Summary

While the dangers of asbestos have been well recognized for over five decades, the industry was able to largely ignore or suppress these until studies in the 1950s by Doll in England and by Selikoff in the U.S. (sponsored by the asbestos insulators union) established the relationship between asbestos and asbestosis and cancers at various sites. Since then the industry has employed scientific expertise to advocate its position in professional journals and to successfully resist and prevent effective government regulations. With an estimated 8 to 11 million workers having been exposed since World War II, the toll of asbestos occupational disease and cancer is now reaching epidemic proportions. Workers have begun to exercise legal initiatives, including third-party and medical malpractice suits, resulting in multimillion dollar awards.

Concerns are also mounting on the dangers to the public from "low-level" asbestos contamination of air and water and from asbestos-containing consumer products such as hair dryers.

As the realization is dawning that asbestos is too expensive in terms of disease and death to continue using, the industry is beginning to develop the strategies of relocating in lesser developed countries and promoting the use of fiberglass as an asbestos substitute in the United States. There are serious unresolved questions as to the dangers of fiberglass and there are possibilities that it may be no less hazardous than asbestos.

The massive human toll taken by asbestos is probably the single most important incentive to the development of coherent national policies recognizing preventive medicine as a major future component in the delivery of health care.

Vinyl Chloride

One of the most common pejoratives applied to modern times is "the plastic age." And just as age-old natural materials like wood, glass, and metal have been replaced by cheaper synthetic plastics, this seems to imply, so has the value of our lifestyle been degraded. Many things we now touch or use daily are made of plastic. Yet the plastic transformation of our society has only taken place over the last forty years. The consequences to society of its plasticization, in terms both of high energy and health costs, have only recently been appreciated. One of the major costs is from cancer.[1]

Manufacture

Plastics are the leading product of the modern petrochemical industry. This era of plastics dawned in the 1930s with the realization that petroleum was the cheapest and simplest starting material for the synthesis of the vast array of organic chemicals now used by modern industry. Petroleum then gradually replaced coal, which had been previously used for this purpose, and is now almost the exclusive basis of the organic chemical industry.

Vinyl chloride (VC) lies at the heart of the plastics industry. VC was first discovered in 1837. Large-scale production, however, did not start until the 1930s, when it was synthesized from chlorine and ethylene, largely for the manufacture of synthetic rubber.[2] Production levels increased rapidly after World War II at a rate of 15 percent per year, reaching a total of about 7 billion pounds this year.

VC is a simple chemical consisting of a small single molecular unit (a *monomer*) which is normally a gas but can be shipped and stored as a liquid under pressure. VC reacts under conditions of heat and pressure and in the presence of plasticizing chemicals to form large molecular chains (*polymers*) of polyvinyl chloride (PVC) resins. The polymerization of the VC monomer never proceeds to completion, but always leaves some unreacted VC entrapped in the PVC resin. Depending on the production process, this unreacted VC has concentrations ranging as high as 8,000 parts per million (ppm).[3] This residual VC can be dissolved out of the polymer or released by heating it.[4]

PVC resins are molded, injected, and extruded in the fabrication of a wide array of plastic products. These include phono-

graph records, wire and cable insulation, floor coverings, garden hose, furniture, upholstery fabrics, car bodies, coating of fabrics and wallpaper, containers, bottles, and food wrappings. It is difficult to get an accurate count of the number of U.S. workers involved in all the various stages of PVC production. There are about seventeen VC manufacturing plants, employing about a thousand workers, and forty PVC plants, employing about six thousand workers.[5] There are no figures available, however, on the number of fabricating plants or on the number of their employees. These must run into the hundreds of thousands.

