RESEARCH REPORT

Oil combustion and childhood cancers

E G Knox


Study objectives: To identify specific toxic atmospheric emissions and their industrial sources in Great Britain. To link them with each other and with the birth addresses of children dying from cancer. To identify specific causal agents and sources.

Design: Birth and death addresses of children dying from cancer were linked to emissions hotspots for specific chemicals: and to related source installations. Among those who moved house, distances from each address to the nearest hazard were compared. Relative excesses of close-to-hazard birth addresses showed high prenatal or early postnatal risks. Relative risks for individual and for combined exposures were measured.

Setting and subjects: Atmospheric emissions hotspots (UK, 2001) published as maps on the internet, were converted to coordinates. Industrial sites were identified through trade directories and map inspections. Child cancer addresses for 1955–80 births were extracted from an earlier inquiry and their postcodes converted to map references.

Main results: There were excess relative risks (RR) within 0.3 km of hotspots for carbon monoxide, PM10 particles, nitrogen oxides, 1,3-butadiene, benzene, dioxins, benz(a)pyrene, and volatile matter; and within 1.0 km of bus stations, hospitals, heavy transport centres, railways, and oil installations. Some excesses were attributable to mutual confounding, but 1,3-butadiene and carbon monoxide, mainly derived from engine exhausts, were powerful independent predictors. They were strongly reinforced when associated with bus stations, hospitals, railways, oil installations, and industrial transport centres; RR = 12.6 for joint exposure 0.5 km exposure to bus stations and 1,3-butadiene.

Conclusions: Childhood cancers are strongly determined by prenatal or early postnatal exposures to oil combustion gases, especially from engine exhausts. 1,3-butadiene, a known carcinogen, may be directly causal.

Previous studies showed that many childhood cancers are probably initiated in early infancy or in the fetus through direct or through maternal exposure to toxic atmospheric discharges. Places of birth were strongly and specifically associated with high local emissions of oil combustion products and of volatile fuels and solvents. They were also closely associated with installations and industrial processes known to generate combustion based or volatile materials, or involved in large scale transport or motor transport. This contrasted with weak or absent associations with inorganic industrial emissions. Four of the associated substances (1,3-butadiene (BUT), benz(a)pyrene (BZP), dioxins (DXN), benzene (BZN)) are known or suspected carcinogens in experimental animals or among industrial workers. These, and other associated substances, (nitrogen oxides (NOx), carbon monoxide (CO), non-methane volatile organic substances (VOC), and fine particulates (PM10)) are derived largely from fuelling and operating internal combustion engines (ICE).

Unanswered questions arose mainly from the close geographical links between different emissions and between them and the industrial sites, and from difficulties in separating their effects. For example, was BZN truly leukaemogenic, as generally believed, or were its associations secondary to geographical confounding with other ICE related materials? Were the associations between childhood cancer and municipal incinerators due to discharges of DXN or to the engine exhausts of vehicles supplying them? Were observed associations with hospital sites a function of their incinerators or of their traffic, or both? At what ranges were the different exposures effective?

Such uncertainties reduce in the end to two fundamental questions namely (1) which chemicals initiated the cancers? (2) what were their physical sources? This study aims to answer these questions.

METHODS

The case material was recovered from an earlier investigation of all 22 458 children dying from leukaemia or other cancer between 1953 and 1980 in Great Britain before their 16th birthday; but this analysis is limited to the subset born from 1955 onwards. Home address and birth address postcodes (PCs) were identified and their map references extracted from the Central PC Directory. Inexact birth addresses supplied by witnesses other than parents were sometimes coded to the nearest central "IAA" PC: with a consequent risk of false association with city centre pollution sources. Such PCs, from such witnesses, were excluded. Urban PC map references represent actual addresses within about 200 metres, although rural PCs are less precise.

The cancers were originally classified into 10 diagnostic subtypes, each analysed separately in the previous paper, but they are grouped here to two main classes, the reticuloendothelial (RE) cancers (leukaemias and lymphomas), and the other "solid" tumours.

