

Wheeler Report Re PVC-VC in
Maliko v. UCC - 1978

P0110-00111
UCC Documents

UCC
067885



SOUTH CHARLESTON PLANT

**UNION CARBIDE CORPORATION
CHEMICALS AND PLASTICS**

P.O. BOX 8004, SOUTH CHARLESTON, W. VA. 25303

Re: Maliko, Mazanowski, Wilkinson and Schaffer vs Union Carbide

"POLYVINYL CHLORIDE - VINYL CHLORIDE MONOMER"

Literature Survey and Engineering Opinions

Prepared by

R. N. Wheeler, Jr.

April 10, 1978

UCC
067886

TABLE OF CONTENTS

	Page
SUMMARY AND CONCLUSIONS	3
R. N. WHEELER, JR. - QUALIFICATIONS	6
LITERATURE SURVEY	
POLYVINYL CHLORIDE INDUSTRY AND PROCESSES	8
VINYL CHLORIDE TOXICOLOGY	16
POLYVINYL CHLORIDE TOXICOLOGY	24
UNION CARBIDE CORPORATION CHRONOLOGY OF PVC ACTIVITIES	27
ENGINEERING OPINION	
ODORS	32
PNEUMOCONIOSIS	34
ESTIMATES OF EXPOSURE TO VINYL CHLORIDE MONOMER	43
LITERATURE CITED	53
ATTACHMENTS	62

POLYVINYL CHLORIDE - VINYL CHLORIDE MONOMER

LITERATURE SURVEY AND ENGINEERING OPINIONS

SUMMARY AND CONCLUSIONS

LITERATURE SURVEY

Polyvinyl Chloride Industry and Processes

1. Union Carbide Corporation solution polymerized PVC resins which were all bagged had negligible potential for vinyl chloride emission.
2. Union Carbide Corporation process bulk resins which are generally sold in bags had little potential for vinyl chloride emission.
3. Union Carbide Corporation suspension homo- and co-polymer PVC resins which are generally handled in bulk contained appreciable residual vinyl chloride monomer. The operations carried out by ATC or OTD were transferring from bulk shipboard containers to bulk hopper trucks, bulk hopper rail cars and to multi-wallpaper bags on pallets. Vinyl chloride emissions from PVC resins at ambient temperatures are very low. Resins were not heated in the ATC operations.

Vinyl Chloride Toxicology

1. Based on the information available to it, the VC-PVC industry and Union Carbide Corporation acted in a responsible manner. In retrospect, speedier action might have expedited the report of the carcinogenic properties of vinyl chloride by no more than one year.
2. Vinyl chloride exposure has been connected with angiosarcoma in humans. No other type of neoplasm has been identified as specifically caused by human vinyl chloride exposure.

Vinyl Chloride Toxicology (continued)

3. Vinyl chloride exposure has been related to acroosteolysis in humans.

Polyvinyl Chloride Toxicity

1. Polyvinyl chloride or its usual copolymers display a very low order of toxicity.
2. Polyvinyl chloride has caused no identified excessive mortalities in the PVC fabricating industry and by inference would show similar properties at ATC.
3. Polyvinyl chloride in the form of respirable dust has been related to pneumoconiosis in workers and laboratory animals.

Union Carbide Corporation Chronology of PVC Activities

1. Union Carbide Corporation initiated or participated in actions to study and control the hazards related to vinyl chloride and polyvinyl chloride exposure as a responsible corporate citizen.
2. There is no evidence that Union Carbide Corporation failed to act upon knowledge it had or obtained, concealed its knowledge from others or acted in a negligent manner with regard to the hazards relating to vinyl chloride or polyvinyl chloride exposure.

ENGINEERING OPINIONS

Odors

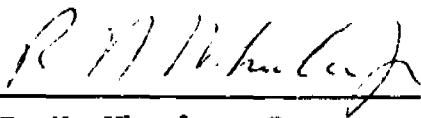
Based on the material data and the specific complaints regarding the vinyl chloride resins, the nasal and bronchial irritation noted by the plaintiffs was due to trace amounts of vinyl acetate and acetic acid given off by resins. Additionally, hydrogen chloride could be emitted during maintenance operations or equipment failures.

Pneumoconiosis

1. Research has not clearly shown that there is a relationship between pneumoconiosis and PVC resin dust.
2. The ingestion of vinyl chloride monomer via inhalation of respirable PVC dusts is negligible since such dust contains less than one ppm by weight of vinyl chloride monomer.
3. The Union Carbide Corporation PVC resins handled and bagged by ATC contained very little, if any, respirable dust.
4. Local ventilation was provided at ATC for operations that were dusty.
5. Exposure to the PVC resin handled by ATC for Union Carbide Corporation is unlikely to cause chronic human disability via inhalation.

Estimates of Exposure to Vinyl Chloride Monomer

Based on the material analysis, of potential employee exposure and the work done by Gollob Analytical Service Corporation, the ATC employees at Perth Amboy were not exposed to toxic concentrations of vinyl chloride and were only rarely exposed in excess of the permissible exposure limits set by OSHA, October 21, 1974.


R. N. Wheeler, Jr.

RNWheeler, Jr./ws

UCC
067890

R. N. WHEELER, JR.

Environmental Protection and Occupational Health,
Project Manager

Union Carbide Corporation
P. O. Box 8004
South Charleston, West Virginia 25303

Coordinator with Union Carbide Corporation on environmental protection, health problems, and regulations. Represents Union Carbide Corporation to OSHA, EPA and NIOSH on such problems.

Graduate Chemical Engineer from Virginia Polytechnic Institute in 1943. Employed by Union Carbide Corporation in various capacities for thirty-five years. Experienced in manufacture of polyvinyl chloride, polyvinyl acetate, polyvinyl butyral, polyvinyl alkyl ethers, polyethylene oxide and related monomers.

ORGANIZATION ACTIVITIES

- I. Organization Resources Counselors
Washington, D. C.
 - 1. Vinyl Chloride Task Force
 - 2. PVC Fabricating Industry, Epidemiology Steering Committee
- II. Society of Plastics Industries VC-PVC Producers Association
New York, New York
 - 1. VCM-PVC-EPA Technical Committee
 - 2. Health Study Committee
- III. Manufacturing Chemists Association
Washington, D. C.
 - 1. Vinyl Chloride Technical Committee
 - a. VCM Research Program Coordinator
 - b. VCM Safety Data Sheet Subcommittee
 - 2. Styrene Toxicity Technical Panel

UCC
067891

- IV. Society of the Plastics Industry
New York, New York
 - 1. Acrylonitrile Copolymer Group
 - 2. Toxicology Committee
 - a. Vinyls Subcommittee
 - b. Vinyl Acetate Study Task Force
- V. Organization Resources Counselors
Washington, D. C.
 - 1. Acrylonitrile Task Force
 - 2. OSHA Generic Carcinogen Proposal Task Force

PUBLICATIONS

- 1. Wheeler, R. N., Jr. and Sutherland, M. E.: "Control of Vinyl Chloride Emissions in Distribution Operations", American Institute of Chemical Engineers, Houston Meeting - 1974.
- 2. Wheeler, R. N., Jr.: "Specialty Vinyl Chloride Resin Processes, Effects of Governmental Regulations", American Chemical Society, San Francisco Meeting - 1976.
- 3. Wheeler, R. N., Jr.: "Automatic Monitoring Systems for Vinyl Chloride", American Institute of Chemical Engineers, New York Meeting - 1977.
- 4. Wheeler, R. N., Jr., Barr, J. T., Laundrie, R. W. and Snyder, P. J.: "Chemical Safety Data Sheet SD-56 Vinyl Chloride", Manufacturing Chemists Association, Revised April 1, 1977 (in process of publication).

POLYVINYL CHLORIDE INDUSTRY AND PROCESSES

Polyvinyl chloride is a ubiquitous part of our environment today in that it appears in clothing, upholstery, wire insulation, food containers, coatings, phonograph records, flooring and an infinite variety of other items. Despite this wide application, there is little public understanding of the size and the complexity of the industry. In order to discuss the health related problems of the industry, it is necessary to review the technology of the PVC industry and to identify the sources of problems and factors contributing to them.

The polyvinyl chloride industry consists of five major functions: 61/

- (1) Manufacture of vinyl chloride monomer.
- (2) Transport of the vinyl chloride monomer to the user.
- (3) Manufacture of synthetic resins from vinyl chloride and other monomers (PVC).
- (4) Transport of the resins (PVC) from the manufacturing plant to the user.
- (5) Manufacture of finished and semi-finished plastic articles for the ultimate use of the consumer.

1. Manufacture of vinyl chloride monomer is carried out in large chemical plants with capacities of 200,000 tons or more per year where ethylene, a gas, is treated with chlorine, oxygen and hydrogen chloride yielding ethylene dichloride (1,2, dichloroethane), a liquid. This ethylene dichloride is cracked (dehydrochlorinated) in a furnace yielding vinyl chloride monomer and hydrogen chloride (both gases). These are collected, separated and purified. The hydrogen chloride is recycled. The vinyl chloride monomer is liquified with pressure and refrigeration. Most of these plants in the United States are located on the Gulf Coast where easy access to salt and to hydrocarbon feed stocks exists. The plants themselves are built in the open and all

operations are carried out under pressure in closed equipment and pipelines thus employee exposure to vinyl chloride monomer is minimal. This, in turn, explains why there have been no reported health problems associated with the manufacture of vinyl chloride.

2. Transport of the vinyl chloride monomer to the resin manufacturing facility is carried out via pipeline if the plant is nearby or via tankers, barges, rail tank cars or tank trucks if the plant is remote from the vinyl chloride manufacturing plant. The primary modes of transport are rail tank cars and pipelines carrying liquified vinyl chloride under pressure. Aside from fires and explosions due to rupture of the transport vessel and subsequent ignition of the vinyl chloride, there have been no health problems associated with this function.

3. Manufacture of synthetic resins from vinyl chloride and other monomers involves reacting these monomers in agitated pressure vessels in the presence of catalysts and converting these liquids and/or gases to solid resins. A considerable amount of heat is generated by the reaction and this is removed by cooling the vessel. As the monomer is consumed during the reaction, the rate of reaction slows down and the heat transfer surfaces of the vessel become coated with solid resin; thus, after some optimum reaction time, the unconverted monomer is removed from the reacting mass by heat and vacuum and the resin (PVC) is recovered as a dried white powder or as a liquid latex of solution. There are presently four main processes for the manufacture of PVC resins:

- (1) Suspension polymerization
- (2) Emulsion polymerization
- (3) Bulk polymerization
- (4) Solution polymerization

These processes along with pertinent variations will be discussed later with particular reference to the resin properties obtained by each process. For the present, one should consider

the facts that a solid is produced in the reactor and a considerable amount of heat is generated by the reaction. These facts require that after one or more batches of resin are made in a reactor, the solid resin adhering to the walls and in the openings be removed manually by scraping, washing with high pressure water or by washing with a solvent to dissolve the resin scrap; i.e., reactor cleaning. It is in the reactor area of the process and more particularly, the cleaning operating with worker exposure to vinyl chloride concentrations of 1,000 ppm or more that workers have contracted acroosteolysis 82/, 94/ and angiosarcoma. 70/

4. Transportation of the resins (PVC) to the manufacturer of finished and semi-finished plastic articles varies with the properties of the resin and the amount consumed by the plastic manufacturer. A plant manufacturing vinyl film and sheeting using millions of pounds per year of suspension or bulk process PVC resins would be shipped rail hopper cars or hopper trucks of raw material. A plant manufacturing vinyl coated cloth for furniture would be shipped PVC dispersion resins in 40-pound paper bags because dispersion resins are very difficult to handle in bulk. A coating manufacturer using costly specialty PVC resins would be shipped material in 50-pound paper bags. Resin delivery in bags is more costly than in hopper trucks. Hopper trucks are more costly than rail hopper cars and rail hopper cars are more costly than containerized water transportation. As a result of transportation economics, large resin producers establish distribution centers such as the Perth Amboy facilities of ATC to minimize the cost of product delivery to the ultimate consumer. While there is some potential for exposure to vinyl chloride monomer given off by the PVC resin, there have been no health problems identified in this area.

