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JAN 21 1980



Imperial
Chemical
Industries
Limited

Plastics
Division

Mr J T Seawell
Chemists Manufacturing Association
1825 Connecticut Av NW
Washington DC 20009
USA

Your ref.

Our ref

Tel ext

Date

JS/SEW/DSO-16

3162

11 January 1980

Dear Mr Seawell

PVC DUST

Thank you for the copy of your minutes of the meeting my colleagues and I attended on 28 November. We did enjoy our discussion with your VC Research Coordinators group and thank you for your friendly reception and hospitality.

Dr Adams had hoped to write to you concerning your PVC dust minute but regrettably he had to go off to Germany for a few days and he asked me to write and give you his views.

Getting to grips with this rather complex IOM study without previous immersion in it poses a number of problems for a secretary. I congratulate you on your write-up. Dr Adams, like myself, wonders whether a page of your notes has gone missing at the end of 4.0. You dealt in some detail with the "breathlessness" findings to which little importance is attached because the information is derived from a somewhat subjective examination by questionnaire. However you did not deal at all with the most important finding viz the effect of PVC dust on "lung function". While we in ICI are sceptical about the radiological findings, we accept fully the "lung function" results. Dr Adams did describe the lung function work in the following terms:

"FEV1 and FVC were inversely related to the index of PVC dust exposure. Dust effect was studied after standardization for age, height and weight by multiple regression. This demonstrated a small but significant dust effect on FEV and they were also able to demonstrate that the dust effect was most marked in cigarette smokers and not apparent in non-smokers. The magnitude of loss in FEV1 was small. After taking into account the effects of age, height and weight, the overall regression with dust index was - 0.0040 litres per dust index unit. This was equivalent to a loss of 52 ml of FEV1 for the mean dust index. An illustration of this would be equivalent to a loss for all men of 5.2 ml of FEV1 per year during exposure to the mean of the dust index over a period of 10 years. In cigarette smokers considered separately, this loss was 8.9 ml of FEV1 per year.

These figures may be compared with the mean losses in the group due to age or smoking. The mean loss due to age is 35 ml and loss due to smoking 20 cigarettes a day, 6.2 ml a year.

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PYC - This also showed a significant inverse relationship to dust index among currently exposed men.

Gas Transfer - No significant changes were noted in this test."

I realise you do revert to "lung function" in summary form at the end of 4.1 but you had not previously dealt with it at length whereas you do give an exhaustive report on the less significant breathlessness results. I hope the above comments will be helpful to you when you come to discuss confirmation of the minutes at your next meeting.

One small point which always amuses us. We are described as "IMPERICAL". More often IMPERIAL turns out as EMPIRICAL! Perhaps we are empirical and pragmatic in our approach and it does show through!! The word "imperial" is not popular these days and leads to many a psychological block and Freudian slip. (By the way in 6.0, the company name is VINATEX).

We hope you are well and look forward to meeting you again in the not too distant future.

With best wishes.

Yours sincerely



J Stafford
Division Manager
Health & Environment Protection

Dated: December 10, 1979.

Frank H. Madden,

*AID Advisory Committee Representative,
Joint Committee on Agricultural
Development, Board for International Food
and Agricultural Development.*

(79 Dec. 79-2000) Filed 12-17-79 9:44 am)

MAILING CODE 4710-02-01

DEPARTMENT OF LABOR

Occupational Safety and Health Administration

Occupational Exposure to Vinyl Chloride and Polyvinyl Chloride

AGENCY: Occupational Safety and Health
Administration, Department of Labor.

ACTION: Request for information on vinyl
chloride and polyvinyl chloride.

SUMMARY: This notice requests information on vinyl chloride (CAS No. 75-01-4) and on polyvinyl chloride (CAS No. 9002-86-2). Vinyl chloride (VC), a synthetic chemical, was once synthesized by the addition of hydrogen chloride to acetylene. Currently, the most common route of production is by the halogenation of ethylene. In this latter process, ethylene is reacted with hydrogen chloride and oxygen to give ethylene dichloride, which is subsequently cracked thermally to produce vinyl chloride and hydrogen chloride. Vinyl chloride is used primarily to produce polyvinyl chloride (PVC), a plastic resin, through conversion of the VC monomer into a polymer or copolymer form. Vinyl chloride is also used in the production of methyl chloroform and in the production of resins as a comonomer with vinylidene chloride. PVC is used in the manufacture of a variety of industrial and consumer products, such as containers, wrapping film, electrical insulation, and pipes.

