Memorandum

AIR PRODUCTS

To: DISTRIBUTION Dept.:

From: H. J. SMITH Dept./Ext.. CHEMICALS MANUFACTURING/8374

Date: 6 OCTOBER 1988

Subject: VCM

Distribution:

R. L. Duggan - 42
M. W. Ferris - 42
B. D. Helms - 139
H. Ortega - 139

cc: M. R. Chmura
J. T. Wharton

Attached for your use is a small package of information on VCM. Note especially the VI alert concerning Greenpeace. Also included are the final published versions of VI bulletins on Community Health Effects and Effects on Human Offspring.

H. J. Smith
H. J. SMITH

HJS:mcs
8728L

Attachments

FORM 1020-* 84

Memorandum

AIR PRODUCTS

To: Distribution

Dept.:

From: A. J. Diglio

Dept./Ext.: Corp. Environmental / 8339

Date: 5 October 1988

Subject: Vinyl Chloride Issue

Distribution

M. R. Chmura
B. Z. Drozdowicz
E. I. Handwerk
P. S. Handwerk
R. H. Schenck
J. H. Smith
J. K. Spata
L. B. Tepper
J. T. Wharton

cc: P. L. T. Brian
T. J. Marriott, Jr.

Please find attached the following information:

- The Vinyl Institute Executive Advisory

  Green Peace is planning some demonstrations on an international scale against PVC modules.

- Community health effects of vinyl chloride

- Potential effects of vinyl chloride on human offspring

- BNA Chemical Regulation Reporter 9/16/88

  Dow vinyl chloride study finds that their workers exposed to VCM did not have any increased risk of cancer.

Please let me know if you need anything else.

A. J. Diglio

AJD:tjs
0690q

Attachments

FORM 1822 (7.84)

AP00022289

The Vinyl Institute A Division of The Society of The Plastics Industry, Inc.
Wayne Interchange Plaza II
155 Route 46 West • Wayne, New Jersey 07470

Sept 30, 1988

To: Vinyl Institute Executive Board
Vinyl Institute Committee Chairman

EXECUTIVE ADVISORY

The purpose of this advisory is to inform you of some late-breaking and significant developments.

1. Norsk Hydro VCM Plant Explosion

Upon learning on September 19 of the explosion at Norsk Hydro's VCM plant in Norway, we contacted Harold Clayton of Norsk Hydro in England and Willem Prinselaar of EVC to get further details. We received a call from Mr. R. Saether, Head of the Petrochemicals Division of Norsk Hydro advising that as a result of the fire and explosion, there was some "damage to the PVC plant" but no injuries or fatalities. Mr. Saether stated that the plant will be down for at least one month, their adjacent chlorine plant is down as well and they will have to cut back on their PVC production as well.

On September 28, we received a telefax from Ian Owston, Purchasing and Distribution Manager, Hydro Polymers, Vinyls Division in England indicating that he was "seeking to contact all U.S. producers of PVC resins with a view to making some short/medium term purchases." We replied supplying him with requested contact information on VI member companies who are PVC producers. You may have already heard from him or can expect to be contacted shortly.

Finally, in a telephone conversation yesterday with Willem Prinselaar, I learned that the explosion was apparently caused by corrosion of pipes in the EDC plant. Mechanical completion of the repairs are now expected to take 2 to 3 months. Willem further advised that because of environmental concerns in Norway and the company's environmental record, it may take some additional time for the Norwegian authorities to allow the repaired plant to resume production.

As you may be aware, the Norsk Hydro plant has a reported capacity of 1 billion lbs. of VCM per year.

2. European Vinyl Manufacturers to meet with Greenpeace

Willem Prinselaar informed me yesterday that certain European vinyl manufacturers had learned that Greenpeace, the environmental activist organization, was planning some demonstrations on an international scale against PVC producers. Rumors were that Greenpeace was planning

demonstrations in front of PVC production plants such as chaining themselves to plant fences and gates, etc. Some of you may be aware that Greenpeace representatives, opposed to ocean dumping, recently tied up New York City rush hour traffic by having twelve demonstrators dangle from ropes off the Triborough Bridge.

Through the European Council of Vinyl Manufacturers, Greenpeace was contacted in various countries with the aim of sitting down and establishing a dialogue. This was categorically rejected by all except for Greenpeace, International in England.

A meeting is now scheduled to be held on October 14 with European Council of Vinyl Manufacturers representatives and Greenpeace. I am advised that two U.S. Greenpeace representatives will be attending that meeting: a Dr. Costner of Greenpeace in Washington and a Mr. Marco Kaltofen who is identified as being with Boston Chemical Data Corporation. At the first meeting it is expected that both groups will exchange ideas about how to establish a dialog and provide Greenpeace with information hopefully to change their views about PVC.

Because U.S. representatives of Greenpeace will be at this meeting, I have asked that Dr. Freiesleben of EVCM call me and advise me of the outcome in the event that Greenpeace approaches the Vinyl Institute or any of its member companies in the future.

We will, of course, keep you advised and ask you to let us know if your individual companies are contacted by Greenpeace.

3. Lead and Cadmium in Incinerator Ash Could Spell Problems for PVC

As you know, EPA has a Task Force developing a a Municipal Solid Waste Strategy. Their draft report entitled "The Solid Waste Dilemma: An Agenda for Action" has just been issued and public hearings are to be held in October in Long Beach,CA, Washington, D.C. and Atlanta,GA. Page 62 of the draft report contains the following statements: "EPA has initiated studies of certain waste materials to allow for the evaluation of potential bans on specific waste stream constituents. EPA studies are initially focused on potential sources of lead and cadmium."

While lead and cadmium use as stabilizers for PVC are not the major potential sources of these metals in incinerator ash, we must recognize that there are perceptions that they are. A report on "The Contribution of the Plastics Component of MSW To The Heavy Metal Content of MWC Ash" is being completed by a consultant to SPI, Dr. William Considine. The draft version of this report which I have reviewed indicates that pigments are the largest contributor of cadmium to plastics where they are used in ABS, HDPE,PP, LDPE and styrenics. Heat stabilizers of the Barium/Cadmium type account for 15% of the cadmium consumed in the U.S. Lead based heat stabilizers are primarily used in wire insulation and the report by Dr. Considine estimates that only 1% of the lead consumed in the U.S. is used in stabilizer manufacture.

