Health Impact of the Proposed Airport Link

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Overview

This report is divided into three Sections:

Sections A and B of the report are literature reviews of the health effects of ambient pollutants, which:

- provide a summary of the background information and current air quality goals/standards for benzene, carbon monoxide (CO), formaldehyde, nitrogen dioxide (NO_2), particulate matter (PM) including PM_{10} and $PM_{2.5}$, toluene and xylene;

- summarise the current levels of benzene, CO, formaldehyde, NO_2, PM_{10}, $PM_{2.5}$, toluene and xylene around Brisbane and other Australian cities; and

- reviews the health effects of benzene, CO, formaldehyde, NO_2, PM_{10}, $PM_{2.5}$, toluene and xylene.

Section C of the report is an assessment of the potential health effects arising from increases in ambient benzene, CO, formaldehyde, NO_2, PM_{10}, $PM_{2.5}$, toluene and xylene from the proposed Airport Link. Section C:

- Provides a summary of the forecast worst case changes in ambient benzene, CO, formaldehyde, NO_2, PM_{10}, $PM_{2.5}$, toluene and xylene from the proposed Airport Link, as modelled by Holmes Air Sciences.

- Examines the potential health effects as a result of changes to the ambient levels of ambient benzene, CO, formaldehyde, NO_2, PM_{10}, $PM_{2.5}$, toluene and xylene from the proposed Airport Link.

Summary

Summary of the health effects resulting from changes to ambient benzene, CO, formaldehyde, NO_2, PM_{10}, $PM_{2.5}$, toluene and xylene.

Overall Conclusion

Regional air pollution as a result of the proposed Airport Link is not expected to have an impact on community health. Holmes Air Sciences provided worst case forecast changes in regional ambient benzene, carbon monoxide, formaldehyde, nitrogen dioxide, coarse and fine particulate matter, toluene and xylene as a result of the proposed Airport Link. The worst case changes in ambient air pollutants were forecast to be very small and were equivalent to 0.001% to 3.7% of the National air quality goals. These worst case increases were used to predict the acute and chronic health impacts based on a range of known published relationships between air pollutants and health. The increased risks of acute adverse health events, such as hospital admissions or mortality were correspondingly small, in the order of 1 in 5 million to 1 in 300 million on the day and at the location where the forecast worst case occurs. The forecast impact on symptoms of asthmatic children, a sensitive subgroup within the community, was also found to be small, representing a worst case acute effect of a 0.008% increase in lower respiratory tract symptoms. Long term health effects on cancer, mortality and lung function growth in children were also forecast to be negligible.

Summary of the Health Effects Resulting from Regional Changes to Ambient Air Pollutants.

A conservative approach was used to model the health impacts of ambient regional air pollutants from the proposed Airport Link. Holmes Air Sciences (HAS) provided predicted ground level concentrations for the criteria pollutants: CO, NO_2, PM_{10} and $PM_{2.5}$ at four specific sites and four time periods (2004, 2012, 2016 and 2026). For the air toxics: benzene, formaldehyde, toluene and xylene data was provided for 2004 and 2012 at three sites. The worst case increases in air pollutants were used for assessing the potential worst case health impact. Where improvements in air quality were forecast by HAS, they were not used to offset the worst case estimates of adverse health effects.

The models used for estimating the health effects were based on published: epidemiological studies in Brisbane, other Australian cities or overseas cities, long term studies of mortality and lung function growth from the United States; challenge chamber studies and panel studies. Where more than one health effect estimate was available the most conservative estimate, that is, the one that gave the largest adverse health impact, was used.

Consistent with the HAS working paper, the health effects resulting from predicted worst case increases in ambient air pollutants were examined. The pollutants considered were benzene, CO, formaldehyde, NO_2, PM_{10}, $PM_{2.5}$, toluene and xylene. Four sites were considered for the criteria pollutants and three for air toxics. The sites included Albert Bishop Park, Bowen Hills, Eagle Farm, Kalinga Park and Kedron. The health effects were modelled for the worst affected sites.

Both acute and long term health effects were examined. The acute health effects examined were:

- Mortality and hospital admission; and
- Lung function, symptoms and GP visits.

The long term effects considered were:

- Mortality
- Cancer incidence; and
- Lung function growth in children.

Health effects at sensitive receptors as a result of regional changes in pollutants

For the health effect modelling, the worst case forecast increases in emissions across the 4 sites were:

- Annual average benzene: 0.03μg/m^3;
- 8 hour carbon monoxide (CO): 0.1mg/m^3;
- Annual average formaldehyde: 0.03μg/m^3;
- 1 hour maximum nitrogen dioxide (NO_2): 9 μg/m^3;
- Annual average NO_2: 1.0 μg/m^3;
- 24 hour PM_{10} μg/m^3: 0.1 μg/m^3;
- Annual average PM_{10}: 0.1 μg/m^3;
- 24 hour toluene: 0.04 μg/m^3;
- Annual average toluene: 0.047 μg/m^3;
- 24 hour xylene: 0.027 μg/m^3; and
- Annual average xylene: 0.035 μg/m^3.

In all cases the forecast increases in ambient air pollutants were small (0.001% to 3.7%), relative the current air quality goals.

Benzene

Conclusion: Based on the worst case scenario, the maximum forecast increases in cancer risk as a result of the proposed Airport Link is 0.24 additional leukaemia cases per 1 million people over 70 years, i.e. 1 additional leukaemia case per 4.2 million people exposed to the worst case increase in ambient annual average benzene over a 70 year period. This is a negligible increase in health risk.

Justification: The forecast maximum increase in annual average ambient benzene as a result of the proposed Airport Link is 0.03μg/m^3 at Kedron (Table 29). It is known that a 1μg/m^3 increase in benzene sustained over a 70 year period would result in approximately 8 additional leukaemia cases per 1 million people. Therefore the worst case increase in annual ambient benzene of 0.03μg/m^3, if sustained over a 70 year period this would be expected to result in approximately 0.24 additional leukaemia cases per 1 million people.

CO

Conclusion: Based on the worst case scenario, the maximum forecast increases in cardiovascular hospital admissions as a result of the proposed Airport Link is 0.005 persons/100,000, ie 1 additional cardiovascular admission per 20 million people exposed to the worst case increase in ambient 8 hour CO. This is a negligible increase in health risk.

Justification: The worst case forecast increase in ground level CO was 0.1 mg/m^3 averaged over an 8 hour period (Table 28). The forecast 8-hour maximum CO level resulting from the proposed Airport Link is equal to 1% of the Australian Ambient Air Quality National Environment Protection Measure (AAQ NEPM) of 10 mg/m^3. CO is known to have an impact on respiratory, asthma and cardiovascular hospital admission and cardiovascular mortality. However, the relatively small increase in CO as a result of the proposed Airport Link was predicted to result in an increase in hospital admission of between 0.003- 0.005 persons/100,000 people exposed and an increase in cardiovascular mortality 0.002 persons/100,000 people exposed (Table 30).

Formaldehyde

Conclusion: The forecast worst case contribution of the proposed Airport Link to ambient 24 hour formaldehyde is not expected to have an impact on health.

Justification: The forecast increase of 0.03μg/m^3 (Table 29) represents 3% of the unit risk factor for formaldehyde as a carcinogen which equates to an additional risk of cancer equivalent to 0.39 additional cancer cases per 1 million people exposed to this increase over a 70 year period and therefore no increase in cancer risk.

NO_2

Conclusions: The worst case increases in acute adverse health events resulting from increases to ambient NO_2 from the proposed Airport Link are forecast to be very small. For every 5 million people actually exposed to the worst case increase in NO_2 on that day, one additional cardiovascular hospital admission is forecast. For every 10 million people exposed to the worst case increase in NO_2 on that day, one additional death is forecast. These are negligible increases in health risk.

The worst case increase in long term health effects is likely to be negligible, since the forecast increase in ambient annual average NO_2 is equivalent to 1.4% of the levels reported to have an adverse impact on lung function growth in children.

Justification: Both 1 hour average NO_2 and annual average NO_2 were forecast to increase as a result of emissions from proposed Airport Link, however the increases are small. The predicted maximum increase of 9 $\mu g/m^3$ for 1-hour maximum NO_2 in 2026 at Kedron (Table 28) is equivalent to 6.6% of the highest 1 hour maximum recorded at the EPA's Brisbane CBD monitoring station in 2004 (Table 4) and 29% of the highest median 1 hour maximum recorded at the South Brisbane monitoring station. Epidemiological models of the acute health effects of ambient NO_2 predict that on the days when the maximum increase in NO_2 occurs, there will be an increase in hospital admissions for cardiovascular, respiratory diseases in people aged 65 and over and asthma. An impact on mortality is also forecast. The background rate of these events is relatively small and therefore the daily increase in each event is also small. The forecast incremental increase in hospital admission for cardiovascular diseases, respiratory admissions in people aged 65 and over and asthma are forecast to be 0.02, 0.01 and 0.02 persons per 100,000 people exposed to the maximum increase on the days when the maximum increases in NO_2 occurs (Table 31). The incremental increase in mortality is forecast to be 0.01 person/100,000 people exposed to the worst case increase in NO_2.

A 1.0 $\mu g/m^3$ increase in annual average NO_2, as result of the proposed Airport Link was also forecast (Table 28). Previous studies on the growth of lung function in adolescents living in different communities in southern California reported a 71.5 $\mu g/m^3$ increase in annual average NO_2, over an eight year period, resulted in a 6% increase in the number of 18 year olds who had a clinically significant lower lung function. Based on HAS' forecast annual average level of NO_2 and the published Californian studies, the increase from the proposed Airport Link is around 1.4 % of the value reported in Californian study and is therefore not likely to have a significant impact on lung function growth of adolescents.

PM_{10}

Conclusions: Small increases in the risk for mortality, hospital admission and respiratory symptoms are forecast. The increased mortality risk is 1 additional death per 287 million people exposed to the worst case PM_{10} increase, while the most adverse increase in hospital admission is equivalent to 1 cardiovascular admission per 192 million people exposed to the worst case PM_{10} increase.

The long term effects of the increase in annual average PM_{10} as a result of emissions from the proposed Airport Link are forecast to be extremely small.

Justification: For PM_{10} a the worst case increase in 24-hour concentration was forecast to be 0.1 $\mu g/m^3$ (Table 28), while the worst case annual average was also forecast to be 0.1 $\mu g/m^3$ (Table 28). The increase in daily PM_{10} is forecast to result in small increases in the risk of mortality, asthma, cardiovascular and respiratory admissions to hospital and visits to the doctor for asthma. However, the increased risk of these events is small. For hospital admissions, the increased risk ranges from 0.0002 to 0.0005 persons per 100,000 people exposed on the days when the maximum increase in PM_{10} actually occurs (Table 32). For mortality the increased risk is 0.0004 persons per 100,000 exposed. Small increases in GP visits for asthma and in respiratory symptoms are also forecast.

Increases in annual average PM_{10} are know to have a long term effect on lung function growth in children. In southern California a 51.5 $\mu g/m^3$ increase in annual average PM_{10} exposure over an 8 year period was associated with 6% more children having a clinically significant reduction in lung function by age 18 years. The forecast increase in annual average PM_{10} for 2026 resulting from the proposed Airport Link is 0.1$\mu g/m^3$, which represents 0.19% of the increment recorded in the Californian studies and is therefore not likely to have a significant impact on lung function growth of adolescents.

$PM_{2.5}$

Conclusion: A small increase in cardiovascular admissions is forecast, which is equivalent to 1 additional hospital admission per 92 million people exposed to the worst case increase in $PM_{2.5}$. Negligible increases in hospital admission for asthma, and all respiratory conditions are also forecast.

Justification: Consistent with the worst case scenario, it was assumed that the forecast increase of 0. $\mu g/m^3$ in 24-hour PM_{10} was all due to $PM_{2.5}$ (Table 28). There is less known about the health effects of $PM_{2.5}$, since until recently $PM_{2.5}$ was not extensively monitored. A study of three Australian cities, not including Brisbane, found a10 $\mu g/m^3$ increase in $PM_{2.5}$ resulted in a 5.1% increase in cardiovascular admission in all ages, while there was no significant effect on total mortality. Studies in Melbourne, which approximated levels of $PM_{2.5}$, reported a 15 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 2.4% and 13.9% increase in all respiratory and asthma admissions, respectively. Based on a 0.1 $\mu g/m^3$ increase in $PM_{2.5}$, associated with the proposed Airport Link, the potential adverse health effects are therefore equivalent to increases in hospital admissions of between 0.001-0.0006 per 100,000 people exposed (Table 33).

The long-term effect of a 0.1 $\mu g/m^3$ in annual average $PM_{2.5}$ (Table 29) is a 0.04% increase in long term total mortality.

Toluene

Conclusion: The forecast worst case contribution of the proposed Airport Link to ambient 24-hour and annual average toluene are not expected to have an impact on health.

Justification: The forecast worst case increases of 0.04 $\mu g/m^3$ and 0.047 $\mu g/m^3$ in 24-hour and annual average toluene, respectively, represents increases equivalent to 0.001% and 0.01% of the AAQ NEPM Monitoring Investigational Levels (Table 29), which were set in consideration of no adverse health effects.

Xylene

Conclusion: The forecast worst case contribution of the proposed Airport Link to ambient 24-hour and annual average xylene are not expected to have an impact on health.

Justification: The forecast increases of 0.0027 and 0.0035$\mu g/m^3$ in 24-hour and annual average xylene, respectively, represent increases equivalent to 0.0025% and

0.004% of the AAQ NEPM Monitoring Investigational Levels (Table 29), which were set in consideration of no adverse health impacts.

Health effects as a result of changes to roadside pollutants

Numerous studies have demonstrated that living near a busy road is detrimental to the health of adults and children. The purpose of this section of the report is to examine the additional health risk due to increases in roadside pollutants associated with the proposed Airport Link.

Holmes Air Sciences (HAS) provided data on the forecast levels of pollutants using the Caline dispersion model (see HAS Report). This provides an estimate of near roadway levels of pollutants. Eleven roads were considered and modelling was performed at distances 10, 30 and 50m from the kerb of the roads (Table 34).

The health effects resulting from changes in roadside pollutants are likely to affect fewer people than the regional changes in pollutants. However, the changes in roadside pollutants, since they are next to major roads are often higher than regional changes in pollutants. To estimate the likely health impact the proximity of child care centres, schools, aged care facilities and hospitals to the major roads was also considered. Both acute and long term health effects were examined, using the same methodology as regional health effect modelling.

CO

Conclusion: The forecast worst case contribution of the proposed Airport Link to near road CO levels is not expected to have an impact on health.

Justification: The maximum increase in near road CO was 0.21 mg/m^3 10m from Gympie Road and was forecast to result in extremely small increases in hospital admissions for asthma, all respiratory diseases, cardiovascular diseases and mortality. The size of the increases ranged from 0.01-0.003 persons per 100,000 people exposed to the forecast worst case increase in CO (Table 36). Given the relatively localised increase in the roadside pollutants, this increase is extremely unlikely to have measurable impact on community health.