5. The manufacture of finished and semi-finished plastic articles from PVC resins is a relatively simple operation, but many different procedures are used depending upon the type of PVC resin used and the properties desired in the final product. Most of the suspension and bulk polymerized resins are mixed with plasticizers (low volatile

organic esters such as dioctyl phthalate), stabilizers (such as organic compounds of tin, lead, cadmium or calcium), antioxidants (such as complex phenols), pigments (such as carbon black or chrome yellow) and processing aids (such as soaps or fatty acids) and heated to form a compound which is molded, extruded or calendered. It is this mixing operation which drives off most of any contained residual vinyl chloride monomer. This is also the point where the employee is exposed to a wide variety of other potentially hazardous materials. Dispersion (emulsion) PVC resins are mixed with similar materials to make a thick liquid compound which needs only to be heated to form a plastic article. Solution polymerized resins are dissolved in solvents and applied as coatings. Work related health problems have occurred in plastic manufacturing plants, however, unless they are specifically identified with exposure to vinyl chloride monomer via bioassay they are probably the result of exposure to other materials since monitoring studies have shown very low vinyl chloride levels or none at all in the workspace air. 74/, 14/

The PVC resin processes and the PVC products from these processes are reviewed as follows: 61/

(a) Suspension polymerization is the major process used for the manufacture of PVC resins. It involves the charging of two parts water and one part vinyl chloride monomer to an agitated reactor along with catalyst and suspending agents such as polyvinyl alcohol. The mass is reacted at 50 to 60°C until about 90% of the contained monomer is converted to resin. The resin water mixture is heated and subjected to vacuum until the unconverted monomer is substantially removed. The resin is then removed from the water and dried in rotary or flash driers by exposure to heated air. Process costs are low and a wide range of products can be produced. The resin particle size can be varied from 50 to 150 microns with varying degrees of porosity and varying composition if other monomers are used. The residual unconverted vinyl chloride (RVCM) is removed with difficulty due to the large

particle size and the low degree porosity. Commercial PVC by this process contained approximately 1,000 ppm by weight RVCM prior to 1974. The resin is widely used in bulk for its low cost, ease of handling and freedom from dust.

(b) Emulsion polymerization is the second most widely used process as well as one of the oldest for manufacture of PVC resin. Again, about two parts water and one part monomer (VCM) are charged to an agitated reactor along with surfactant (soap) so that with agitation a monomer water emulsion is formed. Catalyst in the form of a water soluble salt or organic peroxide is added and the mass reacted at 40 to 60°C. until 70 to 90% of the contained monomer is converted to resin. Resin particles formed are very small (0.2 to 2 microns) and a relatively stable synthetic latex is formed. The reacted latex is stripped of its unconverted monomer to a level of 5,000 ppm by weight on a resin basis prior to 1974. The latex product could be directly formulated into coating materials, could be coagulated and the resin recovered by dewatering and drying or could be spray dried directly to recover the resin. In Europe, the resin is used for all types of fabricating technics while in the United States the latex or the spray dried resin is used for coating or for fabrication via plastisol technics. The resin is characterized by its small particle size (1 to 10 microns), high content of soap (1 to 2%) and its very low residual unconverted vinyl chloride content. The very small particle size coupled with spray drying and grinding cause almost complete removal of the RVCM (<1 ppm by weight). Dustiness, surfactant odor and content, poor bulk handling properties and cost have limited the application of the resin from the process to manufacture of specialty items from resins in bags.

(c) Bulk polymerization was developed by Union Carbide Corporation in 1937 as a continuous one stage reaction. Vinyl chloride polymer is insoluble in vinyl chloride monomer thus on partial polymerization of the monomer, a resin monomer slurry is formed. This resin monomer slurry is fed to a tank of hot water where the monomer is boiled out leaving a resin-water slurry. The contained resin is dewatered and dried in a flash drier. The resin produced by this process is free of suspending aids and surfactant, is very porous, has a particle size of 60 microns and contains less than 10 ppm RVCM. With aeration, the RVCM content of the resin falls rapidly to zero. Manufacturing costs are high, limiting resin use to specialty items. Distribution entails both bulk and bag handling.

In Europe, the bulk process developed by Pechiney St. Gobain, is a two-step high conversion batch process involving no water. The first polymerization stage converts 10% of the monomer to polymer. This mixture is then charged to a second polymerizer where the reaction is carried to 60 to 90% monomer conversion. Resin in the second reactor is subjected to heat and vacuum to remove unconverted monomer. Resin particles are 50 to 150 microns and very porous. Prior to 1974, this process resin was very high in RVCM (or 5,000 ppm). Costs are low and the bulk product is competitive with suspension resin. A number of United States producers use this process.

Neither the UCC process nor the PSG process are amenable to producing resins containing appreciable quantities of comonomer.

(d) Solution polymerization is a process unique to Union Carbide Corporation. It was commercialized in 1933. Vinyl chloride monomer, comonomer, solvent and catalyst are fed to a continuous reactor system. The

polymer formed is soluble in the reacting mass so that the reactor product is a viscous resin-solvent solution. This solution is distilled to remove the unconverted vinyl chloride monomer and the resin product recovered by treating the resin solution with water and drying the product. The resin particle is very porous, is always a copolymer, is free of soaps and suspending agents, has a particle size of 75 microns and contains less than 1 ppm RVCM. Manufacturing costs are relatively high and the product finds its greatest use as a coating material; i.e., paints and lacquers where it is used in bag quantities.

The emission of unconverted vinyl chloride from PVC resins during the course of normal mechanical handling and storage is an important consideration in view of the chronic toxicity of vinyl chloride monomer and the exposure of workers to PVC. Vinyl chloride monomer is not soluble in pure polyvinyl chloride thus it can only be adsorbed on the surface or mechanically entrapped in the resin particle. The larger the resin particle and the less porous it is, the more slowly the contained vinyl chloride is emitted. Studies of monomer stripping at ambient temperatures have shown that the rate of emission is very slow and appears to reach a steady state concentration; i.e., a sample of suspension polymerized resin containing 50 ppm by weight RVCM emitted no VCM at 20°F. Thus, it could be said that regardless of the RVCM concentration in a PVC resin at ambient temperature, the contained vinyl chloride is emitted to the air slowly, if at all. This rate is further slowed by the presence of a comonomer which tends to solubilize the RVCM. 62/, 13/

In 1974 and 1975, Union Carbide Corporation conducted extensive studies of vinyl chloride emission from its resins. In 1975, it was able to establish to OSHA's satisfaction that no hazard from vinyl chloride exposure

existed from its solution and emulsion polymerized resins.
77/, 93/

Emission studies of its suspension PVC resins showed the average RVCM content on shipment from the plant was 860 ppm. When these resins arrived at Perth Amboy and were sampled, the average RVCM content was 542 ppm showing a loss of 37% per month when handled in bulk. Storage of these resins in bags showed an 85% reduction in RVCM per week storage. The bulk process resins were not studied, however, assuming the same rate as suspension resin in bulk, they averaged 15 ppm RVCM on shipment and 9 ppm on bagging at Perth Amboy. The normal period from production to bagging was 32 days, including six days prior to ship loading, seven days on the ship and nineteen days in storage at Perth Amboy prior to bagging. Resin inventory at Perth Amboy averaged 1.25 months or 47.5 days. 2/, 3/, 4/, 5/

Summarizing the salient points of the discussion, the following are to be noted:

Resins handled by ATC during the period involved in litigation are:

(a) Union Carbide Corporation solution polymerized PVC resins, which were all bagged, had negligible potential for vinyl chloride emission.

(b) Union Carbide Corporation process bulk resins, which are generally sold in bags, had little potential for vinyl chloride emission.

(c) Union Carbide Corporation suspension homo- and co-polymer PVC resins, which are generally handled in bulk, contained appreciable residual vinyl chloride monomer.

The operations carried out by ATC or OTD were transferring resins from bulk shipboard containers to bulk hopper rail cars and to multi-wallpaper bags on pallets.

Vinyl chloride emissions from PVC resins at ambient temperatures are very low. Resins were not heated in the ATC operations.

VINYL CHLORIDE TOXICOLOGY

Vinyl chloride despite its reported synthesis in 1835 did not become an article of commerce until 1928 when Union Carbide Corporation started research and pilot scale manufacture in 1928. This work was followed by commercial manufacture of vinyl chloride polymers in the United States by Union Carbide Corporation and in Germany during 1933. From this early start, the VC-PVC industry has grown to its present day level as one of the largest of the plastics industries.

The earliest study of the toxicity of vinyl chloride was that reported by F. A. Patty. 51/ He found that human exposure to vinyl chloride at 2.5% for 3 minutes produced dizziness and disorientation and that guinea pigs exposed for eight hours at 0.5% showed no effect. Guinea pigs exposed to 2.5% for eight hours died. Patty also cited work by Schaumann 63/ that noted no hepatic or renal lesions occurred in mice or rats exposed at 0.5% for four hours daily over five to eight days. The apparent low toxicity of vinyl chloride as compared to chloroform, a widely used anesthetic at that time, led to its consideration as an anesthetic by Lehman and Flury 31/ after Peoples and Leak 53/ had confirmed its apparent low toxicity. Von Oettingen in 1933 cautioned against its use as an anesthetic. 88/ Later studies by Oster and Carr in 1947-1949 showed vinyl chloride used as an anesthetic caused

severe cardiac arrhythmia. 48/ As late as 1955, vinyl chloride was still under consideration as an anesthetic. Von Oettingen cautioned as regards to its effect on the circulatory system. 87/

Many researchers cite the report of Tribukh in 1949 74/ that 73 workers in a PVC compounding and calendering plant showed hepatitis, hypertension, anemia, gastritis and skin lesions as evidence of the highly toxic nature of vinyl chloride retained in the PVC. The compounding was done at temperatures of 160°C. using diphenylene* and chlorinated naphthalene as well as various pigments and stabilizers. Chlorinated naphthalene is well known as a severe liver toxicant and such symptoms have been observed in others exposed to such materials. Between 1953 and 1959, other problems related to additives were noted but there was no reported liver disease. The availability of better plasticizers which were less toxic no doubt contributed to the lack of further problems. 30/

The American Congress of Governmental Industrial Hygienists set the threshold limit value of vinyl chloride at 500 ppm for an eight-hour exposure based on human experience and animal data. This was believed to be a safe level for worker exposure. 70/

Mastromatteo in 1960, following the accidental death of two vinyl chloride workers, exposed rats, mice and guinea pigs to 10%, 20% and 30% vinyl chloride for 30 minutes. 37/ At the higher concentrations, the animals showed narcosis and respiratory failure. All animals showed lung, hepatic and renal congestion.

Prodan reported LD₅₀ of mice, rats, guinea pigs and rabbits based on two-hour exposure. 57/, 58/ The vinyl chloride concentration varied from 12% for mice and 24% for rabbits. Surviving animals rapidly regained their normal appearance on cessation of exposure. A 90-day guinea pig exposure at 10% vinyl chloride resulted in liver and kidney lesions and lung fibrosis.

Torkleson, et al, in 1961, initiated the first studies of vinyl chloride chronic toxicity. They reported micropathological changes in rats after 4.5 months at the then acceptable 500 ppm exposure level. Their minimum level was 100 ppm where rat livers showed only a slight increase in weight. No effect was noted in

* Diphenylene is not a known material and may be an erroneous reference to chlorinated diphenyl which was used as a plasticizer and was also a liver toxin. 30/

the other species (rabbits, dogs and guinea pigs) at this level. The researchers suggested a maximum worker exposure of 100 ppm with an eight-hour TWA of 50 ppm. 73/

Lester, et al, in 1962 reported that repeated exposure of rats to 2% and 5% vinyl chloride concentrations cause no significant problems. They noted liver changes but decided they were not significant. Lester recommended a 500 ppm Threshold Limit Value. 32/ The ACGIH chose to give more weight to Lester's work than to Torkleson's and cut the recommended vinyl chloride TLV from an eight-hour TWA to a 500 ppm ceiling value. 70/ This value was subsequently written into the Occupational Safety and Health Act of 1970 as a Standard.

Suciu, et al, in 1963 have been widely cited as sounding the alarm for vinyl chloride chronic toxicity but they reported on a cohort of vinyl chloride workers exposed for one year at a reported average concentration of 900 ppm. 64/ Based on prior knowledge, the worker dizziness and other symptoms were not unexpected. A later report in 1967 indicated that the acute exposure symptoms reported earlier were disappearing in an estimated exposure environment of 38 ppm.

The year 1966 marked the observed onset of acroosteolysis and Reynaud's syndrome. Cordier, et al, reported skin lesions and acroosteolysis in reactor cleaners. 82/ B. F. Goodrich's Dr. R. H. Wilson reported his observations to the Manufacturing Chemists Association. 94/ He had noted evidence of acroosteolysis since 1962. The Manufacturing Chemists Association in a meeting at Cleveland, late in 1966, reviewed the apparently new occupational disease with VC-PVC industry representatives. These industry representatives agreed to cooperatively sponsor an investigation of the purported disease and the University of Michigan Institute of Industrial Health was employed to conduct an epidemiological study. 82/

After a wide-ranging investigation, the University of Michigan reported to MCA in 1969 that identification of the causative agent or agents was not conclusive; however, it would seem in order to reduce the severity of VC exposure by reactor cleaners and personnel in the reactor area. The value of 50 ppm recommended by

Torkleson, et al, was endorsed. They also recommended that efforts to produce the disease in laboratory animals be supported by industry.

