V. BASIS FOR THE RECOMMENDED STANDARD

The first modern approach to the setting of an asbestos standard was proposed by the British Occupational Hygiene Society (BOHS 1968) in terms of fiber concentration. In 1968, a subcommittee of the Society evaluated data on 290 men at work in an asbestos factory. These data were provided by company sources. All the men had been employed after January 1933, following implementation of dust control measures mandated by the Factory Inspectorate in 1931. Estimates of the fiber exposure of these workmen were also provided by the company. Of the 290 individuals, 8 were stated to have x-ray evidence of asbestos disease and 16 had rales. Noteworthy in the 1968 data was the preponderance of individuals who had been employed less than 20 years. Only 118 of the 290 persons had worked for longer than 20 years and a scant 13 has been employed for 30 or more years.

After a review of these data, the BOHS proposed a standard which was adopted with minor modifications by the British government in 1969, and implemented in May 1970. All fibers between 5 and 100 microns in length were counted by light microscopy. The standard required no action to be taken below 2 fibers/cc. Between 2 fibers/cc and 12 fibers/cc, control measures commensurate with the exposure circumstances (time and frequency of worker exposure) were prescribed; above 12 fibers/cc, full application of control measures, including respiratory protection, was mandatory. The BOHS predicted that the risk of being affected, to the extent of having the earliest clinical signs of asbestos exposure (rales), would be less than 1% for an accumulated exposure of 100 fiber-years/cc (2 fibers/cc for 50

years, 4 fibers/cc for 25, etc). Data (Lewinsohn, 1972) from the same factory which formed the basis for the BOHS standard demonstrate that a greater prevalence of abnormalities now exist (Table V-1). These data, in addition to demonstrating a dose-response relationship for radiographically detected abnormalities consistent with asbestosis, further showed a 17% prevalence of abnormal radiographic findings (6% consistent with asbestosis) in individuals employed since 1950.

Weill et al (1975), when considering lung function and irregular small opacities, reported that there was little evidence of a dose-response relationship below 100 mppcf-years. They further concluded that a concentration of 5 fibers/cc could be cautiously considered as "safe". Ayer and Berg (1976), however, reported data which suggest that the BOHS standard, of an average cumulative exposure of 100 fiber-years/cc, for chrysotile asbestos may prevent significant decreases in pulmonary function only when combined with periodic spirometry and further reduction of exposure for affected workers. Holmes (1973) has since stated that the data upon which the BOHS standard was based were inadequate to set a standard to prevent asbestosis. The BOHS-recommended standard of 2 fibers/cc was based on data related only to asbestosis and the Society clearly cautioned that, since a quantitative relationship between asbestos exposure and cancer risk was not known, it was not possible at that time to specify an air concentration which was known to be free of increased cancer risk. (BOHS 1968)

Howard et al (1976), in a follow-up examination of the textile workers previously studied by Doll (1955) and Knox et al (1965, 1968) for cancer, and by Lewinsohn (1972) for asbestosis, reported a statistically

significant increase in the risk of developing lung cancer (1.8 times the expected) among those first entering scheduled areas from 1933 to 1950. In the same study, they also reported an excess of deaths due to lung cancer (1.9 times the expected) after 15 or more years from initial exposure among those who started work subsequent to 1950, a period of improved industrial engineering control technology and regulation.

In a study of miners exposed to amphibole fibers (amosite) in the cummingtonite-grunerite ore series, with airborne concentrations of less than 2.0 fibers/cc (average concentration, 0.25 fibers/cc) and 94% of the fibers shorter than 5 μm in length, Gillam et al (1976) have demonstrated threefold increases in the risks of mortality from both malignant and nonmalignant respiratory diseases.

Newhouse (1969, 1973) and Newhouse et al (1972) have shown that the cancer risk to factory workers following mixed exposure to chrysotile, amosite, and crocidolite is dose-related. The women reported to have heavier exposures (as judged by their occupations) showed a sixfold excess of cancer following only 15 years' latency, whereas those with moderate or low exposures required 25 years' latency to demonstrate an excess. The rate of mesothelioma increased with both the severity and the length of exposure. However, even with as little as two years of asbestos exposure, six mesotheliomas occurred among female employees.

