Update of the Texaco mortality study 1947–93: part II

EDITORS—It is with a sense of “deja-vo” that I read the paper by Divine et al. on the update of the Texaco mortality study. I was particularly struck by their conclusions about lung cancer among the maintenance trades. Although large numbers of mesotheliomas were found among these workers, the authors concluded that there was no increased risk of lung cancer because the SMRs were <100.

This conclusion is similar to that of Tsai et al. who, despite the findings of excess mortality from mesothelioma and increased lung cancer mortality among maintenance workers, in comparison with operators at another Texas refinery, concluded that asbestos exposures were “not sufficient to produce lung cancer”. It is also similar to that of Raabe et al. who stated that, although they had found an increase in lung cancer mortality among maintenance craft workers, the “temporal patterns of these SMRs and findings from other studies of similar cohorts provided evidence that the excesses were most likely to be unrelated to work in maintenance craft jobs”.

Workers in the petroleum refinery industry, when grouped together, have had a lower risk of lung cancer than members of the general population. This has been found once again in the paper by Divine et al. Given this phenomenon, it is appropriate to make internal comparisons—that is, to compare the lung cancer experience of maintenance workers exposed to asbestos with that of operators who, despite the findings of excess mortality among these workers, the authors concluded that the excesses were most likely to be unrelated to work in maintenance craft jobs.

I have performed some calculations with the data in Table 1 of Divine et al., and I find that in their study there is a significant trend in risk of lung cancer with duration of employment in the maintenance trades. The table below shows the data. I have subtracted the experience of the maintenance employees from that of the total cohort to produce the SMR for unexposed workers. The SMR for lung cancer increases with the duration of time employed in the maintenance trades. I used equation number (3.12) in the monograph of Breslow and Day to compute a test for trend in these SMRs. I used the integers 1 to 4 to rank the exposure levels. The value $x^2$ in the trend test was 18.1 on 1 degree of freedom (p<0.001). I thus conclude that the study of Divine et al. contributes yet another piece of evidence showing that workers exposed to asbestos in the refinery and petrochemical sector are at increased risk of lung cancer.

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Table 1 Lung cancer among workers in the Texaco mortality study

<table>
<thead>
<tr>
<th>Worker group</th>
<th>Observed</th>
<th>Expected</th>
<th>SMR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unexposed</td>
<td>152</td>
<td>274</td>
<td>56</td>
</tr>
<tr>
<td>Maintenance, 0–4 y</td>
<td>219</td>
<td>322</td>
<td>68</td>
</tr>
<tr>
<td>Maintenance, 5–12 y</td>
<td>91</td>
<td>121</td>
<td>75</td>
</tr>
<tr>
<td>Maintenance, ≥20 y</td>
<td>75</td>
<td>84</td>
<td>89</td>
</tr>
</tbody>
</table>

Table 1 Lung cancer mortality by duration of employment in specific maintenance crafts

<table>
<thead>
<tr>
<th>Specific craft</th>
<th>0–4 y</th>
<th>5–19 y</th>
<th>≥20 y</th>
<th>Trend test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obs (SMR 95% CI)</td>
<td>Obs (SMR 95% CI)</td>
<td>Obs (SMR 95% CI)</td>
<td>$x^2$</td>
</tr>
<tr>
<td>Insulator</td>
<td>48 (90–162)</td>
<td>31 (77–149)</td>
<td>10 (133–245)</td>
<td>7 (128–264)</td>
</tr>
<tr>
<td>Painter</td>
<td>30 (76–108)</td>
<td>19 (69–140)</td>
<td>8 (100–193)</td>
<td>3 (87–194)</td>
</tr>
<tr>
<td>Structural steel, pipe fitter, machinist</td>
<td>202 (78–269)</td>
<td>105 (80–165)</td>
<td>46 (71–99)</td>
<td>51 (78–102)</td>
</tr>
<tr>
<td>Electrician, instrument</td>
<td>35 (78–108)</td>
<td>11 (89–160)</td>
<td>9 (58–151)</td>
<td>5 (36–128)</td>
</tr>
<tr>
<td>Boilermaker, welder, lead binder</td>
<td>52 (65–85)</td>
<td>23 (60–98)</td>
<td>19 (83–150)</td>
<td>10 (53–98)</td>
</tr>
<tr>
<td>Equipment operator</td>
<td>59 (73–94)</td>
<td>42 (76–103)</td>
<td>9 (50–98)</td>
<td>8 (102–202)</td>
</tr>
<tr>
<td>Cleaner</td>
<td>93 (68–154)</td>
<td>84 (87–130)</td>
<td>5 (58–136)</td>
<td>147 (147–246)</td>
</tr>
<tr>
<td>Pipefitter, boilermaker</td>
<td>161 (81–268)</td>
<td>97 (78–263)</td>
<td>33 (81–155)</td>
<td>31 (90–118)</td>
</tr>
<tr>
<td>All maintenance</td>
<td>419 (74–871)</td>
<td>215 (76–867)</td>
<td>99 (64–527)</td>
<td>107 (82–97)</td>
</tr>
</tbody>
</table>