In 1973, OSHA regulated vinyl chloride as a carcinogen, based primarily on evidence of excess liver angiosarcoma incidence following VC exposure (29 CFR 1910.1017). OSHA did not regulate exposure to PVC dust in the same standard. Therefore, it continues to be regulated as an inert or nuisance air contaminant (29 CFR 1910.1000, Table Z-3).

Research since 1973 has provided considerably more information concerning the carcinogenic and other toxic effects of VC and PVC exposure. OSHA is currently in the process of evaluating the available evidence pertaining to the potential occupational health hazards of VC and of PVC and, by this notice, is requesting information related to several important issues.

DATE: The information requested in this notice must be submitted in quadruplicate on or before February 10, 1980.

ADDRESS: The information requested in this notice should be submitted to the Docket Officer, Docket No. H-034, Room S6212, U.S. Department of Labor, OSHA, 200 Constitution Avenue, N.W., Washington, D.C. 20210, (202-523-7894).

FOR FURTHER INFORMATION CONTACT: Dr. Peter Infante, Office of Carcinogen Identification and Classification, Directorate of Health Standards Programs, Room N3718, U.S. Department of Labor, OSHA, 200 Constitution Avenue, N.W., Washington, D.C. 20210, (202-357-0325).

SUPPLEMENTARY INFORMATION:

Background

Vinyl chloride (C_2H_3Cl ; CAS No. 75-01-4; chloroethene) is a colorless gas at room temperature and pressure. After synthesis from VC monomer, polyvinyl chloride ($[C_2H_3Cl]_n$; CAS No. 9002-86-2; chloroethene homopolymer) is in the form of white or colorless granules. Residual vinyl chloride monomer can become trapped in the PVC particles. However, recent processing methods can reduce considerably the amount of residual VC trapped in the polymer resins.

The vinyl chloride industry can be divided into three major components: VC synthesis, VC polymerization, and PVC fabrication. It has been estimated that in the U.S., 15 plants manufacture vinyl chloride, 43 plants polymerize polyvinyl chloride, and at least 7,500 plants are engaged in the fabrication of PVC. During 1978, 3.47 million tons of VC and 2.94 million tons of PVC were produced.

The present permissible limit for occupational exposure to vinyl chloride is 1.0 ppm averaged over any 8-hour period, and 5.0 ppm averaged over any period not exceeding 15 minutes (29 CFR 1910.1017). For polyvinyl chloride dust, the standard is 15 mg/m³, averaged over any 8-hour period (29 CFR 1910.1000).

Laboratory Studies

Experimental bioassays have demonstrated the induction of cancer by VC at levels of exposure lower than those previously reported. In VC inhalation studies of rats, Maltoni (1) has induced liver angiosarcomas at 25 ppm and mammary carcinomas at one ppm. In addition, several different test systems have provided evidence for the mutagenic potential of VC, i.e., *E. coli*, *S. typhimurium*, *S. pombe*, insects, plants, and cultured mammalian cells (2). VC also has been demonstrated to have a

transplacental effect in rats. Inhalation exposure of pregnant rats to VC concentrations of 8,000 and 10,000 ppm have resulted in VC-dependent tumors in their offspring (1).

There is also evidence from animal studies that PVC may induce non-malignant respiratory disease. It has been reported that after seven months of exposure, rats and guinea pigs caged in work areas where sacks were being filled with PVC powder developed pulmonary pathological changes and granulomatous lesions containing foreign particles thought to be PVC dust (3).

Epidemiologic Studies

Several investigations of employee populations have indicated that VC/PVC exposure is associated with an increased carcinogenic risk to several organ sites in addition to the liver. A NIOSH retrospective cohort study of workers from four VC polymerization plants showed an excess number of deaths due to cancer of the liver, lung, lympho-hematopoietic system, and central nervous system (4).