McDonald (1973) stated that the risk of developing lung cancer was essentially confined to persons with a dust index above 200 mppcf-years, and Enterline et al (1973) showed no direct dose-response for respiratory cancer below 125 mppcf-years. In a review of these two papers, Schneiderman (1974) concluded that, instead of being consistent with a

threshold level at which no cancer risk exists, these data did not provide evidence for a threshold or for a "safe" level of exposure. He pointed out that in the paper by Enterline et al (1973) there is no dose group for which the Standardized Mortality Ratio (SMR) is below 100 (100 = normal), but that the 95% confidence limits on the SMR's included 100 for two of the three dose groups below 125 mppcf-years. One of the dose groups (25-62.4) had a statistically significant excess mortality from lung cancer, whereas for the other two this mortality rate was insignificantly elevated above the expected values. Regarding McDonald's paper, Schneiderman stated that it is hard to determine what is excess since no expected numbers for each group were given upon which to base this comparison.

Among amosite workers with employment of 3 months or less, Selikoff (1976) reported excess cancer risks of 3.87, 1.68, and 1.65 times those expected for cancer of the lung, colon and rectum, and all sites, respectively.

Anderson et al (1976) have reported a significant excess of radiographic abnormalities of the chest characteristic of asbestos exposure (pleural and/or parenchymal) 25 - 30 years after the onset of household contamination. These abnormalities were observed in 35% of 326 otherwise healthy workers who had household contacts with amosite asbestos. In addition, four pleural mesotheliomas were found in this group.

VI. THE RECOMMENDED STANDARD

Available studies provide conclusive evidence that exposure to asbestos fibers causes cancer and asbestosis in man. Lung cancers and asbestosis have occurred following exposure to chrysotile, crocidolite, amosite, and anthophyllite. Mesotheliomas, lung and gastrointestinal cancers have been shown to be excessive in occupationally exposed persons, while mesotheliomas have developed also in individuals living in the neighborhood of asbestos factories and near crocidolite deposits, and in persons living with asbestos workers. Asbestosis has been identified among persons living near anthophyllite deposits.

Likewise, all commercial forms of asbestos are carcinogenic in rats, producing lung carcinomas and mesotheliomas following their inhalation, and mesotheliomas after intrapleural or ip injection. Mesotheliomas and lung cancers were induced following even 1 day's exposure by inhalation.

The size and shape of the fibers are important factors; fibers less than 0.5 μm in diameter are most active in producing tumors. Other fibers of a similar size, including glass fibers, can also produce mesotheliomas following intrapleural or ip injection.

There are data that show that the lower the exposure, the lower the risk of developing cancer. Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure.

In view of the above, the standard should be set at the lowest level detectable by available analytical techniques, an approach consistent with NIOSH's most recent recommendations for other carcinogens (ie, arsenic and vinyl chloride). Such a standard should also prevent the development of asbestosis.

Since phase contrast microscopy is the only generally available and practical analytical technique at the present time, this level is defined as 100,000 fibers >5 μm in length/m^3 (0.1 fibers/cc), on an 8-hour-TWA basis with peak concentrations not exceeding 500,000 fibers >5 μm in length/m^3 (0.5 fibers/cc) based on a 15-minute sample period. Sampling and analytical techniques should be performed as specified by NIOSH publication USPHS/NIOSH Membrane Filter Method for Evaluating Airborne Asbestos Fibers - T.R. 84 (1976).

This recommended standard of 100,000 fibers >5 μm in length/m^3 is intended to (1) protect against the noncarcinogenic effects of asbestos, (2) materially reduce the risk of asbestos-induced cancer (only a ban can assure protection against carcinogenic effects of asbestos) and (3) be measured by techniques that are valid, reproducible, and available to industry and official agencies.

