, Rhode Island		20
Keysor-Century Corporation Saugus, California		20
Total		3120

[a]Actual operating capacities are usually somewhat lower. Published capacities are often larger because (1) the design presumes continuous production of one type of resin whereas all producers make various types of resins in each plant, and (2) scheduled and unscheduled shutdowns are not taken into consideration in nameplate capacity figures.

Source: SRI

SOURCE: SRI.

FIGURE 2 LOCATIONS OF VINYL CHLORIDE AND POLYVINYL CHLORIDE PLANTS IN THE UNITED STATES

In both of the ethylene-based processes, ethylene dichloride is first obtained and then is cracked to vinyl chloride and hydrogen chloride.

In the oxychlorination process, ethylene dichloride is made by the reaction of air and hydrogen chloride with ethylene in a catalytic process. The catalyst is a mixture of copper chloride and other chlorides.

In manufacture of vinyl chloride by direct chlorination of ethylene, ethylene dichloride is first obtained by the catalytic vapor or liquid-phase reaction of ethylene and chlorine. Metallic chlorides, such as ferric, aluminum, copper, or antimony, are usually used as the catalyst.

In the third process, which is still used by Monochem, Inc. at Geismar, Louisiana, vinyl chloride is produced by the reaction of acetylene with hydrogen chloride in the presence of mercuric chloride catalyst.

Polyvinyl Chloride Resins

Vinyl chloride, which is a gas at room temperature, is used in liquid form under pressure when it is polymerized to form PVC by one of the four basic processes: suspension polymerization (accounting for the majority of total PVC produced); emulsion polymerization; bulk polymerization; or solution polymerization. In all processes, the polymerization is initiated by free radicals and proceeds at temperatures of 40-70°C with the evolution of heat. The term, polyvinyl chloride resins, includes vinyl chloride homopolymers with the repeating unit, $-CH_2CHCl-$, and copolymers of vinyl chloride with varying amounts of another monomer (e.g., vinyl acetate, ethylene, propylene, vinylidene chloride, or acrylates).

Suspension polymerization is carried out in an aqueous system in which monomer droplets are maintained in suspension by means of a protective colloid in conjunction with brisk agitation. Generally, suspension polymerization is operated as a batch process employing glass-lined or stainless steel reactors. The reactor is first charged with deionized water, and then a protective colloid, a buffer, and an initiator are added. The vinyl chloride and, in the case of copolymer production, the second monomer are then intermittently introduced in controlled ratios. The mixture is brought to the polymerization temperature (about 50°C), and after variable induction periods depending on the initiator used, the polymerization starts. The heat generated by polymerization is removed from the system to maintain the desired reaction temperature. The polymerization time can vary by as much as one-half (e.g., 8 vs. 16 hr) depending on the initiator used; in recent years, so-called co-initiation systems have reduced time in the reactor to about 6 hours.

A dispersion of relatively large polymer particles in water is obtained by the suspension polymerization process. After unreacted monomer is driven out of the slurry (and recovered), the slurry is centrifuged, and the polymer is flash dried in an air stream at about 80°C. The dry polymer is screened and shipped, either in bulk or packed in multiwall paper bags.

Emulsion polymerization is basically similar to the suspension process except that relatively large amounts of two emulsifying agents are used, one of which is soluble in the monomer, the other in water. This system very effectively prevents the coalescence of polymer particles, resulting in resins of a very small particle size. The drying methods (frequently by spray driers) are also designed to maintain a small particle size. Because complete removal of the emulsifiers is never achieved, resins made by this process are not used where high clarity is needed (such as in packaging films) or where low water absorption is required (such as in electric wire insulation). The initiator systems used in this process are different from those used in suspension polymerization-they are typically redox-type, employing persulfates.

In the bulk polymerization process, vinyl chloride is polymerized without the addition of other liquids. In this two-stage process, the initial liquid-phase reaction is followed by polymerization in an autoclave that is designed to agitate the then-dry, powdery mass effectively until the conversion from monomer to polymer reaches a level of about 90%. Heat exchange is provided by the distillation of monomer and its recondensation within the reactor and in external condensers. Bulk polymerized PVC resins resemble suspension resins in appearance and are characterized by high uniformity and purity of particles.

In the solution polymerization process, the monomers are first dissolved in an organic solvent (such as n-butane or cyclohexane) in an autoclave. After the addition of a peroxide initiator and heating of the stirred solution to about 40°C, polymerization begins, and the polymer precipitates as the reaction proceeds. Solution polymerization is used exclusively for the production of specialty copolymers of vinyl chloride with vinyl acetate. These copolymers are very pure and uniform, and their chief value lies in their unique solubility and film-forming characteristics.

A significant trend in the polymerization processes for vinyl chloride has been toward larger reactors. Reactors typically have had a capacity in the range of 7,570 to 28,388 liters (2,000 to 7,500 gallons), but reactors with capacities of 98,410 and even 132,475 liters (26,000 and 35,000 gallons) have been installed recently. (With a given amount of total plant capacity, larger reactors would probably reduce the total emission of vinyl chloride during the polymerization operation.)

Compounding of PVC Resins

Polyvinyl chloride resins are compounded with a number of auxiliary materials before they are converted to end products. These materials include plasticizers, light stabilizers, heat stabilizers, pigments, and fillers. Compounding of PVC generally begins with premixing followed by very intense mixing, because uniform distribution is mandatory for trouble-free processing and uniform product quality. Of the many types of PVC in use, most are relatively dry in the premix stage and have a free-flowing, sandy consistency. To achieve intimate mixing, they are subjected to a hot mixing step at fusing temperature.

Conversion to End Products

Compounded PVC resins are converted to end products by several processes. These include extrusion (about 53%), calendering (about 18%), dispersion (about 13%), injection molding (about 6%), compression molding (about 4%), coating and adhesives (about 4%), and blow molding (about 2%).

Figure 3 shows the production and use of vinyl chloride.

Emissions and Their Sources

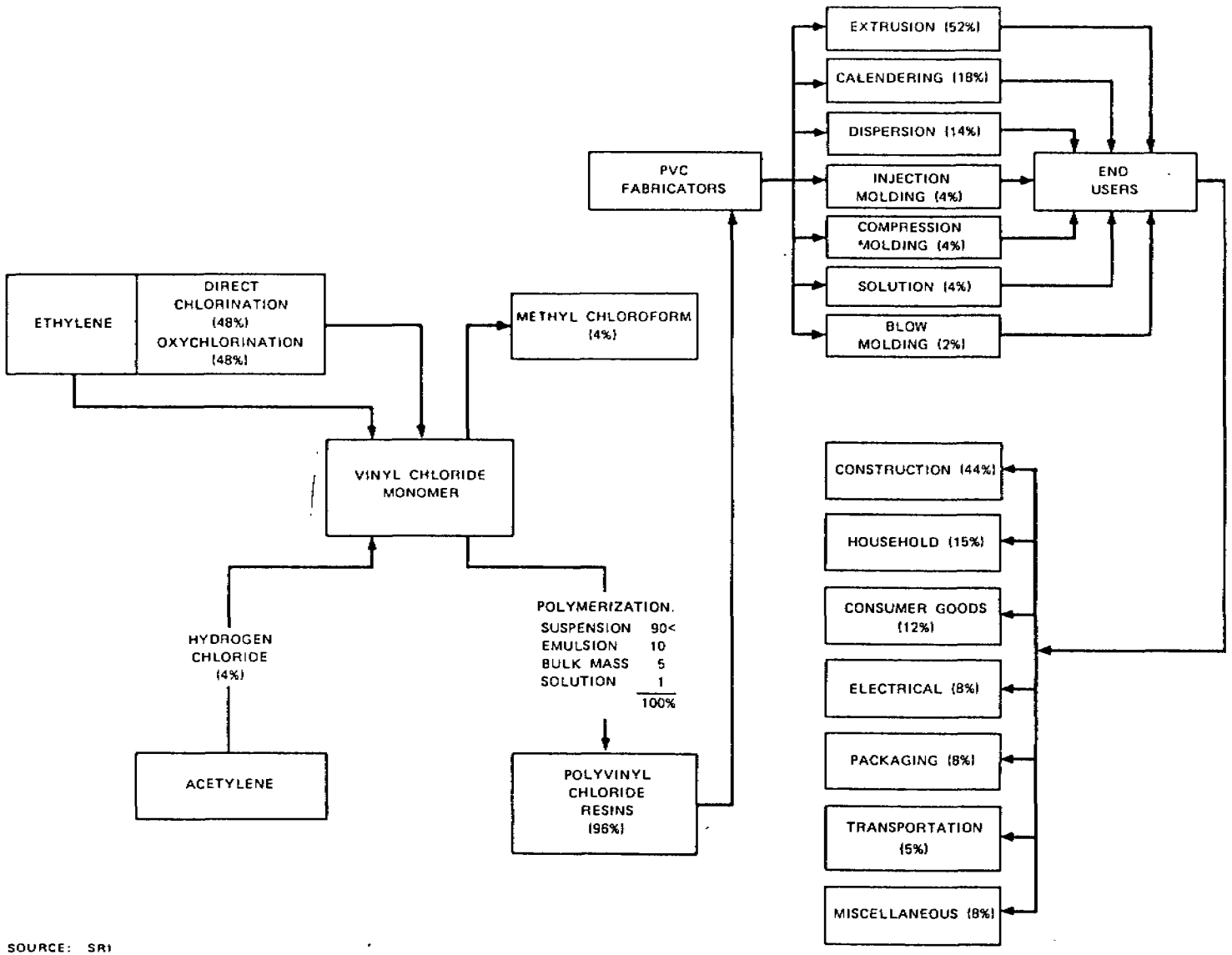
The areas of potential for human exposure to emissions of vinyl chloride may be ranked as follows:

- When vinyl chloride is polymerized to form PVC--the major emission source and the process in which the highest occupational exposures have been recorded.
- Production of vinyl chloride itself--the three current manufacturing processes for this are not ordinarily serious emission sources of the monomer.
- Fabrication of PVC products--a possibility for emissions and exposures, though not a major one, arising when unreacted, entrapped monomer is released during the fabrication process.
- The general environment and miscellaneous sources--including inhalation of contaminated air in the neighborhood of a plant; ingestion of food or water contaminated with vinyl chloride that has migrated from pipe, containers, or wrapping; or, until 1974, inhalation resulting from use of aerosol containers containing the gas as a propellant.

Polymerization of Vinyl Chloride to PVC

As indicated above, it is the polymerization process that is the largest emission source. The Environmental Protection Agency (EPA) has estimated that 85% of U.S. emissions emanate from PVC production facilities.[3]

Most occupational exposures occur in the area of the polymerization reactors. They have been especially high during the hand cleaning of reactors to remove accumulations of PVC scale on the reactor walls. This hand-cleaning procedure was common until the mid-1960s, when cases of acroosteolysis led to the development of high-pressure spray washers; it continued in some plants until the implementation of the OSHA Temporary Standard in early 1974, often without provision of protective equipment. The task was usually assigned to the newest employees, so that nearly all workers employed in polymerization plants before 1960 were given this



SOURCE: SRI

FIGURE 3 PRODUCTION AND USE OF VINYL CHLORIDE

experience. This task is still required intermittently, because of lack of complete removal of scale by the washers, but it is currently performed only by workers in impervious isolation garments, with adequate respiratory protective equipment.

Exposures during the hand-cleaning operation, which requires climbing through a manhole into the reactor vessel, were commonly several thousand parts per million. In addition, it has been estimated that general exposure levels in the PVC plants during the 1940s and 1950s were "...frequently over 1000 ppm and commonly in the 100 ppm range...."[4]

Current exposures are dramatically less than those past exposures as a result of nearly universal industry compliance with the requirements of the OSHA Standard. Daily time-weighted-average occupational exposures have been reduced to levels near to or below 1 ppm for most workers, although such exposures to 10 ppm are not uncommon.[5] The greatest exposures continue to be experienced by the reactor operators, operator's helpers, workers who transfer the incoming vinyl chloride monomer to storage vessels, maintenance personnel, and workers in the compounding steps.

The sources of vinyl chloride emissions at a typical PVC plant and their relative contributions to the total uncontrolled emissions are shown in the following tabulation:[3]

Source	Percent of Total
Fugitive emission sources (leakage, loss during sampling, vaporization from wastewater, and the like)	39%
Sources following the stripper (blind tank, dryers, bulk storage, and the like)	32%
Monomer recovery system	13%
Stripper	8%
Relief valve discharge	5%
Reactor opening	3%
	100%

Production of Vinyl Chloride Monomer

The three production processes for the commercial manufacture of vinyl chloride are not ordinarily serious emission sources of the monomer. The EPA estimates that 11% of all emissions can be attributed to these plants.[3]

Occupational exposures generally occur after production, as the finished monomer is piped to storage or transportation, or during maintenance. Data reported to OSHA on exposures before the emergency temporary standard was imposed indicated peak exposures over 200 ppm for laboratory technicians, over 150 ppm for loading operations, and near 50 ppm for quality control specialists.[4] The following tabulation shows some levels found in a recent survey, conducted for NIOSH, of three vinyl chloride plants in which it will be noted that time-weighted-average exposures range from 0.07 ppm to 26.46 ppm:[6]

Job Category	VCM Breathing Zone Concentration Ranges (ppm)	
	Single Measurement	Time-Weighted Average
Loader	0.06-84.77	0.30-26.46
Plant operator	0.01-18.2	0.07-11.03
Lab technician	0.03- 4.36	0.07- 0.40
Chromatographer	0.02- 1.01	0.06- 0.28
Shift supervisor	0.03- 0.55	0.09- 0.11
Pipefitter	0.01- 0.41	0.17- 0.30
Instrument man	0.02- 0.25	0.17

The sources of vinyl chloride emissions from a typical vinyl chloride monomer plant and their relative contributions to the total uncontrolled emissions are shown below:[3]

Source	Percent of Total
Vinyl chloride formation and purification	54%
Fugitive emission sources	27%
Ethylene dichloride purification	11%
Oxychlorination reactor	8%
	100%

Fabrication of PVC Products

The fabrication of final products from intermediate PVC stock has not been considered a serious source of either emissions or exposures. (Neither EPA nor OSHA has included fabrication plants in its regulations.) In large part, this has been due to uncertainty as to the extent and nature of exposures during fabrication processes and emissions from them. Both agencies are currently attempting to define the contribution of

fabricating plants to the occupational and environmental exposure burdens. That there may be a potential problem is evidenced by two cases of angiosarcoma known in workers who had occupational exposure only in fabrication.[7]

The most severe exposures and emissions would be expected at those fabrications plants where unblended PVC resin is received and then carried through to fabrication of the final product. The personnel most likely to be exposed are those working in the blending and compounding processes, where the PVC resin is heated and mixed with additives. These processes present an ideal opportunity for the release of any unreacted vinyl chloride monomer that may be present in the PVC. Employees working in storage areas may also be exposed to vinyl chloride released from poorly stripped resin.

The NIOSH survey mentioned above[6] also covered fabrication plants. About four-fifths of the samples from time-weighted-average exposures were below 0.01 ppm, which was the lower detectable limit of the sampling and analytical method used. However, there were substantial interplant, as well as intraplant, differences, with exposures in similar job categories ranging from less than 0.01 to 2.44 ppm. The time-weighted-average concentrations of vinyl chloride for various job categories at the seven fabrication plants in the survey are shown in the following tabulation:

Job Classification	Average Concentration (ppm)
Calender personnel	0.85
Pelletizer personnel	0.39
Compounding personnel	0.16
Laboratory personnel	0.03
Molding personnel	0.02
Plastisol dipping personnel	0.01
Extrusion personnel	0.01
Maintenance personnel	0.01
Miscellaneous personnel	0.01

Since vinyl chloride emissions from fabricating plants result from residual monomer in the raw material coming from PVC plants, they will be minimized indirectly as vinyl chloride is controlled more closely at the PVC facilities.

General Environment and Miscellaneous Sources

Current exposure pathways of vinyl chloride to the general public are through inhalation of contaminated air in the neighborhood of a plant or through ingestion of contaminated food or water. Also, there are some miscellaneous minor exposures that may arise in the occupational or general environment; they include the disposal of partially polymerized

waste sludge and occupational exposures during the use of vinyl chloride to produce methyl chloroform or the production of vinyl chloride as a by-product.

Gaseous vinyl chloride emitted into the air at vinyl chloride and PVC plants is estimated to account for 96% of all atmospheric emissions of the monomer.[3] It is distributed into the atmosphere surrounding the emission source in patterns and amounts that depend on the process involved, the amount of vinyl chloride produced, the nature of the plant area from which it is released, topography, and meteorological conditions.

Preliminary monitoring results in 1974 at seven industrial complexes involving ten PVC resin and two vinyl chloride plants indicated that the levels of vinyl chloride in the ambient air near the plants fluctuated sharply. This was apparently due to the periodic opening of reactor kettles in the PVC plants, accidental plant discharges, variations in the production process, and meteorological conditions. While almost all of the samples collected contained detectable levels of vinyl chloride, more than 90% of the instantaneous observations and 97% of the 24-hour samples show amounts less than 1 ppm. Although a level of 33 ppm was found in one sample at one PVC plant, repeated sampling indicated that this level was unusually high and that the average at this site was less than 1 ppm.[8]

Although most samples were obtained within one-half mile of the plant, in one case a level of 3.4 ppm was recorded at a distance of three miles from the plant. However, the average of several readings at this site was approximately 0.5 ppm. The EPA has estimated average exposures to the 4.6 million people who live within 5 miles of vinyl chloride plants at 0.017 ppm.[9]

Vinyl chloride levels in the air in the general community are usually below detectable levels, and the EPA has estimated general average population exposure at 0.5 ppb. Inhalation of vinyl chloride, based on the assumption of 20 m^3 of air inhaled per day containing 0.5 ppb vinyl chloride, is 0.03 mg/day.[8]

In a 1973 EPA study of combustion products from the incineration of plastics, vinyl chloride was identified in the flue gas from incinerators.[10] The quantity of vinyl chloride from a representative PVC homopolymer varied as a function of temperature, as shown in the tabulation below:

Combustion Product--Vinyl Chloride
(in mg/g of sample polymer)

25-280°C	280-350°C	350-430°C	430-510°C	510-580°C
0.04	0.25	0.17	0.02	--

The quantity of vinyl chloride also varied with the form of fabricated plastic article but was primarily dependent on entrapped monomer concentration and temperature. Measurements of the concentration of vinyl chloride in the ambient air in the vicinity of municipal incinerators have not been made.

Exposures through consumer products, such as cosmetics, and through foods and beverages are dependent on the migration rates of vinyl chloride from PVC containers and products. However, very few data exist to show such migration. It is believed that the extent of migration depends upon the residual monomer in the PVC containers, the length of time of storage, and the industrial processes used. The total oral daily human intake of vinyl chloride for Europeans has been estimated at less than 100 μg,[11] and, according to the World Health Organization, intake of vinyl chloride from food may be on the order of 1 μg per person per day.[12] The preliminary report of the range of vinyl chloride concentrations in some consumer products reported by the U.S. Food and Drug Administration is shown in Table 5.[13] These data are not adequate to determine a dietary exposure to vinyl chloride.

