

An investigation of the association between traffic exposure and the diagnosis of asthma in children

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This study investigated whether proximity to traffic at residence location is associated with being diagnosed with asthma as a young child. A survey of parents of children (aged 5–7) in kindergarten and first-grade in 13 schools was completed in Anchorage, Alaska, and Geographical Information System (GIS) mapping was used to obtain an exposure measure based on traffic density within 100 m of the cross streets closest to the child's residence. Using the range of observed exposure values, a score of low, medium or high traffic exposure was assigned to each child. After controlling for individual level confounders, relative to the low referent group, relative risks (95% confidence intervals) of 1.40 (0.77, 2.55) and 2.83 (1.23, 6.51) were obtained in the medium and high exposure groups, respectively. For the null hypothesis of no difference in risk, a significance level of 0.056 was obtained, which suggests that further investigation would be worthwhile. Children *without* a family history of asthma were more likely to have an asthma diagnosis if they resided in a high traffic area than children who had one or more parents with asthma. The relative risk for children without a family history of asthma is 2.43 (1.12, 5.28) for medium exposure and 5.43 (2.08, 13.74) for high exposure. For children with a family history of asthma, the relative risk is 0.66 (0.25, 1.74) for medium exposure and 0.67 (0.12, 3.69) for high exposure. The *P*-value for the overall "exposure-effect" (i.e. both main effects AND interaction terms) is 0.0097.

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Introduction

Several studies have indicated that children who live near traffic have greater symptoms and increased hospitalizations for asthma (Edwards et al., 1994, Brauer et al., 2002). The severity of asthma symptoms is also associated with ambient benzene concentrations (Thompson et al., 2001; Delfino et al., 2003). This paper reports the results of a study that investigated the association between a traffic-density related measure of exposure and the prevalence of diagnosed asthma among school children 5 to 7 years of age in Anchorage, Alaska (an area with high ambient benzene concentrations),

adjusting for gender, parental asthma, a smoker in the home, and family income.

Traffic is known to produce a mixture of both particulate and gaseous air pollution. In Anchorage, gasoline has an unusually high (35–50%) aromatic content (benzene content 5%), causing volatile organic compounds (VOCs) to be a large component of air pollution. The predominant source of VOC pollution is gasoline emissions both from traffic and from cold starts. The average annual ambient benzene concentration in Anchorage is eight parts per billion, higher than any other US city (Air Quality, 1998). The predominant particulate matter is coarse fraction (2.5 μm < PM < 10 μm) generated from road surfaces. Other air pollutants that have been associated with asthma severity, such as PM_{2.5} and ozone, are very low to nonexistent in this area. The fine fraction of PM₁₀ averages less than 15% of the measured particulate mass. Ozone is not formed at this latitude (61°N) because of lack of sunlight during winter inversions when the pollution is highest (Air Quality, 2000). Ultrafine particulate pollution has not been measured in Anchorage. Owing to the geographic considerations, long-haul transportation around the state is done by barge or air cargo planes rather than by trucks. Table 1 shows measured values for carbon monoxide, fine particulates (PM_{2.5}), and

1. Abbreviations: PM₁₀, particulate matter less than 10 μm in aerodynamic diameter; PM_{2.5}, particulate matter less than 2.5 μm in aerodynamic diameter; vm, vehicle meters (vehicles per day multiplied by meters of roadway within the buffer).

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Table 1. Reference table for carbon monoxide and PM_{2.5} in Anchorage Alaska in 2003.

Average quartile 2003 Anchorage air quality measurements	24-h CO (ppm)	PM- _{2.5} (reference R&P) ($\mu\text{g}/\text{m}^3$)	Total organic and elemental carbon ($\mu\text{g}/\text{m}^3$)
1st quartile	0.56	6.90	2.75
2nd quartile	0.90	5.66	3.74
3rd quartile	1.16	6.39	4.54
4th quartile	1.61	9.46	6.87

elemental and organic carbon (a surrogate for diesel exhaust) for each season in Anchorage in 2003. The fine particulates average less than half of the $15 \mu\text{g}/\text{m}^3$ average annual action level set by the Environmental Protection Agency.

