

Analysis of incidence of childhood cancer in the West Midlands of the United Kingdom in relation to proximity to main roads and petrol stations

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Abstract

Objectives—To investigate whether there is an excess of leukaemias in 0–15 year old children among those living in close proximity (within 100 m) of a main road or petrol station.

Methods—Data for 0–15 year old children diagnosed between 1990 and 1994 in the United Kingdom West Midlands were used. Postcode addresses were used to locate the point of residence which was compared with proximity to main roads and petrol stations separately, and to both together. Odds ratios (ORs) were calculated with solid tumours as a control, and incidence ratios (IRs) with population density as a control.

Results—The method based on solid tumours as a control showed ORs of 1.61 (95% confidence interval (95% CI) 0.90 to 2.87) and 1.99 (95% CI 0.73 to 5.43), for those living within 100 m of a main road or petrol station respectively. When population was used as a control, the estimated IRs for leukaemia were 1.16 (95% CI 0.74 to 1.72) and 1.48 (95% CI 0.65 to 2.93) for residence within 100 m of a main road or petrol station respectively, but neither reached significance at the 95% level. Results for residence in close proximity to both a main road and petrol station were inconsistent, but there were few. The influence of socioeconomic factors as represented by the Townsend deprivation index on leukaemia incidence was not significant and the results were not explicable on the basis of impact of social class.

Conclusions—The results are suggestive of a small increase in risk of childhood leukaemia, but not solid tumours, for those living in close proximity to a main road or petrol station. This increase in risk is not, however, significant and a larger study is warranted to establish the true risk and causes of any increase in risk.

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It is now accepted that occupational exposure to high concentrations of benzene can lead to an excess of non-lymphocytic leukaemias.¹ For example, workers exposed to in excess of 400 000 ppb-years benzene in the Goodyear Pliofilm plant showed a standardised mortality

ratio (SMR) of 6.64 (95% confidence interval (95% CI) 1.33 to 19.39).² Despite the fact that most leukaemias in children are of the acute lymphocytic variety, a type not associated with occupational benzene exposure, there have been suggestions that environmental exposure to benzene vapour may be a cause of childhood leukaemia. Wolff³ showed significant correlations between acute myeloid leukaemia, acute lymphoblastic leukaemia, chronic lymphoid leukaemia, low grade non-Hodgkin's lymphoma, and all lymphoproliferative disease and the number of cars per household in each United Kingdom county. Wolff³ postulated that benzene exposure within the car might be responsible for these correlations. In a more complex study, Alexander *et al*⁴ estimated relative risks (RRs) for childhood acute lymphoblastic leukaemia by Poisson regression for groups of electoral wards classified by isolation and car ownership. Multivariate analyses include adjustment for socioeconomic status. No evidence of increased risk of childhood acute lymphoblastic leukaemia was found in areas where more households own cars. It was concluded that the previous explanation for the small area variation of childhood acute lymphoblastic leukaemia in terms of geographical isolation and interpretation in terms of exposure to common infections continued to be justified.

Other geographical analyses of risk of childhood cancer have looked for evidence of involvement of an infectious process^{5,6} or for an association of high risk with proximity to either nuclear or industrial installations.^{7–9} Apart from the well known excess of childhood leukaemias around the Sellafield nuclear reprocessing plant, none of the nuclear sites examined in England and Wales showed evidence of an increased risk of childhood leukaemias and non-Hodgkin's lymphomas in the area covered by a 25 km circle around each plant.⁸ Knox and Gilman⁹ found an association of increased risk of childhood leukaemias and solid cancers with proximity to a range of industrial sites. Relative excesses were consistently associated with proximity to sites producing two main types of industrial atmospheric effluent: (a) volatiles derived from petroleum, and (b) kiln and furnace smoke and gases, and effluents from internal combustion engines. However, no evidence was found of an association with proximity to benzene works.

