

The fibrosis of the lungs that is associated with asbestosis is, however, indistinguishable radiologically from cryptogenic fibrosing alveolitis (an uncommon disease of unknown cause) and the differential diagnosis is a matter of weighing probabilities.

I am sure the lawyers had a field day debating the modern versus historical definition and the diagnostic criteria for asbestosis as well as the relative merits of the single result taken from a thin section of lung tissue and its associated analytical variability compared with the value provided by Mr Howie's clearance model. They may have even turned their minds to determining which of Mark Twains (1835–1910) thoughts was most applicable, such as 'It is the differences of opinion that makes the horse races'. or 'Get your facts first and then distort them as much as you please'. or 'There is something fascinating about science. One gets such wholesale returns of conjecture out of such trifling investment of fact'.

ALAN ROGERS

*Alan Rogers OH&S Pty Ltd,
PO Box 2128,*

*Clovelly, NSW 2031,
Australia*

E-mail: arogersohs@hotmail.net.au

REFERENCES

- Churg A. (1991) Analysis of lung asbestos content [Editorial]. *Br J Ind Med*; 48: 649–52.
- Doll R, Peto J. (1985) Asbestos effects on health of exposure to asbestos. London: Health and Safety Commission. pp. 2 and 31. ISBN 0 716 1075 6.
- Howie RM. (2005) Asbestos lung residue and asbestosis risk. *Ann Occup Hyg*; 49: 95–7.
- Mulhausen JR, Damiano J. (1998) A strategy for assessing and managing occupational exposures, 2nd edn. Appendix III. Uncertainty Analysis, American Industrial Hygiene Association. Fairfax, VA: AIHA Press. ISBN 0 932627 86 2.
- Rogers A. (1984) Determination of mineral fibre in human lung tissue by light microscopy and transmission electron microscopy. *Ann Occup Hyg*; 28: 1–12.

doi:10.1093/annhyg/mei005

Reply

Received 14 February 2005

I fully accept from the literature that analysis of the lungs of asbestotics can reveal many millions of asbestos fibres per gram of dry tissue (mf gdt^{-1}). Whether the plaintiff in this case actually had asbestosis is the field of the medical experts: my interest was purely to determine if the lung residue data reported by the laboratory could have arisen from an exposure of ~ 40 fibres ml^{-1} years $^{-1}$.

The technical content of the laboratory's lung residue report consisted of a total of $8\frac{1}{2}$ lines, $1\frac{1}{2}$ lines of which described the tests carried out and the balance was effectively quoted in Howie (2005), and a table of total fibre content by fibre type: no data being given on fibre length. (An anonymized copy of this report has been lodged with the Editor.)

I would expect a proper scientific report on lung residues to provide information such as the means and standard deviations observed for fibre residue densities in asbestotics and to assess the plaintiff's observed lung fibre densities at the, say, 95 or 99% levels for each fibre type. The laboratory report provided no such information or assessment.

When I originally addressed the lung residue data, I carried out a Medline and manual literature search to see if I could find the type of data I consider the laboratory report should have provided. The most relevant paper I found was that of Churg and Vedal (1994) who reported that the geometric means (geometric standard deviations) for amosite

fibre residues in the lungs of shipyard workers and insulators with asbestosis and airway fibrosis were 10 mf gdt^{-1} (6.6) and 4.3 mf gdt^{-1} (12), respectively. That is, the plaintiff's observed amosite density of 0.61 mf gdt^{-1} was only 1.5 SD below the geometric mean of 10 mf gdt^{-1} for patients with asbestosis and 0.8 SD below the geometric mean of 4.3 mf gdt^{-1} for patients with airway fibrosis.

From the Churg and Vedal data, I concluded that the plaintiff's amosite residue clearly lay within the statistical range for both asbestosis and airway fibrosis. Given that the plaintiff's last likely occupational exposure to asbestos had ceased about 20 years prior to his death, I was mainly concerned that the laboratory report completely failed to address the subject of fibre clearance. Therefore, in Howie (2005) I concentrated on this issue.

