The health impacts of local traffic pollution on primary school age children

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Suggested running head: Health impacts of local traffic pollution on primary school children

Key words: traffic pollution, health impact, child health

Word Count: 4883

Abstract

There is a large body of medical research on the health impacts of local traffic air pollution. Vehicle emissions, road dust and tyre fragmentation together cause an increase in atmospheric levels of carbon monoxide, sulphur dioxide, nitrates and particulate matter in both solid and liquid forms. For those exposed to such pollution, there is a documented increase in mortality and morbidity from both respiratory and cardiac diseases. There is also evidence that the effects are greater on children. Despite these findings, there has been comparatively little work done in estimating the effect on student health of school location, with most studies focusing instead on residential location. By combining a random sample of primary schools in Sydney with detailed traffic volume data, we estimate the impact of traffic pollution on the health of primary school children in Sydney.

1 Introduction

This study examines the effect of local traffic pollution on the health of primary school age children in Sydney. The health effects of urban air pollution generally, and traffic pollution specifically, have been the subject of many studies (Kunzli et al., 2000; Brunekreef and Holgate, 2002). It is now generally known that adverse health impacts (along with congestion) is a major externality caused by traffic. Most studies have estimated health impacts on the population generally (see Fisher et al. (2007)), as estimating health impacts on demographic sub-groups in a population is not possible. It is, however, well known that children are more seriously affected by air pollution than adults, and so this group in particular has been the subject of many studies. Most child-specific studies have focused on residential location. This study looks at the impact of traffic pollution at school on student health.

We follow, in Section 1.1, with a brief overview of the types of pollution attributable to motor vehicles. This is followed, in Section 1.2, by a review of the effects of pollutants on population health. Our specific analysis of primary schools in Sydney then commences in Section 2.

1.1 Atmospheric Pollution from Motor Vehicles

Air pollution from motor vehicles consists of a mixture of gases, liquid droplets and solid particles. Many of these substances have a known association with disease in humans (Brook et al., 2004).

Carbon monoxide, sulphur dioxide and oxides of nitrogen are released from the exhaust pipes of internal combustion engines. Ozone, which is the principal component of photochemical smog, is

produced by the action of solar ultraviolet radiation on nitrous oxide. All of these gases have been shown to be toxic to humans.

Both solid and liquid particles of various sizes are released into the atmosphere from motor vehicles. Primary particles, which arise from engine emissions, consist of droplets of unburned diesel or petrol mixed with solid carbon (soot). Secondary particles arise from tyre fragmentation and also from the suspension of road dust. Although there are literally thousands of chemicals in the mixture, organic compounds such as aromatic hydrocarbons and inorganic chemicals such as heavy metals are known to exhibit toxicity.

Airborne particles with a diameter of less than 10 microns can easily penetrate into the trachea and main bronchi of the human lung where they usually form a deposit. Such particles are known as PM_{10}. Particles of less than 2.5 microns ($PM_{2.5}$) can reach the small airways and even the alveoli where gas exchange occurs. Very small particles (less than 0.1 microns) have a high surface to mass ratio which increases their toxic potential. Such particles can even pass directly into the circulation.

1.2 Diseases Caused by Air Pollution

There have been hundreds of publications linking atmospheric pollution with disease in humans. Interest in this subject rose sharply after the London fog of 1952 which was associated with a huge increase in morbidity and mortality from both cardiovascular and respiratory diseases (Bell and Davis, 2001).

Asthma is a common disease that is characterised by inflammation of the lungs. This results in spasm of the smooth muscle in the small airways (known as bronchospasm) and the production of a

sticky mucous. These two effects cause airway obstruction resulting in cough, wheeze and breathlessness. The disease can be severe and deaths do occur. Although the condition has a genetic basis, attacks are initiated by various triggers including allergy, infection, exercise and air pollution. Both hospital admissions and outpatient attendances for asthma have been associated with the level of regional air pollution. This is typified by a large American study of the relationship between hospital encounters for children with asthma and the degree of exposure to traffic related air pollution. The study found that adjusted hazard ratios for inter-quartile range increases in nitrogen oxides and carbon monoxide were 1.10 and 1.07 respectively (Delfino et al., 2009). This study and numerous others (Edwards et al., 1994; English et al., 1999; Lin et al., 2009) clearly show that the severity of asthma in children is related to the level of exposure to motor vehicle emissions.

