carried out by SAS.\textsuperscript{11, 12} The programs and related information documenting the analytical process are available from JL. Please send a floppy diskette for storage.

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Chrysotile asbestos revisited

Sir,—It is difficult to find a material that has stimulated as much interest, and raised so much controversy, as asbestos. Mentions of health related effects in the literature date back almost to the beginning of this century. Yet results of recent studies are still on the agenda of international scientific meetings and published in current medical journals. Also, several if not most animal and in vitro studies on other fibrous materials include asbestos fibres as “positive controls” in their experimental design. The more recent human studies and updates on health related effects of occupational exposure to asbestos can now rely on longer periods of follow up, and on somewhat better defined exposure data to specific asbestos types.

A case in point is the recent update of the largest cohort of chrysotile asbestos workers ever undertaken.\textsuperscript{1} The preliminary results were the subject of a presentation in September 1992 at the 9th International Symposium on Epidemiology in Occupational Health, held in Cincinnati. The status of this unique cohort had been reviewed four times, the latest follow up was in May 1992 and included 2827 additional deaths, bringing the total to 7312. Cancer risks were re-evaluated. For six classes of exposure up to 300 mcpp f, the authors were unable to detect any excess lung cancers. Applying a conservative estimate for conversion of 1 mcpp to 3 f/ml, the exposure levels below which no excess lung cancers were detected would be 900 f/ml, or \(\sim 45\) f/ml for 20 years. While awaiting the publication of the full study later this year, this preliminary report should not be construed as an invitation to relax the exposure limit of 1 f/ml for chrysotile, as recommended by a group of experts convened by the World Health Organisation in 1989. It does indicate, however, that the recommended exposure limit was indeed a realistic and acceptable one.

I mentioned animal studies on man made fibrous materials, which sometimes include at least one asbestos fibre type as “positive control”; this is another area that needs to be revisited. For example, in a recent inhalation study on the allegedly minor health related effects of man made vitreous fibres (MMVF\textsubscript{s}), the authors include for comparison the results of concurrent studies on the allegedly severe effects from one refractory ceramic fibre sample, and from chrysotile asbestos.\textsuperscript{2} Close scrutiny of the experimental design, however, reveals that the results reported are from animals exposed six hours a day, five days a week, for 24 months to \(\sim 250\) f/ml for the MMVF\textsubscript{s}, \(\sim 180\) f/ml for refractory ceramic fibre, and 10 000 f/ml for chrysotile asbestos!

Another report\textsuperscript{3} indicates that after 24 months at a dose of 100 f/ml (\(\sim 0.9\) mg/m\(^3\)), of aramid (Kevlar) fibres in rats fibrosis had developed along with cystic keratinising squamous tumours. In view of the other inhalation experiments on MMVF\textsubscript{s} mentioned, an interesting experiment (which has never been carried out) would be to test the effects of a 24 month inhalation exposure to chrysotile at similar fibre number dosage (see table). With regard to inhalation studies on rock and slag fibres, the International Labour Office report indicates that “Available data are insufficient to draw conclusions on the relative potency of various types, because the true exposure (number of respirable fibres) was not characterized in most studies.”

It is worth going further into the details of the units of dosage used when reporting results. Coffin and colleagues have for many years warned against inappropriate comparison of the pathologic potential of different fibre preparations when only gravimetric units were used to report biological effects. For instance, an in vitro study published in 1988 on the comparison of mass of number of fibres in the cytotoxic response of lung cells from Chinese hamsters to erionite, crocidolite, and chrysotile, showed that on the basis of fibre numbers, erionite required fewer fibres than crocidolite, and that chrysotile required a \(>50\)-fold higher number of fibres to produce cytotoxic effects similar to those obtained with crocidolite. By comparison with erionite, the difference was \(>300\)-fold.\textsuperscript{4}

More recently, Coffin et al\textsuperscript{5} reported the results of an in vivo study on induction of mesothelioma after intrapleural and intratracheal injections in the rat. Erionite was 500 to 800 times more tumorigenic, and crocidolite was 30 to 60 times more so than chrysotile on the basis of the ratio of tumours to numbers of fibres. The fibre preparations used contained \(3.3 \times 10^6\) f/mg for erionite, \(8.6 \times 10^6\) f/mg for crocidolite and \(1090 \times 10^6\) f/mg for chrysotile.