Suppression by the Industry of Data on the Carcinogenicity of Vinyl Chloride

In May, 1970, an Italian toxicologist, Pier Luigi Viola (employed by Solvar Manufacturing Corporation), reported at an international cancer congress in Houston, Texas, that long-term intermittent exposure of a small group of rats to 3 percent of VC in air (30,000 ppm) resulted in the production of cancers in a wide range of organs.[6]

This first report on the carcinogenicity of VC created little impact because Viola had claimed that the type of tumors induced were peculiar to rats and without human significance, and also because of the very high levels of VC to which the rats were exposed. In fact, Viola had induced tumors at much lower concentrations, extending down to less than 5,000 ppm. These facts were discussed in detail at a Manufacturing Chemists Association meeting in November, 1971, where it was concluded that they should not be published, as this would otherwise "lead to serious problems with regard to the vinyl chloride monomer and resin industry . . . and force an industrial upheaval via new laws or strict interpretation of pollution and occupational health laws."[7] Union Carbide further expressed its concerns that in view of the large stake it had in "areas most likely to be affected, such as food, food packaging, fiber and aerosols [it] would be seriously hurt by arbitrary or panic-induced government restrictions."

Prior to this, there had apparently been no studies in the plastics industry on the possible carcinogenicity of VC, although tens of thousands of workers the world over had been exposed to it for

over three decades and at least seven fatal cases of liver cancer had occurred in VC/PVC workers prior to 1970.[8] Disturbed by Viola's public report, a consortium of giant European chemical industries, led by Italy's semi-governmental Montedison, and with contributions from British, Belgian, and French firms, financed a major study initiated in July, 1971, by Cesare Maltoni of the Bologna Cancer Institute.*[9] An extensive series of tests were conducted in which adult, infant, and pregnant mice, rats, and hamsters were exposed to VC in inhalation chambers at concentrations ranging from the relatively low 50 ppm level to the toxic 100,000 ppm (10 percent) level. By late 1972, Maltoni had confirmed and extended Viola's results. VC was shown to be a potent carcinogen, inducing a rare type of liver cancer (called angiosarcoma), as well as more common liver cancers (hepatomas) and cancers of other organs, including kidney, brain, and lung. The cancers were subsequently found even at the lowest level then tested, 50 ppm. The overall tumor incidence was concentration-dependent, with higher doses increasing the incidence and rate of development of the cancers.[10]

In October, 1972, the Manufacturing Chemists Association, the major trade association of the U.S. chemical industry, entered into an agreement with the European consortium to share their information, but not to disclose it without prior consent.[11] In January, 1973, the U.S. industry representatives visited Maltoni and were given full details of his experimental studies. The Manufacturing Chemists Association (and Maltoni) subsequently failed to disclose this information, in spite of the fact that NIOSH, in the same month, had publicly requested all available data on the toxic effects of VC. In March, 1973, the Manufacturing Chemists Association recommended to NIOSH a precautionary label for VC that made no reference to toxic or carcinogenic effects on animals or humans.

Four months later, however, representatives of the Manufacturing Chemists Association met in secret with Marcus Key, then Director of NIOSH and now Professor of Occupational Medicine at the University of Texas, Houston, and a small group of his sen-

*An additional reason for initiating these tests, revealed by Maltoni in 1975, was his hitherto undisclosed finding in 1969 of malignant cells (of a large cell adenocarcinomatous type not seen in smokers) in the sputum of VC/PVC workers, suggestive of early lung cancer or pre-cancerous changes.

ior staff. They were informed that Maltoni had induced tumors in rats exposed to VC levels of 250 ppm. At one stage of the meeting, an industry representative, V. K. Rowe of Dow Chemical Company, adjourned to a separate office for a private meeting with Key.[12] Rowe subsequently commented that "this private discussion of the carcinogen problem was worth the whole effort." Finally, industry concluded that as a result of a meeting "the chances of precipitous action by NIOSH on VC was materially lessened. NIOSH did not appear to want to alienate a cooperative industry or they did not want to know too much unpublished data."

In March, 1973, following complaints of an unpleasant taste, Schenley Distillers found 10-20 ppm levels of VC in their liquors sold in PVC bottles. On the basis of these findings, and still knowing nothing about the carcinogenicity data, FDA then banned the use of these liquor bottles. In December, 1973, the Society of Plastics Industry, the plastics industry trade association, provided the FDA with data on migration of VC from PVC containers to food, but made no reference to problems of carcinogenicity.

