

## OTHER ORIGINAL PAPERS

# Association between reported exposure to road traffic and respiratory symptoms in children: evidence of bias

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**Background** Many studies showing effects of traffic-related air pollution on health rely on self-reported exposure, which may be inaccurate. We estimated the association between self-reported exposure to road traffic and respiratory symptoms in preschool children, and investigated whether the effect could have been caused by reporting bias.

**Methods** In a random sample of 8700 preschool children in Leicestershire, UK, exposure to road traffic and respiratory symptoms were assessed by a postal questionnaire (response rate 80%). The association between traffic exposure and respiratory outcomes was assessed using unconditional logistic regression and conditional regression models (matching by postcode).

**Results** Prevalence odds ratios (95% confidence intervals) for self-reported road traffic exposure, comparing the categories 'moderate' and 'dense', respectively, with 'little or no' were for current wheezing: 1.26 (1.13–1.42) and 1.30 (1.09–1.55); chronic rhinitis: 1.18 (1.05–1.31) and 1.31 (1.11–1.56); night cough: 1.17 (1.04–1.32) and 1.36 (1.14–1.62); and bronchodilator use: 1.20 (1.04–1.38) and 1.18 (0.95–1.46). Matched analysis only comparing symptomatic and asymptomatic children living at the same postcode (thus exposed to similar road traffic) showed similar ORs, suggesting that parents of children with respiratory symptoms reported more road traffic than parents of asymptomatic children.

**Conclusions** Our study suggests that reporting bias could explain some or even all the association between reported exposure to road traffic and disease. Over-reporting of exposure by only 10% of parents of symptomatic children would be sufficient to produce the effect sizes shown in this study. Future research should be based only on objective measurements of traffic exposure.

**Keywords** Child, preschool, asthma, cough, vehicle emissions, bias, epidemiological methods, questionnaires

## Introduction

Although the individual health risks of air pollution are small its public-health consequences are substantial.<sup>1,2</sup> The recent reduction in classical air pollutants such as SO<sub>2</sub> or NO<sub>2</sub> has masked an ongoing increase in exhaust emissions from road traffic, a complex mixture of pollutants that are not all individually measured. Several authors have, therefore, investigated whether exposure to road traffic is associated with respiratory illness. Their findings have been contradictory,

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some reporting considerable effects<sup>3-7</sup> while others found small or no effects.<sup>8-12</sup> One reason for these discrepancies could be varying misclassification of exposures, especially where self-reported exposure was used.<sup>3-6</sup>

Although population mean estimates of air pollution correlate with objective measures,<sup>13-15</sup> individual estimates vary widely and are associated with a number of factors in addition to measured air pollution.<sup>14,16</sup> Most important is the possible over-reporting of exposure by symptomatic participants, because of the publicity given to air pollution and respiratory health. Such differential reporting would tend to exaggerate any association between exposure and disease.<sup>17</sup> Heinrich *et al.* have recently shown that self-reported and modelled assessment of exposure to air pollution are only weakly associated, but the possibility of reporting bias by symptom status has not been investigated in studies on respiratory symptoms in children, the most common subpopulation involved in air pollution studies, and its impact has not been quantified. Our aim was to investigate in a large population-based survey: (i) whether parent-reported road traffic density at home was associated with the prevalence of respiratory symptoms in pre-school children and (ii) whether any such association could be explained by biased reporting of traffic density.

## Methods

### Population and study design

In April 1998 we sent a respiratory questionnaire to a random sample of 8700 children aged 1.00–4.99 years, born and resident in Leicestershire, UK, using the Leicestershire Health Authority child health database as the sampling frame (Table 1). Parents were told that we were interested in coughs, colds, wheezes, and allergies in young children but not told that we had an interest in road traffic and air pollution. The methods of this survey have been reported in detail elsewhere.<sup>18,19</sup> South Asians, the largest ethnic minority group in the UK were oversampled. The Leicestershire Health Authority Research Ethics Committee approved the study.

