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Recent evidence for adverse effects of residential proximity to traffic sources on asthma

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Purpose of review

A growing body of evidence indicates that residential proximity to traffic sources increases the risk for asthma and asthma exacerbations. In this review we have considered publications from 2006–2007 that examined the impact of residential traffic-related exposures on asthma occurrence and severity.

Recent findings

In these studies, exposures were estimated using traffic metrics based on residential distances from major roads and freeways, traffic densities around homes, and models of traffic exposure. Overall, residential proximity to traffic sources was associated with increased asthma occurrence and exacerbations in both children and adults. Land-use regression models were superior to individual traffic metrics in explaining the variability of traffic-related pollutants. Susceptibility may also play a role in variation in the effects of traffic on asthma.

Summary

There is consistent evidence that living near traffic sources is associated with asthma occurrence and exacerbations. Future studies have the opportunity to improve exposure estimates by measuring traffic-related pollutants near homes and schools and including time/activity patterns in prediction models. Further research is also warranted to investigate the differential impact of traffic by genetic and other susceptibility factors and to identify specific pollutants that underlie the adverse effect of traffic on asthma.

Keywords

air pollution, asthma, freeway, traffic, wheeze

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Introduction

Asthma is the most common chronic obstructive pulmonary disease. Despite much research, the environmental determinants of asthma remain to be firmly established. A growing body of evidence indicates that residential exposures to traffic emissions are associated with increased risk of adverse respiratory health outcomes including asthma incidence, severity and persistence. The effects of traffic on asthma are biologically plausible as traffic-related pollutants are associated with airway inflammation mediated by oxidant stress [1[•]] and reduced lung-function level and growth [2[•],3[•]]. These biological mechanisms are implicated in asthma etiology and severity. Associations of traffic-related ambient air pollutants [particulate matter (PM₁₀, PM_{2.5}, which are particulate matters with a 50% cut-off aerodynamic diameter of <10 and <2.5 μm, respectively), nitrogen dioxide (NO₂), and carbon monoxide (CO)] on asthma occurrence and exacerbations (symptoms and hospitalizations) have been reported; however, there are other important sources (biomass combustion, regional transport, etc.) of these pollutants in addition to local traffic emissions.

In recent years, the impact of traffic-related exposures near home on asthma occurrence and exacerbations has been investigated in epidemiologic studies using different traffic metrics. Some of these metrics include distances of homes from the nearest major road and freeway, use of buffers of variable diameters around homes to compute traffic density, and a modeling approach to estimate traffic-related exposure near homes. In this review, we summarized papers published in 2006–2007 which reported the effects of local traffic-related exposures on asthma occurrence and exacerbations [4^{••},5^{••},6,7,8[•],9^{••}–11^{••},12–15,16[•]].

Literature search strategy

Through PubMed and Medline searches, we reviewed original articles in peer-reviewed journals published between January 2006 and August 2007. For exposures, the search terms used were traffic, freeways, major road, vehicular emissions, diesel, diesel exhaust particles, ultrafine particles, volatile organic compounds (VOCs), combustion products, polyaromatic hydrocarbon (PAH),

and nitrogen dioxide. For outcomes, asthma and wheeze were used as search terms.

Papers not considered

Studies that examined associations with ambient air pollution without assessing the local impact of traffic [17–25] were not considered. We also did not consider papers in languages other than English [26], papers presenting data from ecologic studies [27,28], and papers that discussed exposures that are relevant to studies of proximity to traffic but measured in nontraffic settings (farm setting [29], indoor air [30–33]). We also excluded one paper that used doctors' house calls for asthma because this outcome does not represent population-based asthma prevalence [34].