Viral infections of the respiratory tract are very common in children but most of these are confined to the nose and throat. The incidence of chest colds (that is the spread of infection to the lungs) in children has been shown, in an Australian study, to be related to levels of atmospheric pollution as low as 18.6 $\mu g/m^3$ of PM_{10} particles. The study also found there is a significant increase in the incidence of chest colds and night time cough for every additional 10 $\mu g/m^3$ of PM_{10} (odds ratio 1.43 and 1.34 respectively) (Lewis et al., 1998). Numerous international studies have found a link between respiratory problems in children and residential or school proximity to vehicle traffic (Kim et al., 2004; Peel et al., 2005; Lin et al., 2009).

For children and adults with pre-existing respiratory diseases such as asthma, emphysema, bronchiectasis and cystic fibrosis, deaths increase by 3.4% for every increase in air particles by 10 $\mu g/m^3$ (Brook et al., 2004).

Lung cancer is one of the most common malignant tumours in humans and it is also one of the most lethal. Data from the American Cancer Society in 2002 estimated an 8% annual increase in the long term risk of death from lung cancer for each increase in exposure to $PM_{2.5}$ particles of 10 µg/m^3 (Pope III et al., 2002).

The American Heart Association has estimated that there are 60,000 deaths annually in the USA due to particle inhalation (Brook et al., 2004). There is now evidence from several studies to indicate that air pollution may accelerate the development of coronary atherosclerosis and that a transient exposure to high levels of particulate matter is associated with an increased risk of acute myocardial infarction (heart attack). The large epidemiological study of traffic pollution in Europe by Kunzli et al. suggested that 3% of total mortality (20,000 cases per year) could be attributed solely to traffic pollution (Kunzli et al., 2000).

2 Data & Method

2.1 Sampling of schools

Schools are selected using random stratified sampling of primary schools across the four main school regions covering the Sydney metropolitan area: Sydney, South-West, Northern Sydney, and West. Twenty primary schools were randomly selected from each of these regions from a list of primary schools obtained from the NSW department of education and training. Schools for special purposes (i.e. special education, arts, sports) were not included.

For each of the schools selected, we calculated the distance from the school 'centroid' to the nearest major road. The school 'centroid' was chosen through a combination of examination of

aerial photographs, personal inspection, and virtual inspection through the use of Google Streeview. The general aim was to select a point representative of the exposure of an average student at the school. Where a school was comprised of a number of buildings, this was the point at the centre of these buildings. In schools with a large established building and some demountable classrooms, the distance to the main building was taken.

2.2 Population considered

School populations consist of both children and adults such as teachers, other employees and volunteers (usually parents). This paper, describing work in progress, only considers the effect of traffic pollution on school children.

2.3 Pollutants considered

Our focus in this paper is on the health effects of localized traffic pollution. Because of this, we will not consider the health effect of general urban-area pollution, but will instead concentrate on the net effect of localized traffic pollution.

The main pollutants already mentioned in the introduction are: sulphur dioxide, carbon monoxide, oxides of nitrogen, ozone, and particulates (PM_{10}, $PM_{2.5-10}$, $PM_{2.5}$). Because we are interested in the local effects of school proximity to traffic, we need to consider which of these pollutants are likely to have local area effects. Table 1 shows which pollutants are considered in this study.

Pollutant	Considered?	Reason
Sulphur dioxide	No	Australia's vehicle fleet is predominantly petrol-based, with diesel use restricted almost exclusively to heavy commercial vehicles. As a consequence, sulphur dioxide emissions in Australia are relatively low, and we do not consider them in this study.
Carbon monoxide	Yes	Carbon monoxide is emitted directly from the exhaust pipes of vehicles. It is dispersed through diffusion and wind, but wind is by far the more important factor[1]. Carbon monoxide levels are measurably higher in the vicinity of busy roads (Zhu et al., 2002), and are known to affect respiratory health in adults and children.
Nitrogen oxides	Yes	Nitrogen oxides are, like carbon monoxide, emitted directly from the exhaust pipes of vehicles, and are primarily wind dispersed. As in most other studies, we focus specifically on nitrogen dioxide, which, like carbon monoxide, is measurably higher near busy roads (Gilbert et al., 2007), and affects the respiratory health of adults and children.
Ozone	No	Ozone is produced by the action of solar ultraviolet radiation on nitrous oxide, but this process takes place over a time period long enough for us to consider ozone a wide-area pollutant.
Particulates	No	Numerous studies into the health effects of pollution have concluded that particulate matter is an important cause of increased morbidity and mortality (Kunzli et al., 2000; Brook et al., 2004; Fisher et al., 2007). However, the dispersal mechanism for particulate matter results in widespread dispersal, rather than local concentration. As a consequence, particulate concentrations near main roads are typically similar to general background levels (Kim et al., 2004), at least for particulates larger than 50 nanometres (Zhu et al., 2002, page 4329, Fig. 5). PM_{10} particles are 200 times this size. Thus, while particulate matter is known to have adverse effects on adults and children, it is generally unrelated to local traffic, and so we do not consider it.