Rather than wait for a ban on these metals to happen, I plan to explore the technical realism of the phasing out of the use of such metals from vinyl plastics at the next meeting of the Vinyl Institute Technical Committee in Cleveland on October 18. In the interim, I would ask that each of you consider whether the Vinyl Institute should adopt a policy position on this issue, viz. voluntary phase out, restriction on uses, elimination, in advance of any governmental action. I will plan to put this on the agenda for the next Executive Board meeting for discussion.

If you have any questions or comments on any of the items in this advisory, please call.

Roy D. Gottesman

RTG:g

The Vinyl Institute

TECHNICAL INFORMATION

COMMUNITY HEALTH EFFECTS OF VINYL CHLORIDE

Prepared By:

The Vinyl Institute

Health, Safety & Environment Committee

Issued: August 1, 1986

The Vinyl Institute, A Division of The Society of the Plastics Industry, Inc.
Wayne Interchange Plaza II, 155 Route 46 West, Wayne, New Jersey 07470, (201) 890-9299

I. SUMMARY

THIS DOCUMENT REVIEWS THE TOXICITY AND HUMAN HEALTH EFFECTS OF ABIENT EXPOSURE TO VINYL CHLORIDE (VC), THE RAW MATERIAL USED IN THE PRODUCTION OF POLYVINYL CHLORIDE (PVC). IT SUMMARIZES THE EXTENSIVE AND RIGOROUS FEDERAL REGULATION OF THE VC/PVC INDUSTRY, AND COMPARES QUANTITATIVE RISK ASSESSMENTS WITH ACTUAL HEALTH OBSERVATIONS OF INDIVIDUALS IN NON-OCCUPATIONAL SETTINGS.

A REVIEW OF THE WORLD SCIENTIFIC LITERATURE SHOWS NO COMMUNITY HEALTH IMPACTS ASSOCIATED WITH EXPOSURE TO VC EMISSIONS FROM VC/PVC MANUFACTURING FACILITIES.

II. INTRODUCTION

VINYL CHLORIDE IS THE BASIC BUILDING BLOCK FOR PRODUCING THE MOST VERSATILE PLASTIC YET DEVELOPED -- POLYVINYL CHLORIDE AND ITS COPOLYMERS WITH OTHER MONOMERS. MOST OF THE SEVEN BILLION POUNDS OF VC PRODUCED ANNUALLY IN THE UNITED STATES IS CONVERTED INTO PVC USED IN THOUSANDS OF PRODUCTS IN THE HOME AND IN INDUSTRY -- PRODUCTS SUCH AS WALLCOVERINGS, UPHOLSTERY, FLOORING, HOUSE SIDING, WATER PIPES, SEWER PIPES, LUGGAGE, CLOTHING, AUTOMOTIVE PARTS, AND MEDICAL DEVICES, FOOD WRAP, WINDOWS, DOORS AND WIRE INSULATION, GARDEN HOSES, AND PHONOGRAPH RECORDS.

PVC IS A POLYMER PRODUCED FROM VC THROUGH A CHEMICAL REACTION CALLED POLYMERIZATION. VC IS CONVERTED INTO PVC BY SUSPENSION, EMULSION, BULK OR SOLUTION POLYMERIZATION METHODS. PVC RESINS CAN BE EXTRUDED, MOLDED OR CALENDARED INTO DIVERSE SHAPES, SIZES, AND COLORS. MECHANICAL CHARACTERISTICS CAN BE CONTROLLED TO PRODUCE FORMS THAT ARE RIGID, FLEXIBLE, OR IN A LIQUID FORM SUCH A LATEXES, PASTES AND ADHESIVES.

VINYL CHLORIDE BECAME OF INDUSTRIAL IMPORTANCE APPROXIMATELY FIFTY YEARS AGO WHEN SEMON (1933) DISCOVERED THAT THE POLYMER COULD BE CONVERTED INTO USEFUL ARTICLES BY PLASTICIZATION WITH PHTHALATE ESTERS. COMMERCIAL DEVELOPMENT BEGAN FIRST IN EUROPE AND THEN IN THE UNITED STATES IN THE LATE 1930's. IT WAS NOT UNTIL THE EARLY 1950's THAT WIDESPREAD CONSUMER APPLICATIONS DEVELOPED. PVC IS NOW A MATURE PRODUCT, AND ITS GROWTH RATE FALLS IN STEP WITH THE GROSS NATIONAL PRODUCT.

III. HEALTH HISTORY

ACUTE TOXICITY

VINYL CHLORIDE IS A STRONG ANESTHETIC AT 8-12% IN ANIMALS AND HUMANS. DEATH FOLLOWS RAPIDLY AFTER UNCONSCIOUSNESS SETS IN IF EXPOSURE IS NOT REDUCED QUICKLY (PATTY ET AL., 1930). NO MAJOR HISTOLOGICAL CHANGES WERE REPORTED AFTER 100 DAYS AT EXPOSURES OF 50,000 PPM (KUEBLER, 1964). REVERSIBLE LIVER EFFECTS AT 100-500 PPM LED TO A RECOMMENDATION OF A 50 PPM TWA EXPOSURE LIMIT (TORKELSON, OYEN, AND ROWE, 1961), BUT THE AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS ADOPTED INSTEAD A RECOMMENDATION BY YALE SCIENTISTS OF 500 PPM. THIS IS THE VALUE LATER ACCEPTED BY OSHA AND IT SERVED UNTIL 1974. LEHMAN AND FLURY (1943) TERMED VINYL CHLORIDE TO BE "ONE OF THE LEAST DANGEROUS OF THE CHLORINATED HYDROCARBONS".

