Dear Editor,

Some key material presented in this article [1] is ambiguous, vague, and confusing. The conclusions are unsupportable.

The “lung dust analysis” is never defined. Although both asbestos bodies (ABs) and asbestos fibers were reportedly counted, lung fibers were not characterized with regard to the number or type; only ABs were used in the data analysis.

The histologic analysis, the second leg of a two-legged stool, is based on flawed criteria. The authors use the Asbestosis Committee of the College of American Pathologists and Pulmonary Pathology Society (CAP/PPS) criteria rather than the more credible Pneumoconiosis Committee of the College of American Pathologists and the National Institute for Occupational Safety and Health (CAP/NIOSH) criteria to classify asbestosis grade 1, necessary in Germany for the diagnosis of asbestos-related lung cancer in the absence of asbestosis, pleural plaques or cumulative asbestos exposure of > 25 fiber-years [2–4]. The CAP/PPS criteria have been criticized on the basis of: histologic criteria that combine CAP/NIOSH grade 0 and grade 1 asbestosis into a single category: grade 0, and the requirement of an average concentration of at least 2 ABs/cm² of lung tissue [5]. This concentration of ABs would equate to 4000 ABs/gm wet lung, which is 200 times the upper limit of the range of 0–20 ABs/gm of wet lung observed by Roggli and Sanders in their laboratory among patients with no history of asbestos exposure [6]. The diagnostic criteria chosen by the authors for asbestosis grade 1 are controversial and restrictive and explain the low frequency of asbestosis found in their study [4].

The authors fail to provide support for their statement: “Asbestos fibers, including chrysotile/white asbestos which was the main type used in Germany, have a very long half-life in the human lung” [1, p. 300–1].

- As noted above, only ABs were counted and used in their data analysis. Amphibole fibers form ABs; chrysotile, as a general rule, does not.
- Asbestos fibers were counted using the differential inference contrast technique with polarizing imaging; a subset was analyzed with electron microscopy. For that subset, there presumably is the data on the fiber number by type of fiber. This data was not presented.
- The authors’ own data shows decreasing numbers of ABs with the lapse of time following the last exposure. One explanation for this finding is the low biopersistence of asbestos in the human lung. The explanation chosen by
the authors is the aging German population, the explanation for which they fail to provide pertinent evidence. The Figure 4 [1] presents relationships between ABs and the presence or absence of asbestosis as a dichotomous variable indicative of a threshold. However, several authors, including Stayner et al., who examined the relationship between exposure to chrysotile asbestos and asbestosis in a large cohort of the U.S. textile workers, found no evidence of a threshold for asbestosis or for lung cancer [7]. The authors ignore the overwhelming scientific evidence of low biopersistence of chrysotile fibers in the human lung. Accordingly, the diagnosis of asbestos-related disease (ARD) based on the fiber analysis of lung tissue systematically results in a false-negative diagnosis [3,5,8–16]. In conclusion, we consider that the study by Feder et al. [1] uses flawed methods to make the diagnosis of asbestosis grade 1 more difficult. It also misrepresents the low biopersistence of chrysotile asbestos in the human lung. Although ABs and/or pathology may identify cases of asbestos-related lung cancer, they should never be used as required criteria. In doing so, the rightful access to workers’ compensation for asbestos-related lung cancer and other ARDs in Germany and elsewhere in the world is incorrectly denied.

Key words:
Asbestos, Asbestos bodies, Asbestos related diseases, Histopathology, Lung fiber burden, Asbestos workers

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