Table 1: Pollutants resulting from traffic.

2.4 Heath Effects Model

As discussed in the introduction, traffic pollution is associated with many illnesses, and also with increased mortality. However, estimating increased mortality in children as a result of pollution is difficult, as increased mortality is only likely to present itself later in life. There is evidence that childhood exposure to local traffic pollution results in reduced lung function later in life (Gauderman et al., 2006), but no study has been able to characterise the relationship precisely enough for us to include estimates of these long-run effects in our study. Given that the effect of childhood exposure to pollution on long-term mortality and morbidity is difficult to estimate, we choose to use a more immediate measure – hospitalisations for asthma – as an indicator of the health impact of traffic pollution. We focus specifically on admissions due to asthma as this allows us to use hazard ratios from a recently published study (Delfino et al., 2009). Hospital admissions for respiratory illnesses generally are associated with traffic pollution levels (see Peel et al. (2005) and Lin et al. (2009)), so asthma admissions are a useful proxy indicator.

The specific study we rely on (Delfino et al., 2009) gives hazard ratios of 1.07 for each 0.056 mg/m^3 of CO and 1.1 for each 4 µg/m^3 of NO$_2$.

2.5 Traffic and pollution

CO and NO$_2$ levels are dependent on three things: fleet emissions (the emission profile of vehicles passing the school); traffic volume (the number of vehicles passing the school during school hours), and; proximity (the distance of the school from the vehicles). Other factors, such as local weather and topography will have an effect, but for simplicity these factors were not considered.

Emissions of CO and NO_x can be calculated directly using Australian national emissions standards for motor vehicles. However, actual road-side measurements in Australia conducted by Bluett et al. (2008) suggest that this method underestimates actual pollutant levels. We use the estimates in Bluett et al. (2008, page 70), which are based on remote sensing data. In the absence of detailed data describing the fleet profile on Sydney's roads, we assume a uniform fleet composition identical to that used in Bluett et al. (2008, page 77).

The NSW Roads and Traffic Authority publishes traffic volume data for many of Sydney's roads (see NSW Roads and Traffic Authority (2005)). Where hourly traffic volume data is available, mean weekday hourly traffic volume is calculated for the 8 hours 8-4. Average traffic volume between these hours is assumed to be indicative of the pollutant levels experienced by students at school. Students are assumed to be exposed to pollution from this traffic for a period of 6.5 hours per day. Where hourly volume data is unavailable, daily traffic volume data is used, with traffic volume in the hours 8-4 assumed to be 50% of total 24-hour traffic. If no traffic volume data is available, local traffic volumes are assumed to be 750 vehicles per hour for non-local roads[2]. Traffic volume on local roads is not considered, as pollutant levels near these will be representative of general background pollutant levels.

Knowing fleet emissions and traffic volume allows us to calculate CO and NO_2 levels at the road. In order to calculate pollutant levels at a distance from the road, a pollutant dispersal model must be used. Complicated dispersal models are available, which simulate the physical dispersion of pollutants and account for local topography and wind, but we use a simpler method. We consider the road a point source of emissions, with the school 'centroid' (described above in Section 2.1) being indicative of the exposure level of students at that school. Given on-road concentrations of

CO and NO$_2$, we assume CO and NO$_2$ both decay exponentially with distance from the road. Empirical studies suggest this is a reasonable assumption: Zhu et al. finding a decay exponent of -0.033 for CO (Zhu et al., 2002, page 4331, Fig. 7a) and Gilbert et al. finding a decay exponent of -0.0254 for NO$_2$ (Gilbert et al., 2007, page 2671, Table 1). Figure 1 shows the exponential decay of CO and NO$_2$ with distance from traffic.

Figure 1: Decay model for CO and NO$_2$ with distance from the road.

When calculating pollutant levels, all roads within 100m are considered. Roads further than 100 metres from the school are not considered, as beyond this distance, the exponential decay results in low pollution levels even for high volume roads. Existing literature also typically supports the claim that it is only the closest roads that have a statistically measurable impact on health (English et al., 1999; Ryan et al., 2005).

2.6 Method: Example

Because the method used to calculate the health effects of exposure to CO and NO$_2$ is quite complex, we believe it is useful to provide an example of how we calculate pollution levels and the resulting increase in the chance of hospitalisation for asthma. The school we will use for this example is Coogee Public School, in the Sydney suburb of Coogee. Figure 2 shows aerial photography of the area surrounding the school. The school itself is circled, and the approximate centroid is shown.

