could explain an increased frequency of pleural plaques in exsmokers because damaged clearance mechanisms unaided by continued smoking may lead to higher "effective" doses of asbestos in the tissues.

The clinical significance of a relationship between the prevalence of pleural plaques and smoking depends on the clinical significance of plaques. The immediate importance of these lesions is negligible but there is some suspicion that plaques may be indicators of an enhanced risk of lung cancer[10,11] and mesothelioma.[11]

With respect to lung cancer risk the evidence is equivocal. Kiviluoto et al[10] found 700 cases of calcified plaque in 6,000 adults during a mass chest x-ray survey in Tuusniemi commune, Finland, in 1962. The lung cancer incidence between 1962 and 1977 was almost identical to the incidence in controls without plaques in Maaninka commune. 13 and 14, respectively. It may be important to note that this study was limited to calcified plaques. In a case-control study of 69 lung cancer cases there was an elevated risk of lung cancer of 2.8 only when there was pulmonary fibrosis in addition to calcified plaques. Information on smoking habits was not provided.

Edge[11] has reported an increased incidence of lung cancer in 429 older British shipyard workers with pleural plaques unassociated with pulmonary fibrosis found between 1964 and 1971 who were compared with a matched control group of 429 men in another city where there was neither known asbestos exposure nor plaques. After excluding six men with lung cancer who had clinical evidence of tumor at the time observation was begun in the men with plaques, the relative risk was 3.25 (13 cases in the men with plaques compared to 4 in the controls, chi square with Yates' correction = 3.84, $p = 0.05$). However, this comparison is probably invalid because only 231 of the 429 men with plaques were discovered by routine chest films and the remaining 198 were found by clinic or hospital films whereas all 429 controls had routine films. Thus, there was undoubtedly some selection bias operating to increase the incidence of lung cancer in the men with plaques. Comparing only the groups that had routine films, six (2.6%) of 231 men with plaques and four (0.9%) of 429 controls developed lung cancer, a difference which is not statistically significant at the 0.05 level. Furthermore, half the lung cancer cases in the men with plaques were diagnosed in the first two years of follow-up and therefore these individuals presumably had their tumors at the time observation began. After exclusion of the early cases, the relative risk, as compared to the incidence of lung cancer in the general population, remained at 1.0 until after six years of follow-up it rose to 2.1 (6 cases compared to 2.8 expected), which also is not statistically significant at the 0.05 level. Most importantly, no smoking habit information was available for adjustment of the relative risk.

A similar problem indicating selection bias in the men with plaques operated with respect to the incidence of mesothelioma: most of the cases occurred early in the follow-up period and only two developed after six years of follow-up. However, mesothelioma is so rare in the general population that even two cases in the men with plaques is suspicious, especially since mesothelioma is not related to smoking.

Thus, at present the prognostic significance of pleural plaques with regard to the risk of lung cancer is doubtful. In the absence of smoking-habit data, no conclusions are warranted. If plaques are related to smoking as well as to asbestos exposure and if lung cancer is similarly related, then any association between plaques and lung cancer is likely to be spurious.

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