THERE ARE NO OTHER KNOWN ACUTE HUMAN PHYSIOLOGICAL EFFECTS FROM VINYL CHLORIDE EXPOSURE. THE ODOR THRESHOLD IS ABOUT 1,000 PPM. THE HIGH HEAT OF VAPORIZATION CAUSES A SUBSTANTIAL PART OF A LARGE SPILL TO LIQUIFY AND PRESENTS THE DANGER OF FROSTBITE. VINYL CHLORIDE IS FLAMMABLE OVER THE RANGE OF 3.6-33% IN AIR, AND EXTREME CARE MUST BE TAKEN TO AVOID SPILLS AND LEAKS FOR THAT REASONS. MOST MEASUREMENT AND WARNING SYSTEMS WERE DESIGNED TO HOLD PLANT ATMOSPHERES BELOW THE FLAMMABLE LIMITS. RETROSPECTIVE ESTIMATES OF TYPICAL TIME-WEIGHTED AVERAGE PERSONAL EXPOSURES FOR POLYMERIZATION WORKERS IN ENGLAND HAVE BEEN ESTIMATED (BARNES, 1980) AS FOLLOWS:

1945 TO 1955	-	1,000 PPM (OR ABOVE)
1955 TO 1960	-	400 TO 500
1960 TO 1970	-	300 TO 400
MID 1973	-	150
1975	-	5

IN SOME JOBS, PARTICULARLY THE CLEANING OF POLYMERIZATION REACTORS, EXPOSURES IN THE THOUSANDS OF PPM RANGE WERE EXPERIENCED FOR SHORT PERIODS. (SEE PURCHASE, ET AL, 1985 AND BARR, 1986 FOR REVIEWS OF THE TOXICITY OF VC).

CHRONIC HEALTH EFFECTS

THE FIRST CLEAR INDICATION OF CHRONIC HEALTH PROBLEMS ASSOCIATED WITH VC CAME IN THE 1960'S IN MEN WHO ENTERED VC POLYMERIZATION

VC-RELATED CASES (FALK, ET AL 1981), BUT THIS TASK WAS TAKEN OVER FIRST BY JOHN STAFFORD OF ICI, ENGLAND (FOREMAN, ET AL 1985) AND LATER BY BRIAN BENNETT ALSO OF ICI. THE 1986 UPDATE OF VC-RELATED ASL CASES SHOWS A TOTAL OF 38 CASES IN THE UNITED STATES AND 120 WORLDWIDE. ALL OF THESE CASES INVOLVE HIGH OCCUPATIONAL EXPOSURES TO VC.

THE AVERAGE ASL LATENCY PERIOD (YEARS FROM FIRST EXPOSURE TO DIAGNOSIS) IN THE UNITED STATES HAS BEEN 25 YEARS, BUT WITH A MEDIAN OF ABOUT 22 YEARS. THE LATENCY PERIOD IN EUROPE, PARTICULARLY IN GERMANY, HAS BEEN SOMEWHAT SHORTER, APPROXIMATELY 19 YEARS. ALL THE U.S. OCCUPATIONAL CASES, AND ALMOST ALL SUCH CASES IN THE REST OF THE WORLD ARE CLOSELY ASSOCIATED WITH THE JOB OF REACTOR CLEANING, WHICH WAS ONCE DONE MANUALLY AT THE END OF THE POLYMERIZATION CYCLE. THERE IS CLUSTERING OF CASES IN RELATIVELY FEW PLANTS AND THE MAJORITY OF PLANTS HAVE HAD NO CASES. DIFFERING WORK PROGRAMS AND JOB PROGRESSIONS MAY HAVE HAD SOME EFFECT ON REDUCING RATES AT VARIOUS PLANTS.

AN INDUSTRY-SPONSORED EPIDEMIOLOGICAL SURVEY OF WORKERS IN THE VC/PVC INDUSTRY COVERED 8,384 MEN WITH AT LEAST ONE YEAR OF EXPOSURE BEFORE 1973 (TABERSHAW AND GAFFEY, 1974). THE EXPECTED EXCESS OF ASL WAS FOUND. THERE WAS ALSO SUGGESTIONS OF AN EXCESS OF CANCERS AT OTHER SITES. THIS STUDY WAS EXPANDED TO 10,173 WORKERS (COOPER, 1981), WHERE SUGGESTED EXCESS OF BRAIN AND RESPIRATORY CANCERS CONTINUED TO BE SEEN WITHOUT, HOWEVER, AN ASSOCIATION BETWEEN THE BRAIN CANCER AND EXPOSURE. IN ADDITION, MOST OF THE LUNG CANCER CASES COME FROM THE SAME FACILITY, WITH MANY PLANTS HAVING NO CASES. A FOLLOW-UP STUDY OF THIS EXPANDED COHORT TO DETERMINE THE STATUS OF THE WORKERS AS OF THE END OF 1980 IS UNDERWAY.

SEVERAL STUDIES HAVE BEEN MADE OF THE GENERAL POPULATION USING ASL AS THE MARKER DISEASE IN AN EFFORT TO DETECT AN ASSOCIATION WITH POSSIBLE ENVIRONMENTAL EXPOSURE TO VC. THERE WAS NO ASSOCIATION WITH LIVING NEAR A PLANT MANUFACTURING OR USING VC IN THE GENERAL U.S. SURVEY CONDUCTED BY THE CENTER FOR DISEASE CONTROL (POPPER ET AL, 1978; FALK, ET AL, 1981). BRADY ET AL, (1977) SURVEYED 26 ASL DEATHS IN NEW YORK STATE BETWEEN 1970 AND 1975, AND FOUND FIVE WHO LIVED NEARER PLANTS HANDLING VC THAN DID THEIR MATCHED CONTROLS, BUT COULD NOT ESTABLISH A DIRECT CONNECTION WITH THE DISEASE TO EXPOSURE. TEN CASES OF ASL IN WISCONSIN WERE EXAMINED FOR POSSIBLE CONNECTION WITH VC EXPOSURE, AND NONE WAS FOUND (FIECHTNER ET AL, 1976). BAXTER ET AL,

(1977) FOUND NO RELATIONSHIP BETWEEN DISTANCE OF RESIDENCE FROM VC EMITTERS AND THE 47 CASES OF ASL IN THE GENERAL POPULATION OF GREAT BRITAIN REPORTED IN 1963-1973. A LATER UPDATE (BAXTER ET AL, 1980) FOUND ONE CASE WHERE THE PERSON HAD LIVED THE LAST SIX YEARS OF HIS LIFE NEAR A PVC PLANT AND THREE CASES WHERE THE MEN HAD WORKED IN THE PLASTICS FABRICATING INDUSTRY, BUT FOR WHOM THERE WERE NO RECORDS TO INDICATE EXPOSURE TO VC.