On January 22, 1974, B. F. Goodrich announced that since 1971 three PVC workers in their Louisville, Kentucky, plant had died of angiosarcoma of the liver. On that same day, the Manufacturing Chemists Association revealed the by now fifteen-month-old Maltoni data to NIOSH.

According to a recent special committee report of the American Association for Advancement of Science, the Manufacturing Chemists Association "appears to have deliberately deceived NIOSH regarding the true facts. . . . Because of the suppression of these data, tens of thousands of workers were exposed without warning, for perhaps some two years, to toxic concentrations of vinyl chloride."[13]

Distortion by the Industry of Data on the Carcinogenicity of Vinyl Chloride

The first published case reports of angiosarcoma of the liver in VC/PVC workers generated an intensive epidemiological investi-

gation of cancer mortality in the industry in several countries. A British Petroleum Corporation study, published in *Lancet* in 1975, claimed that the longer one worked in a VC plant, the less was the risk of getting cancer.[14] Closer examination by NIOSH, however, questioned the interpretation of the English data. Expected death rates in workers exposed to VC for fifteen or more years were artificially reduced, and rates in workers with lesser exposures were artificially inflated. Re-analysis of the data by NIOSH, using standard methods, showed a clear excess of mortality from all causes and all cancers, including digestive system and lung cancers, besides angiosarcoma of the liver (Table 5.3).

Exaggeration by the Industry of the Costs of Regulating Occupational Exposure to VC

As information on the carcinogenicity of VC began to surface, OSHA was prodded into action. On April 5, 1974, OSHA reduced permissible exposure levels from 500 ppm to a new emergency standard of 50 ppm. Under pressure from labor and independent scientists, who insisted that the new standard was too high for safety, on May 10, 1974, OSHA proposed a VC standard "at no detectable level" (in the range of 0 to 1 ppm) to be achieved by the use of engineering controls, rather than worker use of respirators. Faced with strenuous industry opposition and alarming economic impact analyses, OSHA did not proceed to adopt this standard, but on October 4, 1978, instead issued the more lenient final 1 ppm standard.[15] Even at that time, however, it had been shown that exposure of small numbers of rats to 5 ppm VC induced an increased incidence of cancer, and there was no evidence then (or now) of any safe exposure level for VC, or indeed to any other carcinogen.†

Nevertheless, industry objected strongly to the "no detectable level" proposal on the grounds that it was unnecessary, too expensive, and beyond their compliance capability. To bolster these claims, contracts were given out by the Society of the Plastics In-

† Later studies by Maltoni and others demonstrated carcinogenic effects, including breast cancer, in rodents following inhalation of VC at levels as low as 1 ppm.

Table 5.3 *Comparison of Causes of Death in VC Workers Reported by British Petroleum (BP) and Recalculated by NIOSH*

Cause of Death, Reported by BP and Recalculated by NIOSH	Standard Mortality Ratios* for Workers Exposed to VC for Varying Times		
	Years of Exposure		
	9 or less	10–14	15 or more
All causes			
BP	112	107	61
NIOSH	79	137	353
All cancers			
BP	123	58	73
NIOSH	86	76	428
Digestive system cancer			
BP	106	47	121
NIOSH	75	62	702
Lung cancer			
BP	129	101	62
NIOSH	90	133	366

Source: J. K. Wagoner, P. F. Infante, and R. Saracci, "Vinyl Chloride and Mortality." *Lancet* 2 (1975): 194–95.

* Values greater than 100 indicate excessive deaths relative to expected population values.

dustry, Inc. to Arthur D. Little, Inc., of Cambridge, Massachusetts. Also, OSHA gave out a contract to Foster D. Snell, a division of Booz, Allen and Hamilton, for the purpose of estimating the economic impact of the new standard. The consulting firms predicted costs as high as $90 billion and losses up to 2.2 million jobs, persuasive arguments in the depressed economic climate of 1974. However, these estimates were shown by the subsequent experience of the plastics industry to be gross exaggerations.[16] In spite of massive industry lobbying and pressures, OSHA stood firm on the new 1 ppm standard, which was passed on April 1, 1975.

