RESEARCH REPORT

Oil combustion and childhood cancers

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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, benzo(a)pyrene, and volatiles; 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 $<$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 based combustion gases, especially from engine exhausts. 1,3-butadiene, a known carcinogen, may be directly causal.

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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.[1] 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 rail or motor transport.[2 3] This contrasted with weak or absent associations with inorganic industrial emissions. Four of the associated substances (1,3-butadiene (BUT), benzo(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[4 5]; 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 ''1AA'' 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,[1] 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).[6] The maps were downloaded as bitmaps, unwanted areas (Northern Ireland, Orkney, Shetland, North Sea oil/gas platforms) were edited out, and colour coded pixels

Abbreviations: BUT, butadiene; BZP, benzo (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

representing the highest emissions were translated to grid coordinates, as described elsewhere.[1] The emission codes represent tonnes or kg or g/km^2/year. The maps are resolved to a precision of 413 metres per pixel and hotspots based upon kilometre squares were typically represented as groups of four or nine red pixels. The linear distance between an address and the centre of the nearest ''hot'' pixel was adopted as the measure of exposure. The associated errors are within about $\pm$0.5 km.

The limitation to post-1954 births ameliorated the effects of interim environmental changes, almost all of which were improvements. Some hotspots may have been lost but most of those still recorded in 2001 would have been present earlier. Their validity was confirmed by the results.[1] The use of distances and not values avoids artefacts arising from local quantitative improvements in the interval between the dates of the two datasets.

Data relating to industrial sites were obtained from several sources. Railways and motorways were digitised from maps using a digitising tablet.[1 2] Hospitals (except day hospitals and cottage hospitals) were identified through a web site directory of PCs of all NHS and private hospitals, subsequently translated to coordinates.[7] Incinerator locations were supplied by colleagues.[3] Bus and coach stations and termini were identified from national timetables and located directly on Ordnance Survey (OS) and A to Z street maps. Factories undertaking hazardous processes were identified through trade directories (see previous reports) and large hazards such as oil storage facilities, power stations, and airports were located directly on OS maps.[2] A new category of heavy transport foci was constructed from among these last groups. It comprised docks, harbours, ferries, breweries, brick lime cement and pottery works, steelworks and other smelters casters and forgers, bread and biscuit makers, car and aircraft factories, Ministry of Defence and nuclear establishments, rail yards and goods terminals, chemical manufacturers, cotton manufacturers, power stations: and others. For all these datasets except for the births and the deaths, secondary maps were constructed in the common format by placing them on an NAEI based outline map. This provided comparable resolution for all environmental hazards used in the subsequent multivariate analyses, and helped eliminate errors and duplicates.

Relative risks (RR) around emissions hotspots were estimated among children who moved house by comparing numbers of short range birth-to-hazard and death-to-hazard distances. The validity of this approach depends upon the premise of a short term migration equilibrium among the general child population: a premise validated and discussed previously.[1] Against such a background, an excess of short range birth addresses represents a cancer associated selection. For very short distances the birth-death ratio is an adequate estimator of the RR, but estimates within wider boundaries were based upon near/far: birth/death odds ratios (OR). Short range birth excesses are associated with a high ratio of outward to inward migrations, relative to the hazard. Outward/inward comparisons restricted to children crossing a specific circum-hazard boundary exclude many less informative cases, and the outward/inward ratio supplies an efficient alternative RR estimator.

In addition to measuring and comparing the cancer predictive strengths and effective ranges of individual emissions and installations, simultaneous examinations of pairs of exposures were carried out. They were designed to dissect mutual confounding between exposures, to separate primary from secondary associations, and to detect mutual augmentations and synergisms. They supply the main new findings and conclusions of this report. The pair examination method is illustrated in an appendix.

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-effective range ORs gave little indication of independent prediction (VOC:BZN: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.[8]

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 = 2.6:2.0 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

Table 1 Distance distributions of births and deaths relative to nearest emissions hotspots