NO_2

Conclusion: The forecast worst case contribution of the proposed Airport Link to near road 1-hour maximum NO_2 levels is not expected to have a significant impact on health. The long term health impact of increased near roadway annual average NO_2 is difficult to quantify, however previous studies, with 12 times higher changes in NO_2, have found an impact on lung development in children.

Justification: The predicted maximum increase in near road 1-hour maximum PM_{10} was15.32 $\mu g/m^3$ at a distance of 10 m from Gympie Road. Hospital admissions for all respiratory diseases, asthma, cardiovascular diseases and mortality were predicted to increase, however the magnitude of the increases were extremely small and ranged from 0.02-0.04 person per 100,000 people exposed to the forecast worst case increase in NO_2 (Table 38).

The worst case maximum increase in roadside annual NO_2 was 5.80μg/m^3 at a distance of 10m from Gympie Road. Previous studies have found a 71.5 μg/m^3 increase in annual NO_2, over an eight year period, resulted in lower lung function growth in adolescents. It is unclear whether the much smaller increases in near road annual NO_2, associated with the Airport Link, would have an impact on lung function growth in adolescents.

PM_{10}

Conclusion: The forecast worst case contribution of the proposed Airport Link to near road 24-hour maximum or annual average PM_{10} levels is not expected to have a significant impact on health.

Justification: The predicted maximum increase in near road 24-hour PM_{10} was1.72 μg/m^3 at a distance of 10 m from Gympie Road. Hospital admissions for all respiratory diseases, cardiovascular diseases and mortality were predicted to increase, however the magnitude of the increases were extremely small and ranged from 0.004-0.009 person per 100,000 people exposed to the forecast worst case increase in PM_{10} (Table 38). The forecast impact on acute symptoms in adults and children with asthma were also negligible. The worst case increase in annual average PM_{10} was 0.72 μg/m^3 at a distance of 10 m from Gympie Road, this is not likely to have a significant impact on lung growth or mortality, since it represents a small increase in comparison to levels known to have a long term impact on health.

$PM_{2.5}$

Conclusion: The forecast worst case contribution of the proposed Airport Link to near road 24-hour maximum or annual average $PM_{2.5}$ levels is not expected to have a significant impact on health.

Justification: $PM_{2.5}$ was not modelled by HAS, however it was assumed conservatively that all the near road PM_{10} was all $PM_{2.5}$, therefore the size and location of forecast increases was as per PM_{10}. The magnitude of increases in hospital admission for asthma, other respiratory diseases and cardiovascular diseases as result of the worst case increase in $PM_{2.5}$ was very small. Increases of between 0.009-0.02 persons per 100,000 people exposed to the worst case increase were forecast (Table 39). The long term increase in $PM_{2.5}$ impact was also forecast to be negligible.

Glossary

A

age standardised risk	is the risk of an event occurring within an age group expressed as a fraction of the number of people in that age group.
AHR	airways hyperresponsiveness
airway	abnormality of the airways which makes them

hyperresponsiveness	narrow too easily and too much in response to various stimuli; an abnormality seen in people with asthma
airways	air conducting tubes or passages of the lungs
all cause mortality	deaths due to all causes (except accidents)
allergen responsiveness	the extent to which airways narrow in response to allergen exposure
allergen	an environmental substance (usually a protein) which the body's immune system recognises and reacts adversely to
allergic	being capable of recognising and reacting to an allergen
ambient	in the air as it exists in our breathing zone under usual circumstances
Ambient Air Quality National Environment Protection Measure	An Australian air quality standard.
ante-natal	before birth, during pregnancy
arterial oxygen saturation	level of oxygen in the blood
asthma	Asthma is a chronic disorder of the airways which causes them to narrow too easily and too much in response to a wide range of stimuli. It is manifest as episodes of wheeze, chest tightness, shortness of breath and, sometimes, cough which are accompanied by reduced lung function. Airways of people with asthma usually exhibit a special form of inflammation which is present even when the person has no symptoms.
asymptomatic	not experiencing any symptoms

B

bronchitis	cough with phlegm

C

cardiovascular disease	heart disease, or circulatory diseases comprise all diseases of the heart and blood vessels, including coronary heart disease, stroke (or cerebrovascular disease), heart failure, and peripheral vascular disease.
cardiovascular mortality	death due to heart attacks and related diseases
chronic asthma	asthma with permanently impaired lung function

chronic bronchitis	long standing cough with phlegm
chronic obstructive pulmonary disease	Chronic obstructive pulmonary disease (COPD) refers to a group of diseases characterised by an irreversible reduction in expiratory airflow. Included in this disease are patients with emphysema, many of those with chronic bronchitis and some with chronic asthma.
cohort study	a well defined group of subjects are followed over time to measure the relation between an exposure (eg pollution level) and an outcome (eg symptoms or lung function)
confounding	where the presence of another risk factor confuses the true relationship between the factor under study and the outcome
COPD	chronic obstructive pulmonary disease

D

diagnosis	disease label
disability	inability to perform tasks
dyspnea	breathless ness
dysrhythmia	abnormal heart beat rythm

E

ED	Emergency Department
EIS	Environmental Impact Statement
Emergency Department	also known as Casualty, Accident and Emergency or Emergency Room
emphysema	disease in which lung tissue is gradually destroyed (usually due to smoking); causes breathlessness
exacerbations	temporary deterioration in illness state
expiratory airflow	rate at which air can be exhaled (blown out)
exposure chamber	a device for exposing experimental subjects to known concentrations of gases (eg ozone or nitrogen dioxide)

F

Forced expiratory volume- one second	rate of exhalation of air in one second, after a full, deep inhalation, a measure of expiratory airflow

Forced vital capacity	total volume of air which can be forcibly exhaled after a full, deep inhalation

H

heterogeneous	a non-uniform mixture of various different components
hydroscopicity	tendency to take up water

I

impaired lung function	lung function lower than expected for age, gender and height
impairment	reduced function
ischaemic heart disease	or coronary heart disease consists of mainly heart attack and angina. A heart attack occurs when a vessel supplying blood to the heart muscle suddenly becomes blocked by a blood clot. Angina is a temporary chest pain or discomfort caused by a reduced blood supply to the heart muscle.

L

lower respiratory tract	airways within the lungs (ie below the nose, mouth and throat)
lowest-observed-adverse-effect level	lowest concentration of a pollutant which causes a detectable adverse effect
lung function	a measure of respiratory health

M

mass median aerodynamic diameter	a measure of particle size
measure of association	a way of expressing the relation between an exposure (eg pollutant) and an outcome (eg symptoms or lung function) in a numeric form
meta-analysis	a systematic overview of related investigations which summarises the information they contain in one or more measures of association
mortality	deaths

N

NH&MRC	National Health and Medical Research Council
nitrogen dioxide	gaseous pollutant
no-observed-adverse-effect level	greatest concentration of a pollutant which causes no detectable adverse effect.
NO_2	nitrogen dioxide

O

occupational exposures	exposures occurring at work
odds ratio	a statistical measure of increase in prevalence of an outcome associated with change in level of pollutant
oxidant	a chemical which oxidises
oxidising	a type of chemical reaction

P

panel study	see cohort study
particulate	airborne particle
peak expiratory flow rate	the maximum flow rate during a exhalation with effort; a measure of lung function
PEFR	peak expiratory flow rate
PM_{10}	particulate matter with MMAD less than 10μm
ppm	parts per million; a measure of gas concentration
prevalence	the proportion of a population who have a condition at any given time

Q

questionnaire	a series of standardised questions used to collect information for analysis

R

randomised controlled trial	an experimental research design in which subjects are randomly allocated (ie by chance) to one of two or more conditions and followed over time to measure an outcome or response
randomly selected	selected or allocated by chance (eg toss of coin or

	throw of a dice)
rate ratio	ratio of the rate in one group to the rate in another group; in the usage here it refers to the ratio of the admission or death rate on two days separated in pollutant concentration by a specified amount (shown in the increment column of the tables).
reactive chemical species	chemical entities which are unstable and tend to react with other substances
regression coefficients	a measure of association between two variables (eg pollutant concentration and lung function) derived from a form of statistical analysis known as regression
relative risk	a statistical measure of increase in occurrence of a condition associated with exposure to a pollutant. Usually used for prospective cohort or cross sectional studies and over short time periods.
respiratory mortality	deaths due to respiratory disease
risk factors	factors which, if possessed by an individual, increase the likelihood of that individual having a specified disease or other outcome

S

sulphur dioxide	sulphur dioxide
standard error	a statistical measure of uncertainty about an estimate
sulphur dioxide	a gaseous pollutant

T

Time series analysis	a form of analysis which examines factors influencing change in an observation (eg hospital admission rates) over time
toxic	harmful and damaging to living things
particles	total suspended particulates, a measure of particulate pollution

W

weighted average	average of several estimates giving greater weight to the more certain estimates and lesser weight to the less certain estimates

$\mu g/m^3$	micrograms per cubic metre; a measure of particle or gas concentration in air
10 minute mean	average over a 10 minute period
24 hour mean	average over a 24 hour period
95 percent CI	95 percent confidence interval
95 percent confidence interval	the range of values within which the actual measure lies; it is based on the estimated value and the uncertainty of the estimate (std error). Where the confidence interval for a rate ratio excludes the value 1 we can be fairly certain that exposure is related to outcome (admission or death rates) in that study (ie $P < 0.05$).

Abbreviations

AAQ NEPM	Ambient Air Quality National Environment Protection Measure
AHR	airways hyperresponsiveness
AAQ NEPM MIL	AAQ NEPM Monitoring Investigation Level
APHEA	Air Pollution and Health European Approach
ASR	age standardised risk
BS	Black smoke, an measure of particulate matter
bsp	particles in the air as assessed by back scatter of light
CI	confidence interval
CVD	cardiovascular disease
CO	carbon monoxide
COMEAP	Committee on the Medical Effects of Air Pollutants
COPD	chronic obstructive pulmonary disease
CVD	cardiovascular disease
ED	Emergency Department
EIS	Environmental Impact Statement
EU	European Union
FEV_1	forced expiratory volume in one second
FVC	forced vital capacity
GN	Golden North - location of an air qaulity monitor in Port Pirie
HRA	health risk assessment
ICD 9	International Classification of Diseases, Version 9
ICD 10	International Classification of Diseases, Version 10
IHD	Ischaemic heart disease
JPSS	John Pirie Secondary School - location of an air qaulity monitor in Port Pirie
LOAEL	lowest-observed adverse effect level
MMAD	mass median aerodynamic diameter
NAAQS	National Ambient Air Quality Standards of the United States
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure

NH&MRC	National Health and Medical Research Council
NO_2	nitrogen dioxide
NOAEL	no-observed-adverse-effect level
OR	odds ratio
PEFR	peak expiratory flow rate
PM_{10}	particulate matter with MMAD less than 10µm
ppb	parts per billion; a measure of gas concentration
pphm	parts per hundred million; a measure of gas concentration
ppm	parts per million; a measure of gas concentration
RR	relative risk
SAMMS	South Australian Monitoring and Surveillance System
SE	standard error
SO_2	sulphur dioxide
Particles	total suspended particulates, a measure of particulate pollution
$\mu g/m^3$	micrograms (one microgram is one millionth of a gram) per cubic metre; a measure of particle or gas concentration in air
US EPA	United States Environment Protection Agency
95% CI	95 percent confidence interval
WHO	World Health Organisation

Section A: Literature Review of the Health Effects of Ambient CO, NO_2, PM_{10} and $PM_{2.5}$.

Background

Carbon Monoxide (CO)

Carbon monoxide (CO) is an odourless, colourless and tasteless gas. It is produced by the incomplete combustion of fossil fuels. Carbon monoxide is absorbed through the lungs of humans and animals, where it reacts with haemoglobin (the oxygen-carrying molecule in the blood) to reduce the blood's oxygen-carrying capacity. Hence, it affects the delivery of oxygen to the body's organs and tissues. At concentrations exceeding about $100cm^3/m^3$ (0.01%) it is highly toxic. Its affinity for haemoglobin (with which it forms carboxyheamoglobin) is between 200 and 300 times that of oxygen and it has the effect of reducing the oxygen-transport capacity of The main source of carbon monoxide in the ambient air of a city, such as Brisbane, is petrol-fuelled motor vehicles; smaller quantities are produced by diesel-fuelled vehicles and other combustion processes. Motor vehicles account for up to 90 percent of all carbon monoxide emissions. Technological developments, such as improved engine design and catalytic converters, have reduced carbon monoxide emissions in recent years[1].

Carbon monoxide levels, therefore, tend to be greatest in areas of high traffic density[2].

Nitrogen Dioxide (NO_2)

Nitrogen dioxide is a brownish gas with a pungent odour. In the atmosphere, nitrogen dioxide exists in equilibrium with nitric oxide — a colourless, odourless gas. The mixture of these two gases is commonly referred to as nitrogen oxides, or NOx. Nitrogen dioxide is produced from the combustion of fossil fuels. Motor vehicle emissions account for 70% of NO_2 production [3] and NO_2 is a strong marker of road traffic [4]. During high temperature combustion, NO_2, NO and other nitrogen oxides (NO_X) are generated. Part of the NO is converted to NO_2 through reactions involving oxygen and ozone. NO_2 is water insoluble and a strong oxidising agent, which may penetrate deep into the lungs. NO_2 and other NO_X are precursors for a range of secondary pollutants which have adverse effects on human health [4]. In the presence of sunlight NO_X react with hydrocarbons and oxygen to form other photo oxidants, such as such ozone and nitric acid. There are often strong correlations between NO_2 and PM[5].

Particulate Matter (PM)

Particulate matter (PM) is a complex mixture of solids and/or liquids suspended in air. Airborne PM is produced through natural processes and as a result of human activity. Because Australia is a dry continent, its atmosphere contains a significant amount of

particulate matter in the form of windblown dust. Bushfires, hazard-reduction burning in forests and agricultural practices also introduce particles into the atmosphere. In industrial and urban areas, the combustion of fossil fuels (e.g. by power stations and motor vehicles), industrial operations, incinerators and earth-moving activities all contribute to airborne particulate matter levels. Also, in coastal areas, the atmosphere can contain a significant level of sea-salt particles[6].

The size and composition of particulate matter is an important determinant of the health effects.

Size

Particle size is measured as the aerodynamic diameter of the particles and governs where the particles will be deposited in the respiratory tract and also the transport and removal of the particles from the air. The site of deposition in the respiratory tract influences the acute symptoms provoked, retention within the airways and possibly the long-term health consequences. Based on aerodynamic diameter, particle pollution is divided into three groups PM_{10}, $PM_{2.5}$ and $PM_{0.1}$.