### Questionnaire

The questionnaire was designed in 1990 for use in pre-school children and was slightly adapted by adding core questions from the International Study of Asthma and Allergies in Childhood (ISAAC).<sup>20,21</sup> It included questions on the 12 month period prevalence of wheeze, doctor-diagnosed asthma, bronchodilator use, night cough, and chronic rhinitis. 'Possetting or vomiting' in the first year of life was included as a symptom not expected to be related to air pollution in the minds of the children's parents. Exposure to road traffic was assessed by the question, 'How would you describe the location of your house: (i) in a street with very dense traffic (main road); (ii) in a street with moderate traffic (residential road); (iii) in a quiet street with little or no traffic'. The questionnaire also included sections on socio-demographic conditions (parental education, single parents, overcrowding), family history of atopic disease, and a number of known or suspected environmental risk factors for respiratory disease.

### Analysis

Statistical analysis was performed using STATA, version 8.0 for Windows (STATA Corporation, TX, USA). First, we investigated whether reported traffic was associated with increased prevalence of respiratory outcomes in all responders ( $n = 6811$ ; Table 2). Relevant outcomes were wheeze, night cough, chronic rhinitis, and bronchodilator use in the past 12 months, doctor-diagnosed asthma ever, and possetting in the first year of life, with results expressed as proportions and odds ratios (ORs) with 95% confidence intervals (95% CIs), comparing symptom prevalence between exposure categories. Chi-square tests for trend and likelihood ratio tests were used to assess evidence of association. Then, the following putative confounders were entered one by one into the model and those changing the OR perceptibly were included in the final regression models: ethnicity, place of residence, age and sex of the child, paternal and maternal smoking, gas cooking, gas heating, household pets, number of siblings, enrolment in nursery school or day-care, breastfeeding, parental education, and single parenthood.

The association between reported traffic exposures and symptoms that we found might be due to (i) a true causal association between traffic-related air pollution and health, (ii) over-reporting of traffic exposure by families of symptomatic children, or (iii) a combination of the two. To investigate these possibilities, we matched the children by postcode, assuming that within this small area (a 7-digit postcode covers up to 15–16 dwellings) the true exposure to road traffic would be very similar and, therefore, the OR of the effect of traffic exposure, comparing symptomatic with asymptomatic children should be 1.0. Any remaining association between symptoms and reported traffic exposure in this matched analysis, using a conditional logistic regression model, would therefore suggest over-reporting of exposure by parents of symptomatic children.

For this analysis, we could use only data from postcodes where at least one symptomatic child and one asymptomatic child were living. As these subgroups differed from the total sample, including more families living in an inner city area and more south-Asian children (Table 1), we analysed the subgroup in two ways. First, we performed an analysis ignoring the matching (logistic regression) to assess whether the association between traffic and symptoms was similar to the one obtained in the full study population. Second, we performed a conditional logistic analysis that accounted for the matching on postcode to assess whether the association persisted once matching on postcode was appropriately accounted for. For that we compared the ORs from this matched analysis with the results of an unmatched analysis within the same subpopulation (Table 3). We tested these models for effect modification by including interaction terms.

## Results

The response rate, after discounting 200 invalid addresses, was 80% (6811/8500) and there were <2% missing answers for most questions. Forty-one per cent of the children reported little or no traffic, 48% moderate, and 11% dense traffic at their home address (Table 1).

**Table 1** Description of the total study population ( $n = 6811$ ) and of the subgroup used for the matched analysis ( $n = 1660$ )<sup>a</sup>

