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Air pollution and respiratory health of young children

Think globally, breathe locally

R J Delfino

Why the worldwide health impact of air pollution on young children begins in our neighbourhoods

To date, most epidemiological studies of paediatric pulmonary disease and air pollution have focused on the impacts of air pollutants that are both regulated and monitored on a daily basis by governments. This includes particle mass concentrations and gaseous air pollutants such as ozone and sulfur dioxide. In most studies this has led to the use of available regional air monitoring data. Generally, these approaches have resulted in important new information about ongoing health impacts in many nations or have confirmed previous findings of adverse associations with respiratory morbidity. This has justified calls for greater involvement by citizens, local community organisations, industry, and governments to curb air pollutant exposures.[1] However, there is increasing evidence that concentrations of air pollutants—especially particle components and size distributions not routinely monitored—have spatial distributions that can vary by neighbourhood. In urban areas the most prevalent determinant of sub-regional air pollutant concentrations is local traffic. Roadway traffic will continue to increase worldwide, as evidenced by projections that transportation energy consumption in emerging economies such as China will increase at an annual rate from 2002 to 2015 of 4.4 quadrillion Btu (1055.1 Joule/Btu).[2]

WHAT IS IMPORTANT ABOUT PROXIMITY TO TRAFFIC?

Concentrations of ultrafine particles <100 nm in diameter are influenced strongly and positively by distance from roadways.[3] There is also growing evidence that photochemically generated ultrafine particles are a potentially important regional exposure.[3] Ultrafine particles have low mass concentrations in air compared with regulated particulate matter <10 μm in diameter (PM_{10}). However, the large surface area per unit mass of ultrafine particles can carry to the lungs large quantities of toxic air pollutants including oxidant gases, organic compounds, and transition metals.[4] This toxicity of ultrafine particles is combined with their high pulmonary deposition efficiency and high particle number concentration which are magnitudes higher than larger particles. The traffic related increase in ultrafine particles is additional to a parallel increase in a plethora of pollutant gases and volatile and semi-volatile organic compounds that can undergo chemical transformations and can attach to the elemental carbon core of ultrafine particles.

Many organic compounds associated with vehicle emissions such as polycyclic aromatic hydrocarbons, along with transition metals, have been identified as having adjuvant effects on cytokine mediated airway inflammation, in part through oxidative stress mechanisms.[5–8] This process has been linked to the enhancement of allergic respiratory responses to airborne allergens and may be involved in the onset of atopy.[9] Evidence for this has come primarily from studies that have used diesel exhaust particles as a model exposure since this source is particularly rich in redox cycling compounds.[8]

The development of respiratory allergic phenotypes is thought to begin early in life through a failure of the T cell population to mature adequately from a Th2 to Th1 subtype, thus driving an imbalance toward Th2 immunity.[10] The adjuvant effects of air pollutants on this shift may begin in utero, as supported by evidence from a cohort study of an increased risk of childhood asthma and wheezing following in utero exposure to environmental tobacco smoke.[11] In addition to risks attributable to a developing immune system, young children are also susceptible to adverse health effects of air pollutants because they generally have higher levels of activity compared with adults, and have greater minute ventilation rates per body weight leading to higher doses of irritants. Submicron particle dose in the pulmonary region has been predicted to be particularly high among 3 month old children compared with adults.[12] These factors are coupled with the likelihood that young children often play outside near traffic sources.

THE RESPIRATORY HEALTH OF CHILDREN AND EXPOSURE TO TRAFFIC RELATED AIR POLLUTION

In this issue of *Thorax* Pierse *et al*[13] provide evidence for the importance of traffic related air pollutants near the home to the respiratory health of children aged 1–5 years surveyed in 1998 and again in 2001 when aged 4–8 years. The authors found that parent reported prevalence and incidence of cough without a cold and the incidence of wheeze were positively associated with an increase in the estimated concentration of PM_{10} from sources near the homes of children aged 1–5 years. The estimations of PM_{10} were based on a dispersion model that was tailored for the study area (Leicester) and was primarily based on traffic flow and wind speed and direction in relation to the home address. However, as discussed above, ultrafine particles and associated toxic air pollutants are more strongly influenced by proximity to traffic than larger particle size fractions included in PM_{10}.