Of further concern is the finding that the carcinogenic risk may extend to industries fabricating polyvinyl chloride. A proportionate mortality study of plastic workers in Great Britain, which included PVC fabricators, demonstrated a statistically significant excess of stomach cancer (5). Also, an excess of mortality from digestive system cancer among PVC fabricators of both sexes was found by Chiuzze et al. in the U.S. (6). Among white females in the study, observed deaths from breast and urinary tract cancers were also greater than expected. Waxweiler et al. performed a detailed study of the excess lung cancer risk previously observed at a synthetic plastics and rubber plant (7). Analysis of lung cancer cases by cell type demonstrated a greater frequency of adenocarcinomas (Type 3) and large cell undifferentiated (Type 4) cancer. Neither cell type is believed to be strongly associated with cigarette smoking. Of 12 chemicals analyzed, only PVC dust exposure proved to be statistically significant. The authors suggested that the excess of Type 3 and 4 lung cancer cases in this plant was related to PVC dust exposure.

A recent epidemiologic study suggests that PVC may be related to pneumoconiosis (8). Employees of a polyvinyl chloride production factory in Italy were submitted to chest X-ray examination. Twenty subjects were diagnosed as having pneumoconiosis. All of these cases had worked five or more years in departments with demonstrated PVC dust pollution. No

pneumoconiosis was observed in subjects who worked in areas free of PVC dust. These findings are consistent with earlier case reports of pneumoconiosis among workers exposed to PVC dust (9,10). The Institute of Oncology and Tumor Center in Bologna, Italy has demonstrated a higher frequency of cytologically abnormal sputum cells among the workers employed in the VC/PVC industry in contrast either to workers in the general chemical industry or to individuals who are heavy smokers (11).

Epidemiologic studies also exist which demonstrate the mutagenicity of VC in humans. Chromosomal aberrations in lymphocytes of male workers, in excess of the number observed in non-exposed workers, have been reported in several studies (2). Moreover, a study of miscarriages among the wives of men occupationally exposed to VC detected a significant excess in fetal mortality following the husband's exposure to VC (12).

Information requested on vinyl chloride and polyvinyl chloride

The data recently received by OSHA suggest that a reassessment of the known health effects of vinyl chloride and polyvinyl chloride is appropriate at this time. Additional information in several areas is needed before a reassessment can be completed. The requested information includes, but is not limited to, the following:

(1) Experimental test results for carcinogenicity of vinyl chloride at atmospheric exposures of less than 50 ppm.

(2) Studies of transplacental carcinogenic and teratogenic effects in humans or animals at any level of exposure to vinyl chloride.

(3) Experimental studies of carcinogenicity and other toxic manifestations for any level of polyvinyl chloride exposure. These effects should include, but are not limited to, mutagenicity, teratogenicity, embryotoxicity, and other transplacental effects, as well as cytotoxic and cytogenetic effects on sperm cells. To the greatest extent possible, complete information concerning the industrial source of the polyvinyl chloride, the size and characteristics of the particles, and exposure levels or concentrations of PVC and residual VC should be included for each study.

(4) Epidemiologic studies of either vinyl chloride or of polyvinyl chloride (i.e. cohort, cross-sectional, or case-control).

(5) Case reports and case series of brain, lympho-hematopoietic, lung, and

liver cancers by facility and relevant demographic variables, such as age, sex, race, date of diagnosis, date of death, date of first exposure, and length of exposure for either vinyl chloride or polyvinyl chloride.

(6) Mutagenicity study results of vinyl chloride or polyvinyl chloride as measured by the analysis of human body fluids, e.g., direct mutagenic testing with peripheral blood lymphocytes, non-disjunction in humans with YFF sperm test, and *in vivo* cytogenetics.

(7) Body burden measurements of vinyl chloride in humans.

(8) For operations involving polyvinyl chloride, the types of resin in use, the concentration of vinyl chloride trapped in the resin, and the concentration of VC and PVC dust in the atmosphere where individuals are working. This information should include estimates of the particles sizes and concentrations of particles that fall within the respirable range.

(9) In PVC bagging and milling operations, atmospheric levels of PVC and VC, monitoring devices used to detect VC, and the type of respirator protection program for individuals working in these operations. This should include the sensitivity and validity of analytic techniques in use.

