criterion. Citing a study by Samet et al., which used a relatively large cohort, Kilburn argued that smoking neither produced the x-ray appearance of pulmonary fibrosis nor contributed to fibrosis resulting from asbestos exposure.

Pearle (Ex. 84–079) studied 141 shipyard workers who were referred for medical exams because of suspected asbestos-related lung disease. The shortest duration of exposure in this group was 7 years. Chest x-rays were taken on all subjects and pulmonary function data were collected, including FVC, FEV_1, and diffusion capacity. X-rays were examined for pleural thickening and interstitial abnormalities consistent with asbestosis. Smoking groups were defined in terms of nonsmokers, light smokers, moderate smokers, and heavy smokers. Three asbestos exposure groups were also defined as being mild, moderate, or heavy, based on the duration of exposure (0–14 years, 15–19 years, and 30+ years, respectively). Three percent of the nonsmokers had interstitial disease, all of whom were concentrated in a heavy exposure group. By contrast, 8–12 percent of the smokers had significant interstitial disease, with the highest prevalence in the mild and moderate asbestos exposure groups. These differences between nonsmokers and smokers, however, were not statistically significant. The prevalence of pleural disease in heavy smokers was 25 percent, compared with 9 percent in nonsmokers. This difference was statistically significant. The prevalence of pleural disease among the light and moderate smoking groups was similar to that in heavy smokers. The largest difference in the prevalence of pleural disease between heavy smokers and nonsmokers is found in the group with mild asbestos exposure. These prevalence measures were not adjusted for age, however, and it cannot be concluded definitively that the statistically significant difference in prevalence between heavy smokers and nonsmokers is attributable to smoking history alone.

Berry et al. (Ex. 84–020) studied 379 men employed in an asbestos textile mill. Two cohorts were defined; those first employed before 1951 and those employed on or after 1951. The mean cumulative exposure for the earlier cohort was approximately twice that of the more recent cohort. Smoking histories were available for 376 men. Five smoking groups were defined: Never smoked, 1–4 cigarettes per day, 5–14 cigarettes per day, 15+ cigarettes per day, and ex-smokers. In the most recent cohort, the prevalence of crepitations, possible asbestosis, certified asbestosis, and small radiological opacities was higher among heavy and ex-smokers compared with light smokers (1–4 cigarettes per day) and nonsmokers. For example, 15 percent of heavy smokers had certified asbestosis versus none in the nonsmoking and light smoking groups. By contrast, there were no apparent differences in the prevalence of asbestosis or other conditions among the five smoking groups from the earlier cohort, which incurred a higher mean cumulative exposure, was older, and had a longer period of followup than the more recent cohort. This study suggests that, although there may be differences in the prevalence of asbestosis among smokers and nonsmokers who have been exposed recently to asbestos, the prevalence of asbestosis among smokers and nonsmokers tends to be more similar as the latency period increases or at higher levels of exposure to asbestos.

One additional study received since the November proposal is pertinent to this issue. Nicholson and his colleagues obtained chest x-rays and administered pulmonary function tests to 916 brake line repair and maintenance workers and approximately 205 nonexposed blue collar workers (Ex. 172–B). Chest x-ray abnormalities were defined to include parenchymal changes of 1/0 or greater, pleural thickening, pleural plaques, and pleural calcification. Predicted values for spirometry were based on the revised analysis by Miller et al. (1980) of the 1971 data of Morris, Kuski and Johnson (Ex. 172–B).

The percentage of workers with any evidence of chest x-ray abnormality among those with garage employment was 24.2 percent compared with 18.8 percent among workers with no stated asbestos exposure or garage employment (Ex. 172–B). This overall difference between the two groups is accounted for by differences in the prevalence of parenchymal abnormalities (19.0 percent vs. 15.3 percent) rather than pleural abnormalities (8.4 percent vs. 8.9 percent). However, significant differences existed in the percentages of pleural abnormalities among those employed in work having direct asbestos exposure (22.2 percent) or shipyard employment (25.2 percent) and those employed only in garage work (8.4 percent) or having no asbestos exposure (8.9 percent).

These results were interpreted by the authors to mean that "pleural abnormalities often appear from relatively low asbestos exposures and can exceed parenchymal abnormalities in prevalence at long times from onset of exposure" (Ex. 172–B, p. 29). Similar results were obtained after standardizing for age and smoking history.

The pulmonary function test data, when standardized for smoking, indicated virtually identical results for the unexposed controls, the brake repair workers, and individuals exposed or possibly exposed to asbestos (Ex. 172–B). The investigators note that these findings are not surprising because "forced vital capacity is usually a less sensitive determination of asbestos-related changes than the presence of x-ray abnormalities and forced expiratory volume in 1 second relates to exposures other than asbestos" (Ex. 172–B, p. 46). Although this study (Ex. 172–B) provides evidence that asbestos causes chest x-ray abnormalities over and above those that may be caused by smoking, the data were not sufficient to show that asbestos-exposed workers who smoke suffered more lung impairment than either asbestos-exposed nonsmokers or non-exposed smokers (Ex. 172–B).

In summary, OSHA finds that there is limited though conflicting evidence that asbestos workers who smoke have a higher risk of dying from asbestosis, as well as a higher prevalence of crepitations, lung function decrements, and small radiological opacities than their nonsmoking co-workers.

F. Relationship of Fiber Size and Type of Risks from Asbestos-Related Disease

1. Evidence for a Differential Risk by Fiber Type

In the November proposal (48 FR 51110), OSHA reviewed numerous epidemiological studies concerning the toxicity and carcinogenicity of different asbestos fiber types. OSHA concluded that all fiber types, alone or in combination, have been observed in studies to induce lung cancer, mesothelioma, and asbestosis in exposed workers, with the exception of anthophyllite, which has been observed to induce lung cancer and asbestosis, but not mesothelioma (OSHA/NIOSH, Ex. 84–200; for amosite: Seidman et al., Exs. 84–87, 261–A; Anderson et al., Ex. 84–17; and Murphy et al., Ex. 84–311; for chrysotile: McDonald et al., Ex. 84–65; McDonald and Fry, Ex. 84–64; Liddell et al., Ex. 84–59; Nicholson et al., Ex. 84–72; Rubino et al., Ex. 84–86; Dement et al., Ex. 84–37; Acheson and Gardner, Ex. 84–15; and Berry and Newhouse, Ex. 84–21; for crocidolite: Jones et al., Ex. 84–138; Hobbs et al., Ex. 84–132; McDonald and Newhouse, Ex. 163; Berry and