I thus conclude that the excesses were most likely to be unrelated to work in maintenance craft jobs.
Childhood leukaemia, population mixing, and paternal occupation

The possible infective origin of childhood leukaemia has been the subject of much recent research. Fear and Roman reply—In our paper published in an earlier issue of Occupational and Environmental Medicine, we examined the role of father’s occupational social contact on the development of childhood leukaemia from data covering the whole of England and Wales.1 We found no evidence for an association between death during childhood leukaemia and father’s occupational social contact. Kinlen states that because we examined data for the whole of England and Wales rather than for specific “population-mixing situations associated with an excess of childhood leukaemia” it is unsurprising that we found no association. However, given the worrying nature of the suggestion that parents may transmit a workplace acquired leukemogenic agent to their offspring,2 we think that our result, showing that, in general, children of men who have high levels of occupational social contact are not at an increased risk of leukaemia, is important. Further, the first study published on this topic, based within an area identified as having an excess of childhood leukaemia, found no evidence that parental occupational social contact—either with adults or with children— influenced the risk of leukaemia in their offspring.3

The suggestion that infections may be involved in the aetiology of childhood leukaemia, particularly acute lymphoblastic leukaemia (ALL), is not new. Kinlen’s population mixing hypotheses, and those of Greaves,4 are currently the subject of much research. To date, however, no specific agents or mechanisms have been identified.4

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Childhood leukaemia, population mixing, and paternal occupation

The possible infective origin of childhood leukaemia has been the subject of much recent research. Fear et al have taken up my suggestion that my necessarily limited examination of childhood leukaemia relative to levels of paternal occupational contact in the general population1 has been extended and (as in my study) found no evidence of a general increase in risk when fathers have jobs involving contact with many people. However, in reporting my hypothesis they did not bring out that the principal question considered in my study did not concern the general population, but rather whether there was a relation with paternal occupational contacts “in population-mixing situations associated with an excess of childhood leukaemia”.1 I investigated this question1 with data from previous studies that were carried out to test the view that it is a rare response to some unidentified infection, and that situations of unusual urban-rural population mixing would favour excesses by promoting contacts between infected and susceptible people. This occurs when urban and rural areas are separated by a grid of rural communities that are likely to contain a higher proportion of susceptible people—such as in rural new towns or the North Sea oil industry in northern Scotland. Within the excesses associated with such unusual situations, there was a higher proportion of children with fathers in high contact occupations, as noted in certain established infections.1

Evidence of a relation with occupational levels of contact (although worth checking formally) would not be predicted in data on childhood leukaemia from the whole of England and Wales, dominated as they are by urban areas. In such areas of high population density, even if exposed to population mixing, the prevalence of susceptible people would tend to be lower, and of immune people to be higher, so that the high level of herd immunity would reduce the likelihood of epidemics. It would be unfortunate if infrequent but informative situations were neglected as a result of a misconception that if infection is present, then evidence should be observable generally. In this connection, it may be worth recalling that those who did the classic work on feline leukaemia virus stressed that nothing about the pattern of the usual (sporadic) cases of leukaemia in domestic urban cats suggested infection; only in the special situation of multicat households did the disease show epidemic features.

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