In an EPA survey of 80 public water supplies in 1974, small quantities (between 0.27 and 5.6 ppb) of vinyl chloride were found in three of four samples from the municipal water supplies of Miami and Philadelphia.[14] The sources of the substance have not been identified.

EPA and FDA are undertaking test programs to determine the migration of vinyl chloride from PVC water pipe. The results so far show that migration of residual vinyl chloride does occur and that the extent of the migration depends on the residual level in the PVC.[8]

Waste effluents from vinyl chloride and PVC plants were monitored for vinyl chloride. Levels were found to vary considerably depending upon the in-plant handling of wastes and treatment of wastewater. The highest level of vinyl chloride found in wastewater leaving the plant site was 20 ppm. More typically, levels of 2 to 3 ppm were found. The levels of vinyl chloride entrapped in sludge and other solid wastes from the reactor kettles ranged from 100 ppm to, in one case, 3000 ppm.[6]

With regard to other miscellaneous sources of vinyl chloride emissions--sources that either use vinyl chloride for production of methyl chloroform or that produce vinyl chloride as a by-product--preliminary reports indicate that there are eight such plants in the United States and that they account for approximately 3% of the total estimated 1974 emissions from all sources.[13] The EPA has not proposed vinyl chloride emission standards for these plants.

Dispersion and Degradation of Vinyl Chloride

In the paragraphs that follow, the ultimate dispersion and degradation of vinyl chloride in the atmosphere, water, and soil is discussed briefly.

Table 5

RANGE OF VINYL CHLORIDE CONCENTRATIONS
IN SOME CATEGORIES OF CONSUMER PRODUCTS

Product	Range[a]	Sensitivity
Cosmetics[b]	N.D.[c] to 4 ppm	0.1 ppm
Mouthwashes[d]	N.D. to 7 ppm	0.05 ppm
Biologic products	N.D.	0.02 to 0.4 ppm
Vinegar	N.D. to 8.4 ppm	Unknown
Oil	6.5 to 10 ppm	Unknown
Meat products	N.D. to 0.4 ppm	Unknown
Water pipe (residue)	1 to 100 ppm	0.05 ppm
PVC films	1 ppm to 4 ppm	Unknown
Sheeting	1 ppm	Unknown
Cap liners (food and beverage jars)	N.D.	--

[a]Preliminary data--not verified by Food and Drug Administration.

[b]Time in bottle--3 to 48 months.

[c]Not detectable.

[d]All samples purchased before November 1974.

Source: Chapter reference 13

Atmosphere

Vinyl chloride boils at -13.9°C and thus is a gas at normal atmospheric pressure. It is slightly more than twice as dense as air.

Results on the atmospheric reactions and rates of disappearance of vinyl chloride in the ambient atmosphere are inconclusive. The peak absorption of the substance in the ultraviolet region is far below the solar cutoff (approximately 290 nm), so that vinyl chloride would not undergo reaction in sunlight in the absence of other reactive chemical species.

Laboratory photochemical chamber experiments have shown different photodegradation rates of vinyl chloride in the presence of high concentrations of different reactants (including NO_2, O_3, and NO) that are commonly found in ambient air. The half-life of vinyl chloride was reported to be 3 to 6 hours, and the reaction products identified included carbon monoxide, formaldehyde, formic acid, formyl chloride, and hydrogen chloride.[15]

Two chemical reaction mechanisms that appear to be potentially important for removal of vinyl chloride in ambient air have been identified. They are:

- Reaction with HO· radical, which is most often present at 4 parts per trillion (10^{-14} molar) concentrations,[16] and
- Reaction with ozone (O_3) usually found in the range of 10-100 parts per hundred million ($4 - 40 \times 10^{-9}$ molar).[17]

The concentration of vinyl chloride in the immediate vicinity of emission sources may be sufficiently high to deplete the photochemical oxidants, and under these conditions the substance can be considered a stable pollutant. Under strong nocturnal inversions during the fall and winter, buildup of vinyl chloride from emission sources may thus be of particular concern. The half-life of vinyl chloride in more "typical" ambient conditions has not been measured but is estimated at about 20 hours.

Water and Soil

The solubility of vinyl chloride in water is 1.1 g/liter. The quantity (up to that limit) that dissolves in water will depend upon the partial pressure, or concentration, of the gas above the solution. If the partial pressure of the vinyl chloride gas above the water is reduced--for instance, if a stream runs from an area with high airborne concentrations of vinyl chloride to a less contaminated area--vinyl chloride will escape into the air. The rate of bulk exchange of gaseous vinyl chloride between atmosphere and water is about twice that of oxygen, and thus the escape will be rapid.[18]

Preliminary results of EPA investigations support the suggestion that vinyl chloride will escape rather quickly from agitated or aerated water at room temperature. Half-lives ranging from about 5 minutes to about 5 hours were observed. There was no significant difference in the rate of vinyl chloride losses from distilled water, river water, or vinyl chloride plant effluents stirred at similar rates.[19]

In addition to the bulk exchange of gaseous vinyl chloride between air and water, the chemical reactions of vinyl chloride in water have been considered. These can include reactions with water impurities or the processes of hydrolysis, oxidation, or photolysis and might act to increase or decrease the residence time (and environmental availability)

of vinyl chloride in water. Certain metal ions do have the ability to combine with vinyl chloride; for example, soluble silver and copper salts increase the solubility of vinyl chloride in water by forming complexes. Under environmental conditions, however, few of the complexation reactions are expected to be important. The estimated half-lives for hydrolysis and oxidation of vinyl chloride in water are relatively long (see Table 6), and photolysis is considered an unimportant mechanism for loss of vinyl chloride in water.

Vinyl chloride may persist long enough to accumulate biologically via direct absorption or via the food chain. This might result from the slow, continuous release of vinyl chloride from sediments and sludges, combined with either a continuous input of vinyl chloride into water or poor, erratic mixing in lakes or ponds. However, data are not yet available to indicate the extent (if any) of inorganic particulates, and sorption to bacteria, algae, or fungi has not been detected. Bacterial degradation of vinyl chloride was found to be negligible, and vinyl chloride did not affect bacterial growth under test conditions in aqueous systems.[18]

Microbiological degradation of vinyl chloride in soil may be of significance in depletion of vinyl chloride over long time periods. However, information regarding such biodegradation is not available.

From the above, it can be seen that the most important mechanism for removal of vinyl chloride from water is volatilization and that other mechanisms are of limited potential importance. It can also be seen that vinyl chloride will have a limited potential for general environmental effects as a water pollutant, principally because it is removed so rapidly and dispersed into the air. This suggests that inhalation of vinyl chloride in air is likely to be the most important exposure pathway for the general public, as it is for those persons who are occupationally exposed.

Source/Exposure Model

Figure 4 presents a conceptual model based on the foregoing estimates for the flow of vinyl chloride from emission through the environment to man. The model is qualitative, with exposures based on empirical and estimated data.

Table 7 shows the relative importance of vinyl chloride exposure for people in different environments.

In Figure 4 and Table 7, the need for control of airborne emissions to limit exposures of humans is indicated, even for the general public (i.e., those persons not occupationally exposed).

Table 6

ESTIMATED ENVIRONMENTAL PERSISTENCE OF VINYL CHLORIDE

Phase	Reaction	Rate Constant (liter/mole-sec)	Reactant Conc. (Molar)	Half-life of VC (days)	References[a]
Water	Hydrolysis	10^{-9}	$H^+ = 10^{-7}$	10^8 years	20
Water	Hydrolysis	10^{-4}	$H^+ = 10^{-3}$	80	21
Water	Oxidation	1	$RO_2 = 10^{-10}$	8×10^4	22
Air	Oxidation	10^9	$HO = 10^{-14}$	0.8	23
Air	Oxidation	1200	$O_3 = 2 \times 10^{-9}$	3.3	24

[a]For reactant concentrations.

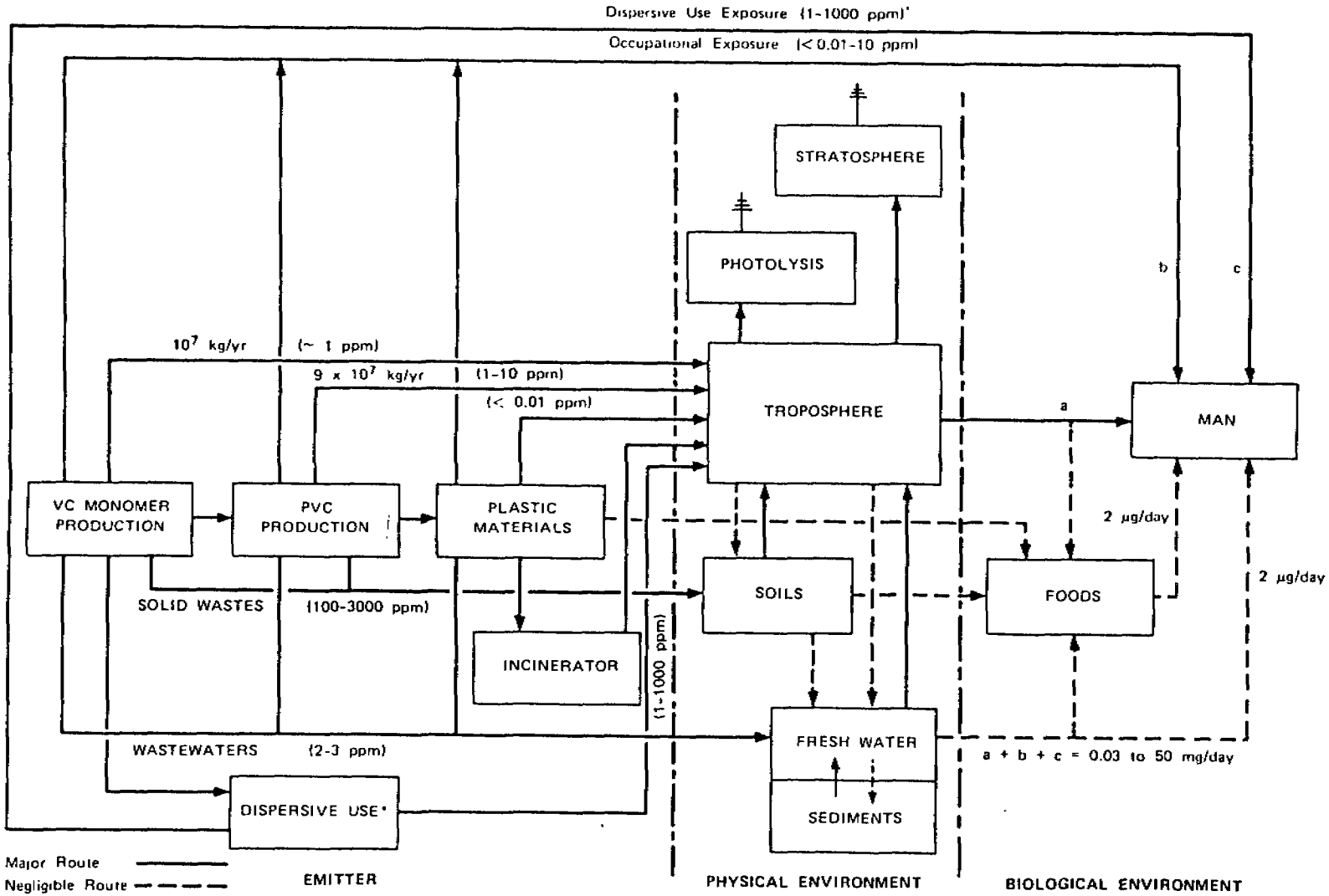


FIGURE 4 ENVIRONMENTAL FLOW OF VINYL CHLORIDE TO MAN

Table 7

RELATIVE IMPORTANCE OF VINYL CHLORIDE EXPOSURE FOR A "STANDARD MAN"
(Assuming Levels of Late 1975)

Source of Exposures	Workers (Time-Weighted-Average Exposures)						People 5 Miles from Plants		General Public	
	VCM		PVC		Fabricators					
Air										
Workplace										
Concentration[a]										
Range	0.01-30 ppm		0.05-10 ppm		0.01-2 ppm		--		--	
Average	0.5 ppm = 1.28 mg/m^3		2.0 ppm = 5.12 mg/m^3		0.01 ppm = 0.03 mg/m^3		--		--	
Average daily intake[b]		6.144 mg		24.576 mg		0.123 mg		--		--
Community										
Concentration										
Range	c		c		c		d		e	
Average	0.005 ppm = 0.013 mg/m^3		0.010 ppm = 0.026 mg/m^3		0.001 ppm = 0.003 mg/m^3		0.017 ppm = 0.044 mg/m^3		0.0005 ppm = 0.001 mg/m^3	
Average daily intake[f]		0.198 mg		0.395 mg		0.040 mg		0.870 mg		0.026 mg
Drinking water										
Concentration	1 ppb (1 µg/liter)		1 ppb		1 ppb		1 ppb		1 ppb	
Average daily intake[g]		0.002 mg		0.002 mg		0.002 mg		0.002 mg		0.002 mg
Food										
Concentration	1 ppb (1 µg/kg)		1 ppb		1 ppb		1 ppb		1 ppb	
Average daily intake[g]		0.002 mg		0.002 mg		0.002 mg		0.002 mg		0.002 mg
Total average daily intake		6.346 mg		24.975 mg		0.167 mg		0.874 mg		0.030 mg
(Population exposed)	(900)		(5,600)		(100,000)		(4,600,000)		(210,000,000)	

[a]Concentrations computed from chapter Reference 6 for vinyl chloride monomer and fabrication workers; estimated by SRI from multiple sources for PVC workers.

[b]Assuming an average daily respiratory volume of 20 m^3, and an average work week of 40 hours, an average daily respiratory volume of 4.8 m^3 while working was estimated.

[c]Assuming an average near plants of 0.017 ppm, that some workers would live near to the plants, and that the concentrations would be highest near PVC plants and lowest near fabricating plants.

[d]From chapter Reference 9.

[e]From chapter Reference 8.

[f]An average daily respiratory volume (while not working) of 15.2 m^3 was assumed (20 m^3 - 4.8 m^3).

[g]Assume an average concentration of 1 ppb and average daily intake of 2 liters of water and 2 kg of food.

Source: SRI

Chapter III

NONCARCINOGENIC EFFECTS OF VINYL CHLORIDE

Animals, Microorganisms, and Plants

In addition to tumorigenesis, other biological effects of vinyl chloride have been observed in several different animal species, microorganisms, and plants. Effects of vinyl chloride and polyvinyl chloride (PVC) and the byproducts of their production have been studied to determine toxicity, mutagenicity, metabolism, and teratogenicity.

Animals

Animals exposed by inhalation to anesthetic doses of vinyl chloride have exhibited tracheal irritation, cardiac irregularities, pulmonary edema, and hyperemia of liver and kidneys.[1] Animals acutely and chronically exposed to vinyl chloride by inhalation have been found to have increased liver weight, interstitial hepatitis, central lobular degeneration of the liver, fibrosis and necrosis of the liver, degeneration of bone, of connective tissue, and of nerves, and chronic interstitial nephritis.[2,3] These effects of exposure are seen predominantly in male animals.

Systemic aberrations, which may be precursors to angiosarcoma, are observed in the vasculature of essentially every tissue and organ in the body.[4] Chronic effects include increased blood epinephrine, fibrosclerosis of the vessels, and hypertension.[5] These systemic vascular effects are very similar to the vascular effects seen in humans. The systemic vascular effects appear in correlate with the systemic distribution of angiosarcoma in both animals and man. It is not known whether vinyl chloride or some metabolite may be responsible for the observed vascular effects in animals and man, although a metabolite(s) of vinyl chloride is suspected to be involved in carcinogenesis.

A wide range of agents increase the toxicity of vinyl chloride. Pretreatment with phenobarbital, ethanol, polychlorinated biphenyls, or certain pesticides (i.e., hexachlorobenzene) increases liver injury in animals subsequently exposed to vinyl chloride.[1]

Mutagenesis

Vinyl chloride may be mutagenic,[6,7,8] but the issue has not been resolved. Using bacterial mutagenicity tests, some investigators have observed equivocal results from tests with and without liver

microsomal preparations. Such results do not resolve the question of possible metabolic activation by bacterial enzymes.

Following metabolic activation, vinyl chloride is mutagenic. The metabolites involved may be chloroethylene oxide, chloracetaldehyde, or chloroethanol.[7,9,10,11] The metabolites may act as alkylating agents similar to bis-chloromethylether, a strong mutagen as well as a carcinogen. Chloroethylene oxide may be produced from vinyl chloride by the mixed function oxidase system (MFOS). Chloroethanol and chloracetaldehyde may be produced from vinyl chloride via chloroethylene oxide or independently by an alcohol dehydrogenase pathway. In rats, the latter pathway appears to be operative at concentrations below 50 ppm. The MFOS is apparently not a major pathway for vinyl chloride metabolism until inhalation exposures approach 200 ppm.

Teratogenesis

To date, little experimentation has been performed to investigate the deleterious effects of vinyl chloride on embryonal and fetal development, and there is no unequivocal evidence for or against teratogenic action. Fetal effects were studied in three animal species by maternal inhalation at several concentrations.[12] To assess the possibility that ethanol might alter the metabolism and thereby enhance the toxic or teratogenic potential of vinyl chloride, some animals were simultaneously exposed to 15% ethanol in drinking water. Both maternal and embryonal toxic effects were induced. Also, though gross structural malformations were not observed, minor skeletal abnormalities occurred in excess among mice exposed to 50 ppm with ethanol and to 500 ppm with and without ethanol, and among rats exposed to 2500 ppm with ethanol.

Microorganisms and Plants

No adverse effect has been demonstrated on plant growth when PVC piping has been used for irrigation. However, numbers of soil bacteria are reduced and nitrifying bacteria are increased.[13] Studies have shown that vinyl chloride causes injury to plants in a manner similar to ethylene.[14,15] Byproducts from vinyl chloride production dumped into the ocean affect the early development of marine life.[16,17] Vinyl chloride byproducts accumulate in marine animals,[18] as do other chlorinated compounds such as DDT and polychlorinated biphenyls. Furthermore, vinyl chloride byproducts have been shown to increase bacteria cell membrane fragility, which has been postulated as a mechanism of disease in higher animals, including man.[19]

Toxicity to Humans

It has long been recognized that vinyl chloride gas is capable of producing acute toxic effects in humans. However, a full understanding of the nature and extent of its chronic toxicity has been years in unfolding and has come almost exclusively from observations among occupationally exposed individuals.

Major nonmalignant effects clearly associated with occupational exposure to the gas include: acute intoxication; disturbances of liver function; acroosteolysis, often with Raynaud's phenomenon, and scleroderma; and alterations in pulmonary function, with or without abnormal chest x-ray findings. Other effects, such as alterations in the cellular elements of the blood have been reported among vinyl chloride workers by some authors, but confirmation by other authors has not been uniform.