The study by Pierse *et al*[13] adds to a growing trend in epidemiological studies of air pollution to reduce reliance on available regional air monitoring data by supplementing or supplanting it with data intended to estimate exposures closer to the individual participant. The goal of methods to estimate personal exposure is to reduce misclassification of air pollutant exposures. Exposure misclassification is expected to occur when the same regional exposure is applied to all study subjects regardless of distance to air monitors and of potentially important local pollutant sources. This can often limit studies to make cross sectional comparisons between cities rather than within cities, and opens the door for unmeasured confounding by factors that differ between regions. The ideal exposure measurement is to use personal air monitors, but this becomes impractical with a large study population such as the cohort of 4400 children studied by Pierse *et al*.[13] Methods to achieve a better estimation of personal exposures include local source dispersion models[14] and microenvironmental models that

predict ambient (outdoor) and non-ambient particle exposure using, in part, time-activity and pollutant measurement data at various indoor and outdoor locations.[15]

Although the clinical relevance of increases in parent reported symptoms is unclear, the findings of Pierse *et al*[13] add coherence to other findings for different health outcomes, some with clear clinical relevance. High home or school traffic density has been associated with the prevalence of diagnosed asthma or allergic sensitisation in cross sectional and case-control studies of children and adults (reviewed by Delfino *et al*[16] and Heinrich and Wichmann[17]). Brauer *et al*[18] conducted a birth cohort study in the Netherlands beginning with around 4000 women in their second trimester. They found predicted traffic related exposure to $PM_{2.5}$, $PM_{2.5}$ absorbance (black carbon soot as a marker of diesel exhaust), and NO_2 was weakly associated with the incidence of wheezing, physician-diagnosed asthma, ear/nose/throat infections, and flu or serious colds by 2 years of age. Exposure estimations were based on measured pollutants from 40 sites selected on variability in traffic, then linked with traffic near subject homes using geographical information systems (GIS).[14 19]

In another birth cohort study Gehring *et al*[20] also used GIS based exposure modelling to estimate traffic related air pollutants at the birth addresses of 1756 infants living in Munich. They found that estimated $PM_{2.5}$, $PM_{2.5}$ absorbance, and NO_2 were significantly associated with cough without infection and dry cough at night at 1 year of age (odds ratios 1.32–1.40), but associations were weaker at 2 years of age. These results are consistent with those of Pierse *et al*[13] who additionally found positive associations for incident cough without a cold at ages 3–5 (odds ratio 1.71, 95% CI 1.26 to 2.31). The results are particularly comparable because exposure modelling methods aimed to achieve better spatial resolution of traffic exposures.

Ryan *et al*[21] studied over 600 infants at 6 months of age in Cincinnati, Ohio and found that infants living <100 metres from stop-and-go bus and truck traffic had a significantly increased prevalence of wheezing without a cold (odds ratio 2.50, 95% CI 1.15 to 5.42) when compared with unexposed infants. There was no association for infants living 100–400 metres from a high volume of moving traffic, suggesting that ultrafine particles and associated diesel emissions were driving the association with wheezing. Findings also suggest that roadways other than expressways (freeways) are important. This is supported by evidence that ultrafine particle number concentrations are high at intersections, including times when gasoline powered vehicles accelerate hard or from a standing start.[3]

GOING FORWARD

The importance of the above studies of airway disease in young children is that, until recently, most air pollution research has focused on older children and adults. The articles reviewed above also point to a growing view that air pollutant exposures are best assessed closer to where children live and go to school. Determining the importance of air pollutants from nearby traffic for the development of chronic respiratory diseases will require following cohorts from pregnancy and birth onward. This is because asthma and respiratory allergic diseases are expected to have their beginnings in early life, possibly from exposures in utero, in combination with genetic susceptibilities. It will also be necessary to assess other community level differences that lead to health disparities—for example, in paediatric asthma.[22]

In light of the evidence outlined above, the impact of high traffic exposure on a neighbourhood scale on paediatric respiratory health could be larger than estimates using regional pollution data. The global impact could be considerable given the ongoing growth of urban populations, increasing dependence on fossil fuel, and considerable worldwide variability in air pollution exposure near roadways. This is of greatest concern in developing nations that have not yet instituted strict emission control programmes.[23]

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