The Society of the Plastics Industry then petitioned the 2nd Circuit Court of Appeals to review the 1 ppm final OSHA standard stating:

> The evidence clearly demonstrates that the Standard is simply beyond the compliance of the industry. . . . The general increase in raw materials costs (an unavoidable result of this Standard), and the costs of monitoring, respiratory protection, medical surveillance and record keeping all militate against the likelihood that the bulk of this segment of the industry would be able to survive economically. The evidence is clear (that these various industries) would at the very least suffer economic disaster if not close down completely.

Within one year, the VC/PVC manufacturing industry had successfully met the new standard, and without any major economic dislocation or plant shutdowns. B. F. Goodrich, one of the industry giants, redesigned its manufacturing technology to enclose VC manufacturing and handling processes and plug possible sources of leaks. Additionally, a "stripping" process was developed to reduce levels of the unreacted VC monomer in the PVC resin and to decrease VC loss in the process. The initial capital costs of the compliance technology were only $34 million. Contrary to the estimates of the industry consulting firms, B. F. Goodrich found that the new clean-up technology actually cut labor costs and could be profitably leased. (In spite of this, B. F. Goodrich increased the price of its PVC products in 1976, claiming higher production costs and blaming these on regulatory standards.) The experience of Union Carbide is similar. In late 1975, R. N. Wheeler, a senior technical official expressed surprise as to how unexpectedly easy it had been for his company to comply with the 1 ppm standard. However, earlier intercompany reports had documented the economic advantages of compliance through recovering and recycling VC which would have otherwise been discharged into the environment. Estimates of the economic impact of compliance had ignored the major costs to industry from losses of VC gas, with resulting air pollution of the plant and adjacent community. They had also ignored or discounted the major costs to society, both direct and indirect, from failure to regulate, with resulting VC-induced disease. Finally, these estimates did not reflect the emergence of a small and booming industry manufacturing the monitoring equipment now required to meet OSHA regulations.

It is clear that, despite uniform and overwhelming protestations and prophecies to the contrary, the industry had no major difficulty in meeting the 1 ppm standard. It is also clear that the industry is flourishing with high production, strong demand, growing capacity, and steady prices. The slump in the PVC market in 1974 and 1975, reflecting the 1973 oil embargo and a general pattern of recession, has been replaced by a boom, well recognized in chemical trade publications since 1976. (See for instance, *Chemical and Engineering News,* July 24, 1978, p. 12, and September 4, 1978, p. 13.)

Cancer and Other Diseases Due to VC

Long before the 1974 reports of angiosarcoma in VC/PVC workers, VC was known to be toxic to experimental animals and to humans at relatively high concentrations. Studies on experimental animals in the early 1960s, by Dow Chemical Company and others, established that VC induced toxic effects in the liver and kidney at concentrations as low as 500 ppm, then the U.S. occupational standard.[17] At higher levels skin and bone abnormalities were also noted.

Acute toxic effects, such as headaches, disorientation, dizziness, drowsiness, and loss of consciousness had been noted in VC/PVC workers as long ago as 1949.[18] In the same year, the Russian scientific literature reported the occurrence of chronic gastritis, hepatitis, and skin lesions from VC exposure. In 1957 there appeared the first report of a new occupational disease among vinyl plastic workers called acroosteolysis, characterized by a thickening or clubbing of the fingers and toes, spasmodic contraction of the blood vessels, painful bone changes, and arthritis of the knuckles. Additional cases of acroosteolysis have since been noted from plastic plants all over the world. By 1966, it was recognized that liver damage, hepatitis and cirrhosis, was common among VC/PVC workers and in some cases occurred after only one or two years of employment.

