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29 CFR Parts 1910, et al.

Occupational Exposure to Asbestos; Final Rule

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crocidolite and amosite asbestos be prohibited (Ex. 21 A, p. 9). In particular, two papers by Mossman, et. al, are cited as the basis for the claim that a scientific "consensus" believes that chrysotile carries a reduced carcinogenic risk (Ex. 1–153, 151). Thus AIANA states that "since OSHA issued its 1984 asbestos risk assessment, the scientific consensus that chrysotile asbestos poses lesser risks has solidified" (Ex. 142 at 3).

However, OSHA notes that various participants in this rulemaking, including NIOSH and Dr. Nicholson, disputed the existence of such a consensus. Dr. Nicholson and others including Dr. Landrigan, in a letter to Science, (Ex. 1–155), dispute various interpretations of data in Mossman et al.'s paper, and challenge the conclusion that chrysotile asbestos carries little cancer risk. Nicholson et al, point out that human studies show excess lung cancer risk that is proportionate to exposure across all fiber types, and that animal tests confirm these relationships. OSHA believes that the scientific community has not achieved "consensus" on these issues.

Among the studies submitted in support of the lowered risk of chrysotile asbestos, are those of Churg, and others showing that the lung burden of mesothelioma victims is predominantly amphibole, even though high chrysotile exposure levels were reported. As noted above, this line of argument was presented in the earlier asbestos rulemaking, and OSHA had concluded that lung burden studies are inconclusive. Additional response to this argument is provided by Dement who notes that "(t)he biological significance of post-mortem lung fiber burden data has yet to be established. These data are not useful as a predictor of disease for several reasons. Chrysotile is known to split longitudinally and partially dissolve in the lung whereas amphiboles remain in the lungs for years without significant dissolution * * *. Measurements of tissue fiber burdens many years after first exposure may bear no relationship to the carcinogenic events which likely have taken place many years before clinical manifestation of cancer." (Ex. 1–273)

BCTD pointed out in its post-hearing brief, that "Dr. Landrigan testified, while the observation that chrysotile does not last as long in the lungs as other forms of asbestos is not new knowledge (Tr. 1074), there is recent evidence that chrysotile is "the most effective of the three major fiber types at migrating to the pleura, that it is present in substantial amounts in pleural plaques and mesotheliomas even in circumstances where it is not present or minimally present in the lungs themselves" (Tr. 1074).

The Agency also notes that the HEI report, in summing up its discussion of its literature search of studies examining the issue of the relative potency of chrysotile in inducing mesothelioma, stated: "(t)he evidence that chrysotile rarely causes pleural mesothelioma is not conclusive "* * * and concluded that the absence of mesothelioma in one of the "two cohorts of heavily exposed asbestos workers who worked only with chrysotile * * * seems likely to be due at least in part to chance" (Ex. 1–344 p. 6–23).

HEI concluded that "the mesothelioma risk for chrysotile was an issue of disagreement; some members of the Literature Review Panel held the view that a lower estimate should be recommended, as it would be more consistent with available data. The crucial issues, neither of which can be resolved unequivocally, are (1) what proportion of the mesotheliomas observed in groups such as the U.K. textile workers and the U.S. insulation workers were caused by their exposure to crocidolite or amosite; and (2) whether the best general estimate of the ratio of mesothelioma to excess lung cancer caused by chrysotile is provided by the Quebec miners and millers (about 1:4 or 1:5), or by the South Carolina textile workers handling Quebec fiber (zero)" (Ex. 1–344 p. 6–32).

Thus, although there is some evidence linking chrysotile to a lower mesothelioma rate than some amphibole fiber types, OSHA believes that there is insufficient evidence to show that chrysotile does not present a significant mesothelioma risk to exposed employees. Furthermore, the major disease linked to asbestos exposure, lung cancer, occurs at the same frequency among employees exposed to equivalent doses of chrysotile or to amphibole asbestos fiber types. Indeed, evaluation of all of the evidence indicates that chrysotile asbestos presents a similar significant risk of lung cancer and asbestosis as other forms of asbestos. Since these adverse health effects constitute the majority of diseases related to asbestos exposure, OSHA is still of the opinion that chrysotile exposure should be treated the same as other forms of asbestos.