However, some difficulties arise in that specific work practices and innovative engineering control or process changes are needed. But because of the well-documented human carcinogenicity from all forms of asbestos, these difficulties should not be cited as cause for permitting continued exposure to asbestos at concentrations above 100,000 fibers >5 μm in length/m^3.

This standard was not designed for the population-at-large, and any extrapolation beyond general occupational exposures is not warranted. The standard was designed only for the processing, manufacturing, and use of asbestos and asbestos-containing products as applicable under the Occupational Safety and Health Act of 1970.

REFERENCES FOR CHAPTERS V AND VI

1. 1969 Standard for asbestos dust concentration for use with the asbestos regulations. Department of Employment and Productivity, Her Majesty's Factory Inspectorate. Technical Note 13, 1970

2. Lewinsohn HC (1972): The medical surveillance of asbestos workers. Soc Health 92:69

3. Weill H, Ziskind MM, Waggenspack C, Possiter CE (1975): Lung function consequences of dust exposure in asbestos cement manufacturing plants. Arch Environ Health 30:248-52

4. Ayer H, Berg J (1976): Cumulative asbestos exposure and forced vital capacity. Submitted for publication to Arch Environ Health

5. Holmes S (1973): Environmental data in industry, in Bogovski P, Timbrell V. Gilson JC, Wagner JC (eds): Proceedings of the Conference on Biological Effects of Asbestos. Lyon, pp 135

6. Newhouse ML (1969): A study of the mortality of workers in an asbestos factory. Br J Ind Med 26:294

7. Newhouse ML (1973): Cancer among workers in the asbestos textile industry, in Bogovski P, Gilson JC, Timbrell V, Wagner JC (eds): Proceedings of the Conference on Biological Effects of Asbestos. Lyon, pp 203

8. Newhouse ML, Berry G, Wagner JC, Turok ME (1972): A study of the mortality of female asbestos workers. Br J Ind Med 29:134

9. Howard S, Kimben LJ, Lewinsohn HC, Peto J, Doll R (1976); A Mortality Study among Workers in an Englosh Asbestos Factory. Oxford University, (in press)

10. Doll R (1955): Mortality from lung cancer in asbestos workers. Br J Ind Med 12:81-86

11. Knox JF, Doll RS, Hill ID (1965): Cohort analysis of changes in incidence of bronchial carcinoma in a textile asbestos factory. Am NY Acad Sci 132:526-35

12. Knox JF, Holms S, Doll R, Hill ID (1968): Mortality from lung cancer and other causes among workers in an asbestos textile factory. Br J Ind Med 25:293-303

13. Gillam JD, Dement JM, Lemen RA, Wagoner JK, Archer VE, Beljer HP (1976): Mortality patterns among hard rock gold miners exposed to an asbestiform mineral. Ann NY Acad Sci 271:336-44

TABLE VI-1

B.O.H.S. ASBESTOS STANDARD
X-RAY FINDINGS IN AN ASBESTOS TEXTILE FACTORY
DECEMBER 1970 (MALES)

Years of Exposure	No.	X-ray Findings			
		Normal	Pleural Fibrosis*	Pulmonary Fibrosis	Total Abnormal**
0 - 9	613	548	10	0	65(11%)
10 - 19	189	122	18	20	67(36%)
20 - 29	114	51	30	21	63(55%)
30 - 39	42	9	17	17	33(78%)
40 - 49	12	2	6	3	10(83%)

* Consistent with asbestos exposure
**Including changes not considered due to asbestos exposure
Adapted from reference 2

☆U.S. GOVERNMENT PRINTING OFFICE: 1986-646-117/40905

DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH
ROBERT A. TAFT LABORATORIES
4676 COLUMBIA PARKWAY, CINCINNATI, OHIO 45226

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE, $300

U.S. MAIL

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF H.E.W
HEW 396

THIRD-CLASS MAIL

DHEW (NIOSH) Publication No. 77-169