Much is known about personal exposure to benzene. In the United States the total exposure assessment methodology (TEAM) study^{10,11} measured the sources of personal

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exposure, showing a wide range of sources including active and passive smoking, personal activities—such as use of paints, solvents, other consumer products, and refuelling the car, as well as breathing outdoor air and air in the car. Personal exposures generally exceeded, but were correlated with concentrations in outdoor air. In a study in the United Kingdom, Leung and Harrison¹² carried out both direct measurement and modelling of personal exposure to benzene and other monoaromatic hydrocarbons. The results showed that although some microenvironments—such as adjacent to petrol pumps when refuelling the car—showed highly increased concentrations, integrated personal exposure was influenced far more by microenvironments in which the subjects spent large periods, the home being the most important, with the workplace also being significant for those who worked outside the home. Measurements of the spatial distribution of benzene in the atmosphere have shown that the highest outdoor concentrations within urban areas tend to occur adjacent to main roads,¹³ or in the vicinity of petrol stations.¹⁴ The influence of such ground level sources usually extends for about 100 m before concentrations fall to the local background.¹⁵ This result derives from several monitoring and modelling studies of traffic generated pollutants in open country. The dispersion of pollutants in urban terrain is more complex, but none the less the same general guidelines can be expected to apply. Concentrations within houses are strongly influenced by those out of doors, and hence the occupants of homes located within 100 m of a main highway or petrol station would be expected to experience higher exposures to benzene and related hydrocarbons than occupants of comparable homes at greater distances from such sources.

As the epidemiological studies already referred to have not given a wholly consistent answer to the proposition that benzene exposure may influence incidence of childhood leukaemia, and some have been criticised on methodological grounds, it was thought appropriate to conduct a further analysis of incidence of childhood cancer relative to sources of hydrocarbons. In our study, residence within 100 m of a main highway or petrol station was taken as a surrogate for increased exposure to benzene and other hydrocarbons present in evaporative losses of petrol, or to exhaust emissions from road traffic.

Methods

CASES OF LEUKAEMIA AND SOLID CANCER

Data from the West Midlands Cancer Intelligence Unit for 0–15 year old children diagnosed between 1990 and 1994 were used. Cases were taken from the Birmingham, Walsall, Solihull, Dudley, Wolverhampton, and Sandwell District Health Authorities (total population 2 251 000) as full data on petrol stations were available only for these areas. Classifications from the ninth revision of the international classification of diseases (ICD-9) were included for lymphoid, myeloid, monocytic, and unspecified leukaemia, including

those of uncertain behaviour, and for solid cancers and benign neoplasms. In the United Kingdom all cancer registries, together with the Office for National Statistics use the ICD for both childhood and adult tumours to be consistent across databases rather than the international CCC 1996 coding classification.

GEOREFERENCING DATA USING THE UNIT POSTCODE AND CENSUS GEOGRAPHY

The areas of enumeration districts (EDs), wards and health authorities are used in the study for aggregation of data. The EDs are the smallest areas that can be used to use population census data. Wards are constructed from EDs and in the West Midlands have an average population of 15 600. Health authorities are constructed from wards and form the administrative areas by which health services are organised in the United Kingdom. To integrate data into a geographic information system, both the petrol stations and the cancer cases and controls were allocated a grid reference using their unit postcode. This was accomplished with a table called the Central Postcode Directory (CPD). This consists of all the postcodes in the West Midlands and their grid references. The postcodes from the cancer registration database are matched to identical entries in the CPD. A unit postcode (the full seven or eight digit version—for example, B15 2TT) represents about 13 household children in the West Midlands. Grid references were allocated to unit postcodes in the following way. A 100 m² grid is used to identify in which grid square the first household of a unit postcode is located. This unit postcode is then assigned the grid reference of the south west corner of the grid square. This means that all postcodes whose first house is situated in a particular grid square are allocated the same grid reference. If a postcode happens to be towards the north west of the grid square then the reference will be inaccurate. This inaccuracy was offset by adding 50 m to the easting and northing of the grid reference as recommended by Gatrell *et al.*¹⁶ and used in a study of cancer around a TV transmitter mast by Dolk *et al.*¹⁷

Full postal addresses of petrol stations operational in 1997 within the West Midlands were purchased from Thames Communications (Gravesend, Kent). The local authority planning departments provided information on the closure and construction of petrol stations during the period 1990–7, which allowed development of a database containing addresses of stations fully operational during the years 1990–4 inclusive. Each petrol station was then allocated a grid reference with Matchcode (a software package developed by Capscan, London). Matchcode can improve address data by adding a postcode if one is missing or improving the address if it is slightly inaccurate. At the same time it can add a grid reference to an address. This is done by the software referring to a CD ROM that contains all the addresses in the United Kingdom. This is called the Post Office address file (PAF) and is what the CPD is derived from. After the post-

code was allocated to the stations with Match-code, the 50 m adjustment was made.

