Asbestos Manhattan Industrial Products Company

Environmental Services
North Charleston, S. C.

Mr. Joe Wronski

September 30, 1976

Hoechst Fiber

Attached are seven (7) asbestos dust samples collected during insulation operation at Hoechst Fiber in Spartanburg, S. C. on September 29, 1976.

The results of these samples are very much needed by Mr. Milt Scowcroft of our Marketing Department for court testimony. Any priority on analysis would be appreciated.

Pat Cooper
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Attachments

cc: W. Krucke
M. Scowcroft



1971

APPENDIX 7

EXHIBIT T 205

Documentation of Threshold Limit Values

REVISED EDITION

COMMITTEE ON THRESHOLD LIMIT VALUES

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MACKSYAM-120

PLAINTIFF'S EXHIBIT 5-28-97

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While data on the actual concentrations causing acute intoxication are lacking, post event concentrations of 70 to 300 ppm (Morse and Setterlind, fatal cases(11)), 5 ppm (Kipling and Fothergill(8)) and 0.5 ppm (Elkins(3)) were reported. When urine samples were analysed at early stages concentrations of arsenic ranging from 0.5 to 2 mg/liter were the rule, but occasionally much higher values were found.

Bulmer et al.(12) reported a number of cases of chronic poisoning, with severe anemia. Urinary arsenic levels averaged 2.3 mg/l, dropping to 0.06 mg/l three days later. Greig et al.(13) recorded three relatively mild chronic cases with arsenic in urine levels of 0.5 mg/l.

Since arsenic appears to be excreted rather freely in the urine (Elkins(14)) the levels found in the urine of intoxicated workers could have resulted from inhalations of concentrations below 1 mg/m^3 or 0.25 ppm. The recommended TLV of 0.05 ppm (0.2 mg/m^3) is nearly half the limit of other inorganic arsenic compounds, which are considered substantially less toxic.

Other recommendations: Cook (1945) 1 ppm; Smyth (1956) and Elkins (1959) 0.05 ppm; U.S.S.R. (1966) 0.1 ppm; Czechoslovakia (1968) 0.05 ppm.

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ASBESTOS (all forms)

5 fibers/ml (longer than 5 μ)

Asbestos is a broad term embracing a number of fibrous mineral silicates that differ in chemical composition. The three types of greatest commercial importance are chrysotile, a hydrated magnesium silicate; crocidolite, a sodium iron silicate; and amosite, an iron magnesium silicate. Of these chrysotile is by far the most widely used in industry. Other asbestos minerals include tremolite, anthophyllite and actinite.

That exposure to asbestos is associated with development of a potentially disabling pneumoconiosis in man has been amply demonstrated by industrial experience(1, 2, 3, 4, 5, 6, 7, 8, 9). A threshold limit of 5 mppcf was recommended by Dreessen et al.(6), in 1938 after studying 541 employees in four asbestos textile plants where excessive exposures to chrysotile occurred. Only three doubtful cases of pneumoconiosis were found at the time of the study in those exposed to dust concentrations under 5 mppcf, whereas numerous well-charted cases were found above 5 mppcf. Counts were from impinger-collected samples in ethyl alcohol and distilled water. Both fibrous and nonfibrous particles were counted, but the latter greatly predominated. Although chemical analyses of collected samples of airborne dust corresponded to those of settled dust, it is believed that dust counts of particulates by impinger can be expected to give only an indirect measure of the risk of asbestosis because impinger sampling collects particulates other than asbestos.

A conference on the biologic effects of asbestos(10) in 1965 called attention to the very real proximity that the 5 mppcf limit recommended by Dreessen is inadequate to give complete working-life-time protection against all forms of asbestos. Medical data on which the limits had been based were inadequate; more than half of the asbestos workers studied were under 30 years of age and thus provided an insufficient exposure time for asbestosis to develop. Of the 108 workers exposed to <5 mppcf, 68 had worked < 5 years; 101, < 10 years; only 4 had >10 years exposure. Seven of 26 workers exposed to 5 - 9.9 years had asbestosis; 3 of 10 workers exposed to 10 - 19.9 mppcf for <5 years had asbestosis(9). Moreover it was a "point-in-time" study; many of the ill were missing and the dead uncounted, hence not considered in the over-all evaluation of the limit.