Findings from cohort studies

Brauer *et al.* [4**] followed a birth cohort of 3538 children in the Netherlands. Initially, PM_{2.5}, soot and NO₂ were measured at 40 sites to capture maximum variability of traffic sources. In a land-use regression (LUR) model, three GIS-based traffic metrics near monitoring stations (number of high-traffic roads within 250 m, presence of major road within 50 m and housing density within 300 m for home address at birth) and an indicator variable for region could explain 73, 81 and 85% of the variability in annual average PM_{2.5}, soot and NO₂, respectively. This modeling approach was used and validated earlier [35–37]. This model was then applied to similar GIS-based traffic metrics near residence at birth to estimate annual average air pollution concentrations at residence location. These three pollutants were highly correlated ($r=0.93-0.97$) and showed a similar magnitude of associations with asthma and wheeze outcomes by 4 years of age. Per interquartile range (IQR) increase in NO₂ (i.e. 10.6 µg/m³) was associated with a 19% [95% confidence interval (95% CI), 1.05–1.34] and 28% (95% CI, 1.04–1.56) increase risk of wheeze and doctor-diagnosed asthma, respectively. Approximately half of the study population did not move from their residences at birth, and similar results were found in this restricted sample.

In a prospective birth cohort study, Ryan *et al.* [5**] used the UNMIX receptor model to determine sources of elemental carbon from truck and bus traffic using data collected at 24 monitoring sites in the Cincinnati metropolitan region in Ohio, USA. Subsequently, elemental carbon levels attributable to traffic sources (ECAT; a marker for exposure to diesel exhaust particles) were calculated using UNMIX and chemical mass balance models. LUR models fitted with GIS-based traffic metrics (average daily truck count on major roads within 400 m and length of bus routes within 100 m of home) and elevation could explain 75% of the variability in log-transformed ECAT levels at the monitoring sites. The

final LUR model was validated by simulation studies using data from 19 monitoring sites to predict ECAT levels in the remaining sampling locations. Using the parameter estimates from the final LUR models and the GIS-based exposures, ECAT levels at the infants' home were estimated. There was a significant increase in the risk of wheeze without cold in the first year per 0.1 µg/m³ increase in modeled ECAT levels near home. Compared with children exposed to 0.2 µg/m³ ECAT levels, those exposed to 0.9 µg/m³ were at a 4.3-fold (95% CI, 1.06–17.26) increased risk of wheeze without cold.

In an earlier paper [38], the authors reported that children exposed to 'stop-and-go traffic' (residence within 100 m of a bus route and/or 100 m of a state route with less than 50 miles/h speed limit) near homes had a 2.5-fold (95% CI, 1.15–5.42) increased risk of wheeze without cold compared with 'unexposed' children (living >400 m from an interstate route and >100 m from state and bus routes). Children exposed to moving traffic (living within 400 m of an interstate route and/or state route with ≥50 miles/h speed limit) had no statistically significant increased risk. Although modeled ECAT levels were significantly higher in children exposed to stop-and-go and moving traffic than those unexposed, no difference was found in modeled ECAT levels between stop-and-go and moving-traffic exposures. The authors argued that children exposed to stop-and-go traffic were more likely to belong to a background of low socioeconomic status without air conditioning, which may have resulted in higher exposures. The authors also acknowledged that the LUR models may have underestimated ECAT levels because air sampling sites were mainly located near interstate highways. In addition, the type of traffic (stop-and-go compared with moving) was not evaluated for estimating ECAT levels at sampling sites and infants' homes, which could have showed differences in ECAT levels.

Using linear regression, Morgenstern *et al.* [6] found that GIS-derived exposures [e.g. distance to nearest federal road, motorway (freeway), different buffers for length of rural/county/state roads, and household and population densities] could explain 51 and 36% of the variability of PM_{2.5} and NO₂, respectively. The variability in pollutants explained by these traffic metrics were lower than studies discussed above, and this could be due to not considering traffic counts and/or type in the model. These two pollutants were weakly correlated ($r=0.45$). Residential levels of these pollutants were estimated for 3577 infants who participated in two prospective birth cohorts in Germany. Residential distance of less than 50 m from the nearest main road was associated with a 23% increased risk (95% CI, 1.00–1.51) of asthmatic bronchitis at 2 years of age but not at age 1. NO₂ (per IQR of 5.7 µg/m³) and home distance of less than 50 m

from nearest major road were associated with increased risk of sneezing/runny nose at age 2, whereas PM_{2.5} (per IQR of 1.04 µg/m³) was associated with increased risk of sneezing/runny nose at ages 1 and 2. However, residential distance from a main road and modeled pollutants were not associated with wheeze.