The CPD is provided free to NHS organisations and Universities and is often used in geographical studies as a method of grid referencing postcodes. Resources are available that allow users to grid reference a postcode to an claimed accuracy of 0.1 m (ordnance survey address point product), but the expense of this product prohibits its use in the NHS and academia.

ROADS OF HIGH TRAFFIC DENSITY

Traffic flow data for the West Midlands was obtained from the Joint Data Team, Birmingham. This exists as counts of vehicles along links of road throughout the West Midlands. The data pertaining to the roads used in this study were extracted and average traffic flows calculated.

The Ordnance Survey dataset STRATEGI was used as a source for the digital road network. The following road categories were chosen as being of high traffic density: motorways, primary routes (dual and single carriageways), and single and dual carriageway A roads. In all, this selected 755 sections of road with mean traffic flow 23 400 vehicles a day (10% percentile 11 500; 90% percentile 38 800) as derived from the Joint Data Team statistics for the individual road links.

DATA ANALYSIS

Spatial analysis was performed with a geographic information system (ArcView V2.1). Other manipulation of data was carried out with the software package Microsoft Excel. To investigate prevalence of childhood leukaemia relative to locality of a petrol station and proximity of main roads, the geographic information system was used to perform two different kinds of spatial analysis with different control groups.

In the first kind of analysis, the spatial distribution of leukaemias was analysed with solid tumour cases as a control. Leukaemia and control cases were plotted (with grid references

obtained from addresses at the time of diagnosis) and a spatial query was then performed to determine the number of cases and controls within a 100 m radius of a petrol station or a zone 100 m from a main road. In the second kind of analysis, leukaemia cases, petrol stations, and main roads were again entered into the GIS. The number of new leukaemia cases within 100 m of a source was again evaluated, but further separated according to the District Health Authority area. To include the population data as a control, the method of Dolk *et al*¹⁷ was adopted. To calculate the expected number of leukaemia cases within 100 m of a possible source, a population for such an area has to be calculated. Actual population counts do not exist for small user defined areas so they have to be estimated. The Postcode to Enumeration District Directory from the Office of National Statistics was used which contains the number of household children in each unit postcode. With census information for the ED that the postcode falls in, an average number of people per household can be calculated. From this we calculate the number of people per postcode. Finally with ED census information the number of 0–15 year old children can be estimated.

Townsend deprivation indices were available for electoral wards from the NHS Executive West Midlands Regional Office. To evaluate any influence of socioeconomic factors, the leukaemia incidence was plotted against the Townsend score for each ward. The Townsend index¹⁸ is a measure of multiple deprivation calculated from census variables. High scores reflect severe deprivation. Some other researchers in the United Kingdom have used the Carstairs index derived from Scottish data, whereas the Townsend index is derived from English data. Both indices have four census variables, three of which are common to both, and we would not anticipate any main differences from the use of the Carstairs index.

STATISTICAL METHODS

Two different approaches were used to investigate whether an excess incidence of leukaemia was present around main roads or petrol stations. In the first method odds ratios (ORs) were calculated with a case-control method. Each subject was classified as positive or negative according to their proximity to a main road or petrol station and defined as a case if they had been diagnosed with leukaemia or a control if the diagnosis was of a solid tumour. The OR was then calculated according to equation (1) where A were patients with leukaemia living within 100 m of a road or petrol station; B were patients with solid tumours living within 100 m of a road or petrol station; C were patients with leukaemia living away from a road or petrol station; and D were patients with solid tumours living away from a road or petrol station.

$$OR = AD/BC \quad (1)$$

The 95% confidence interval (95% CI) for the OR was calculated from

Table 1 Population (0–15 years old) within each district health authority and estimated to live within 100 m of source

