Correspondence

Do amines induce occupational asthma in workers manufacturing polyurethane foams?

Sir,—Candura and Moscati refer (1984;41:552–3) to various publications according to which exposure to amines resulted in occupational asthma. In particular the findings by L Belin and coworkers are mentioned.

The amines described in the seven publications quoted in the paper belong without exception to the class of aliphatic amines whereas the authors assume that toluenediamine (TDA), which belongs to the class of aromatic amines, might also cause occupational asthma. There was, according to the bronchial provocation test as carried out by the authors, no indication that this assumption was correct. This finding is in line with the experience that aromatic amines in general do not cause asthmatic symptoms.

The authors claim that TDA is formed from toluenediisocyanate (TDI) during the chemical process by which polyurethane foams are produced. In reality, amine intermediates which are formed by the reaction of isocyanates with water react further with isocyanate groups to produce a urea.1 A recent study has shown that airborne TDI does not form TDA in an atmosphere that contains up to 70% relative humidity.2 Therefore, there is no evidence that occupational exposure to TDA may occur in plants in which polyurethane foam is manufactured.

There is also a miscalculation in the paragraph specifying the TDA concentrations for the bronchial provocation test. Under the assumption of a total hydrolysis of TDI to TDA, 0.07 mg TDA will form 0.05 mg TDA (analogously 0.1 mg TDA is obtained from 0.14 mg TDI). The TDA concentrations (1.31 and 3.21 mg/m3) used in the bronchial provocation test were therefore 25–30 times higher than those corresponding to 0.005/0.01 ppm TDI under the purely theoretical assumption of total hydrolysis.

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References


Mesothelioma and asbestos fibre type

Sir,—If Dr Finkelstein1 intended to emphasise the similarity in the exposure–response relationships for cancer mortality, particularly of the respiratory tract, in our cohort studies of two American asbestos textile plants2,3 we would agree with him. The patterns of relative risk, whether based on SMRs, which at least take account of age and era, or on proportional mortality rates, which do not, were remarkably similar in these two plants. Moreover, as we pointed out, the experience of these workers, dose for dose, was far worse than that of chrysotile miners and millers4 or chrysotile friction product workers.5

If his intention was to imply that the difference between 14 deaths from mesothelioma in the Pennsylvania plant (plus five more in women not reported in this paper) compared with one case in the South Carolina plant was due to misdiagnoses rather than the use of small quantities of amphibole fibre at the former, we would agree with him. It is true that in the Pennsylvania cohort there was evidence that the number of mesotheliomas was underestimated, but this was certainly not so in South Carolina. Both we and the NIOSH team who independently studied the same plant6 combined the local necropsy records and reviewed cohort death certificates for any suggestion of missed cases. Between us, we could find only the one case. This was not because local pathologists were strangers to the diagnosis or blind to asbestos related cancer. The first case of lung cancer attributed to asbestos was reported by Lynch and Smith,7 pathologists from the local Medical College of South Carolina. When mesotheliomas were linked with asbestos, this department made and has continued to make substantial efforts to find cases. A considerable number of mesotheliomas were found from the local area where there are shipyards, but none were found in employees of the textile plant.

The controversy on mesothelioma and fibre type is indeed bedevilled by many problems of which, we agree, exposure estimation is perhaps the most serious. The case against the amphiboles, however, depends not only on our cohort studies but on much other evidence in which diagnostic biases do not appear to have been a major factor.8 This includes the disastrous experience, described by Dr Finkelstein, of Ontario asbestos cement workers exposed to chrysotile and crocidolite.9

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Job related mortality risks of Hanford workers and their relation to cancer effects of measured doses of external radiation

Sir,—According to Gilbert and Peterson,1 who disapprove of the latest attempt by Mancuso, Stewart, and Kneale (MSK) to control for job related mortality risks other than external radiation,2 one of the reasons why their tests of cancer risks for radiation workers at Hanford have not confirmed the latest MSK findings for “radiosensitive cancers” is because their choice of controlling factors is more appropriate than the MSK choice. There is, however, a much stronger reason—namely, lack of conformity between the two groups of cancers which are supposed to correspond to an ICRP classification of “tissue sensitivity to cancer induction effects of radiation”3 (so called A series or radiosensitive cancers).