PM_{10} includes particles with an aerodynamic diameter of 10μm or less and therefore measures of PM_{10} include $PM_{2.5}$ and $PM_{0.1}$ (Figure 1). The precise definition of PM_{10} is particles that pass through a size selective inlet with a 50% efficiency cut-off at 10μm aerodynamic diameter [7]. Particles above 10 μm in diameter are predominantly deposited in the upper airways, such as in the nose, pharynx and trachea (Figure 2). Particles between 2.5-10μm are inhalable and are deposited in the in the larger airways (Figure 2).

$PM_{2.5}$ is all particles equal to or less than 2.5 μm in aerodynamic diameter, or, more strictly, particles which pass through a size selective inlet with a 50% efficiency cut-off at 2.5 μm aerodynamic diameter. $PM_{2.5}$ is therefore a subset of PM_{10} (Figure 1). Smaller particles referred to as respirable, range in aerodynamic diameter from 0.1 to 2.5 μm and are deposited deeper into the respiratory tract, such as the bronchi and alveoli of the lungs (Figure 2). $PM_{2.5}$ have more adverse health consequences than larger particles and are released as a result of combustion processes, such as motor vehicle exhausts and solid fuel heaters.

$PM_{0.1}$, referred to as ultrafine particles, have an aerodynamic diameter of less than 0.1 μm. $PM_{0.1}$ is therefore a subset of both $PM_{12.5}$ and PM_{10} (Figure 1). $PM_{0.1}$ account for the largest number of airborne particles, however the combined mass of these particles is often recorded as insignificant in comparison to the larger particles [8]. Due to limitations in methods for quantifying ultrafine particles, very few of the studies have examined the role of ultrafine particles in health outcomes [8].

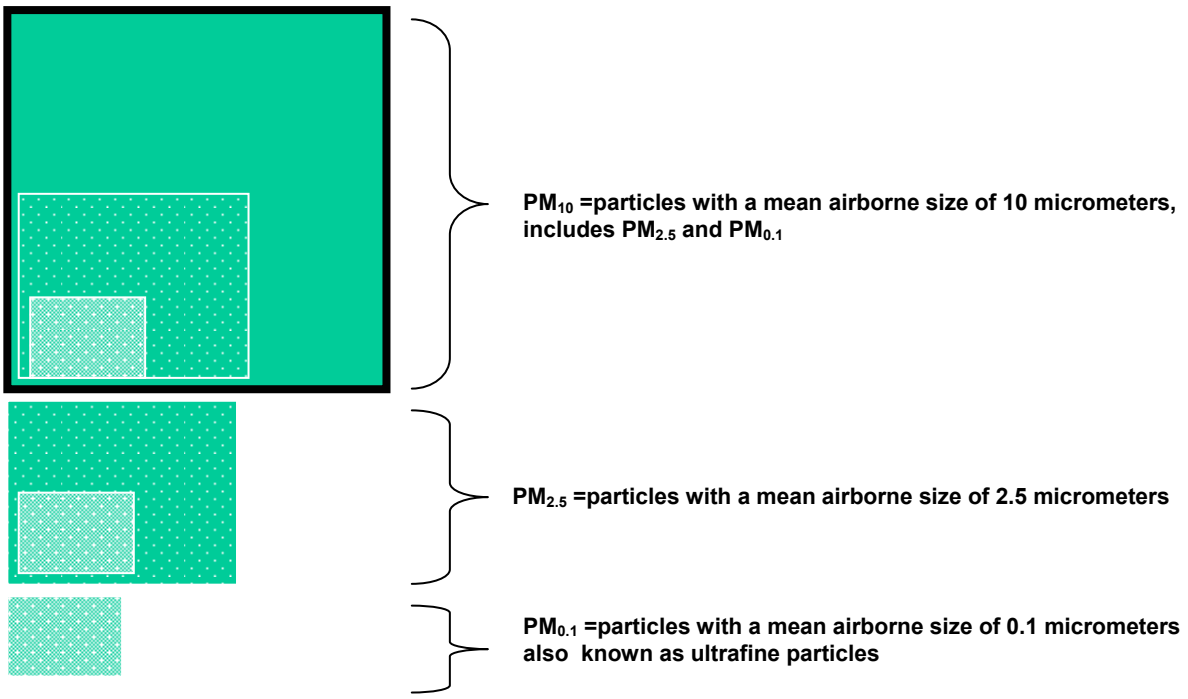

Figure 1: Categories of airborne particulate matter.

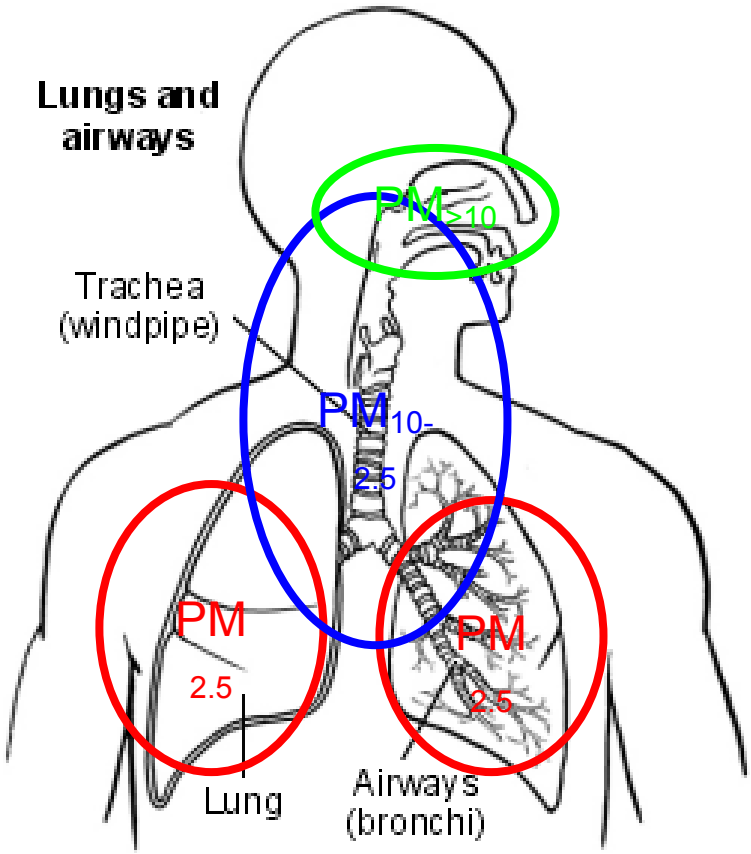

Figure 2: Penetration of particulate matter into the respiratory tract.

Composition

PM can be either primary or secondary in nature. Primary particles are emitted directly into the atmosphere either by natural or man-made processes. Secondary particles have a predominantly man made origin and are formed in the atmosphere from the oxidation and subsequent reactions of sulphur dioxide, nitrogen oxides and volatile organic compounds (VOCs). The particles themselves are therefore a complex mixture of organic and inorganic compounds in solid or liquid states. Types of particulate pollution range from relatively large such as mineral dusts, such as occur during dust storms, to small particles released as a result of condensation of metals or organic compounds following high temperature combustion.

In addition to the different chemical composition of particles (as a result of their formation process) they may also be carriers of biological and non-biological mediators of inflammation. Biological mediators of inflammation include endotoxins and allergens, while non-biological mediators are some metal ions and polyaromatic hydrocarbons[4] .

The ability of diesel particles to function as allergen carriers and to enhance the inflammatory response to allergens is well documented[4].

There also appears to be differences in the nature of the inflammatory response generated against smaller versus larger particles which are carrying allergen. While smaller particles carrying allergens may penetrate deeper into the respiratory tract they do not necessarily induce a greater immediate inflammatory response. In laboratory studies on cat allergic people with asthma, Lieutier-Colas *et al*. (2003)[9] found that 10 micrometer droplets of cat allergen were 20 times more potent than 1.4 micrometer droplets for inducing immediate inflammatory responses. The smaller particles, however, were more potent at inducing late inflammatory responses[9].

Metal ions have also been implicated in increasing the inflammatory properties of the inhaled particles. $PM_{2.5}$ particles rich in cadmium, nickel, zinc and copper were found to induce almost twice the inflammatory response as particles with lower levels of these metal ions[10].

International guidelines/standards

Australia

The Ambient Air Quality National Environment Protection Measure (AAQNEPM) was introduced in 1998 by the National Environment Protection Council (NEPC) and provides the criteria for the national ambient air quality standards for air pollutants to which most Australians are exposed (Table 1). In Queensland there are also Environmental Protection (Air) Policy 1997 air quality goals (Table 1) relating to human, which are similar to the AAQNEPMs.

The AAQNEPM standards were set after consideration of the health effects of these pollutants based on reports prepared by Streeton (1997) [11], the Technical Review Panel of the NEPC and the existing NH&MRC goals (1995).

There is no current ambient air NEPM for $PM_{2.5}$ and in the past these finer particles have not been separately measured from PM_{10}. In 2003 the Environment Protection and Heritage Council (EPHC) introduced Advisory Reporting Standards for $PM_{2.5}$, which are:

25 μg/m^3 averaged over one day (24 hours).
8 μg/m^3 averaged over one year.

The goal of the Advisory Reporting Standards are to gather sufficient data nationally to facilitate a review of the Advisory Reporting Standards as part of the development of a $PM_{2.5}$ ambient air NEPM, which is scheduled to commence in 2005. Monitoring of $PM_{2.5}$ commenced in all States and Territories in either January or July 2004.

Table 1: Australian Ambient Air Quality NEPMs for CO, NO_2 and PM (NEPC, 1998 [12] and NEPC, 2004[13]).

Pollutant	Ambient Air Quality NEPMs		Qld Environment Protection Policy (Air)	
	Averaging period	Maximum concentration	Averaging period	Maximum concentration
CO	8 hour	9 ppm (not to be exceeded more than one day per year)	8 hour	8 ppm (10,000 μg/m^3)
NO_2	1 hour	0.12ppm (246 μg/m^3) (not to be exceeded more than one day per year)	1 hour	0.16 ppm (320 μg/m^3) (not to be exceeded more than 9 hours per year)
			4 hour	0.046 ppm (95 μg/m^3) (not to be exceeded more than 1 day per year)
	1 year	0.03ppm (62 μg/m^3)	1 year	0.015ppm (30 μg/m^3)
PM_{10}	1 day	50 μg/m^3 (not to be exceeded more than five days per year)	24 hour 1 year	150μg/m^3 50 μg/m^3
($PM_{2.5}$)^	1 day 1 year	25 μg/m^3 8 μg/m^3		

* Monitoring Investigation Level (see above). For the purposes of this Measure the annual average concentrations are the arithmetic mean concentrations of 24-hour monitoring results.

^Advisory Reporting Standard (see above).

International Standards

Australia's air quality standards are similar to overseas standards (Table 2). In addition, some overseas standards have considered different time frames which are designed to reflect either the nature of the dose response relationship, or the types of exposures encountered in the community. Of note are:

- The short-term CO guidelines, which are 87ppm averaged over 15 minutes and 50 ppm averaged over 30 minutes (Table 2). These CO standards reflect the nature of exposure to CO as a result of heavy traffic congestion in city canyons and road tunnels. The CO guidelines have been adopted by the Permanent International Association of Road Congresses (PIARC).

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- The uncertainty regarding short-term standards for NO_2. There is considerable uncertainty about the health effects of acute exposure to elevated levels of NO_2 as occurs, for example, in road tunnels. The Swedish National Road Administration recently considered this issue and provided options that ranged from 150-1000 $\mu g/m^3$ for 30-15 minute exposures, noting the lack of information and the need for a compromise in setting this standard [14].

Table 2: Summary of overseas air quality standards. From Cains et al., (2003)[15] and O'Meara et al., (2003) [16].

Pollutant	Level	Time Period	Country	Institution
CO	9 ppm	8 hr	Australia	NEPC
	9 ppm 35 ppm	8 hr 1hr	US	USEPA
	87 ppm 50 ppm 25 ppm 10 ppm	15 min 30 min 1 hr 8 hr	International guidelines	WHO [17]
	9 ppm 20 ppm	8 hr 1 hr	US (California)	SCAQMD
	10 ppm	8 hr	UK	UK
NO_2	120ppbv	1 hr	Australia	NEPC
	30 ppbv	1 yr	Australia	NEPC
	200 µg/m^3	1 hr	WHO regions	World Health Organisation WHO
	40 µg/m^3	1 yr	WHO regions	WHO
	250ppbv	1 hr	US (California)	SCAQMD
	150 ppb	1 hr	UK	UK
	98ppbv	1 hr	International	WHO [17]
PM_{10}	50 µg/m^3	24 hr	Australia	NEPC
	50 µg/m^3	24 hr (99th percentile)	WHO regions	World Health Organisation WHO
	20 µg/m^3	1 yr	WHO regions	WHO
	150 µg/m^3	24 hr	US	USEPA
	50 µg/m^3	24 hr	US (California)	Southern California Air Quality Monitoring Department.
	50 µg/m^3	24 hr	UK	
	40 µg/m^3	1 yr	UK	UK Dept. of Environment, Transport and the Regions
				UK Dept. of Environment, Transport and the Regions
	150 µg/m^3	24 hr	US	USEPA
	50 µg/m^3	24 hr	NZ	NZ Ministry for the Environment
	75, 150 & 250 µg/m^3	24 hr	China	Level depends on classification of area: Class 1 are tourist, historic, and conservation areas. Class 2 are residential urban and rural areas. Class 3 are industrial areas and heavy traffic areas.
$PM_{2.5}$	25 µg/m^3	24 hour	Australia	NEPC Advisory reporting standard for non-peak sites
	8 µg/m^3	1yr	Australia	NEPC Advisory reporting standard for non-peak sites
	65 µg/m^3	24 hr	US	US EPA
	15 µg/m^3	1 yr	US	US EPA
	30 µg/m^3	24 hr	Canada	Canadian Council of Ministers of the Environment

	25 µg/m^3	24 hr	New Zealand	NZ Ministry for the Environment, proposed interim guideline.
	25 µg/m^3	24 hour (99th percentile)	WHO regions	WHO
	10 µg/m^3	1yr	WHO regions	WHO

[1]Not determined

Exposure to CO, NO_2 and PM

CO

Ambient

In 2004, carbon monoxide was monitored at four sites in south-east Queensland: Pinkenba, Brisbane CBD, South Brisbane and Woolloongabba, and one site in Toowoomba. The Brisbane CBD, South Brisbane and Woolloongabba sites are close to major inner-city traffic corridors carrying from 50,000 to over 100,000 vehicles per day, while the Pinkenba and Toowoomba sites are situated further from carbon monoxide emission sources[6]

Table 3: Ambient carbon monoxide concentration (8 hour average) statistics for south-east Queensland sites, 2004[6].

