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INDUSTRIAL HYGIENE QUARTERLY

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PLAINTIFF'S EXHIBIT
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after it has been taken into the lungs than when it is ingested. Less than 10% is absorbed from the gut, whereas a much higher percentage may be absorbed from the lungs, depending on the conditions of exposure (e.g., chemical form in which cadmium is inhaled, particle size, and site of deposition within the lungs). Irrespective of any effect it may have on the lung, most inhaled cadmium passes eventually to the liver and kidneys. It is well established that cadmium tends to accumulate in these tissues--especially in the cortex of the kidneys of men exposed to cadmium in inhalation. Theoretically, damage to the lung might occur during the passage of cadmium from the air spaces into the blood-vessels or lymphatics, without the accumulation of the metal within the lung. Alternatively, cadmium might accumulate in the lung and yet be responsible for no damage there. Nevertheless, if prolonged exposure to cadmium were involved in the causation of emphysema one would be somewhat surprised if there were no correlation between the amounts of the metal found in lung tissue and the severity of the disease. Curiously, there is little information on the concentrations of cadmium in the lungs of cadmium workers dying with emphysema of various degrees of severity. It is indisputable that smoking predisposes to emphysema, and it is also clear that traces of cadmium are present in tobacco smoke. It is therefore not surprising either that smokers accumulate more cadmium in their tissues--kidneys, liver, and lungs--than non-smokers or that more cadmium is found in the tissues of emphysematous subjects with a history of heavy smoking than in those subjects without emphysema and with a history of non-smoking or less heavy smoking. --Cond. from text

61/74 Dimethylformamide-Induced Abdominal Pain and Liver Injury. H.P. Potter. Arch. Environm. Health 27: 340-341, Nov. 1973. 7 refs.

Dimethylformamide (DMF), a widely used and versatile industrial solvent, has been reported to cause abdominal pain and hepatic abnormalities. An accidental dermal and respiratory exposure to DMF produced severe abdominal pain, hypertension, leukocytosis, and hepatic damage in a fabric coating worker. Pain began 62 hours after DMF exposure and was associated with positive results from a Watson-Schwartz test for urine porphobilinogen. --Author's abst.

INDUSTRIAL DUSTS AND INHALANTS

62/74 Type of Asbestos and Respiratory Cancer in the Asbestos Industry. P.E. Enterline and V. Henderson. Arch. Environm. Health 27: 312-317, Nov. 1973. 18 refs.

Differences in the physical properties and chemical composition of the various types of asbestos indicate that there should be differences in their carcinogenic potentials. This study of the mortality experience of 1,348 retirees from the asbestos industry shows that, after adjustment for cumulative dust exposure, men exposed only to chrysotile asbestos had a respiratory cancer mortality rate 2.4 times the expected, whereas men exposed to a combination of chrysotile and crocidolite asbestos had a mortality rate 5.3 times the expected. Since crocidolite was used largely in the asbestos cement industry, this was studied separately. Men who manufactured asbestos cement shingles and sheets containing only chrysotile asbestos had a respiratory cancer risk 1.4 times the expected, whereas men who manufactured asbestos cement pipe containing both chrysotile and crocidolite asbestos had a respiratory cancer risk 6.1 times the expected. --Authors' abst.

63/74 Mesothelioma Following Exposure to Asbestos: A Review of 72 Cases. M. Borow, et al. Chest 64: 641-646, Nov. 1973. 25 refs.

In 1967, the authors reported 17 patients with mesothelioma, of which nine cases were pleural and the remainder peritoneal. Since then, they have treated 36 additional cases, making a total of 53 patients. A search through the medical records of neighboring hospitals brought to light another 19 cases, making the total number 72. Fifty-one of these were pleural (71 percent) and 21 were peritoneal (29 percent). Sixty-four (89 percent) of these patients were men, and information of occupational exposure was present for all but nine, where no history was available. Eight of the patients were women, three of whom had a history of occupational exposure to asbestos, two had environmental exposure to asbestos, and the remaining three had an inadequate history in their medical records. Of this group of 72 patients, there were 68 deaths with autopsies. Four are still living, but with disease. As has been documented before, their patients were exposed to asbestos 20 or more years before the malignancy appeared. Although most of these subjects smoked two or more packs of cigarettes per day, three were nonsmokers. Apparently, the incidence of this malignancy is increasing. Since a nearby asbestos mill has been in operation for half a century and has been the source of all their case material, the authors wondered if there had been other cases of mesothelioma in which a correct diagnosis had not been made in earlier years. They reviewed all the autopsy records from 1948 to 1963 and found only two cases which now must be considered as mesothelioma. Inasmuch as it has been only a few years since the asbestos industry has instituted measures to reduce the incidence of pulmonary asbestosis, they expect to see increasing numbers of patients with mesothelioma for the next 20 to 30 years. They providing that these health measures are sufficient, there should be a dramatic reduction in the incidence of this malignancy. --Cond. from text