Do occupational exposures in nuclear refineries contribute to mortality from brain cancer?

Editor—Mortality from brain cancer in workers has been found more often than expected in several nuclear processing plants. 1 Exposure to chemicals and radiation were examined, one by one, but each alone was not the causative agent of the disease. 2,3 The possibility of an association between brain cancer and exposure to electromagnetic fields has been examined in several recent studies, 2,4 but definitive conclusions about a causal relation have not yet been reached.

Brain cancer was noticed to occur more frequently than expected in nuclear workers in Ontario (observed 5, expected 1-12, standardised mortality ratio (SMR) 446, 95% confidence interval (95% CI): 147 to 1054). This finding was unexpected. These 1190 uranium millers were included in a larger study of underground uranium miners to find out whether exposure to uranium dust in the mills could have increased mortality from kidney disease. The SMRs for all causes, all cancer, and most other specific cancer sites were below 100 for the uranium mill workers. No brain cancers were found (expected = 1-51) in men who worked in other mills in Ontario where ores such as gold, nickel, or zinc were processed. Nor was an excess of brain cancer found in Ontario uranium miners (observed 14, expected 23-72, SMR 59). An excess of mortality from brain cancer has also been found in men who worked in tank houses where nickel is electrolytically refined (observed 8, expected 2-19). 6

Two of the deaths from brain cancer in the Ontario uranium mill workers occurred before 1950, and another in a man over 55. Two of the cancers were glioblastoma multiforme, one a glioma, one a medulloblastoma, a rare tumour occurring in children and young adults, and the other a brain tumour whose histology was unclassified. The work histories available to us were not detailed enough to identify characteristics in which the workers who died of brain cancer differed from the other mill workers. The group of men who died of brain cancer did not work elsewhere in the mining industry in Ontario. One worked underground in a uranium mine and another was a painter for 28 months and gold mill worker for 141 months. Employments in the uranium mills ranged from four months to 74 months for these five men. Four of the five deaths from brain cancer occurred less than 10 years after the men last worked in the uranium mines. The cause of death for a man who died of brain cancer was not recorded on the death certificate for two men and ranged from two months to 6-5 years before death for the other three men.

Exposure records for external y radiation have been included in the National Dose Registry since the routine monitoring of the radiation exposures of uranium mine and mill workers has begun in 1981. We obtained exposure records from the National Dose Registry for 92% of the 1190 Ontario uranium mill workers. The average lifetime exposure to radon from all occupational sources was 20 working level months and the average cumulative external y radiation exposure between 1981 and 1995 was 17 mSv. All of the men who died of brain cancer began working in the Ontario uranium mills in the late 1950s before their internal y radiation levels were routinely measured and recorded in the National Dose Registry. Exposure levels for all workers exposed to radiation in Canada have decreased since the 1950s but deciding whether work in the uranium mills is the most probably cause of brain cancer is difficult. In any case, the uranium millers' low levels of exposure to external y radiation reflect the low levels of radioactive elements in the uranium ores mined in Ontario. Exposure to radon has not been associated with brain cancer, 6 but several studies have shown an association between brain cancer and exposure to y radiation. 8 However, no association between brain cancer and exposure to radiation has yet been found in the survivors of the atomic bombings of Nagasaki and Hiroshima.
Lung cancer in asbestos cement workers in Denmark

Editor—This paper is a tribute to Edith Raffin and Elsebeth Lynge who have been involved in all three analyses of mortality and cancer morbidity in this Danish asbestos cement worker population, and to Johannes Clemmesen, father of their Cancer Register. The history of studies of asbestos cement workers tends to follow a pattern. When the health of the population studied is found to be pluperfect or its excess mortality not significant, there has been a tendency to leave well alone. (The astute epidemiologist, after conducting a preliminary analysis that seems to show that asbestos exposure was good for you, would decline to proceed further until he had verified the integrity of the population. But that is another story).

The attraction of studying asbestos cement workers (and for that matter asbestos textile workers) was the possibility of being able to evaluate the toxicity of chrysotile. In the event, when excess cancer mortality was found, it would be recalled that for a period there may have been exposure to amphibole.

This population of Danish asbestos cement workers overall, had the potential for mixed chrysotile and amphibole exposure, but it does include a subset of workers employed exclusively before the introduction of amphibole. Could the authors inform us whether analysis of this valuable group casts any light on the hazards of exposure purely to chrysotile asbestos?

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BOOK REVIEWS

Book review editor: R L Maynard

Incidence of lung cancer in Danish asbestos cement workers employed during periods when chrysotile only was used

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of cases of lung cancer</th>
<th>SIR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Employed 1929-44:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>12</td>
<td>6.48</td>
</tr>
<tr>
<td>Women</td>
<td>0</td>
<td>1.0 (0.96-3.2)</td>
</tr>
<tr>
<td>Employed 1980-4:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>0</td>
<td>0.24</td>
</tr>
<tr>
<td>Women</td>
<td>0</td>
<td>0.05</td>
</tr>
</tbody>
</table>

If, for example, the period 1941–4. During the years 1945–79 chrysotile primary (±88% of all asbestos used); but for all years also a small amount of amosite (±1%), and for 1950 to 1969 some crocidolite (±1%) were used. During the years 1980–4 again chrysotile only was used.

As reported, from 1943 to 1990 a total of 223 lung cancer cases were diagnosed among the male cohort members (standardised incidence ratio (SIR) 1.7; 95% confidence interval (95% CI) 1.5–2.0).

We have now also tabulated the lung cancer incidence for people employed only during the years where chrysotile only was used at the factory. This involves 163 people who started employment between 1928 and 1940 and ended employment before 1945; and 262 people who started employment between 1980 and 1984. We have taken advantage also of the fact that specific job titles were recorded for the early employment period.

There were a total of 12 lung cancer cases; all among men employed 1928–44. This gave an increased SIR of borderline significance (SIR 1.9; 95% CI 0.96–3.2). The excess number of cases came from workers employed in the asbestos cement production and in the maintenance.

The numbers are thus small, but the data clearly indicate that the excess lung cancer risk found for the total cohort was found also for the subgroup exposed exclusively to chrysotile.

BIBLIOGRAPHY