Monitoring location	Maximum (ppm)	Second highest (ppm)	Percentiles						Minimum (ppm)	Annual average (ppm)
			99.9 (ppm)	99 (ppm)	95 (ppm)	90 (ppm)	75 (ppm)	50 (ppm)		
Pinkenba	2.2	1.8	1.6	0.9	0.3	0.2	0.0	0.0	0.0	0.1
Brisbane CBD	3.3	3.2	2.8	1.5	0.7	0.4	0.2	0.1	0.0	0.2
South Brisbane	4.6	4.3	3.7	2.2	1.3	1.1	0.8	0.5	0.0	0.6
Woolloongabba	4.7	4.3	3.8	2.7	1.6	1.2	0.8	0.4	0.0	0.6

Ambient CO in Brisbane (Table 3), as recorded by air quality monitoring stations in the CBD and at Woolloongabba, did not exceed the AAQNEPM in 2004[6] . For the Brisbane CBD, South Brisbane and Woolloongabba the highest 8-hour CO recordings were 3.3 ppm, 4.6 ppm and 4.7 ppm, respectively, in 2004. This ranges from 37% and 52% of the AAQNEPM for CO [6]. There were no exceedences of the AAQNEPM (Air) 8-hour air quality goal measured at any of the four monitoring sites during 2004.

Carbon monoxide concentrations tend to be higher during winter due to a greater frequency of still conditions. This limits dispersion of pollutants from the vicinity of the emission source (Table 3). Levels of CO are elevated in areas where there is high road traffic and restricted air movements, such as inner city canyons, car parks and road tunnels. The differences in levels recorded at the four monitoring sites (Table 3) reflect their respective distances from sources of carbon monoxide. The Brisbane CBD site is over 100m from a major road, the South Brisbane site is approximately 30m from the South East Freeway, and the Woolloongabba site is only 4m from the nearest traffic lane of Ipswich Road. Emissions from industry and motor vehicles are well dispersed prior to reaching the Pinkenba site.

Ambient CO levels in Brisbane have declined over the period 1998 -2004 [6] Over the period 1998-2004 there have been no exceedances of the AAQNEPM for CO in the Brisbane CBD, South Brisbane or Woolloongabba.

Other sources of high CO exposure

In underground and multi-storey car parks, enclosed ice arenas and various other indoor microenvironments, in which combustion engines are used under conditions of insufficient ventilation, the mean levels of carbon monoxide can rise above 115 mg/m^3 (100 ppm) for several hours, with short-lasting peak values that can be much higher. In homes with gas appliances, peak carbon monoxide concentrations of up to 60–115 mg/m^3 (53–100 ppm) have been measured[17]. Environmental tobacco smoke in dwellings, offices, vehicles and restaurants can raise the 8-hour average carbon monoxide concentration to 23–46 mg/m^3 (20–40 ppm)[17].

NO_2

Ambient

In south-east Queensland the QLD EPA monitored nitrogen dioxide at 11 sites (Mountain Creek, Deception Bay, Eagle Farm, Pinkenba (industry site), Brisbane CBD, South Brisbane, Rocklea, Springwood, North Maclean, Flinders View and Mutdapilly), one Toowoomba site (North Toowoomba), four Gladstone region sites (South Gladstone and Clinton in Gladstone, and two Targinie sites (Stupkin Lane and Swans Road)), and one site in Townsville (Pimlico).

Table 4: Ambient 1-hour maximum nitrogen dioxide concentration statistics for south-east Queensland monitoring sites, 2004 [6].

Monitoring location	Maximum (ppm)	Second highest (ppm)	Percentiles						Minimum (ppm)	Annual average (ppm)
			99.9 (ppm)	99 (ppm)	95 (ppm)	90 (ppm)	75 (ppm)	50 (ppm)		
Mountain Creek	0.041	0.039	0.034	0.025	0.016	0.012	0.006	0.003	0.00	0.005
Deception Bay	0.045	0.041	0.036	0.027	0.020	0.016	0.009	0.003	0.000	0.006
Eagle Farm	0.061	0.061	0.054	0.039	0.029	0.025	0.018	0.011	0.000	0.013
Pinkenba	0.057	0.054	0.044	0.030	0.024	0.021	0.014	0.007	0.000	0.010
Brisbane CBD	0.067	0.064	0.054	0.040	0.031	0.026	0.018	0.011	0.000	0.013
South Brisbane	0.060	0.059	0.048	0.037	0.031	0.026	0.021	0.015	0.000	0.016
Rocklea	0.049	0.049	0.046	0.034	0.025	0.021	0.013	0.007	0.000	0.009
Springwood	0.038	0.038	0.035	0.029	0.022	0.018	0.011	0.004	0.000	0.007
North Maclean	0.046	0.041	0.028	0.020	0.014	0.010	0.006	0.003	0.000	0.005
Flinders View	0.054	0.049	0.042	0.030	0.023	0.019	0.013	0.007	0.000	0.009
Mutdapliy	0.040	0.035	0.031	0.022	0.015	0.011	0.007	0.003	0.000	0.005

The maximum 1-hour NO_2 level reached in 2004 was 0.067 ppm at the Brisbane CBD monitoring station (Table 4), which is 56% of the AAQNEPM of 0.12 ppm (229 μg/m^3). There have been no occasions when the EPP (Air) 1-hour goal for nitrogen dioxide of 0.12ppm was exceeded from 1995-2004 at any of the monitoring sites in south-east Queensland. In south-east Queensland, there has been an overall decline in ambient levels of nitrogen dioxide since 1995. Monitoring has not detected any definite correlation between traffic volume and nitrogen dioxide concentrations. Given that motor vehicles are the major contributor of nitrogen oxides in the region, advances in engine design and emission control are largely keeping pace with

increasing vehicle numbers. Further tightening of vehicle emissions through new ADRs, which come into force from 2005, should help maintain this balance.

The 1-year average AAQNEPM of 0.03 ppm was not exceeded and the highest 1-year average was 0.016 ppm at South Brisbane which is 53% of the AAQNEPM [6].

Other sources of high NO_2 exposure

Unflued gas heating and cooking are a major source of NO_2 exposure and have been found to be associated with asthma symptoms, night-time cough, wheeze and the development of airway hyper-responsiveness in children[18, 19].

Levels of NO_2 in Australian homes are high and frequently above the WHO guideline of 0.11 ppm (210 μg/m^3) for indoor NO_2. The Department of the Environment and Heritage (DEH) recently examined NO_2 in 116 suburban houses (148 house-days) with unflued gas heating or cooking in Melbourne, Sydney, country Victoria and Canberra (Figure 3). The highest recorded 1 hour average NO_2 was 0.93 ppm (1780 μg/m^3), while the average level was 0.19 ppm (363 μg/m^3). Many people are exposed to these high indoor levels of NO_2 for several hours[20].

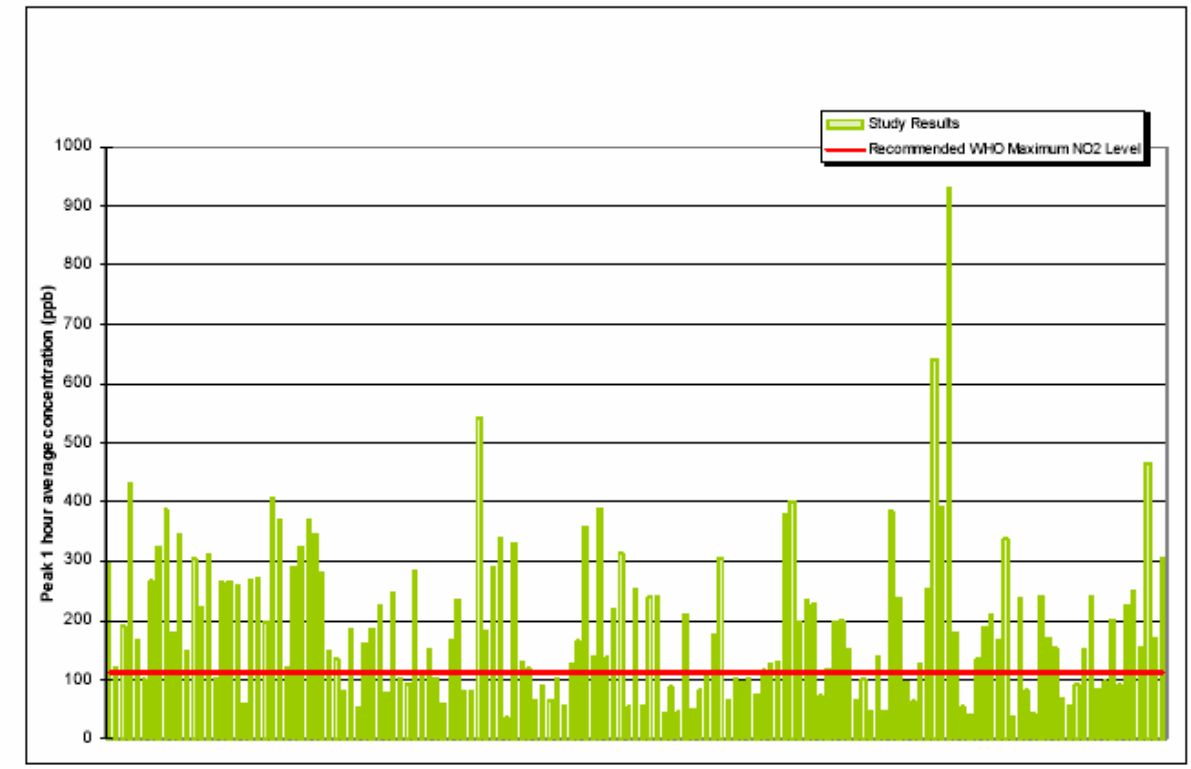

Figure 3: Indoor NO_2 levels in homes with unflued gas heating or cooking. From Technical Report No. 9, Unflued Gas Appliances and Air Quality in Australian Homes (2004)[20].

Ambient PM_{10}

Airborne particulate matter monitoring in Brisbane conducted by the QLD EPA covers three particle size ranges: particles less than 10µm in diameter (PM_{10}), particles less than 2.5µm in diameter ($PM_{2.5}$) and visibility-reducing particles (typically 0.1–2.5µm in diameter). Particulate matter monitoring locations during 2004 were:

- PM_{10} particles at nine south-east Queensland sites (Mountain Creek, Eagle Farm, Pinkenba (industry site), Brisbane CBD, South Brisbane, Woolloongabba, Rocklea, Springwood and Flinders View), one Toowoomba site (North Toowoomba), three Gladstone region sites (South Gladstone and Clinton in Gladstone, and Targinie (Stupkin Lane)), one Rockhampton site (Parkhurst), one Mackay site (West Mackay) and four Townsville sites (Pimlico, Townsville Port (industry site), South Townsville and Garbutt);
- $PM_{2.5}$ particles at two south-east Queensland sites (Rocklea and Springwood) and one Toowoomba site (North Toowoomba); and
- visibility-reducing particles at five south-east Queensland sites (Mountain Creek, Eagle Farm, Brisbane CBD, Rocklea and Flinders View), one Toowoomba site (North Toowoomba), two Gladstone region sites (South Gladstone and Targinie (Stupkin Lane)) and one Mackay site (West Mackay).

Stagnant, stable meteorological conditions are most favourable to a build-up of particulate matter in the atmosphere. This occurs most frequently in winter and early spring, particularly during the night or early morning.

The maximum PM_{10} concentration in Brisbane in 2004 was 79.9µg/m^3 recorded at Eagle farm. On most days in Brisbane and surrounds, ambient PM_{10} did not exceed the AAQ NEPM of 50µg/m^3 (Table 5). The majority of exceptions were due to smoke from bush fires or hazard reduction burning. Of the nine days when 24-hour average PM_{10} levels exceeded 50µg/m^3 at one or more monitoring sites in south-east Queensland, six were directly attributable to smoke from bushfires or hazard reduction burning. The remaining three days were caused by an unidentified local dust source close to the Woolloongabba site (as levels at the nearby South Brisbane and Rocklea sites were considerably lower), industrial activities near the Eagle Farm site, and dust from building site works near the Mountain Creek site respectively[6].

Table 5: Ambient PM_{10} concentration (24 hour maximum) statistics for south-east Queensland monitoring sites, 2004[6].

Monitoring location	Maximum (µg/m3)	Second highest (µg/m3)	Percentiles					Minimum (µg/m3)	Annual average (µg/m3)
			99 (µg/m3)	95 (µg/m3)	90 (µg/m3)	75 (µg/m3)	50 (µg/m3)		
Mountain Creek	66.6	43.4	39.1	29.0	22.6	17.5	13.6	3.8	15.2
Eagle Farm	79.6	57.7	47.7	38.7	34.8	27.4	20.8	7.2	22.8
Pinkenba	54.3	52.8	46.8	37.0	32.6	25.2	19.4	6.4	21.3
Brisbane CBD	56.6	50.1	48.2	30.3	25.1	19.9	15.9	9.8	17.3
South Brisbane	63.5	50.7	42.3	33.6	29.2	24	19.6	9.1	20.6
Woolloongabba	65.4	57.9	50.1	35.3	32.1	26.6	21.1	6.6	22.3
Rocklea	47.3	46.4	44.4	33.4	28.5	22.5	17.7	7.0	19.1
Springwood	40.2	38.0	36.2	27.8	24.3	18.7	15.6	6.6	16.6
Flinders View	64.1	63.0	39.7	32.3	28.5	21.1	16.9	5.6	18.4

In most of the urban areas of Australia, where PM_{10} levels are monitored, they do exceed the current 1-day Air NEPM standard of 50 $\mu g/m^3$ (Figure 4), however the highest levels recorded are often in rural locations and are associated with bushfires wind-blown dust and for Launceston, solid wood heaters (Figure 5). Exceedences in the major cities occur on 1-5 days per year and are normally also associated with bushfires and wind-blown dust. The median level of PM_{10} was from 13 to 21$\mu g/m^3$ with higher median levels recorded at city locations (Figure 6).