Published data clearly indicate that asbestos fibres can be cleared from the lung, with chrysotile fibres being cleared more rapidly than amphibole fibres. For example, McDonald and McDonald (1996) commented that 'The fact remains that as chrysotile is of low persistence, concentrations found at death may be unrelated to what was inhaled over a lifetime and, indeed, may predominantly reflect only very recent exposure.'; Churg (1994) commented that 'The available data suggest that chrysotile is deposited in the parenchyma but is cleared extremely rapidly, with the vast bulk of fibres removed from human lungs within

weeks to months after inhalation; by comparison, amphibole clearance half-lives are of the order of years to decades.'; Churg and Vedal (1994) suggested a clearance half-life of about 20 years for amosite; and Du Toit (1991) concluded that for crocidolite 'allowance for the period which lapsed between date of last exposure and death revealed a lung clearance rate amounting to a half-life of about 6 years'. It should be noted that Du Toit (1991) and Churg and Vedal (1994) considered clearance only after the end of exposure. If clearance during exposure been considered, it is likely that shorter half-lives would have been calculated, particularly so in the case of the latter authors where the mean exposure duration had been 20 years (range 1–50 years).

In the calculations in Howie (2005), I used the half-time data from Bernstein (2004) as being a current and comprehensive publication in this field.

As most of the publications on lung residues do not state the periods of exposure to asbestos and between cessation of exposure and death, it is generally not possible to estimate the likely fibre burdens to which each subject had been exposed.

Irrespective of the numerical value taken for the half-time for each substance, I consider that any attempt to use lung residue data as a index of exposure must take into account the clearance which could have occurred both during exposure to the substance(s) of concern and between the end of exposure to such substance(s) and biopsy or death.

A further problem, not addressed in the laboratory report, is that a number of publications have stated that the number of fibres observed in the lung tends to reduce with the severity of the asbestosis. The plaintiff in this case was not diagnosed as having asbestosis until about 20 years after cessation of his last likely major occupational exposure to asbestos. I therefore presume that his asbestosis was of 'moderate' or less, severity.

I attempted to find references on Medline which would direct me to papers from which I could assess the likely fibre residue densities for patients with 10 or 20% disability due to asbestosis. I was unable to find any such data. The most explicit summary of the interpretation of lung residue I have found was that of Pairon and Dumortier (1999), who stated that 'A positive result does not mean existence of

asbestos-related disease. A negative result does not exclude previous significant asbestos exposure, clearly identified by an occupational questionnaire (particularly for exposure to chrysotile).'

From the literature I fully accept the observation that 'The lungs of asbestotics are likely to contain many millions of asbestos fibres per gram of dry tissue'; however, based on the available published information I am unsure of the validity of the negative corollary that 'unless the lungs contain millions of asbestos fibres per gram of dry tissue the patient is unlikely to have had asbestosis'. It will be appreciated that this is the negative corollary on which the laboratory conclusion seems to have been based.

On a minor point, I must respond to Dr Rogers' comment regarding my back-calculated chrysotile exposures over the periods 1943–1979 and 1957–1979. In Howie (2005), I explicitly stated that the very high assumed initial lung burdens for exposures ending in 1979 suggest that there was ongoing exposure between 1979 and 1990.

ROBIN HOWIE

12 Morningside Road,
Edinburgh EH10 4DB,
UK

E-mail: robin.howie@btconnect.com

REFERENCES

- Bernstein DM. (2004) Asbestos. Available on request from: <http://www.asbestoswatchdog.co.uk/>
- Churg A. (1994) Deposition and clearance of chrysotile asbestos. *Ann Occup Hyg*; 38: 625–33, 424–5.
- Churg A, Vedal S. (1994) Fiber burden and patterns of asbestos-related disease in workers with heavy mixed amosite and chrysotile exposure. *Am J Respir Crit Care Med*; 150: 663–9.
- Du Toit RS. (1991) An estimate of the rate at which crocidolite asbestos fibres are cleared from the lung. *Ann Occup Hyg*; 35: 433–8.
- Howie RM. (2005) Asbestos lung residue and asbestosis risk. *Ann Occup Hyg*; 49: 95–7.
- McDonald JC, McDonald AD. (1996) The epidemiology of mesothelioma in historical context [Review]. *Eur Respir J*; 9: 1932–41.
- Pairon JC, Dumortier P. (1999) Role of biometric analysis in the retrospective assessment of exposure to asbestos. *Rev Mal Respir*; 16: 1219–35.

doi:10.1093/annhyg/mei006