smoking habits, but also regarding the occurrence of parenchymal small lesions according to ILO. As seen from the tables, the relative risk for lung cancer for patients with changes compatible with asbestosis (1/0 or more) was 2.03, and for those without asbestosis it was 1.56.

All the studies cited here and seen in the table agree fairly well. One must therefore conclude that lung cancer risk is increased also in patients without asbestosis.

Mechanism of asbestos-related lung cancer

It has been claimed that fibrosis in the lung parenchyma is unlikely to cause cancer in the large bronchi, where a large part of asbestos-related cancers are seen (58, 152, 153). In tracheal organ cultures, necrosis and desquamation of surface cells accompanied by basal cell hyperplasia can be seen after 1 week (154), and this occurrence is presumably independent of any accompanying parenchymal changes. These changes presumably cause an increase in the susceptibility of epithelial cells of the bronchi to be transformed by environmental carcinogens (31). Fibrosis of the lungs and cancer of the bronchi can thus be seen as end points of 2 unknown mechanisms that may work independently (153).

Concluding remarks

There is an increasing body of evidence which indicates that asbestos at low exposure levels produces a slight increase in the relative risk of lung cancer. The relative risk of cancer in asbestosis patients is higher, but it is unclear whether this higher risk is attributable entirely to higher fiber burden within lung tissue (a dose-response effect) or whether there is also an adjuvant effect of fibrosis by way of cytokine production, over and above the dose effect.

This conclusion is not a purely academic question but has important practical consequences in 2 different areas. One is in the legal world and the other is in the world of industrial hygienists. The legal consequences have an important bearing for many persons. Accepting a no-threshold hypothesis for lung cancer would open the field for a large number of patients with low-grade exposure to claim compensation for lung cancers caused by asbestos exposure, even when the principal cause of the tumor is smoking. However, science should try to find the facts independent of how the law might be affected by these findings. In other words, law should follow science, not the other way around. The legal world has to come to terms with whether — and if so, how much — a small increased risk, such as an increased risk of 10% or 20%, should be compensated. Proposals have been made (33).

The more important fact is that even if an increased risk of 10% or 20% is not very important for a person, it will result in a large number of bronchial carcinomas in the general population where smoking, unfortunately, is far from eliminated. Given this fact, society cannot conclude from the present data that lung cancer risk is increased only when exposure is heavy enough to cause asbestosis. Anyone claiming such an unproved hypothesis shoulders a heavy responsibility. All exposure to asbestos must be minimized, and if asbestos is to be used, stringent precautions must be taken.

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