Figure 2: Coogee Bay Public School

Hourly traffic volume data for nearby Coogee Bay Road is available, and averaging the hours 8-4 gives an hourly average traffic volume of 747 vehicles per hour, which, assuming fleet emissions identical to Bluett et al. (2008, page 77), gives traffic related concentrations of CO and NO_2 of 0.06 mg/m^3 and 11.0 $\mu g/m^3$, respectively. Note that these pollution levels are attributable solely to traffic on Coogee Bay Road, and do not include general background pollutant levels. We wish to deliberately exclude background pollutants, as our focus is on pollution attributable directly to nearby roads. The school centroid is 70 metres from the nearest major road (Coogee Bay Road). The distance decay functions shown in Figure 1 indicate that CO and NO_2 concentrations at this distance will be 10% and 17%, respectively, of the on-road concentrations. Taking distance into account, and assuming a 6.5 hour school day, the increase in daily CO exposure attributable to the school's proximity to Coogee Bay Road is $0.1 \times 0.06 \times 6.5/24.0 = 0.0016$ mg/m^3 , and the additional exposure to NO_2 is $0.17 \times 11 \times 6.5/24.0 = 0.5$ mg/m^3. Note that we convert the exposure during school hours to an equivalent increase in daily exposure levels. Some medical studies use daily exposure levels to estimate hazard ratios (e.g. Lin et al. (2009)), while others use the maximum hourly pollutant concentration during the day (e.g. Peel et al. (2005)). We take the former approach. Given that additional CO exposure is 0.0016 mg/m^3 per day, and daily NO_2 exposure is 0.5 $\mu g/m^3$ higher, we can estimate, using results from Delfino et al. (2009), the increase in the chance of hospitalisation for asthma. Delfino et al. (2009) estimate that each 0.056 mg/m^3 increase in CO results in a hazard ratio of 1.07, and each 4 $\mu g/m^3$ increase in NO_2 results in a hazard ratio of 1.1. Applying these estimates to the exposure levels calculated for Coogee Public results in a total hazard ratio of 1.015.

Note that our method assumes that indoor pollutant levels are the same as outdoor concentrations. The study of pollutant exposure at school by Janssen et al. found high correlation between indoor and outdoor pollution levels in schools in the Netherlands (Janssen et al., 2001).

3 Analysis & Results

There were 33997 students enrolled in the 80 schools sampled for this study. For each of the 80 schools the increase in the chance of hospitalisation for asthma (odds ratio) was calculated. Figure 3 shows the change in the odds ratio across the 80 schools. The x-axis gives the odds ratio for each school (shows as black squares), ranked from lowest to highest. The y-axis shows the cumulative proportion of students covered.

INSERT FIGURE 3 NEAR HERE

Figure 3: Cumulative distribution of odds ratios for the 80 schools sampled

We can see from Figure 3 that over half of sampled primary school students attend schools that have pollution levels equal to general background pollution. Those schools with higher than background pollution levels typically have odds ratios in the range $1.0 - 1.07$. A small minority of students ($\approx 5\%$) are schooled in locations that result in pollutant exposure causing odds ratios over 1.07. The highest odds ratio calculated for the sampled schools was 1.2.

INSERT FIGURE 4 NEAR HERE

Figure 4: Mean odds ratios for students in each of the four school regions covering the Sydney metropolitan area

The mean odds ratio for all students at all surveyed schools is 1.02. Figure 4 shows this average, as well as the regional average for each of the four school regions surveyed. We see that schools in the 'Sydney' region experience the highest levels of exposure to pollutants. This is to be expected, for several reasons. Firstly, schools in this region are on more valuable land, and, as a consequence, school blocks are smaller, and more heavily developed, with less 'buffer' space between classrooms/playgrounds and nearby streets. Secondly, areas closer to the city centre have more space devoted to roads, to cater for the greater volume of traffic that central areas. Thirdly, traffic volumes are higher on roads closer to the city centre, as they carry intra-urban traffic rather than just local traffic. The combination of high traffic volume and proximity to roads results in higher exposure levels for schools located close to the city centre (and, to a lesser extent, other major centres). Figure 5 illustrates the typical difference in layout between a school in the Sydney school region and a school in the Western Sydney region.

INSERT FIGURE 5 (a,b) NEAR HERE

Figure 5: Fig. (a) shows the layout of Vardys Road public school, typical of schools in Sydney's Western region. Fig. (b) shows the more compact layout of Earlwood public school, approx. 10 km from Sydney's CBD.