THE LACK OF RELATIONSHIP BETWEEN RESIDENCE NEAR VINYL CHLORIDE OPERATIONS AND CASES OF UNKNOWN ETIOLOGY WAS CONFIRMED. SARIC ET AL, (1976) STUDIED THE DEATHS DURING THE YEARS 1968-1971 IN AN AREA SURROUNDING A PVC PLANT THAT HAD BEEN IN OPERATION SINCE 1949 AND IN WHICH THREE WORKERS HAD DIED OF ASL. NO RELATIONSHIP WAS FOUND FOR LIVER OR FOR LUNCH OR BRONCHIAL CANCER AND PLACE OF RESIDENCE FOR THE GENERAL POPULATION. A SIMILAR STUDY FOR COMMUNITIES NEAR A SWEDISH PLANT THAT HAD OPERATED SINCE 1945 AND HAD FOUND FOUR ASL CASES SHOWED (ELINDER AND PERSHAGEN, 1978) NO UNEXPECTED ELEVATION OF FETAL MORTALITY, DEATHS FROM ALL CANCERS, OR CANCER OF THE LIVER OR LUNGS DURING THE YEARS 1961-1984. 'PANCREATIC CANCER' IN MALES WAS ELEVATED IN THE AGE GROUP OVER 60. ALL ASL CASES IN HOLLAND SINCE 1950 (27 CASES) WERE STUDIED, AND NONE HAD ANY TRACEABLE CONTACT WITH VC (DALDERUP ET AL, 1976). ITURRA (1976) OBSERVED AN EXCESS OF CANCER DEATHS IN A CITY IN CANADA WITH A PVC PLANT COMPARED TO A SIMILAR NEARBY CITY. THIS DIFFERENCE WAS PRINCIPALLY FOUND IN MALES AGED 20 TO 64, WHICH IS NOT INDICATIVE OF A GENERAL POLLUTION EFFECT. THE AUTHOR DREW NO CONCLUSION AS TO WHY THE CONDITION EXISTED.

REPRESENTATIVES OF THE ENVIRONMENTAL PROTECTION AGENCY HAVE STATED THAT IT HAS BEEN UNABLE TO ESTABLISH A LINK BETWEEN LIVING NEAR VC MANUFACTURING AND USING PLANTS AND ASL.

THERE ARE ABOUT 20 CASES OF ASL PER YEAR IN THE UNITED STATES THAT CANNOT BE ASCRIBED TO ONE OF THE KNOW CAUSES OF THE DISEASE. THERE ARE ALSO ABOUT 5 IN EUROPE EACH YEAR. ACCORDINGLY, THERE WILL BE ONE CASE OF ASL AMONG THE 5 MILLION - 5 MILE NEIGHBORS OF VC/PVC FACILITIES ABOUT EVERY TWO YEARS BY CHANCE ALONE. THIS HAS BEEN SEEN IN THE STUDIES IN NEW YORK BY BRADY, ET AL, (1970), AND IN CONNECTICUT (HEATH AND LANDRIGAN, 1974). THESE STATES HAVE CANCER REGISTRIES, WHICH ARE OF GREAT VALUE. IN ONE CASE, A JURY AWARD WAS MADE TO THE ESTATE OF AN INDIVIDUAL WHO DIED OF ASL, AND WHO HAD LIVED THE LAST FOUR YEARS OF HIS LIFE NEAR A PVC PLANT. INASMUCH AS THAT PERSON ALSO HAD OCCUPATIONAL EXPOSURE TO VC AND EXPOSURE TO OTHER ASL CAUSATIVE AGENTS, IT CANNOT BE CONCLUDED THAT AMBIENT VC EXPOSURE CAUSED HIS ASL (IN RE GRASSO, CIVIL ACTION NO. 78-1562, D.N.J.).

A THOROUGH STUDY (CHIAZZE, ET AL, (1977), CHIAZZE, (1980) OF MORE THAN 15,000 EMPLOYEES OF PVC FABRICATORS FOUND NO EVIDENCE OF VC-RELATED HEALTH EFFECTS IN THAT GROUP, WHICH WAS ESTIMATED TO HAVE BEEN EXPOSED TO AT LEAST 15 PPM VC FOR MANY YEARS.

THE DISEASE ASL IS OFTEN DIFFICULT TO DIAGNOSE (BLOCK 1974; HEATH, FLAK AND CREECH, 1975), IS ALMOST INVARIABLY FATAL WITHIN A SHORT TIME, AND PRESENTS A VARIETY OF SYMPTOMS, INCLUDING PORTAL FIBROSIS AND HYPERTENSION WITH SPLENOMEGALY AND VARICES, PROLIFERATION OF THE SINUSOIDAL LINING, MEGALOCYTOSIA AND THROMBOCYTOPENIA (THOMAS AND POPPER, 1975; GEDIGK ET AL, 1975). METASTASIS IS FREQUENTLY INVOLVED. THESE SYMPTOMS ARE VERY SIMILAR TO THOSE SEEN IN THE MOUSE (SCHAFFNER, 1978) AND RAT (FERON AND KREES, 1979) AND THE PATHOLOGY ALSO IS SIMILAR (GORDON ET AL, 1975). NO REALLY ADEQUATE EARLY WARNING TESTS HAVE BEEN DEVISED (WHELAN ET AL, 1976; LANGBEIN ET AL, 1983; TAMBURRO AND GREENBERG, 1981), ALTHOUGH THE GAMMAGLUTAMYL TRANSPEPSIDASE TEST IS PROMISING, TOGETHER WITH ICG CLEARANCE AND SGOT. RADIOGRAPHIC LIVER SCANS AND TOMOGRAPHY AND SONOGRAPHY (KOISCHWITZ ET AL, 1981) ARE SAID TO BE USEFUL CONFIRMATORY TESTS.

IN SUMMARY, VC IS A CLASSICAL PROCARCINOGEN, AND IS CLEARLY A HUMAN CARCINOGEN, CAUSING ASL IN A SMALL PERCENTAGE OF HIGHLY-EXPOSED WORKERS. THERE IS SUGGESTIVE EVIDENCE THAT IT MAY BE A WEAK GENERAL CARCINOGEN AT HIGH CONCENTRATIONS, PERHAPS THROUGH AN IMMUNOSUPPRESSIVE MECHANISM, BUT MORE DATA ARE REQUIRED TO CONFIRM THIS SUSPICION. SEVERAL STUDIES OF LARGE POPULATION HAVE NOT SHOWN A CONNECTION BETWEEN GENERAL AMBIENT EXPOSURE AND AN INCREASED INCIDENCE OF CANCER.