Dr. P. L. Viola, an industrial physician, 86/, 85/ was employed by Solvay, et cie, to study the acroosteolysis problem. In 1970 he reported that he had exposed 25 rats to 30,000 ppm vinyl chloride for twelve months. Thirteen died from cardio respiratory complications and two from abdominal hemorrhages. He noted lesions of the bone and connective tissue similar to acroosteolysis. 85/ He also noted degenerative processes of the parenthyma in the brain, liver and kidneys. Later that year at the Tenth International Cancer Congress in Houston, Texas, he reported that 26 rats exposed to 30,000 ppm vinyl chloride developed skin tumors (65%) and that 26% of those affected developed respiratory tract tumors. 83/, 84/ The epidermoid tumors were located in the vicinity of the ears. He stressed that the results applied only to rats and no implications to human pathology can be extrapolated. Later that year in an apparently unpublished paper, he noted that a maximum allowable concentration of 500 ppm for worker exposure gave an adequate margin of safety. 86/

The Manufacturing Chemists Association invited Dr. Viola to the United States to discuss his studies in greater detail. On May 5 and 6 of 1971, he reviewed his work in detail and referred to other research work underway in general terms. He claimed to have seen tumors in rats exposed to 5,000 ppm but reaffirmed his belief that 500 ppm TLV provided an adequate margin of employee safety from tumor induction.

In November 1971 the Manufacturing Chemists Association convened a meeting of VC-PVC industry representatives to discuss the apparent vinyl chloride problem revealed by Dr. Viola. Dr. Le Fevre of Solvay, et cie, discussed European observations and stated generally that further European vinyl chloride toxicity studies were underway. Dr. Viola's work was discussed and the appearance of tumors in rats exposed to 5,000 ppm vinyl chloride confirmed. 91/ The group formed an Ad Hoc Committee under

R. N. Wheeler, chairman, to plan a cooperative research program.

The MCA Committee proposed a program involving a three level three species chronic inhalation toxicity study, a study of vinyl chloride metabolism and an industry-wide epidemiological study of mortality at a cost of \$350,000.

Kramer and Mutchler in 1972 reported on a cohort of Dow employees exposed to vinyl chloride. 27/ They reported that life-time exposure to 300 ppm vinyl chloride may result in slight changes in physiological and clinical parameters. The possibility of impairment of liver function must be considered. The ACGIH as a result of the Kramer and Mutchler's report lowered its recommended TLV for workers to 200 ppm 8-hour TWA. 70/

Sponsorship of the Manufacturing Chemists Association study did not meet the financial objective of \$350,000 so the decision was made to do the long-term inhalation study and postpone the other studies pending collection of additional money. An MCA representative was sent to Europe to determine the nature of their program. He returned without any new information.

During 1973 MCA, having collected sufficient sponsorship, contracted for the long-term inhalation toxicity, the metabolism and the epidemiological studies. The inhalation study was late in getting started due to relocation of the contractor and his failure to provide proper facilities.

An MCA group composed of corporation executives established liaison with the European consortium sponsoring vinyl chloride research under Dr. C. Maltoni. Informal reports on Dr. Maltoni's work revealed that the rats under test had shown tumors at the 250 ppm exposure level. These results and the substance of the planned research were discussed with Dr. M. Key and Associates at NIOSH in mid-summer. 92/

On January 23, 1974, B. F. Goodrich Company reported that five Louisville, Kentucky PVC plant employees had died of angiosarcoma, a rare form of cancer. The preliminary reports on the European research confirmed that this cancer may be related to vinyl chloride exposure. 35/

A preliminary report from Dr. C. Maltoni stated that rats exposed to long-term inhalation of vinyl chloride showed zymbal gland carcinomas, nephroblastomas, angiosarcomas, angiomas, hepatomas, brain neuroblastomas and skin carcinomas. Mice developed angiosarcomas, angiomas, lung adenomas, mammary carcinomas and skin tumors. Hamsters developed angiosarcomas, skin tumors and lymphomas. Common to all species were angiosarcomas. Nephroblastomas were common to rats and mice but are not known to appear in humans. Angiomas were also common to rats and mice. 64/

The appearance of angiosarcomas in the mice used in the MCA test confirmed Maltoni's work. 64/

Juhe, Lange, et al, reporting in late 1973 on the vinyl chloride disease notes that at exposures of 60 to 1,000 ppm, they had seen fibrotic livers via laparotomy and seen possible lung fibrosis in employees of PVC plants. 26/

Skin absorption by vinyl chloride was studied by Hefner, et al, of Dow Chemical. A Rhesus monkey exposed to 7,000 ppm vinyl chloride via skin for two hours was estimated to be equal to the inhalation by a man of 0.185 ppm for eight hours. 95/

With the connection between animal bioassay and human disease via angiosarcoma concern then shifted to the nature and extent of vinyl chloride caused neoplasms in the work force and in the people living in the area of VC-PVC facilities.

Waxweiler, et al, in 1976 reported on a survey of four PVC plants whose workers were exposed to high levels of vinyl chloride. Based on 136 deaths, they reported excesses of brain, central nervous system, respiratory, liver and lymphatic neoplasms. 90/

Gamble, et al, in 1976 reported that vinyl chloride workers did not show chronic respiratory effects related to their exposure but smoking caused acute reductions. 19/

A study of Union Carbide Corporation workers at the South Charleston, West Virginia Plant showed excesses of angiosarcomas. All malignancies had a standardized mortality ratio of 87.7 compared to 111 for all other PVC-VC industry workers. Only leukemia and lymphomas were in excess but only eight cases were reported.

covered 1,314 workers and 211 deaths. Forty-nine percent of the study group had twenty years or more employment in vinyl chloride work. 16/

Fox and Collier in 1977 reported that an epidemiological study of vinyl chloride workers in Great Britain showed excess liver cancers but no evidence that vinyl chloride was a general carcinogen. The study covered 7,714 workers and 409 deaths. 18/

The Manufacturing Chemists Association industry-wide VC-PVC worker study on 10,173 workers and 669 deaths showed excessive standardized mortality ratios for brain tumors, miscellaneous cancers and leukemia but the numbers of cases were small. 72/, 96/ The conclusion of this study was that there maybe some support for the hypothesis that vinyl chloride is a general carcinogen. Considering that the previously noted Union Carbide Corporation study made up part of this study and the Union Carbide Corporation workers made up a major portion of those exposed for a long period, the conclusions from the comparison of the two studies are not consistent.

The PVC fabricating industry was the subject of two studies. One by Organization Resources Counselors in the United States reported no angiosarcomas in 4,592 deaths. 14/, 45/, 46/ Proportional mortality ratios showed excess deaths in digestive, respiratory and all other neoplasms as well as deaths from circulatory disease. There was a significant depression in deaths from all other causes indicating a lack of complete data. Dr. G. M. Paddle in a letter to R. N. Wheeler observed that the apparent excesses in PMR's *-1 were a common feature of industrial employee studies. 50/

Baxter and Fox in 1976 reported that there was no excess risk of lung, brain or liver cancer in PVC fabricators in Great Britain. In the period 1963-1973, they found only one case of angiosarcoma relatable to VCM out of an average of four cases per year for the period. They noted that most of the four per year were misdiagnosed and there were actually only fourteen cases of AS *-2 in ten years. 9/, 10/

Brady, et al, in 1977 reported on twenty-six New York State angiosarcoma cases. Three had had contact with vinyl chloride in

*-1 Proportional Mortality Ratio

*-2 Angiosarcoma

UCC
087907

their work and five lived within 4,500 feet of a VCM or PVC plant. They concluded that since the rate of AS cases in New York was 0.25 per million of population versus the national average rate of 0.14, there must be other causes of AS than Thorotrast, arsenicals and vinyl chloride. 97/

MacMahon 34/ and Downs, 15/ et al, in critical reviews of the reports on mutagenesis, fetal anomalies or fetal death reported while there was some evidence of VC caused chromosome aberrations there is no evidence of VC caused fetal wastage or birth defects. 89/, 24/

A tabulation of VC-PVC related angiosarcoma cases is as follows: Year Company and Location

1961	Goodyear, Niagara Falls, New York
1964	B. F. Goodrich, Louisville, Kentucky
1968	Union Carbide, South Charleston, West Virginia
	Goodyear, Niagara Falls, New York
	B. F. Goodrich, Louisville, Kentucky
1969	Firestone Plastics, Pottstown, Pennsylvania
	B. F. Goodrich, Louisville, Kentucky
1970	Goodyear, Niagara Falls, New York
1971	B. F. Goodrich, Louisville, Kentucky
1973	B. F. Goodrich, Louisville, Kentucky
	B. F. Goodrich, Louisville, Kentucky
1974	Union Carbide, South Charleston, West Virginia
1975	B. F. Goodrich, Louisville, Kentucky
	B. F. Goodrich, Louisville, Kentucky
	B. F. Goodrich, Louisville, Kentucky
1976	Union Carbide, South Charleston, West Virginia
	B. F. Goodrich, Louisville, Kentucky
	Great American Plastics, Massachusetts
1977	Union Carbide, South Charleston, West Virginia
	Union Carbide, South Charleston, West Virginia

From this tabulation of angiosarcoma cases, it is evident that only B. F. Goodrich or Goodyear could have concluded that

they were experiencing an excess of angiosarcoma cases and not subject to random cases prior to 1974. Approximately 26 cases of angiosarcoma are diagnosed each year in the United States from all causes.

From this survey of vinyl chloride toxicology, the following conclusions may be drawn:

1. Based on the information available to it the VC-PVC industry and Union Carbide Corporation acted in a responsible manner. In retrospect, speedier action might have expedited the report of the carcinogenic properties of vinyl chloride by no more than one year.
2. Vinyl chloride exposure has been connected with angiosarcoma in humans. No other type of neoplasm has been identified as specifically caused by human vinyl chloride exposure.
3. Vinyl chloride exposure has been related to acroosteolysis in humans.

POLYVINYL CHLORIDE TOXICOLOGY

Toxicity of polyvinyl chloride was studied and reported more or less simultaneously by Smyth, H. F., Jr., and Weil, C. S. at Mellon Institute of Research 67/ and by Harvard University for B. F. Goodrich Company. 21/ In both cases, PVC resin was fed to animals for two years and in both cases no effect attributable to the resin was noted. One resin (VYNU) was an emulsion polymerized vinyl acetate copolymer and the other was vinylidene chloride copolymer suspension polymerized. Both studies involved feeding 75 micron resin over a long period so certainly the particles

were persorbed as noted by Volkheimer. 64/ There was no analysis of the resins for residual vinyl chloride monomer content.

In 1955, Oppenhiemer, et al, reported that PVC was carcinogenic when imbedded in rat tissue as solid discs. 43/ This study resulted in PVC being identified in later literature as a carcinogen. The effect noted resulted from implantation of a solid foreign body. Implantation of perforated discs did not result in cancers. 44/ Recently, United States dimes (Roosevelt) were identified as carcingogenic on implantation.

In 1953, Boettner, et al, 98/ and in 1974, K. L. Paciorek, et al, 99/ reported that the products of combustion and thermal degradation of PVC were largely hydrogen chloride. Very small amounts of vinyl chloride were produced by thermal oxidation of PVC but the presence of hydrogen chloride would make human inhalation of the vinyl chloride produced problematical. Hydrogen chloride even at concentrations of 10 or 20 ppm in air is extremely irritating thus any time equipment containing PVC is heated, such as is required in welding, large amounts of hydrogen chloride are generated.

In 1969, Popow reported on the effect of PVC dust on the respiratory system of the rat. 56/ The PVC resin involved had 92% of its particles less than 5 microns in diameter thus it was largely respirable. Suggestions in Nature (1975) 41/ and in the New Scientist (1975) 42/ that PVC dust was harmful, again appear to be based on PVC resin containing large amounts of respirable particles. Studies by Adams and Purchase in 1975, 1/ as well as Pigott, appear to refute any biological activity related to pure PVC dust. 55/ Any insoluble respirable dust however appears to lead to pneumoconiosis, a fact not entirely related to PVC dust.

Epidemiological studies of PVC fabricating workers done in the United States by Organization Resources Counselors 14/, 45/, 46/, 50/ and in Great Britain by Baxter and Fox 10/ show that there are no significant PVC problems in the PVC fabricating industry. These studies must be interpreted, however, as being not as precise the work done in the PVC producing industry; i.e., both the United States and the British study deal with proportional excesses in a disease category and may not reflect a real increase

or decrease in death rates over a comparative population.