Findings from case-control studies

Modig *et al.* [7] conducted a nested, matched, incident case-control study of asthma among adults (20–60 years) living in Luleå, Sweden. Incident cases of asthma were verified clinically and age- and sex-matched controls were selected from the Swedish population register. A total of 203 cases and 203 controls were enrolled. Traffic flow was calculated by summing mean vehicle count/24 h on weekdays on each road within a 200 m buffer of a home. A vehicle count of 100 vehicles/24 h was assigned for about 50% sample with no traffic-flow data. Correlation of traffic flow with estimated annual average NO₂ (modeled on a 1-week measurement outside home) was weak ($r=0.38$). Although exposure to high traffic flows (75th percentile cut-off, 9700 vehicles/24 h) was associated with a nonsignificant 50% increased risk of asthma, modeled NO₂ was not associated with asthma.

Findings from cross-sectional studies

In a school-based survey of 756 children (age 5–7 years) attending 13 schools in Anchorage, Alaska, USA, Gordian *et al.* [8[•]] calculated traffic density within 100 m of the closest intersection to the child's residence (measured as vehicle meters) by summing the product of the length of each road segment within the buffer and the traffic count of that road. Exposure to 'high' traffic (vehicle meter $>8 \times 10^6$) was associated with a 2.8-fold increased risk of asthma (95% CI, 1.23–6.51) compared with children exposed to 'low' traffic (vehicle meter $<4 \times 10^6$). This association varied by parental asthma (interaction P value = 0.01) and by exposure to smoking at home (interaction P value = 0.05). Exposure to high traffic was associated with a more than 5-fold-increased risk of asthma in children without parental asthma and in those exposed to smoking at home; however, no significant association was found in children with parental asthma or those unexposed to smoking at home.

Among 4762 children (5–7 years) living in 13 southern Californian communities, McConnell *et al.* [9^{••}] found that children living within 75 m of a major road were at increased risk of lifetime (doctor-diagnosed asthma by study entry) and prevalent (12 months before study entry) asthma and current wheeze compared with those living more than 300 m from a major road. These associations, which were stronger in long-term residents, varied significantly by parental asthma and by sex. Girls and children without parental asthma had a significantly increased risk

of asthma outcomes if they lived within 75 m of a major road. In contrast, living near a major road was not associated with asthma/wheeze outcomes in boys and in children with parental asthma. In addition, a line source dispersion model that accounted for traffic volume and wind speed and direction was used to model total NO_x levels and NO_x from freeway and nonfreeway sources. The modeled pollutant levels were poorly correlated with home distance from a major road. Only NO_x from nonfreeway sources was significantly associated with lifetime and prevalent asthma and current wheeze.

In two recent papers, investigators of the southern Californian Children's Health Study (including the authors of this review) reported that the relationships of genes involved in oxidant stress and PAH metabolism pathway with asthma varied significantly with traffic near homes [10^{••}, 11^{••}]. In one paper, we reported that the association between a functional promoter polymorphism in the transforming growth factor β1 (TGFβ1) gene (i.e. C-to-T base substitution at position –509) and childhood asthma varied significantly by distance of home from the nearest freeway [10^{••}]. Children with the –509TT genotype living within 500 m of a freeway had a more than 3-fold-increased lifetime asthma risk (95% CI, 1.29–7.44) compared with those children with the –509CC/CT genotype living more than 1500 m from a freeway. In a second paper, the associations between two genes in the PAH metabolism pathway [epoxide hydrolase (EPHX1) and glutathione S-transferase P1 (GSTP1)] and asthma varied by home distance from a major road [11^{••}]. EPHX1 metabolic phenotypes were determined using genotypic combinations of two functional polymorphisms (T113C and A139G), as described earlier [39]. Among children living within 75 m of a major road, those with high EPHX1 activity had a 3.2-fold (95% CI, 1.75–6.00) higher asthma risk than those with low/intermediate activity. The study also reported that children with high-EPHX1 phenotype and GSTP1 105Val/Val genotype who lived less than 75 m from a major road had almost a 9-fold-increased risk compared with those with low/intermediate EPHX1 activity and GSTP1 105Ile/Ile genotype living more than 75 m from a major road.