Anchorage is a relatively new metropolitan area with a culturally diverse ethnic population and no geographic boundaries based on racial or economic status. Low income people are not isolated to specific neighborhoods but are dispersed throughout the city (Census, 2000). This allows for the consideration of asthma disease in relation to traffic apart from race and income that are often strongly associated with increased proximity to traffic.

Methods

Exposure Assessment

Traffic counts on roadways were obtained from the Alaska Department of Transportation and the city Planning Office. Students from the traffic-engineering department at the University of Alaska Anchorage measured traffic on roads that had not been measured by the State or the Municipality in order to accurately account for all traffic exposure. Traffic counts on streets in Anchorage varied from around 500 to 51,000 vehicles per day. Very low volume residential streets with no through traffic and cul-de-sac streets were not measured for traffic counts. However, a minimal count of 100 vehicles was assigned to such streets, so that no streets had zero traffic; we later used a three-level discretization of exposure, and the *ad hoc* assignment of 100 vehicles did not change any child's classification.

We used the Geographical Information System (GIS) ArcView (GIS version 3.3 Published by Environmental Systems Research Institute (ESRI) 2002 Redlands, CA, USA) to map the coordinates of the nearest cross streets to each child's home. We calculated a traffic exposure variable by drawing a 100-m buffer zone around each intersection of interest. The approximate length of one city block in Anchorage is 100 m. Anchorage Air Quality Section studies have shown that PM₁₀ measured at 10 ft (3 m) from the road declined by 50% at 165 ft (50 m) from the road. Increased respiratory symptoms have been detected at distances up to

300 m from major roads (Brunekreef et al., 1997), and an additional traffic variable based on a 300-m buffer was also calculated. All the road segments that fell within the 100-m buffer were multiplied by the traffic count of that road at that point and then summed to create a measure of traffic exposure; this method allowed for weighting the effect of major roads by their proximity. The measurement of the length of each road falling within the buffer was automatically calculated by GIS.

For the outcome and adjustment data, surveys were conducted in 13 elementary schools in the Anchorage School District. The schools were chosen to include wide geographical and economic diversity of students. The survey included demographic questions, symptom questions, and questions about parental asthma, a smoker in the household, smoking in the house, housing type, house pets, cockroaches, mold, heating, humidifier use, and family income range as a measure of socioeconomic status. The Institutional Review Board at the University of Alaska Anchorage reviewed the research and permitted us to survey without signed informed consent because the surveys were completely anonymous. Computer-assisted data entry of the surveys was carried out using the Cardiff software (Cardiff.com).

A total of 1106 surveys were received. The overall return rate by school for surveys was 75%, with a range of 54–92% across schools. Bilingual teachers translated surveys and letters into the most common non-English languages in the schools (Spanish, Mien, Hmong, Laotian, and Russian) to accommodate non-English speaking parents when necessary.

Statistical Analysis

The primary outcome variable was “ever having had a diagnosis of asthma,” and only children who had been residents in their current home for at least 1 year were considered. We used logistic regression (e.g. Hosmer and Lemeshow, 2000) to model the lifetime probability of being diagnosed with asthma, as a function of the traffic count in vehicle-meters (vm), and additional confounder and precision variables that were known to be associated with diagnosis of asthma. Here we define a confounder in the usual sense, as a variable that may distort the magnitude of effect, while a precision variable is one that is associated with the outcome but not the traffic exposure. In such a model, a generic component represents the log odds ratio associated with a unit change in the corresponding variable, while controlling for all other variables. We present two sets of analyses: the first corresponding to our initial analysis plan where we consider main effects for traffic exposure and the second where we consider the possibility of interactions with other variables. The initial analysis plan was based on sample size considerations, and we view the subsequent investigation of interactions as primarily exploratory. Model parameters were estimated using the S-Plus statistical software package, v6.2 (Insightful Corp, Seattle, WA, USA).