Finally, there is some evidence that workers exposed to vinyl chloride may exhibit an increased frequency of chromosomal aberrations; that wives of vinyl chloride workers may experience an excess fetal death rate; and that mothers living in communities near PVC-production facilities may be at an increased risk of giving birth to children with birth defects. It must be emphasized that, at the time of this writing, a causal relationship between vinyl chloride exposure and these latter phenomena--chromosomal aberrations, excess fetal death rate, and birth defects--has by no means been established.

Acute Intoxication

Vinyl chloride gas is an anesthetic which is slightly irritating to the moist membranes of the eyes and respiratory tract. Its not unpleasant odor can be detected by some at concentrations as low as 300 ppm, but others cannot detect its odor until the gas reaches much higher concentrations. It has been reported that workers exposed to vinyl chloride gas can detect its presence at concentrations below the odor threshold because it produces a sensation of tingling and heat over the skin of the lower extremities.

As the concentration of gas increases, those exposed may become mildly euphoric and, at still higher levels, a typical syndrome of inebriation may appear.[20] When anesthetic concentrations of the gas are reached, about 7%, narcosis develops rapidly and may terminate in death due to respiratory failure. The use of vinyl chloride as a surgical anesthetic was considered in the 1930s, but apparently it was never used for this purpose in humans. The ability of the gas to produce cardiac arrhythmias was later recognized, at which time it was recommended that it not be considered for use as a surgical anesthetic in man.[50]

Two accidental fatal poisonings attributed to occupational exposure to vinyl chloride gas were reported in 1950.[22] Both fatalities occurred in PVC polymerization workers, one who was overcome by the gas while cleaning out a reaction vessel, and the other while inspecting a

tank containing effluent from the polymerization process. Both victims were discovered within a few minutes after having been in contact with fellow workers, suggesting that they had encountered very high concentrations of vinyl chloride gas and collapsed immediately. The autopsy findings in both cases were similar but nonspecific, consisting primarily of congestion of internal organs and failure of the blood to clot.

In 1963, six human volunteers were exposed to concentrations of vinyl chloride gas up to 20,000 ppm.[23] Exposure periods were limited to five-minute periods, twice daily, separated by a six-hour interval and continued for three successive days. At concentrations about 8,000 ppm, acute toxic effects including dizziness, nausea, headache, and diminution of vision were observed.

Disturbances in Liver Function

Workers chronically exposed to vinyl chloride gas may exhibit disturbances in liver function as well as alterations of liver structure. Functional disturbances are nonspecific in nature and often fail to reflect the true extent of structural damage.[40,42] Several investigators,[30,31] have suggested that this may be explained by the observation that the principal anatomic lesion of vinyl-chloride-induced liver disease is fibrosis, with relative sparing of the functional cells of the liver, the hepatocytes. One investigation[31] for example, after examining the microscopic structure of liver tissue from patients with vinyl-chloride-related liver disease, with and without angiosarcoma, reported the presence of a "peculiar" hepatic fibrosis but noted that the hepatocytes were not severely injured. Notwithstanding this phenomenon of hepatocyte sparing, it is apparent that ultimately these functional cells become involved in the disease process to an extent that can be detected by conventional tests of liver function such as dye excretion studies (bromsulphalein and indocyanine green) and plasma enzyme activities (serum glutamic oxaloacetic transaminase [SGOT], serum glutamic pyruvic transaminase [SGPT], alkaline phosphatase).[20,25,29,30,32,33,40,41,42]

Creech and his colleagues[29,41,43] have emphasized the importance of persistent biochemical abnormalities in the assessment of vinyl-chloride-induced liver damage. They conducted a program of medical examinations including a battery of liver function screening tests on 1,183 PVC plant employees, 309 of whom worked directly in PVC production areas. On the initial examination, 26% of the entire group of 1,183 study participants had one or more abnormal liver function test result. The percentage of production area employees with abnormal liver function test results did not differ from the overall group experience in this initial screening program. However, on repeat examination of employees who had one or more abnormal liver function test results on the initial screening, persistent abnormalities were almost three times more frequent among PVC production area workers than among those in areas not directly related to PVC production (11% vs. 4.5%).

Vinyl-chloride-related alterations of liver structure have been demonstrated by hepatic arteriography,[44,46] radioisotopic scintiscan techniques,[44,46] peritoneoscopy,[45] and by direct examination of biopsy and autopsy specimens.[30,31,41,47] Although each of these procedures can provide useful information on the nature and extent of liver involvement, at the time of this writing none is considered, of itself, to be specifically diagnostic (pathognomonic) of vinyl chloride injury.

Clinical manifestations of portal hypertension, such as hemorrhage from esophageal varices may accompany vinyl-chloride-induced liver injury as may pronounced spenomegaly.

Acroosteolysis

In the early 1960s, a symptom complex consisting of a Raynaud's-like phenomenon involving the hands, scleroderma-like changes in the skin of the hands and forearms, and osteolytic and sclerotic lesions of the bones of the extremities and sacroiliac joints were described by a number of authors in this country and elsewhere.[24,25,26] This symptom complex was called occupational acroosteolysis and appeared to be confined to workers engaged in cleaning PVC polymerization reactor vessels. The period from the beginning of exposure to the first symptoms was found to be as short as one month or as long as three years.[27] A year or two after those affected had been removed from exposure, most of the abnormalities disappeared, and the bones showed signs of healing.

The authors of one study[26] believe the cause of occupational acroosteolysis to be a combination of three factors: chemical insult, physical insult, and, due to the relatively low incidence of the disease among those exposed, personal idiosyncrasy. In a personal communication, one of the coauthors of the study, Dr. John L. Creech, expressed his belief that occupational acroosteolysis was not caused by vinyl chloride gas itself, but rather by vinyl acetate, which is formed as a condensation product in the reaction vessel when vinyl chloride polymer is heated.

The relationship, if any, between occupational acroosteolysis and vinyl-chloride-induced liver disease is unclear.

Changes in the Lung

Both radiographic and functional changes of the lung have been reported in vinyl-chloride-exposed workers.

One group of investigators[34] obtained spirometry and maximum expiratory flow volume curves in 348 workers in a vinyl chloride polymerization plant. Their major finding was decrease in air flow in over 50% of the workers examined. This abnormality correlated with age and duration of exposure. A relationship with smoking was noted only in younger workers with exposure of less than ten years. Among workers aged 40 and

over, and among workers with exposure of more than 20 years, the prevalence of this impairment was similar in smokers and nonsmokers. The authors suggested this to be evidence that occupational, or other factors aside from smoking, were operative.

Another team of scientists[35] conducted a survey of vinyl chloride polymerization workers which included chest roentgenograms, a complete smoking history, a chronic bronchitis questionnaire, and pulmonary function studies. They noted a definite increase in roentgenographic changes associated with length of exposure to vinyl chloride. These changes were primarily comprised of small linear reticular and rounded opacities. The prevalence of positive smoking history was found to be higher in workers with abnormal chest roentgenograms. No statistically significant difference was found between chronic bronchitis as diagnosed by questionnaire and abnormal chest roentgenograms. Pulmonary function studies showed obstructive changes in almost half of the workers examined. No correlation between chest roentgenographic changes and pulmonary function abnormalities could be established. The abnormalities observed prompted the authors to suggest that a similar mechanism may be active in the pulmonary tissue of vinyl-chloride-exposed workers as is recognized at other sites.

Hematologic Changes

Hematologic changes among vinyl chloride workers have been reported by several authors. The most striking changes have been those reported by investigators from the Federal Republic of Germany (FRG).[32,33,48] One group[32] found that 81% of 70 patients examined exhibited slight to severe thrombocytopenia, independent of the presence of splenic enlargement and accompanied by normal bone marrow morphology. They hypothesized that these findings were the result of thrombocyte destruction not only in the spleen, but at other sites as well. Another group of investigators, reporting from the U.S.A., noted the FRG reports of thrombocytopenia but found only one such case in their series of 354 vinyl chloride workers.[35]

Other hematologic changes in vinyl chloride workers have also been reported, including anemia,[20,35] leukopenia,[20,32,35] and reticulocytosis.[32,48] For the most part, however, these findings have represented only infrequent, minimal deviations from normal values.

Chromosomal Aberrations, Excess Fetal Death Rate, and Birth Defects

Chromosomal aberrations,[49] increased fetal death rate,[28] and birth defects[36] have all been reported as possible effects of occupational or community exposure to vinyl chloride.

In one study,[49] chromosome analyses were conducted on seven men occupationally exposed to vinyl chloride and on three control subjects, all of whom worked in the same plant. The seven exposed subjects had

worked with vinyl chloride for 7-29 years. Periodic clinical examinations had showed no evidence of acroosteolysis or liver dysfunction. Chromosomal analyses of cells from the exposed and control groups revealed 9.52% and 1.94% aberrations, respectively. This difference was found by the authors to be significant (<0.001).

Stimulated by reports[37,38] that vinyl chloride is mutagenic in microbial test systems, a team of investigators examined the pregnancy outcome among wives of workers exposed to vinyl chloride.[28] These investigators compared fetal death rates prior to and subsequent to the husband's exposure to vinyl chloride. They found that prior to the husband's exposure, fetal death rates (6.1%) were similar to a control group (6.9%) not exposed to vinyl chloride, but subsequent to exposure, fetal death rates were significantly increased (15.8%) when compared both with the control group and with pre-exposure rates.

The authors postulated several mechanism by which fetal loss might be related to vinyl chloride exposure. They considered fetal toxicity or germ-cell mutagenesis in the mother through indirect vinyl chloride exposure from the father unlikely because the high volatility of vinyl chloride would preclude its being inadvertently transported from the workplace to the home via clothing, etc. They considered the leading possibility to be germ-cell damage in the father as a direct consequence of vinyl chloride exposure in the workplace.

Several technical weaknesses heavily becloud the validity of the results reported in this study. No interviews were conducted with worker's wives; data on total number of conceptions and pregnancy outcome were ascertained by interviews with male employees. Moreover, according to the authors "no data were obtained concerning maternal age, except indirectly through paternal age." Lastly, although as correctly pointed out by the authors, fetal loss is known to increase with increasing paternal age, in this study the results were directly the opposite. The fetal death rate was higher among wives of husbands less than 30 years of age than among wives of older workers (20% vs 13%). In a later communication,[39] the authors suggested that if vinyl chloride exposure were truly associated with the observed excess in fetal mortality, the greater effect in younger workers could, in thebry, reflect the placement of younger workers in less desirable work exposure situations for seniority reasons. Nonetheless, the implications raised by this report must be considered highly speculative until additional supporting evidence can be developed.

A study[36] of residents living in communities adjacent to vinyl chloride facilities revealed an excess number of congenital malformations when compared to vital statistics data from both the state and the balance of the counties in which the communities were located. This excess included defects of the central nervous system, genital organs, and upper alimentary tract. A significant excess of club foot was also observed. The authors could not attribute the noted excess to race, maternal age, or regional differences in reporting of birth defects.

This study has many important limitations. No information on ambient air levels of vinyl chloride was presented, nor was consideration given to other factors that are known to contribute to the incidence of congenital malformations, such as infectious agents, other industrial exposures, background radiation, or genetic factors.

A subsequent study by the U.S. Public Health Service, Center for Disease Control's hospital-based birth defects monitoring program[51] confirmed a moderate increase in central nervous system congenital malformations in one of the communities included in the above investigation. None of the interviewed parents of affected infants had ever worked at either of the two vinyl chloride plants located in the community, nor did they live within two miles of either plant. The investigators concluded that, in their study, they could find no association between vinyl chloride exposure and congenital malformations.

Chapter IV

CARCINOGENICITY OF VINYL CHLORIDE

Animal Studies

There exists adequate experimental evidence to confirm that vinyl chloride is carcinogenic in mice and rats when administered by inhalation. A dose-response relationship has been indicated in mice receiving doses of 50 to 10,000 ppm of vinyl chloride.

The tumors observed in mice are predominantly liver angiosarcomas, mammary adenocarcinomas, and lung adenocarcinomas. Other types of tumors have also been observed.

In rats, the tumors observed are predominantly liver angiosarcomas, Zymbal gland tumors, nephroblastomas, lung adenocarcinomas, and osteochondromas. At the lowest level of exposure, 50 ppm, a single nephroblastoma and a single liver angiosarcoma were observed in rats. The frequency of these and the other tumors increased with increasing levels of exposure. There is also evidence that vinyl chloride is carcinogenic in rats by both transplacental and oral routes of administration.

Finally, preliminary results indicate the carcinogenicity of vinyl chloride in hamsters following inhalational administration. Liver angiosarcomas as well as skin epitheliomas, lymphomas, and melanomas have been observed in experiments still in progress.

Mice

In mice receiving doses of 50 to 10,000 ppm of vinyl chloride the tumors observed were primarily lung angiosarcomas, mammary adenocarcinomas, and lung adenocarcinomas (Table 8).[1]

In a subsequent report on the progress of the same experiment,[3] additional tumors were observed including subcutaneous angiomas, skin squamous carcinomas and acanthomas, renal angiosarcomas, hepatic angiomas, liver fibroangiomas, one heart fibroangioma, and an ossifying interscapular angioma (Table 9).

In a related inhalation study, a total of 200 mice of both sexes (CDI Swiss Charles River) were exposed to either 0, 50, 200, or 2,500 ppm of vinyl chloride vapor for seven hours per day, five days per week for nine months. Preliminary data were published following eight months of exposure. At this time, when tissues from only a few animals had been

Table 8

INCIDENCES OF TUMORS IN MICE EXPOSED TO VINYL CHLORIDE
FOR 30 WEEKS AND DYING WITHIN 34 WEEKS

Concentrations of VCM (ppm) given 4 hrs/day on 5 days/week	Total # of animals at start	Survivors at 34 weeks	Adenomas & adenocarci-nomas of the lungs	Mammary adeno-carcinoma	Angio-sarcoma of the liver	Others	Total Tumors
10,000	60	26	12	5	0	3	20
6,000	60	32	12	4	1	2	19
2,500	60	37	6	2	0	0	8
500	60	40	8	1	2	0	11
250	60	44	4	2	0	0	6
50	60	42	0	1	0	0	1
Controls	150	122	0	0	0	0	0

Source: Chapter Reference 1

Table 9

INCIDENCES OF TUMORS IN MICE EXPOSED TO VINYL CHLORIDE
FOR 37 WEEKS AND SURVIVING UP TO 41 WEEKS

Concentrations of VCM (ppm) given 4 hrs/day on 5 days/week	Total # of animals at start	Survivors at 41 weeks	Adenomas & adenocarcinomas of the lungs	Mammary adenocarcinoma	Angiosarcoma of the liver	Vascular tumors of other types and/or sites	Others	Total Tumors
10,000	60	8	27	9	4	7	2	49
6,000	60	16	22	8	2	1	4	37
2,500	60	24	12	4	4	1	2	23
500	60	28	16	2	4	1	1	28
250	60	33	11	6	3	1	0	21
50	60	29	0	7	0	4	0	11
Controls	150	108	1	0	0	0	0	1

Source: Chapter Reference 3

examined, a dose-response relationship was observed in a number of mortalities with neoplasms. The tumors observed at all dose levels included lung adenomas, liver angiosarcomas, and mammary gland adenosquamous carcinomas. The number of each of these types of tumors increased with dose. At the time of publication, no tumors of any type had been observed grossly in any of the untreated control mice.[2]

Rats

An inhalation study was performed in rats in which a group of 26 males (Wistar) was exposed to an atmospheric concentration of 30,000 ppm of vinyl chloride for four hours per day, five days per week, for 12 months. In the 17 surviving rats, skin tumors (subsequently classified as Zymbal gland tumors) developed in all, lung tumors (predominantly adenocarcinomas) developed in seven, and osteochondromas developed in five rats. No tumors were observed in the 25 untreated control animals killed at an unstated time.[1]

Male and female adult Sprague-Dawley rats were exposed by inhalation to concentrations of 10,000, 6,000, 2,500, 500, 250, or 50 ppm of vinyl chloride for four hours daily, five days per week, for 52 weeks. The rats were found to exhibit the incidence of tumors shown in Table 10.

There is evidence for the carcinogenicity of vinyl chloride in rats following prenatal exposure. Pregnant female rats (Sprague-Dawley) were exposed by inhalation to 6,000 to 10,000 ppm of vinyl chloride for four hours daily, between the 12th and 18th days of pregnancy. A single subcutaneous angiosarcoma was observed in the offspring of each exposure group at 75 weeks after the start of the experiment.[1,3] This indicates that vinyl chloride or metabolic conversion products traverse the placental barrier.

There is evidence that vinyl chloride is carcinogenic via the gastrointestinal tract.[4] Vinyl chloride dissolved in olive oil was administered five times per week to 320 13-week old rats in four groups (40 males and 40 females) in concentrations equivalent to 50.0, 16.6 and 3.3 mg/kg body weight. After 50 weeks, one angiosarcoma of the liver was observed in one male animal in the group given 16.6 mg/kg. This is equivalent to 863 mg total over a 52-week period. One angiosarcoma of the thymus gland was observed in a female animal receiving 50 mg/kg, which is equivalent to three times that given the male animal. This oral dosage is equivalent to the total inhalational dose that induced both liver angiosarcomas and renal nephroblastoma, i.e., 800 mg.[3]

Hamsters

Preliminary results from inhalational studies in hamsters suggest the carcinogenicity of vinyl chloride in this species[3] (Table 11). A total of 268 male golden hamsters received vinyl chloride at various

Table 10

INCIDENCES OF TUMORS IN RATS EXPOSED TO VINYL CHLORIDE
FOR 52 WEEKS AND SURVIVING UP TO 130 WEEKS

Concentrations of VCM (ppm) given 4 hrs/day on 5 days/week	Total # of animals at start	Survivors at 130 weeks	Zymbal gland tumor	Nephro-blastoma	Angio-sarcoma of the liver	Angio-sarcoma at other sites	Others	Total Tumors
10,000	69	0	16	4	7	0	6	33
6,000	72	0	7	4	13	2	1	27
2,500	74	0	2	6	14	3	1	26
500	67	0	3	4	7	2	1	17
250	67	1	0	6	4	2	2	14
50	64	3	0	0	0	0	0	0
Controls	68	1	0	0	0	0	0	0

Source: Chapter Reference 1

Table 11

INCIDENCES OF TUMORS IN HAMSTERS EXPOSED TO VINYL CHLORIDE FOR 28 WEEKS

Concentrations of VCM (ppm) given 5 days/ week	Total # of animals at start	Survivors after 28 weeks	Angio-sarcoma of the liver	Tricho-epithelioma (skin)	Lymphoma	Melanoma	Total animals with tumors	Total Tumors
10,000	35	19	0	1	0	0	1	2
6,000	32	21	0	1	1	1	1	4
2,500	33	19	0	0	0	0	0	0
500	33	23	1[a]	0	1	0	1	3
250	32	18	0	0	0	0	0	0
50	33	23	0	0	1	0	0	1
Controls	70	49	0	0	0	0	0	0

[a] [As of] date of final draft.

Source: Chapter Reference 3

concentrations for 30 weeks. The results after 28 weeks of exposure are presented in Table 11.