	Total sample ( $n = 6811$ )			Subgroup ( $n = 1660$ )			P-value <sup>b</sup>
	<i>n</i>	%	95% CI	<i>n</i>	%	95% CI	
<b>Traffic exposure</b>							
No	2818	41.4	(40.2–42.5)	595	35.8	(33.5–38.2)	<0.001
Moderate	3234	47.5	(46.3–48.7)	876	52.8	(50.4–55.2)	
Dense	759	11.1	(10.4–11.9)	189	11.4	(9.9–12.9)	
<b>Sex</b>							
Female	3261	47.9	(46.7–49.1)	800	48.2	(45.8–50.6)	0.768
Male	3550	52.1	(50.9–53.3)	860	51.8	(49.4–54.2)	
<b>Age</b>							
1–1.99	4110	60.3	(59.2–61.5)	1001	60.3	(57.9–62.7)	0.968
2–4.99	2701	39.7	(38.5–40.8)	659	39.7	(37.3–42.1)	
<b>Ethnicity</b>							
Whites	4986	73.2	(72.2–74.3)	1051	63.3	(61.0–65.6)	<0.001
South Asians	1825	26.8	(25.7–27.8)	609	36.7	(34.4–39.0)	
<b>Place of residence</b>							
Inner city	3526	51.8	(50.6–53.0)	1023	61.6	(59.3–64.0)	<0.001
Other	3285	48.2	(47.0–49.4)	637	38.4	(36.0–40.7)	
<b>Maternal education<sup>c</sup></b>							
≤16 years	3579	52.5	(51.4–53.7)	952	57.3	(55.0–59.7)	<0.001
>16 years	3232	47.5	(46.3–48.6)	708	42.7	(40.3–45.0)	
<b>Gas cooking</b>							
No	1686	24.8	(23.7–25.8)	317	19.1	(17.2–21.0)	<0.001
Yes	5125	75.2	(74.2–76.3)	1343	80.9	(79.0–82.8)	
<b>Household pets</b>							
No	4239	62.2	(61.1–63.4)	1134	68.3	(66.1–70.6)	<0.001
Yes	2572	37.8	(36.6–38.9)	526	31.7	(29.4–33.9)	
<b>Mother smoking</b>							
No	5469	80.3	(79.4–81.2)	1311	79.0	(77.0–80.9)	0.120
Yes	1342	19.7	(18.8–20.6)	349	21.0	(19.1–23.0)	

<sup>a</sup> Proportions are calculated from the subgroup used for matched analysis on wheeze, but results are very similar for the other subsamples (asthma diagnosis, bronchodilators, chronic cough, rhinitis, possetting).

<sup>b</sup> Comparing the subgroup used for the matched analysis with the rest of the children.

<sup>c</sup> Age when finishing full-time education.

### Results from the whole study population ( $n = 6811$ )

Prevalence of reported wheeze, asthma diagnosis, bronchodilator use, night cough, and chronic rhinitis was higher in children reported as living on roads with moderate and dense traffic compared with those reporting little or no traffic (Table 2). Adjustment for a large number of confounders did not change these findings. Comparing the categories 'moderate' and 'dense' traffic exposure, respectively, with 'little or no', the ORs (95% CIs) for current wheezing were 1.26 (1.13–1.42) and 1.30 (1.09–1.55); for asthma diagnosis 1.29 (1.11–1.50) and 1.14 (0.90–1.45); for bronchodilator use 1.20 (1.04–1.38) and 1.18 (0.95–1.46); for night cough 1.17 (1.04–1.32) and 1.36 (1.14–1.62); and for chronic rhinitis 1.18 (1.05–1.31) and 1.31 (1.11–1.56). Possetting in the first year of life was not related to reported traffic density.

### Results from the subgroup, where children could be matched by postcode ( $n = 1660$ )

Depending on the prevalence of the different symptoms, a varying number of postcodes including at least one symptomatic child and one asymptomatic child were used for the matched analysis. These were: for wheeze 627 areas with 1660 children, for asthma diagnosis 396 areas with 1047 children, for bronchodilator use 420 areas with 1147 children, for night cough 615 areas with 1662 children, for rhinitis 698 areas with 1832 children, and for possetting 379 areas with 989 children.

Using unconditional logistic regression analysis in these subgroups, the strength of the association between traffic exposure and outcomes was similar to that in the total study population, although CIs for the ORs included 1 in most cases, owing to the lower statistical power (Table 3, unmatched analysis).

**Table 2** Association between self-reported traffic exposure at the child's home and 12 month prevalence of symptoms, bronchodilator use, and asthma diagnosis in children aged 1–4.99 years (total study population,  $n = 6811$ )