Locations of national chemical emission "hotspots" were obtained from maps for 2001 published on the web site of the National Atmospheric Emissions Inventory (NAEI). The maps were downloaded as bitmaps, unwanted areas (Northern Ireland, Orkney, Shetland, North Sea oil/gas platforms) were edited out, and colour-coded pixels to identify nearness of exposed addresses. The map references (PCs) were converted to coordinates with a tolerance of 200 metres. Any address whose coordinates were more than 50 km from the nearest PC was excluded.

Abbreviations: BUT, butadiene; BZP, benz(a)pyrene; DXN, dioxins; BZN, benzene; NOx, nitrogen oxides; CO, carbon monoxide; VOC, volatile organic substance; PM10, fine particulates; ICE, internal combustion engine; PC, postcode

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Results

The 12,018 reliably located post-1954 records included 5849 non-migrants plus 6169 who moved at least 0.1 km between birth and death. Distributions of distances between migrant addresses and nearest hazards displayed several close range birth-death asymmetries. Disaggregation according to birth-to-death migration distance then showed that the asymmetries were limited to the 5125 children who had moved >1.0 km. Emission related results for this group are shown in table 1 in descending order of their short distance (<0.3 km) birth/death ratios. The list was headed by DXN, BUT, and CO. Within the available map resolution a distance of 0.3 km means that these addresses occupied the same “hot” pixel as the hazard itself, or an adjacent one. DXN, BUT, and BZP showed additional birth excesses at greater ranges.

Relative risks were next estimated among subsets of children whose two addresses were on opposite sides of declared circumferences drawn around the hazards. This procedure discards many non-informative cases and supplies more robust RR estimates. The first three distance classes of table 1 were consolidated and re-tested among those crossing a 1.0 km boundary, to give the RR estimates shown in the last column of the table.

Except for DXN emissions, which are generated by organic combustion in the presence of chlorine containing compounds, the emission types shown in table 1 are mainly or partly derived from fuelling and operating ICEs, so the effects of the individual materials are difficult to separate. Pairs of the seven ICE related emissions were examined simultaneously to distinguish independent cancer predictors from those whose associations depended upon their shared locations. Each emission type was examined at distances beyond the effective range of the paired hazard. This was set variously at 0.3 km, 0.5 km, and 1.0 km, with similar results, and table 2 gives results for 0.3 km. The separation technique is illustrated in the appendix.

Many out-of-range ORs gave little indication of independent prediction (VOC:BZ:BN:BZP:NOx) but three (CO:BUT:PM10) displayed independent activity. The DXN pattern was irregular: probably because of smaller numbers. ORs were also calculated for migrants where both addresses were located together on one side or other side of a 1.0 km hazard boundary, and the greatest values again related to hazard pairs involving CO or BUT, and especially the combination of CO with BUT. This is probably because both are almost specifically derived from engine exhausts, whereas the other emissions have important alternative sources.

Table 3 lists birth and death distance distributions for different combustion and volatile associated industrial activities. The final column gives consolidated results for migrants crossing a 1.0 km circumscribed boundary. These sites show broader proximity distributions than those in table 1, possibly reflecting the radial movements of vehicles operated by them, or high local concentrations of other vehicles. The most striking result is the extraordinary concentration of cancer births within 0.3 km of bus/coach stations; (OR = 12.5; CI = 7.7 to 20.3). This was followed (at 0.3 km) by hospitals (OR = 2.62.0 to 3.3) and heavy transport centres (OR = 1.6:1.1 to 2.4). Railways showed only moderate relative excesses, perhaps because diesel did not fully displace steam until after 1960 but they display large numbers of close birth contacts. Incinerators, by contrast, were related to few nearby births or deaths.

Mutually augmenting relations between the emissions (table 1) and the installations (table 3) were displayed in combined two-hazard distance matrices, condensed to different critical boundaries. Table 4 gives results for 0.5 km and shows that double hazard proximities were
frequently more effective than their separated components. BUT and CO greatly increased the predictive power of installations with which they were associated, at the same time increasing their own effects. Individual toxicities and emission levels must vary within each class of hotspot, and powerful joint effects such as this presumably point to an especially powerful common source. The DXN effects were again erratic and installation related risk enhancements were more strongly associated with ICE sources rather than incinerators. Technical improvements in waste incineration may have contributed to this. The cancer risk associations of hospitals may also now depend chiefly upon their motor traffic and not, as previously supposed, upon their incinerators.3 Indeed, DXN may be less of a direct child hazard than is often supposed. Long term surveillance after the massive DXN contamination at Seveso recorded only small excesses of lymphohaemopoietic cancers in adults, and no excesses in children.9

