In vitro experiments with fibres

<table>
<thead>
<tr>
<th>Reference</th>
<th>Dosage used</th>
<th>Fibre number</th>
<th>Observations after 24 months exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>MMVF10 (2)</td>
<td>30 mg/m³</td>
<td>232 f/ml</td>
<td>Wagner PGS 2.5 to 3</td>
</tr>
<tr>
<td>MMVF11 (2)</td>
<td>30 mg/m³</td>
<td>246 f/ml</td>
<td>Wagner PGS 2.5 to 3</td>
</tr>
<tr>
<td>RCF (2)</td>
<td>30 mg/m³</td>
<td>187 f/ml</td>
<td>Wagner PGS 4</td>
</tr>
<tr>
<td>Aramid (3)</td>
<td>Not stated</td>
<td>100 f/ml</td>
<td>Lung tumours 16 (13%)</td>
</tr>
<tr>
<td>Chrysotile (2)</td>
<td>10 mg/m³</td>
<td>10 600 f/ml</td>
<td>Mesothelioma 2 (1.6%)</td>
</tr>
</tbody>
</table>

The experiments that was never carried out

0.18 mg/m³ 200 f/ml

Wagner PGS = Wagner Pathology Grading Scale as follows: Cellular changes 1 normal; 2 minimal: macrophage response; 3 mild: inflammation, bronchiolisation. Fibrosis 4 minimal; 5 mild: linking fibrosis; 6 moderate: connective; 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.

In vitro

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2. Hesterberg TW, Miller WC, McConnell EE, Chevalier J, Hadley JC, Bernstein DM, Thévenaz P, Anderson R. Chronic inhalation toxicity of size-separated glass fibers in...  5 f/day; 5 days/week

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 Kilburn et al reported 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, Weil of Tulane University Medical School completed a study of over 1250 current workers at five US manufacturing plants; Weil 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.

KENETH MENTZER
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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  Controversing 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...
positive findings had "possible" asbestos exposure. Obviously the message should be turned around, 60% of workers with positive chest x-ray films were exposed only to fibreglass. They had no asbestos exposure.

The issue of airborne asbestos fibres seems to be a convenient invention to divert attention from fibreglass. Rather the key health problem is the finding that 49% to 83% of fibres in the fibreglass being used at Cicero were respirable but below detectability by phase contrast microscopy.

That 80% of workers with irregular opacities on chest x-ray films had smoked cigarettes is analogous to the usual industrial or construction cohort exposed to asbestos. As cigarette smoking by itself does not cause irregular opacities, it is plausible to suggest that it is synergistic with fibreglass just as it is for asbestos, and that it enhances the fibre induced irregular opacities.

There are few comparative studies of chest x-ray films and pulmonary function in industrial populations who have not been exposed to any asbestos but to contend that they are characterised by this degree of function impairment is ludicrous. We recommended before that the subjects of studies referred to but not referenced by Mentzer be investi-gated by independent scientists for exposure to asbestos or to man made fibres.

We examined and reported irregular opacities and small airway obstruction in one cohort of five fibreglass manufacturing plants studied by Weill et al. The atmosphere was continuously contaminated with asbestos during dismantling, repair, and construction of fibreglass production lines. Thus we find the statement attributed to Weill, of no abnormalities, incomprehensible.

The final series of Mentzer's statements are reminiscent of the position of the asbestos industry from the 1930s to the 1960s despite the compelling evidence of asbestosis, cancer, and mesothelioma provided by Merewether, Dreessen et al, Doll, Wagner et al, Newhouse and Thompson, and Selikoff et al. Society has learned in this century that such views by industry of human risk reflect concerns for corporate comfort and survival, not the safety and health of human beings. Must we repeat the whole sad asbestos litany for man made mineral fibres?

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