Probability of $\beta_2$ microglobulinuria as related to urinary cadmium concentration.

carried out by SAS.$^{11, 12}$ 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 centu-
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%) Mesothelioma 2 (1.6%)</td>
</tr>
<tr>
<td>Chrysotile (2)</td>
<td>10 mg/m³</td>
<td>10 600 f/ml</td>
<td>Fibrosis and cystic keratinising squamous tumours</td>
</tr>
<tr>
<td>Chrysotile</td>
<td>0.1-18 mg/m³</td>
<td>200 f/ml</td>
<td>Wagner PGS 4 Lung tumours 13 (18%) Mesothelioma 1 (1.4%)</td>
</tr>
</tbody>
</table>

The experiments that were carried out

The experiment that was never carried out

Wagner PGS = Wagner Pathology Grading Scale as follows: Cellular change 1 normal; 2 minimal: macrophage response; 3 mild: inflammation, bronchiolisation. Fibrosis 4 minimal; 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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2 Hesterberg TW, Miller WC, McConnell EE, Chevalier J, Hadley JC, Bernstein DM, Thevenaz P, Anderson R. Chronic inhalation toxicity of size-separated glass fibers in


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.

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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