The primary exposure of interest is the traffic exposure based on the 100-m buffer zone, and ranged between 40,500 and 15,273,918 μm . Since this range is very large, it is unlikely that a single odds ratio would be appropriate to relate traffic exposure to the diagnosis of asthma across the complete range. Hence, we split the exposure variable into three categories: low ($<4 \times 10^6 \mu\text{m}$), medium (4×10^6 to $8 \times 10^6 \mu\text{m}$), and high ($>8 \times 10^6 \mu\text{m}$). The choice of these cutpoints was based on initially splitting the range of the traffic exposure into roughly four equal intervals; due to the sparseness of data in the upper ranges of exposure, the two intervals in the upper end of exposure were collapsed to produce the final three-level categorization. An alternative to categorizing the exposure would be to use smoothing-based methods (Harrell, 2001). However, since the continuous exposure variable is not a personal assessment, we also expect that with discretization (which involves a loss of information), we will reduce the possibility of exposure misclassification; although without a gold standard there is no way to verify this. We parameterized each model by fixing the low exposure category to be the referent group and compared the remaining two categories with the referent. Consequently, there are two odds ratios of interest: (1) the relative change in the odds of asthma comparing an individual in the medium category to an individual in the low category and (2) the relative change in the odds of asthma comparing an individual in the high category to an individual in the low category. Confounders/precision variables based on published literature were gender, parental asthma, presence of a household smoker, and income (Gergen et al., 1998; Jaakkola et al., 2001). For the first set of analyses (without interaction terms) we considered two sets of models: an unadjusted model using traffic exposure alone and an adjusted model that includes the additional confounder and precision variables outlined above. Further analyses with other confounders included were considered, but the results were stable across these choices. A further analysis adopted exactly the same modeling approach but used the 300-m buffer zone based exposure measure. The range of the traffic exposure based on the 300-m buffer zone was 106,350 to 57,778,372 μm . Again, due to the large range of counts, exposure was categorized into three levels: low ($<14 \times 10^6 \mu\text{m}$), medium (14×10^6 to $29 \times 10^6 \mu\text{m}$), and high ($>29 \times 10^6 \mu\text{m}$).

The second set of analyses explored potential interactions between the 100 m-based traffic exposure and a variety of confounders and precision variables. We focused primarily on those variables included as adjustment variables in the first set of analyses, and in each case consider interactions with the three-level categorical exposure measure. Each interaction was considered separately, and each model included adjustment for at least each of gender, parental asthma, household smoker and income. Evidence for interactions was assessed by initially computing a *P*-value for the overall significance of

“exposure”, which, in these models, included both the main effects *and* the interaction terms. Subsequently, evidence for effect modification was assessed directly by considering the contribution of the interaction terms, above and beyond that of the main effects.

Results

Table 2 provides baseline characteristics arranged by asthma diagnosis status on confounding/precision variables, which were included as adjustment variables in the model. Table 2 also provides stratum-specific asthma rates and *P*-values to assess the univariate associations between each variable and diagnosis of asthma. The overall asthma rate among the children of the 1043 respondents in the data set is 11.1%. However, when the data set was modified to include only those children who had been living at their current address for more than 1 year, the asthma rate among the 756 remaining children (72.5% of respondents) who met this criterion was 10.6%. Table 2 shows gender was missing for six children, and there were slightly more males than females (395 vs. 355). Although the asthma diagnosis rate appears to be slightly higher among females than males (11.0% vs. 10.1%), the association is not statistically significant at the 0.05 level. There are 40 children for whom parental asthma status is missing. Among the asthmatics with recorded parental asthma, 49.3% had at least one parent previously diagnosed with asthma, compared to only 15.5% of the unaffected children who had at least one parent previously diagnosed. The asthma rates comparing children with and without at least one parent previously diagnosed with asthma were 25.9% and 6.2% ($P < 0.001$). There is fairly strong evidence to suggest substantially increased risk associated with having at least one parent being previously diagnosed with asthma (OR 5.14; 95% CI 3.01, 8.78). The asthma rates among children with and without at least one household smoker were 12.6% and 9.9%, although this association is not statistically significant ($P = 0.276$). Although the association between income category and asthma is statistically significant ($P = 0.031$), the rates do not exhibit a clear monotonic trend: the asthma rate in the lowest income was 16.8% and decreased to 6.7% in the third highest income category, while the asthma rate in the highest income group (which has relatively sparse data) increased to 12.5%. Children living less than 1 year in their current address were disproportionately in the lowest income families (36.4% vs. 25.0%), but the transients were not disproportionately asthma cases or noncases for all groups.

