Do people living near inner city main roads have more asthma needing treatment? Case-control study

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Hospital admissions for asthma in east London are 80% above the national rates. This may reflect the high incidence of acute asthma. Recent reports of a higher prevalence of wheeze^{1,2} or hospital admissions in children in association with traffic flow or proximity of residence to roads³ have highlighted concerns about the possible health effects of road traffic in the London Borough of Tower Hamlets.

In each of two computerised general practices in Tower Hamlets around 20% of the population have received computer prescriptions for bronchodilators, inhaled steroids, or inhaled anti-inflammatory drugs since 1990. The diagnostic computer coding for asthma showed a prevalence of treated asthma of 9% in one practice and 17% in the other (unpublished observation). We examined whether the proximity of residence to main roads was associated with these high prescribing rates for asthma in the two inner city practices.

Subjects, methods, and results

This case-control study took place in June 1994 in two adjacent general practices located near major roads; they have a combined list of 15 300 patients and 10 principals. Since 1990 both practices have routinely used EMIS (Egton Medical Information Systems) consulting room computers. We included all eligible

patients aged 2 to 64 who had had computer consultations in the preceding year. Cases had a computer record of prescriptions for asthma drugs in the previous year and a computer diagnosis of asthma. Control subjects had no computer record of asthma drugs and no recorded diagnosis of asthma. We collected data on age, sex, practice, smoking, and residential postcode from the notes. Data were linked by postcode to information on deprivation from the 1991 census and using the ARC geographic information system, with the shortest distance of residential postcode to a busy road carrying more than 1000 vehicles an hour at peak times. Postcode grid references within 10 m were available for 92% of cases (978) and 91% of controls (5685). The remainder were within 100 m.

Table 1 summarises baseline characteristics and results. Overall, 51% of subjects (3172/7299) in the study came from practice 2, whose patient population made up 43% of the whole (6558/15 393 registered patients). Most cases and controls lived in districts rated to be in the bottom fifth of the Carstairs deprivation scores (98% (1047/1066) v 97% (6032/6233)). Cases tended to be younger and male, and proportionately more of the adult cases were current smokers. Smoking habit did not differ significantly in relation to distance of home from roads.

In children under 16 the unadjusted odds ratio for being treated for asthma when living 150 m or less from busy roads compared with more than 150 m from them was 0.94 (95% confidence interval 0.75 to 1.19); in adults the odds ratio was 0.81 (0.68 to 0.97) (data not shown). The odds ratios did not differ significantly from unity after adjustment for age, age group (in adults) sex, and practice (table 1). Point process methods to analyse the effect of living at different distances from roads also showed no evidence of spatial clustering.⁵

Comment

This study shows no increase in risk of asthma with living close to busy roads. Although table 1 shows analyses based on residence 150 m or less from busy roads or more than 150 m away from them, we also examined risk with distance measured as a continuous variable; spatial point process methods fit an exponential function for the decline in risk with distance.⁵ With this regression approach there was also no evidence of spatial clustering of cases of asthma near roads when we adjusted for age, sex, and smoking habit. These negative findings are consistent with an ecological study of the prevalence of wheeze near the M25 motorway, but they contradict other reports.¹⁻³ The use of routine data from general practice computers allowed basic covariates to be adjusted in a way that is impossible when using many routine data sources. The definition of cases and controls made misclassification between them unlikely. Possibly, some true cases were missed because of the requirement for computer recording of both treatment and asthma diagnosis. There is no reason to suspect that this was a source of bias. On the other hand, distance of residence from a busy road is a crude proxy for exposure to traffic related pollution, especially among adults for whom work and commuting patterns may be more relevant. Misclassification of exposure is therefore inevitable.

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Table 1—Details of cases with active asthma and controls. Values are numbers (percentages) of patients unless stated otherwise

Covariate	Cases (n=1066)	Controls (n=6233)	χ^2 Statistics or adjusted odds ratio (95% confidence interval)
Practice:			
1	416 (39)	3171 (51)	
2	650 (61)	3062 (49)	$\chi^2=51.5$; $P<0.001$
Sex:			
Male	513 (48)	2750 (44)	
Female	553 (52)	3483 (56)	$\chi^2=5.7$; $P=0.016$
Age (years):			
2-15	436 (41)	1138 (18)	
16-40	358 (34)	3349 (54)	
41-64	272 (26)	1746 (28)	$\chi^2=291$; $df=2$; $P<0.001$
Smoking (adults ≥ 16):			
Never smoked	199 (32)	1763 (35)	
Ex-smoker or current non-smoker	82 (13)	450 (9)	
Current smoker	314 (50)	2064 (41)	
No record	35 (6)	818 (16)	$\chi^2=65.3$; $df=3$; $P<0.001$
Deprivation:			
Median enumeration district Carstairs score (interquartile range)	6.5 (4.9-8.8)	5.8 (4.5-8.0)	
Home postcode distance from busy roads:			
Median distance (interquartile range) (m):			
Age 2-15	191 (116-282)	190 (114-315)	
Age 16-64	191 (116-330)	178 (100-318)	
Age 2-15:			
≤ 150 m	172 (39)	464 (41)	
> 150 m	264 (61)	674 (59)	0.96 (0.78 to 1.22)*
Age 16-64:			
≤ 150 m	237 (38)	2165 (43)	
> 150 m	393 (62)	2930 (58)	1.00 (0.84 to 1.19)†

*Adjusted for sex and practice.