The Amboy Terminaling Company employees were engaged in a transportation and distribution function and were not a part of the PVC producing industry or the PVC fabricating industry. In the first industry vinyl chloride monomer is the raw material and there is opportunity for excessive worker exposure. In the fabricating industry, the PVC resin is mixed with other materials and heated to fuse the mixture into a plastic mass thus driving off any contained vinyl chloride monomer and potentially exposing workers to excessive vinyl chloride concentrations. At ATC the workers were merely exposed to the resin powder at ambient temperature thus exposure to vinyl chloride would not approach the levels found in the PVC producing industry and probably be much less than or equal to the PVC fabricating industry.

A summary of salient points is as follows:

1. Polyvinyl chloride or its usual copolymers display a very low order of toxicity.
2. Polyvinyl chloride has caused no identified excessive mortalities in the PVC fabricating industry and by inference would show similar properties at ATC.
3. Polyvinyl chloride in the form of respirable dust has been related to pneumoconiosis in some workers and laboratory animals.

UNION CARBIDE CORPORATION CHRONOLOGY OF PVC ACTIVITIES

- 1928 Started pilot plant production of vinyl chloride monomer and development of resins made from vinyl chloride - South Charleston, West Virginia.
- 1933 Commercial production of vinyl chloride by alkali dehydrochlorination of ethylene dichloride and the manufacture of vinyl chloride resins by the solution polymerization process - South Charleston, West Virginia.
- 1936 Expanded solution polymerization resin plant - South Charleston, West Virginia.
- 1937 Commercial production of vinyl chloride resins by continuous bulk polymerization - South Charleston, West Virginia.
- 1937 Chemical Hygiene Fellowship under Dr. H. F. Smyth established at Mellon Institute of Research at Pittsburgh, Pennsylvania.
- 1940 Studies of skin sensitization by PVC resins and compounds initiated at Mellon Institute of Research by Dr. H. F. Smyth.
- 1941 Initiated production of vinyl chloride by thermal dehydrochlorination of ethylene dichloride - South Charleston.
- 1942 Initiated production of vinyl chloride by synthesis process using acetylene and hydrogen chloride.
- 1943 Built new continuous bulk polymerization plant for vinyl resins - South Charleston, West Virginia.
- 1943 Built new emulsion polymerization plant for vinyl resins.
- 1945 Built new facility for manufacture of synthetic fiber from vinyl chloride and acrylonitrile - South Charleston, West Virginia.
- 1946 Built new vinyl chloride monomer plant, a solution polymerization resin plant and a continuous bulk polymerization resin plant in Texas City, Texas.
- 1947 Discontinued manufacture of vinyl chloride by caustic dehydrochlorination of ethylene dichloride and manufacture of bulk polymerized resins at South Charleston, West Virginia.
- 1947 Smyth, H. F., Jr. and Weil, C. S. reported on work at Mellon Institute "Chronic Oral Toxicity to Rats of Vinyl Chloride - Vinyl Acetate Copolymer", Toxicology and Applied Pharmacology 9, 501-504, 1966.

- 1954 Expanded solution polymerization plant for resins in Texas City.
- 1955 Initiated manufacture of vinyl resins by the suspension resin process at South Charleston, West Virginia.
- 1957 Converted the emulsion polymerization plant to a dispersion resin plant at South Charleston, West Virginia.
- 1960 Expanded the solution polymerization plant at Texas City, Texas.
- 1961 Perth Amboy Bulk Terminal started operation under OTD management.
- 1962 Initiated manufacture of vinyl resins by the suspension resin process at Texas City, Texas under technology license from Wacker Chemie.
- 1965 Expanded the suspension vinyl resin plant at Texas City, Texas, reduced capacity for bulk resins by fifty percent at Texas City, Texas and discontinued manufacture of suspension vinyl resins at South Charleston, West Virginia.
- 1966 Union Carbide Corporation representatives met with the Manufacturing Chemists Association to discuss reported acroosteolysis at the B. F. Goodrich Chemical Company plants. Support for an epidemiological study was pledged by Union Carbide Corporation.
- 1967 Discontinued manufacture of vinyl chloride at South Charleston, West Virginia.
- 1968 Expanded the suspension vinyl resin plant at Texas City, Texas.
- 1968 An employee of Union Carbide Corporation at South Charleston, West Virginia plant dispersion PVC unit died of liver cancer, subsequently diagnosed as angiosarcoma of the liver, a rare type of cancer.
- 1969 Discontinued manufacture of vinyl chloride at Texas City, Texas.
- 1969 The epidemiological investigation of the PVC industry co-sponsored through MCA by Union Carbide Corporation showed no confirmed cases of acroosteolysis in any Union Carbide Corporation polymerization plants.
- 1971 Union Carbide Corporation co-sponsored a visit to the U.S.A. by Dr. P. L. Viola to discuss his reported oncogenic effect of vinyl chloride exposure to rats with the Manufacturing Chemists Association Occupational Health Committee.

- 1971 An Ad Hoc Planning Group for Vinyl Chloride Research was formed in MCA under the chairmanship of Mr. R. N. Wheeler, Jr., of Union Carbide Corporation. The Group recommended chronic toxicity studies, a metabolism study and an epidemiological study of the effects of vinyl chloride at an estimated cost of \$350,000.
- 1972 Union Carbide Corporation agreed to co-sponsor the chronic toxicity and other studies proposed by MCA.
- 1973 Union Carbide Corporation representative, Mr. R. N. Wheeler, along with representatives of MCA, Dow, Ethyl and Imperial Chemical Industries met with Dr. M. Key and Associates of the National Institute for Occupational Safety and Health to discuss the status of the possible vinyl chloride chronic toxicity problem.
- 1974 Union Carbide Corporation supplied the National Institute for Occupational Safety and Health and the Occupational Safety and Health Administration with written testimony and free access to its plants using vinyl chloride. It also reported promptly the second death of an employee from angiosarcoma of the liver.
- Union Carbide Corporation through Organization Resources Counselors co-sponsored an epidemiological study of the PVC fabricating industry as well as provided access to its records for the study.
- 1975 Union Carbide Corporation complied with the Occupational Safety and Health Administrations Standard for Vinyl Chloride.
- 1976 Discontinued the manufacture of vinyl chloride copolymer fiber and dispersion vinyl resins at South Charleston, West Virginia.

Conclusions:

1. Union Carbide Corporation initiated or participated in actions to study and control the hazards related to vinyl chloride and polyvinyl chloride exposure as a responsible corporate citizen.
2. There is no evidence that Union Carbide Corporation failed to act upon knowledge it had or obtained, concealed its knowledge from others or acted in a negligent manner with regard to the hazards relating to vinyl chloride or polyvinyl chloride exposure.

ENGINEERING OPINION

Maliko, Mazanowski, Wilkinson and Schaffer

versus

Union Carbide Corporation

R. N. Wheeler, Jr.

UCC
067915

Engineering opinion and data interpretation is necessary in three areas:

1. Odors reported by the plaintiffs.
2. Pneumoconiosis as a possible cause of emphysema reported by the plaintiffs.
3. Estimated levels of vinyl chloride exposure experienced by the plaintiffs.

ODORS

The operations at Amboy Terminaling Company involved handling vinyl chloride resins from three different processes, polystyrene, polyethylene and bisphenol A. Several of the plaintiffs have complained of nasal and bronchial irritation caused by the odors. In an effort to define the odors and the sources of irritation, the following are listed:

Vinyl Chloride Resins

Vinyl chloride polymers have no odor themselves but these do contain materials or may decompose into materials which have very distinctive odors:

- (a) Vinyl chloride monomer has a pleasant sweet odor 76/ detectable by odor at 2,000 ppm by volume. Prolonged inhalation tends to anesthetize a person's olfactory senses causing the odor threshold to rise as high as 4,000 ppm by volume.
- (b) Vinyl acetate monomer has a sour sharp irritating odor detectable by odor at 0.55 ppm by volume. 23/
- (c) Acetaldehyde has a sweet green plant odor detectable at 0.2 ppm by volume. This odor is usually present in conjunction with vinyl acetate. 23/
- (d) Acetic acid has a sour vinegary odor detectable by odor at 1.0 ppm. This odor is usually present in conjunction with vinyl acetate. 23/
- (e) Acetone has a pungent fruity odor detectable at 140 ppm by volume. This odor would occur only in solution polymerized vinyl chloride resins. 23/

(f) Isopropanol has a sharp musty odor detectable at 28 ppm by volume. This odor would occur only in solution polymerized vinyl chloride resins. 23/

When vinyl chloride resins are overheated, as in a fire, a welding operation or simply a hot surface such as a motor, they emit hydrogen chloride which is quite pungent and irritating. Hydrogen chloride odor is detectable at 10 ppm. 23/

Polyethylene Resins

Polyethylene resins have a characteristic paraffin wax odor. Some residual ethylene may be emitted but its pleasant olefinic odor detectable at 700 ppm by volume is largely masked by the resin's waxy odor.

Polystyrene Resins

Polystyrene resins have no characteristic odor except that of residual monomeric styrene. Styrene monomer has an aromatic naphthalene (mothballs) odor detectable at 0.05 ppm by volume. 23/

Bisphenol A

Bisphenol A has a phenolic medicine-like odor presumably from the small amount of phenol present in the material. Phenol odor is detectable at 0.05 ppm by volume. 23/

Based on the foregoing data and the specific complaints regarding the vinyl chloride resins, the nasal and bronchial irritation noted by the plaintiffs was due to trace amounts of vinyl acetate and acetic acid given off by resins. Additionally, hydrogen chloride could be emitted during maintenance operations or equipment failures.

PNEUMOCONIOSIS

Pneumoconiosis caused by PVC resin was initially studied by Jerzy Popow 56/ with rats exposed to resin particles most of which were less than 5 microns in diameter in 1969. Szendi 71/ reported on a human case of pneumoconiosis but failed to obtain data on the resin particle size except via lung tissue inspection. He identified oval or polygonal particles 15 to 25 microns in diameter in 1971. Nature 41/ in August, 1975 reported on the biological reactivity of PVC dust. This report didn't direct itself to pneumoconiosis and dealt with a water extractible fraction in the resin sample. The New Scientist reported in June, 1975 that PVC dust felled mice based on work by Frongia which has not been formally reported. 42/ Adams and Purchase 1/ and Pigott 55/ don't believe that PVC dust is a hazard with regard to pneumoconiosis.

The United States Atomic Energy Commission in 1961 defined "Respirable Dust" as that portion of the inhaled dust which penetrates to the nonciliated portions of the lung. 33/

Respirable dust was defined as follows:

<u>Particle Size</u>	<u>Percent Respirable</u>
10.0 microns	0
5.0 microns	25
3.5 microns	50
2.5 microns	75
2.0 microns	100

Insoluble respirable dust particles are carried past the body's normal air filtering system and are ultimately deposited in the alveoli of the lungs from thence there is no bodily mechanism to eject them. Collection of insoluble dusts in the

lung alveoli leads to reduced lung capacity such as is experienced in phenumoconiosis.

Physiological damage via polyvinyl chloride dusts may occur if the dusts carry a high concentration of vinyl chloride monomer into the lungs where the vinyl chloride monomer passes into the bloodstream or if an appreciable volume of dust deposits in the lung alveoli reducing the lung's capacity to exchange oxygen for carbon dioxide in the bloodstream.

Union Carbide Corporation and others have produced and marketed PVC dispersion resins for many years. These resins are produced by polymerizing vinyl chloride to a resin particle which is smaller than 1 micron in diameter. The water dispersion of these particles is stripped of unconverted monomer to a level of 2,000 to 6,000 ppm residual vinyl chloride monomer on a dry resin basis. This stripped latex is spray dried to remove the water. In the process of spray drying, the small resin particles are agglomerated into particles which vary from 1 micron to 20 microns. The dried resin is then ground to a median particle size of 2 microns. In the process of drying and grinding, the dispersion PVC resin essentially all of the contained vinyl chloride monomer is released to the air. Thus, when the resin is packaged in paper bags, the residual vinyl chloride monomer content is less than 1 ppm by weight. Dispersion PVC resin is almost always packaged in paper bags because resin bulk handling equipment used for all other PVC resins cannot be used for this material. Based on knowledge of dispersion resins, I believe that PVC particles which are in the respirable size range contain less than 1 ppm by weight residual vinyl chloride monomer thus appreciable ingestion of vinyl chloride via dust inhalation is not possible. 93/

The Union Carbide Corporation PVC resins handled and packaged by Amboy Terminaling Company were produced by the UCC continuous bulk polymerization process, the UCC continuous solution polymerization process and by a conventional suspension polymerization process. Tests of representative resins from these processes performed at UCC's laboratory showed the following

content of dusts in the less than 10 microns range. Results of these tests are as follows:

<u>Process</u> <u>UCC Bulk Press</u>	<u>Percent Particles</u> <u>10 Microns or Less</u>
VYNW Resin	0.4
UCC Solution Process	
VYDH	mil
Suspension Process	
Homopolymer QSAN-7	mil
Copolymer VSJE	0.004

Attached are photomicrographs made of the various resins discussed:

Figure I - shows the particles of UCC dispersion resin QYNV magnified 50 times.