IV. FEDERAL REGULATION OF VC/PVC INDUSTRY

THE PRIMARY FEDERAL AGENCIES REGULATING THE VC/PVC INDUSTRY ARE THE OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA), WHICH IS PART OF THE U.S. DEPARTMENT OF LABOR, THE U.S. ENVIRONMENTAL PROTECTION AGENCY (EPA), AND THE FOOD AND DRUG ADMINISTRATION (FDA). OSHA REGULATION FOCUSES ON WORKER HEALTH WHILE EPA ADDRESSES THE CONTROL OF CHEMICALS OUTSIDE THE WORKPLACE. FDA OVERSEES USES OF PVC THAT INVOLVE FOOD, DRUGS, COSMETICS, AND MEDICAL DEVICES.

A. THE OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION

THE ALLOWABLE OCCUPATIONAL EXPOSURE FOR VINYL CHLORIDE OF 1 PPM ON AN 8-HOUR TIME WEIGHTED AVERAGE (TWA) IS SET BY THE

OSHA WORKPLACE STANDARDS AT 29CFR1910.1017. THIS WAS ADOPTED IN 1974, AFTER EXTENSIVE PUBLIC HEARINGS, AND BECAME EFFECTIVE IN APRIL 1975. OSHA FIRST SET AN EMERGENCY TEMPORARY STANDARD OF 50 PPM AND PROPOSED A PERMANENT LIMIT OF NONDETECTABLE EXPOSURE BY A TEST SENSITIVE TO 1 PPM. OSHA THEN PROMULGATED A FINAL STANDARD OF AN 8-HOUR TWA OF 1 PPM, AND A 15-MINUTE CEILING OF 5 PPM.

IN BRIEF, THE REGULATION SETS:

1. A LEVEL OF 0.5 PPM VC BELOW WHICH NO ACTION IS REQUIRED. THIS GENERALLY EXEMPTS MOST PVC FABRICATION PLANTS AND LABORATORIES AND MANY MONOMER PLANTS.

2. A REGULATED AREA WHERE EXPOSURES ARE ABOVE 0.5 PPM WHICH RESTRICTS ENTRY TO AUTHORIZED PERSONS.

3. MEDICAL EXAMINATION REQUIREMENTS AND EXPOSURE RECORD RETENTION FOR SPECIFIED EMPLOYEES.

4. A LIST OF ACCEPTABLE RESPIRATORS.

5. MONITORING AND ALARM SYSTEMS FOR THE WORKPLACE, AND ROUTINE MEASUREMENT OF WORKER EXPOSURE.

6. LABELING AND SIGNS FOR REGULATED AREAS AND CONTAINERS OF VINYL CHLORIDE AND PVC.

7. WORK PROCEDURES FOR HAZARDOUS OPERATIONS.

8. TRAINING PROGRAMS FOR EMPLOYEES.

OSHA ALSO HAS A HAZARD COMMUNICATION STANDARD (HCS), 29CFR1910.1200, WHICH PROVIDES LABELING REQUIREMENTS COMPLEMENTARY TO THE OSHA VINYL CHLORIDE STANDARD. ARTICLES MADE FROM PVC ARE EXEMPT FROM LABELING REQUIREMENTS UNDER THE STANDARD.

B. ENVIRONMENTAL PROTECTION AGENCY

EPA REGULATES THE RELEASE OF VINYL CHLORIDE UNDER SEVERAL STATUTES, INCLUDING THE CLEAN AIR ACT, CLEAN WATER ACT, SAFE DRINKING WATER ACT, RESOURCE CONSERVATION AND RECOVERY ACT

(RCRA), COMPREHENSIVE ENVIRONMENTAL RESPONSE, COMPENSATION AND LIABILITY ACT (CERCLA OR SUPERFUND), AND THE TOXIC SUBSTANCES CONTROL ACT (TSCA).

1. AIR STANDARD (40CFR61.60)

THE EPA STANDARD ESTABLISHED IN 1976 SPECIFIED THE FOLLOWING CONDITIONS:

A. FUGITIVE EMISSIONS CONTROLS BY LEAK PATROLS AND DESIGN STANDARDS FOR PUMP AND COMPRESSOR SEALS, AGITATORS, AND LOADING DEVICES.

B. WORK PRACTICES FOR VESSEL OPENINGS AND SAMPLING.

C. STRIPPING REQUIREMENTS FOR RESIDUAL MONOMER IN RESINS AND WASTEWATER.

D. ABATEMENT OF SPECIFIED POINT SOURCE EMISSIONS TO 10 PPM.

E. PROHIBITION OF RELIEF VALVE DISCHARGES, EXCEPT FOR EMERGENCIES.

F. EXTENSIVE MONITORING, REPORTING AND RECORDKEEPING REQUIREMENTS.

G. SPECIFIC ANALYTICAL PROCEDURES.

EPA ESTIMATED THAT THIS STANDARD WOULD RESULT IN A 95% REDUCTION OF VC EMISSIONS TO THE ATMOSPHERE FROM VC/PVC MANUFACTURING PLANTS AND REDUCE THE 5 MILE ANNUAL AVERAGE VC AMBIENT AIR CONCENTRATION FROM 17 PARTS PER BILLION (PPB) TO LESS THAN 1 PPB.

2. WATER REGULATIONS

VINYL CHLORIDE IS LISTED AS A PRIORITY POLLUTANT UNDER SECTION 307(A) OF THE CLEAN AIR ACT, AND A WATER QUALITY CRITERIA DOCUMENT HAS BEEN PREPARED. THIS SUBJECTS VC AND PVC MANUFACTURING PLANTS TO SPECIAL CONSIDERATIONS WHEN WASTE WATER DISCHARGE PERMITS ARE ISSUED PURSUANT TO EPA REGULATIONS.