In a second inhalational study in hamsters still in progress, a total of 200 Golden Syrians were exposed to either 2,500, 200, 50 or 0 ppm of vinyl chloride vapor seven hours per day, five days per week for 12 months. After only eight months of treatment, one liver angiosarcoma was observed in a male hamster exposed at 2,500 ppm. Control data were not adequately reported.[2]

Epidemiology and Case Reports

In this section, information on the association between exposure to vinyl chloride monomer and the occurrence of cancer in humans is critically reviewed. Three types of studies have yielded this information:

- Case Reports--reports of clinical experience, with anecdotal opinions on the relationship of some cancers to vinyl chloride.
- Cohort Studies--epidemiologic investigations designed to determine whether vinyl chloride workers die of cancer at a higher than expected rate.
- Community Studies--epidemiologic investigations which search for elevated cancer mortality rates among dwellers in communities where there is known to be a potential for occupational and community exposure to vinyl chloride--that is, in communities in which a vinyl chloride facility is located.

The evidence that vinyl chloride causes angiosarcoma of the liver in humans may be considered conclusive. There is strong evidence implicating the chemical as a cause of cancer of the central nervous system, especially glioblastoma multiforme. The evidence for cancer of the lung and lymphatic system is suggestive but not conclusive. For other liver and biliary cancers, the evidence is weak. The evidence that vinyl chloride exposure is a cause of the buccal cavity and pharynx is too weak to be considered important.

Case Reports

In January 1974, Creech and Johnson reported three cases of angiosarcoma* of the liver in workers employed in a polyvinyl chloride

*Angiosarcoma is a malignant tumor composed of neoplastic endothelial cells. It is also known by the synonyms hemangiosarcoma and malignant hemangioendothelioma. The tumor may originate in organs other than the liver, although to the time of this writing, all reported vinyl-chloride-associated angiosarcomas appear to have originated in the liver.

(PVC) polymerization plant in Louisville, Kentucky.[5] This type of tumor was known to be extremely rare, appearing at a rate of only 0.05-0.2 new cases per million population[6] (25 to 30 new cases reported in the U.S. annually).[7] Thus, it became immediately apparent that this cluster of cases was unlikely to be a result of chance, and further studies were undertaken by these and other authors.

As a result of these studies, which included investigations of other groups of vinyl chloride workers as well as experimental exposure to laboratory animals, vinyl chloride was strongly implicated as an agent capable of causing angiosarcoma of the liver in both man and animal. By December 1975, 45 cases of angiosarcoma of the liver had been discovered throughout the world,[8] some in individuals living and some among the records of the deceased. All had documented occupational exposure to vinyl chloride.

The possibility that other ingredients or contaminants associated with PVC production were the cause of the tumors was rejected because none of these materials was present in other than minute concentrations compared to vinyl chloride itself, and none appeared to be capable of inducing angiosarcoma in experimental animals. Also, a detailed investigation of some of the earlier reported U.S. cases revealed no significant exposure to substances known to be toxic to the liver,[7] particularly the two types of compounds historically recognized to be capable of inducing angiosarcoma of the liver in humans--thorium dioxide (Thorotrast)[9] and arsenic compounds.[10]

Further investigations established that exposure to vinyl chloride during the polymerization process was but one of several types of exposure that could lead to the induction of angiosarcoma. For example, several cases were reported in workers involved in the production of vinyl chloride itself and two in workers engaged in fabricating PVC into the vast array of products for which it is used.[11] All evidence presently available indicates that these latter cases were attributable to exposure to residual unreacted vinyl chloride released from the PVC during fabrication, not to the PVC itself--there is no evidence to implicate PVC as a cancer hazard.

There have been reports of angiosarcoma of the liver occuring in individuals who, although not occupationally exposed to vinyl chloride, resided near a vinyl chloride facility--a fabrication plant in one case[12] and a polymerization plant in another.[13] There have also been reports of cases of carcinoma of the liver (not angiosarcoma) in individuals occupationally exposed to vinyl chloride,[14] in people living in the neighborhood of a vinyl chloride plant,[15] and among family members of vinyl chloride workers.[14] It should be emphasized that these reports in themselves are not sufficient to imply a definite causal relationship.

Cohort Studies

The purpose of a cohort study is to compare the mortality patterns (incidence of the disease, death rate, and cause of death) of one group of individuals with another. The seven cohort mortality studies described here compare the mortality patterns of certain groups of vinyl chloride workers with U.S. population figures. Table 12 summarizes the major features of these seven cohort mortality studies.

Ideally, a cohort study of vinyl chloride workers should:

1. Include information on a large number of exposed workers so that the presence of a relatively rare cancer can be detected, and to minimize the possibility that by chance alone a cause of death will appear with an unusually high frequency; and

2. Identify all, or virtually all, of those workers exposed to vinyl chloride for a substantial period of time, preferably 10 to 20 years.

None of the studies discussed here fully meet both of these desirable characteristics, but several come close, and the study findings are sufficient to indict vinyl chloride as an agent capable of causing angiosarcoma of the liver in man.

Tabershaw & Gaffey [17,18]--Manufacturing Chemists Association Study

This study examined the records of 9,677 workers in 37 vinyl chloride and PVC plants and compared their mortality experience with U.S. population figures. Only workers who by the end of 1972 had experienced at least one year of exposure to vinyl chloride were included in the study. In all, 707 deaths were known to have occurred in this cohort. An excess of deaths from cancer of the digestive organs and peritoneum was found, which was entirely explained by angiosarcoma of the liver. Also identified was an increased risk of developing cancer of the respiratory system among exposed workers. This risk increased slightly with increasing intensity and duration of exposure. The death rate from lymphoma followed a similar but more pronounced pattern, the rate more than doubling in the group of workers with the highest intensity and longest duration of exposure. There was no increased risk found for leukemia.

A two-fold excess in the number of deaths from cancer of the central nervous system was found and was statistically significant. However, the excess was not related to duration or estimated intensity of exposure to vinyl chloride.

Table 12

SUMMARY TABLE OF COHORT STUDIES OF OCCUPATIONAL EXPOSURE TO VINYL CHLORIDE

Study	Cohort Size	Cohort Qualification[a]	Estimation of Exposure Intensity	Estimation of Exposure Duration	Follow-up of Cohort	Total Deaths	Increased Risk of Cancer: Angiosarcoma of Liver	Other Liver and Biliary	Lung	Central Nervous System	Lymphatic and Hematopoietic
Tabershaw-Cooper May 1974	9,109 employees, 36 plants	Greater than 1 yr exposure prior to 12/31/72	Est. by Industrial Hygiene & Safety Personnel	Compared 5 yrs w/5+ yrs	95.7% traced; 97.3% death certificates found	525	Yes	Not analyzed separately but digestive system excess explained by angiosarcoma	Yes	Included in "other & unspecified" which shows increased risk	Leukemia no Lymphoma
Proportional Mortality (Harvard School of Pub. Health) 8/17/74	Total employees unknown; 2 plants	All deaths 1947-1973 active or retired employees	Not available	Not available or examined	100% of all active and actual. None of others	161	Yes	Yes, but explained by angiosarcoma	Yes	Brain cancer excess	Yes
Goodyear Mt. Sinai Study 1/31/75	257 employees, 1 plant	All employees 1/9/46-63 exposed at least 5 yrs	Only by department	Only examined latency and restriction of cohort (i.e. 5 yrs exposed)	255 out of 257 traced (99%)	24	3 out of 24 total deaths	No deaths	No deaths	1 death from glioblastoma	2 deaths
Dow July 1975	594 employees, 72 exposed to both arsenicals and VC; 1 plant	All employees in areas potential VC exposure 1942-60	Based on Air available sampling data	1 yr vs. 1+ yrs used only	97% traced	88 total; 79 among those not exposed to arsenicals	None found. (death certificates)	None reported	Resp. system no	None reported	None reported
BF 12/13/75	2,120 employees, 1 plant	All male employees exposed to VC from 1948 to 10/73	Only by job categories	Compared 10 yrs 10-14 15+	7 lost, (99% traced)	152; 136 under age 75 (age to which analysis restricted)	None 1 death after study period	Not examined separately (no excess for digestive system)	No, but slight excess in those exposed before 1956	None reported	None reported
NIOSH in press (1976)	1,294 employees, 4 plants	All employees with 5 or more yrs. employment who have passed 10th anniversary of employment date	None	Only examined latency of 15+ yrs separately	7 lost; (99% traced)	136	Yes	Yes	Yes	Yes (at least 9 of 10 glioblastoma multiforme	Yes

[a]Different cohorts may contain some of the same individuals

Finally, a slightly increased risk of cancer of the buccal cavity and pharynx was found in workers with the lowest intensity and shortest duration of exposure to vinyl chloride. This led the authors to suggest that this finding was likely to be the result of chance rather than a consequence of vinyl chloride exposure. Other studies have not found an increased risk among vinyl chloride workers to cancers of this category.

The findings reported in this study are enhanced in importance by the large size of the cohort and the very high percentage of the members of the cohort accounted for (95%). Also, an important aspect of the study was the inclusion of an estimated exposure index. This index, although likely to be inconsistent somewhat from plant to plant and from year to year because it was necessarily based on subjective estimates rather than actual air sampling, permitted some notion of the effects of duration and intensity to be included in the data analysis.

Monson, et al[19]--B. F. Goodrich Study

This proportional mortality study was an analysis of 161 deaths occurring between 1947 and 1963 among B. F. Goodrich employees who had worked in either the company's vinyl chloride plant or in its PVC polymerization plant. Both of these plants were located in Kentucky. The percentage of deaths attributable to various cancers and other causes was calculated and compared with U.S. population figures. It was found that a higher-than-expected percentage of the B. F. Goodrich workers had died from cancer of the liver and biliary tract. (This excess persisted even after subtracting all known cases of angiosarcoma of the liver from the analysis.) A four-fold increase in the percentage of workers who had died of cancer of the brain was found, as was a 50% increase in cancer of the lung. Cancer of the lymphatic and hematopoietic system also showed a 50% increase.

Although it must be emphasized that proportional mortality studies such as this do not express the risk of developing cancer or any other disease, the complete ascertainment of cause of death in active and retired employees over the entire study interval was a major strength of this study. Aspects of the study which tend to weaken the significance of the positive findings are: (1) no information was obtained on living workers who had left B. F. Goodrich employment before retirement; (2) no information was obtained on intensity or duration of exposure; and (3) many persons were apparently included in the study who had never been exposed to vinyl chloride.[20]

Nicholson, et al[21]--Goodyear Study

The population studied was 257 individuals, each with a history of at least five years of exposure to vinyl chloride in a PVC polymerization plant during the period 1946 to 1963. In each case, 10 or more years had passed since commencement of employment.

Twenty-four deaths were identified, including three confirmed cases of angiosarcoma of the liver, one brain cancer, and two lymphomas. The authors suggested that a causal relationship between vinyl chloride exposure and the appearance of these relatively rare cancers was likely.

A major achievement was that 255 out of 257 (99%) members of the cohort were successfully located and their current health status determined. The requirement for over five years of exposure was also important in that it increased the likelihood that any given member of the cohort had a reasonable degree of contact with vinyl chloride. The absence of air sampling data and the availability for analysis of only 24 deaths, limit the extent to which the findings of this study can be considered representative of the true mortality patterns for workers in PVC polymerization plants.

Ott, et al[22] and Holder[23]--Dow Study

The population in this study was 594 chemical workers exposed to vinyl chloride at a single vinyl chloride manufacturing facility between the years 1942 and 1960. Expected deaths in this study were determined from U.S. white male mortality rates. Eighty-eight individuals were known to have died; death certificates were located for 86 of these. An exposure rating of low, intermediate or high, depending upon existing industrial hygiene data, was assigned to each member of the cohort.

The authors of the study concluded that workers exposed to vinyl chloride at levels in excess of 220 ppm experienced an "apparent increase in overall malignancy rate." However, this increase was not found to be statistically significant. Angiosarcomas of the liver were not found at any level of exposure.

The value of this study is enhanced by the availability of air-sampling data specific for vinyl chloride exposure as well as by a successful followup of over 95% of the cohort. Some misclassification of dose-response relationship is apparent, and to some extent this confuses the study results. For example, in order to cope with the fact that many employees had been exposed to a rather wide variety of concentrations of vinyl chloride at different times during their employment, the authors assigned each employee to the exposure group representing the highest level of exposure that he or she had experienced for more than one month. Then, in assessing duration of exposure, the authors only considered duration at the highest level to which the employee had been exposed. As a result, a worker who, for example, had spent two months at the highest

exposure level and 20 years at the intermediate exposure level, is classified as "high exposure--less than one year," even though, of course, his exposure had been of much longer duration. Because of this method, an unknown proportion of employees are misclassified by duration. The extent of this misclassification may be sufficient to explain the lack of observed risk gradient with increased duration of exposure.

A major confounding variable in this study was that 72 of the workers included were also exposed to arsenicals, which themselves are known carcinogens. Therefore, it was necessary to reduce the study population from 594 to 522, a requirement recognized by the authors and, accordingly, incorporated in the analysis. Of these 522, 228 had less than one year of exposure and may not have been truly part of the population at risk. Ninety-eight of these workers had ten or more years of exposure, and 34 were exposed for a minimum of 20 years. The authors did not analyze these groups separately; rather they reported the cancer risk of all workers with one or more years of exposure. Separate analysis of the data from workers with longer exposure would have been helpful to the interpretation of the study results.

Duck, et al[24]--British Petroleum Study

This study included 2,120 male employees exposed to vinyl chloride at a PVC polymerization plant in Wales during the period 1948 to October 1973. Until 1968, vinyl chloride was also produced in this plant. The authors compared the mortality experience in the exposed population with that experienced by males ages less than 75 years in England and Wales, 1955-1972. They found no excess of cancer deaths overall, nor was an increase in deaths from cancer of any specific site observed.

This study has several major strengths. First, only seven of 2,120 subjects were lost to followup and, secondly, many of the participants had long exposure periods--582 had 10 or more years exposure, and 336 had 15 years or more. Weaknesses of the study include the absence of information on air concentrations, which precluded the inclusion of exposure intensity in the analyses. Moreover, the authors were unable to realistically separate jobs by intensity or duration of exposure.

Waxweiler et al[25]--National Institute for Occupational Safety and Health (NIOSH) and Center for Disease Control (CDC) Study

This study by the NIOSH and CDC included 1,294 workers employed in four PVC polymerization plants. Each study subject had five or more years of exposure, and, for each, a period of ten years had elapsed since the date of first exposure. In addition to the cohort study, all deaths from cancer of any site, or liver disease of any type, recorded on a death certificate were investigated further, regardless of whether the case met the cohort-inclusion criteria of five years exposure and ten years latency.

Of the 14 histologically confirmed cases of biliary and liver cancer among workers from the four plants, 11 cases of angiosarcoma were found, all of which were in workers who had worked as reactor cleaners. The authors also identified a three- to five-fold increased risk of cancer of the central nervous system (3 cases). In 9 of 10 brain cancer cases that included 7 noncohort cases, a diagnosis of glioblastoma multiforme was made. (No histological diagnosis was available for the tenth case.) The authors compared this finding with the results of an autopsy series reported by Manuelidis and Solitaire[26] in which only about one-third of the tumors of the central nervous system were found to be glioblastoma multiforme. The authors also found a 50% increase in risk for respiratory system cancers and for cancers of the lymphatic and hematopoietic system.

This study has a number of major strong points: (1) the authors were able to locate greater than 99% of the cohort, and (2) the population of the study was not significantly diluted by minimally exposed individuals. Also, the study compared the findings for one plant--which contributed more than two-thirds of the total person-years of exposure--with the population of three other plants. Similar results were found in each of the two groups, suggesting that the excess risks observed were not peculiar to a specific plant, but rather resulted from vinyl chloride exposure.

Data on air concentrations of vinyl chloride were not available; therefore, dose-response relationships could not be examined.

Pasternack[27]--Firestone Study

This proportional mortality study examined all 85 deaths occurring between 1947 and 1974 among active and retired employees of a chemical and plastic plant complex. According to the author, employees in these 2 plants were all exposed to vinyl chloride and were the only employees in the complex so exposed. In addition, the mortality experience of 348 tire-plant workers, unexposed to vinyl chloride, was examined for comparison.

Utilizing information obtained from death certificates, the author compared the proportion of the 85 deaths, by cause, with the proportion of U.S. white male deaths, by cause, during 1968. He also compared the proportion of deaths, by cause, between the 85 vinyl chloride workers and the 348 tire-plant workers. One death from angiosarcoma of the liver and one death from brain cancer were found. A slight excess of cancers of the respiratory and digestive system was present in both groups when compared with the U.S. white male population figures. However, there was no significant excess in deaths from any specific cancer when the mortality experience of the 85 vinyl chloride workers was compared with that of the 348 tire-plant workers.

Like the Monson, et al[19]--B. F. Goodrich study, this study accounted for every death in active and retired employees, a definite strength. A confusing factor, however, is the choice of tire-plant

workers for comparison since tire workers are thought to be at increased risk of cancer development themselves.

Community Studies

Aspects of a study of the distribution of birth defects in residents living in three Ohio communities,[28] where vinyl chloride production facilities are either located or are nearby, are reported elsewhere in Chapter III. For the purpose of this section, however, the cancer mortality data reported in this study are relevant.

Thirty-eight deaths from tumors of the central nervous system were reported among the residents of these communities during the period 1958 to 1973 in whites aged 45 and older. Based on the rates for Ohio, only 24 deaths from cancer of this site would be expected. This difference is statistically significant ($p < 0.01$).

This study has many important limitations. No information on ambient air levels of vinyl chloride was presented, nor is it known whether the plants use vinyl chloride or PVC. Also, no consideration was given to other environmental factors that might contribute to the incidence of cancer, such as infectious agents, other industrial exposures, background radiation, genetic factors, or exposure to other chemicals. Personal habits such as smoking were not considered.

Dose-Response Relationships

The existence of a dose-response relationship between exposure to vinyl chloride and development of angiosarcoma of the liver has often been suggested. Support for this notion is found in the observation that the earliest reported victims of angiosarcoma all began their work in the industry as polymerization vessel cleaners, and were, in the course of that work, exposed to extremely high concentrations of vinyl chloride gas. Moreover, one research study[7] noted an inverse relationship between the length of time spent as a polymerization vessel cleaner and the period between first exposure and tumor diagnosis. However, no information appears to be available on risk as a function of time spent as a vessel cleaner, and this question was not examined in any of the studies reviewed here. Thus, while it is not known just how likely it is that a vessel cleaner will develop the tumor, it appears that if he is going to do so, the length of time he spent at that occupation will influence how quickly the tumor will appear.

Several cohort studies were able to examine dose-response relationships, although in a relatively imprecise fashion. The Tabershaw and Gaffey[17, 18] study found a dose-response relationship with intensity and duration of exposure in the case of several cancers. Listed in order from the strongest to the weakest relationship, these are:

- Digestive system cancers (entirely explained by angiosarcoma of the liver).
- Cancers of "other and unspecified sites" (nearly one half of these cases were brain cancers).
- Lymphoma.
- Lung cancer.