The distribution analyses were repeated separately for the main tumour classes. As in earlier studies (where each individual tumour type was examined) they showed no differences.1 7 Results were also obtained for children up to 60 months at death, over 60 months, and up to 18 months. As before there were no evident differences in their hazard proximity patterns.1

**DISCUSSION**

These results confirm the relative proximities of child cancer births to substance specific hotspots from oil based emissions, and to industrial sites known to discharge such materials.1–3 They identify the most powerful of the single and combined exposures and specify the distance distributions between hazards and births. They separate the direct effects of single and combined exposures from indirect statistical associations.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Distance distributions of births and deaths relative to nearest emissions hotspots</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distances (km)</td>
<td>-0.3</td>
</tr>
<tr>
<td>Dioxins (107B map points)</td>
<td></td>
</tr>
<tr>
<td>Births</td>
<td>123</td>
</tr>
<tr>
<td>Deaths</td>
<td>38</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>3.24</td>
</tr>
<tr>
<td>BUT</td>
<td>1.85 to 2.70</td>
</tr>
<tr>
<td>Carbon monoxide (13371)</td>
<td></td>
</tr>
<tr>
<td>Births</td>
<td>1123</td>
</tr>
<tr>
<td>Deaths</td>
<td>477</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>2.35</td>
</tr>
<tr>
<td>CO</td>
<td>2.06 to 2.53</td>
</tr>
<tr>
<td>PM10 particles (39749)</td>
<td></td>
</tr>
<tr>
<td>Births</td>
<td>1976</td>
</tr>
<tr>
<td>Deaths</td>
<td>1113</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>1.78</td>
</tr>
<tr>
<td>BUT</td>
<td>1.67 to 2.00</td>
</tr>
<tr>
<td>Benzoperylene (13813)</td>
<td></td>
</tr>
<tr>
<td>Births</td>
<td>826</td>
</tr>
<tr>
<td>Deaths</td>
<td>563</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>1.47</td>
</tr>
<tr>
<td>CO</td>
<td>1.55 to 1.94</td>
</tr>
<tr>
<td>Nitrogen oxides (53875)</td>
<td></td>
</tr>
<tr>
<td>Births</td>
<td>2809</td>
</tr>
<tr>
<td>Deaths</td>
<td>1936</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>1.45</td>
</tr>
<tr>
<td>BUT</td>
<td>1.74 to 2.16</td>
</tr>
<tr>
<td>PM10 (53279)</td>
<td></td>
</tr>
<tr>
<td>Births</td>
<td>3515</td>
</tr>
<tr>
<td>Deaths</td>
<td>3023</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>1.16</td>
</tr>
<tr>
<td>CO</td>
<td>1.34 to 1.71</td>
</tr>
<tr>
<td>Benzene (59811)</td>
<td></td>
</tr>
<tr>
<td>Births</td>
<td>3672</td>
</tr>
<tr>
<td>Deaths</td>
<td>3152</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>1.16</td>
</tr>
<tr>
<td>CO</td>
<td>1.32 to 1.71</td>
</tr>
</tbody>
</table>

*ORs < 1.0 in subset who migrated >1.0 km and who had one address within 1.0 km of the nearest hazard, and the other address outside this distance.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Odd ratios* for emissions-Y outside the effective ranges† of emissions-X</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emission-X</td>
<td>BUT</td>
</tr>
<tr>
<td>BUT</td>
<td>–</td>
</tr>
<tr>
<td>CO</td>
<td>2.15</td>
</tr>
<tr>
<td>PM10</td>
<td>1.71</td>
</tr>
<tr>
<td>NOx</td>
<td>1.59</td>
</tr>
<tr>
<td>BZP</td>
<td>1.40</td>
</tr>
<tr>
<td>BZN</td>
<td>1.30</td>
</tr>
<tr>
<td>VOC</td>
<td>1.23</td>
</tr>
<tr>
<td>DXN</td>
<td>2.20</td>
</tr>
</tbody>
</table>