Figure II - shows the particles of UCC dispersion resin QYNV magnified 500 times. This second picture shows discrete particles in the ≤ 10 micron respirable range.

Figure III - shows particles of UCC bulk polymerized QYNL resin magnified 50 times. The tests indicate this type of resin contains 0.4% by weight particles less than 10 microns.

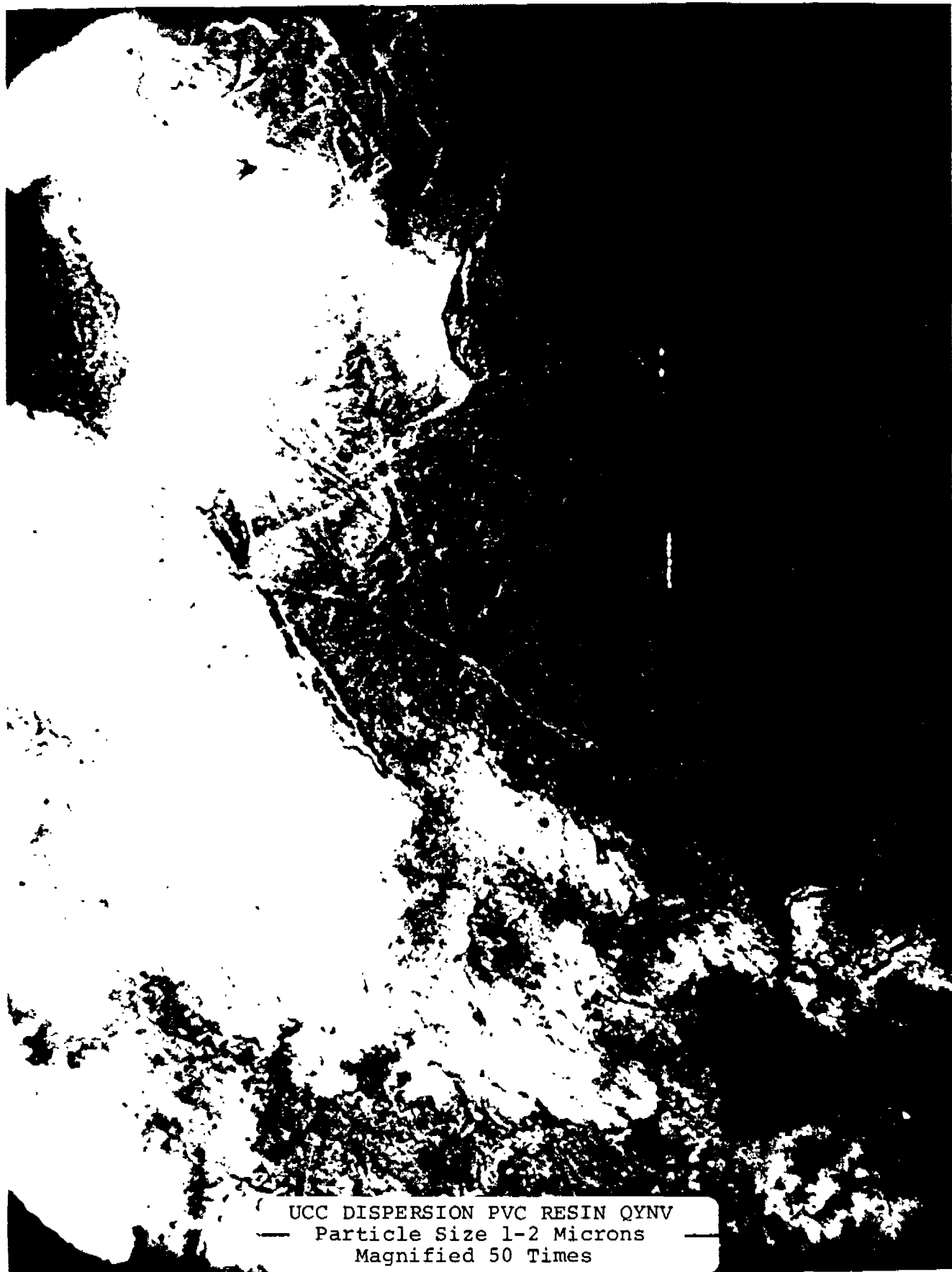
Figure IV - shows the particles of UCC solution polymerized resin VYHD magnified 50 times. The tests showed no particles 10 microns or less present.

Figure V - shows the particles of UCC suspension polymerized resin QSAN magnified 50 times. The tests showed no particles 10 microns or less present.

To create airborne respirable dust, there must be a source of dust particles less than 10 microns in diameter and the resin containing the source of respirable dust exposed to a turbulent air stream which later becomes a part of the workspace air. Resin at rest in bags or even lying on the floor will contaminate the workspace air with some form of aeration. Process dust sources such as the bagging operation at ATC were provided with exhaust hoods.

Summary Statements:

1. Research has not clearly shown that there is a relationship between pneumoconiosis and PVC resin dust.
2. The ingestion of vinyl chloride monomer via inhalation of respirable PVC dusts is negligible since such dust contains less than one ppm by weight of vinyl chloride monomer.
3. The UCC PVC resins handled and bagged by ATC contained very little, if any, respirable dust.
4. Local ventilation was provided at ATC for operations that were dusty.
5. Exposure to the PVC resin handled by ATC for Union Carbide Corporation is unlikely to cause chronic human disability via inhalation.