In addition to contentions that OSHA's risk assessment had overstated asbestos risks because it treated the risks from all asbestos fiber types equally, other contentions were made that the earlier risk assessment may have understated the risks from asbestos, because it ignored evidence of the incidence of pleural plaques, and other asbestos disease which occurred in workers exposed at low levels, primarily as building custodians. The earlier risk assessment in 1984 focused on whether there was a significant risk of cancer and asbestosis at various levels of cumulative exposure. During this hearing, various labor groups stated their position that the presence of pleural plaques in asbestos exposed employees is not only a marker of asbestos exposure, but also an independent "material impairment" because they are associated with a greater risk of lung function impairment and pleuritic pain. Pleural plaques are focal areas of fibrous thickening of the pleura, the membrane lining the lung. Further, suggestions were made that OSHA should reduce its PELS to correspond to these increased risks of "material impairment" which occurred at lower exposure levels (see e.g., Ex. 143 at 35–37).

Evidence submitted during the rulemaking consisted of testimony and studies which in the view of some participants showed lung function decrement and resulting excess disease among workers exposed at low levels. For example BCTD witness Dr. Christine Oliver described various studies and concluded:

> Pleural plaques * * * were a predictor for increased mortality from lung cancer and malignant mesothelioma in subsequent years * * * pleural plaques have also been shown to be associated with decrement in lung function * * * At the very least, pleural plaques are a marker for exposure, sufficient to increase risk for lung cancer and for malignant mesothelioma, and they have also been associated with loss of lung function (Tr. 1035–6).

Dr. Oliver recommended medical surveillance of those exposed to asbestos in their capacity as custodians in buildings.

The studies considered by Dr. Oliver consisted of one involving 120 Boston public school custodians (Tr. 1026) which she conducted and found pleural plaques in 33% (N=40) of the group. Further she noted that in 21% (of the 40, or 12 individuals) there was no known exposure to asbestos outside work as school custodian. In 18% of the group and 17 % of those with no outside exposure to asbestos, she observed a restrictive pulmonary defect, significantly associated with duration of employment as school custodian. Other studies described by Dr. Oliver, in the docket include: a study of 666 New York school custodians, reporting only x-ray data (Ex. 47). For all groups of workers, the lung abnormality seen on x-ray was associated with duration of

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work as custodian: a study of 1,117 insulation workers (likely to have had extensive asbestos exposure) by Dr. Irving Selikoff, in which workers were followed for up to 27 years prospectively, in which pleural plaques were found and which were concluded to be predictive of lung cancer mortality (Tr. 1036 and Ex. 124A); a study, by Balmes (Ex. 124 DD, Tr. 1036, Ex. 1–374) of approximately 900 school district employees in California were determined as likely to have been exposed to asbestos. The authors concluded, "More than 11 percent of workers known to have sustained exposure to ACM in school building, without history of exposure to asbestos prior to school district employment, and with at least 10 years of employment with the district had radiographic evidence of parenchymal asbestosis and/or asbestos-related pleural thickening" (Ex. 1–374, p. 547). After adjusting for smoking and age, the relative risk was 1.3 times greater for those with 10 years or more employment compared with those who had just begun working for the school district.

In addition to the occurrence of pleural plaques which are viewed as presenting an independent material impairment of health due to low level asbestos exposures, Dr. Oliver cited other studies which correlated low level asbestos exposure with mesothelioma. Thus, a study by Dr. H. Anderson (Tr. 1032 and Ex. 124 EE, Ex. 1–374 using information on mesothelioma cases from a Wisconsin Cancer Registry, analyzed 359 deaths from 1959 to 1989. Using death certificate occupational information, the researchers hypothesized 41 as likely to have been exposed to asbestos in buildings. For 10 (34%), no other likely source of asbestos exposure was identified. The paper concluded that "individuals occupationally exposed to in-place ACBM are at risk for the subsequent development of mesothelioma" (Ex. 1–374, p. 570).