*ORs > 2.00 shown in bold. ORs < 1.30 shown in italics. †Effective range taken to be 0.3 km.
The task was hampered by the common physical sources of some ICE related emissions (especially CO, PM10, and BUT) and by non-exclusivity (for example, VOC includes BZN and BZP). However, three short range birth exposures (CO:DXN:BUT) emerged as strong independent cancer predictors. CO and BUT are almost uniquely derived from engine exhausts and the importance of this source was confirmed by the mutually augmenting combinations with bus stations, hospitals, heavy transport centres, and railways. This points strongly towards ICE exhausts as an important initiating cause of childhood cancers, in contrast with the weaker associations of fuel evaporation sources, including benzene. Diesel exhausts are particularly incriminated.

### Table 3 Distance distributions of births and deaths relative to nearest hazard

<table>
<thead>
<tr>
<th>Distances (km)</th>
<th>–0.3</th>
<th>–0.5</th>
<th>–1.0</th>
<th>–2.0</th>
<th>–5.0</th>
<th>–5.0–1.0</th>
<th>0.95 CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Bus stations (444 map points)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>18</td>
<td>43</td>
<td>209</td>
<td>635</td>
<td>1805</td>
<td>2415</td>
<td>212</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>12.06</td>
<td>3.00</td>
<td>1.81</td>
<td>1.03</td>
<td>0.93</td>
<td>0.86</td>
<td>3.14 to 3.68</td>
</tr>
<tr>
<td>(b) Hospitals (1248)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>85</td>
<td>142</td>
<td>549</td>
<td>1311</td>
<td>1954</td>
<td>1084</td>
<td>516</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>2.49</td>
<td>2.23</td>
<td>1.60</td>
<td>1.02</td>
<td>0.78</td>
<td>0.79</td>
<td>2.23 to 2.48</td>
</tr>
<tr>
<td>(c) Heavy transport (1019)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>65</td>
<td>114</td>
<td>471</td>
<td>1090</td>
<td>1528</td>
<td>2622</td>
<td>241</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>1.59</td>
<td>1.48</td>
<td>1.77</td>
<td>1.30</td>
<td>0.88</td>
<td>0.86</td>
<td>1.88 to 2.16</td>
</tr>
<tr>
<td>(d) Industrial solvent use (486)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Deaths</td>
<td>17</td>
<td>89</td>
<td>368</td>
<td>784</td>
<td>1465</td>
<td>2262</td>
<td>241</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>1.78</td>
<td>1.44</td>
<td>1.67</td>
<td>1.20</td>
<td>0.96</td>
<td>0.90</td>
<td>1.85 to 2.17</td>
</tr>
<tr>
<td>(e) Incinerators (70)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>0</td>
<td>10</td>
<td>34</td>
<td>169</td>
<td>678</td>
<td>4234</td>
<td>43</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>–</td>
<td>0.70</td>
<td>1.97</td>
<td>1.28</td>
<td>0.97</td>
<td>0.99</td>
<td>1.76 to 2.58</td>
</tr>
<tr>
<td>(f) Railway lines</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deaths</td>
<td>5</td>
<td>9</td>
<td>54</td>
<td>222</td>
<td>975</td>
<td>3860</td>
<td>43</td>
</tr>
<tr>
<td>B/D ratio</td>
<td>1.43</td>
<td>0.88</td>
<td>1.67</td>
<td>0.81</td>
<td>0.76</td>
<td>0.82</td>
<td>1.62 to 1.77</td>
</tr>
</tbody>
</table>

*As in table 1.

### Table 4 Cancer associations with industrial sites (Y) and emission hotspots (X). Odds ratios* inside/outside 0.5 km† of both hazards