UCC DISPERSION PVC RESIN QYNV
— Particle Size 1-2 Microns —
Magnified 50 Times

FIGURE I

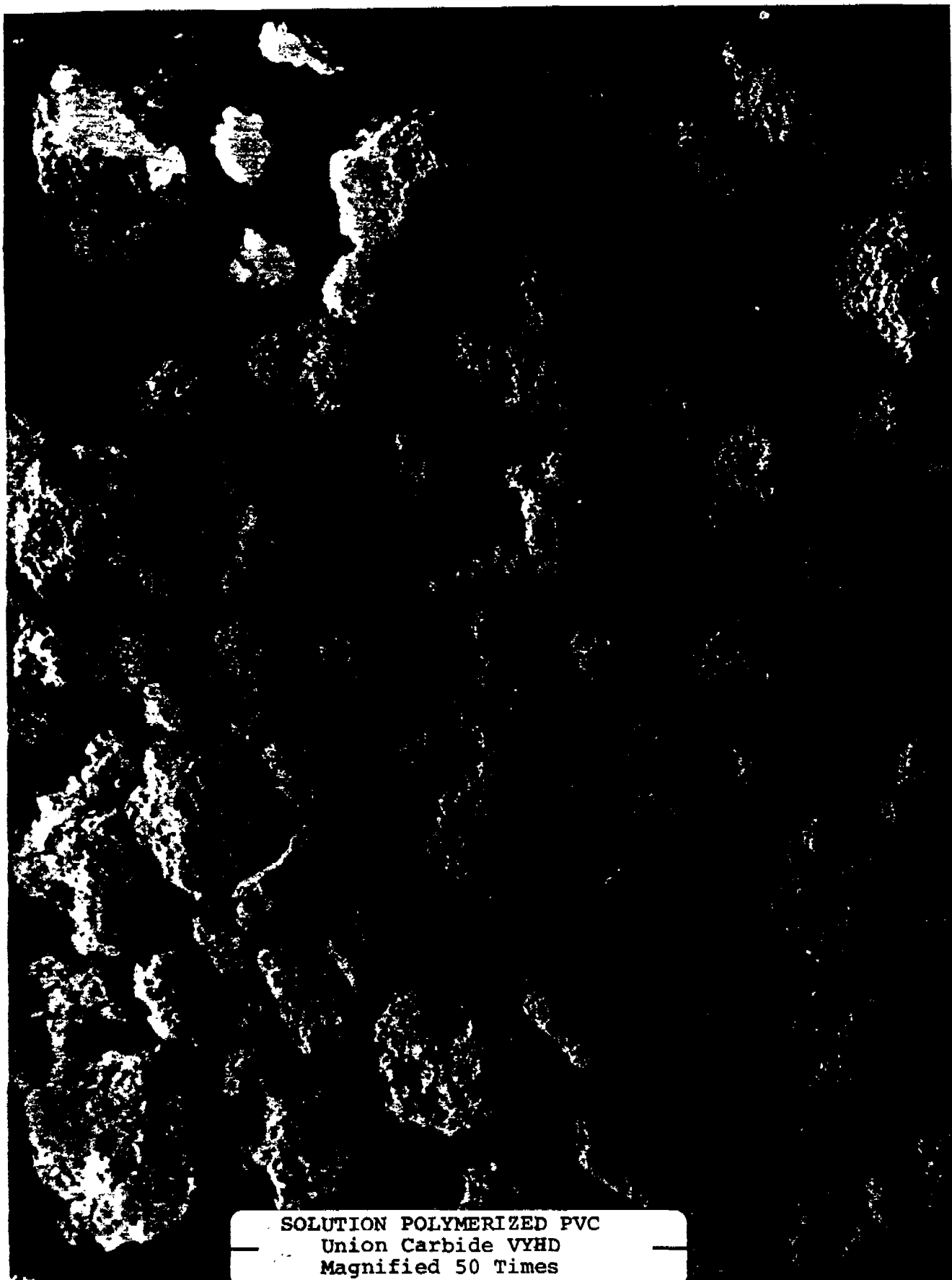
39



UCC DISPERSION PVC RESIN QYNV
Magnified 500 Times

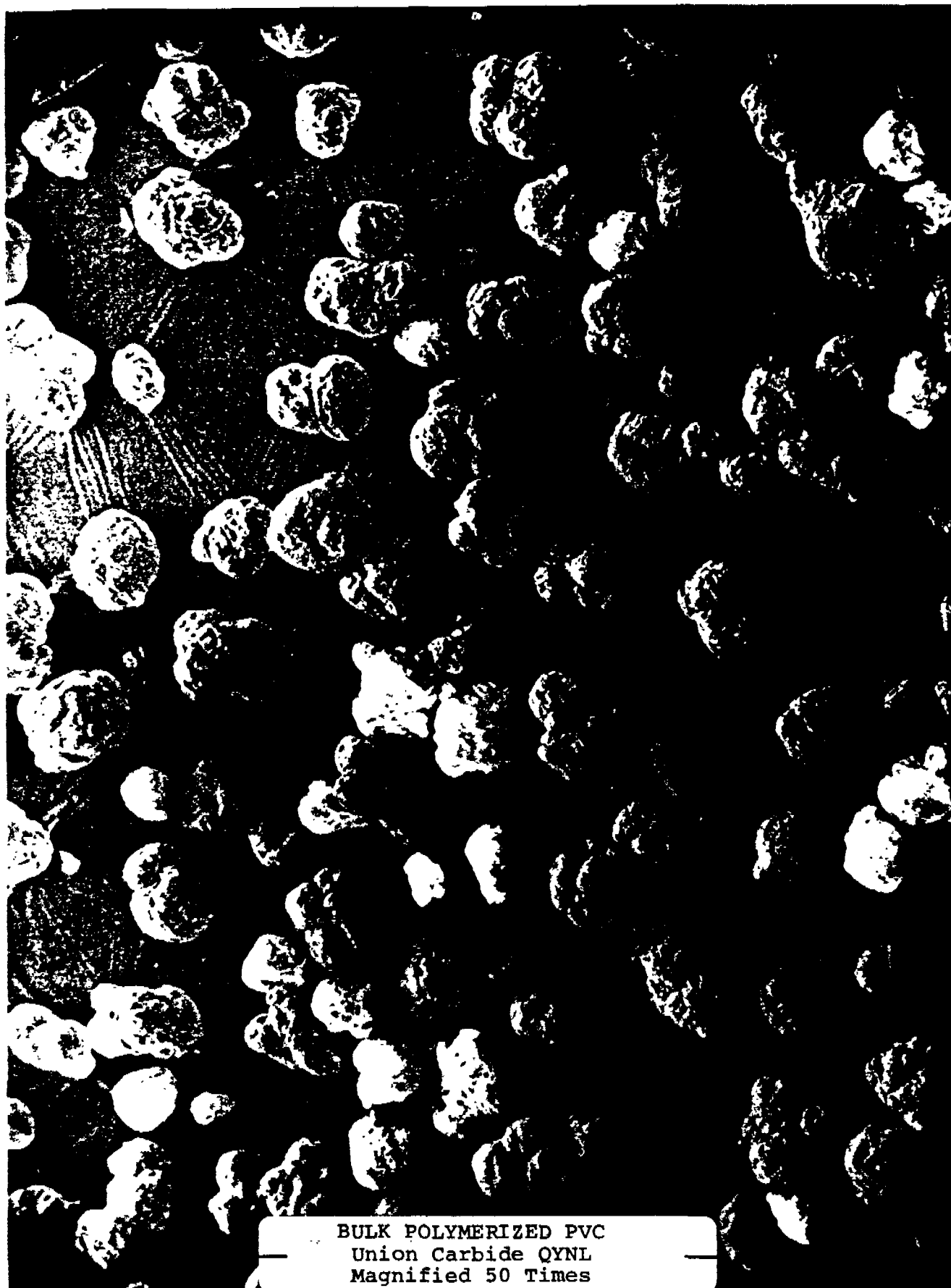
FIGURE II

UCC
067924



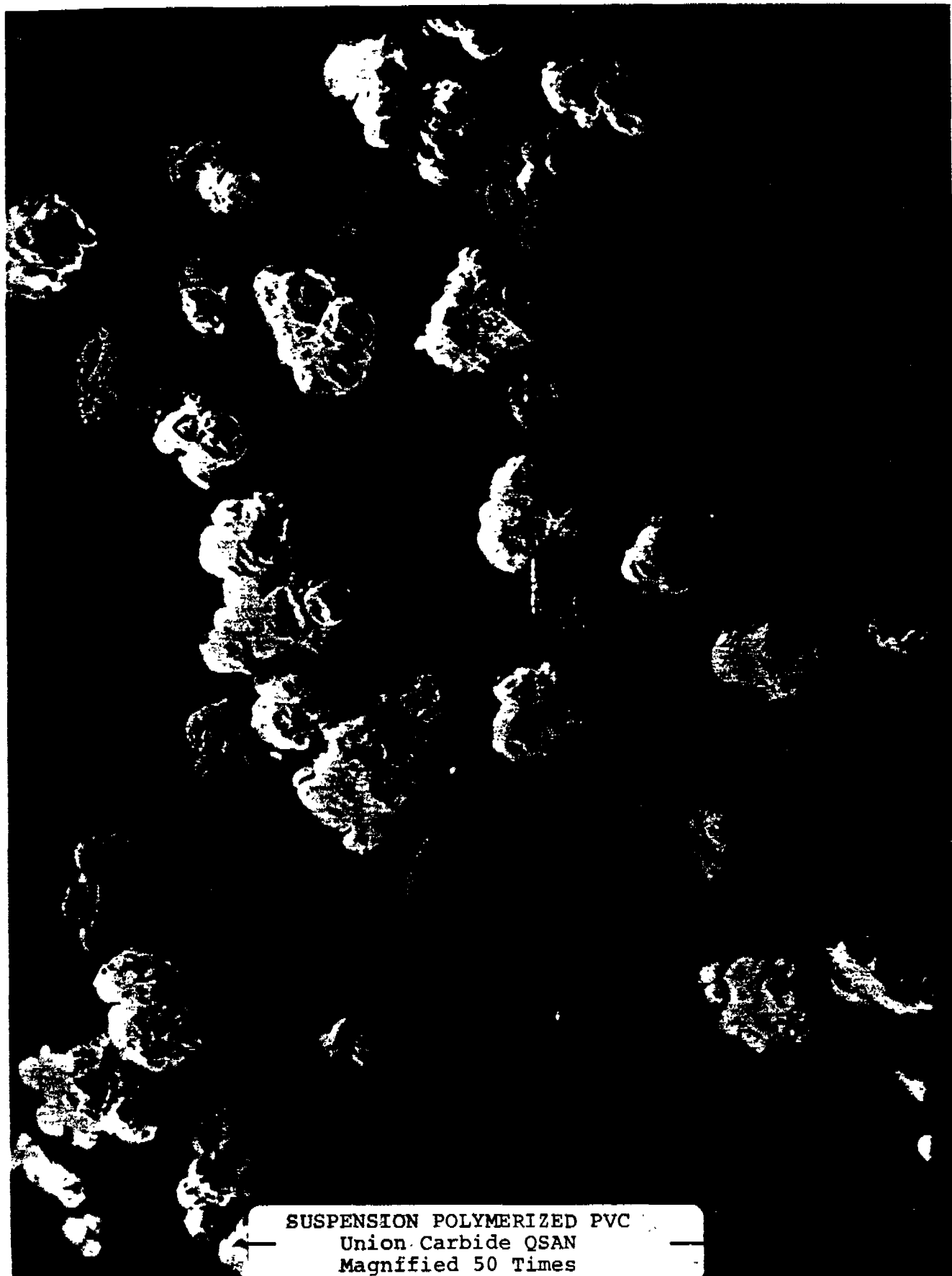
SOLUTION POLYMERIZED PVC
Union Carbide VYHD
Magnified 50 Times

FIGURE III



BULK POLYMERIZED PVC
Union Carbide QYNL
Magnified 50 Times

FIGURE IV



SUSPENSION POLYMERIZED PVC
Union Carbide QSAN
Magnified 50 Times

FIGURE V

UCC
067927

ESTIMATES OF EXPOSURE TO VINYL CHLORIDE MONOMER

The Perth Amboy ATC facilities are located on 35 acres of land adjacent to the bay. Tankers with deck loads or container ships are docked and unloaded by a large traveling crane which removes the containers (van boxes) from the ship and places them on the ground. At this point, the box is picked up by a straddle carrier and moved to the storage yard where as many as 1,500 containers may be stored in the open.

On receipt of an order for a bulk shipment, hopper truck or hopper rail car, the appropriate box of resin is selected by the straddle carrier operator and placed on a tilting mechanism. The box is tilted and the product transferred from the box to the bulk carrier by gravity. (The product is literally poured from one container to another.) At the end of the transfer, the containers are closed. The empty van box is returned to the manufacturing plant by ship and the rail car or truck is moved to the ultimate customer. This operation is carried out in the open air.

Resin packaging and package storage was carried out in a large masonry building encompassing approximately 250,000 square feet. This building was compartmentalized as follows:

(1) Three large warehouse storage rooms of 60,000 square feet each. Warehouse room "A" was used for vinyl chloride resin bag storage. Warehouse rooms "B" and "C" were used for polyethylene, polystyrene and bisphenol A bag storage. These rooms were ventilated only by the open loading dock doors.

(2) One palletizing room of approximately 40,000 square feet contains six automatic bag palletizers. The filled multi-wall kraft paper bags of product enter the room via belt conveyor and are carried into the

palletizer where 45 bags are stacked on a wooden pallet. These pallets are then transported by fork truck to the warehouse area. Room ventilation is provided only by open doors.

(3) Three bagging rooms for vinyl resins have approximately 850 square feet each. These rooms, each of 16,500 cubic feet, are ventilated by a fan to provide one air change every 3.83 minutes. The vinyl resin enters the room through a closed chute into a closed hopper over the bagging machine. The closed hopper vents through a bag filter to an exhaust system. The multi-wallpaper bags are fabricated with an opening in one end consisting of a paper or plastic sleeve. The bagging machine fill nozzle fits into this opening and the product flows from the bagging machine into the bag. When the bag is at the proper weight, the flow of product shuts off and the bag is pulled off the fill nozzle by the operator. The material in the bag collapses the filling part on the bag, thus closing the bag. The filled bag is carried by conveyor through a metal detector, a roller flattener and thence to the palletizer in the next room. In order to make the produced resin flow from the packing machine into the bag, compressed air is injected into the resin contained in the packer compartment causing the powdered solids to become fluidized; i.e., flow like water. This fluidization plus a slight positive pressure within the packer forces the product through the fill nozzle into the bag. The bag walls are porous and the fluidizing air carried into the bag with the product passes through the bag walls to the outside leaving the product in the bag. For each bag filled, approximately 1.25 cubic feet of air are discharged into

the room. If too much fluidizing air flow is used, if the bag wall porosity is too low, or if the bag is poorly glued, it bursts during filling, fouling the immediate area with product. The dust collection system is designed to handle minor spills and the dribbles from the bags and packer on disengaging but when several bags burst, this must be cleaned manually using the vacuum collection system.

In the resin bagging operation the appropriate bulk container (van box) of resin is selected by the straddle carrier operator from those in the storage yard. The box is transported to a tilting frame outside the resin bagging room. The box is connected to the bagging machine through the building wall by a duct. To start the bagging operation, the box is tilted and the contained resin flows from the box through the duct into closed hopper on top of the bagging machine. When the box is empty, it is lowered to a level position and removed to the storage yard by the straddle carrier.

The vinyl chloride resins handled, packaged and warehoused at ATC facilities in Perth Amboy, New Jersey, were manufactured by one of these three processes:

1. Solution polymerized vinyl chloride copolymer.
2. Continuous bulk polymerized vinyl chloride copolymer or homopolymer.
3. Suspension polymerized vinyl chloride homopolymer or copolymer.

This description of ATC operations and facilities was provided by Mr. T. F. Archer, Union Carbide Corporation representative to ATC.

At the time these resins were shipped, the residual vinyl chloride was estimated to be: 2/, 3/, 93/

(a) Solution resin	< 1 ppm RVC
(b) Bulk resin	15 ppm RVC
(c) Suspension resin	860 ppm RVC

The suspension resin was made up of three varieties: vinyl chloride homopolymer, vinyl acetate copolymer and ethylene copolymer. The estimate in "c" above is higher than homopolymer and ethylene copolymer but lower than vinyl acetate copolymer. Since vinyl acetate copolymer gives up its residual vinyl chloride less readily than the others, its contribution to the workspace air contamination in a unit time is lower. Vinyl chloride is not soluble in vinyl resins but is slightly soluble in vinyl acetate thus the vinyl acetate containing resins give up the unconverted vinyl chloride less readily. Over a period of time, vinyl chloride resins emit the unconverted vinyl chloride at varying rates depending upon the amount present, the size of the resin particle, the porosity of the resin particle, the molecular weight of the resin and the amount of residual vinyl acetate present. For purposes of this discussion, the rate of emission has been based on ethylene copolymer resin which contains up to 1,000 ppm RVC and gives it up readily thus creating a worst case situation. 3/

Study has shown that ethylene copolymer yields 37% of its contained RVC in container storage per month. It yields 20% of its residual VCM with one hour of aeration at ambient temperature. It also loses 85% of its RVC in one week of storage in bags in a warehouse.

Storage and handling of vinyl resin in containers and bulk storage is estimated as follows:

Bin storage awaiting analysis	2 days
Containers at ship dock	4 days
Containers on the ship	7 days
Containers stored at ATC	19 days
(container inventory at ATC averaged 1.25 months sales)	—
 TOTAL Container Storage	 32 days

Resin RVCM at time of bagging at ATC was:

1. Solution resin (1.00-0.39) (< 1)	< 0.6 ppm
2. Bulk resin (1.00-0.39) (15)	9.0 ppm
3. Suspension resin(1.00-0.39) (860)	525.0 ppm

In a typical year, during the period 1960 to 1975, the material bagged was:

Solvent resin	61.5%
Bulk resin	12.8%
Suspension resin	25.7%

Resin handled and shipped in bulk is assumed to cause no exposure to dust or VCM since operations were outdoors and the resin was not aerated during handling.

The resin bagging operation was carried out in a room 21' 4" x 39' 2" x 19' 9" for a total volume of approximately 16,500 cubic feet. With ventilation at a rate of one air change every 3.83 minutes, the amount of vinyl chloride emitted required to maintain a concentration in the bagging room of 1 ppm would be:

$$\frac{(16,500)}{(3.83)} (0.000001) = 0.00431 \text{ ft. }^3/\text{min.}$$

The resin feeds from a closed vented hopper to the bagging machine when it is aerated and bagged at a rate of 50 pounds per 15 seconds with 1.25 cubic feet of air. Assuming the conveyor is running at a minimum speed when the bagging rate is 400 pounds or 8 bags per minute, the 36-foot conveyor would contain six bags in the room allowing six feet of conveyor per bag. The conveyor storage plus two bags being filled would expose 400 pounds of aeriated resin per minute which is losing 20 percent of its contained RVCM per hour thus:

$$\frac{(400)(525)(0.20)(378)}{(1,000,000)(62.5)(60)} = 0.00423 \text{ cubic feet/minute}$$

Comparing this worst case, volume RVCM emitted versus 1 ppm volume shows the concentration of VCM in the bagging room would be 0.98 ppm when bagging suspension resin.