AS PART OF ITS REGULATION OF CARCINOGENS IN DRINKING WATER, EPA HAS PUBLISHED A FINAL RECOMMENDED MAXIMUM CONTAMINANT LEVEL (RMCL - A NON-BINDING GUIDELINE) FOR VC IN DRINKING WATER OF ZERO (SEE 50FR46880, NOVEMBER 13, 1985 FOR THIS AMENDMENT TO 40CFR 141.50).

HOWEVER, EPA INDICATED THAT A "JUSTIFIABLE" WAY TO DETERMINE THE ABSENCE OF VINYL CHLORIDE WOULD BE BY SETTING A DEFINED, STATE-OF-THE-ART DETECTION LIMIT SENSITIVE TO APPROXIMATELY 1 PPB. (49FR24,330, 24,347 - JUNE 12, 1984). EPA HAS ALSO PROPOSED A MAXIMUM CONTAMINANT LEVEL OF 1 PPB FOR VINYL CHLORIDE IN DRINKING WATER. (SEE 50FR46,902 - NOVEMBER 13, 1985).

3. WASTE AND SPILL REGULATION

THE EPA ISSUED A RULE UNDER WHICH CERTAIN VC MANUFACTURING DISTALLATION RESIDUES ARE LISTED AS HAZARDOUS WASTES WHEN DISPOSED (49FR5308). THIS RULE REQUIRES THAT ALL SUCH WASTES ARE TO BE DISPOSED OF ONLY BY RCRA-APPROVED PROCEDURES.

WHEN DISPOSED OF, COMMERCIAL GRADE VC IS CLASSIFIED AS A HAZARDOUS WASTE UNDER THE RESOURCE CONSERVATION AND RECOVERY ACT (RCRA), BECAUSE OF ITS TOXIC AND IGNITABLE CHARACTERISTICS. ANY DISPOSAL IS SUBJECT TO REGULATION UNDER RCRA.

EPA HAS PROPOSED ADDITIONAL RCRA REGULATIONS (51 FR 21648, JUNE 13, 1986) WHICH APPLY TO ALL WASTES CONTAINING VC. THESE PROPOSED REGULATIONS DEFINE WASTES AS HAZARDOUS WHEN THE VC LEVEL IN THE EXTRACT BY A SPECIFIED TEST METHOD EXCEEDS 50 PPB. CONGRESS HAS SPECIFIED AN INTERIM 1 POUND REPORTABLE QUANTITY FOR VINYL CHLORIDE. RELEASES TO THE ENVIRONMENT IN EXCESS OF 1 POUND ARE REGULATED UNDER CERCLA.

4. NEW PRODUCT MANUFACTURE

THE EPA ALSO ADMINISTERS THE TOXIC SUBSTANCES CONTROL ACT (TSCA) WHICH ESTABLISHES HEALTH AND ENVIRONMENTAL REGULATIONS FOR BOTH NEW AND EXISTING SUBSTANCES. NO ONE MAY MANUFACTURE OR USE A SUBSTANCE WHICH IS NOT ON THE AGENCY'S OFFICIAL INVENTORY, UNLESS THE PREMANUFACTURING NOTICE PROCEDURES ARE FOLLOWED.

BEEN REDUCED BY 99.99% (RATHER THAN THE 95% ESTIMATED BY EPA), THE ACTUAL RISK IS LESS THAN 0.1 CASE OF CANCER IN THE NEXT 70 YEARS AMONG THE 5 MILLION PRESUMED TO BE EXPOSED TO VC FROM LIVING WITHIN 5 MILES OF A VC/PVC FACILITY.

DR. RICHARD WILSON OF HARVARD (1979) HAS ATTEMPTED TO HELP PEOPLE UNDERSTAND THIS METHOD OF STATING THE RISKS OF EVERY DAY OCCURRENCES. EACH OF THE FOLLOWING ACTIVITIES FOR EXAMPLE, IS PREDICTED TO RESULT IN ONE DEATH PER MILLION PEOPLE: SMOKING 1.4 CIGARETTES (DUE TO CANCER, HEART DISEASE); DRINKING 1/2 LITER OF WINE (DUE TO CIRRHOSIS OF THE LIVER); TRAVELING 6 MINUTES BY CANOE, 10 MILES BY BICYCLE, 300 MILES BY CAR, OR 1,000 MILES BY JET (DUE TO AN ACCIDENT); AND HAVING ONE CHEST X-RAY TAKEN IN A GOOD HOSPITAL (DUE TO CANCER BY RADIATION).

WE CONCLUDE, THEREFORE, THAT THERE IS NO BASIS FOR CONCERN BY PERSONS LIVING NEAR VC-USING OR PRODUCING FACILITIES FOR ANY HEALTH EFFECTS FROM EXPOSURE TO AMBIENT CONCENTRATIONS OF VC NOW BEING EXPERIENCED.

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TECHNICAL INFORMATION

POTENTIAL EFFECTS OF VINYL CHLORIDE ON HUMAN OFFSPRING

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POTENTIAL EFFECTS OF VINYL CHLORIDE ON HUMAN OFFSPRING

Summary

Vinyl chloride monomer (VCM) is one of the most intensely studied chemicals that is used in industry today. Although it is a known human and animal carcinogen, many other alleged health concerns are not supported by animal or human data. Early or preliminary studies of offspring by some investigators have suggested that VCM causes birth defects and miscarriages. However, critical peer review and subsequent research have shown that there is no basis for such fears.

What we do know from decades of research on VCM is that studies with pregnant animals have shown that VCM can undergo placental transfer and, at high levels, cause cancer in their offspring. Other animal studies have indicated that VCM does not produce any mutations in subsequent generations.

Furthermore, numerous animal and human studies have shown that VCM does not cause birth defects or reproductive effects. While some human studies have claimed that VCM causes birth defects and miscarriages, close examination by numerous experts have found these early reports to be seriously flawed and not supportive of an effect.

Transplacental Carcinogenicity

The carcinogenic potential of VCM is well recognized. Many studies have shown that it causes cancer in a number of different animal species and conditions (Viola et al., 1971; Maltoni and Mehlman, 1984; Keplinger et al., 1975). The fact that VCM causes angiosarcoma in man is evidenced by numerous studies (Creech and Johnson, 1974; Delorme and Theriault, 1978; Fox and Collier, 1977; Noria et al., 1976). Studies by Maltoni (1974) and Maltoni and Mehlman (1984) also have shown that VCM is transplacental carcinogen. When pregnant rats were exposed to high levels of VCM, an increased incidence of cancer was observed in the offspring.