The asbestos conference(10) further called attention to the rising worldwide rate of increase in lung cancer among asbestos workers, particularly bronchial cancers, and also noted with alarm a widespread incidence of pleural and diffuse mesotheliomas, tumors heretofore overlooked and only recently (1960) associated with exposure to asbestos. The epidemiology of the early cases was especially revealing in pointing out the scope of asbestos exposures: many of the mesotheliomas cases had no industrial association, but were "neighborhood" cases or cases traced to users of asbestos. Exposures to asbestos extend far beyond those of miners and millers of asbestos or textile weavers, and include carpenters (sawing asbestos board), insulators and pipe laggers, dockers handling asbestos cargo, brake-lining workers, rubber compounders and several others.

The Committee on Hygienic Standards of the British Occupational Hygiene Society from an evaluation of medical evidence from Great Britain and data from the U.S. Public Health Service Study(6) recommended the following criteria for limiting exposure to chrysotile asbestos assuming a 1% risk of contracting asbestos disease during a 30-year working exposure(11).

Dust Category	Chrysotile Concentration[a] Fibers/ml[b]
Negligible	< 0.5
Low	0.5 - 2.0
Medium	>2 - 10
High	>10

a) Averaged during a three-month period
b) Greater than 5μ in length

From these criteria, a limit of 100 fibers/ml-years was proposed, hence a limit of 2 fibers/ml averaged over a 50-year period.

In arriving at an asbestos limit based on American experience and considerations applicable to the American industrial scene, the following facts predominated. Recent evaluation of the health experience in asbestos plants indicated that the 5 mppcf limit was not sufficiently low to protect workers exposed for 20 years, a period rarely exceeded in America. Selmar and Cooper (12) supported this review in a report of asbestosis occurring among insulation workers from levels that were deemed highly unlikely to have exceeded a time-weighted average of 5 mppcf. A retrospective investigation of the association between lung cancer in asbestos workers and smoking by Selikoff et al.(13) revealed that asbestos neoplasias were strongly associated with smokers.

From a study of asbestos fiber concentrations in six different industries in 32 plants, Ayer et al.(14) found average counts above 5 fibers/ml in 61 departments, and below 5 in 148. The highest counts generally were found in insulation, textile and friction plants. Lower levels were obtained in shingle and mill board plants and cement pipe plants, and the lowest in packing and rubber plants.

Williams and associates(15) compiled data from two Pennsylvania asbestos textile plants, going back to 1920 in one plant and 1946 in the other. Even though exposures for the most part were below 5 mppcf and in many cases below 2 mppcf, 64 cases of asbestosis were reported from the two plants. The authors concluded that a limit of 5 mppcf would provide inadequate protection and even the 2 mppcf TLV might not be substantiated.

An Ad Hoc select review committee of the Bureau of Occupational Safety and Health(16) proposed a standard of 6 fibers/ml. longer than 5 μ. This concentration has been found equivalent to 1 mppcf by the conventional light field impinger count.

Inasmuch as the particles counted by the impinger procedure often include extraneous material, a limit of 1 mppcf might be unrealistic in processes where dust other than asbestos was present, even in relatively low concentrations. A TLV of 5 fibers, longer than 5 microns (μ), per cc. as determined by collection on membrane filters and counted using phase-contrast illumination at 430X magnification(17) is recommended.

On present evidence, this interim standard should afford protection against asbestosis and reduce to an acceptably low risk the development of neoplasms. Complete elimination of risk cannot be assured at the present time from this or any other potential carcinogenic agent, because of incomplete understanding of dose-response relationships of such agents.

Although some authorities believe that different varieties of asbestos have different biological effects, at this time there appears to be insufficient information to justify differentiating among them in establishing the TLV.

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ASPHALT (PETROLEUM) FUMES

5 mg/m³

Asphalt is a native mixture of hydrocarbons which occurs as an amorphous, brownish black solid or semisolid(1, 2). It results from evaporation of the lighter hydrocarbons from petroleum and partial oxidation of the residue. Petroleum asphalt thus is to be differentiated from tar or pitch, which results from the destructive distillation of coal. Asphalt fumes arise in the course of manufacture or from the secondary heating of asphalt. Thus asphalt fume exposure occurs not only in association with its manufacture but also in road building, roofing, and in the coating of construction metals.

Animal inhalation studies have not yielded sufficient evidence of asphalt-induced lung cancer, and only limited investigation has been done on the chemical properties and the metabolic changes