For solution resin, the concentration would be:

$$\frac{(400)(\leq 0.6)(0.20)(378)}{(1,000,000)(62.5)(60)} = \leq 0.000005 \text{ cubic feet/minute}$$

or

$$\leq \frac{0.000005}{0.00431} = \leq 0.001 \text{ ppm VCM}$$

For bulk resin, the concentration would be:

$$\frac{(400)(9)(0.2)(378)}{(1,000,000)(62.5)(60)} = 0.000072$$

or

$$\frac{0.000072}{0.00431} = 0.017 \text{ ppm VCM}$$

The average annual exposure for a bagger would be:

$$(0.615)(0.001) + (0.128)(0.017) + (0.257)(0.98) = 0.25 \text{ ppm}$$

with a maximum of 0.98 ppm.

In the palletizing room, the area was 40,000 square feet with a 20 foot ceiling yielding volume of 800,000 cubic feet. Since ventilation was random, one air change per hour is assumed. The worst case situation would involve operating three lines on vinyl resin bags thus suspension resin at $(525)(1.0-0.2) = 420$ ppm RVCM and bulk resin at 9 $(1.0-0.2) = 7.2$ ppm RVCM. Operating each line at 400 pounds per minute would involve exposure of 1,125 pounds of each resin type for six minutes. The bagged resin is losing its RVCM at a rate of 85% per week or 0.05% for six minutes. Thus:

$$\begin{aligned} & \frac{(420)(0.05)(378)(1,125)}{(1,000,000)(100)(62.5)} + \frac{(0.48)(0.05)(378)(1,125)}{(1,000,000)(100)(62.5)} \\ & + \frac{(7.2)(0.005)(378)(1,125)}{(1,000,000)(100)(62.5)} = 0.00145 \text{ cubic feet/6-minutes} \\ & \text{or } 0.014549 \text{ cubic feet/hour} \end{aligned}$$

$$\frac{(0.014549)(1,000,000)}{800,000} = 0.02 \text{ ppm}$$

for one air change per hour.

For one air change per day, the maximum concentration at the end of the working day would be:

$$\frac{(0.014549)(8)(1,000,000)}{266,667} = 0.44 \text{ ppm}$$

for an average exposure of 0.22 ppm

The warehouse situation cannot be modeled mathematically due to the variety of unknowns. The personal monitoring data collected at ATC by Gollob Analytical Service Corporation reflects the minimal exposure.

	<u>Concentration</u>	<u>Sample Time</u>
Report of April 25, 1974		
Suspension resin bagger	0.8 ppm	(200) minutes
Solution resin bagger	0.4 ppm	(20) minutes
Report of May 7, 1974		
Bulk resin bagger	0.45 ppm	(20) minutes
Solution resin bagger	0.21 ppm	(20) minutes
Report of May 22, 1974		
Suspension resin bagger	0.2 ppm	(10) minutes
Solution resin bagger	0.4 ppm	(10) minutes
Report of July 23, 1974		
Solution resin bagger	0.7 ppm	(?)
Suspension resin bagger	1.3 ppm	(?)
Report of August 29, 1974		
Bulk resin bagger	0.1 ppm	(?)
Solution resin bagger	ND*	(?)

* ND - not detectable

UCC
067935

Report of October 30, 1974

Solution resin bagger	0.3 ppm	(13) minutes
Suspension resin bagger	3.7 ppm	(20) minutes

Report of November 14, 1974

Fork truck operator	1.9 ppm
---------------------	---------

Report of December 9, 1974

Fans installed in warehouse

Solution resin bagger	0.47 ppm TWA 8
Suspension resin bagger	1.47 ppm TWA 8

Report of December 27, 1974

Fans in warehouse not operating

Suspension resin bagger	2.28 ppm TWA 8
Solution resin bagger	0.24 ppm TWA 8
Fork truck operator	0.96 ppm TWA 8

Report of June 2, 1975

Suspension resin bagger	0.76 ppm TWA 8
-------------------------	----------------

Report of July 22, 1975

Bulk resin bagger	0.11 ppm TWA 8
-------------------	----------------

Report of September 30, 1975

Warehouse ventilation change noted.

Suspension resin bagger	0.45 ppm TWA 8
-------------------------	----------------

Report of December 17, 18 and 19, 1975

Change in packing room ventilation noted.

Fork truck operator	ND *	TWA 8
Suspension resin bagger	ND *	TWA 8

Report of January 22, 1976

Fork truck operator	0.1 ppm TWA 8
---------------------	---------------

Report of July 7, 1976

Bulk resin bagger	0.05 ppm TWA 8
Fork truck operator	0.06 ppm TWA 8

* ND - not detectable

UCC
067936

Summary of Gollob Measurements

<u>Job</u>	Average Concentration		<u>Range</u>
	<u>VCM</u>		
Suspension resin bagger	1.22 ppm	ND* ———	3.7 ppm
Solution resin bagger	0.34 ppm	ND* ———	0.7 ppm
Bulk resin bagger	0.18 ppm	0.05 ———	0.45 ppm
Fork truck operator	0.6 ppm	ND* ———	1.9 ppm

* ND - not detectable

Based on the foregoing analysis of potential employee exposure and the work done by Gollob Analytical Service Corporation, the ATC employees at Perth Amboy were not exposed to toxic concentrations of vinyl chloride and were only rarely exposed in excess of the permissible exposure limits set by OSHA, October 21, 1974.

UCC
067937

LITERATURE CITED

- * 1. Adams, W.G.F. and Purchase, I.F.H.: "Is PVC Dust a Hazard?", Unpublished Communication, Imperial Chemical Industries, Ltd., Welwyn Garden City, Herfordshire, England, July 23, 1975.
- * 2. Arnold, R. M.: "Current Data of the Residual Vinyl Chloride in Suspension/Non-Solvent Vinyl Resins", letter to Mr. M. E. Eisenhour, Union Carbide Corporation, Texas City Plant, dated March 14, 1974.
- * 3. Arnold, R. M.: "Analysis of Suspension Vinyl Resins for VCM Content Following Storage", Resins Laboratories, UCC, C&P Div., Texas City Plant, dated April 29, 1975.
- * 4. Arnold, R. M.: "VCM Content in QSOF-7 Resin" - during bag storage at Perth Amboy Terminal. Letter to Messrs. E. Bell and G. Scott, Union Carbide Corporation, October 17, 1975.
- * 5. Arnold, R. M.: "Comparison of Original Blend and Shipping Container for Residual VCM in QYNL/VYNW Non-Solvent Vinyl Resins", Union Carbide Corporation, March 4, 1976.
- * 6. Arnold, R. M.: "Samples Forwarded for Evaluation of the Head Space Technique Proposed by EPA". Letter to Mr. A. E. Gabany, Union Carbide Corporation, March 12, 1976.
- * 7. Arnold, R. M.: "Particle Size Distribution UCC PVC Resins", Texas City Plant, Union Carbide Corporation, December 8, 1977.
- 8. Baretta, E. D., et al,: "Monitoring Exposures to Vinyl Chloride Vapor - Breath Analysis and Continuous Air Sampling", American Industrial Hygiene Association Journal, p. 537, Vol. 30, 1969.
- 9. Baxter, P. J., et al,: "Angiosarcoma of the Liver in Great Britian 1963-1973:", British Medical Journal, pp. 919-921, Vol. 2, 1977.
- 10. Baxter, P. J. and Fox, A. J.: "Angiosarcoma of the Liver in P.V.C. Fabricators", The Lancet, January 31, 1976.
- 11. Verband Kunststofferzeugende Industrie E.V. (VKE) "VC/PVC: Beispiel einer Problemlosung", Frankfurt/Main, 1975.
- 12. Carr, J., et al,: "Chemical Constitution of Hydrocarbons and Cardiac Automaticity", Journal of Pharmacology Exptl, Ther. 97, 1, 1949.

* Copy Attached

UCC
067938

13. Chan, R., et al,: "Stripping of Vinyl Chloride Monomer from Resins in Aqueous Slurries and from Dry Resin Powders", Air Products and Chemicals, Inc., October 1974.
14. Chiazze, L., Jr., Nichols, W. E., and Wong, Otto: "Mortality Among Employees of PVC Fabricators", Journal of Occupational Medicine, Vol. 10, No. 9, September 1977.
15. Downs, T. D., et al,: "Vinyl Chloride, Birth Defects and Fetal Wastage, A Critical Review", The Society of the Plastics Industry, September 16, 1977.
- * 16. Equitable Environmental Health, Inc.,: "Epidemiologic Study of Vinyl Chloride Workers Employed by Union Carbide Corporation, South Charleston Plant", 2180 Milvia Street, Berkeley, California 94704, December 1976.
17. Falk, Henry, et al,: "A Cross-Sectional Epidemiologic Survey of Vinyl Chloride Workers", United States Department of Health, Education and Welfare, National Institute for Occupational Safety and Health, Cincinnati, Ohio, April 1977.
18. Fox, A. J., et al,: "Mortality Experience of Workers Exposed to Vinyl Chloride Monomer in the Manufacture of Polyvinyl Chloride in Great Britain", British Journal of Industrial Medicine, pp. 1-10, Vol. 34, 1977.
19. Gamble, J., Shuguey L., McMichael and Waxweiler: "Effect of Occupational and Non-Occupational Factors on the Respiratory System of Vinyl Chloride and Other Workers", Journal of Occupational Medicine, Vol. 18, No. 10, October 1976.
- * 20. Gehring, P. J., et al,: "Resolution of Dose-Response Toxicity Data for Chemicals Requiring Metabolic Activation: Example - Vinyl Chloride", Dow Chemical U.S.A., Midland, Michigan 48640, July 12, 1977.
- * 21. Harvard University for the B. F. Goodrich Company: "Animal Feeding Studies on Geon 202" (Geon 202 is a copolymer of vinyl chloride-vinylidene chloride.) B. F. Goodrich Company, Cleveland, Ohio, 1947.
22. Health and Safety Executive: "Vinyl Chloride, Code of Practice for Health Precautions", London, 1975.
- * 23. Hellman, T. M.: "The Characterization of the Odor Properties of 101 Union Carbide Corporation Petro-chemicals Using Sensory Methods:, Project No. 510A20, R&D Dept., C&P, Union Carbide Corporation, South Charleston, WV, July 10, 1973.

24. Infante, P. F., et al,: "Carcinogenic, Mutagenic and Teratogenic Risks Associated with Vinyl Chloride", Mutation Research, pp. 131-142, Vol. 41, May 13, 1976.
- * 25. Johnson, L. F., and Arnold, R. M.: "A Study of Residual VCM in Suspension Resin", letter to Mr. M. E. Eisenhour, Texas City Plant, Union Carbide Corporation, June 18, 1974.
26. Juhe, S., Lange, G., Stein, G., and Veltman, G.: "On the So-Called Vinyl Chloride Disease", University Skin Clinic of Bonn, Deutsche Medizinische Wochenschrift, 98 (43), pp. 2034-2037, 1973.
27. Kramer, C. G., et al,: "The Correlation of Clinical and Environmental Measurements for Workers Exposed to Vinyl Chloride", American Industrial Hygiene Journal, January 1972.
28. Kuzmach, A. and McGaughy, R.,: "Quantitative Risk Assessment for Community Exposure to Vinyl Chloride", United States Environmental Protection Agency, 1975.
29. Lee, C. C., Bhandari, J. C., et al,: "Carcinogenicity of Vinyl Chloride and Vinylidene Chloride", Journal of Toxicology and Environmental Health; Pharmacology & Toxicology, Midwest Research Institute, Kansas City, Missouri 64110 and Environmental Toxicology Branch, National Institute of Environmental Health Sciences, Research Triangle Park, N.C. 27709, ABSTRACT, 32, 1977.
30. LeFaux, R.: "Practical Toxicology of Plastics", Iliffe Books Ltd, London, pp. 142-144, pp. 37-371, 1968.
31. Lehman, K. B. and Flury, F., Toxicology and Hygiene of Industrial Solvents, Williams and Wilkins Company, 1943.
32. Lester, D., et al,: "Effects of Single and Repeated Exposures of Humans and Rats to Vinyl Chloride", American Industrial Hygiene Association Meeting, Washington, D.C., May 1962.
33. Linch, A. L.,: "Evaluation of Ambient Air Quality by Personnel Monitoring", pp. 141-145, C.R.C. Press, Inc., 1975.
- * 34. MacMahon, Brian, M.D.: "Vinyl Chloride and Human Reproduction", The Society of the Plastics Industry, Vinyl Chloride - Polyvinyl Chloride Safety Group, August 15, 1977.

