

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE  
CENTER FOR DISEASE CONTROL  
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH  
CINCINNATI, OHIO 45226

HEALTH HAZARD EVALUATION DETERMINATION  
REPORT NO. HE 78-31-623

OLIN CORPORATION  
ASHTABULA, OHIO 44004

October 1979

I. TOXICITY DETERMINATION

NIOSH conducted a medical evaluation of Oil Chemical and Atomic Workers (Local 7-629), members who allegedly were suffering adverse health effects (nervous and respiratory system complaints) from exposure to toluene diisocyanate, phosgene, and toluene 2-4 diamine. Only 15 of 57 OCAw workers participated in the evaluation conducted April 27-28, 1978. Thirteen of 15 individuals examined reported having experienced upper respiratory tract symptoms; seven reported symptoms of chronic bronchitis and seven reported chest tightness associated with work and wheezing; four of 14 individuals exhibited one or more abnormal pulmonary function test results. The rate of reported respiratory signs and symptoms is higher than expected in this subpopulation and suggests adverse health effects. However, since the number of workers who chose to participate was small, it cannot be determined whether an abnormal rate of respiratory signs and symptoms existed in the total population of 57 workers.

II. DISTRIBUTION AND AVAILABILITY OF DETERMINATION REPORT

Copies of this Determination report are currently available upon request from the National Institute for Occupational Safety and Health (NIOSH) Division of Technical Service, Information Resources and Dissemination Section, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days, the report will be available through the National Technical Information Service, (NTIS), Springfield, Virginia. Information regarding its availability through NTIS can be obtained from NIOSH, Publications Office at the Cincinnati address.

Copies of this report have been sent to:

- a) Vice President, Oil Chemicals and Atomic Workers Union,  
1126 16th Street N.W., Washington, D.C. 20036.
- b) President, Local 7-629, 1352 Norwood Drive, Ashtabula, Ohio  
44004.
- c) Olin Chemicals Group, P.O. Box 206, Ashtabula, Ohio 44004.
- d) U.S. Department of Labor - Region V.
- e) NIOSH - Region V.

For the purpose of informing the approximately 18 "affected employees" the employer shall promptly "post" for a period of 30 calendar days this determination report in a prominent place(s) near where the exposed employees work.

### III. INTRODUCTION

Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6), authorizes the Secretary of Health, Education, and Welfare, following a written request by an employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The National Institute for Occupational Safety and Health received such a request from an authorized representative regarding worker exposure to toluene diisocyanate (TDI), phosgene, and toluene 2-4 diamine. The request stated that workers at the plant were on strike (for about two months) and were suffering from hyperactivity, nervousness, and irritability, and requested evaluation of workers for respiratory symptoms associated with TDI and phosgene exposure.

On April 27-28, 1978, three Harvard physicians on contract to NIOSH and a NIOSH project officer proceeded to an off-plant site in Ashtabula to conduct medical examinations.

Information on plant processes and exposures was obtained from two long-term employees.

Fifty-seven members of the local Union were invited to participate in the medical examination. Fifteen union members received pulmonary function tests, and self administered and physician administered questionnaires. One worker underwent a physical exam by a contract physician on April 28. A verbal summary of actions taken was presented to a representative of the local Union President and other interested union members on the evening of April 27. A preliminary report summarizing actions taken was forwarded to representatives of labor and management June 12, 1978. No known change in the status of the labor dispute has taken place since this date.

**IV. HEALTH HAZARD EVALUATION**

**A. Plant Process**

Raw materials - coke, chlorine, 2,4 toluene diamine (TDA) and mono-chlorobenzene (MCB) - are used. Exposure of the front end operator to TDA may occur during unloading of the railroad car and during purification of the meta isomer before use. Coke is used to generate carbon-monoxide in the CO plant. The CO plant operator may be intermittently exposed to high concentrations of CO. CO and chlorine from enclosed tank cars are piped into a phosgene reactor monitored by the phosgene plant operator. Leaks may periodically occur in this enclosed process. Phosgene is pumped through water and Freon<sup>®</sup> coolers to a TDI reactor vessel where meta-TDA and MCB are also piped in. At this point, the "front end" operator is responsible for MCB and phosgene recovery. Upon formation, the TDI is purified on columns and the residue is removed, dried, and transported. Hydrochloric acid is a byproduct. This part of the operation exposes the "back end" operator and "helper" to TDI. The flow of the entire operation is monitored by a panelboard operator. Several maintenance personnel, depending on activity, receive multiple exposures throughout the plant.

The workers are supplied with a fresh pair of coveralls each day which are laundered by the company and showers exist at the plant. Separate lockers are provided for street and work clothes. Air-supplied respirators are used intermittently throughout the plant. Compressed air is obtained from General Tire Company next door. The plant has a dispensary, where a nurse delivers acute medical care for injuries and (minor) illnesses.

**B. Medical Design and Methods**

Fifteen members of OCAW Local 7-629 who attended a union meeting on April 27, 1978, were interviewed and received lung function tests. A self-administered questionnaire of general medical history and review of systems was followed by a physician administered questionnaire on work-related respiratory, neurological and dermatological symptoms. All 15 workers performed pulmonary function tests using a Stead-Wells spirometer to record FEV<sub>1</sub> and FVC. The prediction equations of Kory *et<sup>6</sup> al.* were used to generate expected lung function values. One worker with a history of liver disease and dermatological problems underwent a physical exam. All workers examined were advised of the results of their examination by letter.