Table 3 provides a description of the 100-m buffer traffic exposure after categorization into three levels (low, medium, high). The column percentages in Table 3 indicate that asthma cases tend to experience higher exposure than those without asthma, with 12.5% of asthma cases being classified

Table 2. Baseline characteristics for children aged 5–7 years living at the current address for 1 year or more.

	No asthma (%)	Asthma (%)	Combined number	Asthma rate (%)	P-value ^a
Total	676 (89.4%)	80 (10.6%)	756	10.6	
<i>Gender</i>					0.702
Male	355 (52.9%)	40 (50.6%)	395	10.1	
Female	316 (47.1%)	39 (49.4%)	355	11.0	
	671	79	750		
<i>Parental asthma</i>					<0.001
No	545 (84.5%)	36 (50.7%)	581	6.2	
Yes	100 (15.5%)	35 (49.3%)	135	25.9	
	645	71	716		
<i>Household smoker</i>					0.276
No	475 (70.9%)	52 (65.0%)	527	9.9	
Yes	195 (29.1%)	28 (35.0%)	223	12.6	
	670	80	750		
<i>Income</i>					0.031
< \$20K	119 (18.6%)	24 (30.0%)	143	16.8	
\$20–49K	278 (43.4%)	35 (43.7%)	313	11.2	
\$50–100K	195 (30.4%)	14 (17.5%)	209	6.7	
> 100K	49 (7.6%)	7 (8.8%)	56	12.5	
	641	80	721		

^aBased on a χ^2 test for independence between the covariate and asthma outcome.

Table 3. Description of the 100-m buffer zone exposure after categorization into three levels.

	No asthma (%)	Asthma (%)	Combined number	Asthma rate %
<i>Exposure</i>				
Low	471 (69.7%)	49 (61.2%)	520	9.4
Medium	161 (23.8%)	21 (26.3%)	182	11.5
High	44 (6.5%)	10 (12.5%)	54	18.5
	676	80	776	

as high exposure compared to only 6.5% of those with no asthma diagnosis. The same ordering of asthma rates is observed in the medium exposure group though it is less marked.

Table 4 presents the results from the main logistic regression analyses (i.e. those without interactions). The unadjusted analysis of the 100-m buffer zone traffic exposure is based on 756 children for whom both exposure and asthma diagnosis were measured and who had resided at the same location for 1 year or more (see Table 2). The adjusted analysis is based on 671 children for whom, in addition to exposure, asthma diagnosis and length of residence > 1 year, gender, parental asthma, presence of a household smoker, and household income were also recorded.

In the unadjusted analysis, there is an association for those in the high exposure group (relative to the low exposure

group), with an estimated odds ratio (OR) of 2.19 (95% CI: 1.04, 4.60). However, the overall association across all three levels, although displaying a monotonic relationship, is not statistically significant ($P=0.139$). After adjusting for confounding/precision variables, we found that there is no significant difference between the low exposure group and medium exposure group, in terms of the odds of a diagnosis of asthma. However, we found that the odds ratio of an asthma diagnosis for a child in the high exposure group was 2.83 times higher (95% CI: 1.23, 6.51) than the referent of a child in the low exposure group, controlling for the combined effects of gender, parental asthma, presence of a household smoker, and household income. Although the point estimates suggest a trend, the overall association between the three levels of exposure and asthma is on the borderline of conventional levels of significance, with a $P=0.056$.

The OR for asthma diagnosis with exposure to high traffic within 300 m (not in Table 4) compared to low traffic was 2.45 (95% CI: 0.99, 6.10) with $P=0.149$ after adjustment for gender, parental asthma, a smoker in the house, and family income.

We also considered the possibility of confounding that might occur by having a doctor’s diagnosis as an outcome measure when low-income children may not have access to a physician. We included the symptoms of daily or weekly wheeze not associated with colds, daily cough not associated with colds, and/or asthma medications taken regularly in an expanded definition of the outcome measure. The expanded

Table 4. Logistic regression analysis of the 100-m buffer based traffic exposure on asthma diagnosis.