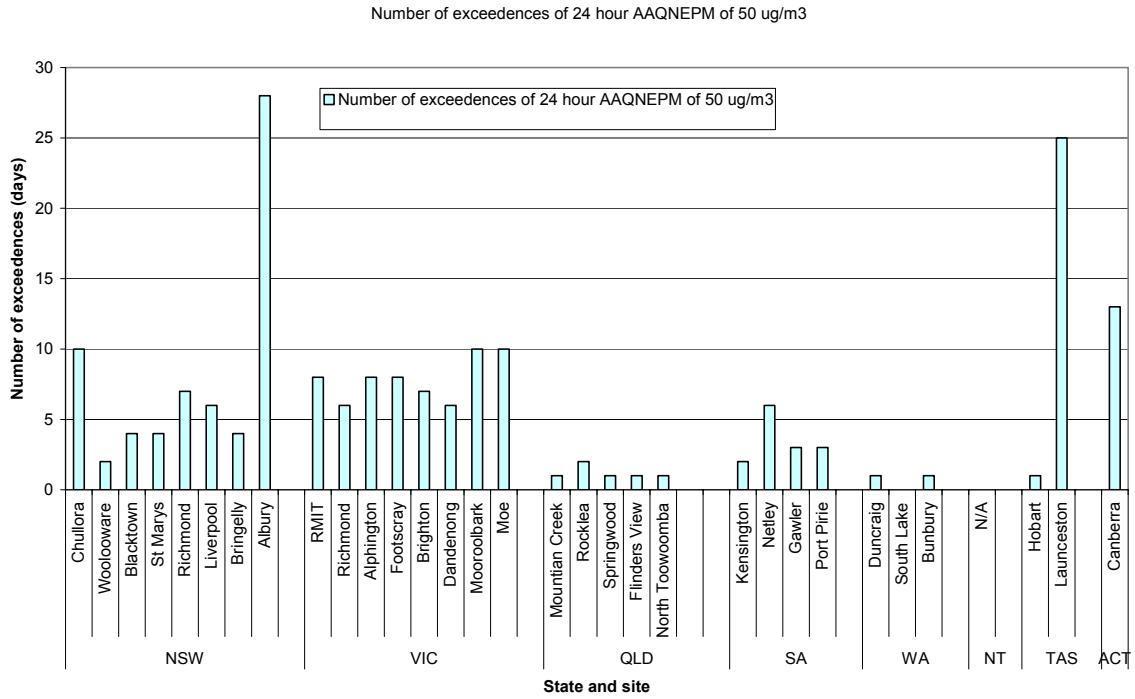

Figure 4: Number of times the 24 hour PM_{10} concentration exceeded 50 $\mu g/m^3$ in Australian Cities in 2003[1, 21-27]. The Ambient Air Quality National Environment Protection Measure (AAQ NEPM) is 50$\mu g/m^3$.

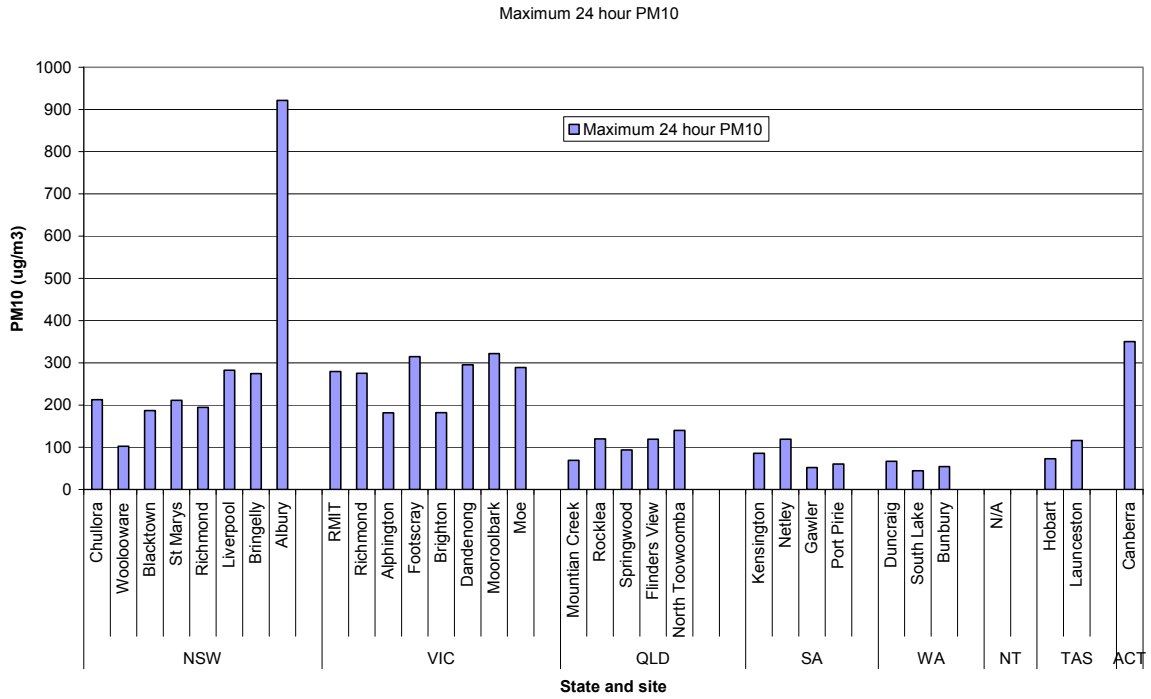

Figure 5: Maximum 24 hour PM_{10} in Australian Cities in 2003[1, 21-27]. The Ambient Air Quality National Environment Protection Measure (AAQ NEPM) is 50μg/m^3.

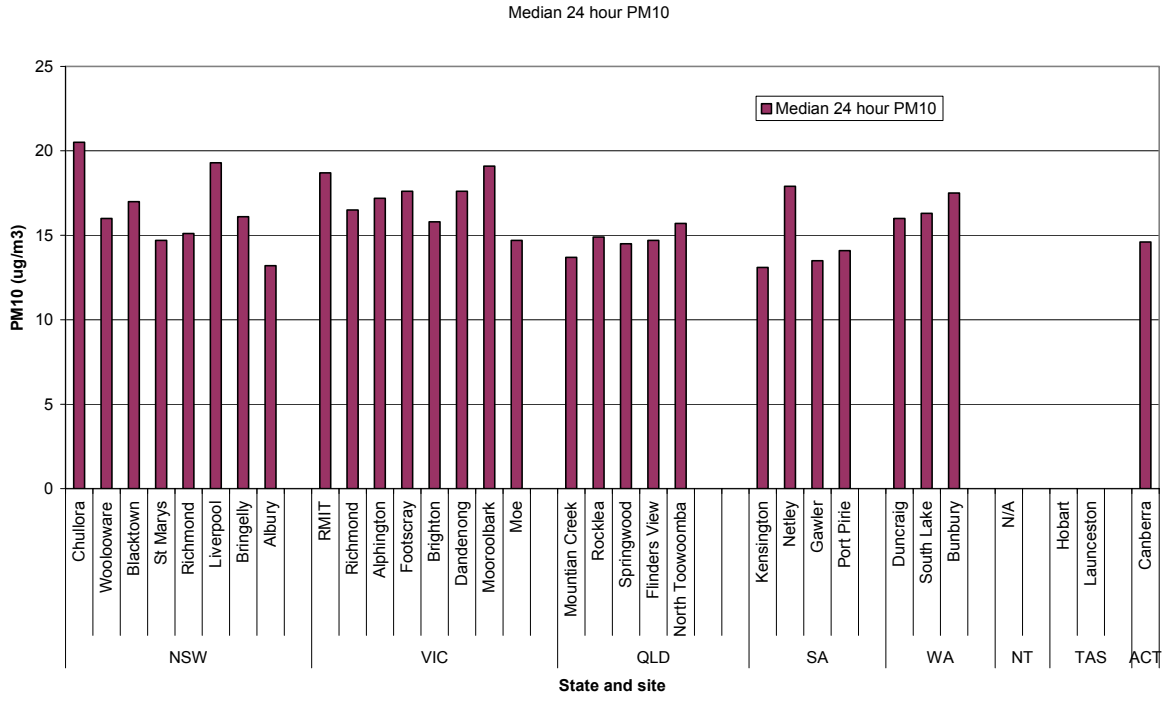

Figure 6: Median 24 hour PM_{10} in Australian Cities in 2003 [1, 21-27]. The Ambient Air Quality National Environment Protection Measure (AAQ NEPM) is 50μg/m^3.

While median ambient PM_{10} levels in Australia are below the AAQNEPM, they have not decreased over time (for example, Figure 7) as has occurred for a number of

other pollutants such as, lead, carbon monoxide and nitrogen dioxide. This has resulted in State EPA's expressing concern over the levels of PM_{10} in their major cities.

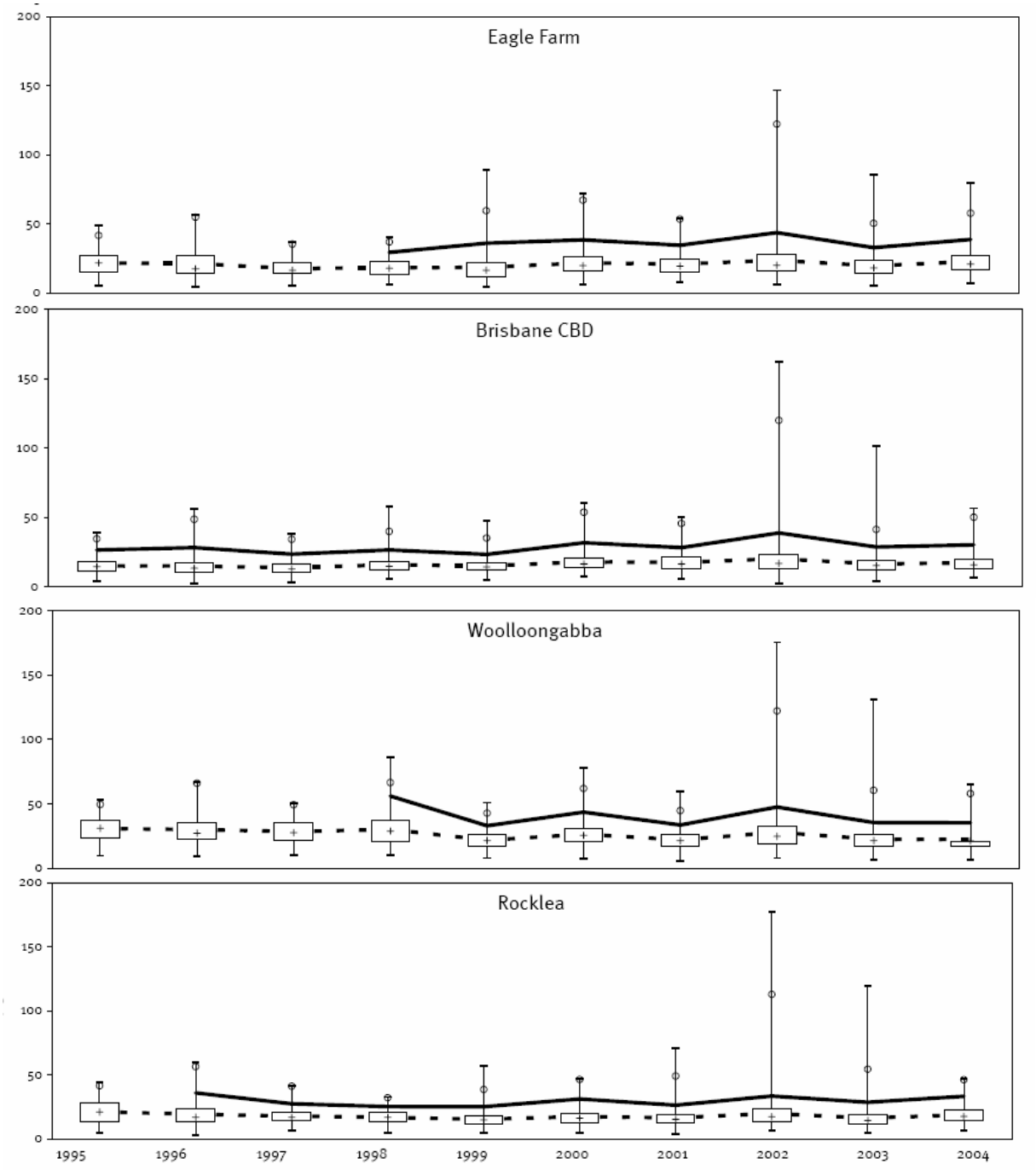

Figure 7: Median 24 hour PM_{10} (μg/m^3) at sites around Brisbane, over the period 1995-2004[6].

Ambient $PM_{2.5}$

During 2004, $PM_{2.5}$ was monitored at Rocklea and Springwood in south-east Queensland (Table 6). $PM_{2.5}$ concentrations exceeded the Air NEPM 24-hour average advisory standard of 25μg/m3 on five days at the Rocklea site. All were due

to bushfire smoke. The Air NEPM annual average advisory standard was also exceeded at the Rocklea site.

Table 6: Ambient $PM_{2.5}$ concentration* (24 hour maximum) statistics for south-east Queensland monitoring sites, 2004[6].

Monitoring location	Maximum (μg/m3)	Second highest (μg/m3)	Percentiles					Minimum (μg/m3)	Annual average (μg/m3)
			99 (μg/m3)	95 (μg/m3)	90 (μg/m3)	75 (μg/m3)	50 (μg/m3)		
Rocklea	34.9	32.0	30.6	19.9	14.0	10.4	7.6	2.8	9.0
Springwood	22.8	19.5	17.9	12.6	10.5	7.6	4.3	1.6	6.5

*Raw TEOM $PM_{2.5}$ data have been offset on the basis of observed 'baseline' values specific to each monitoring site. In 2004 these offset values were: Rocklea +2.7μg/m^3 and Springwood +1.0μg/m^3.

$PM_{2.5}$ levels in several Australian cities are given in Table 7. Although there is no AAQ NEPMs for $PM_{2.5}$, the advisory reporting standard for 24 hours is 25μg/m^3. The 24-hour average concentration occasionally exceeds the advisory reporting standard of 25 μg/m^3.

Table 7: $PM_{2.5}$ concentration in Australian cities in 2003[1, 21, 22, 24].

Location	Maximum (μg/m^3)	Median (μg/m^3)	# times exceeded 25 μg/m^3
South-east Queensland			
Rocklea	33.1	3.9	1
Springwood	20.6	4.8	0
Victoria			
CBD South East	44.6	7.0	4
Inner East Metro	38.4	5.7	4
Inner West Metro	51.1	4.8	3
South Metro	39.4	5.1	2
Outer East Metro	33.5	7.6	3
Western Australia			
North East Metro	27.3	7.6	1
North Metro	25.2	8.3	1
South West Region	37.6	7.8	3
New South Wales			
Chullora	81.0	9.7	6
Earlwood	39.4	9.6	9
Woolooware	67.7	9.4	5
Richmond	61.9	8.1	10
Westmead	67.8	10.3	4
Liverpool	50.1	11.9	11

Australian versus Overseas PM Levels

Internationally, out of 24 cities considered, Melbourne and Sydney rank respectively in 21st and 18th position for annual average PM_{10} concentration (Figure 8).