Nitrogen dioxide is the major cause of increased hospitalisations for asthma: of the total mean odds ratio of 1.01961, 1.01626 comes from exposure to NO_2. This is because of the low carbon monoxide emissions assumed from Australia's predominantly petrol-fuelled vehicle fleet (Bluett et al., 2008, page 78).

It is worthwhile remembering that we did not have road-specific data on traffic composition, and have assumed a uniform vehicle fleet. For schools on roads with a disproportionately high number heavy commercial vehicles, pollutant levels could be significantly higher than estimated. Also worth noting is that because pollution declines exponentially with distance from the road, and we estimate pollutant exposure levels only for a single point at each school, we underestimate the exposure of some school-children (those in buildings closer to the road than the school centroid) and overestimate it for others. Because pollution declines exponentially with distance, the underestimation can be significant. To see this, consider the Coogee Public school building marked with the letter A in Figure 6. While the school centroid (already shown in Figure 2) is 70 metres from Coogee Bay Road, this building is only 10 metres from the road. This results in pollutant levels ≈ 4.6 times higher than those at the school centroid, meaning that the odds ratio for hospitalisation for children in these classrooms is ≈ 1.07 rather than the general level of 1.015 estimated based on the school centroid. This same effect will occur at many of the schools in our sample. While our method of using the school centroid provides a useful (if conservative) base estimate of odds ratios, it is clear that a more detailed analysis would need to take into account the intra-school distribution of school-children. This would be a significant undertaking, requiring student numbers by building and classroom. We have not yet collected this data, but the simpler preliminary analysis presented here still provides useful initial results.

INSERT FIGURE 6 NEAR HERE

Figure 6: Closest school building at Coogee Public school (marked with an 'A') is 10m from Coogee Bay Road

4 Discussion

The general level of exposure to pollutants in the schools sampled in this survey results in a mean odds ratio of ≈ 1.02. There might seem little cause for concern with such a low odds ratio. However, the nature of school-to-school variation in pollution levels is such that focusing on the mean odds ratio is misleading. What the results in this study suggest is that the health hazards posed by traffic pollution are born disproportionately by children attending a small number of schools. As discussed in Section 2, the method employed in this study systematically underestimates the pollutant levels experienced by children attending schools close to roads. Given that health effects vary greatly between schools, and are further likely to vary greatly within schools (depending on classroom location), we can conclude that the health impacts of traffic pollution are borne disproportionately by a small group of children within a small number of schools. Given this fact, there is a strong case for targeted pollution monitoring in those schools/classrooms closest to high-volume roads. If actual monitoring indicates high pollutant levels in these 'at-risk' classrooms, pollution filters, or other targeted measures such as strategic tree planting, could be employed to reduce exposure in the worst affected buildings. Because there will only be a small number of such buildings in a small proportion of schools, a cost-benefit analysis of targeted schemes is likely to be compelling. A non-targeted alternative to filtered air-conditioning would be to set, and police, mandatory emissions standards. It is well known that a small number of vehicles is responsible for a large share of gross emissions. Bluett et al., for example, estimate that 10% of Australian vehicles are responsible for 70% of CO, 60% of particulate matter, and 50% of nitrogen oxides (Bluett et al., 2008, page 58).

In this study, we have focused on estimating odds ratios for hospitalisations for asthma, but this specific indicator is also a good indicator for respiratory damage generally. We know that children & infants, with their developing respiratory systems, are at risk of suffering long-term health

complications from exposure to pollutants (Gauderman et al., 2006). That we allow this exposure to occur when this risk could be largely eliminated through targeted action is puzzling. It seems unlikely that this cavalier attitude to childhood exposure to pollution represents a genuine societal 'choice'.

We did not consider particulate matter in this study because of its widespread dispersal. While this is a reasonable assumption for particles measured in µm (i.e. PM_{10}, $PM_{2.5-10}$, $PM_{2.5}$), Zhu et al. found that ultra-fine particulates (< 100 nanometres) do exhibit localized concentration (Zhu et al., 2002). Compared to particulate matter 2.5 µm and larger, there have been relatively few studies on the health effects of ultra-fine particles, and for this reason we could not consider their impact in this study. We do know that ultra-fine particles are small enough to pass into the blood-stream, and as a consequence are highly likely to have adverse health effects, especially on children. This would be a useful area for further research.