	100-m Buffer			
	Unadjusted (<i>n</i> = 756)		Adjusted (<i>n</i> = 671)	
	OR (95% CI) ^a	<i>P</i> -value ^b	OR (95% CI) ^a	<i>P</i> -value ^b
<i>Exposure</i>		0.139		0.056
Low	Referent		Referent	
Medium	1.25 (0.73, 2.15)		1.40 (0.77, 2.55)	
High	2.19 (1.04, 4.60)		2.83 (1.23, 6.51)	
<i>Gender</i>				0.843
Male			Referent	
Female			1.06 (0.62, 1.78)	
<i>Parent asthma</i>				<0.001
No			Referent	
Yes			5.14 (3.01, 8.78)	
<i>Smoker in house</i>				0.439
No			Referent	
Yes			1.25 (0.72, 2.16)	
<i>Income</i>				0.143
< \$20K			Referent	
\$20–49K			0.61 (0.32, 1.16)	
\$50–100K			0.40 (0.18, 0.89)	
> \$100K			0.75 (0.26, 2.14)	

^aMaximum likelihood estimates and Wald-based 95% confidence intervals for logistic regression models.

^b*P*-values are based on likelihood ratio tests for the inclusion of the entire factor.

definition of asthma increased the number of cases by 12, and the overall rate of asthma was increased from 10.6% to 12.6%. The additional cases did not change the results significantly. The OR for a risk of the expanded definition of asthma associated with high traffic exposure (based on a 100-m buffer) was 2.85 (95% CI: 1.28, 6.36) with an overall *P* = 0.053.

Table 5 presents selected results from an exploration of potential effect modification of the 100-m buffer based traffic exposure. While interactions were considered for each of gender, parental asthma, presence of a household smoker and income, we only present stratum-specific results for parental asthma and the presence of a household smoker. Overall there seems to be strong evidence for effect modification by parental asthma (*P*-value 0.01) and marginal evidence for effect modification by the presence of a household smoker (*P*-value 0.055). There was no statistical evidence for effect modification by gender or income (*P*-values 0.179 and 0.374, respectively). Although we do not present them, for both parental asthma and household smoker, stratum-specific exposure distributions were very similar. When interpreting effect modification by parental

Table 5. Selected stratum-specific results of logistic regression-based analysis of potential interactions between adjustment variables and the 100-m buffer based traffic exposure on asthma diagnosis.

Interaction variable ^a	Exposure level	OR (95% CI) ^b	Overall <i>P</i> -value ^c
<i>Parental asthma</i>			0.010
No	Medium	2.43 (1.12, 5.28)	
	High	5.34 (2.08, 13.74)	
Yes	Medium	0.66 (0.25, 1.74)	
	High	0.67 (0.12, 3.69)	
<i>Household smoker</i>			0.055
No	Medium	1.72 (0.83, 3.56)	
	High	1.85 (0.60, 5.73)	
Yes	Medium	0.87 (0.28, 2.67)	
	High	5.58 (1.59, 19.61)	

^aInteraction variables considered separately. Each model included adjustment for at least those variables in first set of analyses: gender, parental asthma, household smoker and income.

^bMaximum likelihood estimates and Wald-based 95% confidence intervals for logistic regression models.

^c*P*-values are based on likelihood ratio tests for the inclusion of the entire exposure factor (i.e. both main effects and interaction terms).

asthma one may loosely interpret the results in terms of a genetic predisposition for asthma among the children. From Table 5 it seems that children who have a family history of asthma do not appear to be affected by traffic exposure as much as children without a family history. The odds ratio for children with no parental asthma is 2.43 (95% CI: 1.12, 5.28) for medium exposure and 5.43 (95% CI: 2.08, 13.74) for high exposure. For children with a family history of asthma, the relative risk was 0.66 (95% CI: 0.25, 1.74) for medium exposure and 0.67 (0.12, 3.69) for high exposure. For children in households without a smoker, we find an indication of increased risk associated with traffic pollution, (ORs of 1.72 and 1.86 for medium and high traffic exposure, respectively), although we cannot rule out no effect in either stratum. Children exposed to a smoker in the house were less affected by exposure to medium traffic OR 0.87 (95% CI: 0.28, 2.67), but seemed to be greatly affected by high traffic exposure OR 5.58 (95% CI: 1.59, 19.61), suggesting a strong synergistic interaction between exposure to traffic pollution and exposure to household cigarette smoke.

