CORRESPONDENCE

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

Editor—It is with a sense of “deja-vu” 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, 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 with minimal or no exposure to asbestos. When this is done, the risk of lung cancer is indeed increased among maintenance workers in the refinery and petrochemical sector.

I have performed some calculations with the data in the table 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 each of the maintenance employee groups. Even for the group of maintenance workers potentially exposed >20 years, there is still an 11% deficit of lung cancer (SMR 89, 95% confidence interval (95% CI) 70 to 112).

It should be noted that the maintenance category that Finkelstein has constructed is a combination of all of the different maintenance trades, except for insulators, each with different job responsibilities and exposures. If the trends for lung cancer for the maintenance subgroups from the complete Texaco mortality study report are examined (unpublished data), none of these categories show an increasing trend in the lung cancer SMR with increasing duration of employment in the subgroup (as shown in table 1).

However, in a previous article, Finkelstein again compared the results for lung cancer in maintenance workers who had ≥20 years of potential exposure to asbestos to those for non-maintenance employees who had ≥20 years of employment in refinery or chemical operations. Because age was related to cumulative exposure, the maintenance group with ≥20 years of potential exposure to asbestos was older than the corresponding non-maintenance group. In fact, 52% of the person-years in the maintenance group was from workers ≥20 years only in the non-maintenance group. Although Finkelstein calculated a relative risk, based on the ratio of two SMRs, 1.24 for the two groups, a more exact age standardized rate ratio for lung cancer is 1.09 (95% CI 0.79 to 1.50) (Tsai SP, Glister DL, Ross CE. Lung cancer among maintenance employees in a refinery and petrochemical plant, unpublished letter.4) This rate ratio does not support increased lung cancer mortality among maintenance employees compared with non-maintenance employees.

Also, in their study of maintenance workers potentially exposed to asbestos, Tsai et al provided similar SMRs for lung cancer by duration of employment for maintenance employees (a group defined exactly as in our report). The SMRs are 88, 77, and 83 for those employed 1–5 years, 5–19 years, and ≥20 years as maintenance workers, and there is no trend of increasing lung cancer with increasing duration of employment. Thus the study of Tsai et al does not support Finkelstein’s allegations that, in general, petroleum industry maintenance workers have an increased risk of lung cancer compared with other workers, which increases with duration of employment as a maintenance worker.

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, &gt;20 y</td>
<td>75</td>
<td>84</td>
<td>89</td>
</tr>
</tbody>
</table>

I have performed some calculations with the data in the table 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 Day4 to compute a test for trend in these SMRs. I used the integers 1 to 4 to rank the exposure levels. The value for χ2 in the trend test was 11 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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Author’s reply—Finkelstein has reviewed the lung cancer results for a select group of maintenance workers in our paper on the update of the Texaco mortality study. In his letter, he suggests that although several papers on petroleum refinery workers have shown no increase in the standardized mortality ratios (SMR) for lung cancer, if internal comparisons of the SMRs were done, this would show an increased risk of lung cancer among maintenance workers in the refinery and petrochemical sector.

It has been well documented that a valid comparison of age-standardised SMRs requires that the underlying age specific mortality rate within each study group is constant or that the age distributions of person-years are similar. The value for trend in these SMRs. I used the integers 1 to 4 to rank the exposure levels. The value for χ2 in the trend test was 12.3 on 1 degree of freedom.

It should be noted that the maintenance group had higher age at death than the non-maintenance group. Because age was related to cumulative exposure, the maintenance group with ≥20 years of potential exposure to asbestos was older than the corresponding non-maintenance group. In fact, 52% of the person-years in the maintenance group was from workers ≥20 years only in the non-maintenance group. Although Finkelstein calculated a relative risk, based on the ratio of two SMRs, 1.24 for the two groups, a more exact age standardized rate ratio for lung cancer is 1.09 (95% CI 0.79 to 1.50) (Tsai SP, Glister DL, Ross CE. Lung cancer among maintenance employees in a refinery and petrochemical plant, unpublished letter.6) This rate ratio does not support increased lung cancer mortality among maintenance employees compared with non-maintenance employees.