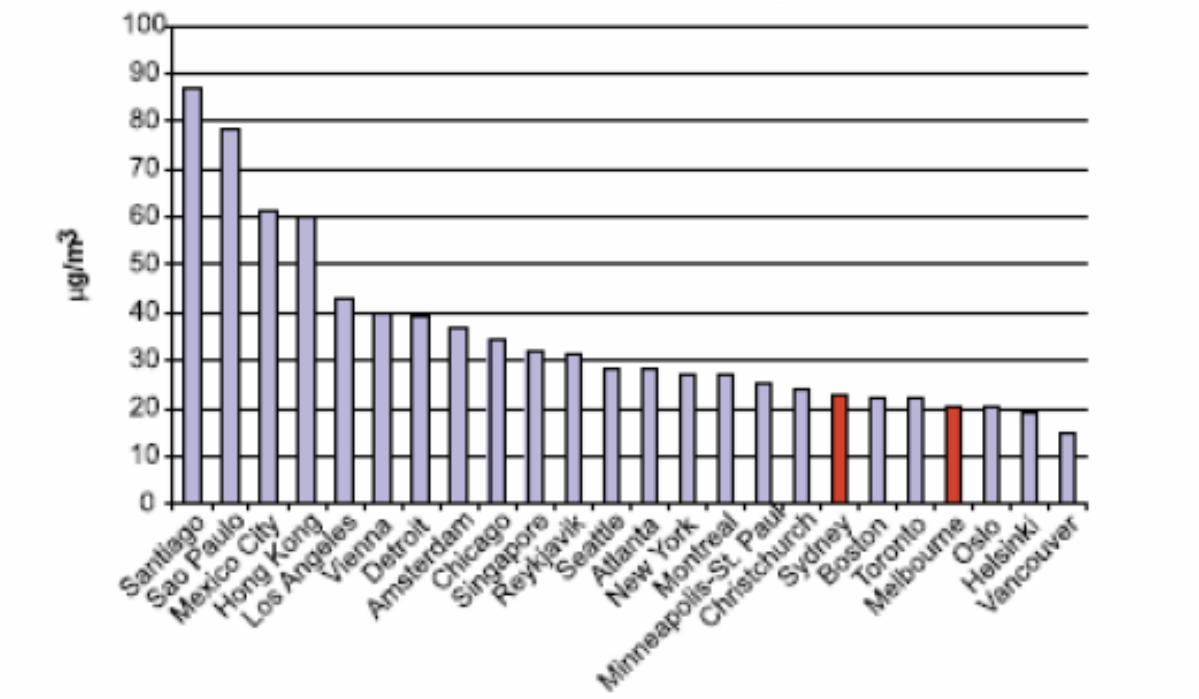

Figure 8: Annual average concentration of PM_{10} in selected cities in 1995. Source: Manins *et al*. 2001[28].

Assessing the effect of air pollution on health

There are three types of studies that examine the effects of air pollution on health. Each type of study has strengths and weaknesses and no single study can be interpreted as conclusive.

Experimental Chamber studies

Challenge chamber or experimental studies involve the exposure of individuals to a known concentration of a single pollutant or mixtures of pollutants under experimental conditions. An individual or a small group of people are exposed to a certain concentration of a single pollutant for a defined period of time. During the experimental studies people are exercising at a moderate or high intensity for the majority of the time they are exposed to the pollutant. Exercising increases the amount of pollutant inhaled and also deposits it further into the lower airways.

The advantages of chamber studies are that they are a controlled exposure study so that the dose of exposure can be accurately assessed. Chamber studies are extremely powerful for determination of dose response relationships, that is, the impact of concentration and time on a health outcome. Challenge chambers also enable quantitative outcomes to be measured such as changes in lung function, which requires some interpretation. For assessing lung function, both the rate and total volume of air that can be forcibly exhaled after a full and deep inspiration are measured. The rate of expiration of air is often expressed as forced expiratory volume in the first second of expiration and termed FEV_1. FEV_1 is often reported as percent of predicted FEV_1, which is based on established values that vary with sex, height and age. The maximum or peak expiratory flow rate (PEFR) is another

measure of the rate expiration. PEFR is the maximum flow rate of forcibly exhaled air following a deep breath. The volume of expired air is termed the forced vital capacity (FVC). Another measure of lung function is airway resistance. Airway hyperresponsiveness is a measure of the response of the lung to broncho-constricting stimuli (AHR).

Many studies examine the association between changes in FEV_1, or PEFR and levels of pollutants. In interpreting these studies it is necessary to assess the importance of percentage changes in FEV_1 or PEFR. Both FEV_1 and PEFR show diurnal variability in healthy subjects and in subjects with asthma and COPD. The US EPA expert panel concluded that a greater than 10 percent fall in FEV_1 should be regarded as an adverse effect (US Congress Office of Technology Assessment, 1989)[29]. The American Thoracic Society (1985, 2000)[30, 31] concluded that reversible loss of lung function associated with symptoms should be considered as adverse. In this report we have accepted that a short-term reduction in FEV_1 of 10% or more, which is attributable to a pollutant exposure, does constitute an adverse health effect.

Much of the variation caused by pre-existing symptoms, personal exposure variations, level of activities, duration of exposure, local changes in concentration of pollutants, mixes of pollutants, medical practices or subjective assessment of outcomes can be minimised in chamber studies. Subjects in the studies are blinded to the concentration of pollutant to which they are exposed and when combined with objective assessment of lung function they are less influenced by participant's perceptions. Participants in the challenge chamber studies may be selected from certain at risk groups in the community, for example those with asthma or hypertension.

The disadvantages of challenge chamber studies are that the number of participants is limited and therefore the application of the data to the wider at-risk population is uncertain. Chamber studies do not replicate the range of environmental exposures and activities undertaken across the community and they do not account for the vast range of other environmental factors that may influence a response to a pollutant or a mix of pollutants. Environmental conditions within chambers such as temperature, humidity and the mixture of environmental pollutants may not depict the mixture of these variables that occurs in the ambient air. Furthermore, it is only feasible to measure short-term outcomes in chamber studies, that is symptoms and changes in lung function. The effect of pollutants on disease exacerbations and serious adverse outcomes cannot be assessed.

Time series studies

Time series studies are a powerful way to examine the influence of a pollutant upon a community. In these studies a community health outcome, such as deaths, emergency department attendances or hospital admissions are related to the ambient levels of pollutants. The data on air pollutants and health outcomes are collected retrospectively. The data are adjusted to take account of a variety of non-pollutant factors that may influence the health outcome, such as influenza epidemics or weather. From these studies two important outcomes can be assessed: which groups in the community are most sensitive to pollutants; and what is the relationship between an increase in the level of a pollutant and a community health outcome. The

sensitivity of the health outcome to a change in pollutant level is normally expressed as an odds ratio or relative risk, which relates the increase in health outcome to a unit increase in pollutant. Odds ratios are often expressed as a percentage, for example, a 1.0 % increase in hospital admissions for asthma and COPD in response to a 10 $\mu g/m^3$ increase in ambient PM_{10}.

Panel or cohort studies

A common form of observational study is the panel or cohort study. In short term studies, a group of volunteers (eg at a summer camp or a school) record symptoms and lung function measurements every day over a period of weeks or months during which time air pollutants and other environmental variables are measured at the site. This is a more powerful way of assessing air pollution effects than chamber studies since they are performed on a larger number of subjects and under real conditions. It is often difficult, however, to separate out the relative importance of specific pollutants and impossible to determine the actual level of exposure.

Long-term cohort studies are also used to assess the cumulative impact of air pollutants on health. In such studies groups of people living in different locations, which have different levels of air pollutants, are followed over periods of up to 20 years to assess the long term impact of air pollution on mortality and development of specific diseases such as lung cancer. The impact on children's health has also been assessed in these studies by examining changes in lung function or the development of diseases such as asthma or leukaemia.

Thresholds: Is there a safe level of pollutant exposure?

A threshold is a level below which no adverse health effect will occur. There is currently no scientific basis for selecting a threshold for the effects of the major air pollutants (including CO, NO_2, PM_{10} and $PM_{2.5}$), if a threshold is defined as a level characterised by an absence of observable effects.

CO

According to the WHO (2000) [17], in healthy subjects, endogenous production of carbon monoxide results in carboxyheamoglobin (COHb) levels of 0.4–0.7% in blood. During pregnancy, elevated maternal COHb levels of 0.7–2.5%, mainly due to increased endogenous production, have been reported. The COHb levels in non-smoking general populations are usually 0.5–1.5%, owing to endogenous production and environmental exposures. To protect nonsmoking, middle-aged and elderly population groups with documented or latent coronary artery disease from acute ischaemic heart attacks, and to protect the fetuses of nonsmoking pregnant women from untoward hypoxic effects, a COHb level of 2.5% should not be exceeded. Table 8 gives guideline values (ppm values rounded) and periods of time-weighted average exposures have been determined in such a way that the COHb level of 2.5% is not exceeded, even when a normal subject engages in light or moderate exercise.

Table 8: Concentrations of CO and time weighted average exposure periods regarded as maintaining COHb levels below 2.5%[17].

Concentration	Time period
100 μg/m^3 (90 ppm)	15 minutes
60 μg m^3 (50 ppm)	30 minutes
30 μg/m^3 (25 ppm)	1 hour
10 μg/m^3 (10 ppm)	8 hours

NO_2

A threshold for NO_2 is difficult to establish due to the complex relationship of NO_2 with other pollutants. The WHO (2003) concluded that there was insufficient evidence to indicate a threshold for NO_2 below which no adverse health effects would occur [4].

PM

The National Morbidity, Mortality and Air Pollution Study (NMMAPS) recently examined whether a threshold existed for PM. Models were developed to test for thresholds for daily time-series data for the 20 largest US cities for 1987-1994, using concentration of diameter PM_{10} as the exposure measure. These studies indicated that linear models without a threshold are appropriate for assessing the effect of particulate air pollution on daily mortality even at current ambient levels [32]. There is no threshold for PM_{10}.

At Risk Groups

Precisely defining the at risk groups in the community is difficult and appears to depend on the nature of the study undertaken. For example, experimental challenge chamber studies suggest that individuals with asthma are not more susceptible to the effect of ozone than people without asthma. Asthmatics, however, appear to be one group in the community who are more likely to be admitted to hospital on high ozone days. Generally those most at risk are children and people with respiratory diseases such as asthma and COPD. This includes people with chronic bronchitis, emphysema and heart disease (Table 9).

It is generally acknowledged that children are more susceptible to the health effects of air pollutants than adults[33]. The following reasons have been proposed:

- Infant's metabolic pathways may be insufficient to detoxify pollutants;
- Children are more metabolically active and therefore inhale more air than adults;
- Children also have smaller calibre airways that may more easily constrict; and
- Children spend more time outdoors.

Table 9: People who appear to be most at risk from the health effects of air pollutants.

Pollutant	Group at risk	Impact on this group
CO	People with cardiovascular disease	Increase angina and increase risk of myocardial infarction
	Pregnant women and foetus	Reduced growth rate of the foetus.
NO_2	People with asthma	Bronchoconstriction following exposure to levels of > 0.26 ppm and allergen challenge.
		Increased risk of hospitalisation
	People with cardiovascular disease	Increased risk of hospitalisation
PM	Children	Reduced lung function growth
	People with chronic respiratory diseases (asthma and COPD)	Increased likelihood of hospitalisation.
	People with cardiovascular or ischaemic heart disease.	Increased likelihood of hospitalisation or death.

Acute effects of short term exposure to above ambient levels of air pollutants

CO

At very high concentrations (well above ambient levels), carbon monoxide causes a large number of acute accidental and suicidal deaths in the general population.

Unlike other gaseous pollutants, CO appears to have no toxic effect on the lungs, but its health effects are through interference with oxygen transport [17]. Inhaled CO combines with haemoglobin in the blood to form carboxyheamoglobin. The toxic effects of CO are due to hypoxia, which becomes evident in organs and tissues with high oxygen consumption such as the brain, the heart, exercising skeletal muscle and the developing fetus. During exposure to a fixed concentration of carbon monoxide, the COHb concentration increases rapidly at the onset of exposure, starts to level off after 3 hours, and reaches a steady state after 6–8 hours.

Carbon monoxide is eliminated unchanged via the lungs. The decline in COHb concentration depends on the rate of carbon monoxide release from haem proteins, alveolar ventilation, oxygen concentration of inhaled air, duration of carbon monoxide exposure, and the level of COHb saturation. The formation of COHb is a reversible process, but because of the tight binding of carbon monoxide to haemoglobin, the elimination half-life while breathing room air is 2–6.5 hours, depending on the initial COHb level. The elimination half-life of COHb is much longer in the fetus than in the pregnant mother [17].

Neurological and neurobehavioural effects
It is well known that severe hypoxia due to acute carbon monoxide poisoning may cause both reversible, short-lasting neurological deficits and severe, often delayed,

neurological damage. At a COHb level of about 10%, carbon monoxide is likely to cause headache, and at somewhat higher levels there will be also dizziness, nausea and vomiting. At a COHb level of about 40%, carbon monoxide starts to cause coma and collapse, and at 50–60% the poisonings are often lethal. The dose–effects of low-level carbon monoxide exposures on human behaviour have been critically reviewed and there seems to be reasonably good agreement that there is no significant impairment of visual or other behavioural functions in healthy young sedentary subjects at COHb levels below 18%. Early studies, however, suggested that these effects start at the much lower level of 3–5% in some people. One obvious reason for the discrepancy between the different studies is that the early studies showing the highest sensitivity to carbon monoxide were single-blind in design, whereas the more recent studies have been double-blind. During exercise there may be errors in behavioural tests at somewhat lower COHb levels than in resting conditions. It is also possible that abnormal cardiovascular function and other disease processes increase the sensitivity of subjects to carbon-monoxide-induced behavioural effects. Psychomotor effects, such as reduced coordination, tracking and driving ability, and impaired vigilance and detection of small environmental changes have been revealed in double-blind studies at COHb levels as low as 5.1–8.2%. From WHO (2000)[17].

The effects of carbon monoxide on cognitive performance have generally been equivocal at COHb levels of 5–20%. At COHb levels of 7% and 10% visual tracking performance can be significantly improved in resting conditions, but in contrast it is significantly impaired if the subjects engage in heavy exercise. Moreover, both response patterns seem to be dependent on the COHb concentration in the blood. From WHO (2000)[17].

Cardiovascular effects
Numerous controlled human studies have been conducted in healthy subjects and in patients with ischaemic heart disease in order to characterize the effects of low-level carbon monoxide exposures on the cardiorespiratory responses to exercise. In these experiments, the subjects have typically been exposed to clean air and carbon monoxide in a chamber or through a face-mask. This has usually been conducted at rest to achieve a predetermined COHb concentration in the blood. After the exposure, the subjects have engaged in an exercise test on a treadmill or cycle ergometer until exhaustion (healthy subjects) or the appearance of angina pectoris or electrocardiographic signs of cardiac ischaemia. From WHO (2000)[17].

In apparently healthy subjects, the maximal exercise time and the maximal oxygen consumption have decreased at COHb levels as low as 5%. The regression between the percentage decrease in maximal oxygen consumption and the percentage increase in COHb concentration appears to be linear, with approximately a one percentage point fall in oxygen consumption per 1% rise in COHb level above 4%. From WHO (2000)[17].

