E. Effects of Cigarette Smoking and Asbestos Exposure

This section discusses scientific evidence describing the influence of smoking on the risk from asbestos-related disease. Because several studies (Exs. 2–5; 84–190; 84–47) were cited in the November proposal as evidence of a multiplicative effect of asbestos exposure and cigarette smoking with regard to producing increased lung cancer risk, several commenters, including the Asbestos Information Association (Ex. 328), argued that OSHA overstated the risk of lung cancer in its qualitative risk assessment by failing to distinguish between the lung cancer risks for asbestos-exposed smokers and nonsmokers. While the scientific data are discussed here, Section VI (Significance of Risk) contains OSHA's response to comments dealing with how the lung cancer risk for asbestos-exposed smokers should be evaluated from a regulatory perspective.

Asbestos-Related Malignant Disease

Several studies were cited in the November proposal as evidence that asbestos-exposed workers who smoke have a higher risk of lung cancer mortality than either asbestos-exposed nonsmokers (Selikoff, Churg, and Hammond, Ex. 2–5; Selikoff, Seidman, and Hammond, Ex. 84–190; Hammond, Selikoff, and Seidman, (Ex. 84–047). The reduced ability of smokers to clear particles from their lungs, compared with the ability of non-smokers to do so, as suggested by Cohen et al. (Ex. 84–031), may help to explain the higher lung cancer risk of asbestos workers who smoke. The Agency also determined that there is no evidence of an association between cigarette smoking and an increased risk of either mesothelioma or gastrointestinal cancer.

To exemplify the multiplicative effect of asbestos exposure and cigarette smoking in producing an increased lung cancer risk, OSHA discussed two studies at length (Hammond et al. Ex. 84–047; Selikoff et al., Ex. 84–190). The Hammond et al. study (Ex. 84–047) examined the smoking histories of 8,220 of 12,051 asbestos insulation workers with a followup of 20 or more years since initial exposure. In late 1966, 6,841 of these workers were either current or past cigarette smokers, 486 had a history of pipe or cigar smoking, and 891 had never smoked regularly. The mortality of these workers was observed during the period 1967–1976. The comparison population, drawn from the American Cancer Society's long-term prospective study, consisted of 73,763 white men who had no more than a high school education, were not farmers, were alive as of January 1, 1967, and had a history of occupational exposure to dust, fumes, vapors, gases, chemicals, or radiation. The age-standardized lung cancer mortality rate for non-smoking controls (i.e., the baseline rate) was 11.3 deaths per 100,000 man-years; the rate for smoking controls was approximately 11 times higher (122.6 per 100,000 man-years). The lung cancer mortality of non-smoking asbestos workers was 5 times higher (58.4 deaths per 100,000 man-years) than that of non-smoking controls (11.3 deaths per 100,000 man-years). Hammond et al. (Ex. 84–047) found that the lung cancer mortality of asbestos workers who smoked was 601.6 per 100,000 man-years, a value that is also about five times higher than the baseline rate of lung cancer mortality for smoking controls.

Selikoff et al. (Ex. 84–190) examined the effects of cigarette smoking and asbestos exposure among 582 amosite production workers, 567 of whom had smoking histories. As in the study by Hammon et al. (Ex. 84–047), the age and cause-specific mortality rates were compared within each smoking status category defined by the American Cancer Society cohort. Selikoff et al. (Ex. 84–190) concluded as follows:

> Here asbestos exposure greatly multiplied the already high risk that would have been present with cigarette smoking alone. . . . This increased risk is very much the same as that seen among asbestos insulation workers [who smoked]. This observation indicates that the increased risk of death from lung cancer among cigarette-smoking asbestos workers is a specific interaction rather than coincidental, and not, for example, the result of other agents in the environment of the construction trades." (Ex. 84–190).

In the November proposal, OSHA presented two ways of calculating the *probability* that any single case of lung cancer in a person with known exposure to asbestos could be attributed to the asbestos exposure. The first way, proposed by Enterline (Ex. 84–128), was based only on relative risk estimates. Using data from Selikoff et al. (Ex. 84–090) on asbestos insulation workers, Enterline estimated that there was a probability of 75 percent that lung cancers were attributable to asbestos exposure; this probability applied both to smoking and non-smoking asbestos workers. However, in the case of asbestos workers who smoked, OSHA deems it inappropriate to dichotomize causation in terms of smoking *or* asbestos exposure because of the synergistic effect between cigarette smoke and asbestos. OSHA therefore presented its method of calculating the probabilities of causation in the November publication (Table 6 and Table 7, 48 FR 51110). Although OSHA's calculations differ from Enterline's calculations of attributable risk by including a factor for synergism, the two probability estimates do not differ by a great extent. According to OSHA's calculations, asbestos exposure contributes to 79.4 percent of lung cancer deaths among asbestos-exposed workers who smoke, and 77.2 percent of lung cancer deaths among nonsmoking asbestos workers.

Lung Disease and Chest X-ray Abnormalities

In the study by Hammond et al. (Ex. 84–047) discussed above, it was also reported that asbestos insulation workers who smoked one or more packs of cigarettes per day had an asbestosis mortality rate 2.4 times higher than that of asbestos insulation workers who had never smoked regularly. Selikoff et al. (Ex. 84–190), however, observed no increased risk of death from asbestosis among amosite production workers who smoked compared to their nonsmoking co-workers.

Weiss (Ex. 84–097) conducted a chest x-ray and questionnaire survey of 100 asbestos textile workers. Chest roentgenograms were examined for evidence of pulmonary fibrosis. Two asbestos exposure groups were defined: those with less than 20 years of exposure and those with 20 or more years of exposure. The age-adjusted prevalence of pulmonary fibrosis among smokers and non-smokers was 40 and 23 percent, respectively. None of the 11 non-smokers with less than 20 years of asbestos exposure had pulmonary fibrosis, in contrast to 29 percent of the smokers with less than 20 years of exposure to asbestos. The median duration of exposure to asbestos was similar for these two groups. Based on these findings, Weiss (Ex. 84–097) concluded that both asbestos exposure and cigarette smoking were associated with pulmonary fibrosis and that asbestos workers who smoked had a higher prevalence of fibrosis relative to that among nonsmoking asbestos workers. Weiss did not indicate whether the difference in the prevalence of pulmonary fibrosis between smokers and nonsmokers was statistically significant. OSHA tested the significance of the reported difference using a chi-square test of proportions and did not find a significant difference (p greater than 0.1). This study and its findings were criticized by Kilburn (Ex. 84–237) because Weiss used a definition of pulmonary fibrosis that differed from the standard International Labour Office