	Birmingham	Dudley	Sandwell	Solihull	Walsall	Wolverhampton
Total population in age range	217583	59297	59665	40263	54610	51170
Population living <100 m from petrol station	8760	2118	2739	760	3102	2221
Population living <100 m from major road	33226	9425	10137	3792	8958	8561
Population living <100 m from petrol station and road	5955	1332	1994	300	1900	1575

Table 2 Numbers of leukaemias and solid cancers relative to location of residence, and calculated odds ratios (ORs)

Location of residence	Leukaemias	Solid cancers (controls)	OR	95% CI
Major road < 100 m	24	31	1.61	0.90 to 2.87
Major road > 100 m	106	220		
Petrol station < 100 m	8	8	1.99	0.73 to 5.43
Petrol station > 100 m	122	243		
Major road and petrol station < 100 m	3	1	5.91	0.61 to 57.3
Major road and petrol station > 100 m	127	250		
Total cases/controls	130	251		

Table 3 Incidence ratio (IR (95% CI)) of childhood leukaemia if living <100 m from source

	Petrol station				Roads of high traffic density				Petrol station and roads			
	Obs	Exp	IR	95% CI	Obs	Exp	IR	95% CI	Obs	Exp	IR	95% CI
Birmingham	4	2.6	1.54	0.42 to 3.92	14	9.8	1.43	0.79 to 2.40	1	1.8	0.56	0 to 3.22
Dudley	1	0.6	1.67	0 to 9.33	4	2.6	1.54	0.42 to 3.92	1	0.4	2.50	0 to 14.5
Sandwell	1	0.6	1.67	0 to 9.33	3	2.2	1.36	0.27 to 4.00	1	0.4	2.50	0 to 14.5
Solihull	0	0.2	—	—	0	0.8	—	—	0	0.1	—	—
Walsall	1	0.9	1.11	0 to 6.22	1	2.6	0.38	0 to 2.23	0	0.6	—	—
Wolverhampton	1	0.5	2.00	0 to 11.2	2	2.7	0.74	0.07 to 2.67	0	0.4	—	—
All districts	8	5.4	1.48	0.65 to 2.93	24	20.7	1.16	0.74 to 1.72	3	3.7	0.81	0.16 to 2.38

$$95\% \text{ CI} = \exp[\ln \text{RR} \pm 1.96(1/A + 1/B + 1/C + 1/D)] \quad (2)$$

An OR of 1.0 would indicate that there is no effect on incidence of childhood leukaemia of living in close proximity to main roads or petrol stations.

The second method used indirect age and sex standardisation to examine the observed number of cases of leukaemia around main roads and petrol stations compared with what would be expected for the population of the health authority as a whole. An incidence ratio (IR) of 1.0 would indicate that there is no difference between the observed number of cases in the exposed group and that which would be expected from the comparison population. We have chosen not to use the term standardised IR as full age distributions were not known and therefore full standardisation has not been carried out.

To estimate the IR, the number of leukaemias in the 0–15 age group expected within a region within 100 m for a petrol station or main road was first calculated from equation (3)

$$E_{\text{pet/road}} = (C_A/P_A)P_{\text{pet/road}} \quad (3)$$

where $E_{\text{pet/road}}$ = expected number of cases, C_A = total number of cases in District Health Authority A (0–15 year age group), P_A = total population of 0–15 year old children in District Health Authority A, $P_{\text{pet/road}}$ = population of 0–15 year old children residing within 100 m of petrol station or road.

The IR was then calculated from

$$\text{IR}_{\text{pet/road}} = O_{\text{pet/road}}/E_{\text{pet/road}} \quad (4)$$

where $O_{\text{pet/road}}$ = observed number of leukaemia cases within 100 m of a petrol station or road.

Due to the small number of cases, the 95% confidence interval on the observed number of cases was read directly from standard statistical tables applying to the Poisson distribution. These were used due to the non-integer nature of the expected values.