The Dow study had access to actual air-sampling data, but, because of the small number of individuals in the cohort, the study was of limited value as an indicator of dose-response relationships. The Duck, et al[24] British Petroleum study found no gradient of response with duration of exposure for cancers of the digestive system or lung, nor in this study was there a gradient of response with duration of exposure for all cancer sites combined.

Discussion

Angiosarcoma of the Liver

Because of the rarity of this tumor, the case reports already available clearly reflect the presence of a greatly increased risk among workers exposed to vinyl chloride gas. The fact that some of the cohort studies failed to detect this increase in risk is probably a result of misdiagnosis, a circumstance clearly verified by Tabershaw and Gaffey.[17] These authors found that on death certificates, angiosarcomas of the liver had been diagnosed as liver carcinoma, or even as cirrhosis.

Other Liver and Biliary Cancer

In the Waxweiler et al[25]--NIOSH and CDC study, an excess of liver and biliary cancers was found. Other studies either found no excess in this cancer category or found an excess that could be explained completely by angiosarcoma of the liver. There have been isolated case reports of cancer of these sites occurring in individuals with possible environmental exposure, such as neighborhood or household exposure; but because these tumors are not so rare as angiosarcoma of the liver, these findings must be considered as only suggestive.

Central Nervous System Tumors

Most studies have found a higher risk of these tumors in vinyl chloride workers than would be expected based on U.S. population figures, but the excess has not been related to duration or intensity of exposure. In those studies that did not find a higher risk, the failure to do so may be a consequence of small cohort size. Thus, it is suggestive, but by no means conclusive, that vinyl chloride is a cause of cancer of this site, especially glioblastoma multiforme.

Lung Cancer

Most studies have found an increased risk of cancers of this site among vinyl chloride workers in comparison with U.S. population figures. The failures of the Nicholson, et al.[21]--Goodyear and, perhaps, the Ott, et al.[22] and Holder[23]--Dow studies to find an increased risk in this category could be due to the small cohort size. There is no clear explanation for the failure to find an increased lung cancer risk in the Duck, et al.[24]--British Petroleum study, other than the possibility that no increased risk of lung cancer truly exists in vinyl chloride workers. Thus, the evidence that vinyl chloride causes lung cancer is suggestive, but far from conclusive.

Cancer of the Lymphatic and Hematopoietic Systems

Most studies show an increased risk of cancer in these categories. The Tabershaw and Gaffey[17, 18] study suggests that the increased risk is for lymphoma, rather than leukemia. Again, the failure to find an increased risk in this category by the Ott et al.[22] and Holder[23]--Dow study may be the result of small numbers. The negative findings of the Duck, et al.[24]--British Petroleum study are unlikely to be explained by methodological problems; thus, they must be considered inconsistent with the findings of the other studies. Accordingly, it can only be said that the increased risk of cancer in these categories among vinyl chloride workers is suggestive--not conclusive.

Cancer of the Buccal Cavity and Pharynx

The Tabershaw and Gaffey[17, 18] study identified increased risk for this site, but only in those groups with the lowest level of exposure and the shortest duration of exposure. No increased risk of cancer of this site was found in any other study, and the authors themselves noted that this finding is unlikely to be due to vinyl chloride exposure.

Chapter V

CANCER CONTROL PROGRAM FOR EXPOSURE TO VINYL CHLORIDE

Control of the health hazard associated with exposure to vinyl chloride depends largely upon reducing exposure in the workplace and in the general environment, and this, in turn, depends principally on application of appropriate engineering control measures. Also important in controlling this health hazard are two other measures--medical surveillance and education. These three control measures are the subject of this chapter.

Engineering Control

Substantial progress has already been made in reducing exposure to vinyl chloride both in the workplace and in the general environment. Effective engineering control is a demonstrated reality in many vinyl chloride facilities, and the technology exists to extend satisfactory control to almost all monomer production plants and PVC manufacturing facilities. Since these sites are the source of about 96% of all vinyl chloride emitted into the nation's atmosphere (see Chapter II), it is of paramount importance that they be considered a primary target for control.

This section is not intended as a detailed process design manual, but as a guide to essential elements of good engineering control practice that will permit informed community review of a total control program. The specific measures to be taken at each site must be dictated in large part by the nature of the process and by the professional judgment of the control engineers responsible for implementation of the control strategy.

Simplified schematic flow charts of some of the major steps in the vinyl chloride-PVC-fabrication chain are given in Figures 5, 6, and 7. Areas where experience has shown that major emissions are likely to occur are indicated.

Monitoring for Vinyl Chloride Exposure

The principal goals of a vinyl chloride monitoring program are to identify and prevent excessive exposures, whether in the workplace or the general environment.

The selection of times and places where monitoring should be undertaken and the selection of appropriate methods require knowledge of the processes in operation as well as the statistical, physical, and chemical bases of the monitoring method. The aim of the monitoring program should be clearly defined, and assistance should be sought from process

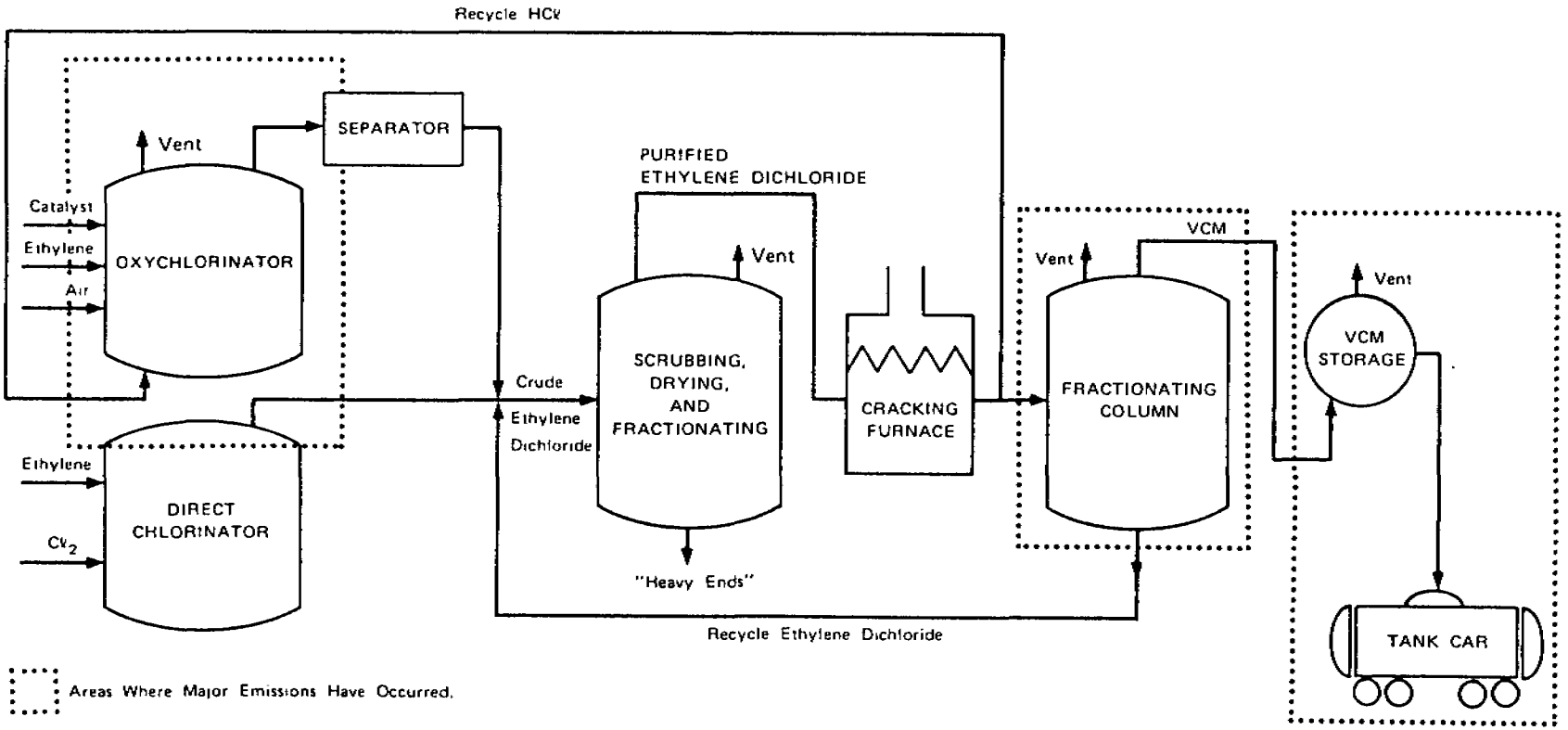


FIGURE 5 SIMPLIFIED "TYPICAL" PROCESS FOR PRODUCTION OF VINYL CHLORIDE MONOMER (VCM)

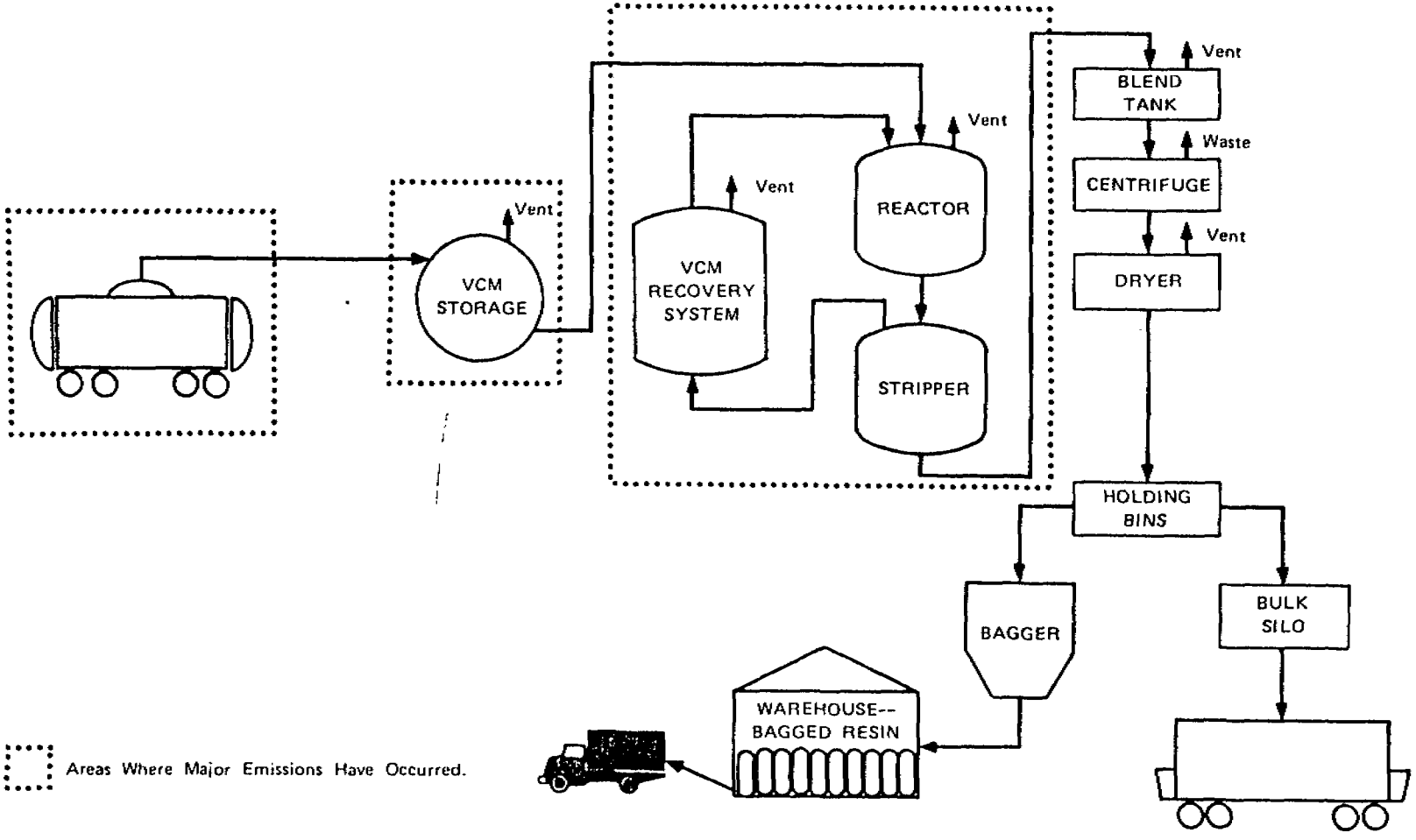


FIGURE 6 SIMPLIFIED "TYPICAL" POLYMERIZATION PROCESS

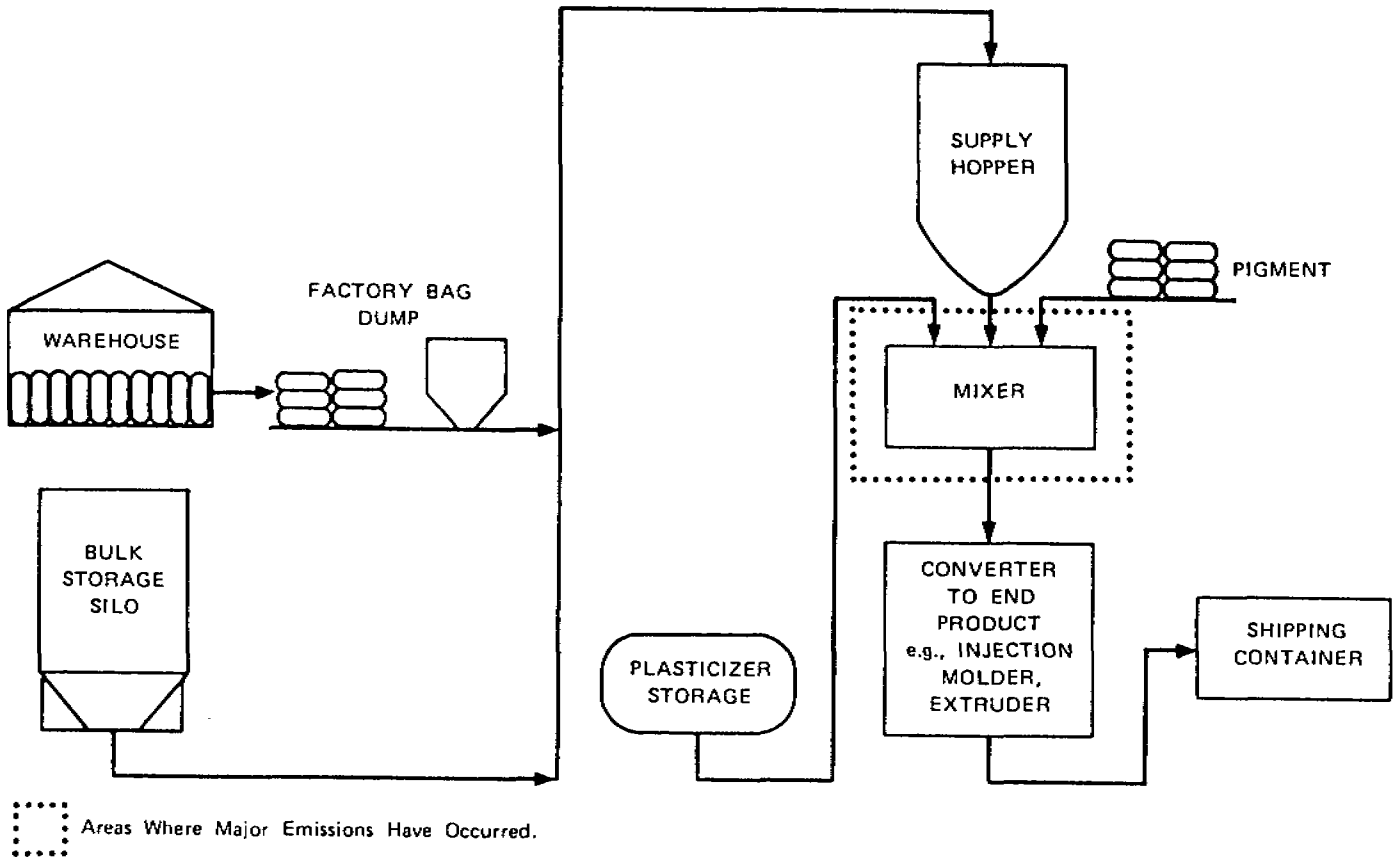


FIGURE 7 SIMPLIFIED "TYPICAL" FABRICATION OPERATION

engineers and regulatory agency representatives in the design of the program. (Agencies that can provide assistance are listed in Appendix E.)

Additional goals of the monitoring program may include evaluation of effectiveness of control methods and definition of exposure for epidemiologic studies. Several chapters in the NIOSH training syllabus, "The Industrial Environment: Its Evaluation and Control,"[1] offer valuable guidance on the general monitoring principles applicable to both occupational and general environments. Determination of compliance with appropriate standards may be accomplished through application of methods found in two other NIOSH publications: "Handbook of Statistical Tests for Evaluating Employee Exposure to Air Contaminants"[2] and "Statistical Methods for the Determination of Noncompliance with Occupational Health Standards."[3] More generally useful statistical techniques are available in the National Bureau of Standards publication "Experimental Statistics."[4]

The Occupational Environment

Two major vinyl chloride monitoring strategies have been applied successfully in programs for control of the occupational environment. Strategy One is the most common and involves collection of samples followed by a separate analytical procedure. Strategy Two relies upon the use of direct reading or continuous monitoring instrumentation, or both.

Strategy One involves the application of one of two different sample collection techniques. The first of these techniques is contained in the NIOSH Manual of Analytical Methods, 1974[7] and is referenced in the OSHA vinyl chloride exposure standard of 1974 as meeting the OSHA-required standards for accuracy. In this method, a known volume of air from the worker's breathing zone is drawn through an activated charcoal tube with the aid of a small pump. The trapped vinyl chloride is then desorbed with carbon disulfide, and an aliquot is injected into a gas chromatograph for analysis. Several modifications of this method have been published.[5,6] In the second sample collection technique, the breathing zone air is collected in an aluminized gas sampling bag without concentration[6] and then analyzed directly by gas chromatography. With either sampling method, sample collection should be planned so that evaluation of both Time Weighted Average (TWA) and 15-minute exposures is possible. The recent development of a monitoring "badge" which does not require the use of a pump for sample collection has been reported.[8]

The advantages of Strategy One are that it may be readily adapted to monitoring individual exposures and that airborne concentrations may be determined with considerable accuracy. Its disadvantages include: (1) the requirement for a skilled chemical analyst; (2) the lag time between sampling and receipt of analytical results (potentially resulting in a delay in application of control measures); and (3) measurements are necessarily intermittent and thus may not identify peak concentration periods.

The direct reading instruments with which to implement monitoring Strategy Two may be either portable, or may be fixed (nonportable) with probes that extend to work areas to be sampled. Continuous monitoring instruments are nearly always fixed with information output recorded in permanent records; the only external indication of activity being an alarm system set to respond to a predetermined vinyl chloride air concentration.

