Estimating age, sex and period specific constant tar cigarette consumption in the UK

In a paper published three years ago (September 1990;45:657-65) we examined trends in smoking associated respiratory diseases up to 1985 in relation to trends in cigarette smoking. In that paper we presented a graph for each sex of cumulative constant tar cigarette consumption (CCTCC) by age and year of death, and by age and year of birth. We were approached by the Lung and Asthma Information Agency for permission to use these graphs with the request that they be updated to 1990. While updating the figures an unfortunate error was noted in how the CCTCC estimates had been calculated. Although this did not significantly affect the estimates, or the conclusions of our paper at all, we are writing to present the corrected and updated CCTCC data (table). For each sex, period, and age group, CCTCC is an estimate of the average total lifetime number of manufactured cigarettes of constant tar smoked (using tar factors of 1 for 1961-65, 0.804 for 1966-70, 0.613 for 1971-5, 0.544 for 1976-80, 0.477 for 1981-5 and 0.423 for 1986-90). The CCTCC data are lifetime consumption estimates directly comparable with mortality estimates obtained by aggregating annual age specific data over five year periods. Details of the basic data from which the estimates were derived, and the precise method used to derive them (incorrectly described in our previous paper) are available on request.

**Alveolar atypical hyperplasia in association with primary pulmonary adenocarcinoma: a clinicopathological study of 10 cases**

I read with interest the description by DR FA Carey and colleagues (December 1992;47:1041-3) of nodules of atypical alveolar cell hyperplasia in 10 patients. The cases as illustrated are identical to those I have reported.1

The authors make the point that their cases were found in formalin inflated lung but were not grossly apparent in most, if not all, instances. They further state that previous reports of such lesions, including my reports,2 were based on lesions found in formalin inflated lung. In fact the great majority of cases in our series were inflated in Bouin’s solution and that point was made in both papers. The issue is of importance since the lesions are not usually grossly apparent in formalin inflated lung but are easy to see in specimens of lung inflated in Bouin’s solution, and this difference has been illustrated.3 The inability to see these lesions grossly will result in an under-estimate of frequency in prospective studies, and if the authors are planning such a study, I would recommend that inflation in Bouin’s solution be considered.

Dr Carey and coworkers comment that it is premature to view these nodules as bronchiolaevolar cell adenomas rather than atypical hyperplasia since they have not been described except in association with carcinoma. I have, in fact, found such lesions in several resections for other types of primary disease (granulomas and metastatic sarcomas, for example), but I cannot understand how this is relevant to the issue of whether or not these lesions are neoplasms.

**Cumulative constant tar cigarette consumption (in thousands) by sex, age and period**

<table>
<thead>
<tr>
<th>Age</th>
<th>Period</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>1951-55</td>
<td>3</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>1961-65</td>
<td>2</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>1971-75</td>
<td>3</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>1981-85</td>
<td>4</td>
<td>12</td>
<td>21</td>
</tr>
<tr>
<td>1991-95</td>
<td>4</td>
<td>15</td>
<td>27</td>
</tr>
<tr>
<td>1951-55</td>
<td>4</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>1961-65</td>
<td>4</td>
<td>16</td>
<td>32</td>
</tr>
<tr>
<td>1971-75</td>
<td>4</td>
<td>18</td>
<td>35</td>
</tr>
</tbody>
</table>
| 1981-85 | 4 | 20 | 39 | 58 | 76 | 95 | 107 | 111 | 104 | 93 | 79 | 64 | 50 | 39 | **ROBERTA R MILLER**

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AUTHORS’ REPLY We accept Dr Miller’s point that lesions of atypical alveolar cell hyperplasia are more easily identified macroscopically in specimens of lung inflated in Bouin’s solution. There are, however, disadvantages to the use of this fixative, not least of which is the fact that it severely compromises nuclear DNA analysis of tumour material. Also, since formalin is widely accepted as the routine fixative of choice, a change to Bouin’s solution for lung specimens is not likely to be widely accepted. It is, nevertheless, possibly true that inflation in Bouin’s solution might be adopted in any prospective search for atypical hyperplasia in pulmonary resection specimens.
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