Additionally, the Poisson probability of from 0 to (r–1) cases of disease was calculated, where r is the number of observed cases, and λ is the expected number. Probability (n cases) = $e^{-\lambda} \cdot \lambda^n/n!$

These probabilities were summed from n=0 to n=r–1 and subtracted from 1.0 to give the probability of $\geq r$ events occurring.

Results

The study was limited to 0–15 year old children for several reasons. Most important is the relatively short time interval between disease induction and diagnosis which makes spatial analysis more realistic than for adult

cases. Secondly, important factors which can confound the analysis of adult cancer data are not present for children. Most important among these are smoking and occupational exposure to carcinogens. Childhood cases of cancer are also more accurately diagnosed and more completely registered than adult cancers.¹⁹

Table 1 shows basic data relating to the populations within the six district health authorities which were included in this study. The area is densely populated and as many as 17% of the population were classified as residing within 100 m of a main road within some health authority areas. Table 2 shows the number of cases and controls according to location of residence when solid tumours were used as controls. The table also shows the ORs and 95% CIs for leukaemia relative to solid cancer. While we were conducting this study, the work of Knox and Gilman⁹ showed that both leukaemias and solid tumours had similar excesses relative to the sources examined in that work. It was therefore thought prudent to use some other form of analysis, and with the procedures outlined above, estimated populations of 0–15 year old children were used to calculate expected numbers of cases of leukaemia. The results of this study expressed as IRs appear in table 3. This procedure was carried out also for solid tumours, showing for all districts combined IR values of 0.77, 0.8, and 0.24 for residences close to petrol stations, main roads, and the two jointly, respectively. None of these was significantly different from 1.0 at the 95% level.

Discussion

Both methods show an increased risk of childhood leukaemia for those living within either 100 m of a petrol station, or within 100 m of a main road. For the case-control method, standard 95% CIs appear in table 2. For the IR method, 95% confidence limits on the observed value were taken from statistical tables for the Poisson distribution, and used to calculate a 95% CI on the IR. Probabilities were calculated for the combined health authorities for proximity to a main road (p=0.28) and proximity to a petrol station (p=0.18). In neither instance, therefore, is the result significant at the 95% level although the data for all district health authorities together approach significance for the case-control method. This finding is broadly consistent with a study of the incidence of leukaemia and lymphoma in young people (0–24 years) within 3 km of the petrochemical plant at

Baglan Bay, South Wales.²⁰ By contrast, a study in Britain by Knox and Gilman⁹ found excesses near to sites producing two main types of industrial atmospheric effluent (petroleum derived volatiles; and kiln and furnace smoke and gases, and effluents from internal combustion engines), but a non-significant excess of childhood leukaemias and solid cancers near to petrochemical works, and no association of increased risk with proximity to benzene installations. Reasons for the differences in findings between studies may be related to problems obtaining accurate population denominators on which to base calculations of expected numbers or in the definition of exposed populations. The study in South Wales took as its exposed population those resident within EDs, wholly or partially within concentric circles of 1.5 or 3 km radius of the centre of works. This included areas outside these zones, and took no account of the wind direction frequencies and pattern of dispersion of gaseous effluents from the petrochemical plant. Because of the long period covered by cases used in the Knox and Gilman study (1953–80), census-based population estimates could not be used, and would not have been available at the fine spatial resolution required; instead a method based on postcodes was used.⁹ This could have created a false apparent excess of cases near to industrial sources if postcodes close to those sources also systematically had higher densities of children living in them than did postcodes which were distant from industrial sources. Aware of this problem, the authors investigated the distribution of postcodes. Areas nearest to industrial sources had a lower density of postcodes per kilometre than other locations, indicating that fewer people lived in these postcodes, and hence that fewer cases would be expected in postcodes near to industrial locations. This, together with the consistency in the nature of the sites around which excesses were found, makes it unlikely that their findings were due to an artefact. None the less, some of the conclusions of Knox and Gilman are surprising. In particular, the finding of a relative excess of solid cancers and leukaemias within 4 km of a motorway, with a significant deficit beyond 4 km, is hard to reconcile with the fact that the impact of main roads on air quality is not measurable above the local background air pollution at distances beyond about 100–200 m.¹⁵ Our study was not without its problems, the population data for the 0–15 year age group was inferred from information for EDs which are considerably larger than postcode districts (by a factor of about 16). It was because of the uncertainties in defining exactly the population numbers in the exposed and control groups that we thought the case-control method would provide valuable supporting information. Another limitation of our study is the lack of age and sex standardisation. This is a problem for childhood cancers as previous studies have shown a slight increase of incidence for males relative to females (1.3:1 male to female ratio) and for the under 5s.¹⁹ Although main geographic

variations in the age and sex distribution of the 0–15 year old children within the West Midlands are unlikely, the lack of discrimination by age and sex may obscure aetiological insights which could be gained from a more refined analysis.