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

<table>
<thead>
<tr>
<th>Specific craft</th>
<th>Obs</th>
<th>SMR (95% CI)</th>
<th>Obs</th>
<th>SMR (95% CI)</th>
<th>Obs</th>
<th>SMR (95% CI)</th>
<th>Obs</th>
<th>SMR (95% CI)</th>
<th>Trend test</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulator</td>
<td>48</td>
<td>90 (66 to 120)</td>
<td>31</td>
<td>77 (52 to 110)</td>
<td>10</td>
<td>133 (63 to 245)</td>
<td>7</td>
<td>128 (51 to 264)</td>
<td>2.25</td>
<td>0.13</td>
</tr>
<tr>
<td>Painter</td>
<td>30</td>
<td>76 (51 to 108)</td>
<td>19</td>
<td>69 (41 to 108)</td>
<td>8</td>
<td>100 (43 to 197)</td>
<td>3</td>
<td>87 (14 to 195)</td>
<td>0.03</td>
<td>0.86</td>
</tr>
<tr>
<td>Structural steel, pipe fitter</td>
<td>202</td>
<td>78 (67 to 99)</td>
<td>105</td>
<td>80 (65 to 97)</td>
<td>46</td>
<td>71 (52 to 99)</td>
<td>51</td>
<td>78 (58 to 102)</td>
<td>0.04</td>
<td>0.84</td>
</tr>
<tr>
<td>Electrician, instrument</td>
<td>38</td>
<td>78 (53 to 108)</td>
<td>11</td>
<td>89 (54 to 160)</td>
<td>19</td>
<td>97 (58 to 151)</td>
<td>5</td>
<td>36 (12 to 83)</td>
<td>3.40</td>
<td>0.07</td>
</tr>
<tr>
<td>Boilermaker, welder, lead burner</td>
<td>52</td>
<td>65 (48 to 85)</td>
<td>23</td>
<td>60 (38 to 90)</td>
<td>19</td>
<td>83 (50 to 130)</td>
<td>10</td>
<td>53 (26 to 98)</td>
<td>0.06</td>
<td>0.80</td>
</tr>
<tr>
<td>Equipment operator</td>
<td>59</td>
<td>73 (55 to 94)</td>
<td>42</td>
<td>76 (54 to 103)</td>
<td>9</td>
<td>50 (23 to 98)</td>
<td>8</td>
<td>102 (44 to 202)</td>
<td>0.13</td>
<td>0.72</td>
</tr>
<tr>
<td>Cleaner</td>
<td>91</td>
<td>68 (54 to 83)</td>
<td>84</td>
<td>67 (58 to 130)</td>
<td>5</td>
<td>58 (18 to 130)</td>
<td>147</td>
<td>147 (80 to 237)</td>
<td>0.50</td>
<td>0.25</td>
</tr>
<tr>
<td>Pipefitter, boilermaker</td>
<td>161</td>
<td>81 (68 to 94)</td>
<td>97</td>
<td>78 (63 to 95)</td>
<td>53</td>
<td>81 (55 to 113)</td>
<td>31</td>
<td>90 (61 to 128)</td>
<td>0.49</td>
<td>0.47</td>
</tr>
<tr>
<td>All maintenance</td>
<td>419</td>
<td>74 (67 to 81)</td>
<td>213</td>
<td>76 (66 to 87)</td>
<td>99</td>
<td>64 (52 to 77)</td>
<td>107</td>
<td>82 (67 to 98)</td>
<td>0.20</td>
<td>0.65</td>
</tr>
</tbody>
</table>
Correspondence

Childhood leukaemia, population mixing, and paternal occupation

The possible infective origin of childhood leukaemia has been the subject of much recent attention.1 Leukaemia has been the subject of much research. To 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 question with data from previous studies that were carried out to test the view that it is a rapid 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 rural groups 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.1

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References


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