Presenting data from the 2001 California Health Interview Survey 2001, Meng *et al.* [12] calculated vehicle miles traveled (VMT/mi²) by summing the product of annual average daily traffic and road segment length for all road segments within a 500-ft buffer around home. High, medium and low traffic densities were based on VMT/mi² values of more than 200 000, between 20 000–200 000, and less than 20 000, respectively. High traffic density as defined here was associated with higher prevalences of asthma-related emergency department visits/hospitalizations in children (0–17 years) and adults (≥ 18 years) and

daily/weekly asthma symptoms in adults. Subsequently in another paper, they reported that high traffic density was associated with over a 2-fold-increased risk (95% CI, 1.38–3.23) of poorly controlled asthma in adults [13]; however, data on children were not reported. Traffic density (as a continuous variable) was poorly correlated with annual average traffic-related pollutants ($r=0.14$, 0.13 and 0.11 for PM₁₀, PM_{2.5} and NO₂, respectively).

Bayer-Oglesby *et al.* [14] examined the associations between three GIS-based traffic metrics (distance to closest main street, main street segments within 200 m of home, and living within 20 m of a main street) and respiratory symptoms (attacks of breathlessness, wheezing with breathing problem, wheezing without cold, regular cough, and regular phlegm) in adults. Whereas per 500 m of main street segments within 200 m of home was associated significantly with a 13% increased risk of attack of breathlessness and living within 20 m of a main street was associated with a 15% increase in risk of regular phlegm, no other significant associations were seen for other outcomes or with distance to main street from home. Given so many odds ratios are presented, chance could not be excluded as an explanation for the findings.

Two studies used parental/self report of traffic intensity near homes [15,16[•]]. In a clinic-based study conducted among 204 Asian (Chinese) children (4–18 years) living in the USA, Brugge *et al.* [15] reported that living near heavy traffic was associated with increased asthma risk (odds ratio = 2.6; $P=0.01$) in a model that included family history of asthma and place of birth but not other potential confounders (e.g. age, sex, socioeconomic status). Kuehni *et al.* [16[•]] found that parents of children with respiratory symptoms over-reported higher traffic intensity than parents of asymptomatic children living in the same zip codes. They concluded that subjective report of traffic exposures could positively bias a null association.

Discussion

Data from these studies provide supporting evidence that residential exposure to traffic is associated with asthma occurrence and exacerbations. However, cohort studies that reported associations with respiratory symptom outcomes in infants need further follow-up data to adequately address the effects of traffic exposures on asthma occurrence in children [5^{••},6]. In terms of exposure assessment, validated LUR models, which incorporated spatial and meteorological data, could better predict residential levels of air pollution from traffic sources. Two studies reported modifying effects of susceptibility factors (parental asthma and sex) in the associations between residential traffic exposures and asthma occurrence [8[•],9^{••}]. In addition, two studies found that the

relationship of functional variants in genes in the oxidant stress pathway (TGF β 1, EPHX1, and GSTP1) and asthma varied by residential traffic exposure.

Review of these studies suggests that inconsistencies in findings across these and previously published papers could be attributed to the use of different methodologies in assessing local traffic-related exposures, use of self/parental report of traffic intensity, missing data on traffic counts, and moderate to poor correlation between each traffic metric and modeled ambient traffic-related pollutants. A publication bias could not be ruled out because most of the reviewed papers found some significant associations. The GIS technique was not fully utilized to determine traffic metrics in some studies [7,8[•], 10^{••},11^{••}]. A strong limitation of these studies lies in presenting data on one traffic metric. This also raises concern of reporting bias because each of these papers reported some statistically significant associations with the traffic metric used.

In addition to individual traffic proximity metrics (distances of road and freeway, and traffic density within a specified buffer around home), models have been used with different traffic and other spatially important factors (elevation, wind speed and direction, population density, and housing density). Although these models could predict 60–80% of the variability in traffic-related pollutants (especially NO₂, NO_x, and particulate matter) in some of these studies, the variables included in these prediction models varied across studies [4^{••},5^{••}]. This could be due to spatial differences by geographical location, differences in traffic density, type of traffic, number of road segments within a given buffer, and use of different modeling approaches. Traffic-related ambient pollutant data that were collected from air monitoring stations located further away from residences [9^{••}] or measured pollutants for 1–2 weeks at residences [7] were used to estimate annual average pollutant levels. This may not have adequately captured traffic-related pollution levels near homes, which may have resulted in less than optimum predictability of the LUR models. In addition, none of the studies to date incorporated activity patterns in time and space (home, school, commute, and workplace) into exposure assessment, which could better determine personal level exposure. Thus, these exposure misclassifications may have attenuated the risk estimates.