There is little published evidence to suggest that pollutant emissions from road traffic or petrol stations are connected with solid tumours in the general population, excluding occupationally exposed groups. Occupational exposure to polycyclic aromatic hydrocarbons is linked with an excess risk of lung cancer. As road vehicle emissions contain polycyclic aromatic hydrocarbons, adults residing alongside main roads may run a slightly increased risk of lung cancer from this source. This condition, however, represents only a tiny proportion of cancers in the 0–15 year age band for whom the brain and spinal cord are the main loci of solid tumours.²¹ Exposure to gasoline vapour in adult workers has been associated with an increase in kidney cancer,²² but this is also a rare condition in children. We therefore think it likely that solid cancers represent a good control for this study and that the ORs presented in table 2 represent a genuine representation of increased risk. The IRs for solid tumours show no excess risk and support this view. The combined use of the two complementary methods of data analysis in our view adds to the confidence with which the results can be viewed.

A plot of incidence of childhood leukaemia in 1000 children by electoral ward versus Townsend deprivation index shows a poor correlation ($r^2=0.04$; $n=130$), but there does seem to be a slight decrease in incidence of leukaemia with increased deprivation (higher Townsend index) as reported in previous studies.²³ In the West Midlands Health Region the more deprived wards tend to be sited in the central conurbation, whereas the more affluent wards tend to be in the rural shire counties. Thus, this finding is consistent with earlier findings of higher incidence of childhood leukaemia associated with lower population densities. It does not indicate any role for exposure to benzene from passive smoking in the aetiology of childhood leukaemia as smoking is more prevalent in homes of lower socioeconomic status (higher Townsend scores) and would produce the opposite slope to that found.

One question rarely, if ever, considered in the spatial analysis of disease in relation to pollution sources is the magnitude of exposure. Thus, many studies have related incidence of disease to distance from source without regard to the spatial distribution of pollutant concentrations. In this work the use of a criterion of 100 m separation between source and point of residence (as indicated by postcode) is consistent with knowledge of the range over which air pollution from a ground level source sustains concentrations above the local background. It is, however, extremely difficult to estimate the increased magnitude of exposure for those living within the 100 m band. The work of Leung and Harrison¹² shows that exposure within the

home is likely to be the main contributor to exposure to aromatic hydrocarbons, including benzene. The extent of exposure arising from the proximity of the road is very hard to estimate, but some indications can be gained from the work of Leung and Harrison.¹³ This work showed that roadside concentrations of aromatic hydrocarbons, including benzene, were highly variable depending on the sampling site. In particular, the openness of the site, and therefore the ease of pollutant dispersion, has a major influence on atmospheric concentrations. Taking the mean of 53 samples collected alongside six main roads in Birmingham indicates a typical ratio of roadside to urban background concentration of benzene of roughly 5, and for toluene roughly 2.5. Allowing for the fact that indoor concentrations often slightly exceed those out of doors due to indoor sources, and the slightly greater distance of most homes from the traffic than the roadside samplers used in the work of Leung and Harrison,¹³ it is likely that the exposure to benzene for those living within 100 m of a busy road is around twice that of those living at greater distance from the road. Long term average concentrations of benzene in the vicinity of petrol stations are broadly similar to those at the roadside, and therefore a similar magnitude of increase might be expected.