The advantage of direct reading and continuous monitoring instrumentation is that an immediate indication of airborne concentration is provided. In addition, continuous monitoring systems have the capability to monitor many areas in closely successive time periods, utilizing a "switching" network of valves and sampling probes, thus providing the flexibility to immediately modify processes when vinyl chloride concentrations are found to be excessive. The disadvantages of this approach include high initial cost, rigidity of installation where systems are fixed, and difficulty in relating measured concentrations to individual exposures.

An effective monitoring system should utilize the best of both strategies to maximize benefits. For example, personal samples may be taken in the breathing zones of workers, while a fixed, continuous monitoring system may be utilized to warn of leaks and to call immediate attention to areas where concentrations are excessive, and hand-carried, direct reading monitors are used to survey plant or process emissions. Table 13 summarizes the advantages and disadvantages of these two monitoring strategies.

Even the most vigorous efforts to reduce vinyl chloride air concentrations to desired levels may not always be fully successful. It is therefore advisable to warn employees when concentrations rise above recommended limits. Continuous monitoring of in-plant air should be carried on with a detecting device equipped to trigger a preliminary alarm at 1.0 ppm, and a warning to immediately don respirators at 5.0 ppm. A properly calibrated, nonspecific flame ionization detector or an infrared spectrophotometer is most often used for this purpose. The usual background level of other hydrocarbons can be partially accounted for in the calibration so that the results will generally reflect only vinyl chloride concentrations. More sophisticated systems using gas chromatographic columns specific for vinyl chloride are preferable and are now readily available.

Several systems, using either flame ionization or infrared are now available from commercial vendors. Most installations are designed specifically for the characteristics of the facility where they are to be installed. An example of the successful application of this concept is the system at Pantasote Corporation in New Jersey, which is described in Appendix C. In addition to its use in protection of individual workers, such a system may have inlets situated at points where emissions may be expected and thus aid in early detection and correction of leaks.

Table 13

ADVANTAGES AND DISADVANTAGES OF MAJOR MONITORING STRATEGIES

	Advantages	Disadvantages
Sample collection followed by separate analytical procedure	Airborne concentrations may be determined with considerable accuracy May be adapted to monitoring individual exposures	Requires a skilled chemical analyst Time lag between sampling and reported results Difficult to obtain measurement of high peak concentrations
Direct reading instrumentation	Immediate indication of concentrations Flexibility to modify processes immediately when concentrations are found to be excessive	Imprecise and subject to interferences Maintenance and operational costs are high
Continuous monitoring	Capability to monitor many areas in closely successive time periods	High initial cost Rigidity of fixed-system installations Difficult to relate concentrations to individual exposures

Source: SRI

The General Environment

The EPA national emission standard for vinyl chloride contains a test method procedure to determine compliance. Samples of stack gas (or ambient air) are to be collected in Tedlar® or aluminized Mylar® bags. Water or waste sludge samples are to be collected in glass vials with Teflon® cap liners. Ambient air samples have also been successfully collected by drawing air through charcoal adsorbent tubes for a 24-hour period.[9]

The analysis of vinyl chloride content in PVC products or foods requires specialized solution techniques for sampling.[10] Ethanol and n-heptane are used as solvents to simulate the action of alcoholic and fatty foods, respectively.[11] These food categories have been shown to have the greatest potential for leaching vinyl chloride from rigid or semirigid PVC products. A recently reported method simplifies the food and polymer sampling procedure and provides an analytical method that is useful for the detection of vinyl chloride in foods with a minimum detectable concentration of 50 parts per billion (ppb).[12]

Analysis

All analytical methods currently acceptable to federal regulatory agencies are based on the use of gas-liquid chromatography with a flame ionization detector.

The OSHA standard requires that methods of sampling and analysis for vinyl chloride have a lower detectable limit of 0.25 ppm with precision at the 95% confidence level of ± 50%.[13] The EPA requires equivalence to the methods given in detail in its standard with no stated precision or sensitivity.[14] A skilled analytical chemist with modern laboratory facilities will be able to detect concentrations of a few ppb in air if adequate sampling methods are employed. The FDA-referenced method[10] is sensitive to 0.05 parts per million (ppm) vinyl chloride in foods and 1.0 ppm in PVC, while other methods permit detection of as little as 10 ppb in vinegars and alcoholic beverages.[15]

Control Measures for Emission Sources

Polymerization Plants

In PVC facilities, the primary sources of emissions requiring application of control procedures include:

- Opening and cleaning of reactor vessel.
- Discharge of relief valve.
- Piping joints.

- Stripping process equipment.

  Fugitive vinyl chloride sources--dryers, bulk storage, blenders, pumps, and valves.

Control will generally embody the use of some or all of the following measures.

Process or Procedure Change--Many of the measures that have been taken to reduce occupational exposures and emissions to the community have involved changes in the processes used in handling vinyl chloride and PVC. Some examples are:

- Using high-pressure water or steam to clean polymerization reactors remotely, rather than the old practice of sending workers into the reactors for manual cleaning.

- Lengthening the reaction time of polymerization so that the amount of monomer entrapped in the PVC is minimized.

- Inserting rupture disks upstream of relief valves to reduce leaks.

- Using exhaust hoods with filters in laboratories where samples of vinyl chloride are prepared for analysis.

- Using closed-loop vapor return systems at loading and transfer stations.

- Substituting closed gauges for open-end slip gauges to measure the contents of a tank car.

- Adding vacuum or steam stripping steps at the end of polymerization to reduce the vinyl chloride content of PVC.

Maintenance and Surveillance--Because vinyl chloride is colorless and odorless at hazardous concentrations and is under pressure at all stages of transfer, storage, and polymerization, a program of constant vigilance and maintenance of all possible sources of leakage must be carefully followed.

Local Exhaust Ventilation--A local exhaust ventilation system designed so that released vinyl chloride is immediately captured and removed from the ambient air will add to the effectiveness of any control system. This has been particularly effective in reducing emissions from tank car transfer operations. Ventilating systems may be designed

to actuate when excessive levels of vinyl chloride are detected at some point in the plant by a continuous monitoring system.

Gas Cleaning--All gases collected by the exhaust ventilation system or vented by relief valves must be cleansed so that no more than 10 ppm of vinyl chloride remains in the emission gases (dilution with additional air is not an acceptable method to reach the 10 ppm concentration). There are three types of emission controls that EPA considers to be technically and economically feasible for this purpose: activated carbon adsorbtion, solvent extraction, and incineration.

Conventional heat exchangers may be used as a preliminary step to reduce the vinyl chloride content of the gas stream, although they are not very efficient because of the high vapor pressure of the vinyl chloride monomer. Carbon adsorption and solvent extraction offer the potential advantage of recycling the collected monomers. There is a possibility of polymerization of the adsorbed monomer on the surface of the carbon, which might lead to rapid loss of adsorption capacity.

Although incineration is probably the most thoroughly tested and immediately feasible of the three types of emission control, it does have economic disadvantages including: loss of monomer, need for fuel to support combustion, and rapid corrosion of incinerator components from HCl formed during combustion.

Too few data are available to completely assess the actual or potential effectiveness of any of these systems in any operation, and it is suggested that the regional EPA office be consulted for applicable standards and recent assessment of currently available technology.

Other Measures--The control of monomer content in PVC should include treatment of waste sludge discharged from incomplete polymerization reactions. Where the vinyl chloride content cannot be effectively reduced, the waste should be disposed of in such a way that human contact is minimized, with attention given to landfill operators, refuse collectors, and others who may be exposed to the waste. The potential for release of vinyl chloride during inefficient combustion of PVC at low temperatures must be recognized if incineration is chosen as the means of disposal.[16]

Significant quantities of vinyl chloride may be released from relief valves if a reaction does not proceed properly and excessive pressure builds up in the reactor.[17] There is a significant risk of explosion in this event, and release to a larger vessel or to the atmosphere is essential. Appropriate piping and gas cleaning capability must be provided in anticipation for this emergency so that the discharged gas stream is reduced to 10 ppm.

Preparation for recharging the reactor vessel after completion of polymerization must employ several emission control procedures.

The reactor concentration of vinyl chloride should be reduced to 50 ppm or less before opening, and the exhaust gases must be vented through a duct or stack to a cleansing operation before release to the outside atmosphere. The actual cleansing of the reactor vessel should be done with a jet of high pressure water or steam.

The requirements of the OSHA standard[13] with regard to labeling of vinyl chloride containers, and control of access to and posting of regulated areas must be followed. Regulated areas must also be isolated physically by barriers and equipped with general ventilation, so that the potential for human exposure to vinyl chloride is minimized.

Vinyl Chloride Production Plants

Many of the measures listed above for PVC plants are also applicable to plants that produce vinyl chloride monomer. Special attention must be given to the opportunities for exposure in the quality control laboratory and during transfer and loading operations. Effective control will often entail a combination of procedural changes and personal protection. As in the PVC plants, a program of surveillance and maintenance to discover and repair leaks is one of the most effective control measures. An integrated program for control in a vinyl chloride production plant has been developed by PPG Industries.[18] This program has elements that are applicable to any use of vinyl chloride monomer.

PVC Fabrication Plants

The most effective method for reducing occupational exposures and environmental emissions from PVC plants, as discussed above, is to reduce the monomer content in the PVC before delivery to the fabrication plant. If this is not done (for instance where old resin stocks are used), then the level of control necessary will depend on the fabricating process under consideration. Isolation of blending processes with provision of local exhaust ventilation and appropriate treatment of waste gases may be the most useful general procedures.

General Population Exposure

The most important measure to be taken to reduce general population exposures, beyond control of emission sources, is to reduce monomer content in fabricated PVC articles. This measure will reduce the potential for the very small, although measurable, exposures that have occurred in the past as a result of migration of vinyl chloride from PVC food containers or water pipe.

For many of the products in which PVC is now used, substitutes exist. Although these substitutes may not fully share the desirable characteristics of PVC, such as chemical inertness and nonflammability, they can be substituted in some applications where these special characteristics of PVC are not essential.

The potential for general population exposures to vinyl chloride will be significantly reduced as the monomer content of PVC articles is reduced. Substitution should, of course, be considered cautiously, since it is an expensive and potentially disruptive process when widely used consumer articles are considered. Some possible substitutes for PVC in various uses are shown in Table 14.

Personal Protective Measures

Even with the installation of appropriate engineering control measures, personal protective devices such as respirators, protective clothing, safety glasses, and face shields should be provided and worn as required by the nature of the operation or condition.

The OSHA standard establish requirements for use of personal respiratory protective devices, and NIOSH has set criteria and standards for their performance. In general, these devices should not be used as a substitute for the engineering control measures, but there are occasions when their use is essential. This is particularly true for operations such as loading, reactor cleaning, quality control sampling, and maintenance, which may involve exposures to high vinyl chloride levels.

Additional Sources of Information

There are local and regional offices of the several federal agencies with specific regulatory or advisory responsibility for aspects of the engineering control of toxic chemicals. They should be consulted for the most recent regulations governing use and control of vinyl chloride. These agencies include:

- Occupational Safety and Health Administration (OSHA)
- National Institute for Occupational Safety and Health (NIOSH)
- Environmental Protection Agency (EPA)
- Food and Drug Administration (FDA).

Researchers in these agencies are deeply involved in evaluation of improved methods of control and monitoring, and often can provide guidance on the most recent acceptable methods. (See Appendix C.)

Medical Surveillance

As has been emphasized earlier in this monograph, medical surveillance of vinyl chloride workers is a developing picture, characterized by intense investigation and continuing methodological refinement.[19-25] For this reason, it would be misleading to attempt to lay out in detail here

Table 14

POSSIBLE PVC SUBSTITUTES

Market	PVC Resin Usage* Millions of Kilograms 1973	1974	Possible Substitutes
Apparel			
Baby pants	12	9	Rubber
Footwear	66	60	Rubber
Outerwear	31	25	Other synthetic fibers
Building and construction			
Extruded foam moldings	26	22	Wood
Flooring	202	156	Wood
Lighting	5	6	Glass, styrene
Panels and siding	39	44	Wood, polyester
Pipe and conduit	520	526	Steel, ABS, polyethylene
Pipe fittings	41	31	Steel, ABS, polyethylene
Rainwater systems, soffits, fascias	16	14	Wood, aluminum
Swimming pool liners	18	18	Rubber
Weatherstripping	16	15	Rubber, urethane
Window, other profiles	26	22	Wood, steel, aluminum
Electrical			
Wire and cable	188	158	Rubber, polyethylene
Home Furnishing			
Appliances	20	21	Other plastics in some applications
Furniture	145	124	Wood, melamine
Wall coverings & wood surfacing films	54	58	Paper, melamine
Housewares	--†	48	Styrene, rubber
Packaging			
Blow molded bottles	39	26	Glass, cans
Closure liners and gaskets	9	10	Rubber
Coatings	9	9	None
Film	59	56	Acrylics, styrene
Sheet	35	35	Polyethylene, nylon polyester
Recreation			
Records	66	70	None
Sporting goods	25	27	Rubber, leather
Toys	38	35	None
Transportation			
Auto mats	18	18	Rubber
Auto tops	15	12	Steel
Upholstery and seat covers	83	80	Nylon, polyesters
Miscellaneous			
Agriculture (including pipe)	66	70	Aluminum, polyethylene
Credit cards	8	9	None
Garden hose	18	15	Rubber, nylon
Laminates	23	20	None
Medical tubing	23	22	None
Novelties	7	7	None
Stationery supplies	18	19	Polyester
Tools and hardware	8	9	None
Export	66	135	None
Other	93	100	None
Total	2,151	2,141	

*Modern Plastics, January 1975, p. 51, and January 1976, p. 43.

†Included in "Other."

a comprehensive medical surveillance program which the occupational health physician could accept and apply without further enquiry. Rather, the purpose of this section is to take note of the federal vinyl chloride standards as promulgated by OSHA in 1974 and to familiarize the physician with some of the medical surveillance activities of a leading vinyl chloride research program now underway.

An imbalance in the content of this section will immediately become apparent to the reader. The recent and dramatic discovery, by Creech, of the association between exposure to vinyl chloride and the appearance of a rare tumor of the liver--angiosarcoma--has tended to focus attention on that organ as the major target of vinyl chloride toxicity. The OSHA standards clearly emphasize this concern, as does the research first reported from Louisville and cited here. However, as noted in Chapters III and IV of this monograph, both carcinogenic and noncarcinogenic effects of vinyl chloride are often expressed in organs other than the liver, and medical surveillance programs should be fully reflective of this recognized potential for widespread injury.

Overview

Employees engaged in work with vinyl chloride should be participants in a well-organized occupational health program, as should employees exposed to any hazardous workplace environment. This program should include a systematic program of medical surveillance designed specifically to detect early evidence of health-related response to vinyl chloride exposure. The nature of this medical surveillance program may properly vary to some extent according to the potential for exposure, and it is here that the application of current technical knowledge and sound medical judgment is of paramount importance.

Because liver dysfunction is known to be an early response to vinyl chloride, prompt detection of alterations in liver function coupled with removal of the employee from exposure has become the principal goal of medical surveillance. Thus, highest priority has been given to efforts to formulate a screening regimen capable of detecting vinyl-chloride-induced liver dysfunction with maximum sensitivity.

OSHA Requirements

In October 1974, OSHA published detailed standards for the control of occupational exposure to vinyl chloride.[26] These standards established requirements for medical surveillance, which, as defined by these standards, includes a general physical examination, performed with special attention to detecting abnormalities of the liver, spleen, kidneys, skin, connective tissues, and pulmonary system. A medical history is to be recorded, including information on alcohol intake, past history of hepatitis, past exposure to potential liver toxins, and past history of blood transfusions and hospitalizations. A work history is also to be recorded.

In addition, the following biochemical tests are to be done: total bilirubin, alkaline phosphatase, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), and gamma glutamyl transpeptidase (GGTP). If abnormalities are found, the standards require that these tests be repeated. If tests remain abnormal, consideration must be given to withdrawal of the employee from contact with vinyl chloride while a more comprehensive medical examination is made. According to these regulations, examinations should be performed every six months on individuals who have been employed in vinyl chloride or PVC manufacturing for ten years or longer and annually for all other employees.

The standards require the employer to obtain a statement of each employee's suitability for continued exposure to vinyl chloride from the examining physician promptly after examination, and to provide a copy of the statement to the employee. Withdrawal of the employee from possible contact with vinyl chloride is required if continued exposure would materially impair health.

A Medical Surveillance Program--Louisville, Ky.

Tamburro and his associates have reported their experience,[22-24] in developing a medical surveillance program for vinyl chloride workers in Louisville, Kentucky. Each participant in the program receives an initial examination and periodic examinations:

- The initial examination of each participant in the program consists of a personal and occupational history, a general physical examination, a chest roentgenogram, and the following laboratory determinations: complete blood count (CBC), urinalysis, SGOT, SGPT, total bilirubin, alkaline phosphatase, and GGTP. Tamburro and his colleagues feel that the initial examination should also include a functional test of liver clearance, of which there are a number available. Among these are indocyanine green (ICG), sulfobromophthalein (BSP) and bile acid, radiobilirubin, or aminoacid clearance.[24]

- Periodic examinations are done at least annually on all participants and at six-month intervals on individuals exposed to vinyl chloride at the OSHA "action level" (an airborne concentration of vinyl chloride of 0.5 ppm averaged over an eight-hour work day)[26] and at six-month intervals on certain employees known to have had high exposures in the past. This periodic examination includes a chest roentgenogram and laboratory determinations identical to those performed during the initial examination. In addition, a radioisotopic liver--spleen scintiscan is included to detect possible deformities in the liver, spleen, or both. The results of

each participant's examination are assessed by the responsible physician and a decision is made with regard to the need for repeat or additional tests or special procedures.

A similar medical surveillance program has been described by investigators in the United Kingdom.[25]

Tamburro and his associates also have given considerable attention to an aspect of medical surveillance that has historically received little or no consideration--the creation of an active data bank. In this computerized bank are all available clinical, biomedical, radiological, and physiological study data for each surveillance program participant. Work is under way to add various estimates of intensity and duration of exposure such as job classification code, employee work history, and chemical exposure history.[27]

With these data, an index of exposure to any of several chemicals can be calculated by which the average and total exposure of any individual or group can be realistically estimated. Computerized printouts of biomedical and exposure information have been designed and utilized for a variety of purposes such as communication of test results to the private physicians of individual employees. The data bank also includes provisions for an early warning system and epidemiological programs.[27]

Educational Control

It is not sufficient for purposes of control that a carcinogenic substance is identified, that standards are promulgated regulating its manufacture and use, and that control measures are taken to eliminate or limit the physical contact between the substance and humans. It is also necessary that methods be developed to educate and thereby modify the behavior of those persons who interface with hazardous substances. However, given the paucity of information on the effectiveness of educational programs dealing with hazardous substances, it is difficult to determine whether behavioral modification would be best accomplished through use of formalized educational programs or of more psychologically oriented techniques.

A succinct definition of the goal of public education regarding cancer has been given elsewhere--i.e., to:

- Educate the public about treatable forms of the disease
- Motivate individuals, especially those at high risk, to participate in screening programs designed to detect early forms of cancer
- Persuade those with recognizable signs or symptoms of ill health to seek early medical advice.[28]

This goal is consonant with the goals of the National Cancer Institute's National Cancer Plan, and it is in this context that the educational aspects of a control program for vinyl chloride-induced disease are presented here.

