UNION CARBIDE CORPORATION
CHEMICALS AND PLASTICS
P.O. BOX 8004, SOUTH CHARLESTON, W. VA. 25303

SOUTH CHARLESTON PLANT

1057

PF
TOX
VINYL CHLORIDE

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

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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 angio-sarcoma 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

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 an s-thetic 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

sever cardiac arrhythma. 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 sever 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 LD_{50} 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 ref rence to chlorinat d 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.

Th MCA Committee proposed a program involving a three level three species chronic inhalation toxicity study, a study of vinyl chloric 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 lif - 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 th 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 th 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 biossay 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 th 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 Stat angiosarcoma cases. Three had had contact with vinyl chlorid in

*-1 Proportional Mortality Ratio

*-2 Angiosarcoma

their work and fiv 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 rat 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 critcal reviews of the reports on mutagenesis, fetal anomalies or fetal death report d 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 evid nt 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 polym riz d vinyl acetate copolymer and the other was vinylidene chlorid copolymer suspension polymerized. Both studies involved feeding 75 micron resin over a long period so certainly the particles

w re persorb d 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 identifi d 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. ., both the United States and the British study deal with proportional exc sses in a diseas category and may not reflect a real increase

or decrease in death rates ov r 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 manufactur 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 establish 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 d hydrochlorination of ethylene dichloride - South Charleston.

1942 Initiated production of vinyl chloride by synthesis proc ss 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 polym rization 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 Appli d 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 canc r, 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 Committe .

1971 An Ad Hoc Planning Group for Vinyl Chloride R search was formed in MCA under the chairmanship of Mr. R. N. Wh eler, 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 Occupation 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.