In the MSK analysis this group was formed from the 8th revision of the ICD list of diseases and causes of death by pooling the following code numbers: 146–9, 151, 153, 157, 162–3, 174, 193, 200–3, and 205. In the G/P analysis the group included the following code numbers: 146–59, 162–3, 174, 193, 200–9 (table). Therefore, although the total number of deaths was larger in the MSK series of 1943–79 deaths (4939) than in the G/P series of 1943–78 deaths (4635), the latter included more radiosensitive cancer (612) than the former (572).

For all deaths the ratio of MSK to G/P cases (1.07) showed no signs of being dose related, but for radiosensitive cancers the ratio was much higher for the upper half of the dose scale (1.12) than the lower half (0.89).

Therefore, if Gilbert and Peterson are anxious to prove that Hanford data are not inconsistent with current estimates of radiation risks—and are not prepared to control for job related mortality risk by the MSK method—they should at least (1) show us the results of applying to their data the latest MSK groupings of radiosensitive and other cancers and (2) include the two groups of deaths from cancer in an analysis that distinguishes between craftsmen and operators and other Hanford workers.

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Comparisons between two versions of Hanford data

<table>
<thead>
<tr>
<th>Dose (rems)</th>
<th>Mancuso, Stewart, and Kneale (MSK)</th>
<th>Gilbert and Peterson (G/P)</th>
<th>Ratio of MSK to G/P cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All deaths</td>
<td>Radiosensitive* cancers</td>
<td>All deaths</td>
</tr>
<tr>
<td>0.00–1</td>
<td>1060 (105)</td>
<td>998 (123)</td>
<td>1.06</td>
</tr>
<tr>
<td>0.01–0.05</td>
<td>555 (66)</td>
<td>522 (79)</td>
<td>1.06</td>
</tr>
<tr>
<td>0.08–0.1</td>
<td>869 (99)</td>
<td>819 (117)</td>
<td>1.06</td>
</tr>
<tr>
<td>0.32–0.4</td>
<td>647 (96)</td>
<td>600 (94)</td>
<td>1.08</td>
</tr>
<tr>
<td>0.64–0.75</td>
<td>689 (77)</td>
<td>652 (84)</td>
<td>1.06</td>
</tr>
<tr>
<td>1.02–1.25</td>
<td>530 (52)</td>
<td>491 (48)</td>
<td>1.08</td>
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<tr>
<td>2.50–3.0</td>
<td>231 (33)</td>
<td>218 (31)</td>
<td>1.06</td>
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<tr>
<td>5.12–10</td>
<td>150 (20)</td>
<td>143 (16)</td>
<td>1.05</td>
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<tr>
<td>10.24–20</td>
<td>96 (17)</td>
<td>92 (15)</td>
<td>1.04</td>
</tr>
<tr>
<td>20.48–40</td>
<td>112 (7)</td>
<td>100 (5)</td>
<td>1.12</td>
</tr>
<tr>
<td>Total</td>
<td>4939 (572)</td>
<td>4635 (612)</td>
<td>1.07</td>
</tr>
</tbody>
</table>


Positions on the dose scale take no account of radiation doses within 10 years of death.

References

Hand injuries in south Wales coal miners

We acknowledge the help of Mrs K Bolt with the preparing and typing of this paper and also the National Coal Board in Wales for permission to use the illustration in the text.

References


Correction

Mesothelioma and asbestos fibre type
In the second paragraph of this letter (August, p 567) it should have read “... small quantities of amphibole fibre at the former, we would not agree with him.”