A limited number of studies have also reported that the relationship between residential traffic exposures and asthma varied by susceptibility factor [8[•],9^{••}]. McConnell *et al.* [9^{••}] found that distance of home from nearest major road had statistically significant associations in girls, in children of nonasthmatic parents, and in children with no history of allergy, whereas no statistically significant associations were found in boys and in children with

no allergy or those with no parental asthma. Gordian *et al.* [8[•]] also found significant associations between traffic exposure and asthma occurrence in children with no parental asthma and in those exposed to smoking at home. Further research is warranted to elucidate the nonsynergistic effects of traffic exposures with genetic susceptibility factors for asthma occurrence in children with parental asthma.

Additionally, stronger associations with traffic were found among long-term residents. This finding may reflect that early life exposure could be a critical window of susceptibility. Therefore, a careful consideration of critical windows of exposures and modifying effects of genetic susceptibility factors for each asthma phenotype (age at onset and persistence of symptom based) could provide a better understanding of the biological pathways of traffic mediated effects. Oxidant stress-mediated airway inflammation is one of the underlying mechanisms for increased asthma risk in children from residential traffic exposures. Therefore, variants in some candidate genes that could potentially increase oxidant stress in the airways may predispose individuals to greater asthma risk. Recent reports from the Children's Health Study support this hypothesis [15,16[•]] and emphasize the importance in examining further the role of other genes in the oxidant stress pathways.

These findings have significant public health and policy implications. One study found housing and population densities predicted air pollutant exposures [4^{••}]. In another study, daily average truck count on major roads within 400 m significantly predicted traffic-related pollutant levels [5^{••}]. In addition, presence of schools near busy streets could also affect children as they spent a considerable amount of time on the playground. Therefore, policy makers need to consider the public health impact of housing and transportation and could set better standards for fuels and engines to reduce traffic exposures. Air-quality regulatory agencies may need to consider monitoring traffic emissions near busy roads and freeways, which is likely to reduce misclassification in exposure assessment near homes.

Conclusion

Current evidence shows that residential proximity to traffic sources increases asthma occurrence and exacerbations. Among children, the risk from traffic appears to be higher in children with no parental asthma. Further investigation into the modifying effects of genes in oxidant stress and inflammatory pathways could provide better understanding of the asthma pathogenesis as well as the differential effects of traffic in atopic and nonatopic asthma. Improvement of the prediction models for exposures by measuring pollutants near homes and

schools and by considering time/activity patterns is also warranted.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 83–84).

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This longitudinal study in children finds that traffic density near homes caused increased levels of exhaled NO and reduced lung volumes in children with asthma.
- 2 Gauderman WJ, Vora H, McConnell R, *et al.* Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *Lancet* 2007; 369:571–577.
This study finds that living within 500 m of a freeway significantly reduces 8-year lung-function growth in children aged 10–18 years.
- 3 Kan H, Heiss G, Rose KM, *et al.* Traffic exposure and lung function in adults: the Atherosclerosis Risk in Communities study. *Thorax* 2007; 62:873–879.
This study finds that traffic density near home significantly reduces lung function in women but not in men.
- 4 Brauer M, Hoek G, Smit HA, *et al.* Air pollution and the development of asthma, allergy and infections in a birth cohort. *Eur Respir J* 2007; 29:879–888.
In this cohort study, validated methods and LUR models were used to estimate annual average air-pollution concentrations at residence locations. Significant associations were found between residential traffic-related pollutant levels near home and asthma and wheeze occurrences in children.
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In this paper, the investigators of the Children's Health Study report that children with a functional promoter polymorphism in the TGFβ1 gene were at higher asthma risk if they lived near freeways. This is one of the first studies to explore gene–environment interaction with residential traffic exposures.
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This study using data from the Children's Health Study finds that children with functional variants in key genes in the PAH metabolism pathway are at increased risk of asthma if they live near major roads. This paper also explored gene–environment interaction with residential traffic exposures.
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