In this paper, we have thus far concentrated on the exposure of children in primary schools because this is a vulnerable group. Expanding our focus and considering the population at large, this study suggests that we should be cautious about encouraging development of residential dwellings close to, or along, major roads. But this is exactly what we are currently doing in Sydney, and we plan to continue doing so, with the Sydney metropolitan plan suggesting that over 50% of new residential development will occur in centres and along major corridors (NSW Department of Planning, 2005), many of which are close to main roads. A naive 'centres and corridors' residential development policy together with chronic under-investment in public transport will expose those in new developments along key corridors such Parramatta Road, Victoria Road, and the Pacific Highway to pollution levels much higher than the background levels experienced even 50m from these roads.

Pollution modelling work by Peter Newtown and others at CSIRO in the 1990s supports this claim (Newton, 1997).

5 Conclusions

In examining the school-based exposure to traffic pollution in Sydney primary schools, we find that most school children are unlikely to experience pollution levels distinguishable from local background levels. However, a small proportion of schools (based on our sample, $\approx 5\%$) do experience high levels of CO and NO_2 to warrant additional monitoring & study. Because pollution levels are only likely to be high in a small number of buildings/classrooms, steps to minimise primary student exposure can likely be achieved without expensive changes (such as school re-location).

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Appendix

Table of key terms

Term	Explanation
Odds ratio	A means of expressing the impact of exposure to pollution. For example, a reported hospitalization odds ratio of 1.1 for each part-per-million of exposure to pollutant A indicates that someone exposed to 4 parts-per-million of pollutant A has a 40% greater chance of being hospitalized than someone not exposed.
CO	Carbon monoxide, a major traffic pollutant considered in this study. CO is a local pollutant, with concentrations highest at road-side, then falling by ~ 50% for each 20m from the road. CO pollution has been linked with increase mortality and morbidity in a large number of studies, but we use hospital admissions as a useful indicator of overall health effects, as reliable estimates are not available for longer-term health impacts on children.
NO_x	Nitrogen oxides, a major traffic pollutant considered in this study. NO_x is a local pollutant, with concentrations highest at road-side, then falling by ~ 40% for each 20m from the road. NO_x pollution has been linked with increase mortality and morbidity in a large number of studies, but we use hospital admissions as a useful indicator of overall health effects, as reliable estimates are not available for longer-term health impacts on children.
SO_2	Sulphur dioxide, a major traffic pollutant. Mainly associated with engines that use diesel fuel. In Australia diesel fuel is used predominantly in commercial vehicles. This pollutant was not considered in this study, as road-specific information on commercial vehicle traffic volumes was not available.
Particulates	Very small particles are considered a form of pollution. The most commonly studied particles are those between 2.5 ($PM_{2.5}$) and 10 microns (PM_{10}). Traffic is one of the main sources (e.g. fragmented tyre, suspended dust and petrol), but there are many others (e.g. bush-fires, wood heaters, industrial processes). The health impact of particulate matter varies depending on the size of the particles, but all are known to be harmful, with health impacts generally increasing with decreasing size. Particulate matter concentrations are generally *not* local – they disperse generally and concentrations are not measurably higher close to the pollution source. Very small particles (< 100 nanometres) do exhibit local concentration, but these ultra-fine particles are not yet as well studied.

Endnotes

[1] Even on still days, local wind turbulence created by traffic movement and heat gradients will cause dispersion.

