Authors' reply.—Hathaway draws attention to an apparent inconsistency in the classification of exposures in our study. This arose because, as explained in the paper, the method for assigning exposures in the nested case-control study differed from that in the cohort analysis. In the cohort analysis, factory-specific job exposure matrices were derived in the same way as for the nested case-control study, but because the cohort was divided into different occupational groups, the nested case-control study only included those who were definitively exposed to acid mists. In the nested case-control study exposures were classified as zero, low, high, or uncertain, taking into account not only the jobs in which men had worked, but also the periods during which those workers were employed. We questioned staff at the factories about these jobs in more detail than would have been practical for the full cohort. Among other things this caused one deceased cohort worker, originally classified as unexposed, to be assigned to the uncertain category; and another, whose exposure had been uncertain, to be classed as having low exposure.

We must stress that all exposures in the nested case-control study were assigned blind to the case or control status of the subject. Because they were assessed from more detailed information than could be incorporated into the cohort analysis, they should be more reliable.

Hathaway also questions our inclusion of cancers of the lip and mouth with those of the larynx and nasopharynx. This decision was made after the analysis was completed and out, and was based on an assumption that if acids cause cancer of the upper aerodigestive tract, the most likely mechanism is by an irritant effect. High exposure to acid mists is known to cause dental erosion, and it seemed natural to include cancers of the lip and mouth. Hathaway points out that lip cancer is caused by sun exposure and pipe smoking. There is no reason to think that within the workforce studied, men exposed to acid mists would have had higher exposure to sunlight or pipe smoking.

The statistical power of our study was limited, but we stand by our conclusion. The findings are consistent with those of other studies indicating a hazard of aerodigestive cancer from acid mists, but they suggest that any risk from exposure to sulphuric acid and hydrochloric acid below 1 mg/m$^3$ is small.

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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. Exposure to chemicals and radiation were examined, one by one, but each alone was not the causative agent of the disease. 2, 4 The possibility of an association between brain cancer and exposure to electromagnetic fields has been examined in several recent studies, but definite conclusions about a causal relation have not yet been reached.

Brain cancer was noticed to occur more frequently than expected among uranium mill 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 1196 observations included cases only 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 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 the period under study, and the other three occurred in men over 55. Two of the cancers were glioblastoma multiforme, one a gliona, one a medulloblastoma, a rare tumour occurring in children and young adults, and the other two were other brain tumours.

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. For the workers 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. Employment 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 was diagnosed as 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 levels for external $\gamma$ radiation have been included in the National Dose Registry since the routine monitoring of the radiation exposures of uranium mine and mill workers began 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 $\gamma$ 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 exposure 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 or not a group of uranium millers is difficult. In any case, the uranium millers' low levels of exposure to external $\gamma$ 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, but several studies have shown an association between brain cancer and exposure to $\gamma$ radiation. 7 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.

Electromagnetic fields, especially electric 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 data showed that mill workers were exposed to magnetic fields around some heavy duty electrical machinery used in the mining industry was between 1 and 10 $\mu$T and levels of electromagnetic fields in the electrolytic nickel refineries were in general lower. In the 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 before death was most strongly associated with mortality from brain cancer, the relative risk increasing by 1.94/T-year. 8 This suggests that magnetic fields may act as a tumour promoter. 9 A Canada-France collaborative study found a non-significant increased risk of brain cancer in workers whose cumulative exposure to magnetic fields was above 15.7 $\mu$T-years (odds ratio 1.95, 95% CI 0.76 to 5.0). 11

The question of whether or not the increased risk of brain cancer among workers 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 found in uranium miners and the exposures in the uranium mills were much higher than in the uranium mines. Neither do the exposures to $\gamma$ radiation seem to be large enough to produce an excess of brain cancer. However, future work in the exposure of exposure levels, 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 electromagnetic metal refineries. Electromagnetic fields, radon progeny, $\gamma$ radiation, and exposure to chemicals, including solvents, can be considered in these studies. The populations would need to be combined to get one large enough for a valid statistical analysis.

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4 Carpenter AV, Flanders WD, Frome EL, Cole...
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 Clemmensen, 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 where chrysotile only was used

<table>
<thead>
<tr>
<th>Number of cases of lung cancer</th>
<th>Group</th>
<th>Obs</th>
<th>Exp</th>
<th>SIR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Employed 1929-44:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Workers</td>
<td>Men</td>
<td>12</td>
<td>6.48</td>
<td>1.9 (0.96-3.2)</td>
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<tr>
<td>Asbestos cement</td>
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<td>8</td>
<td>4.04</td>
<td>2.0 (0.9-3.9)</td>
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<tr>
<td>Cement only</td>
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<td>2</td>
<td>1.55</td>
<td>1.3 (0.47-4.7)</td>
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<tr>
<td>Maintenance</td>
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<td>0</td>
<td>0.06</td>
<td></td>
</tr>
<tr>
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<td>2</td>
<td>0.23</td>
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</tr>
<tr>
<td>Women</td>
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<td>0</td>
<td>0.18</td>
<td></td>
</tr>
<tr>
<td>Employed 1980-4:</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td>0</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td>0</td>
<td>0.05</td>
<td></td>
</tr>
</tbody>
</table>


Authors’ reply—The cohort of asbestos cement workers from Denmark includes 7887 men and 576 women employed between 1928 and 1984. During the years 1928-40 chrysotile only was used in asbestos cement production. No asbestos was used during the war years 1941-4. During the years 1945-79 chrysotile primary (≈ 88% of all asbestos used); but for all years also a small amount of amosite (≈ 11%), 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 0.9-3.2). 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.

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Smoog 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 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 the 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 Hypersensitisation. 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
Do occupational exposures in nuclear refineries contribute to mortality from brain cancer?
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