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

Editor—Mortality from brain cancer has been found more often than expected in several nuclear processing plants.1 Exposure to chemicals and radiation was 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,4 but definite conclusions about a causal relation have not yet been reached.

Brain cancer was noticed to occur more frequently than expected among uranium 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 miners 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 cancers, and most other specific cancer sites were below 100 for the uranium mill workers. No brain cancer deaths 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).5

Two of the deaths from brain cancer in the Ontario uranium mill workers occurred in men under the age of 35, and one occurred in men over 55. Two of the cancers were glioblastoma multiforme, one a gloma, one a medulloblastoma, a rare tumour occurring in children and young adults, and two other brain tumours were unspecified. 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 whole cohort. Thereafter, the reason why the tumour 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 tumour in the 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 γ radiation have been included in the National Dose Registry4 since the routine monitoring of the radiation exposures of uranium mine and mill workers was 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 γ 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 external γ 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 during work periods for uranium mill workers was difficult. In any case, the uranium millers' low levels of exposure to external γ 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, however, several studies have shown an association between brain cancer and exposure to γ radiation.6 However, no association between brain cancer and exposure to radon has yet been found in the survivors of the atomic bombings of Nagasaki and Hiroshima. Furthermore, electromagnetic fields and extremely low frequency fields in two Ontario mines were surveyed but no measurements of the intensity of those fields in any of the uranium mills were made. The survey showed that electromagnetic magnetic fields around some heavy duty electrical machinery used in the mining industry was between 1 and 10 μT and levels of electromagnetic fields in the electrolytic nickel refineries were very low. In recent United States study found a significant excess of brain cancer in workers in the highest exposure category. Exposure to magnetic fields two to 10 years after death was most strongly associated with mortality from brain cancer, the relative risk increasing by 1-94/T-year.7 This suggests that magnetic fields may act as a tumour promoter.7,8 A Canada-France collaborative study found a non-significant increase in risk of brain cancer in workers whose cumulative exposure to magnetic fields was above 15-7 μT-years (odds ratio 1-95, 95% CI 0-76 to 5-0).9

The question of whether or not the increased mortality from brain cancer in workers involved in nuclear processing plants is due to occupational exposures remains unanswered. It is unlikely that exposure to radon is directly related to the development of brain cancer as no excess has been seen in uranium miners and the exposures in the uranium mills were much lower than in the uranium mines. Neither do the exposures to γ radiation seem to be large enough to produce an excess of brain cancer.10 The exposure to electromagnetic fields, however, may be larger than those found in other studies, in which excesses of brain cancer have been found. The case-control method should be used to investigate exposures that might be associated with brain tumours in nuclear processing plants and electrotycl metal refineries. Electromagnetic fields, radon progeny, γ radiation, and exposures to chemicals, including solvents, can all be considered and the populations would need to be combined to get one large enough for a valid statistical analysis.

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

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of cases of lung cancer</th>
<th>Obs</th>
<th>Exp</th>
<th>SIR (95% CI)</th>
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<tr>
<td>Employed 1929-44:</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>12</td>
<td>6.48</td>
<td>1.9 (0.96–3.2)</td>
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<tr>
<td>Women</td>
<td>8</td>
<td>4.04</td>
<td>2.0 (0.9–3.9)</td>
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</tr>
<tr>
<td>Men</td>
<td>2</td>
<td>1.55</td>
<td>1.3 (0.47)</td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>2</td>
<td>0.98</td>
<td>3.0 (0.3–10.9)</td>
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<tr>
<td>Men</td>
<td>223</td>
<td>0.24</td>
<td>0.24</td>
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<tr>
<td>Women</td>
<td>0.05</td>
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BOOK REVIEW

Book review editor: R L Maynard


Smog Alert: Managing Urban Air Quality is a useful introductory book on urban air pollution. The style is apocalyptic and the author delights in providing, especially in the early chapters, details of appalling population growth and worsening air quality in the rapidly expanding cities of the countries in transition from an agricultural to an industrial economy. The author has provided an extensive footnote references, which I like, to the “grey literature” but almost no references to the original scientific literature. This is by contrast with his book Atmospheric Pollution: A Global Problem which provides detailed referencing. The author has included a wide range of government reports in his footnotes: again, I like this, but use of the secondary literature alone makes it difficult to check statements made in the text.

Does the book provide a balanced account? Looking closely at those areas with which I am familiar, I am afraid that it does not. For example, the section dealing with asthma and air pollution leaves the impression that the worldwide, rising tide of asthma is caused by air pollution. A secondary source not quoted by the author is the Department of Health Report on Asthma and Outdoor Air Pollution. This report made clear, by a detailed examination of the primary literature, that links between air pollution and the prevalence of asthma were far from established. This point has also been made in a recent report of an International Programme of Chemical Safety Workshop on Environmental Chemicals and Respiratory Hypersensitivity. The prevalence of asthma is increasing in the United Kingdom and yet the trend in pollution levels in the United Kingdom urban areas has not been dramatically upward during the past 10 or so years. Other sections of the book also