Symptoms	Reported traffic	Sample ( $n$ )	Cases $n$ (%)	OR (95% CI) Unadjusted	OR (95% CI) Adjusted <sup>a</sup>
Wheeze	Little or none	2818	758 (26.9)	1.0	1.0
	Moderate	3234	1019 (31.5)	1.25 (1.12–1.40)	1.26 (1.13–1.42)
	Dense	759	237 (31.2)	1.23 (1.04–1.47)	1.30 (1.09–1.55)
	$P$ (trend)		<0.01	<0.01	
Asthma diagnosis <sup>b</sup>	Little or none	2818	345 (12.2)	1.0	1.0
	Moderate	3234	501 (15.5)	1.31 (1.13–1.52)	1.29 (1.11–1.50)
	Dense	759	106 (14.0)	1.16 (0.92–1.47)	1.14 (0.90–1.45)
	$P$ (trend)		0.01	0.02	
Bronchodilator use	Little or none	2818	431 (15.3)	1.0	1.0
	Moderate	3234	583 (18.0)	1.22 (1.06–1.40)	1.20 (1.04–1.38)
	Dense	759	136 (17.9)	1.21 (0.98–1.50)	1.18 (0.95–1.46)
	$P$ (trend)		0.01	0.03	
Night cough	Little or none	2818	673 (23.9)	1.0	1.0
	Moderate	3234	896 (27.7)	1.22 (1.09–1.37)	1.17 (1.04–1.32)
	Dense	759	240 (31.6)	1.47 (1.24–1.76)	1.36 (1.14–1.62)
	$P$ (trend)		<0.01	<0.01	
Rhinitis	Little or none	2818	825 (29.3)	1.0	1.0
	Moderate	3234	1072 (33.1)	1.20 (1.07–1.34)	1.18 (1.05–1.31)
	Dense	759	273 (36.0)	1.36 (1.15–1.61)	1.31 (1.11–1.56)
	$P$ (trend)		<0.01	<0.01	
Possetting	Little or none	2813	443 (15.7)	1.0	1.0
	Moderate	3234	494 (15.3)	0.97 (0.84–1.11)	0.98 (0.85–1.13)
	Dense	759	107 (14.1)	0.88 (0.70–1.11)	0.92 (0.73–1.16)
	$P$ (trend)		0.30	0.54	

<sup>a</sup> Adjusted for age, sex, ethnic group, maternal education, pets, gas cooking, number of siblings, and overcrowding.

<sup>b</sup> Ever in life.

Conducting a conditional logistic regression analysis after matching the children by postcode (Table 3, matched analysis) resulted in equal or even larger effects than in the unmatched analysis. The adjusted ORs (95% CIs), comparing children exposed to dense traffic with those exposed to little traffic, were 1.40 (0.88–2.23) for wheeze, increasing to 1.90 (1.06–3.42) for bronchodilator use and 2.26 (1.22–4.21) for asthma diagnosis, two features associated with more severe wheeze. ORs for night cough and rhinitis were smaller [1.33 (0.85–2.08) and 1.40 (0.91–2.17), respectively]. This implies that parents of children with more severe respiratory problems are particularly prone to overestimate traffic exposure.

We did not find consistent evidence of an effect modification, which would suggest more misclassification in subpopulations defined by paternal or maternal education, parental smoking, parental asthma, or living in an inner city, but statistical power for performing interaction tests was low.

## Discussion

In this population-based survey of pre-school children prevalence of respiratory symptoms, bronchodilator use, and asthma diagnosis were associated with reported exposure to road traffic, even after controlling for a large number of confounders. When we repeated the analysis after matching

the children by postcode, an objective marker for comparing traffic exposure, the strength of associations remained similar or increased, especially for those with more severe symptoms. This suggests that the parents of children with respiratory symptoms over-reported their children's exposure to road traffic or that a third unmeasured factor, like 'negative affectivity',<sup>22</sup> was present that led families to over-report both respiratory symptoms and traffic exposure.

## Methodological considerations

The strengths of this study include its population-based sampling strategy, large sample size, good response rate, and inclusion of large numbers of South Asians, the largest group of ethnic minority population in the UK. Our results are, therefore, likely to be representative for the UK. The full postcode allowed us to allocate children's houses to small geographic areas, covering up to 15 dwellings. We assumed that true domiciliary exposure to traffic-related air pollution was uniform within a single postcode. Although there are certainly situations where traffic exposure might vary within a postcode, owing to increasing horizontal distance from a major road or differing vertical distance in multi-storey buildings the difference should be less within postcodes than between postcodes. Therefore, the ORs should be lower in the matched

**Table 3** Association between self-reported traffic exposure and 12 month prevalence of symptoms, bronchodilator use, and asthma diagnosis in subgroups of the population, using unconditional logistic regression (A) and conditional logistic regression, matched by 7-digit postcode (B) ( $n = 1660$ )