Patients with cardiovascular disease, especially ischaemic heart disease, are expected to be particularly sensitive to carbon monoxide. Atherosclerotic narrowing of the coronary arteries and impaired dilatation mechanisms restrict blood flow to the myocardium and prevent physiological compensation for lowered oxygen delivery caused by elevated levels of COHb. In exercise, these subjects experience

myocardial ischaemia, which can impair myocardial contractility, affect cardiac rate and rhythm, and cause angina pectoris. Early studies suggested that low-level carbon monoxide exposures resulting in COHb levels of 2.5–3.0% shorten the time to onset of exercise-induced chest pain in patients with angina pectoris. From WHO (2000)[17].

The design and results of the five most important clinical studies conducted in patients with ischaemic heart disease are summarized in Table 10.

Despite the obvious differences between these studies, they all show a significant shortening in the time to onset of angina at mean post-exposure COHb levels of 2.9–5.9% (post-exercise COHb levels in Table 10 are somewhat lower). This represents mean incremental increases of 1.5–4.4% COHb from the pre-exposure baseline levels. The potential arrhythmogenic effects associated with low-level carbon monoxide exposures have not been fully resolved at COHb levels of <5%. Effects on resting and exercise-induced arrhythmias in ten patients with coronary artery disease and no baseline ectopia have been reported at 3.5% and 4.9% COHb levels (post-exercise concentrations). In contrast, others showed in 41 nonsmoking patients with documented coronary artery disease and various levels of baseline ectopia that the frequencies of both single and multiple ventricular depolarizations increased significantly at a mean post-exercise COHb level of 5.0% but not at 3.5%. In another study, however, no additional effect was found of either 3% or 5% COHb over the exercise-induced increases in single or multiple ectopic beats experienced by patients with myocardial ischaemia and baseline ectopia. From WHO (2000)[17].

According to some epidemiological and clinical data, carbon monoxide from recent smoking and environmental or occupational exposures may contribute to cardiovascular mortality and the early course of myocardial infarction. It is not known whether this contribution is due to arrhythmogenic effects or to some longer-term effects, as suggested by some authors. In patients with severe ischaemic heart disease, carbon monoxide poisonings have been lethal at COHb levels of 10–30%, while usual COHb levels in lethal poisonings are around 50–60%. Stern et al. (1998) [34] investigated the effects of occupational carbon monoxide exposures on deaths from arteriosclerotic heart disease among 5529 New York City bridge and tunnel officers in the period 1952–1981. Among the more exposed tunnel officers there was a 35% excess risk compared with the New York City population, whereas among the less exposed bridge officers the risk was not elevated. The elevated risk among the tunnel officers declined significantly within five years after cessation of the occupational exposure, and there has also been a significant decline since 1970, when the introduction of new ventilation systems lowered the carbon monoxide levels in tunnels and tunnel booths. The 24-hour average carbon monoxide concentrations inside the tunnels were around 57mg/m^3 (50 ppm) in 1961 and 46 mg/m3 (40 ppm) in 1968. During rush hour traffic in 1968, carbon monoxide concentrations in tunnel toll booths were as high as 74–189 mg/m^3 (65–165 ppm) and in 1970 the mean concentration over 38 days was 72 mg/m^3 (63 ppm). However, the mean COHb levels measured among smoking and nonsmoking tunnel officers in 1970 and 1981 were generally lower than 5%. Current data from epidemiological studies and laboratory animal studies do not suggest that common environmental exposures to carbon monoxide have atherogenic effects on humans. From WHO (2000)[17].

Developmental effects
The pregnant mother, the fetus *in utero* and the newborn infant are at high risk of adverse health effects from atmospheric carbon monoxide exposures. During pregnancy, the endogenous production of carbon monoxide can be elevated as much as 3-fold, the concentration of maternal haemoglobin is often reduced, and the mothers have physiological hyperventilation. As a result of these changes, maternal COHb levels are usually about 20% higher than the non-pregnant values. From WHO (2000)[17].

Carbon monoxide diffuses readily across the placental membranes, and the carbon-monoxide binding affinity of fetal haemoglobin is higher than that of adult haemoglobin. Moreover, carbon monoxide is cleared much more slowly from fetal blood than from maternal blood. At steady state, fetal COHb levels are up to 10–15% higher than maternal COHb levels. There are theoretical reasons and supporting laboratory animal data to suggest that the fetus and the developing organs are especially vulnerable to carbon monoxide. The developing brain seems to have the highest sensitivity of all organs. There is a well established and probably causal relationship between maternal smoking and low birth weight at fetal COHb levels of 2–10%. In addition, maternal smoking seems to be associated with a dose-dependent increase in perinatal deaths and with behavioural effects in infants and young children. Carbon monoxide is probably one of the most important aetiological factors for these effects, although there are numerous other toxic pollutants in tobacco smoke. From WHO (2000)[17].

Table 10: A summary of results of the five most important double-blind clinical studies on the effects of low-level carbon monoxide exposures on patients with documented ischaemic heart disease and exercise-induced angina. From WHO (2000)[17].

Reference	Exposure		Exposure duration and activity	Subject characteristics	Effects of CO exposure (symptoms, ECG changes, etc.)
	CO(mg/m^3)[a]	COHb (%)[b]			
Anderson[35]	0 57 115	1.3 2.9 4.5	4-hour exposure at rest, post-exposure exercise on a treadmill	10 males, mean age 49.9 years (5 smokers 5 nonsmokers), reproducible angina	Time to onset of angina shortened at COHb 2.9% and 4.5% (P < 0.005) and duration of angina prolonged at COHb 4.5% (P < 0.01). Deeper ST-segment depressions with CO in 5 subjects.
Kleinman[36]	0 115	1.4 2.8	1-hour exposure at rest, post-exposure incremental exercise on a cycle ergometer	24 males, mean age 58.8 years (nonsmokers for at least 6 months), reproducible angina	Time to onset of angina shortened by 5.9% (P = 0.046), no significant effect on duration of angina, oxygen uptake at angina reduced by 2.2% (P = 0.04). Time to 0.1 mV ST-segment depression shortened by 19.1% (P = 0.044) in 8 subjects.
Allred[37]	0 134 290	0.6 2.0 3.9	50- to 70-minute exposure at rest, pre- and post-exposure incremental exercise on a treadmill	63 males, mean age 62 years (nonsmokers), reproducible angina	Time to onset of angina shortened by 4.2% (P = 0.054) at COHb 2.0% and by 7.1% (P = 0.004) at COHb 3.9%. Time to threshold ischaemic ST-segment changes shortened by 5.1% (P = 0.02) at COHb 2.0% and by 12.1% (P < 0.0001) at COHb 3.9%. Significant dose relationships in the changes of both the onset of angina (P = 0.02) and the onset of ST-segment changes (P < 0.0001).
Sheps[38]	0 115	1.6 5.2	1-hour exposure at rest, post-exposure incremental exercise on a cycle ergometer	25 males and 5 females, mean age 58.2 years (nonsmokers for at least 2 months), ischaemia in a screening test	No significant changes in time to onset of angina, duration of angina, maximal exercise time, maximal ST-segment depression, time to significant ST-segment depression, or maximal left ventricular ejection fraction. 3 subjects experienced angina only on CO exposure, actuarial analysis including these subjects showed shortening in time to onset of angina in the study group (P < 0.05).
Adams[39]	0 115-229	1.6 5.2	1-hour exposure at rest, post exposure incremental exercise on a cycle ergometer.	22 males and 8 females, mean age 58 years, non-smokers for at least 2 months, ischaemia in a screening test.	Maximal exercise time shortened by 6.5% (P < 0.05), level and change in left ventricular ejection fraction at submaximal exercise reduced (P = 0.05). Shortening in time to onset of angina (P < 0.05) according to actuarial analysis.

[a]Carbon monoxide, 1 mg/m^3 = 0.873 ppm.
[b]Carboxyhaemoglobin concentrations are from venous blood samples taken immediately after exercise; in the study of Anderson et al. *(40)* samples were taken only immediately after carbon monoxide exposure.

NO_2

It appears that NO_2 itself is not a potent inducer of lung inflammation, bronchoconstriction or an inducer of respiratory symptoms in either asthmatic or non-asthmatics (Table 11). Some studies have demonstrated bronchoconstriction in healthy people resulting from exposure of 1.5-5 ppm, while others have observed no effects at 4 ppm [40].

People with asthma, compared to non-asthmatics, appear to be more susceptible to increased airway hyperresponsiveness following exposure to NO_2 and a bronchoconstricting agent such as an allergen [40, 41]. Studies in asthmatic subjects have failed to see changes in lung function as a result of exposure to NO_2, alone, in the range of 300-1230 mg/m^3 (0.24 - 0.6ppm) for exposure periods of 15-60 minutes [42-45], although there is individual variation. In people with severe asthma, exposure to 0.6 ppm for 1 hour did not result in significant changes in lung function or symptoms [43]. Furthermore, exposure of people with mild asthma to 500μg/m^3 (0.24ppm) for 30 minutes or 0.4 ppm for 1 hour followed by allergen challenge results in clinically insignificant [46] changes in lung function or symptoms.

There is conflicting data on the susceptibility of people with COPD to acute exposure to NO_2. Early studies suggest people with COPD to be more susceptible than healthy people, however later studies have not supported these results[40].

The impact of acute exposures, such as occurs on a 5-10 minute trip through a road tunnel, has not been tested.

Koenig et al. (1988)[47] tested the sensitivity of 10 healthy adolescents and 10 adolescents with asthma to ozone and nitrogen dioxide. The adolescents were exposed via a mouthpiece to three different atmospheres (filtered air, ozone, and nitrogen dioxide, at either 0.12 or 0.18 ppm) each for 2 x 30 minute exposures on separate days at least one week apart. Before, during, and after exposure lung function was measured. Pulmonary function was not significantly altered in either the asthmatic or the healthy non-asthmatic adolescents as a result of either the 0.12 or 0.18 ppm exposures. After exposure to 0.18 ppm nitrogen dioxide there was a 3 percent decrease in the forced expiratory volume in one second in asthmatic subjects. This change was not significant. It was concluded that there were no differences in pulmonary function responses between asymptomatic, allergic asthmatic adolescents and healthy adolescents exposed to either ozone or nitrogen dioxide under the conditions of these studies.

Avol et al. (1989)[48] exposed 34 asthmatic volunteers aged 8 to 16 on separate occasions to clean air (control), to 0.30 ppm nitrogen dioxide (NO_2) in otherwise clean air, and to polluted Los Angeles area ambient air on summer mornings when NO_2 pollution was expected. Exposures lasted 3 hrs, with alternating 10-minute periods of exercise and rest. In ambient pollution exposures, 3-hr average NO_2 concentrations ranged from 0.01 to 0.26 ppm, with a mean of 0.09 ppm. Ambient exposures did not significantly affect lung function, symptoms, or bronchial reactivity to cold air, relative to the control condition. Responses to 0.3 ppm NO_2 exposures were equivocal. Lung function declined slightly during the first hour at 0.3 ppm, but

improved over the remaining 2 hr. Compared to other conditions, symptoms were not increased during 0.3 ppm exposures, but were increased during the 1-week period afterward.

Morrow and Utel (1989)[49] investigated the symptom responses and changes in the pulmonary function of people with mild to moderate asthma, people with chronic obstructive pulmonary disease (COPD) and people with no-respiratory-impairment (controls), when exposed to 0.3 parts per million (ppm) (560 micrograms/m^3) nitrogen dioxide or air in a double-blind crossover design. Subjects were randomly exposed to air or NO_2 for 4 hours during intermittent exercise, with a one day observation period between exposures. All four-hour exposures included several pre determined periods of exercise and pulmonary function measurements. This group had a mean age of 60.0 years and consisted of 13 men and 7 women. No significant symptomatic or physiologic responses to nitrogen dioxide could be detected in either the young or the elderly control group. The asthmatic group as a whole did not manifest significant reductions in lung function after exposure to 0.3 ppm nitrogen dioxide compared to their pre exposure base-line data or to their responses after a comparable four-hour exposure to air.

Tunnicliffe et al. (1994) [42] exposed 8 people with asthma to air, 0.100 ppm nitrogen dioxide, or 0.4 ppm nitrogen dioxide for 1 hour, in double-blind, random order, then immediately challenged them with house dust mite allergen. Baseline forced expiratory volume in 1 s (FEV_1) was not affected by any of the gas mixtures. The mean early asthmatic response (maximum percentage change in FEV_1 during first 2 h after challenge) was -14·62% (SD 8·03) after air, -14·41% (7·86) after 0.1 ppm nitrogen dioxide, and -18·64% (7·28) after 0.4 ppm nitrogen dioxide. The difference between air and 0.4 ppm was small (-4·01%), but significant (95% CI -1·34 to -6·69%, $p<0·009$), but those between air and 0.1 ppm and between 0.1 and 0.4 ppm were not significant (0·21 [-3·10 to 3·53]% and -4·23 [-8·75 to 0·29]%). The mean late asthmatic response (maximum % change in FEV_1) to challenge after air was -2·85% (3·95), after 0.1 ppm nitrogen dioxide -7·76% (6·92), and after 0.4 ppm -8·13% (6·64). The difference in means between the air and 0.4 ppm exposures was significant (-5·28 [-0·73 to -9·83]%, $p<0·02$) but those between air and 0.1 ppm (-4·90 [-10·60 to 0·78]%) and 0.1 and 0.4 ppm (0·37 [-3·06 to 3·80]%) were not.

Jenkins et al (1999)[50] exposed eleven mild atopic asthmatic patients for 6 hrs, in randomized order, to air or 0.2 ppm NO_2, followed immediately by bronchial allergen challenge. Subsequently 10 of these patients were exposed for 3 h to air or 0.4 ppm NO, followed immediately by bronchial allergen challenge. All exposures were carried out in an environmental chamber, with intermittent moderate exercise, and a minimal interval of 2 wks between exposures. Exposure to NO_2 alone had no significant effect on lung function; however there was considerable individual variation (Figures 9 and 10). Exposure for 6 hrs to 0.2 ppm NO_2 did not lead to any significant increase in the airway response of these individuals to inhaled allergen, when compared with exposure for 6 h to air. In contrast, exposure for 3 hrs to 0.4 ppm NO_2 significantly decreased the dose of allergen (in log cumulative breath units [CBU]) required to decrease FEV_1 by 20% (allergen $PD_{20}FEV_1$), compared with exposure to air (geometric mean CBU: 3.0 for air versus 2.78 for NO_2 ($p=0.018$).