Since 1974, seven independent retrospective epidemiological studies have identified over seventy deaths from angiosarcoma of

the liver in VC/PVC plants in the United States and elsewhere.‡ While most have involved polymerization workers, others have occurred in workers exposed to lower concentrations of VC, including four cases in PVC fabricating plants, where PVC levels were probably below 10 ppm. Cancers at various other sites including the lung, brain, kidney, and digestive tract, besides lymphomas and leukaemias, have also been found in VC/PVC workers.* The lung cancers were mainly of a type (adenocarcinoma and undifferentiated large-cell) not generally associated with smoking, and were found in workers engaged in a wide range of VC/PVC operations, including PVC milling and packaging. While the occupational exposures preceding these various cancers have generally ranged from thirteen to twenty years, there have been four reports of liver angiosarcoma following less than six years of exposure. The true incidence of VC/PVC cancers in exposed workers is still unknown, because of considerations of latency, and also because of the ever-increasing number of cancers reported in organs other than the liver, which can no longer be considered the exclusive or even predominant site of VC/PVC-induced cancers.[19] It should be noted that the wide range of VC cancers found in workers is, in general, similar to the types and distribution of cancers induced in animal tests.

Despite the growing evidence of chronic human disease and the many thousands of workers at risk worldwide, the industry failed for more than thirty years to undertake any carcinogenicity tests in animals. Such tests could as easily have detected the carcinogenic effects of VC in the 1940s as they eventually did in the 1970s.

There are still elements of the academic community who minimize the risks of VC exposure. A recent brochure from the University of Louisville's Vinyl Chloride Project, a multimillion dol-

‡ At least seven fatal cases of primary carcinoma of the liver, subsequently diagnosed as angiosarcoma, had been recognized in VC/PVC workers before 1970. Six of these cases occurred in the United States between 1961 and 1968, and the other in Sweden.

* An excessive incidence of brain tumors (gliobastoma multiforme) has been noted among VC workers in Union Carbide and Monsanto plants in Texas City, Texas, and is now being investigated by OSHA and NIOSH. There have been ten deaths from the brain tumor among workers at the Carbide plant since 1962.

lar NCI-funded program for the prevention and detection of VC-induced occupational cancer and other diseases, states:

> The low incidence of cancer development among people with prolonged exposure to vinyl chloride is a reflection of the body's ability to remove the chemical agent safely without developing cancer. Some individuals do not have this capability and develop cancer after years of exposure.[20]

The brochure makes no reference to the fact that the present apparent "low incidence of [VC] cancer" may simply reflect its long latency period. Additionally, the independence of this University project has been questioned following recent disclosure of its receipt of undisclosed support from the Manufacturing Chemists Association.†

Cancer and Other Diseases Due to PVC

While in the past concerns have focused on VC itself as the major toxic and carcinogenic agent to VC/PVC workers, there is growing realization of other serious hazards posed by PVC dust to far greater numbers of workers in operations involving the packaging and milling of PVC, and its fabrication into innumerable plastic products. These hazards most probably reflect the presence of unreacted VC entrapped at concentrations up to about 1 percent (10,000 ppm) in the PVC resin or products, and which can be released by their heating or processing, or can be dissolved from inhaled PVC dust by tissue fluids. Additionally, VC gas can be absorbed on PVC or on other dusts which may act as inert carriers.

Experimental studies have shown that inhalation of PVC dust or its instillation into the lungs of rodents can induce acute inflammation followed by chronic granulomatous changes. Similarly, a wide range of abnormalities has been recognized in the lungs of workers exposed to PVC dust in a variety of operations involving bagging and packaging of PVC resins and their fabrication of PVC

† This was admitted at an NCI site visit meeting at the University of Louisville on September 26 and 27, 1977.

products, where dust levels have been found to range as high as 20 mg/m³, and where the dust may be so fine that most is inhaled deep into the lungs. The lung abnormalities reported include reduction of lung function, radiological changes, and chronic lung disease (pneumoconiosis). "Meat-wrapper's asthma" is a possibly related problem well recognized among supermarket and wholesale meat workers who, in packaging cuts of meat, use a "hot wire" or "cool rod" to cut and seal the sheet of Saran Wrap‡ or other PVC plastic film used for wrapping.[21] In the cutting process, particularly when high temperatures are used, irritant fumes, including hydrochloric acid, are given off. While the industry has readily ascribed the asthma to such irritants, it is not yet known whether there are any carcinogenic hazards posed by unreacted VC, or other related carcinogenic agents, such as vinylidene chloride, which may be liberated from the plastic film by the heat cutting.