<table>
<thead>
<tr>
<th>X</th>
<th>Y</th>
<th>Overall</th>
<th>CO</th>
<th>BUT</th>
<th>NOx</th>
<th>BZN</th>
<th>BZP</th>
<th>VOC</th>
<th>DXN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bus stations</td>
<td>6.01</td>
<td>12.42</td>
<td>12.58</td>
<td>10.36</td>
<td>9.75</td>
<td>8.68</td>
<td>4.43</td>
<td>8.36</td>
<td>13.65</td>
</tr>
<tr>
<td>Hospitals</td>
<td>2.48</td>
<td>5.27</td>
<td>5.31</td>
<td>4.47</td>
<td>4.41</td>
<td>3.39</td>
<td>3.38</td>
<td>3.26</td>
<td>5.10</td>
</tr>
<tr>
<td>Oil sources</td>
<td>2.37</td>
<td>–</td>
<td>4.78</td>
<td>6.23</td>
<td>5.28</td>
<td>3.81</td>
<td>2.16</td>
<td>1.90</td>
<td>–</td>
</tr>
<tr>
<td>Solvents</td>
<td>1.59</td>
<td>2.71</td>
<td>2.85</td>
<td>2.29</td>
<td>2.30</td>
<td>2.29</td>
<td>1.85</td>
<td>2.06</td>
<td>1.59</td>
</tr>
<tr>
<td>Railways</td>
<td>1.56</td>
<td>3.29</td>
<td>3.41</td>
<td>2.84</td>
<td>2.49</td>
<td>1.98</td>
<td>2.31</td>
<td>1.92</td>
<td>3.22</td>
</tr>
<tr>
<td>Transport</td>
<td>1.95</td>
<td>2.40</td>
<td>2.60</td>
<td>2.26</td>
<td>2.48</td>
<td>2.29</td>
<td>1.77</td>
<td>2.02</td>
<td>4.90</td>
</tr>
<tr>
<td>Incinerators</td>
<td>1.74</td>
<td>2.52</td>
<td>2.57</td>
<td>2.73</td>
<td>2.51</td>
<td>2.21</td>
<td>1.63</td>
<td>2.24</td>
<td>2.88</td>
</tr>
<tr>
<td>Host/Bus/Train</td>
<td>1.76</td>
<td>3.54</td>
<td>3.70</td>
<td>3.05</td>
<td>2.65</td>
<td>2.12</td>
<td>2.57</td>
<td>2.06</td>
<td>3.65</td>
</tr>
<tr>
<td>All ICE</td>
<td>1.66</td>
<td>3.24</td>
<td>3.24</td>
<td>2.80</td>
<td>2.46</td>
<td>2.01</td>
<td>2.46</td>
<td>1.95</td>
<td>3.59</td>
</tr>
<tr>
<td>B/D/ratio</td>
<td>1.47</td>
<td>1.13</td>
<td>0.91</td>
<td>0.68</td>
<td>0.77</td>
<td>0.76</td>
<td>1.76</td>
<td>1.59</td>
<td>1.94</td>
</tr>
</tbody>
</table>

*These ratios serve as estimates of relative risk (RR) within combined proximity classes. The proportion of exposure attributable cases within each class is (RR–1)/RR. Thus, for cases born within 1.0 km of both a bus station and a BUT hotspot, 92.1% (11.58/12.58) can be attributed to the joint exposure. (Also see appendix). ORs >3.0 are shown in bold. Composite variables are shown separately at the feet of the primary lists. †Because of small numbers, incinerator and DXN ORs are based upon 1.0 km. Other ORs based on small numbers are omitted.
The main ill effects of ICE related emissions occurred within a nominal 0.3 km of their hotspots, suggesting a rapidly dispersing ground level source. A single responsible component cannot finally be identified but CO is not generally regarded as a carcinogen while 1,3-butadiene combines the statistical and biological properties of a direct agent. Combined with sub-0.5 km exposures to bus/coach stations, it generated a 12-fold increased risk.

Calculations of population attributable risks remain uncertain because of mutual confounding between emission types, incomplete source data, and the limited geographical resolution of the emissions hotspots. They returned values of 7% to 30% for industrial installations and 11% to 35% for emissions hotspots. These values show the general importance of their role but they cannot legitimately be combined to provide a composite assessment. A more exact treatment must await assembly of a fuller and more detailed inventory of specific sources.

A literature search returned no other migration based studies of contrasting toxic birth exposures. Migration studies of the population mixing infective hypothesis of leukaemia have not generally considered chemical exposures. However, several case-control studies based on cancer onset locations have been reported. One, from Denver USA, estimated benzene exposures from high volume roads within 750 feet of child cancer addresses and it yielded significant estimated benzene exposures from high volume roads within a nominal 0.3 km of their hotspots, suggesting a rapidly dispersing ground level source. A single responsible component cannot finally be identified but CO is not generally regarded as a carcinogen while 1,3-butadiene combines the statistical and biological properties of a direct agent. Combined with sub-0.5 km exposures to bus/coach stations, it generated a 12-fold increased risk.