SBA submitted a critique of these studies which they commissioned by Drs. H. Weill and J. Hughes (Ex. 122). They suggested potential biases in these studies, that Dr. Oliver's study subjects were volunteers, the study had a low participation rate, they had used a non-standard classification system, and did not adequately account for age in relating restriction to lung function. These reviewers concluded that spirometric functional measurements were not related to the presence of plaques and that reduced lung volume could result from other factors. Drs. Weill and Hughes also examined the other studies, and argued that Dr. Selikoff's were "fatally flawed" due to the potential for development of unmeasured changes during the 27 year period of follow-up, and that both the Anderson and Balmes studies failed to adequately adjust for age, smoking and other direct asbestos exposures. Other reports cited by BCTD were dismissed because of potential sources of bias.

Dr. Oliver rebutted these arguments (Ex 143, Attachment F). She argued that she had adequate controls, adequately accounted for age and demonstrated that pleural plaques were significantly associated with both latency and duration of work as custodian in the total group and in the group with no known other exposure, that lung restriction was significantly associated with duration of work as a custodian, and that pleural plaques mark increased risk for lung cancer mortality.

Dr. Levin also responded to the reviewer's criticism of his studies with Dr. Selikoff (Ex. 143, Attachment G). He pointed out that all x-rays had been read by a single reader, Dr. Selikoff, and that there is no evidence that smoking without asbestos exposure increases appearance of the small irregular opacities in the lung seen on the x-rays in their study. He further noted that in his study only actively working custodians were included and were therefore a "survivor" group and would therefore not be expected to report pulmonary dysfunction frequently. He claimed that relatively unexposed subject groups would not be expected to have more than an upper limit of 3% pleural plaques.

Dr. Anderson also responded to the Weill/Hughes comments (Ex. 143, Attachment H). He asserted that the review fails to explain how biases would significantly increase odds ratios in the study, that misclassification often is random and biases toward not detecting a difference between the study and control groups. He also questioned existence of evidence that smoking without asbestos exposure causes pleural thickening or irregular opacities.

The review of available literature, including the studies mentioned above by the Health Effects Institute, resulted in its the estimation that the prevalence of pleural plaques in the general population to be about 5% (Ex. 1–344, p. A2–9). Although HEI advised caution in interpreting the existing studies due to lack of specificity and sensitivity of methods used and couched its conclusions in cautious terms, they concluded: "* * * there is now persuasive evidence implicating asbestos-related pleural disease as an independent cause or indicator of functional impairment and possibly even disability * * * On the individual level, pleural disease may be the only indication of asbestos exposure, may explain symptoms and function impairment, and may predict future deterioration in lung function" (Ex. 1–344 p. A2–12).

OSHA agrees that health effects such as lung function impairment and pleuritic pain would be considered "material impairment," if substantial evidence supports the link to pleural plaques. OSHA concludes that the scientific data indicate that pleural plaques are primarily associated with asbestos exposure, and that they have occurred and still may at relatively low exposure levels.

However, OSHA does not believe that the data are available to permit OSHA to do a separate risk assessment for these effects which would in a major way add to the present assessment. The risk assessment on which OSHA has based its significant risk determinations for the 1986 and newly revised standards, calculated the incidence of mesothelioma, lung and other cancers and asbestosis, diseases based on a substantial amount of both mortality and exposure data. The data concerning lung function decrement and pleural plaques lack exposure information and would make quantitative risk estimates for these health effects less precise than the data for other forms of asbestos-related disease upon which OSHA is relying.

A separate risk assessment is also unnecessary. OSHA believes that the revised regulations are already regulating at the margin of what is feasible, in terms of levels to be achieved, and controls which are required. OSHA has imposed necessary, feasible and well supported work practices for custodial work, which should reduce custodial exposures well below the historic levels (indeterminate) which may have been experienced by the workers studied in the above reports.

More generally, there would be remaining significant risk at this new 0.1 f/cc exposure limit if there were not other provisions to these standards. However, the exposure limit is accompanied by mandated work practice controls and requirements for hazard communication, training and other provisions. Together these will very substantially reduce that remaining significant risk, although the exact amount of that reduction cannot be quantified. In addition, it would be difficult to measure accurately in the industrial setting levels lower than those in these standards. OSHA believes

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