The silica carcinogenicity issue in Japan

In the opinion of this writer, the recent action by the Japanese government amending relevant laws designating lung cancer as a compensable complication of pneumoconiosis is a big step towards improvement. Pneumoconiosis victims who develop lung cancer will be compensated under the national compensation plan. In Japan, there are currently an estimated 12,000 pneumoconiosis victims. However, I would like to express here reservations regarding some aspects of the amendment to the law. This is aimed at the: (1) overall lung cancer risk among silica containing dust exposed workers; (2) lung cancer risk among dust exposed workers, in particular for non-silicotics and silicotics; (3) lung cancer risk among pneumoconiotics; and (4) lung cancer risk separate from non-silicotics and silicotics. Further, combined with the aforementioned negative assertion on animal and mutagenicity studies, the Committee drew the conclusion that “there is no evidence to support the carcinogenicity of silica itself”, and “further findings are needed for judgment”. The most serious problem with the reasoning behind the recommendation is the reliance on eight epidemiological studies showing lung cancer risk separately for non-silicotics and silicotics. However, it is clear that some of these studies were designed to specifically address the issue of dissociating risks between non-silicotics and silicotics, with the notable exception of the study by Cook and colleagues, in which lung cancer risk was detected in relation to cumulative exposure among non-silicotics. In the remaining studies where such data were lacking, most authors acknowledged the possibility that the exposure profile of subjects was represented by the status of fibrosis, so the presence/absence of silicosis should be regarded as a marker of high/low cumulative exposure. The argument that silicosis is, but silica itself is not, a risk factor for lung cancer cannot be advanced from a pooled risk calculated for the non-silicotics in these studies. Further, since such studies tend to underestimate the fact that the distinction between the presence/absence of fibrosis is arbitrary because fibrosis occurring at microscopic levels often escapes radiological detection. The fairly limited scope of the available epidemiological literature warrants that the silica carcinogenicity issue be treated in perspective, combining findings from the broader spectrum of silica exposed subjects, including non-silicotics and silicotics. The rebuttal of the animal and mutagenicity studies as failing to provide evidence of silica carcinogenicity only lessens the scientific credibility of the Committee’s argument. Finally, I reiterate that significant progress has been made administratively in Japan to provide improved opportunities for follow up of pneumoconiotic victims and better compensation if and when they develop lung cancer. Needless to say, such action falls into the realm of secondary and tertiary prevention. Further steps should be taken to reevaluate silica carcinogenicity and incorporate it into administrative measures aimed at primary prevention.

K Takahashi
Department of Environmental Epidemiology, University of Occupational and Environmental Health, Chiba, Japan.

References

www.occenvmed.com