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A literature search returned no other migration based studies of contrasting toxic birth exposures. Migration studies of the population mixing infective hypothesis of leukaemia have not generally considered chemical exposures. However, several case-control studies based on cancer onset locations have been reported. One, from Denver USA, estimated benzene exposures from high volume roads within 750 feet of child cancer addresses and it yielded significant RRs for all cancers (5.9) and for leukaemias (8.3). A similar diffusion model, used in Italy, yielded a significant leukaemia RR of 3.91. A Swedish study based upon NOx concentrations gave a significant child cancer RR of 3.8. Other case-control reports based on smaller numbers have led to “suggestive but inconclusive” results; and yet others to “no evidence of” conclusions. A recent French study of leukaemias examined both current and previous addresses and found a significant excess of dwellings adjacent to petrol and vehicle repair garages.

One large scale case-control study in California related the birth addresses of 4369 under 5 year cancers, to traffic exposure estimates similar to those used in the Denver study; but it reached a negative conclusion. The contradictions between the California findings, and those of this study and of the Denver studies could reflect combinations of recent catalyst use, different proportions of petrol and diesel traffic, different starting/stopping frequencies, or different hazard distance distributions. Another Californian study showed that fine particulates and CO were concentrated within 300 metres of major highways, beyond which a combination of dispersion and particle coagulation reduced concentrations to the background level.

The main public health implication from these findings is a requirement for improved control of ICE effluents, especially for diesel burning transport including buses, lorries, and locomotives; and for more detailed and frequent monitoring of 1,3-butadiene in particular. The dominant approach to control has been to specify maximum ambient air concentrations in workplaces, currently set at 1 ppb were not designed to prevent childhood cancers. Although they protect the working environments of pregnant women, they were not based upon estimated risks to unborn children, and may not be adequate even there. In any case, most emissions related cancer initiations probably arise from intermittent local exposures external to the working environment, mainly from industrial effluents and from vehicle engine exhausts. Substances other than 1,3-butadiene may also be involved. Controls should be directed now towards sources and outputs rather than upon ambient measurements.

Table 5

<table>
<thead>
<tr>
<th>Hazard</th>
<th>Proportion all births</th>
<th>Migrant RR</th>
<th>Attributable proportions</th>
<th>Attributable population risk per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bus stations</td>
<td>9.85</td>
<td>3.14</td>
<td>68.15</td>
<td>6.71</td>
</tr>
<tr>
<td>Hospitals</td>
<td>21.38</td>
<td>2.23</td>
<td>55.16</td>
<td>11.79</td>
</tr>
<tr>
<td>Railways</td>
<td>58.35</td>
<td>1.80</td>
<td>44.44</td>
<td>25.93</td>
</tr>
<tr>
<td>HE/Bus/Train</td>
<td>65.72</td>
<td>1.72</td>
<td>42.86</td>
<td>28.16</td>
</tr>
<tr>
<td>ICE</td>
<td>70.09</td>
<td>1.76</td>
<td>43.18</td>
<td>30.27</td>
</tr>
<tr>
<td>CO</td>
<td>45.21</td>
<td>2.46</td>
<td>59.35</td>
<td>26.83</td>
</tr>
<tr>
<td>BUT</td>
<td>32.55</td>
<td>2.28</td>
<td>56.14</td>
<td>18.27</td>
</tr>
<tr>
<td>PM10</td>
<td>58.35</td>
<td>1.83</td>
<td>45.36</td>
<td>26.46</td>
</tr>
<tr>
<td>NOx</td>
<td>71.38</td>
<td>1.94</td>
<td>48.45</td>
<td>34.59</td>
</tr>
<tr>
<td>BZn</td>
<td>83.33</td>
<td>1.50</td>
<td>33.33</td>
<td>27.78</td>
</tr>
<tr>
<td>BZP</td>
<td>26.89</td>
<td>1.73</td>
<td>42.20</td>
<td>11.35</td>
</tr>
<tr>
<td>VOC</td>
<td>81.43</td>
<td>1.51</td>
<td>33.78</td>
<td>27.50</td>
</tr>
</tbody>
</table>

* Migrant plus non-migrant. † (RR – 1)/RR.