It is worth mentioning that the summary of research recommendations of a National Institute of...
In vitro experiments with fibres

<table>
<thead>
<tr>
<th>Reference</th>
<th>Dosage used</th>
<th>Fibre number</th>
</tr>
</thead>
<tbody>
<tr>
<td>MMVF10 (2)</td>
<td>30 mg/m²</td>
<td>232 f/ml</td>
</tr>
<tr>
<td>MMVF11 (2)</td>
<td>30 mg/m²</td>
<td>246 f/ml</td>
</tr>
<tr>
<td>RCF (2)</td>
<td>30 mg/m²</td>
<td>187 f/ml</td>
</tr>
<tr>
<td>Aramid (3)</td>
<td>Not stated</td>
<td>100 f/ml</td>
</tr>
<tr>
<td>Chrysotile (2)</td>
<td>10 mg/m³</td>
<td>10 600 f/ml</td>
</tr>
<tr>
<td>Chrysotile</td>
<td>0.18 mg/m³</td>
<td>200 f/ml</td>
</tr>
</tbody>
</table>

The experiments carried out

Observations after 24 months' exposure: 5 h/day; 5 days/week

Wagner PGS = Wagner Pathology Grading Scale as follows: Cellular change 1 normal; 2 minimal: macrophage response; 3 mild: inflammation, bronchiolisation. Fibrosis 4 minimal: minimal fibrosis; 5 mild: linking fibrosis; 6 moderate: consolidation; 7 severe: marked fibrosis and consolidation; 8 severe: complete obstruction of most airways.

Environmental Health Sciences workshop on fibre toxicology indicates that “A major failing of past experimental studies has been the use of mass as the main dose parameter. Data are needed on fibre comparison by fibre number... Most studies using fibres in vitro have in the past expressed dosage on the basis of fibre mass as opposed to number of fibres per cell, which now appears to be a more valid means of comparison of fibre effects in relation to their potential to cause human disease.”

Without going further into the details of the dosages used and the results reported, these studies indicate that the time has come to revisit the case of chrysotile asbestos, and to compare its health related effects with those of other man made fibres, with fibre number at comparable dosage for comparisons. Some surprises might be revealed from such a comprehensive re-examination of the data. A major international re-evaluation of the case of chrysotile asbestos is in order.

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Pulmonary effects of exposure to fine fibreglass: irregular opacities and small airways obstruction

SIR,—On behalf of the North American Insulation Manufacturers Association (NAIMA), I am writing to express our concern over the publication of an article by Kilburn et al (1992;49:714–20) that examined a group of fibreglass workers at an appliance manufacturing plant in Cicero, Illinois. The study concluded that “commercial rotary spun fibreglass used for insulating appliances appears to produce human disease that is similar to asbestosis.”

The fact is that the conclusion of Kilburn et al is incorrect; fibreglass has not been found to produce human disease similar to asbestosis. Kilburn et al reached their conclusion despite several factors in their study that point to other culprits. For example, at least 40% of the workers with positive findings had known exposure to asbestos. In fact, the levels of airborne asbestos reported to Kilburn et al by the plant were higher than the levels of glass fibres, and yet were not even considered by Kilburn et al in reaching their conclusion. Further, about 80% of the study participants with positive findings were current or former smokers. Finally, the x ray film and pulmonary changes reported by Kilburn et al as abnormal are actually consistent with those that other scientists have reported to be expected in this age and type of population.

NAIMA would like to point out that the findings of Kilburn et al are not consistent with other morbidity studies regarding the health effects of exposure to fibreglass. Recently, Weill of Tulane University Medical School completed a study of over 1250 current workers at five US manufacturing plants. Weill concluded that “…after 10 years of these investigations, we have failed to demonstrate any adverse effect of MMMF [glass fibre] exposure on respiratory health. We have found workers in this industry to be generally healthy, without any detectable evidence of occupationally induced respiratory disease.”

NAIMA joins our European colleagues in support of the existing body of scientific research that finds no cause and effect relation between exposure to fibreglass and lung disease or cancer in humans. Based on the current weight of scientific evidence, NAIMA remains confident that fibreglass products are safe to manufacture and install when the simple instructions outlined on product packages are followed.

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Authors’ reply

SIR,—We appreciate the opportunity to respond to Mentzer. His concern is well founded and NAIMA should be worried about the adverse human health effects of commercial rotary spun fibreglass.1 Controverting the traditional fibre industry position he places the entire causal responsibility for abnormalities in the fibreglass workers on asbestos; not neglecting, of course, the contribution of cigarette smoking. He argues disingenuously that 40% of our workers with