More ominous than the inflammatory lung changes, an excess frequency of cancer deaths has been reported in four retrospective epidemiological studies of workers predominantly exposed to PVC dust.[22] These include lung cancer (of the same type as described in VC workers), and cancers of the urinary tract, digestive system, and lymphatic system.

Public Exposure to VC

The public and the regulatory agencies are now beginning to realize that what goes on in a plant often affects the health of those living nearby in the community. The closer you live to a plant manufacturing VC or PVC or fabricating PVC products, the more VC you are likely to breathe. EPA has estimated that 4.6 million people live within five miles of VC/PVC plants.[23] No estimates are available for the presumably greater number of people living near the uncounted PVC fabricating plants scattered over the country. Measurements by EPA in 1974 and 1975 indicated that in 90 percent of cases, VC levels in the vicinity of VC/PVC

‡ Saran Wrap, a product of Dow Chemical, is a co-polymer of VC and the related vinylidene chloride, which has recently also been shown to be carcinogenic in rodents, inducing angiosarcoma of the liver in addition to other cancers.

plants were below 1 ppm; EPA estimated that average exposure within a five-mile radius would be about 17 parts per billion (ppb). Higher levels, however, were found in 10 percent of communities sampled—33 ppm at a distance of one-third of a mile from the center of one plant.

EPA also estimated that VC/PVC plants were losing 220 million pounds of VC into the air annually, mainly from PVC plants. These considerations, together with reports of excess birth defects in Ohio women living near VC/PVC plants, prompted EPA in October, 1976, to propose regulating VC as a "hazardous air pollutant" and to limit plant emissions to 10 ppm. These regulations would be expected to reduce VC emissions by about 95 percent of previous levels and to reduce community exposure by a similar amount. However, no regulations have yet been developed for plants fabricating PVC products nor for municipal incinerators, which discharge VC into the air from thermal processing and combustion of plastic products, respectively.

The results of a June, 1977, EPA investigation of an elementary school in Saugus, California, located across the road from Keysor-Century Corporation, which manufactures PVC and phonograph records, are disturbing.[24] Levels as high as 2.8 ppm VC have been measured in the air in classrooms. This heavy contamination largely results from VC losses during tank car unloading at the plant in the night.

The occurrence of VC disease in the general population has been suggested by preliminary findings of an incidence of 18 congenital malformations per 1,000 live births in women living in three Ohio communities near PVC plants, in comparison with an incidence of 10.5 per 1,000 in those living at a distance.[25] Most of these malformations were of the brain. In addition, an excessive number of brain tumors in adult males were found in those communities near the PVC plants. A similar excess of birth defects over a period from 1966 to 1974, has been recently noted in Shawinigan, Quebec, where a large VC/PVC plant is located.*[26]

* More recently, five cases of angiosarcoma of the liver have been reported in women who have lived for prolonged periods close to VC polymerizing or PVC fabricating plants, and who have never worked in these plants or been exposed to other carcinogens known to induce angiosarcoma (such as arsenic or thorium dioxide).

These various effects have been associated with the detection of VC in the air of communities adjacent to VC/PVC plants.

Another way in which the public has been exposed to VC is from aerosol propellants in consumer products such as insecticides, hair sprays, disinfectants, and furniture polishes.[27] EPA studies show that during use of hair or insect sprays, users breathed VC in the 100 to 400 ppm range. After much prodding, the FDA banned the use of VC as a propellant in cosmetic products, including hair sprays. However, the FDA did not consider the matter serious enough to order a recall of VC-containing products so that the public could avoid further exposure. The Health Research Group eventually pried out from the FDA the brand names of hair sprays that used VC as a propellant, identifying Clairol as a top selling product. According to a recent survey by Consumer Product Safety Commission staff, no new consumer products containing VC have been manufactured since 1974, and no VC-containing products remain on the market in 1978.