Discussed in the following paragraphs on educational control are: OSHA regulations and education; the understanding of "risk;" results of an important National Academy of Sciences study of cancer education; and, finally, some information resources that could prove helpful to anyone concerned with the educational aspects of cancer control.

OSHA Regulations--Education

In those situations where a chemical substance is regulated by government agencies as a carcinogen, an educational process has already begun through promulgation of regulatory controls. Moreover, the 1974 OSHA regulatory standard for vinyl chloride mandates employee training concerning the health hazards of exposure to the substance including, specifically, the carcinogenic hazard.

Employers subject to this regulation must ensure that employees receive initial and annual training concerning which operations could result in excessive exposure to vinyl chloride; the purpose, proper use, and limitations of respiratory protective devices; the nature of a required environmental and personal monitoring program; the purpose and description of a required medical surveillance program; and conditions that could result in release of vinyl chloride. In addition, the employer is required to provide OSHA a copy of all training materials on request. It could be assumed, then, that the populations at highest risk of vinyl chloride-induced disease are receiving the benefits of a mandatory education/training program through their employers.

Unfortunately, the beneficial effects of these mandatory training programs on the behavior of the employee have not been evaluated. How well such employees have grasped the significance of relative and individual risk should be a matter of great concern to those responsible for worker education.

Understanding "Risk"

Of the numerous aspects of cancer about which the worker should be informed--as enumerated in the following section of this chapter (see particularly Table 16)--one of the more important and complex is how information should be evaluated and interpreted in terms of the risk that is present in the workplace.

In the case of vinyl chloride-related angiosarcoma of the liver, a conceptual understanding of risk by the employee is complicated by the fact that there is an extremely high relative risk, which may approach

200 to 400 times that of unexposed workers, but a very low actual or individual, risk. The average annual incidence of liver angiosarcoma in U.S. males is approximately 0.02/100,000. Given this incidence rate, the expected annual number of new cases of the disease among 7,000 vinyl-chloride-exposed workers--even if all were at highest relative risk (i.e., 400 times that expected)--would be less than 1.

It could be assumed that the recent reduction in exposure concentrations effected by the OSHA regulation would have the effect of greatly reducing the exceedingly high relative risk. But this assumption, in turn, would pertain principally to only those new workers who have benefited from the 500-fold reduction in the permissible exposure concentration. These considerations can be extremely confusing to workers who simply wish to understand whether they risk insidious health hazards during the performance of their jobs.*

A National Academy of Sciences Study

Working under an interagency agreement with the National Cancer Institute, OSHA funded a program in 1975 to determine how best to inform workers concerning carcinogenic hazards in the workplace. Part of this program included a contract with the National Academy of Sciences (NAS) to form a select Committee on Public Information in the Prevention of Occupational Cancer. The work of this Committee was completed during 1977, and a report entitled "Informing Workers and Employers about Occupational Cancer" was published by the Academy.[29]

This report is of utmost importance to this monograph because it brings together a select group of preeminent individuals possessing special expertise in fields of cancer biology, environmental medicine, communication, journalism, occupational medicine, epidemiology, law, and behavioral sciences. At a time when the methods used to inform workers of carcinogenic hazards are largely derived on a subjective basis, the recommendations of this Committee must be considered as the most appropriate available. Hence, pertinent sections of the Committee's report are reviewed here.

Legal and Ethical Bases for Providing Information

The NAS Committee concluded that both public law and published standards clearly establish a requirement for training the employee with regard to the hazards of substances with which they work, and that current laws "clearly contain a legal basis" for providing public information on the prevention of occupational cancer.

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*None of this is to suggest, however, that vinyl chloride should not be avoided, if possible.

From the standpoint of ethics, the Committee agreed that there is a fundamental ethical principle that may best be called the "right to know." The Committee said it believed that the readiness with which the Committee members accepted this principle "parallels the increasing concern of society as a whole with the individual's right of self-determination."

The Committee stated further that it believes it is "unethical and unrealistic" to (1) assume that the right to know requires employers to inform workers about only the few chemicals that nearly everyone agrees are human carcinogens and (2) argue that the right to know requires only that summary information concerning the exposure of workers to suspected carcinogens "be made available for inspection"--it is necessary for government agencies and employers to make reasonable efforts to ensure that workers comprehend the relevant material as well. The ethical test for whether particular information must be divulged, the Committee said, is whether it is relevant to the employee's decision. In this light, the Committee recommended that, in spite of the anxiety that is frequently associated with cancer hazards, information essential to the understanding of risk by the whole group of employees should not be withheld on the chance that it might cause anxiety in some.

Target Audience and Information to be Conveyed

The Committee felt that the most readily indentifiable target audience would be the unionized employee, with the most difficult target audiences being farmers and others engaged in agriculturally related activities such as migrant workers. Families of workers were also indicated as prime target audiences, and the Committee recommended that programs be designed to reach such families.

The Committee recommended that information concerning carcinogenic hazards in the workplace should:

- Provide sufficient awareness to permit job applicants to decide whether they would want to work in the face of the hazard or to allow present employees to decide whether they wish to continue work.
- Tell the employee how to take steps to minimize his own exposure.
- Be sufficient to permit the worker to assist to the limit of his capability in monitoring and improving the environment of his workplace.

The Committee went on to recommend specific items of information that should be presented to employees and employers with regard to both (1) the hazard of occupational cancer in general, and (2) a specific substance once it has been determined that it is, in fact, a carcinogenic. These recommendations are presented in Tables 15 and 16.

Table 15

WHAT THE EMPLOYEE AND EMPLOYER SHOULD KNOW
ABOUT OCCUPATIONAL CANCER IN GENERAL

1. Many carcinogenic substances can be absorbed by man without any warning signals such as coughing, burning, or nausea.

2. If permanent damage is done to a cell by a carcinogenic agent, the defect is passed on to daughter cells in the presence of multiplicaton; the effects of repeated exposure can therefore be additive. Moreover, some agents are not readily eliminated from the body so their concentrations increase with repeated exposure.

3. There is usually a latent period of from 5 to 30 or more years between the first absorption of the carcinogen and the appearance of any sign of the disease.

4. Although there is still some debate within scientific circles, for all practical purposes there is o dose of a carcinogen below which one can say that there is absolutely no risk of it causing cancer. Nevertheless, decreasing the exposure decreases the risk and increasing it increases the risk.

5. The hazard of a carcinogen is in some instances multiplied if if is absorbed in conjunction with other substances, such as cigarette smoke.

6. Some carcinogens can be inadvertently transferred from the work-place to the home in significant quantities.

7. An indication that a substance may produce cancer in man is frequently found in experiments on laboratory animals and cells. When an agent is demonstrated in controlled experiments to produce cancer in animals, that agent should be regarded as possibly carcinogenic in man.

8. In some instances, benign tumors develop into cancer and, in many instances, agents that produce benign tumors increase the risk of cancer.

Source: National Academy of Science (Chapter reference 29).

Table 16

WHAT THE EMPLOYEE AND EMPLOYER SHOULD KNOW
ABOUT A SPECIFIC CARCINOGENIC SUBSTANCE

1. The identification of the substance in question by the name used in the workplace and, where possible, by generic name.

2. Data on the carcinogenicity of the substance including evidence from human epidemiologic studies, animal studies, and other valid techniques.

3. An interpretation of the risks in the workplace implied by the above data.

4. Description of the disease in man, where applicable.

5. The route of exposure in the workplace.

6. Required and recommended measures to reduce exposure, including contaminant and disposal measures, monitoring procedures, and individual protective measures.

Source: National Academy of Sciences (Chapter reference 29).

The Committee recommended that OSHA should take necessary steps to ensure that carcinogenic substances were readily identified, that lists of commercial enterprises that use such substances are kept up to date, and that, in order to enhance the receptiveness of the worker to a specific program: 1) where appropriate, the immediate community should also be informed through local health and other authorities of the hazard; and 2) the fundamental concept of hazardous substances in the environment should be introduced in primary and secondary school curricula.

The Decision to Inform

Because the decision to inform those in affected workplaces of a chemical's hazard involves a commitment of substantial resources, the Committee considered it essential that a single national source, such as the Department of Health, Education, and Welfare, be charged with making that decision. The Committee noted that it should be, to the extent possible, a source that is credible to both management and labor--hence, the Committee said, one not involved in the regulatory process. It was also noted that the decision source should be close to the realities of the workplace, so that it could make an appropriate assessment of risk--at least, the source should announce its decision in a form that could be translated to a specific occupational situation.

Information Sources and Manner of Presentation

The Committee noted that workers are exposed to many kinds of information about the workplace from a number of different sources that are perceived by the worker as having various degrees of expertise and credibility. Included in these information sources are "official" ones such as OSHA, and "unofficial" ones such as, industrial hygienists, family physicians, shop stewards, management, local newspaper columnists, family, and fellow workers.

The Committee stated that, notwithstanding the official sources, it may be that unofficial sources are more influential in actually modifying the worker's decision and behavior with respect to the workplace. The Committee said, however, that it could not identify any data with respect to the effectiveness or credibility of various information sources and that, therefore, multiple sources and institutions should be used to transmit messages to the worker.

It was noted by the Committee that OSHA has responsibilities for the promulgation of standards concerning the appraisal of employees and that NIOSH, in its Criteria Documents, must address this same area. However, the Committee emphasized that there appeared to be no mechanism at present by which the distribution of appropriate documents to all places of employment could be ensured. In this regard, it stated that management and labor both play a role in presenting information to the worker and to the relevant audiences.

Concerning the manner of presenting information to the worker, several recommendations were made by the Committee. To ensure effective communication with the worker, sources beyond those charged with statutory responsibility should be involved--consideration should be given to labor unions; extension services of land-grant colleges and other adult educarion services; state, county, and municipal health departments; and community oriented public service health groups. It was emphasized that, to be effective, information provided by these groups must be essentially the same. And in this regard, the Committee recommended that a central national source of such information be established, adding that the output of this source should be credible to all who use it as a common base.

Although the Committee realized that programs will differ, it said that it was clear that:

- The information program should be initiated at the pre-employment interview and presented recurrently, regardless of the stability of the work force.

- Presentation of the information should take into account the nature of the specific work force--its size, structure, degree of training, etc.

It was recommended that the media used to carry specific messages be tailored to the audience, and that a variety of media and presentation methods might be used. The Committee suggested tht OSHA devote some of its resources to the support of studies that would identify the best presentation techniques for selected types of workplaces.

Implications and On-going Programs

It should be recognized that successful education programs must utilize existing resources of health education and behavior modification including, for example, the American Cancer Society, the national and community health agencies, and university resources located in the community. In particular, the cancer prevention and control programs of the National Cancer Institute's comprehensive cancer centers should be looked to as sources of specialized knowledge in regional collaborative efforts.

A variety of published iformation is available concerning benavioral and motivational factors as they relate to health education. Some selected publications are included in the References for this chapter.

Unfortunately, there is, at present, a dearth of models that relate practical experiences with worker populations exposed to carcinogenic substances to the behavioral and motivational factors that influence these populations towards positive health behavior. Such information is just now being developed in a few programs such as the cancer prevention program at the University of Louisville for vinyl chloride workers. The components

of this program with regard to the medical aspects were reviewed earlier in this chapter; however, an educational component for the program is being developed, and the results of this endeavor will be useful to individuals concerned with cancer control for vinyl chloride workers (as well as for workers exposed to other carcinogenic substances).

The approach to education developed by the Louisville center involves the following areas:

- General information for:
  - All employees
  - Employee's families
  - Former employees.
- Specific educational information for:
  - Families of deceased workers.
  - Individuals at high risk.
  - Employees who have been retired or transferred because of known occupationally-related disorders.
  - Employees with specific nonoccupationally-related health problems.
- Educational programs on self-destructive life styles concerning smoking and alcohol.
- Counseling and rehabilitative programs.
- Professional education programs for:
  - Private and community physicians
  - Industrial nurses and paramedical personnel
  - Health care personnel
  - Internal program personnel.
- Additional services:
  - Computer printouts of information on file for each employee, made available to the employee's physician.

- Development of specialized training brochures for workers describing the medical surveillance program.

- Implementation of specialized meetings for:

  General plant information.

  Employees at high risk who have been removed from the main plant and given other jobs.

  The families of workers.

- Development of professional and community education programs for information exchange involving air pollution boards, industrial medical personnel, the National Academy of Sciences, and the National Research Council.

- Organization of a seminar workshop entitled, "Detection and Prevention of Industrial Chemical Carcinogenesis--Vinyl Chloride Mode."

- Fostering development of community educational programs to assist both business and the general community to understand the cancer control program's goals and objectives.

- Development of a joint program with the B. F. Goodrich Safety Division to motivate worker compliance with safety measures requiring the use of protective respirators.

- Establishment of an advisory board composed of representatives from the union, business, religious, legal, medical and scientific communities. The purpose of this board is to advise and evaluate the education of industry and the general public concerning environmental carcinogenic hazards.

Another major resource in the formulation and implementation of an educational program for the control of cancer caused by vinyl chloride exposure is the Cancer Centers program of the National Cancer Institute (NCI). Under the terms of this program, the NCI funds centers of cancer research and development throughout the United States that meet certain criteria set forth by the agency. The NCI intends that these centers be geographically well distributed to maximize the benefits of their programs to the entire U.S. population.

The increasing interest of other federal agencies in the area of education is being manifested. In 1977, the National Institute for Occupational Safety and Health (NIOSH) solicited proposals for a study to be entitled "Behavioral Procedures for Reducing Worker Exposure to Carcinogens." The stated purpose of the proposal was to develop and test an employee motivation program that would ensure healthful work practices which, in turn, could reduce exposure to carcinogenic agents.

Chapter VI

A VINYL CHLORIDE CONTROL PROGRAM FOR A TOWN OF 25,000--A SCENARIO

The setting is a vinyl chloride polymerization (PVC) plant located in a town of about 25,000 population. Although the plant was originally built in a very sparsely populated area at the edge of town, the town's growth has resulted in construction of homes near the plant fence line.

Over the years, the plant had never been considered a community nuisance, only occasionally emitting visible smoke or vapor. At times, especially on hot, humid days, detectable though not particularly unpleasant odors were noted by drivers passing the plant.

The plant uses large quantities of water from a moderate-size river that flows past the town and, during the first couple of decades of operation, plant effluents passed without treatment directly into the town's sewage tretment system (which, in turn, discharges effluents into the river). About ten years ago, the PVC plant constructed a small treatment facility, primarily to remove solids from the effluent prior to its release into the town's system.

The community now acknowledges that the plant, during several decades of operation, has released vinyl chloride gas into the general environment in quantities not precisely known but believed to be significant. The community also acknowledges that the plant's management has recently acted to minimize this discharge, but there are some cynical citizens who feel that scant attention has been paid to this problem other than to avoid a loss of revenue by loss of a useful raw material.

At the same time, however, the PVC plant has always been well-managed and, historically, has been considered by the community to be a good place to work. Accordingly, the labor turnover rate of about 10% a year is somewhat less than average. The plant workforce has grown from the original 75, of which between 50 and 60 are still employed, to a workforce of about 150. The workers are unionized, with relatively powerful leadership capable of effective negotiations with the management.

The town boasts three hospitals and a modern public health clinic (all of which also serve the surrounding area). There is a local office of the American Cancer Society and the County Medical Society, the latter having appointed an Environmental Health Committee several years ago. The County Health Department, which receives little help from the chronically underbudgeted state health department, is a source of some pride to the community due to its recognized competence in most fields of public health. There are a number of citizen's groups and social organizations, many quite active and some concerned with questions of environmental protection.

There is a small college (without a medical center). The media is represented by, in the town itself, a daily and a weekly newspaper, four radio stations, and a television station. The town and surrounding community are also served by metropolitan newspaper, radio, and TV.

The text below outlines generally what was done by community organizations to create a comprehensive vinyl chloride control program following the report in 1974 that forcefully linked vinyl chloride exposure with angiosarcoma of the liver.

The County Medical Society

The County Medical Society's Environmental Health Committee, recognizing the need for close communication between groups that would be involved in vinyl chloride cancer control, took the initiative to form a special Vinyl Chloride Advisory Committee. The committee's members include:

- A health educator from the college.
- An industrial hygienist from the County Health Department.
- An oncologist from a leading hospital in the area.
- The PVC plant manager.
- Representatives of the union, the American Cancer Society (ACS), and the Kiwanis Club (one of several citizen's groups particularly concerned with environmental health).

This advisory group meets monthly for discussion and to coordinate and assist activities, and has been quite effective in reducing duplication of effort and encouraging cooperation. (Such a committee might have been created by any organization--e.g., the plant itself, the health department, the local college, or the American Cancer Society chapter.)

One of the Advisory Committee's first acts was to set in motion the development of a formalized education program directed to workers at the PVC plant (discussed subsequently under "Plant Management"). Meanwhile, the Medical Society, after educating local physicians on the potential hazards of exposure to vinyl chloride, encouraged them to obtain occupational histories from their patients in order to identify those at greater-than-average health risk and to record any unusual clinical findings. Also, they were encouraged to include in their diagnostic workups those tests most helpful in identifying vinyl chloride injury.

The Medical Society also took it upon itself to communicate with those scientific investigators who are on the forefront of diagnostic investigation into vinyl chloride injury and transmitted their findings to the community physician. In this activity, the Society drew upon the

resources of the Cancer Information Service (CIS), a service affiliated with the Comprehensive Cancer Centers recognized by the National Cancer Institute (see Figure 8) and with the American Cancer Society. Working with the CIS by telephone, the Society has been receiving up-to-date information on research on the detection, treatment, and prevention of cancer and on the rehabilitation of afflicted patients. A listing of the CIS offices is given in Table 17. Another helpful resource to the Medical Society was the group at the University of Louisville in Kentucky that has established, with support from the NCI, an extensive medical surveillance and screening program for the workers at the B. F. Goodrich plant in Louisville.

Other federal agencies concerned with cases of possible vinyl-chloride-related disease--National Institute for Occupational Safety and Health (NIOSH), Food and Drug Administration (FDA), Occupational Safety and Health Administration (OSHA)--were referred to by the Medical Society. (See Appendix C for location of these agencies.)

County Health Department

The County Health Department began to monitor ambient air levels of vinyl chloride in the community, with emphasis, of course, on monitoring near the plant site. Both ambient air and stack emissions are being tested and recorded. Levels of vinyl chloride in the waste water effluents from the plant are monitored systematically, as is the solid waste that is deposited in community waste disposal areas. Working with information obtained fom the district Environmental Protection Agency and Food and Drug Administration offices, the Health Department also monitors food and drinking water to help ensure that regulations are being enforced.

Coordinating with the other members of the Vinyl Chloride Advisory Committee, the Health Department issues news releases--carefully prepared so as not to cause undue alarm--to the local and regional media. The news releases are a major component of the program to convey information about vinyl chloride to the general public. The health hazard represented by vinyl chloride also has been included in the health education program in the schools.