[2] The figure of 750 vehicles per hour was chosen as a representative figure for non-monitored 'regional roads' based on the (first) author's experience working with RTA traffic volume data.
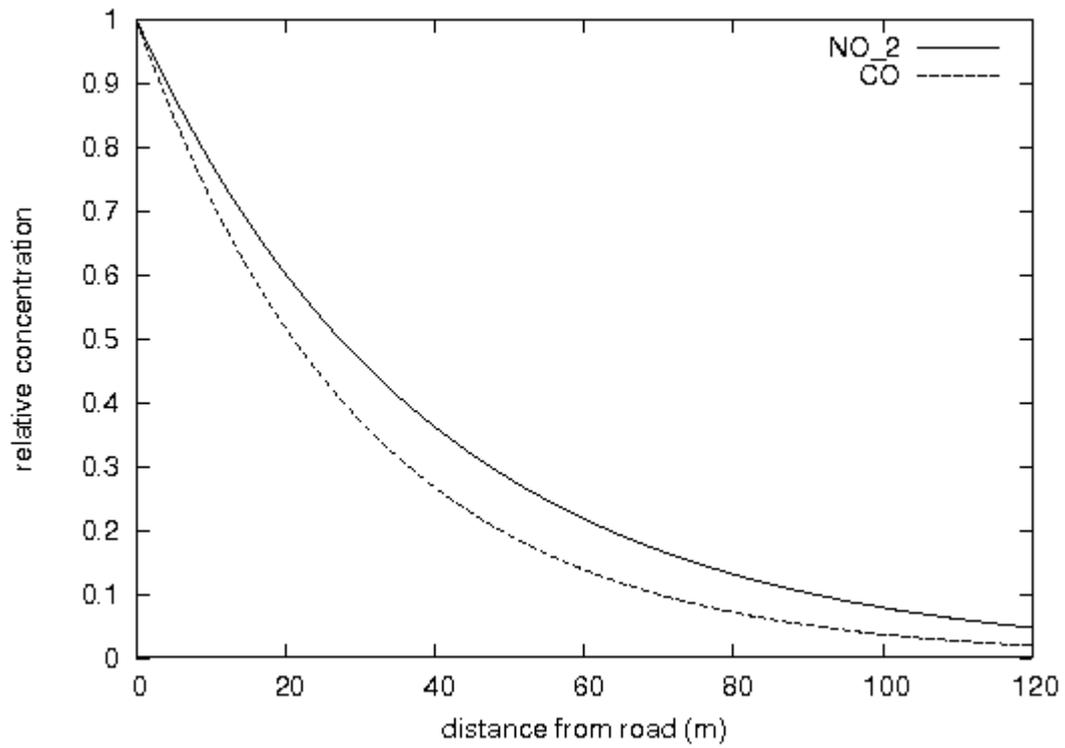
FIGURES AND CAPTIONS

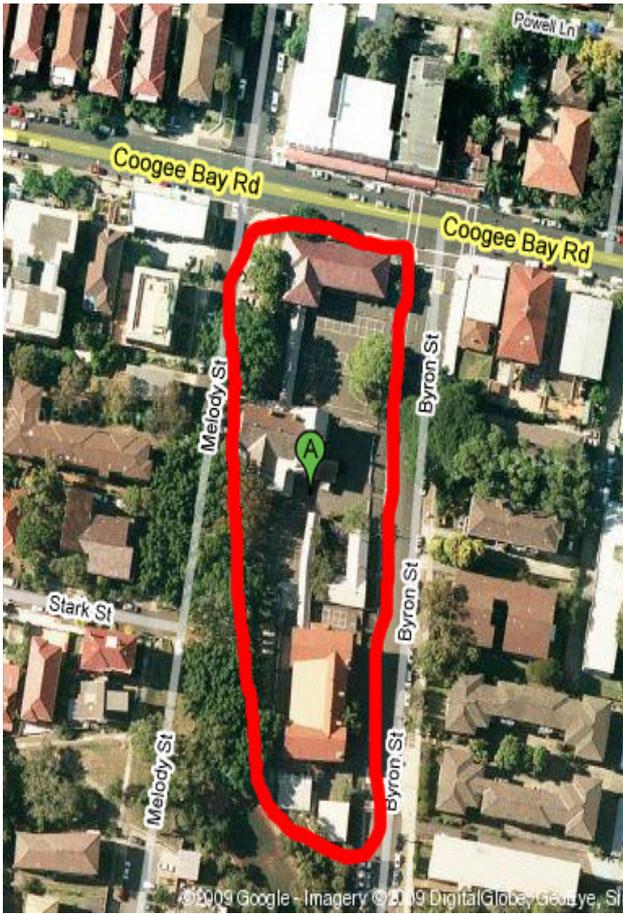Figure 1: Decay model for CO and NO_2 with distance from the road.
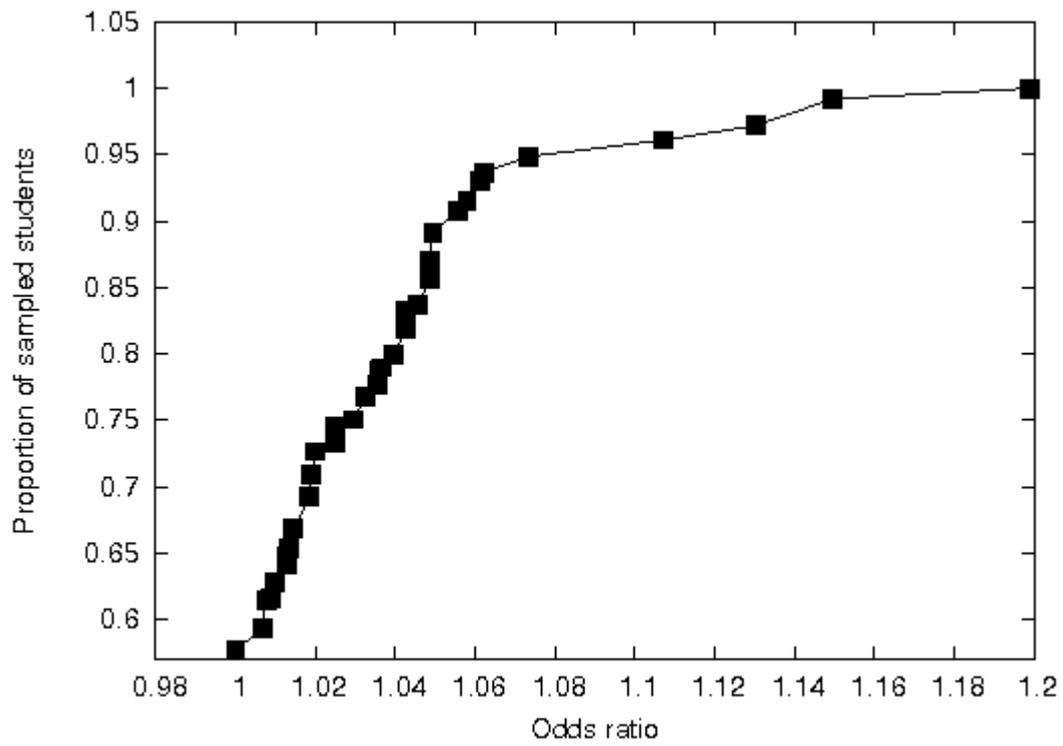
Figure 2: Coogee Bay Public School