Symptoms	Traffic exposure	Sample (n)	Cases n (%)	OR (95% CI) unmatched (A)		OR (95% CI) matched (B)	
				Unadjusted	Adjusted <sup>a</sup>	Unadjusted	Adjusted <sup>a</sup>
Wheeze	Little	595	253 (42.5)	1.0	1.0	1.0	1.0
	Moderate	876	392 (44.8)	1.09 (0.89–1.35)	1.11 (0.90–1.38)	1.08 (0.84–1.41)	1.06 (0.81–1.39)
	Dense	189	96 (50.8)	1.40 (1.01–1.94)	1.47 (1.05–2.06)	1.44 (0.92–2.26)	1.40 (0.88–2.23)
	<i>P</i> (trend)			0.06	0.04	0.16	0.23
Asthma diagnosis <sup>b</sup>	Little	379	156 (41.2)	1.0	1.0	1.0	1.0
	Moderate	559	231 (41.3)	1.01 (0.77–1.31)	1.05 (0.80–1.37)	0.99 (0.70–1.40)	1.02 (0.71–1.45)
	Dense	109	53 (48.6)	1.35 (0.88–2.08)	1.43 (0.92–2.23)	2.15 (1.18–3.93)	2.26 (1.22–4.21)
	<i>P</i> (trend)			0.30	0.19	0.08	0.06
Bronchodilator use	Little	424	159 (37.5)	1.0	1.0	1.0	1.0
	Moderate	593	254 (42.8)	1.25 (0.97–1.61)	1.26 (0.97–1.63)	1.43 (1.03–1.98)	1.42 (1.02–2.00)
	Dense	130	59 (45.4)	1.39 (0.93–2.06)	1.44 (0.96–2.17)	1.88 (1.06–3.35)	1.90 (1.06–3.42)
	<i>P</i> (trend)			0.05	0.04	0.01	0.01
Night cough	Little	588	253 (43.0)	1.0	1.0	1.0	1.0
	Moderate	855	382 (44.7)	1.07 (0.87–1.32)	1.06 (0.85–1.31)	1.15 (0.89–1.50)	1.11 (0.85–1.45)
	Dense	219	102 (46.6)	1.15 (0.85–1.58)	1.10 (0.80–1.52)	1.34 (0.87–2.07)	1.33 (0.85–2.08)
	<i>P</i> (trend)			0.35	0.51	0.15	0.21
Rhinitis	Little	674	288 (42.7)	1.0	1.0	1.0	1.0
	Moderate	931	437 (46.9)	1.19 (0.97–1.45)	1.19 (0.97–1.45)	1.20 (0.93–1.54)	1.18 (0.92–1.52)
	Dense	227	111 (48.9)	1.28 (0.95–1.73)	1.29 (0.95–1.75)	1.38 (0.90–2.13)	1.40 (0.91–2.17)
	<i>P</i> (trend)			0.05	0.06	0.09	0.09
Possetting	Little	379	159 (42.0)	1.0	1.0	1.0	1.0
	Moderate	502	211 (42.0)	1.00 (0.77–1.32)	1.00 (0.76–1.32)	1.00 (0.72–1.40)	0.99 (0.70–1.40)
	Dense	108	44 (40.7)	0.95 (0.62–1.47)	0.98 (0.63–1.52)	0.90 (0.49–1.65)	0.93 (0.49–1.73)
	<i>P</i> (trend)			0.88	0.95	0.83	0.85

Numbers are smaller than in Table 1, because only children from postcodes, where at least one symptomatic child and one asymptomatic child were living, could be included. A 7-digit postcode covers up to 15–16 houses.

<sup>a</sup> Adjusted for age, sex, ethnic group, maternal education, pets, gas cooking, number of siblings, and overcrowding.

<sup>b</sup> Ever in life.

analysis compared with the unmatched analysis. This was not the case, the strength of association was similar in the matched and the unmatched analysis, implying that most or even all of the associations found in this study might be explained by biased over-reporting of traffic density by parents of symptomatic children.

A limitation of the study was the low statistical power in the matched analysis, due to the fact that the sampled children were dispersed over a large area. Only a quarter of the study families could thus be used for the matched analysis.