C. Medical Criteria and Health Effects

1. TDI - TDI may cause irritation of the eyes, respiratory tract and skin. Exposures to high doses may cause acute bronchitis and pulmonary edema. Sensitization to TDI may occur with resulting asthmatic reaction on exposure to low concentrations. Wheezing, dyspnea and cough may be most severe at night following daytime exposure. Excessive decrease in FEV<sub>1</sub> may occur with chronic exposure to greater than 0.003 ppm. Symptoms of chronic bronchitis are also excessively prevalent in TDI exposed workers. Neurological complications have been observed after a single high exposure to TDI in firefighters.
2. Phosgene - Exposure to phosgene above 1 ppm for 10-15 minutes may produce pulmonary edema frequently preceded by a latent period of 5-12 hours. Chronic exposure to phosgene may cause emphysema and fibrosis of the lung.
3. Chlorine - Chlorine is irritating to the skin, eyes, and mucous membranes. In high concentrations chlorine gas may produce cough, chest-pain, dyspnea, bronchitis, pulmonary edema and pneumonia.
4. TDA - 2,4 diamine toluene is a carcinogen in animals.<sup>4,7</sup> Hepatocellular carcinoma has been produced in rats by feeding studies and sarcomas by subcutaneous injection. TDA is mutagenic in the Ames bacterial test after metabolic activation. No epidemiological studies have been done to link TDA to carcinoma in humans. TDA has also been shown to cause liver cirrhosis in animals.

D. Evaluation Results

Of the 15 workers interviewed, the ages ranged from 25-60 years. The mean age was 40 years. Most were long-term employees, having worked at Olin Company between 4-14 years (mean = 9 years). Of the 15, seven described lower respiratory tract symptoms on exposure to greater than usual amounts of phosgene, Cl<sub>2</sub>, TDI or mixtures of these gases and vapors. Upper respiratory tract symptoms were common, with 13 of 15 experiencing running or stuffy nose, sneezing, nosebleeds, hoarseness or dry sore throat apart from a cold. Seven workers described symptoms of chronic bronchitis (cough and phlegm on most days for more than 3 months per year for 2 years). Two of the four were present smokers, three were past smokers, and two had never smoked. Four workers have noted a reduction in bronchitis during the last 6 months when they have not been working. Chest tightness associated with work and wheezing was described by seven workers. Four workers said they got short of breath when hurrying or walking up a slight hill, while one-worker had to walk slower on the level ground because of breathlessness (dyspnea grade 3). Seven of the workers were ex-smokers, four never smoked and four smoke currently.

Technically acceptable pulmonary function studies were obtained in 14 of the workers. Two workers exhibit a FEV<sub>1</sub>/FVC % of less than 70%. Three workers had FEV<sub>1</sub> less than 80% of predicted and two had an FVC of less than 80% predicted. Of the four individuals one was a present smoker, two were past smokers, and one had never smoked.

One worker, who had a history of liver disease and dermatitis that he thought was related to work, underwent a physical exam. He was found to have red, cracked, scaly palms due to an irritative dermatitis. Another abnormal finding was a firm, slightly tender liver edge that extended 2 1/2 inches below the right costal margin.

Due to the low rate of participation of available workers, the inhibitory effect of the strike, and the employment of non-union replacements, and on-going OSHA abatement action, no in-plant evaluation was undertaken.

#### E. Conclusions

A few workers reported dizziness and loss of consciousness which may be work related. Behavioral symptoms in this group appeared to be stress related and not as a direct result of work exposures to chemicals.

Upper respiratory tract irritation at work was common. Almost half of the 15 workers interviewed had experienced at least one acute episode of lower respiratory tract irritation while employed at Olin.

A high prevalence of chronic bronchitis and asthmatic symptoms of the type classically associated with TDI was found in the 15 workers interviewed. Most noted improvement during the strike. It is not known if this subgroup is a representative sample of the entire group of 57 individuals.

#### F. Medical Recommendations

The entire plant population should be periodically surveyed for symptoms of lower respiratory tract disease and lung function abnormalities that may be caused by exposure to TDI, phosgene and chlorine. Exposure to TDI should be ended in workers showing classical symptoms of TDI sensitization or excessive decline in lung function.

The possibility of over exposure of some workers to carbon monoxide should be evaluated with environmental sampling, carboxyhemoglobin determinations, and medical evaluation of workers. Liver function tests should be periodically done on workers exposed to TDA.

TDA is a animal carcinogen and mutagen and thus is a potential occupational carcinogen. TDA levels should be monitored and TDA exposure reduced to the lowest feasible level.

Since the effects of TDA on humans has not been documented, consideration should be given to assess chromosome abnormalities in workers exposed to TDA. The rate of sister chromatid exchanges, chromosome aberrations, and amount of mutagenic metabolites in the urine could also be determined in those workers currently or previously exposed to TDA and in an appropriate comparison group. This could be done to determine whether these workers have evidence of excessive rates of DNA damage.

**V. REFERENCES**

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