Distances (km)	−0.3	−0.5	−1.0	−2.0	−5.0	>5.0	0–1.0*	(0.95 CI)
Dioxins (1078 map points)								
Births	123	83	182	436	1334	2967	360	
Deaths	38	33	118	383	1290	3263	161	
B/D ratio	3.24	2.52	1.54	1.14	1.03	0.91	2.24	1.85 to 2.70
1,3-butadiene (11944)								
Births	1123	284	611	930	1058	1119	1258	
Deaths	477	230	604	1042	1486	1286	551	
B/D ratio	2.35	1.23	1.01	0.89	0.71	0.87	2.28	2.06 to 2.53
Carbon monoxide (13371)								
Births	1798	308	647	743	855	774	1433	
Deaths	853	286	763	1102	1201	920	582	
B/D ratio	2.11	1.08	0.85	0.67	0.71	0.84	2.46	2.23 to 2.72
PM10 particles (39749)								
Births	1976	434	847	948	732	188	1420	
Deaths	1113	493	1008	1295	1013	203	777	
B/D ratio	1.78	0.88	0.84	0.73	0.72	0.93	1.83	1.67 to 2.00
Benzo(a)pyrene (13813)								
Births	826	242	490	586	1500	1481	909	
Deaths	563	200	411	649	1522	1780	525	
B/D ratio	1.47	1.21	1.19	0.90	0.99	0.83	1.73	1.55 to 1.94
Nitrogen oxides (53875)								
Births	2809	426	682	454	416	338	1019	
Deaths	1936	506	982	786	553	362	526	
B/D ratio	1.45	0.84	0.69	0.58	0.75	0.93	1.94	1.74 to 2.16
Non-methane-VOC (53279)								
Births	3515	338	420	260	315	277	668	
Deaths	3023	451	572	353	420	306	441	
B/D ratio	1.16	0.75	0.73	0.74	0.75	0.91	1.51	1.34 to 1.71
Benzene (59811)								
Births	3672	330	365	244	293	221	626	
Deaths	3152	446	559	310	391	267	416	
B/D ratio	1.16	0.74	0.65	0.79	0.75	0.83	1.50	1.32 to 1.71

*B/D ratios $\leqslant$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.

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 2] 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]

The public health consequences of these findings depend jointly upon birthplace RRs and upon the proportions of all children so exposed: (columns 2 and 1 of table 5). Proportions of attributable cases born within 1.0 km of a hazard ((RR−1)/RR) are shown in column 3. Products of columns 3 and 1 give population attributable risks (column 4). The 0.5 km boundary gave lesser values.

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 2 Odd ratios* for emissions-Y outside the effective ranges† of emissions-X

	Emission-X							
Emission-Y	BUT	CO	PM10	NOx	BZP	BZN	VOC	DXN
BUT	–	1.53	1.56	1.36	**2.95**	**2.38**	**2.04**	**2.64**
CO	**2.15**	–	1.90	1.38	**2.91**	**2.38**	**2.55**	**2.61**
PM10	1.71	1.40	–	*1.26*	**2.28**	1.42	1.50	**2.20**
NOx	1.59	*1.29*	1.39	–	1.90	1.37	1.32	1.94
BZP	*1.40*	*1.28*	*0.99*	*1.08*	–	1.61	1.35	1.59
BZN	*1.30*	*1.17*	*1.20*	*1.06*	1.52	–	1.21	1.55
VOC	*1.23*	*1.11*	*1.15*	*0.97*	1.45	*0.77*	–	1.48
DXN	**2.20**	*1.20*	**2.24**	*0.69*	**3.81**	*0.68*	0.87	–

*ORs>2.00 shown in bold. ORs$\leqslant$1.30 shown in italics. †Effective range taken to be 0.3 km.

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

Distances (km)	−0.3	−0.5	−1.0	−2.0	−5.0	>5.0	0.0–1.0*	(0.95 CI)
(a) Bus stations (444 map points)								
Births	217	129	378	651	1674	2076	666	
Deaths	18	43	209	635	1805	2415	212	
B/D ratio	12.06	3.00	1.81	1.03	0.93	0.86	3.14	2.69 to 3.68
(b) Hospitals (1248)								
Births	212	317	880	1331	1526	859	1149	
Deaths	85	142	549	1311	1954	1084	516	
B/D ratio	2.49	2.23	1.60	1.02	0.78	0.79	2.23	2.00 to 2.48
(c) Heavy transport (1019)								
Births	65	114	471	1090	1812	1573	568	
Deaths	41	77	266	837	2070	1834	302	
B/D ratio	1.59	1.48	1.77	1.30	0.88	0.86	1.88	1.62 to 2.16
(d) Industrial solvent use (486)								
Births	64	95	368	784	1465	2349	445	
Deaths	36	66	221	652	1528	2622	241	
B/D ratio	1.78	1.44	1.67	1.20	0.96	0.90	1.85	1.58 to 2.17
(e) Incinerators (70)								
Births	2	7	67	216	659	4174	74	
Deaths	0	10	34	169	678	4234	43	
B/D ratio	–	0.70	1.97	1.28	0.97	0.99	1.76	1.21 to 2.58
(f) Railway lines								
Births	1275	810	1141	979	638	282	1325	
Deaths	928	639	1154	1216	842	346	820	
B/D ratio	1.37	1.27	0.99	0.81	0.76	0.82	1.62	1.48 to 1.77
(g) Oil sources (storage/refinery facilities) (221)								
Births	19	14	65	222	982	3823	93	
Deaths	5	9	54	222	975	3860	63	
B/D ratio	3.80	1.56	1.20	1.00	1.01	0.99	1.48	1.07 to 2.04
(h) Motorway								
Births	167	83	249	563	1504	2559	357	
Deaths	117	122	296	558	1337	2695	393	
B/D ratio	1.43	0.68	0.84	1.01	1.12	0.95	0.91	0.78 to 1.05
(i) Combined bus stations, hospitals, railways (a+b+f)								
Births	1553	885	1179	838	493	177	1295	
Deaths	1009	738	1315	1178	646	239	740	
B/D ratio	1.54	1.20	0.90	0.71	0.76	0.74	1.75	1.59 to 1.92
(j) All ICE (a+b+c+f+h)								
Births	1658	943	1231	764	388	141	1193	
Deaths	1130	832	1356	1120	501	186	679	
B/D/ratio	1.47	1.13	0.91	0.68	0.77	0.76	1.76	1.59 to 1.94

*As in table 1.