Discussion

The prevalence of asthma and allergies has been increasing in westernized countries. Higher rates of asthma are found in urban areas than in rural areas (ISAAC, 1998). An explanation for the different asthma rates may be the higher amount of traffic pollution in urban vs. rural areas. Recent studies of exposures to polycyclic aromatic hydrocarbons

(PAH), a product of diesel combustion, showed that children in inner cities had significantly higher exposure to PAH than did rural children (Chuang et al., 1999). High traffic exposure is a risk factor for the development of wheeze and asthma diagnoses. A study in Japan found that rate of asthma increased among girls living within 50 m of a trunk road (Shima et al., 2003), and an increased incidence of asthma during the follow-up period, that was significantly associated with atmospheric concentrations of nitrogen dioxide (a vehicular exhaust emission) (Shima et al., 2002).

A study in Italy found that children who live on streets with a high frequency of truck traffic may experience adverse respiratory effects with increased lower respiratory tract infections early in life and wheezing and bronchitic symptoms at school age (Ciccone et al., 1998). In the Netherlands, cough, wheeze, runny nose, and doctor-diagnosed asthma were significantly increased for children living within 100 m of the freeway (van Vliet et al., 2000). Another report from the Netherlands indicates that exposure to traffic-related air pollution, in particular diesel exhaust particles, may lead to reduced lung function in children living near major motorways (Brunekreef et al., 1997). In Munich, adolescents who self-reported constant truck traffic exposure had increased prevalence odds ratio of 2.15 for wheezing (Duhme et al., 1996).

In the United Kingdom, primary and secondary school children living within 150 m of a main road had increased risk of wheeze with increasing proximity to a main road (Venn et al., 2001). In France, a study of lifetime traffic exposure in young children found that exposure in the first 3 years of life was significantly associated with a diagnosis of asthma after controlling for known risk factors (Zmirou et al., 2004). In Belfast, Ireland, admissions to the emergency room for asthma were related to air pollutants in a time series analysis. After adjustment for thoracic particulate matter, nitrogen oxides, carbon monoxide, and sulfur dioxide, benzene level was the only variable associated independently with asthma emergency department admissions in children. These authors conclude that the most reliable method of measuring exposure to vehicle exhaust emissions may be measurements of benzene (Thompson et al., 2001). Two studies have been completed in the United States. One found that traffic-related pollution is associated with respiratory symptoms in children (Kim et al., 2004). The other found no increase in asthma prevalence related to residence proximity to high traffic roads in a cohort of low income children, although they reported an increase in medical visits for asthma by traffic-exposed children (English et al., 1999).

VOCs in both ambient and indoor air have been associated with an increased risk of developing asthma. A study in Kanawha valley concluded that children exposed to VOCs had increased risk of chronic respiratory symptoms characteristic of reactive airways (Ware et al., 1993). A recent

study in Australia found that, for every 10 unit increase in VOCs (benzene and toluene) found in the home, children had a two to three times increased risk of developing asthma (Rumchev et al., 2004). There is laboratory support for this association. In chamber studies, a mixture of VOCs was shown to increase lower respiratory symptoms — chest tightness, shortness of breath, and cough — in a dose-response manner both immediately and 2 h after exposure (Pappas et al., 2000). This study was carried out in healthy, nonasthmatic adults exposed for 4 h to a mixture of VOCs at 25 and at 50 mg/m³.

Our population-based study indicates that in an area where vehicular traffic is the sole source of air pollutants and ambient VOCs are the highest in the nation, exposure to high traffic is associated with increased asthma prevalence.

Limitations

The possibility of unmeasured confounders causing bias cannot be eliminated, although we have been able to adjust at the individual level for a number of important known confounders such as smoking status of parents and income. Despite an overall good response rate, selection bias could occur. The derived semiecological exposure is not optimal but we would expect it to be better than a simple “distance to major road” variable. Since the children were relatively young, the use of residential addresses is less limiting than a similar study in adults.

Conclusion

We have evidence of a weak association between asthma prevalence in children 5–7 years of age exposed to traffic in an area where the primary air pollutants are VOCs and coarse fraction particulate matter. Children without genetic predisposition to asthma appear to be most at risk. More epidemiological studies are needed regarding the effects of traffic pollutants, especially VOCs.

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