

Letter to the Editor

Asbestos Lung Residue and Asbestosis Risk

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I read with concern the attempt by Howie (2005) to prove in a courtroom that a negative may actually be a positive.

Our studies, along with those of many other international researchers, on asbestotics, lung cancer and mesothelioma cases indicate that lung clearance and lung residues are complicated issues. However, one consistent finding is that excess quantities of amphiboles remain in the lungs even decades after cessation of exposure; chrysotile is cleared relatively rapidly but even decades after exposure, a few long fibres and fibrils remain as markers of past exposure (Churg, 1991).

Mr Howie has failed to inform the reader that his model produces results that are contrary to the values found in the scientific literature. A search of the literature would reveal that there are in excess of 50 papers of case series reports on the asbestos residues found in the lungs of asbestotics. The values found in these papers indicate that tens of millions to hundreds of millions of asbestos fibres per gram of dry tissue are found in the lungs of asbestotics even many decades after exposure. The UK laboratory involved in this case indicated similar findings in reporting that asbestosis is generally associated with lung fibre levels in excess of 50 million fibres per gram of dry tissue, which by simple comparison is well in excess of the value reported in the lung tissue of the plaintiff.

The estimated cumulative airborne exposures presented in table 2 show the absurdity of the clearance model. For instance, the period 1943–1979 requires 2000 fibres ml^{-1} years⁻¹ of amosite [or 55.5 fibres ml^{-1} time-weighted average (TWA) for every hour of every working day for 36 years, a severe exposure by any industry or standard] to produce a final result of 0.61 million fibres of amosite per gram of lung tissue which is just slightly above the level found in the normal population. This cumulative airborne exposure would in our experience, in examining Australian amosite insulation manufacturing workers, result in a much higher lung burden of around many hundreds

of millions of fibres per gram of dry tissue even 25–35 years after cessation of exposure. The result presented previously from an asbestos cement manufacturing worker with 24 years of heavy (at times TWA ~ 5 – 10 fibres ml^{-1}) exposure to mixed asbestos types was a lung burden of 78 million fibres per gram of dry tissue (of which 14 million were chrysotile and tremolite was less than 0.2 million) some 20 years after cessation of exposure (Rogers, 1984).

When faced with such disparities between the measured results and the theoretically modelled exposures, it would be wise to take into account the recommendations of the American Industrial Hygiene Association committee on testing the validation of predictive exposure models.

One should fully expect, however, that actual exposure data and the distribution of that data should fit within the predicted distributions. If not, there was a serious error in the judgement that assigned the distribution of Type#2 predictor variable. A comparison of the distribution type of these data with the predicted distribution could provide a reality check on the assumption used in the Type#2 uncertainties. Of course, if the industrial hygienist has a good data set, he or she does not need to perform a composite analysis of the elements of uncertainty, as occurred above. (Mulhausen and Damiano, 1998)

One would hope that in viewing Mr Howie's modelled value and other evidence, the court exercised caution based on the prophetic comments by Doll and Peto (1985) during their examination of the data on the risk of fibrosis at low dose levels.

All these (lung) changes can be produced by other conditions, and the recognition of them, other than the history of exposure and the alterations in lung function, is, to a certain extent, subjective and subject to inter and intraobserver error. ... It is conceivable, therefore, that the recognition of these signs is influenced by knowledge of the history of exposure.

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'.

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Reply

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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