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[8]; 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 4 Cancer associations with industrial sites (Y) and emission hotspots (X). Odds ratios* inside/outside 0.5 km† of both hazards

		X							
Y	Overall	CO	BUT	PM10	NOx	BZN	BZP	VOC	DXN
Bus stations	**6.01**	**12.42**	**12.58**	**10.36**	**9.75**	**8.68**	**4.43**	**8.36**	**13.65**
Hospitals	2.48	**5.27**	**5.31**	**4.47**	**4.41**	**3.39**	**3.38**	**3.26**	**5.10**
Oil sources	2.37	–	**4.78**	**6.23**	**5.28**	**3.81**	2.16	1.90	–
Solvents	1.59	2.71	2.85	2.29	2.30	2.29	1.85	2.06	1.59
Railways	1.56	**3.29**	**3.41**	2.84	2.49	1.98	2.31	1.92	**3.22**
Transport	1.55	2.40	2.60	2.26	2.48	2.29	1.77	2.02	**4.90**
Incinerators	1.74	2.52	2.57	2.73	2.51	2.21	1.63	2.24	2.88
HosBusRail	1.76	**3.54**	**3.70**	**3.05**	2.65	2.12	2.57	2.06	**3.65**
All ICE	1.66	**3.24**	**3.24**	2.80	2.46	2.01	2.46	1.95	**3.59**
Overall		2.44	2.37	1.94	1.88	1.54	1.50	1.44	2.14

*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 foot 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.

Table 5 Attributable proportions and risks for different exposures. Proportions (%) of all cancer births*, and migrant RRs inside 1.0 km

Hazard	Proportion all births	Migrant RR	Attributable proportions†	Attributable population risk per cent
Bus stations	9.85	3.14	68.15	6.71
Hospitals	21.38	2.23	55.16	11.79
Railways	58.35	1.80	44.44	25.93
HosBusRail	65.72	1.75	42.86	28.16
ICE	70.09	1.76	43.18	30.27
CO	45.21	2.46	59.35	26.83
BUT	32.55	2.28	56.14	18.27
PM10	58.35	1.83	45.36	26.46
NOx	71.38	1.94	48.45	34.59
BZN	83.33	1.50	33.33	27.78
BZP	26.89	1.73	42.20	11.35
VOC	81.43	1.51	33.78	27.50

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

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.[10] 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).[11] A similar diffusion model, used in Italy, yielded a significant leukaemia RR of 3.91.[12] A Swedish study based upon NOx concentrations gave a significant child cancer RR of 3.8.[13] Other case-control reports based on smaller numbers have led to ''suggestive but inconclusive'' results;[14 15] and yet others to ''no evidence of'' conclusions.[16 17] 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.[18]

Key points

Childhood cancer birthplaces are closely associated with atmospheric emissions from oil based combustion processes and volatiles; and with installations that generate these emissions such as bus/coach terminals, hospitals, motorways, railways, and foci of heavy road transport. There is a specific association with engine exhausts, especially diesel exhausts, and the chief carcinogenic agent is probably 1,3-butadiene.

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.

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.[19] 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,[8] 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.[20]

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 work situations[21]; but this is clearly not sufficient in the context of fetus/child exposure. Control and monitoring measures must be directed towards the sources.

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APPENDIX

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)

		Birth addresses Hazard-X				Death addresses Hazard-X	
		Near	Far			Near	Far
Hazard-Y	Near	a	b	Hazard-Y	Near	e	f
	Far	c	d		Far	g	h
Example							
		Bus station			Bus station		
		⩽0.5	>0.5			⩽0.5	>0.5
BUT	⩽0.5	184	1224	BUT	⩽0.5	18	689
	>0.5	162	3558		>0.5	43	4378

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

	Example	
	OR	**(CI)***
1 OR for nearY in absence of nearX = bh/df	2.19	(1.97 to 2.42)
2 OR for nearY in presence of nearX = ag/ce	2.71	(1.51 to 4.89)
3 OR for nearX in absence of nearY = ch/dg	4.64	(3.30 to 6.51)
4 OR for nearX in presence of nearY = af/be	5.75	(3.52 to 9.42)
5 OR for nearY+nearX v neither = ah/de	12.58	(7.74 to 20.45)
6 OR for nearY overall = {(a+b)(g+h)}/{(c+d)(e+f)}	2.37	(2.14 to 2.62)
7 OR for nearX overall = {(a+c)(f+h)}/{(b+d)(e+g)}	6.01	(4.56 to 7.91)

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.

*Confidence intervals calculated as suggested by Bland and Altman.[22]

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