Animal Studies of Developmental and Reproductive Effects

The exposure of males and/or females to toxic chemicals prior to or following conception may provide an opportunity for deleterious effects to occur. Because VCM has been found to cause mutation in some non-reproductive cells (Bartsch et al., 1976); McCann et al., 1975; Rannug, 1974; Drevon and Kuroki, 1979; Basler and Rohrborn, 1980), studies have been conducted

to determine whether VCM can cause mutations in germ cells (egg, sperm) that could effect viability or produce body anomalies.

In 1976, Anderson et al. examined VC mutagenicity in fertile male mice using the Dominant Lethal Assay. These investigators exposed mice to VC for 6 hours per day for 5 days in concentrations of 3,000, 10,000 and 30,000 ppm. To assure that their test system was functional, two known mutagens were used as positive controls (cyclophosphamide and ethyl methane sulphonate). No mutagenic effects were observed with VCM at any stage of spermatogenesis while dominant lethal effects were observed with the positive controls.

This early work of Anderson et al. (1976) was later confirmed by Himeno et al. (1983) in a dominant lethal study using two different exposure conditions. One group of CD-1 mice was exposed for four hours per day during five consecutive days to 10,000 ppm of VC, and the second group of mice was exposed four hours per day, five days per week over a 10 week period to 5,000 ppm VC. Again, no dominant lethal mutations were observed following VC exposure.

Using another approach, Peter and Ungvary (1980) studied the germinal mutation potential of VCM in the mammalian spot test. Following the mating of inbrad females with rotated-bred males, they exposed groups of females on the 10th day of pregnancy to 0 or 4,600 ppm of VCM for 5 hours or to cyclophosphamide, the positive control. Cyclophosphamide caused a significant increase in the number of white or colored spots, while VCM did not cause an increase in color spots. Also, there was no effect on litter size at birth or at the end of the 3-5 week observation period. The authors concluded that although gene mutation had been reported in fruit flies exposed to VCM, there is no evidence that VCM causes mutations in mammals.

Other studies have focused on the potential effects of maternally inhaled VCM on embryonal and fetal development. In studies by John et al. (1977 and 1981) groups of rats and rabbits were exposed to 0, 50, 500 or 2,500 ppm VC for 7 hours per day during the critical development phase (organogenesis) for each species (days 6 to 15 for mice/rats and days 6 to 18 for rabbits). Additional groups of pregnant females were simultaneously exposed to VCM (2,500 ppm for rats and rabbits and 50 and 500 ppm for mice) plus 15% ethanol in their drinking water over the same period. Ethanol, a known reproductive toxin, was used because it is known to block the primary metabolic pathway for VCM. Although high levels of VCM caused frank maternal toxicity, no significant embryonal toxicity, fetal toxicity or teratogenic effects were observed. Greater maternal embryonal and fetal toxicity but not teratogenic effects were observed following exposure to ethanol and VCM. Because of the limited control data which was provided, it

could not be determined whether this increase in toxic response was largely due to ethanol alone or the inhibition of VCM metabolism (detoxification) of VCM. However, these studies have further underscored the concerns associated with ethanol consumption during pregnancy.

Ungvary et al. (1978) also carried out a detailed study in rats of the potential of VCM to cause birth defects. In their initial experiments, they exposed females on the 18th day of pregnancy to either 2,000, 7,000 or 12,000 ppm VCM for 2.5 hours. Analyses of the maternal blood, amniotic fluid, and fetal tissue demonstrated that VCM could be transferred across the placental barrier from the mother to the fetus. In subsequent experiments they exposed females to 1,500 ppm VCM 24 hours per day over three different periods of their pregnancy and looked for fetal effects. No birth defects occurred as the result of VCM exposure. Fetal toxicity was evident in only one of the three groups.

All of these studies cited above are remarkable in two ways. First, the exposure levels used in these experiments are for the most part several orders of magnitude above levels of VCM in the workplace and many orders of magnitude above that in the community. Such exposure would only be encountered under rare, catastrophic situations and would likely be short-lived relative to these studies. Secondly, even though the females were exposed to very high levels of VCM, the evidence indicates that VCM does not cause dominant lethal mutations or birth defects. Furthermore, these data indicate that the levels of VCM in the workplace and the ambient air are not of concern to the mother, embryo or developing fetus.

Human Studies of Mutagenic, Developmental and Reproductive Effects

Numerous studies of human populations have been conducted to evaluate the potential of VCM to cause birth defects or other reproductive effects. The studies in humans have been carried out with workers employed in polyvinyl chloride (PVC) production or fabrication facilities, and other studies have examined communities in which a PVC production or fabrication facility is located. In contrast to the well controlled conditions of the animal studies in the laboratory, these human studies have had to contend with many confounding factors such as smoking, alcohol, maternal age, maternal infections, lack of exposure information, etc. The ability or inability of the author to deal with these variables has frequently affected the utility of the study and the validity of the authors' conclusions. Many of these problems have been encountered in studies of the effects of VCM on human reproductive outcome.

One of the earliest studies of the potential of VCM to influence human reproductive outcome was conducted by Infante et al.

(1976a). Fetal loss among wives of VCM exposed workers was studied by interviewing husbands employed in VCM polymerization and PVC fabrication facilities in Ohio. No interviews were conducted with wives and no maternal age information was obtained. Among the workers potentially exposed to VCM, fetal loss was observed to be 15.8% compared to 8.8% among the unexposed controls. This effect was observed in men younger than 30 years old.

The above study by Infante et al. (1976a) has been reviewed by a number of investigators and has been found to be inadequate and misleading (Stallones, 1987; Clemmensen, 1982; Haas and Schottenfeld, 1979). The credibility and validity of the conclusions from this survey have been impaired by the absence of maternal interviews and information on maternal age, smoking or alcohol consumption and inappropriate statistical treatment of the data. Furthermore, the authors presumed that all workers had a significant exposure to VCM.