Also, the Health Department cooperates with the Medical Society in disseminating information to physicians in the community. The Department has also set up small but effective epidemiological studies utilizing its own records and reports from community physicians and hospitals.

Finally, the Department's personnel have provided assistance on medical surveillance activities and helped the PVC plant management in establishing control programs to reduce exposure to workers and to reduce emissions to the community.

FIGURE 8 NATIONAL CANCER INSTITUTE, COMPREHENSIVE CANCER CENTERS

Table 17

NATIONAL CANCER INFORMATION SERVICE

CALIFORNIA
LAC-USC Cancer Center
From Area Codes (213), (714) and
(805): 1-800-252-9066
Rest of California: (213) 226-2374

COLORADO
Colorado Regional Cancer Center
1-800-332-1850

CONNECTICUT
Yale University
Comprehensive Cancer Center
1-800-922-0824

DELAWARE
Fox Chase Cancer Center
800-523-3586

DISTRICT OF COLUMBIA
(Includes suburban Maryland
and Northern Virginia)
Cancer Communications for
Metropolitan Washington
(202) 232-2833

FLORIDA
Comprehensive Cancer Center
for the State of Florida
Florida: 1-800-432-5953
Dade County: (305) 547-6920

ILLINOIS
Illinois Cancer Council
Illinois: 800-972-0586
Chicago: (312) 346-9813

MAINE
Maine Cancer Information Center
1-800-225-7034

Table 17 (Continued)

MARYLAND
The Johns Hopkins Oncology Center
800-494-1444

MASSACHUSETTS
Massachusetts Cancer Information Service
1-800-952-7420

MINNESOTA
Minnesota Cancer Council
1-800-582-5262

MONTANA
Montana Cancer Information Service
1-800-525-0231

NEW HAMPSHIRE
1-800-225-7034

NEW JERSEY
Fox Chase Cancer Center
800-523-3586

NEW MEXICO
New Mexico Cancer Information Service
1-800-525-0231

NEW YORK
Roswell Park Memorial Institute
New York State: 1-800-462-7255
Erie County: (716) 845-4400

NEW YORK CITY
Memorial Sloan-Kettering Cancer Center
(212) 794-7982

NORTH CAROLINA
Duke Comprehensive Cancer Center
North Carolina: 800-672-0943
Durham County: (919) 286-2266

Table 17 (Concluded)

PENNSYLVANIA
Fox Chase Cancer Center
800-822-3963

TEXAS
M. D. Anderson Hospital and Tumor Institute
Texas: 1-800-392-2040
Houston: (713) 792-3245

WASHINGTON
Fred Hutchinson Cancer Research Center
Washington: 1-800-562-2875
Seattle Metro: (206) 284-7263

WISCONSIN
Wisconsin Clinical Cancer Center
University of Wisconsin
800-362-8038

WYOMING
Wyoming Cancer Information Service
1-800-525-0231

Persons in states not listed can call the National Cancer Line: (800) 638-6694.

Source: U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health.

Plant Management

First, plant management has recognized its responsibility to protect its employees and has developed controls that more than comply with Occupational Safety and Health Administration standards (i.e., vinyl chloride levels inside the plant no greater than 1-ppm time-weighted average). This has entailed establishing environmental monitoring, designating regulated areas within the plant, and requiring strict standards of work practice. Also, plant management has effectively implemented emission controls to comply with EPA air emission regulations, and plant water effluents and solid wastes are monitored to ensure that only negligible quantities of vinyl chloride are released. Coincident with establishing production and emissio controls, the plant instituted education and medical surveillance programs to help control the vinyl chloride health hazard.

Educational Control

With the advice and assistance of the Advisory Committee for Vinyl Chloride, plant management, in conjunction with the union, established an education program to:

- Increase awareness of the hazards of working with vinyl chloride.
- Inform workers and their families on the nature of controls used by the plant.
- Motivate adherence to safety measures.
- Motivate participation in a medical surveillance program.

The target group for the educational program included not only the plant workers themselves, but also their families and retired workers and their families. (The plant was assisted by the Health Department in identifying and locating former employees for inclusion in the education and medical surveillance program.)

The health educator on the Vinyl Chloride Advisory Committee was retained by the plant to coordinate, conduct, and evaluate the company's program. The educator's duties included enlisting the cooperation and assistance of the workers' union.

The educational program includes information on the emotional aspects of exposure to a known chemical carcinogen, and it provides for personal counseling for both the workers and their families. Since many women in the community, especially wives of workers and women living near the plant, were concerned with reports that vinyl chloride has been linked with miscarriages and birth defects, the plant, in conjunction with the County Health Department and the Medical Sociey, took steps to

inform the public about the (so far) tenuous nature of evidence for this hazard.

In fact, the general educational program was extended to the public at large. For the general public, a telephone medical information service was established by the advisory committee; this service included tapes on the health effects and control of vinyl chloride, the PVC plant's program to control emissions and exposure to workers and to the community, and on cancer in general. For the continuing education of medical and other health personnel in the community, tapes of a more technical nature were provided by the Medical Society.

A recent progress review of the plant education program showed that a "critical-mass" point had been reached--i.e., current workers at the PVC plant, their families, former employees, and their families are knowledgeable about the vinyl chloride hazard, the nature of controls used at the plant, and the necessary safety measures to be continuously taken; also, all targeted persons were participating in the medical surveillance program. Hence, it was possible to reduce the level of formal, direct, effort to the level necessary to convey occasional new items of information, sustain motivation toward safety measures and medical surveillance, and to educate new workers.

Medical Surveillance

Although a formalized plant medical program has been in existence at the PVC plant for a number of years, the findings on vinyl chloride as a cause of angiosarcoma in the liver prompted the establishment of a more closely defined program of medical surveillance, including appropriate biochemical, radiographic, and functional diagnostic procedures.

The medical surveillance program established by the plant management now conforms to the best state of the art currently available--a conformance which is complicated by the fact that the state of the art changes often and, with regard to the major bodily organ of concern, the liver, is in an early stage of development. Hence, the plant physician directing the program is in contact with the University of Louisville group that is actively involved in medical surveillance of vinyl chloride workers, as well as with other sources of information related to his medical surveillance function, such as the Medical Society, the state health department, the National Cancer Institute, NIOSH, and OSHA.

An epidemiologic investigation undertaken at the plant has not, so far, shown any clearcut evidence of vinyl chloride-related injury among the employees.

The Plant Union

The union leadership at the PVC plant played a major role in the entire picture of vinyl chloride control. Since it is to their unions

that workers and their families often turn to for advice of many kinds, the plant's union leadership kept itself well informed on the vinyl chloride problem and was in constant contact with plant management, the plant physician, and with outside sources of information. It lent its strength to, and assisted in developing and implementing, the educational program for workers and their families.

American Cancer Society

The local American Cancer Society carried much of the educational burden in reaching the community in general. It also assisted in the program for education of the PVC plant's workers and their families. The vinyl chloride problem was incorporated into an existing ACS program to approach the community regarding cancer prevention in general.

APPENDICES

Appendix A

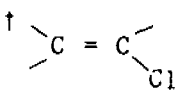
VINYL CHLORIDE-RELATED COMPOUNDS

The evidence that vinyl chloride is a carcinogen in man and animals raises the question of the possible carcinogenicity of other related chemicals in the industrial setting and general environment. The two tables that follow list chemicals that are structurally related to vinyl chloride. These chemicals met one of the following criteria:

- Presence of vinyl chloride moiety.
- Presence of a substituted vinyl group in which substitution is a potential leaving group approximately equivalent to chloride.
- Structure may be a possible vinyl chloride metabolite.

CBDS Number	CAS Number	Chemical Name	U.S. Production* 1973 (grams)	Structural Criteria†
--	75014	Vinyl chloride	2.43×10^{12}	2
--	108054	Vinyl acetate	6.82×10^{11}	1
C04580	127184	Tetrachloroethylene	3.20×10^{11}	1
C04546	79016	Trichloroethylene	2.05×10^{11}	1
C50522	126998	2-Chlorobutadiene	1.8×10^{11} 1974 (est)	1
--	75354	Vinylidene chloride	7.7×10^{10} 1974 (est)	3
C08264	79118	Chloroacetic acid	2.21×10^{10} 1972	3
C50135	107073	Chloroethanol	--**	1
--	--	Dichlorobutadiene	--**	1
--	926578	1,3-Dichloro-2-butene	--**	1
--	78886	2,3-Dichloropropene	--**	1
--	96195	1,2,3-Trichloropropene	--**	2
C50373	593602	Vinyl bromide	--**	3
--	107200	Chloroacetaldehyde	--§	1
--	540590	1,2,-Dichloroethylene	--§	1
--	--	Vinyl iodide	--§	2
--	105384	Vinyl propionate	--§	2

*From 1973 Synthetic Organic Chemicals (U.S. Trade Comm.).

† 

$CH_2 = CH\text{-}X$ where X is a potential leaving group approximately equivalent to Cl.

Possible vinyl chloride metabolite.

**The chemical is listed in the 1973 Synthetic Organic Chemicals, but separate data are not given. This implies that annual production is greater than 1000 pounds or $1000.

§Not in SOC--This implies that annual production is less than 1000 pounds or $1000.

Appendix B

ONE PVC PRODUCER'S APPROACH TO EVALUATION AND CONTROL OF VINYL CHLORIDE EXPOSURE

One of the U.S. producers of PVC, the Eleanora Chemical Division of The Pantasote Company of New York, Inc., recently inaugurated an innovative system for evaluating and controlling the exposure of workers to vinyl chloride.

Description of the System

The Pantasote system yields real-time information on exposure by obtaining data at intervals of several minutes for each workplace. A computer programming system prints a time-weighted average (TWA) for each work area sampled. The system obviates the requirement for daily sampling of individual workers, and it replaces the charcoal tube sampler with a more rapid and accurate evaluating system. This rapid, continuous survey and evaluation technique is coupled with alarms, careful maintenance of equipment, and a stern policy of rule enforcement by the company's management.

The actual sampling time for each work area (or group of work areas) requires only 2 seconds. The frequency of sampling is a factor of the number of stations and the concentration of vinyl chloride. Stations showing a sample value of more than 5 ppm are resampled to determine the accuracy of the sample. As stated above, the system prints out 8-hour TWAs for each area; but in addition, its memory carries and prints out 8-hour TWAs for maintenance men or other individuals who visit any sampling area during their work.

This program is deemed to meet the requirements of the OSHA vinyl chloride standard of October 4, 1974, Section III (4) Monitoring:

> (4) The standard requires that the individual employee exposure levels are to be determined. This may be by personal or area monitoring, just so it is done with a 95% confidence level.

Also in the Standard Section 1910.1017(d)(1) and (4):

> (d) (1)--A program of initial monitoring and measurement shall be undertaken in each establishment to determine if there is any employee exposed, in excess of the action level, without regard to the use of respirators.

(d) (4)--The method of monitoring and measurement shall have an accuracy (with a confidence level of 95 percent) of not less than plus or minus 50 percent from 0.25 through 0.5 ppm, through 1.0 ppm, and plus or minus 25 percent over 1.0 ppm. (Methods meeting these accuracy requirements are available in the "NIOSH Manual of Analytical Methods.")

Major Stages in Developing the System

A comprenensive and thorough evaluation of vinyl chloride concentrations was undertaken in all areas of the plant where exposure was likely to occur. The evaluation extended over several months, and hundreds of samples were taken. The NIOSH method, using personal samplers with charcoal adsorption of the vinyl chloride and subsequent analysis by gas chromatography, was used to determine personal exposure in the areas of potential exposure.

Studies of air movement were made to determine the paths and velocities of contaminated air streams within the buildings. The early studies of vinyl chloride concentration, coupled with knowledge of air movement patterns, served to locate areas of hign exposure and units of equipment that needed maintenance. When indicated equipment repairs nad been made, the sampling program was repeated. Sampling and maintenance continued until exposure within the plant was controlled to a minimum. At this point, the excursions of high exposure found were attributed to human failure.

After the preceeding evaluation and control procedure was completed, the next stage of the company's program was to: establish permanent sampling stations; develop a dystem of sampling and analysis to give real-time evaluations; and attune the system to yield the 95% confidence level required to OSHA regulations.

A portable gas chromatograph with flame ionization detector system was used to pinpoint leaks. It lacked the capability however, to measure or locate random excursions so as to facilitate the use of rapid corrective procedures.

The need was indicated for a continuous rapid monitoring system that would pinpoint excursions in real time at any of the sampling locations. This would enable the evacuation of employees if necessary and allow for repairs to be undertaken.

The Infrared Spectral Resolution gas phase spectrometer had the appropriate capabilities and the sensitivity to determine concentrations in low parts per billion in a metter of seconds. It could be programmed to provide:

- Measurements virtually immune from interferences and thus accurate monitoring without either false low or high readings.

- Monitoring of excursions and 8-hour TWAs.

- Data for permanent hard-copy records in easy-to-read engineering units, maintaining a format of excursion levels and 8-hour TWAs.

The instrument was supplemented with an appropriate system of data gathering and analysis including the reporting of operational information relating to plant concentration values, alarm annunciation, and employee exposure (TWA). The data acquisition system:

- Accepts data from the sensor measurements and converts them into concentration units.

- Determines if alarm levels are needed.

- Interacts automatically with the system operator to alter the sampling sequence of measurements based on current concentration values so as to allow more measurement of areas and sample locations wnere high potential exposure hazards exist. Based on the particular situation and sampling algorithm, the system automatically tests (measures) adjacent sample locations in the vicinity of an excursion and increases the frequency of sampling measurements for that point. All alarm conditions are printed out.

Checklist for Setting Up a Continuous Monitoring System

Setting up a continuous monitoring system must be done systematicaly to quantify the environment of a plant. The following is a checklist for use in setting up such a system.

- Make a preliminary charcoal survey.

- Repair obvious leaks ascertained from the data obtained in preliminary survey and from use of the gas chromatograph flame ionization detector.

- Divide plant into study areas of approximately equal size, and monitor cycles of operation at each station; repeat each sampling cycle at 15-minute intervals as a minimum (maximum of 30-minute intervals).

- Statistically analyze results to determine areas for permanent sampling stations.

- Provide a sample probe and a continuous sampling program that samples each probe for a predetermined length of time.

- Conduct a time-motion study to quantify probability of an employee with a specific job title being in a given area.

- Sample an area for a period of 16 to 24 hours during normal operation.

- Continue this preliminary analysis until the exposure concentration and patterns for the plant are clearly defined. To recap--obtain these three sets of data: concentration in an area as a function of time; probability of an excursion in an area; probability of an employee with a given job title being in a given area.

- Establish a correlation of monitor results and charcoal tube results by continuously sampling with charcoal tubes a number of individuals in areas being monitored.

- Determine (after the equivalency of the two sampling methods is established) the amount of monitoring required to evaluate employees' exposure to within 25% with a 95% confidence level.

- Determine, through sampling, the variation of vinyl chloride concentration with time; i.e., chances of negative, zero, and high concentration.

- Determine through time studies the work path and practice for each operator or worker in the contaminated area; compute the actual time within the area, using time study and sample data to determine a TWA for each employee for short periods, (15 minutes), longer periods (1 hour) and for an 8-hour work day.

- Program the system so that it responds to the plant environment with variations in frequency of sampling--i.e., using its probabilistic intelligence, the system will hunt for excursions, thereby biasing areas of high chance of excursion and minimizing the amount of time required to monitor low-chance areas.

- Set up the systems to print out reports of 15-minute TWAs at each sampling point and at a given time each day; or, for each 8 hours, to print out the average length or number of each excursion and TWA, for each station, and for specific individuals--i.e., maintenance or supervisory personnel that are in many work places in an 8-hour shift.

- Program the system to a three-alarm level--a Blue alert when a concentration of 5 ppm is reached, a Yellow alert at a concentration of 25 ppm, and a Red alert at 1000 ppm, at which time the building is to be evacuated.

- Design the system for continuous monitoring without operator assistance and for programming of all items previously listed.
- Install analytical equipment that is fast, accurate, and specific.
- Design sample handling to be fast, self-cleaning, and continuous.

It is apparent that such a system--operating continuously in the potential exposure area, determining concentrations and TWAs at 15-minute intervals, surveying areas of potential excursions at frequent intervals, and printing out data as previously described--will provide surveillance of real-time exposure in a vinyl chloride plant superior to that of the charcoal tube.

Appendix C

SOURCES FOR FURTHER INFORMATION

Department of Health, Education, and Welfare--Center for Disease Control (CDC)

National Institute for Occupational Safety and Health (NIOSH)

Regional Offices:

I	John F. Kennedy Federal Building Boston, MA 02203	(617) 223-6831
II	26 Federal Plaza New York, NY 10007	(212) 264-4600
III	3535 Market Street Philadelphia, PA 19101	(215) 597-6492
IV	50 7th Street, NE Atlanta, GA 30323	(404) 526-5817
V	300 S. Wacker Drive Chicago, IL 60606	(312) 353-5160
VI	1114 Commerce Street Dallas, TX 75202	(214) 749-3396
VII	601 E. 12th Street Kansas City, MO 64106	(816) 374-3436
VIII	1961 Stout Street Denver, CO 80202	(303) 837-3373
IX	50 Fulton Street San Francisco, CA 94102	(415) 556-6746
X	1321 2nd Avenue Seattle, WA 98101	(206) 442-0420

Food and Drug Administration (FDA)
5600 Fishers Lane
Rockville, MD 20852 (301) 443-3380

National Institute of Health (NIH) (301) 656-4000

National Cancer Institute (NCI)
9000 Rockville Pike
Bethesda, MD

Division of Cancer Control and Rehabilitation
Dr. Diane Fink (301) 427-7997

Environmental Protection Agency
401 M Street, SW
Washington, DC 20460 (202) 755-2673

Regional Offices:

I John F. Kennedy Federal Building
Boston, MA 02203

II 26 Federal Plaza
New York, NY 10007

III Curtis Building
6th and Walnut Streets
Philadelphia, PA 19106

IV 1421 Peachtree Street
Atlanta, GA 30309

V 230 Dearborn Street
Chicago, IL 60604

VI 1600 Patterson Street
Dallas, TX 75201

VII 1735 Baltimore Avenue
Kansas City, MO 64108

VIII 1860 Lincoln Street
Denver, CO 80203

IX 100 California Street
San Francisco, CA 94111

X 1200 6th Avenue
Seattle, WA 98101

Department of Labor--Occupational Safety and Health Administration (OSHA)

Regional Offices:

I 18 Oliver Street
Boston, MA 02110

II 1515 Broadway
New York, NY 10036

III 3535 Market Street
Philadelphia, PA 19104

IV 1375 Peachtree Street, NE
Atlanta, GA 30309

V 230 S. Dearborn Street
Chicago, IL 60606

VI 1512 Commerce Street
Dallas, TX 75201

VII 911 Walnut Street
Kansas City, MO 64106

VIII Federal Building
P.O. Box 3588
Denver, CO 80202

IX Federal Building
P.O. Box 36017
San Francisco, CA 94102

X 506 2nd Avenue
Seattle, WA 98104

Appendix D

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