The FDA also showed reluctance in moving against the use of PVC containers for food products, although it was aware that unreacted VC was leached out of the container walls by the foods or fluids inside. By 1974 the FDA knew that VC levels as high as 9 ppm had been found in vegetable oils sold in rigid PVC containers.[28] High levels were also found in other PVC-packaged products, including beverages and cosmetics. While the FDA proposed a ban on the use of rigid PVC containers for food products and beverages, the use of PVC film for wrapping foods is still sanctioned.

Extensive research is now being done by industry with the object of reducing VC levels in PVC products, especially those intended for food packaging and medical applications.[29] However, as indicated in a recent trade journal article, the prospects of success seem slim:

> At present conventional devolatilizing techniques cannot be successfully offered to PVC. . . . It cannot be expected, however, that a completely monomer-free PVC resin will be a commercial reality in the near future.[30]

Summary

Vinyl chloride (VC) is a simple gaseous chemical which can be polymerized to form a wealth of plastic materials. Despite recognition for several decades of a wide range of chronic toxic effects in animals and exposed workers, it was not until 1970 that the carcinogenicity of VC was first established experimentally. A consortium of European plastics manufacturers then funded further carcinogenicity testing of VC which confirmed and extended the earlier findings. These results, which were shared with the Manufacturing Chemists Association, remained unpublished for over eighteen months until the announcement by B. F. Goodrich in January, 1974, of the occurrence of three fatal cases of an extremely rare cancer, angiosarcoma of the liver, among its VC/PVC workers. On the same day, the Manufacturing Chemists Association revealed the results of tests showing VC to be a potent carcinogen in rodents, inducing angiosarcoma of the liver and a wide range of cancers at other sites at exposures of 50 ppm and below.

In the absence of available information on the carcinogenicity of VC in animals, its effects in exposed workers were only finally recognized because of the occurrence of the three rare angiosarcomas of the liver. Had VC only induced lung cancer, its carcinogenicity would in all probability have remained unrecognized or ascribed to smoking.

The present occupational standard of 1 ppm was promulgated by OSHA in spite of estimates by industry of grossly exaggerated costs and unemployment. While the standard affords some degree of protection to workers in VC/PVC plants, it is clearly excessive. Furthermore, it does not protect the greater number of workers in PVC packaging and fabricating plants, nor does it prevent exposures to VC of the public-at-large, particularly those living in the vicinity of VC/PVC plants, with attendant risks of cancer and also possibly birth defects.

Quite apart from VC itself, there has been recent recognition of the carcinogenic hazards of exposure to PVC dusts in a wide range of operations involving bagging and packaging of PVC resins, and their formulation into innumerable plastic products.

My own company is very conscientious and careful about the chemicals we use in the manufacturing process.

Ellington M. Beavers, Medical Director and Vice President, Rohm & Haas Company, 1974.

Bischloromethylether

It is surprising how quickly the scandals of yesterday become today's respectable textbook classics. A standard 1975 medical text, *Occupational Medicine,* contains the following matter-of-fact account of the discovery of bischloromethylether (BCME) as a potent human carcinogen:

> [Dr. William Figueroa] reported on lung cancer found in chloromethylmethylether workers and suggested another occupational hazard that increases the risk of lung cancer. They surveyed and studied a chemical manufacturing plant with approximately 2,000 employees where periodic chest X-ray surveys had been carried out for many years. In 1962, management became aware that an excessive number of workers suspected of having lung cancer were reported in one area of the plant, and promptly engaged consulting services to identify and resolve what appeared to be a serious problem.[1]

Claims of "prompt" action by this company, Rohm and Haas (R&H), one of the world's dozen largest chemical manufacturing companies, have often been reiterated in the medical literature. The facts prove the contrary. As shown, among others, by Phila-