Policy implications

Atmospheric standards for 1,3-butadiene in workplaces, currently set at 1 ppb were not designed to prevent childhood cancers. Although they protect the working environments of pregnant women, they were not based upon estimated risks to unborn children, and may not be adequate even there. In any case, most emissions related cancer initiations probably arise from intermittent local exposures external to the working environment, mainly from industrial effluents and from vehicle engine exhausts. Substances other than 1,3-butadiene may also be involved. Controls should be directed now towards sources and outputs rather than upon ambient measurements.

ACKNOWLEDGEMENTS

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Conflicts of interest: none.

The author is Emeritus Professor, University of Birmingham, UK.
APPENDIX

<table>
<thead>
<tr>
<th>Example</th>
<th>Hazard-Y</th>
<th>Hazard-X</th>
<th>OR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>a</td>
<td>Near</td>
<td>Far</td>
<td>2.19</td>
</tr>
<tr>
<td>b</td>
<td>Far</td>
<td>Near</td>
<td>1.97</td>
</tr>
<tr>
<td>c</td>
<td>Far</td>
<td>c</td>
<td>2.71</td>
</tr>
<tr>
<td>d</td>
<td>Near</td>
<td>d</td>
<td>2.00</td>
</tr>
<tr>
<td>e</td>
<td>Near</td>
<td>e</td>
<td>4.64</td>
</tr>
<tr>
<td>f</td>
<td>Far</td>
<td>f</td>
<td>6.01</td>
</tr>
<tr>
<td>g</td>
<td>Far</td>
<td>g</td>
<td>12.58</td>
</tr>
<tr>
<td>h</td>
<td>Near</td>
<td>h</td>
<td>16.08</td>
</tr>
</tbody>
</table>

*Confidence intervals calculated as suggested by Bland and Altman.

Relative risks in 2×2×2 tables, as used in tables 2 and 4

Separate condensed tables of hazard distances are assembled for birth and death addresses, each showing near and far classes for hazard-Y and hazard-X, as below. (0.5 km in example)

<table>
<thead>
<tr>
<th>Hazard-Y</th>
<th>Hazard-X</th>
<th>Birth addresses</th>
<th>Death addresses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Near</td>
<td>Far</td>
<td>a</td>
<td>e</td>
</tr>
<tr>
<td></td>
<td></td>
<td>b</td>
<td>f</td>
</tr>
<tr>
<td></td>
<td></td>
<td>c</td>
<td>g</td>
</tr>
<tr>
<td></td>
<td></td>
<td>d</td>
<td>h</td>
</tr>
</tbody>
</table>

Example

<table>
<thead>
<tr>
<th>Bus station</th>
<th>0.5</th>
<th>&gt;0.5</th>
<th>0.5</th>
<th>&gt;0.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>BUT</td>
<td>184</td>
<td>1224</td>
<td>18</td>
<td>689</td>
</tr>
<tr>
<td>BUT</td>
<td>162</td>
<td>3558</td>
<td>43</td>
<td>4378</td>
</tr>
</tbody>
</table>

Odds ratios serve here as estimators of the relative risks (RR) of birth proximities. ORs for different situations are listed below

<table>
<thead>
<tr>
<th>OR</th>
<th>[CI]*</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.19</td>
<td>(1.97 to 2.42)</td>
</tr>
<tr>
<td>2.71</td>
<td>(1.51 to 4.89)</td>
</tr>
<tr>
<td>4.64</td>
<td>(3.30 to 6.51)</td>
</tr>
<tr>
<td>5.75</td>
<td>(3.52 to 9.42)</td>
</tr>
<tr>
<td>12.58</td>
<td>(7.74 to 20.45)</td>
</tr>
<tr>
<td>6.01</td>
<td>(4.56 to 7.91)</td>
</tr>
</tbody>
</table>

In the example, both exposures are active (OR = 2.19, 4.64) when outside the effective range of the other; and each is increased (2.71, 5.75) when within range of the other. Finally, comparing double proximities with double non-proximities, the OR for a double exposure is 12.58.

REFERENCES


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