†Adjusted for sex, practice, and age.

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Commentary: Traffic exposure and asthma: problems of interpretation

David P Strachan

Neither air pollution nor asthma is a new problem, but in the past decade there has been increasing concern that the apparent increase in the prevalence of asthma may be due to increasing exposure to pollution, particularly from motor vehicles.¹

Vehicles emit many pollutants, but in relation to asthma interest has focused on particulate matter and nitrogen oxides (mainly nitrogen monoxide). Nitrogen monoxide is rapidly oxidised to nitrogen dioxide, which in turn, and at a distance, catalyses the formation of ozone. Proximity to major roads or estimates of local traffic density have been used as indicators of exposure to vehicle exhaust fumes in several recent epidemiological studies, including that by Livingstone and colleagues.² The relation of traffic exposure to pollutant exposure, however, is far from straightforward, and this complicates the interpretation of both positive^{3,4} and negative² findings.

Although mean nitrogen oxide and particulate concentrations decrease with distance from the kerb,³ the decline beyond 20 m is small. No correlation between traffic density and ambient nitrogen dioxide concentrations was found in Munich,⁴ although carbon monoxide, benzene, and toluene (pollutants related to vehicles that are not implicated in asthma) were more concentrated in areas of higher traffic flow. Higher traffic density was inversely correlated with concentrations of ozone,⁴ which is formed at some distance

from emission sources and scavenged in city centres by nitrogen monoxide from vehicles.

Hitherto, epidemiological studies have imputed traffic exposure from place of residence. Yet, for many people the environment close to home represents only a small part of their daily exposure to outdoor air. Furthermore, indoor sources (particularly cooking fuels and environmental tobacco smoke) are more important influences on personal exposure to both nitrogen oxides and airborne particulates. Only a small increase in personal exposure to these pollutants was discernible for subjects living close to major roads in Tokyo.⁵

When significant associations between recurrent wheezing and traffic exposure have been reported—for example, among Japanese women³ and German children⁴—the prevalence varies (in relative terms) by up to 50% across the range of traffic exposures. This is a modest difference but arguably too great to be explained by the subtle variations in measured exposures to pollutants related to local traffic density. This does not exclude the possibility that other vehicle related pollutants may have a hitherto unsuspected role in initiating or exacerbating asthma, but it argues for a cautious interpretation and careful consideration of possible confounding factors and reporting artefacts.

On the other hand, the failure of a statistically powerful study such as that by Livingstone and colleagues to show an association between local traffic density and prevalence of disease² does not refute the possibility of a more general link between air pollution and asthma because place of residence is such a poor indicator of personal exposure to traffic related pollutants. Nevertheless, these findings do offer reassurance to city dwellers living close to busy roads that the location of their home does not place them or their children at substantially increased risk of asthma.

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Effect of fish oil on heart rate variability in survivors of myocardial infarction: a double blind randomised controlled trial

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Marine n-3 polyunsaturated fatty acids may protect against ischaemic heart disease.¹ In the diet and reinfarction trial patients with an acute myocardial infarction advised to eat fish rich in n-3 polyunsaturated fatty acids had a 29% reduction in two year all cause mortality compared with controls.² The authors hypothesised that dietary n-3 polyunsaturated fatty acids might reduce malignant ventricular arrhythmias and sudden cardiac death, as reported in animals.³ We investigated a possible antiarrhythmic

effect of dietary n-3 polyunsaturated fatty acids in survivors of myocardial infarction.

Patients, methods, and results

Patients were eligible for study if they had been discharged from the department of cardiology at Aalborg Hospital between November 1991 and August 1993 after a myocardial infarction and had a ventricular ejection fraction below 0.40. We excluded patients aged over 75, patients with pacemakers or permanent tachyarrhythmias, and those with serious non-cardiac disease. Eighty one patients fulfilled the inclusion criteria and 55 gave informed consent to a double blind placebo controlled trial.

Patients were randomly allocated to receive either 5.2 g of n-3 polyunsaturated fatty acids daily (as 4.3 g eicosapentaenoic acid and docosahexaenoic acid, equal to eight capsules of Pikasol, a re-esterified triglyceride (EPAX 5500; Pronova Biocare A/S, Norway)) or olive oil for 12 weeks. Treatment began at the time of allocation. Patients were randomised in blocks of 10 and numbered consecutively. Patients drew treatment

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