Correspondence regarding the article "The asbestos fibre burden in human lungs: new insights into the chrysotile debate"

To the Editor:

The article by Feder *et al.* [1] states that the lung asbestos fibre burden in 23 955 patients was analysed to address fibre type and biopersistence; data from 12 patients undergoing two tissue excisions at intervals at least 4 years were considered.

We believe that the article has serious shortcomings, as follows.

1) Unclear aim. Contrary to the authors' claim, there is no ongoing debate about the biopersistence of chrysotile asbestos among independent, credible scientists. In support of their claim that such a debate exists, the authors rely on an article commissioned, funded and developed in collaboration with asbestos lobbyists.

2) Faulty study design. Significant scientific problems in patient/sample selection and applied methods exist. First, the small sample size: only 12 (0.05%) of the 23 955 cases were analysed with two investigations; only six had electron microscopic examination of tissue. Second, the selection criterion of 500 asbestos bodies per gramme of wet lung is discretionary and arbitrary. Third, relationships between outcome and fibre-years were not examined using a detailed occupational history. No statistical analysis accounting for occupation/exposures, interim exposures and latency periods, and exposure changes over recent decades is reported.

3) Methods. The authors do not explain why they used both field energy (FE) scanning electron microscopy (SEM) and transmission electron microscopy (TEM), nor is it clear which data come from which method in the supplementary table. TEM is regarded as the method of choice by impartial and credible pathologists; the limitations of SEM have been discussed previously [2].

The use of FE-SEM and TEM on autopsy specimens alone does not allow the authors to say anything about the change in the number of chrysotile fibres over time.

The term "pulmonary asbestos fibre concentration" defined as "total of asbestos bodies and bare fibres" is incorrectly conflated with asbestos fibre concentration or burden as generally understood.

4) Results. First, there is no information on fibre length, which would be crucial for understanding outcome since short fibres are more rapidly cleared from the lung [3, 4].

Second, in the six cases analysed with electron microscopy, the fibre counts reported are generally similar to, or even lower than, asbestos body counts. This unusual finding contradicts the literature where total fibres outnumber asbestos bodies by three orders of magnitude [5].

Third, in a large number of ferruginous bodies, the authors were unable to identify the core material. How did the authors define a ferruginous body as asbestos and non-asbestos in those cases not subjected to electron microscopy?

Last, the authors mention that "asbestos grading followed national and international criteria, *i.e.* primarily the Helsinki criteria". It is unclear which criteria were used in each case. There are significant differences

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All asbestos types cause asbestosis (cancer): chrysotile is not biopersistent, so fibre analysis is not diagnostic http://ow.ly/BOLC30grqYg

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between the NIOSH/CAP definition and the definition of Roggli and co-workers and its respective formulation in the Helsinki criteria 2014 [6] (for further details, see [7]).

5) Data analysis, data interpretation and conclusions. First, in the six cases with fibre type differentiation, 10% to 95% were reported to represent chrysotile. In Germany, about 94% of asbestos used was chrysotile. The relative paucity of chrysotile fibres in cases 1 and 2 (33% of the cases) with intervals of 14 and 21 years between the first and second examinations is consistent with low biopersistence of chrysotile fibres.

Second, the authors state "Thus fibre clearance and biopersistence are considered the most important factors for diagnostics and risk assessment of malignant and non-malignant diseases." In fact, diagnosis is based mainly on a thorough occupational history and noninvasive clinical findings; risk assessment is related to fibre concentration in the workplace [8].

Third, the major interpretation of the data by the authors is that chrysotile fibre counts, like those of amphibole fibres, do not change over time in the human lung. Their findings and extensive literature show just the opposite [8].

6) Discussion. The authors claim an ongoing debate about the hazardous nature of chrysotile. Publications not cited by Feder *et al.* [1] (IARC, WHO), and other professional bodies and government agencies contradict this claim, concluding that chrysotile asbestos exposure increases risk for asbestosis, mesothelioma, lung and other cancers [9].

7) Medico-legal relevance. There is grave risk that the publication by Feder *et al.* [1] will influence outcomes in the adjudication of asbestos-related disease in the legal system and that, as a result, the injured worker will suffer unfairly and unjustly. This risk would apply to those with a history of occupational exposure to chrysotile asbestos and in whom few or no asbestos fibres are found in the lung years later. The claim by Feder *et al.* [1] that chrysotile fibres are biopersistent in the lung could be used in courts of law to deny justice to asbestos-harmed victims.

8) Conflicts of interest. The authors fail to disclose significant financial conflicting interests [10].

In conclusion, Feder *et al.* [1] provide misleading findings that fail to refute the generally accepted tenet that chrysotile asbestos fibres are not biopersistent in the human lung. Neither their clinical nor their statistical analyses, nor the literature, support their claims.

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Conflict of interest: Disclosures can be found alongside this article at erj.ersjournals.com

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