### Comparison with other studies

Heinrich *et al.*, using data from Dutch and German cohorts, have recently shown that self-reported and modelled assessments of exposure are only weakly associated. They did not, however, analyse their data by symptom status of the participating children.<sup>15</sup>

Other studies on road traffic and respiratory symptoms in children using self-reported traffic exposure (including our

own unmatched data) tended to find larger effects than surveys relying on objective measurements. For instance, ORs for current wheeze in children, contrasting the categories 'frequent' and 'constant' truck traffic with 'never', were 1.53 and 2.15 in a survey of 12- to 15-year old children in Münster and 1.53 and 1.67 in 13- to 14-year olds in Bochum, Germany (Table 4).<sup>4,6</sup> Hirsch *et al.*, in 5421 children aged 5–11 years, found an OR of 2.09 for wheeze, comparing 'constant' with 'no' truck traffic, while they did not find an association between wheeze and objectively measured exposures to a number of traffic-related air pollutants.<sup>5</sup> Studies using distance to the main road or traffic counts as exposures generally found smaller or no effects.<sup>8–10,12,23</sup> For rhinitis, we found insufficient studies using measured exposure to draw any conclusions.

### Implications for future research

Our findings suggest that systematic over-reporting of exposure to road traffic by families of symptomatic children might have led to biased effect estimates and could explain

**Table 4** Published studies on the association between respiratory symptoms (wheeze, cough, rhinitis) in children and measured or self-reported exposure to road traffic

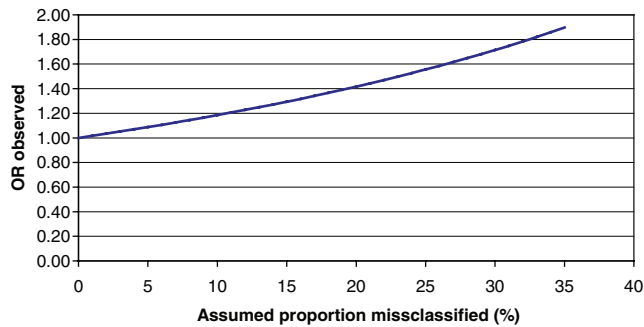
Author	Age (years)	Exposure measure	OR (95% CI)		
			Wheeze	Cough	Rhinitis
<b>Exposure measured</b>					
Nicolai <sup>9,12</sup>	5–11	Rest of study sample	1.00	1.00	
		Low	0.52 (0.19–1.46)	1.18 (0.67–2.05)	
		Medium	1.10 (0.55–2.18)	1.49 (0.93–2.39)	
		High traffic counts	1.70 (0.93–3.11)	1.54 (0.97–2.46)	
Venn <sup>8,23</sup>	4–11	Low	1.00	1.00	
		Medium	1.11 (1.02–1.22)	1.21 (1.02–1.44)	
		High traffic activity	1.13 (1.03–1.24)	1.22 (1.02–1.45)	
	11–16	Low traffic activity	1.00	1.00	
		Medium traffic activity	0.99 (0.92–1.06)		
		High traffic activity	0.94 (0.87–1.01)		
Venn <sup>23</sup>	4–11	per 30 m <sup>a</sup>	1.08 (1.00–1.16)		
	11–16	per 30 m <sup>a</sup>	1.16 (1.02–1.32)		
van Vliet <sup>10</sup>	7–12	Truck traffic	1.13 (0.33–3.88)	1.30 (0.59–2.86)	2.10 (0.74–5.99)
Lewis <sup>12</sup>	4–6	≥150 m <sup>a</sup>	1.00		
		90–149	1.14 (0.96–1.36)		
		30–89	1.02 (0.87–1.21)		
		<30	0.90 (0.69–1.18)		
<b>Exposure self-reported</b>					
Ciccone <sup>3</sup>	6–14	Never	1.00	1.00	
		Sometimes	1.12 (0.95–1.31)	1.26 (1.11–1.44)	
		Often truck traffic	1.25 (1.02–1.53)	1.49 (1.27–1.74)	
Hirsch <sup>5</sup>	5–11	Constant truck traffic	2.09 (1.24–3.53)	1.60 (1.06–2.42)	
Duhme <sup>4</sup>	12–15	Never	1.00		1.00
		Seldom	1.11 (0.88–1.41)		1.26 (1.05–1.51)
		Frequent truck traffic	1.53 (1.15–2.05)		1.71 (1.36–2.15)
		Constant truck traffic	2.15 (1.44–3.21)		1.96 (1.40–2.76)
Weiland <sup>6</sup>	13–14	Never	1.00		1.00
		Frequent	1.53 (1.06–2.20)		1.67 (1.17–2.68)
		Constant truck traffic	1.67 (1.05–2.66)		1.54 (0.97–2.44)
Kuehni (current study)	1–4	Low	1.00	1.00	1.00
		Medium	1.26 (1.13–1.42)	1.17 (1.04–1.32)	1.18 (1.05–1.31)
		High traffic activity	1.30 (1.09–1.55)	1.36 (1.14–1.62)	1.31 (1.11–1.56)