In addition to the above problems, Paddle (1977) asked for clarification of the age adjustment procedure used by Infante et al. (1976a). Infante et al. (1976b) tried to explain the age adjustment procedure, but in doing so revealed that there were four different age-based categories in the original study. The alleged effect upon fetal loss could only be detected in the 25-29 year old age group. This specific response could not be explained on any basis since one normally expects the overall incidence in fetal defects to increase with increasing parental age. Stallones (1987) noted that the fetal loss in the 25-29 year old unexposed group was substantially lower than for any other age group within the unexposed population. Stallones concluded that the study was seriously flawed by the lack of internal consistency, and the study should be discarded on that basis alone.

A recent study was conducted by Lindbohm et al. (1985) which examined spontaneous abortions among women employed in the plastics industry. The study was conducted in Finland during the years of 1973 to 1980 on workers who belonged to the Union of Chemical Workers, and whose potential exposure were to a variety of plastics materials including VC, styrenes, butadienes, acrylonitriles, and polyurethanes. Cases were selected from women who had been union members during their first trimester of pregnancy, and selections were done so that a subject was primarily exposed to only one type of plastic or monomer. Controls were selected from women not having had a spontaneous abortion. Exposure (yes or no only, no quantitation) was determined by having the physicians who treated a given patient fill out a questionnaire.

When the data from Lindbohm et al. (1985) was examined, no difference in the rate of spontaneous abortions could be

determined between the case and control populations for women working with PVC or PVC thermal degradation products. Although the number of women in the study was low, the statistical power of this study was sufficient to detect a two-fold increase in fetal wastage. Therefore, this study indicated that there was no association between VCM exposure and the occurrence of spontaneous abortion.

In addition to these studies of workers, a number of investigators have examined the occurrence of birth defects in people from communities surrounding plants which use VCM to make PVC. Infante (1976) has published a study of birth defects in a number of communities in Ohio which contained PVC production facilities. He has reported that the rate of birth defects in the communities producing PVC was greater than in neighboring communities that did not produce PVC.

One obvious problem with this Infante study was the total lack of any quantitative exposure data as in his previous study. Besides the lack of exposure data, no effort was made to correlate the rate of defects with distance from production facilities. If the defects were correlated with VC exposure, then one might expect a greater frequency of birth defects closer to the production sites with lower frequencies away from a given facility. When Edmonds (1976) examined the Infante data as frequency of defects vs. distance from a given facility, he could find no difference between the exposed and unexposed populations. This observation weakens any putative link between observed birth defects and VC exposure.

Bias is a major concern in the Infante (1976) study because of the way he examined and combined populations for analysis. There are three communities in the study which contained PVC plants, and those cities are Painesville, Ashtabula, and Avon Lake. For comparison communities without PVC plants, 10 cities were chosen for their proximity to the exposed communities. Of the 10 control communities, two of them (Geneva and North Ridgeville) had higher malformation rates than the exposed communities containing PVC facilities. Since North Ridgeville was proximate to Avon Lake, according to Infante, the North Ridgeville data were combined with data from Painesville, Ashtabula, and Avon Lake even though it did not contain any VC operations within its boundary. This combination of data made a more striking difference between the exposed and unexposed groups whereas the difference would have been diminished if North Ridgeville was properly placed with the unexposed data. This gerrymandering in the case of North Ridgeville is of particular concern since it is 10 miles from the nearest plant and since its southwest location is not in the line of typical prevailing winds.

Another point to examine in the Infante study is the question of the CNS defects compared to the control population.

Edmonds' (1976) examination of the data could not find an association between VC exposure and reported CNS defects. Edmonds also believes that localized increases can occur sporadically, but these localized increases do not always signal a potential problem. Moreover, Oakley (1981) examined CNS defects nationwide as part of an ongoing activity of the Center for Disease Control (CDC). Oakley found that the frequency of CNS defects has been decreasing in all regions of the country at a steady rate, and the decrease has been occurring since the 1930's.

Since CNS defects were a major concern of the Infante study, Edmonds et al. (1978) examined the presumed relationship of this defect to VC exposure in offspring of Hanawha County, West Virginia residents. In contrast to Infante et al. (1976a) and Infante (1976), Edmonds examined geographic distribution of CNS malformations in relationship to the PVC plant and attempted to quantitate ambient VC levels in the community. When Edmonds examined the malformation rates from matched pairs of exposed and unexposed residents, he could find no correlation with distance from the plant site. Although he did find some suggestive patterns in which more exposed were northeast and more unexposed south of the plant, no correlation was evident when wind and particulate deposition patterns were examined. VC was measured in the community after one large release, and levels of 0.1 to 0.2 ppm VC were found in grab samples to the northeast of the plant site. Edmonds was also able to determine emission data during his study period (1970-1974). He found that the rate of CNS malformations was declining during the study period, and the decline in CNS defects preceded the decrease in VCM emissions from the plant sites by about nine months. Edmonds concluded from his study that he could not find any correlation between any wind-borne pollutants and the incidence of CNS defects in Kanawha County, West Virginia.

Theriault et al. (1981 and 1983) also examined the presumed relationship of community VC exposure and the appearance of a variety of birth defects. Their study was conducted in Shawinigan, Ontario, Canada during the period of 1966 to 1979. In addition to examining the geographic distribution of defects and attempting to quantitate ambient VC levels in the air like Edmonds et al. (1978), this study also examined the seasonal variations in VC levels and malformation rates.

Geographic distribution of malformation rates were examined in Shawinigan and comparison communities by using school district boundaries. The rate of defects was constant for each school district in Shawinigan as well as the comparison areas, and the ratio of observed to expected defects was always close to one (1). The ratio of observed to expected defects was approximately one (1) for the school district with the PVC plant as well. Further comparisons were made between school districts

with high and low VC levels, and no correlation could be found between the exposed and unexposed groups for CNS or any of the other malformations. One group, however, did show an increase in malformation rates, but it was located furthest from the plant and would have had the lowest degree of exposure. Consequently, no association was found between the geographic distribution of malformations and VCM exposure.

Although Theriault et al. (1983) reported that seasonal variations in birth defects corresponded with variations in atmospheric concentration, they concluded that a causal association between VCM and birth defects was not supportable. VCM concentration did not correlate with the distribution of birth defects and malformation rates were not high near the plant or in areas where VCM levels were the highest. Furthermore, districts with the highest rates were farthest from the plant.

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