Editorial

Comparing biological effects of mineral fibres

Careful examination of the great majority of in vitro and in vivo experimental protocols to assess the biological effects of mineral fibres shows that comparison of the effects (in vitro cytotoxicity, fibrogenicity, and tumour yield) has traditionally been based on a gravimetric basis—that is, by comparing the effects produced by equal mass of tested minerals—for example, $50 \mu g/10^5$ cells; $20 \text{mg dose by intraperitoneal injection; } 20 \text{mg/m}^3$ by inhalation, etc.

The effects reported are often not consistent with epidemiological observations. In recent years the development of sophisticated techniques for tissue mineral analysis has led to some possible explanations for the inconsistencies between animal experimental data and epidemiological evidence, pointing to the different durability of mineral fibres and their different relative lifespan persistence between laboratory animals and man.

We consider that another explanation may also reside in the basis for reporting the observed effects—that is, equal mass v equal number of fibres.

Contrary to experimental data, human experience of exposure to different asbestos fibre types and manmade mineral fibres has shown an impressive difference in pathological potency, chrysotile being much less fibrogenic and carcinogenic, and having little mesothelioma producing potential (if at all) compared with amphiboles. Recently, Wagner et al wrote, "We believe therefore that chrysotile is the least harmful form of asbestos in every respect and that more emphasis should be laid on different biological effects of amphibole and serpentine asbestos fibre."7

Recently, manmade mineral fibres, especially rock/slag wool, have been positively correlated with excess incidence of lung cancer in production workers at low exposure levels, indeed at exposure levels ($\sim 1 \text{f/mL}$) where chrysotile has been shown to produce no excess lung cancer. There is some uncertainty about the estimates of exposure in the early production phase of rock/slag, and there may have been additional contributing factors such as the presence of polycyclic aromatic hydrocarbons or arsenic to the reported excess lung cancer mortality; all these sources may have contributed to the observed hazard, but as Doll said at the 1986 Copenhagen Symposium "... none has produced a quantitatively similar hazard elsewhere, unless exposure was both intense and prolonged."8

The problem is that despite the human data, some regulatory agencies such as the United States Environmental Protection Agency maintain that chrysotile has the same potential as the other asbestos fibre types, and that manmade mineral fibres have lower pathogenic potential based on animal experiments using equal mass doses.

Limited attempts to transform retrospectively gravimetric doses into fibre number-doses have indicated that if based on fibre number the pathogenicity would show that "fibre for fibre" chrysotile would be seen as less potent than the other asbestos fibre types, and possibly some other manmade mineral fibres as well. For instance, Davis et al have reported that rats submitted to dust clouds of chrysotile, crocidolite, and amosite showed more lung fibrosis and tumours after inhalation of chrysotile than with either amphiboles.3 But, they state: "... it was found that the chrysotile dust clouds used in this study contained many more fibres longer than 20 microns than either of the amphibole dust clouds."

In 1986, Peto, using animal data from Wagner,4 analysed the effects per fibre rather than per unit mass relevant to the comparison of asbestos and manmade mineral fibres in the induction of pulmonary tumours by inhalation. The results shown in the table indicate that a similar mass of chrysotile produced a greater incidence of tumour than manmade mineral fibres but that the risk per fibre may have been similar for chrysotile and glass wool, and possibly greater for rock wool. (All groups had been exposed to $10 \text{mg/m}^3$ but the fibre count was more than 10 times higher for chrysotile than for rock wool or glass wool.) While there is some scepticism in the scientific community about such retrospective analysis, it would be most desirable to have more relevant experimental data.

Also in 1986 Goldsmith indicated that "based on fibre or particle counts, manmade mineral fibres appear to be more potent than asbestos with regard to chronic pulmonary disease."5

Finally, in a more recently published in vitro study on the comparison of mass v number of fibres in the cytotoxic response of Chinese hamster lung V79 cells to erionite, crocidolite, and chrysotile, especially for fibres L > 8 microns, W < 0.25 microns, the UICC chrysotile fibres emerge as the least potent.6 Their data show that both samples of erionite required fewer fibres than UICC crocidolite, and that UICC chrysotile required a significantly higher number of fibres than the other three tested materials to produce similar cytotoxicity. For instance, the LD₅₀ values (in fibre number) for V79 cytotoxicity shows the follow-
Lung tumour distribution related to cumulative dosage

<table>
<thead>
<tr>
<th>Material (no of rats)</th>
<th>BAH* (a)</th>
<th>Adenoma† (b)</th>
<th>Adenoma‡ (c)</th>
<th>Adeno-carcinoma (d)</th>
<th>All neoplasms (b + c + d)</th>
<th>Average§ cumulative dose per rat at 12 months</th>
<th>Tumours per 100 fibres/cm³</th>
</tr>
</thead>
<tbody>
<tr>
<td>UICC chrysotile (48)</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>12</td>
<td>656</td>
<td>1.8</td>
</tr>
<tr>
<td>Glass microfibre (48)</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>232</td>
<td>0.4</td>
</tr>
<tr>
<td>Rock wool (48)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>39</td>
<td>0.1</td>
</tr>
<tr>
<td>Glass wool with resin (48)</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>55</td>
<td>0.8</td>
</tr>
<tr>
<td>Glass wool resin free (47)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>41</td>
<td>2.4</td>
</tr>
<tr>
<td>Controls (48)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>41</td>
<td>2.4</td>
</tr>
</tbody>
</table>

*Bronchoalveolar hyperplasia. †Benign. ‡Some features of malignancy. §Fibres/cm³.

Note: This table is reproduced from "Request from HSE for an opinion on the carcinogenic potential of glass fibre, glass wool, slag wool and rock wool. A resume of the relevant biological properties" which was prepared for the Committee on carcinogenicity.

It seems therefore that the stage is set for an increasing trend away from gravimetric data to fibre counts, and that if TLVs are to be expressed in fibre numbers per unit volume of air such TLVs should be based on scientific data which are reported on the basis of fibre number rather than on a mass basis.

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References

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doi: 10.1136/oem.46.10.681

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