18. P. Kotin and G. R. Chase, "Comments on 'Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Mineral' and 'Asbestos Fiber Exposures in a Hard Rock Gold Mine'," Johns-Manville Corp., Health and Safety Department (Denver, Colo., 1976).
19. J. K. Wagoner, et al., "Comments on 'Critique of Mortality Patterns among Hard Rock Gold Miners Exposed to an Asbestiform Mineral' and 'Asbestos Fiber Exposures in a Hard Rock Gold Mine'" (Washington, D.C.: National Institute of Occupational Safety and Health, 1976).
20. J. F. Finklea to Assistant Secretary for Health, "Evaluation of Data on Health Effects of Asbestos Exposure and Revised Recommended Numerical Environmental Limits," December 15, 1976.
21. P. A. Greene, "OSHA Serves a Corporate Client, Ignoring Asbestos in Vanderbilt Industrial Talc" (Washington, D.C.: Public Citizen Health Research Group, 1976).
22. Ibid., p. 18.
23. Occupational Safety and Health Review Commission, Docket 10757 OSHD 20, 947, June 28, 1976.
24. International Agency for Research on Cancer, *Asbestos*, IARC Monographs on the Evaluation of Carcinogenic Risks of Chemicals to Man, vol. 14 (Lyon, France, 1977). This is the best available summary on the carcinogenicity of asbestos.
25. H. Weinstein, "Did Industry Suppress Asbestos Data? Dangers Apparently Long Known," *Los Angeles Times*, October 23, 1978.
26. Kotelchuk, "Asbestos Research."
27. A. Lanza, "Effects of Asbestos Dust on the Lungs," Public Health Reports 50 (1935), p. 1.
28. K. W. Smith, "*Industrial Hygiene—Survey of Man* in a Dusty Area," unpublished Johns-Manville Report, 1949.
29. The Louisville Trust Company, Administrator of the Estate of William Virgil Sampson *vs.* Johns-Manville Corporation, Jefferson Circuit Court, Common Pleas Branch, Seventh Division, Deposition of Kenneth W. Smith, April 21, 1976, pp. 63–64.
30. I. J. Selikoff, J. Churg, and E. C. Hammond, "Asbestos Exposure and Neoplasia," *Journal American Medical Association* 188 (1964), pp. 22–26; I. J. Selikoff, J. Churg, and E. C. Hammond, "The Occurrence of Asbestosis Among Insulation Workers in the United States," *Annals New York Academy of Sciences* 132 (1965), pp. 139–55.
31. Minutes Asbestos Textile Institute Meetings, June 6, 1963 (Air Hygiene Committee), and October 8, 1964 (Air Hygiene Committee).
32. Minutes General Meeting Asbestos Textile Institute, February 4, 1971.
33. Selikoff, Churg, and Hammond, "Asbestos Exposure and Neoplasia," and "Asbestosis in Insulation Workers."

34. J. C. Wagner, C. A. Sleggs, and P. Marchand, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," *Brit. J. Ind. Med.* 17 (1960), pp. 260–71.
35. W. Nicholson, Mt. Sinai School of Medicine, New York, quoted in Brodeur, *Expendable Americans*, p. 172.
36. R. Nader and R. Harris, "Don't Drink the Water, Don't Breathe the Air," *Environmental Action*, September 15, 1973; E. W. Lawless, *Technology and Social Shock* (New Brunswick, N.J.: Rutgers University Press, 1977), pp. 288–307.
37. T. Temple, "Protecting Lake Superior," *EPA Journal* 4 (1978), pp. 5–9. Contains a chronological summary of the Reserve Mining case.
38. Selikoff, "Cancer Risks of Asbestos Exposure"; Nicholson, "Occupational and Environmental Standards for Asbestos"; IARC, *Asbestos*.
39. Nicholson, "Occupational and Environmental Standards for Asbestos"; IARC, *Asbestos*.
40. D. L. Bayliss et al., "Mortality Patterns among Fibrous Glass Production Workers," in Saffiotti and Wagoner, eds., *Occupational Carcinogenesis*, pp. 324–35.
41. P. Kotin, *New Times*, November 25, 1977.
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