Petrol stations are unlikely to generate sufficient traffic on their own to cause a substantial increase in pollutants from vehicle exhaust. It might be expected that petrol stations would be located mostly on main roads, and the data in table 1 indicate that this is the case for >50% of the exposed population. The data for populations living within 100 m of a petrol station does, however, include a substantial additional population and justifies the separate treatment of this category of exposure. Petrol stations are most notable as a source of evaporated hydrocarbons from petrol. On the other hand, main roads are the source of a wider range of pollutants including nitrogen oxides and polycyclic aromatic hydrocarbons as well as volatile hydrocarbons such as benzene. If our finding of a higher RR associated with proximity to a petrol station than to a main road were borne out by a study of higher statistical power, it would be strongly suggestive of an influence of hydrocarbons upon childhood cancer.

The results of this study are broadly consistent with those of other published work. Savitz and Feingold²⁴ reported an OR of 1.7 (95% CI 1.0 to 2.8) for cancer among children exposed to more than 500 vehicles a day. They reported ORs also for leukaemia of 2.1 (95% CI 1.1 to 4.0) and brain tumours of 1.7 (95% CI 0.8 to 3.9). Their results showed a dose-response gradient with traffic in excess of 10 000 vehicles a day giving an OR of 3.1 (95% CI 1.2 to 8.0) for total cancer and of 4.7 (95% CI 1.6 to 13.5) for leukaemia. Feychting *et al*²⁵ used calculated nitrogen dioxide concentrations as an estimate of exposure to traffic pollution and found a relation between RR of cancer in 0–15 year old children relative to exposure to nitrogen dioxide. With those exposed to a 99 percentile of hourly average concentrations

over 1 year of nitrogen dioxide of >49 $\mu\text{g m}^{-3}$ defined as an RR of 1, those exposed to concentrations in the 50–79 $\mu\text{g m}^{-3}$ range were subject to an RR of 1.9 (95% CI 0.8 to 4.5), and for a concentration of $\geq 80 \mu\text{g m}^{-3}$ the RR was 3.8 (95% CI 1.2 to 12.1). Increased but imprecise risk estimates were found for leukaemia and central nervous system tumours. Background concentrations of nitrogen dioxide in the United Kingdom West Midlands conurbation lie within the highest band (99 percentile $\geq 80 \mu\text{g.m}^{-3}$) used in this study. These workers²⁵ also used data published by Wertheimer and Leeper²⁶ to calculate the association between traffic density and childhood cancer from a study designed to investigate the association between exposure to electromagnetic fields and childhood cancer where traffic density was included for control of confounding. In this study exposure to heavy traffic was defined as homes within 40 m of road with a daily traffic count of ≤ 5000 . The effect of thus defined heavy traffic on total cancer mortality in children was calculated as an OR of 1.6 (95% CI 1.1 to 2.3). Information for specific cancer sites was not provided. The results of these studies both for total childhood cancer and for leukaemias are broadly consistent with the result of our own research strongly suggesting that although the results are just below the level of significance normally required, they are reflective of a true effect of road traffic on cancer in children.

Conclusions

The total population of the area studied is 2.25 million and the population of 0–15 year old children included in the data analysis is 482 588. The study included all diagnosed cases of leukaemia in the 0–15 year age group over a period of 5 years. The results suggest a slight increase of risk of leukaemia for those 0–15 year old children living in close proximity to a main road or petrol station, although in no case do the results reach significance. The two methods used based on use of solid tumours and population numbers as controls give broadly similar results except in the case of proximity to both main road and petrol station where the number of cases is very small and the results have very wide 95% CIs. Exposure of the population within 100 m of source is likely to be of the order of double that of other dwellers within the conurbation not living in such high proximity. In the case of petrol stations, increased exposures are primarily to hydrocarbons, including benzene, whereas in the case of main roads, pollutants include combustion products such as NO_x and polycyclic aromatic hydrocarbons as well as volatile hydrocarbons such as benzene. The fact that increased risks are found at both kinds of site (although not significant) is suggestive of volatile hydrocarbons as a causal agent. Although the results of the study are suggestive of a possible slight association of exposure to pollutant (not necessarily benzene) with increased incidence of leukaemia, the data are overall rather reassuring in showing that any such effect, if real, is likely to be small in magnitude. A larger

study is warranted to establish whether there is truly an increase in risk for those living in close proximity to roads and petrol stations and to determine the causes.

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