Figure 3: Cumulative distribution of odds ratios for the 80 schools sampled
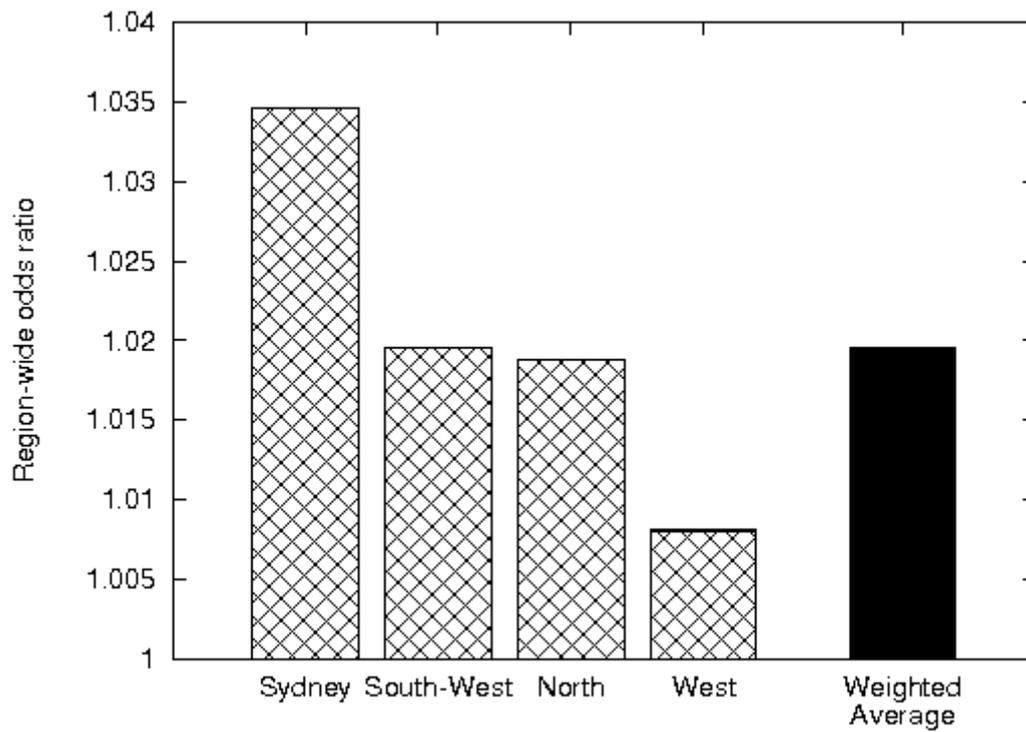
Figure 4: Mean odds ratios for students in each of the four school regions covering the Sydney metropolitan area

a   b

Figure 5: Fig. (a) shows the layout of Vardys Road public school, typical of schools in Sydney's

Western region. Fig. (b) shows the more compact layout of Earlwood public school, approx. 10 km

from Sydney's CBD.

Figure 6: Closest school building at Coogee Public school (marked with an 'A') is 10m from

Coogee Bay Road