CORRESPONDENCE

Does occupational exposure to dust prevent colorectal cancer?

Editor—A recent report found that increased rates of stomach cancer coincide with decreased rates of colorectal cancer in populations exposed to dust.1 The report’s author first noticed this in a cohort of Ontario miners, one of the populations considered in the report.

The table compares mortalities in that cohort with those in the general male population of Ontario. The mortalities for several diseases (silicobrusisosis, stomach, lung cancer, and pneumonia) are increased; mortalities for some other diseases (chronic lymphatic and myelogenous leukaemia, nasal and laryngeal cancer) are greater than in the general population but the increases are not large enough to be considered real and the mortalities for many of the remaining diseases are much less than those in the comparison population. Because the diseases in the first group are associated with exposures in the mines, should one then suggest that exposure to dust protects workers from those diseases such as pancreatic cancer, oesophageal cancer, bladder cancer, heart disease, and cirrhosis of the liver for which mortalities are lower than in the comparison population?

As Finkelstein remarks, because of the healthy worker effect, comparisons of mortalities in working populations with those in the general population give biased results. And as noted in another report,2 additional personal identifying information increases the mortalities found in occupational groups. Perhaps these two factors explain much of the deficit of mortality from colorectal cancer and other diseases found in populations exposed to dust.

Author’s reply—Kusiak and colleagues have performed some brilliant work in collecting and analysing data about the mortality of Ontario miners and I am pleased to receive his remarks. The intent of my paper was to comment on the tendency of epidemiologists to focus on “positive” associations and to ignore “negative” ones. My comments on causation were largely “tongue in cheek”. Nevertheless, as Kusiak points out, there were other disease causes and tumour sites in the Ontario mining population with lower than expected mortality. It is not implausible that some of these might be influenced by dust exposure. The oesophagus lies on the ingestion pathway, and the bladder on the route of excretion. The challenge of course is to identify causality in an environment of confounding and multiple comparisons. Consistency of any of these findings across several cohorts might lead one to explore what factors are confounding the association or to consider the possibility of causality.

MURRAY FINKELSTEIN
Ontario Ministry of Labour, Health and Safety Studies Unit, 400 University Avenue, 7th Floor, Toronto, Ontario M7A IT7, Canada

Back pain and male parenthood

Editor—The article “Backpain and parenthood” written by Finkelstein raises new and interesting issues concerning male parenthood as a risk factor for back pain.1 It was mentioned that this finding had never been reported before. We have reported similar results of an association between self-reported work impairment due to back pain and caring for children at a population of 269 male aircraft assembly workers.2 We had treated this factor as a potential confounder but had not specifically reported

---

2 Kusiak RA, Ritchie AC, Muller J, Springer J. Mortality from lung cancer in Ontario ura-


---

10.1136/oem.52.10.699-b
Preliminary experimental findings using intraperitoneal assays to determine carcinogenic potential of man made mineral fibres: relevance to recent proposals for classification testing

Editor—For many years the potential of different mineral fibre types to induce mesothelioma has been studied with intraperitoneal (IP) injection as a route of administration to rats. Many studies have been conducted in which man made mineral fibres have been reported to cause tumours in the peritoneal cavity, in particular, mesotheliomas. As a result of the belief that inhalation may not provide an adequately sensitive assay for the carcinogenicity of man made fibres, a decision has been made in Germany for the formal classification of fibres based on the results of IP tests. The proposed classification of fibres involves two stages: the calculation of a “carcinogenicity index” (KI) based on chemical composition of the fibres combined with (if available) biological data from IP injection. Peritoneal cavity of a rat is about 10 mL in 12- to 16-week-old animals, and would be lower in younger animals. Given that density of these fibre types is about 2.3 g cm⁻³, the minimum volume which 5300 mg could occupy is 2.3 cm³ or one quarter of the available free space in the peritoneal cavity. So even if the animals could be injected with this mass of material and survive, a significant fraction of the peritoneal free space would be filled with fibres. Studies to determine the maximum mass that a rat could be injected with and survive have not been conducted, but I am certain that animals would not survive injection of the above masses. Even if we used masses as reported in our studies (<200 mg), we have seen significantly reduced life spans in animals that did not develop tumours, suggesting that the maximum tolerated dose for IP injection has been exceeded. The proposed regimen for testing fibres with IP injection would therefore not seem to be practical.

Our studies have also shown information on the sensitivity of the proposed IP test to fibre length. Table 2 shows for both studies, the doses given in terms of mass and fibre number, and the incidences of mesothelioma occurring in the animals. Results from studies on similar materials by Roller and Pott are also given for comparison and were obtained by personal communication (1994).

For the stonewool fibre types, the three groups injected with longer fibres showed a similar dose response of dose, suggesting that the maximum incidence had been achieved in all of these experiments. When shorter fibres were injected, even at a relatively high dose, the incidence of

---

Table 1 Injection data from animals injected with MMVF11 and D6 fibres

<table>
<thead>
<tr>
<th>Material</th>
<th>Study I</th>
<th>Study II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MMVF11</td>
<td>D6</td>
</tr>
<tr>
<td>Total mass of fibre injected (mg)</td>
<td>52</td>
<td>36</td>
</tr>
<tr>
<td>Total fibres injected (×10⁶)</td>
<td>11-4</td>
<td>12-0</td>
</tr>
<tr>
<td>WHO fibres injected (×10⁶)</td>
<td>7-5</td>
<td>8-8</td>
</tr>
<tr>
<td>Median length (µm)</td>
<td>6-86</td>
<td>7-84</td>
</tr>
<tr>
<td>Median diameter (µm)</td>
<td>0-97</td>
<td>0-80</td>
</tr>
<tr>
<td>Geometric mean diameter (µm)</td>
<td>0-89</td>
<td>0-89</td>
</tr>
</tbody>
</table>