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THRESHOLD LIMIT VALUES



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COMMITTEE ON THRESHOLD LIMIT VALUES

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ARSINE

0.05 ppm (Approximately 0.2 mg/m^3)

The extreme, acute toxicity of arsine is well known; 250 ppm for 30 minutes is fatal to man and 3-10 ppm can cause poisoning symptoms in a few hours (1). Nau (2) reported the conditions of an arsine exposure that indicated the previous limit of 1 ppm was too high. Elkins (3) reported a case of severe, but nonfatal, arsine poisoning that resulted from an exposure averaging 0.5 ppm. Elkins has concluded that 0.05 ppm arsine is not an unreasonable low limit on the basis of a study of chronic arsine exposures of Bulmer, et al. (4). Urinary As varied from 0.7 to 4 mg. in men showing toxic signs of exposure of jaundice and anemia. Assuming 75% of the As is excreted in the urine 1 mg. As/l corresponds to 1.33 mg. intake, or 0.133 mg. As/m^3 air if 10 m^3 air is taken as the average amount of tidal air inhaled during a working day. This corresponds to 0.033 ppm arsine, and suppor the recommended limit of 0.05 ppm arsine.

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ASBESTOS

5 mppcf

Asbestos is a generic term applying to a number of mineral silicates that are incombustible in air and can be separated into filaments. The most widely used in industry is chrysotile, a magnesium silicate from serpentine. Other types include amosite (an iron magnesium silicate) crocidolite (a sodium iron silicate), tremolite (a calcium magnesium silicate) and anthophyllite (also an iron magnesium silicate).

Miller and Sayers (1) showed that intraperitoneal injection of amosite, chrysotile and crocidolite in guinea pigs produced the reaction of an inert dust. Vorwald et al. (2) confirmed this for short fibers (under 3μ) but observed that long fibers produced a fibrous reaction Continuing unpublished work by Gardner, these workers demonstrated that long fibers (20 microns and above) produced peribronchiolar fibrosis in lower animals, and developed evidence that this resulted from mechanical rather than chemical action. Asbestos dust containing 0.6 per cent fibers longer than 10 microns, in concentrations of 138 mppcf (or 0.8 million "long" fibers per c.f.), was capable of producing experimental asbestosis in guinea pigs. When the concentration was 6.7 per cent fibers over 10 microns, 40 mppcf, (equivalent to 2.7 million "long" fibers per c.f.), the reaction developed in approximately half the time.

That exposure to asbestos is associated with development of a potentially disabling pneumoconiosis in man has been amply demonstrated by industrial experience (3,4,5,6,7,8,9,10,11). The present threshold limit relates to the prevention of asbestosis. It was recommended by Dreessen et al. (8), after study of 541 employees in three asbestos textile plants using chrysotile. Only three

f pneumoconiosis were found in those exposed to dust concentra-
appcf, whereas numerous well-marked cases were found above 5 mppcf.
rom impinger-collected samples in ethyl alcohol and distilled water.
and non-fibrous particles were counted, but the latter greatly pre-
. While chemical analyses of collected samples of airborne dust corres-
those of settled dust samples, it is believed that dust counts of par-
es by conventional methods can be expected to give only an indirect measure
risk of asbestosis because of the great relative importance of long fibers.

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BARIUM (and Compounds)

0.5 mg/m^3

The clinical entity "baritosis" has been reported in the industrial hygiene literature sporadically since 1934 when Laschke (1) described a case with almost fatal outcome in a baryta worker who had apparently inhaled ample quantities. Other reports of industrial exposure to barium compounds with or without exposure to lithopone have described pulmonary nodulation with or without decrease in lung function, such as dyspnea on exertion (2,3). More soluble forms of barium, as the carbonate, oxide and nitrate, tend to be more injurious, particularly acutely. Dusts of barium oxide are considered potential agents of dermal and nasal irritation (4).

The pharmacologic action of barium is well known (5); chief among the actions of barium is its effect on muscle, particularly cardiac, increasing its excitability. Skeletal, arterial, intestinal, and bronchial muscle are all affected by barium. In addition, effects on the hematopoietic system have been noted, as well as on the cerebral cortex.

Fazekas, et al. (6) have reported that subcutaneous injection of an aqueous solution of barium chloride at a dosage of 5 mg/kg caused acute toxicity with death after 2-2.5 hours. Chronic poisoning was achieved by the injection of solutions at 10, 5, and 2 mg/kg. Rabbits in this series were killed at 98 to 193 days Effects on the central nervous system are described.

The present limit of 0.5 mg Ba/m^3 air was suggested by Hyatt (7), who employed this limit for a number of years at the Los Alamos Laboratories with satisfactory results for the control of exposure to barium nitrate. It is not known what added safety this limit incorporates.

doubtful cases of pneumoconiosis were found in those exposed to dust concentrations under 5 mppcf, whereas numerous well-marked cases were found above 5 mppcf. Counts were from impinger-collected samples in ethyl alcohol and distilled water. Both fibrous and non-fibrous particles were counted, but the latter greatly predominated. While chemical analyses of collected samples of airborne dust corresponded to those of settled dust samples, it is believed that dust counts of particulates by conventional methods can be expected to give only an indirect measure of the risk of asbestosis because of the great relative importance of long fibers.

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