

# LETTER TO THE EDITOR (APRIL 4, 2018) CONCERNING THE PAPER “HISTOLOGICAL FINDINGS AND LUNG DUST ANALYSIS AS THE BASIS FOR OCCUPATIONAL DISEASE COMPENSATION IN ASBESTOS-RELATED LUNG CANCER IN GERMANY”

Dear Editor,

The 2017 publication by Feder et al. [1] reiterates the fallacious and repeatedly disproven claim that the analysis of lung tissue for asbestos fiber levels and asbestos bodies is the appropriate technique for assessing past occupational exposure to asbestos. Contrary to an overwhelming body of scientific evidence [2–11] and the recommendations of the National Institute for Occupational Safety and Health [12] as well as the Collegium Ramazzini [13], these authors mistakenly insist that detection of a certain number of asbestos fibers or asbestos bodies in lung tissue is essential for confirming past occupational exposure to asbestos and that it is superior to a carefully obtained history of occupational exposure.

While it is true that finding asbestos fibers or asbestos bodies in lung tissue might provide evidence of asbestos exposure in cases where no exposure history or other proof of exposure is available, the detection of a certain number of asbestos fibers or asbestos bodies in lung tissue cannot be made a universal requirement for confirming exposure to asbestos because such a requirement ignores two well established biological facts.

First, a universal requirement for finding a certain number of asbestos fibers in lung tissue to confirm asbestos exposure fails to recognize that chrysotile asbestos, the predominant form of asbestos in the world markets today is well documented to have only a short residence time in lung tissue [2–8]. Therefore, measurement of chrysotile fibers in lung is an inherently insensitive analysis that carries high likelihood of underestimating or of failing altogether to diagnose past asbestos exposure even in the case of persons with a well-corroborated history of exposure [13].

Secondly, a universal requirement for finding a certain number of asbestos bodies in lung tissue ignores the well-established fact that chrysotile asbestos rarely forms asbestos bodies [9–11]. Therefore, insisting on the presence of certain numbers of asbestos bodies in lung tissue as an index of past exposure is to force reliance on an insensitive diagnostic technique and may lead to false negative diagnoses [13].

Unnecessary lung biopsies constitute a further potential negative consequence of an undue insistence on lung tissue analysis as a criterion for diagnosing past occupational exposure to asbestos. Lung biopsy is an invasive and poten-

tially hazardous procedure [14]. Since lung biopsy carries the risk of medical complications and is unnecessary for a diagnosis of asbestos exposure, the Collegium Ramazzini has stated that lung biopsy is “never ethically justified solely for medico-legal or compensation purposes” [12]. Feder et al.’s [1] insistence on applying the insensitive and outdated technology of lung tissue analysis to the diagnosis of asbestos-related disease combined with their unsound demand for the presence of a certain number of asbestos fibers and asbestos bodies in lung tissue could lead to missed diagnoses of asbestos exposure in the case of individuals and to very substantial undercounting of the true magnitude of asbestos disease in populations. The undercounting of disease that could result from strict application of this practice has been estimated to be as large as 80% [15–17]. If applied in the adjudication of compensation claims for asbestos disease, a strict requirement for lung tissue analysis could lead to judicial error and societal injustice on a very large scale [13,18,19].

The Collegium Ramazzini emphasizes that a carefully obtained history of occupational exposure to asbestos is the cornerstone of an accurate diagnosis of the diseases caused by asbestos [13]. An occupational history taken by a knowledgeable occupational physician and supplemented as necessary by an exposure assessment conducted by an experienced industrial hygienist is a far more sensitive and specific indicator of lung cancer risk from chrysotile asbestos than lung fiber burden analysis or asbestos body counting [20].

**Key words:**

**Occupational exposure, Lung biopsy, Chrysotile asbestos, Asbestos bodies, Asbestos fiber levels, Undercounting of disease**