35. Manufacturing Chemists Association (MCA): "Vinyl Chloride Chromology", MCA-3846 - 1825 Connecticut Avenue, N.W., Washington, D.C. 20009, May 10, 1974.
36. Maricq, Johnson, Whetstone & LeRoy: "Capillary Abnormalities in Polyvinyl Chloride Production Workers", JAMA, Vol. 236, No. 12, September 20, 1976.
37. Mastromatteo, E., et al,: "Acute Inhalation Toxicity of Vinyl Chloride to Laboratory Animals", American Industrial Hygiene Association Journal, p. 394, Vol. 5, 1960. Bulletin Hygiene, 36:244.
38. Mutchler, J. E., et al,: "Report on the Relation of Exposure to State of Health of Dow Chemical Workers", Gordon Conference, Tilton, New Hampshire, August 1968.
39. National Institute for Occupational Safety and Health: "Recommended Standard for Occupational Exposure to Vinyl Chloride", United States Department of Health, Education and Welfare, 1974.
40. Nass, L.: "Encyclopedia of PVC", Vol. I, p. 480, Muriel Decker, New York, 1976.
41. Nature, "Biological Reactivity of PVC Dust", Vol. 56, August 21, 1975.
42. New Scientist, "PVC Dust Fells Factory Mice Suggesting New Work Hazard", June 1975.
43. Oppenheimer, B. S., et. al,: "Further Studies of Polymers as Carcinogenic Agents in Animals", Cancer Research, Vol. 15, pp. 335-340, 1950.
44. Oppenheimer, E. T., et al,: "Observations on the Effect of Powdered Polymer in the Carcinogenic Process", Cancer Research, p. 132, Vol. 21, 1961.
- * 45. Organization Resources Counselors, Inc.: "Report on a Mortality Study Covering Employees of PVC Fabricators", 1625 I Street, N.W., Washington, D.C. 20006, February 1976.
- * 46. Organization Resources Counselors, Inc.: "Report on a Case Control Study Covering White Female Employees of PVC Fabricators", May 1977.

- * 47. Occupational Safety and Health Administration Emergency Temporary Standard for Exposure of Vinyl Chloride, United States Department of Labor, April 5, 1974.
- 48. Oster, R. H., et al,: "Narcosis with Vinyl Chloride", Anesthesiology, Vol. 8, p. 359, 1947.
- 49. Ott, M. G., Langner, R. R., and Holder, B. B.: "Vinyl Chloride Exposure in a Controlled Industrial Environment", Arch Environ Health, Vol. 30, July 1975.
- * 50. Paddle, G. M.: "Report on a Mortality Study Covering Employees of PVC Fabricators". (Personal Letter to Mr. R. N. Wheeler, Jr. - letter written to: Dr. J. Stafford, Health and Environment Dept., ICI Plastics Division, Welwyn), July 13, 1977.
- 51. Patty, F. A., et al,: "Acute Response of Guinea Pigs to Vapors of New Commercial Organic Compounds", Public Health Report, 45, 1963.
- 52. Patty, F. A.: "Industrial Hygiene and Toxicology", Vol. II, BUS-1307, John Wiley and Sons, New York, New York, 1962.
- 53. Peoples, A. S. and Leake, C. D.: Journal of Pharmacology, Vol. 48, p. 284, 1933.
- 54. Picciano, Flake, Gay and Kilian: "Vinyl Chloride Cytogenetics", Journal of Occupational Medicine, Vol. 19, No. 8, August 1977.
- 55. Pigott, G. H.: "In Vitro, Studies of a Range of Formulations of PVC", American Chemical Society, New York Meeting, April 4, 1976.
- 56. Popow, Jerzy: "Effect of Polyvinyl Chloride (PVC) Dust on the Respiratory System of the Rat", Roczniki Akademii Medycznej im Juliana Marchlewskiego w Bialymstoku, Supplement 24, p. 5, Vol. 48, 1969.
- 57. Prodan, L., et al,: Intoxicatia conica cu clorura de vinil, Ses. St. 1 MF Cley Romania, 1969.
- 58. Prodan, L. and Suciu I, et al,: "Experimental Acute Toxicity of Vinyl Chloride". Toxicity of Vinyl Chloride - Polyvinyl Chloride, New York Academy of Sciences, Vol. 246, pp. 154-158, 1975.

59. Richardson, H. L.: "Angiosarcoma of the Human Liver", U.S. Environmental Protection Agency, April 17, 1974.
- * 60. Roeh, W. V.: "Residual VCM Suspension Vinyl", letter to Mr. Ed Bell - material stored one month in Palmer Warehouse, Union Carbide Corporation, March 26, 1975.
61. Sarvetnick, H. A.: "Polyvinyl Chloride", Reinhold Plastics Applications Series, 1969.
62. Scamehorn, J. F. and Yang, Kang: "Diffusion of Vinyl Chloride in Polyvinyl Chloride Below the Glass Transition Temperature", American Institute of Chemical Engineers, New York Meeting, November 1977.
63. Schaumann, O., MED U. CHEMIE, Vol. 2, p. 139 (cited by Dr. P. L. Viola), 1931.
64. Selikoff, I. J. and Hammond, E. C.: "Toxicity of Vinyl Chloride - Polyvinyl Chloride", New York Academy of Sciences, Vol. 246, 1975.
 - (a) Suci, et al.: "Chemical Manifestations in Vinyl Chloride Poisoning", pp. 53-69.
 - (b) Maltoni, et al.: "Carcinogenicity Bioassays of Vinyl Chloride Current Results", pp. 195-218.
 - (c) Volkheimer: "Hematogeneous Dissemination Ingested Polyvinyl Chloride Particles", p. 164.
 - (d) Kepfinger, et al.: "Interim Results of Exposure of Rats, Hamsters and Mice to Vinyl Chloride", pp. 219-230.
65. Smith, P. M., Williams, D.M.J. and Evan, D.M.D.: "Hepatic Angiosarcoma In a Vinyl Chloride Worker", Bulletin of the New York Academy of Medicine, May 1976.
66. Smyth, H. F., Jr.: "Improved Communications - Hygiene Standards for Daily Inhalation", American Industrial Hygiene Association Journal, p. 129, 17, 1956.
67. Smyth, H. F., Jr. and Weil, C. S.: "Chronic Oral Toxicity to Rats of Vinyl Chloride - Vinyl Acetate Copolymer", Toxicology and Applied Pharmacology, 9, 501-504, 1966.
- * 68. Stafford, John: Letter to Dr. T. Torkelson, Dow Chemical, containing a tabulation of worldwide cases of angiosarcoma, July 29, 1977.

69. Stafford, John: "Vinyl Chloride Disease" or "Vinyl Chloride Directive No. 2", Imperial Chemical Industries Limited (ICI) of London - covering regulations on vinyl chloride for Germany, The Society of the Plastics Industry, Inc., December 8, 1977.
- * 70. Torkelson, T. R.: "Statement of the Manufacturing Chemists Association Before the Subcommittee on the Environment of the Senate Committee on Commerce on Vinyl Chloride", MCA, August 21, 1974.
71. Szende, C. K., et al,: "Pneumoconiosis Developing After Inhalations of Polyvinyl Chloride", Orv. Hetil, 112:85-6, January 10, 1971.
72. Tabershaw/Cooper Associates, Inc.: "Epidemiological Study of Vinyl Chloride Workers", 2180 Milvia St., Berkeley, California 94704, May 3, 1974.
73. Torkelson, T. R., et al,: "The Toxicity of Vinyl Chloride as Determined by Repeated Exposure of Laboratory Animals", American Industrial Hygiene Association Journal, p. 354, 22, Number 5, 1961.
74. Tribukh, S. L., et al, Gigenia Sanit., 10:38, 1949, quoted by Marsteller, et al,: "Splenomegalic Liver Disease", Toxicity of Vinyl Chloride - Polyvinyl Chloride, New York Academy of Sciences, Vol. 246, 1975.
- * 75. Union Carbide Corporation: "Statement of Carl U. Dernehl, M.D., Associate Medical Director, prepared for Presentation at the Department of Labor Hearings on Vinyl Chloride Occupational Exposure Standard, June 25, 1974."
- * 76. Union Carbide Corporation's Testimony, OSHA Public Hearing, Docket OSH-36, June 25, 1974.
77. Occupational Safety and Health Administration, "Standard for Exposure to Vinyl Chloride". (29 CFR 1910.1017) U.S. Department of Labor, Federal Register 39, pp. 35890-35898, pp. 41848, 1974.
78. Occupational Safety and Health Administration, "Vinyl Chloride, Proposed Standard", U.S. Department of Labor Federal Register 39, pp. 16896-16900, 1974.
79. United States Environmental Protection Agency: "Scientific and Technical Assessment Report on Vinyl Chloride and Polyvinyl Chloride", EPK-600/6-75-004, 1975.

80. United States Environmental Protection Agency: "Standard Support and Environmental Impact Statement, Emission Standard for Vinyl Chloride", Vol. II, 1976.
81. United States Environmental Protection Agency: "Standard Support and Environmental Impact Statement, Emission Standard for Vinyl Chloride", 1975.
82. The University of Michigan: "Epidemiological Investigation of the Polyvinyl Chloride Industry in Reference to Occupational Acroosteolysis", The Institute of Industrial Health, February 1969.
83. Viola, P. L.: "Carcinogenic Effect of Vinyl Chloride", Regina Elena Institute for Cancer Research, University of Perugia, Tenth International Cancer Congress, Houston, Texas, May 22, 1970.
84. Viola, P. L.: "Oncogenic Response of Rat Skin, Lungs and Bones to Vinyl Chloride", Cancer Research, pp. 516-522, Vol. 31, 1971.
85. Viola, P. L.: "Pathology of Vinyl Chloride", Medicina del Lavoro, Vol. 61, pp. 174-179, March 1970.
86. Viola, P. L.: "The Vinyl Chloride Disease", Unpublished Communication, Summer, 1970.
87. Von Oettingen, W. F.: "The Halogenated Hydrocarbons, Toxicity and Potential Dangers", U.S. Public Health Service Publication No. 414, Government Printing Office, Washington, D.C., 1955.
88. Von Oettingen, W. F., U.S. Public Health Service Publication No. 411, U.S. Government Printing Office, Washington, D.C., 1933.
89. Wagoner, Infante and Brown: "Genetic Effects Associated with Industrial Chemicals", Industrywide Studies Branch, Div. of Surveillance, Hazard Evaluation and Field Studies, National Institute for Occupational Safety and Health, Cincinnati, Ohio, 1976.
90. Waxweiler, et al.: "A Cross-Sectional Epidemiologic Survey of Vinyl Chloride Workers", Department of Health, Education and Welfare, Cincinnati, Ohio, April 1977.

- * 91. Wheeler, R. N., Jr.: "Manufacturing Chemists Association Occupational Health Committee - Vinyl Chloride Conference", Union Carbide Corporation, November 23, 1971.
- * 92. Wheeler, R. N., Jr.: "Vinyl Chloride Research MCA Report to NIOSH", Union Carbide Corporation, July 19, 1973.
- 93. Wheeler, R. N., Jr.: Letter and Report to Mr. G. Wrenn, U.S. Dept. of Labor, OSHA, Classification of UCC Vinyl Coating Resins, Union Carbide Corporation, February 14, 1975.
- 94. Wilson, R. H., et al., "Occupational Acroosteolysis", Journal of American Medical Association, Vol. 21, No. 8, 1967.
- * 95. Hefner, R., Watanabe, P., Gehring, P.: "Percutaneous Absorption of Vinyl Chloride (VCM) Gas in the Rhesus Monkey", Dow Chemical U.S.A., Midland, Michigan, 1974.
- * 96. Equitable Environmental Health, Inc.: "Epidemiological Study of Vinyl Chloride Workers - Final Report", 2020 Milvia St., Berkeley, California 94704, August 1977.
- 97. Brady, J., et al.,: "Angiosarcoma of the Liver: An Epidemiologic Survey", J National Cancer Inst., Vol. 59, No. 5, November 1977.
- 98. Boettner, E., Ball, G., and Weiss, B.: "Analysis of the Volatile Combustion Products of Vinyl Plastics, Dept. of Industrial Health, School of Public Health, University of Michigan, Ann Arbor, Michigan, 1974.
- 99. Paciorek, K. L., et al.,: "Oxidative Thermal Decomposition of Poly (vinyl Chloride) Compositions", Journal of Applied Polymer Science, Vol. 18, pp. 3723-3729, 1974.

RNWJr./ws

April 10, 1978

UCC
067946

Attachment "A"

GOLLOB ANALYTICAL SERVICE CORPORATION

REPORTS

OF

WORK DONE AT AMBOY TERMINALING COMPANY

UCC
067947

Attachment "B"

LITERATURE CITED

Copies of Unpublished Items

UCC
067948