<sup>a</sup> Distance of home from main road.

some or all of the exposure-disease association in our study. The size of this bias varied for the different health outcomes; while it was not noticed for the non-respiratory symptom of 'possetting', it was intermediate for night cough and rhinitis and largest for bronchodilator use and the diagnosis of asthma, which have received the broadest media coverage with regard to air pollution. Also, diagnosis or medication given by doctors might induce parents to regard the symptoms more seriously and thus to attribute (or misattribute) causes to these problems.

Although it has often been hypothesized that reporting bias might play a role in assessment of effects of air pollution, this has never been shown for studies on respiratory symptoms in children. The different results for different symptoms

suggest that public concern about health effects of air pollution plays an important role. The extent of this bias is, therefore, likely to vary between regions and time periods, so that results from one study cannot be extrapolated to other situations. For instance, a population-based survey in Italy, where information about respiratory disorders and traffic near residences was collected by questionnaire, could evaluate the possibility of reporting bias by matching a subsample of cases and controls by address code. In this study, the raw association between case-control status and reported frequency of lorry traffic was 1.12, decreasing to 1.04 in the matched analysis, suggesting no systematic difference in traffic reporting between parents of symptomatic and asymptomatic children.<sup>3</sup>



**Figure 1** Observed odds ratio (OR) of an association between traffic exposure and symptoms as a function of different levels of assumed exposure misclassification

Misclassification is defined as an unexposed family with a symptomatic child falsely reporting high traffic.

The scenario assumes no true effect (true OR=1.0) and a population with 40% unexposed (low traffic) and 60% exposed (moderate or high traffic) families.

The calculation for the figure is based on a traditional 2x2 table. If the true odds ratio (tOR) is calculated as  $tOR = (a/c) / (b/d)$ , then the observed odds ratio (oOR) with a varying proportion (X) of cases with misclassified exposure is calculated ( $oOR = ((a + X*c)/(c-Xc)) / (b/d)$ ).

With a prevalence of exposure to moderate or high traffic of 60% in our study, a relatively small proportion (10 and 20%, respectively) of families with symptomatic children falsely reporting high traffic exposure would be sufficient to bias the OR from 1.0 (no effect) to 1.2 and 1.4, respectively, effect sizes typically reported in epidemiological studies (Figure 1). With 38% of parents falsely reporting high exposure, the OR would be 2.0.

Our data illustrate that random errors (quantified with *P*-values or CIs) and confounding, the two main issues that are usually dealt with in epidemiology, are not the only threats to valid inference and in fact might be dwarfed by systematic errors such as biased reporting. Systematic errors unfortunately are not routinely considered in the interpretation of research results.<sup>17</sup> Our findings parallel what in more traditional case-control studies, for example in childhood cancer and its association with antenatal risk factors, is termed 'recall bias' if exposures are assessed retrospectively or contemporaneously with the health outcome.

In conclusion, after matching for postcode our results provide evidence that most if not all of the association between reported road traffic and respiratory symptoms in this survey of pre-school children could be the result of a reporting bias. These findings point out that self-reported exposure to road traffic is unreliable and of limited use in aetiological research.

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## KEY MESSAGES

- Our study suggests that reporting bias could explain some or even all of the association between reported exposure to road traffic and respiratory symptoms in children.
- Over-reporting of exposure by only 10% of parents of symptomatic children would be sufficient to falsely suggest an effect with an OR of 1.2, while 20% over-reporting would produce an OR of 1.4.
- Future research should be based only on objective measurements of traffic exposure.
- If existent, this bias also threatens the validity of several other observational studies that showed a positive association between road traffic and asthma.

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