**REFERENCES**

1. Feder IS, Theile A, Tannapfel A. Histological findings and lung dust analysis as the basis for occupational disease compensation in asbestos-related lung cancer in Germany. *Int J Occup Med Environ Health*. 2018;31(3):293–305, <https://doi.org/10.13075/ijomeh.1896.01148>.
2. Wagner JC, Berry G, Pooley FD. Mesotheliomas and asbestos type in asbestos textile workers: A study of lung contents. *Br Med J*. 1982;285(6342):603–6, <https://doi.org/10.1136/bmj.285.6342.603>.
3. Baker DB. Limitations in drawing etiologic inferences based on measurement of asbestos fibers from lung tissue. *Ann N Y Acad Sci*. 1991;643(1):61–70, <https://doi.org/10.1111/j.1749-6632.1991.tb24444.x>.
4. Kohyama N, Suzuki Y. Analysis of asbestos fibers in lung parenchyma, pleural plaques, and mesothelioma tissues of North American insulation workers. *Ann NY Acad Sci*. 1991;643(1):27–52, <https://doi.org/10.1111/j.1749-6632.1991.tb24442.x>.
5. Churg A, Wright JL. Persistence of natural mineral fibers in human lungs: An overview. *Environ Health Perspect*. 1994;102 Suppl 5:229–33.
6. Finkelstein MM, Dufresne A. Inferences on the kinetics of asbestos deposition and clearance among chrysotile miners and millers. *Am J Ind Med*. 1999;35(4):401–12, [https://doi.org/10.1002/\(SICI\)1097-0274\(199904\)35:4 < 401::AID-AJIM12 > 3.0.CO;2-4](https://doi.org/10.1002/(SICI)1097-0274(199904)35:4 < 401::AID-AJIM12 > 3.0.CO;2-4).
7. Roggli VL, Sharma A, Butnor KJ, Sporn T, Vollmer RT. Malignant mesothelioma and occupational exposure to asbestos: A clinicopathological correlation of 1445 cases. *Ultrastruct Pathol*. 2002;26(2):55–65, <https://doi.org/10.1080/01913120252959227>.
8. Suzuki Y, Yuen SR. Asbestos fibers contributing to the induction of human malignant mesothelioma. *Ann N Y Acad Sci*. 2002;982(1):160–76, <https://doi.org/10.1111/j.1749-6632.2002.tb04931.x>.
9. Dodson RF, Hammar SP, Poye LW. A technical comparison of evaluating asbestos concentration by phase-contrast microscopy (PCM), scanning electron microscopy (SEM), and analytical transmission electron microscopy (ATEM) as illustrated from data generated from a case report. *Inhal Toxicol*. 2008;20(7):723–32, <https://doi.org/10.1080/08958370701883250>.
10. Egilman D. Fiber types, asbestos potency, and environmental causation: A peer review of published work and legal

- and regulatory scientific testimony. *Int J Occup Environ Health*. 2009;15(2):202–28, <https://doi.org/10.1179/oeh.2009.15.2.202>.
11. Hammar SP, Abraham JL. Commentary on pathologic diagnosis of asbestosis and critique of the 2010 Asbestosis Committee of the College of American Pathologists (CAP) and Pulmonary Pathology Society's (PPS) update on the diagnostic criteria for pathologic asbestosis. *Am J Ind Med*. 2015;58(10):1034–9, <https://doi.org/10.1002/ajim.22512>.
  12. National Institute for Occupational Safety and Health. Occupational diseases – A guide to their recognition. Washington: The Institute; 1977 [cited 2018 Mar 18]. Available from: <https://www.cdc.gov/niosh/docs/77-181/pdfs/77-181.pdf>.
  13. Baur X, Frank AL, Budnik LT, Weitowitz HJ, Oliver LC, Welch LS, et al. Collegium Ramazzini: Comments on the 2014 Helsinki consensus report on asbestos. *Am J Ind Med*. 2016;59(7):591–4, <https://doi.org/10.1002/ajim.22595>.
  14. Raghu G, Collard HR, Egan JJ, Martinez FJ, Behr J, Brown KK, et al. An official ATS/ERS/JRS/ALAT statement: Idiopathic pulmonary fibrosis: Evidence-based guidelines for diagnosis and management. *Am J Respir Crit Care Med*. 2011;183(6):788–824, <https://doi.org/10.1164/rccm.2009-040GL>.
  15. Baur X. Asbestos-related disorders in Germany: Background, politics, incidence, diagnostics and compensation. *Int J Environ Res Public Health*. 2018;15(1):143, <https://doi.org/10.3390/ijerph15010143>.
  16. Baur X. Response to the letter of R. Merget, I. Feder and A. Tannapfel. *Pneumologie*. 2017;71(2):121–3, <https://doi.org/10.1055/s-0042-124569>.
  17. Weitowitz H-J. [The theory of asbestos bodies is dead – German mesothelioma register – What next?]. *Zbl Arbeitsmed*. 2016[cited 2018 Mar 18];66(4):232–8, <https://link.springer.com/content/pdf/10.1007%2Fs40664-016-0135-3.pdf>. German.
  18. Baur X, Weitowitz HJ, Budnik LT, Egilman D, Oliver C, Frank A, et al. Asbestos, asbestosis, and cancer: The Helsinki criteria for diagnosis and attribution. Critical need for revision of the 2014 update. *Am J Ind Med*. 2017;60(5):411–21, <https://doi.org/10.1002/ajim.22709>.
  19. Baur X. [Asbestos: Socio-legal and scientific controversies and unsound science in the context of the worldwide asbestos tragedy – Lessons to be learned]. *Pneumologie*. 2016;70(6):405–12, <https://doi.org/10.1055/s-0042-103580>. German.
  20. Begin R, Christman JW. Detailed occupational history: The cornerstone in diagnosis of asbestos-related lung disease. *Am J Respir Crit Care Med*. 2001;163(3 Pt 1):598–9.

Philip J. Landrigan<sup>1</sup> and Richard A. Lemen<sup>2</sup>  
on behalf of the Collegium Ramazzini

<sup>1</sup>Schiller Institute for Integrated Science and Society,  
Chestnut Hill, USA  
Boston College

<sup>2</sup>National Institute for Occupational Safety and Health,  
Canton, USA  
(Retired)

Corresponding author: P.J. Landrigan  
Schiller Institute for Integrated Science and Society  
Boston College  
Chestnut Hill, MA 02467, USA  
(e-mail: phil.landrigan@bc.edu)