(10) Types of occupations, job classifications, and industries where exposure to either VC or PVC at any level may occur, and the numbers of employees involved in each vinyl chloride and polyvinyl chloride exposure situation, separated by sex and race.

(11) Appropriate engineering controls, work practices, and personal protective equipment available to reduce levels of exposure to VC or PVC below the current standards, or to the lowest levels feasible.

SUBMITTALS OF INFORMATION REQUESTED

Interested persons are invited to submit written data, views, and comments with respect to the issues described above. All communications should be submitted in quadruplicate, by February 10, 1980, to the Docket Officer, Docket H-034, Room S6212, U.S. Department of Labor, 200 Constitution Avenue, N.W., Washington, D.C. 20210 (202-523-7894).

References

The following documents, referred to in this notice, are available for inspection and copying at the OSHA Technical Data Center, Room S6212, U.S. Department of Labor, 200 Constitution Avenue, N.W., Washington, D.C. 20210.

1. Maltoni, C. Vinyl chloride carcinogenicity: An experimental model for carcinogenesis studies. *Origins of Human Cancer*, Cold Spring Harbor Laboratory, 1977, pp. 119-148.

2. Wagoner, J. K., and P. F. Infante. Vinyl chloride: A case for the use of laboratory bioassay in the regulatory control procedure. *Origins of Human Cancer*, Cold Spring Harbor Laboratory, 1977, pp. 1797-1805.

3. Frongia, N., A. Spinazzola and A. Bucarelli. Lesioni polmonari sperimentali da inalazione prolungata di polveri di PVC in ambiente di lavoro: (Experimental lung damage from prolonged inhalation of airborne PVC dust). *La Medicina del Lavoro*, 65: 321-342, 1974.

4. Waxweiler, R. J., W. Stringer, J. K. Wagoner, J. Jones, H. Falk, and C. Carter. Neoplastic risk among workers exposed to vinyl chloride. *Annals of the New York Academy of Sciences*, 271: 40-48, 1976.

5. Baxter, P. J., and A. J. Fox. Angiosarcoma of the liver in PVC fabricators. *Lancet*, 1: 245-246, 1976.

6. Chiazze, L., W. E. Nichols, and O. Wang. Mortality among employees of PVC fabricators. *Journal of Occupational Medicine*, 19: 623-626, 1977.

7. Waxweiler, R. J., A. H. Smith, H. A. Tyroler, and H. Falk. An epidemiologic investigation of an excess lung cancer risk in a synthetic chemicals plant. Presented at the XIX International Congress on Occupational Health, Dubrovnik Yugoslavia, 25-30 September 1978.

8. Mastroangelo, G., M. Manno, C. Marier, C. B. Bertolucci, C. Comignani, C. Saladino, L. Simonato, and B. Sala. Polyvinyl chloride pneumoconiosis: Epidemiological study of exposed workers. *Journal of Occupational Medicine*, 21: 540-542, 1979.

9. Szende, B., K. Lapis, A. Nemes, and A. Pinter. Pneumoconiosis caused by the inhalation of polyvinyl chloride dust. *La Medicina del Lavoro*, 61: 433-436, 1970.

10. Amaud, A., P. Pommier de Santil, L. Garbe, H. Payen, and J. Charpin. Polyvinyl chloride pneumoconiosis. *Thorax*, 33, 19-23, 1978.

11. Maltoni, C. Precursor lesions in exposed populations as indicators of occupational cancer risk. *Annals of the New York Academy of Sciences*, 271: 444-447, 1976.

12. Infante, P. F., J. K. Wagoner, A. J. McMichael, R. J. Waxweiler, and H. Falk. Genetic risks of vinyl chloride. *Lancet*, 1: 734-735, 1976.

Signed at Washington, D.C. this 12th day of December 1979.

Eula Bingham,

Assistant Secretary of Labor.

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BILLING CODE 4510-26-02

Pension and Welfare Benefit Programs

(Prohibited Transaction Exemption 79-78; Exemption Application No. D-837)

Exemption from the Prohibitions for Certain Transactions Involving College Retirement Equities Fund

AGENCY: Department of Labor.