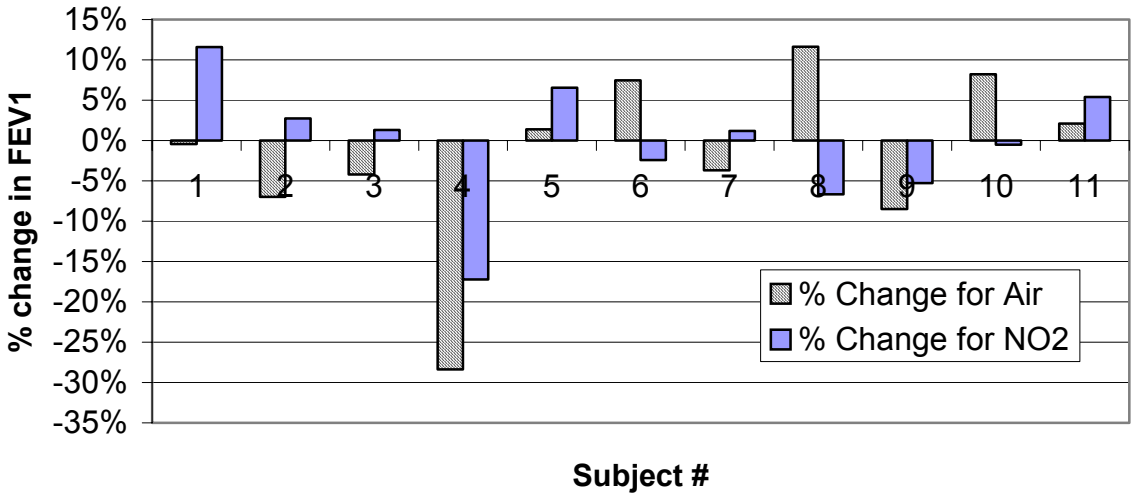

Figure 9: Effect of exposure to 0.2 ppm NO_2 for 6 hours on lung function in people with mild asthma [50].

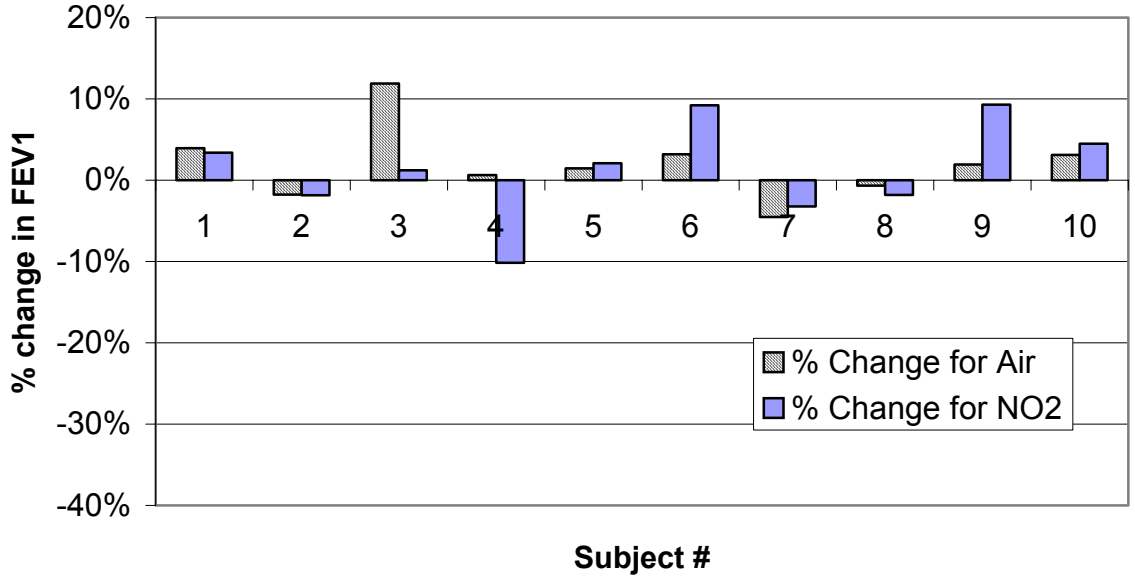

Figure 10: Effect of exposure to 0.4 ppm NO_2 for 3 hours on lung function in people with mild asthma [50].

Strand et al. (1996)[51] exposed 19 subjects with mild asthma to either purified air or 490 $\mu g/m^3$ (0.26 ppm) NO_2 for 30 minutes during intermittent exercise (Table 11). Symptoms, airway responsiveness to histamine, inflammatory mediators in blood, airway resistance and thoracic gas volume (TGV) were measured following histamine challenge. NO_2 exposure alone did not affect sRaw, but TGV was significantly reduced after exposure. Bronchial responsiveness to histamine was significantly increased 5 hrs after NO_2 exposure, when compared to air. NO_2 increased the levels

of one of the inflammatory mediators (Mac-1) on immune cells in blood 30 minutes after exposure when compared to pre-exposure values. No effect was seen on other mediators (tryptase, eosinophil cationic protein (ECP), or myeloperoxidase (MPO)).

In a later study, Strand and colleagues (1998)[52] investigated the effects of NO_2 and allergen on lung function in a repeated exposure model (Table 11). For 4 subsequent days, 16 subjects with mild asthma and allergy to birch or grass pollen were exposed at rest to either purified air or 500 $\mu g/m^3$ NO_2 for 30 minutes in an exposure chamber. Four hours later they were challenged with allergen. Acute changes in lung function (FEV_1) were measured at 15 minutes, while delayed responses were assessed 3–10 hours after allergen. Subjective symptoms and medication use were recorded. Small, but significant reductions in FEV_1 were recorded following repeated NO_2 exposure and allergen challenge. The 4-day mean acute fall in FEV_1 after NO_2 was at -2.5% versus -0.4% for air (p=0.02) and the fall in delayed response was -4.4% versus -1.9% for air (p=0.01). An increase in acute response was seen after a single NO_2 exposure (p=0.03). There was no significant change in symptoms or medication use although there was a tendency (p=0.07) towards increased night-time symptoms of asthma after NO_2 plus allergen.

Barck et al. (2002) exposed 13 people with mild asthma to either 500$\mu g/m^3$ (0.26 ppm) NO_2 or air for 30 minutes, followed by an allergen challenge 4 hours later(Table 11). Lung function was assessed by measuring FEV_1 and airway resistance. Exposure to 500$\mu g/m^3$ of NO_2 for 30 minutes did not result in a decrease in FEV_1, airway resistance or symptoms. Following allergen challenge there were also no differences between NO_2 and air exposure for FEV_1, airway resistance or symptoms. However there appears to be a change in inflammatory response following exposure to NO_2 plus allergen, which was not reflected in changes in lung function. Inflammation was measured by the levels of inflammatory cells in bronchial washes (BW) and bronchoalveolar lavage (BAL) fluid. Surprisingly there was a significant decrease in the total cells and macrophages in BAL after NO_2 plus allergen exposure, suggesting a reduced inflammatory response. Furthermore the number of eosinophils and mast cells did not differ between NO_2 plus allergen and air plus allergen exposures for either BW of BAL. Eosinophils and mast cells are associated with allergic inflammation. There was however a significant increase in the percentage of neutrophils in both BW and BAL. Of the seven inflammatory markers measured, there was a no change in six and a significant increase in ECP in both BW and BAL.

Recently, Barck et al. (2005)[44] exposed 18 people with mild asthma to either air or 500$\mu g/m^3$ (0.26 ppm) NO_2 for 15 minutes on Day 1 followed four hours later by an allergen challenge (Table 11). On Day 2, the air or NO_2 exposures were performed on two occasions followed by an allergen challenge. Exposure to 500$\mu g/m^3$ NO_2 for 15 minutes did not result in a decrease in lung function or symptoms on Day 1 or Day 2. Likewise there was no significant difference between pre-exposure to air or NO_2 in lung function or symptoms following allergen challenge. Exposure to NO_2 followed by allergen challenge resulted in significantly higher levels of ECP in sputum and blood, in comparison to air plus allergen challenge. There were no significant differences between air and NO_2 for total cells, eosinophils or neutrophils in sputum.

The clinical significance of these changes in inflammatory cells and mediators[44, 45] in the absence of changes in lung function or symptoms, remains unclear. It has been suggested that such inflammation may result in airway remodelling in asthmatics [44]. This hypothesis is firstly based on the observations that people with asthma often have airway remodelling and airway inflammation. Secondly, airway remodelling is more prevalent in people who have had asthma for longer, which may indicate that prolonged inflammation in the lungs is associated with airway remodelling. However, the mechanisms of airway remodelling are still to be elucidated and inflammation per se has not been demonstrated to be the cause of airway remodelling in humans.

Table 11: Challenge chamber studies with NO_2

Study	Experimental group	Level and duration of exposure to NO_2.	Health outcomes measured and results.
Barck et al. (2005)[44]	n = 18, atopic, mild-asthmatics.	Day 1: 500 $\mu g/m^3$ for 15 minutes followed by allergen challenge 4 hrs later. Day 2: 500 $\mu g/m^3$ for 15 minutes, 1 hr elapsed, 500 $\mu g/m^3$ for 15 minutes followed by allergen challenge 4 hrs later.	Lung function, airway resistance & symptoms following NO_2 exposure and allergen challenges. Inflammatory mediations and cells in sputum on Days 1, 2 & 3. No effect of NO_2 exposure on lung function, airway resistance, symptoms, or response to allergen challenge. Significant increase in ECP between Days 1 & 3 in both sputum and blood after NO_2 exposure plus allergen challenge. Decreases in the levels of the anti-oxidant MPO in blood.
Pathmanathan et al. (2003) [40]	n = 12, healthy adults, non-asthmatics, non-smokers, exercising.	2 ppm NO_2 for 4hrs/day on 4 days	Inflammatory mediators in the lungs (bronchial biopsies). Significant increases in : IL-5, IL-10, IL-13, ICAM-1
Barck et al. (2002)[45]	n = 13, atopic, mild-asthmatics.	500 $\mu g/m^3$ for 30 minutes. Allergen challenge 4 hrs later.	Lung function, airway resistance, symptoms, inflammatory mediations and cells in bronchial wash & lavage response to allergen challenge 4 hours post NO_2 exposure No effect on lung function, resistance or symptoms, increased neutrophils and mediators.
Svartengren et al. (2000)[53]	n = 20 adults with mild asthma. Exposed to pollutants within a road tunnel.	30 minute exposures to NO_2 313 $\mu g/m^3$ (range 203–462). PM_{10} & $PM_{2.5}$ 170 (range 103–613) & 95 (range 61–218) $\mu g/m^3$, respectively.	Lung function, airway resistance, symptoms pre and post bronchial challenge. No effect on symptoms. Exposure to > 300 $\mu g/m^3$ NO_2 had a significantly greater early reaction, following allergen exposure, as well as lower lung function and more asthma symptoms during the late phase. Exposure to > 100 $\mu g/m^3$ $PM_{2.5}$ increased early reaction.
Blomberg et al. (1999)[54]	n = 12, healthy adults, non-asthmatics, non-smokers, exercising.	2 ppm NO_2 for 4-hours/day on 4 days.	Lung function and inflammatory mediators in the lungs (bronchial biopsies, lavage and washes). Significant reductions in FEV_1 and FVC. Significant increases in neutrophils and

			antioxidants (myeloperoxidase).
Strand et al. 1996[51]	n = 19, people with mild asthma, atopic, non-smokers, exercising.	0.26 ppm NO_2 or air for 30 minutes.	Significant changes in airway response to histamine, thoracic gas volume and blood Mac-1. No change in airway resistance, symptoms, blood ECP, MPO or tryptase
Strand et al. 1998[52]	n = 16, people with mild asthma, atopic, non-smokers, at rest.	0.26 ppm NO_2 or air for 30 minutes on 4 subsequent days followed by allergen challenge.	Small but significant changes in lung function (FEV_1). No change in symptoms or mediation use.
Blomberg et al (1997)[55]	n = 30, healthy adults, non-asthmatics, non-smokers, exercising.	2 ppm NO_2 for 4hours.	Inflammatory mediators in the lungs (bronchial biopsies and bronchial lavage). Significant increases in IL-8, neutrophils, memory T-cells and B-cells.
Rubinstein et al. (1991)[56]	n=5, healthy adults, non-asthmatics, non-smokers, exercising	0.60 ppm NO_2 for 2 hrs /day on 4 days.	No effect on symptoms, airway calibre, or alterations in circulating and BALF lymphocyte subtypes.
Salome et al. (1996) [43]	n=9 adults & 11 children with severe asthma, resting.	0, 0.30, 0.60 ppm NO_2 mixed with ambient air or in combination with combustion products for 1 hr	No significant effect on symptoms or lung function (FEV_1, PEFR). Significant increase in airway reactivity (PD_{20} FEV_1 to histamine) at 0.6 ppm.

Particles

Several experimental challenge chamber studies have measured the acute health effect of exposing individuals to high concentrations of particles generated from diesel engines or concentrated ambient pollutants (Table 12).

Exposure to concentrated particles or diesel particles results in lung inflammation that is dose dependant [57]. Exposure to levels of 100-300 $\mu g/m^3$ of PM_{10} has consistently been found to cause inflammation. This inflammatory response is characterised by neutrophil infiltration into the lungs and occurs in both people with and without asthma (Table 12).

Rudell et al. (1996)[58] exposed healthy non-smokers for one hour to diesel smoke that contained 2.6 x $10^6/cm^3$ particles, 1.9 ppm NO_2, 2.7 ppm NO, 30 ppm CO and other pollutants. They also examined the effect of partial filtration of the smoke, which resulted in a 46% reduction in particles, but no change in the other pollutants. Compared with air, exposure to diesel smoke or partially filtered smoke, resulted in significant increases in unpleasant smells and irritation to the eyes and nose [58]. These symptoms varied enormously between the small number of individuals (n=12) tested [58]. Diesel smoke or partially filtered smoke, when compared with air also resulted in a significant increase in airway resistance, but no change in FEV_1 or FVC.

Table 12: Challenge chamber studies with particles.

Study	Experimental group	Level and duration of exposure PM	Health outcomes measured and results.
Rudell et al. (1996)[58]	n = 12, healthy adults, non-asthmatics, non-smokers, exercising.	Partially filtered diesel exhaust for 1 hour , which contained: 1.4-2.6 x 10^5/cm^3 particles, 1.7-1.9 ppm NO_2, 2.6-2.7 ppm NO and 27-30 ppm CO, plus hydrocarbons and formaldehyde.	Symptoms, lung function and airway resistance. Symptoms of eye and nose irritation were increased although highly variable between individuals and therefore not significantly different. Significant increase in airway resistance.
Salvi et al. (1999)[59]	n = 15, healthy adults, non-asthmatics, non-smokers, exercising	300 μg/m^3 diesel particles (PM_{10}) for 1 hour on two occasions.	Lung function, inflammatory cells and mediators in bronchial lavage and serum. Significant increase in neutrophils, mast cells and lymphocytes in the lung and increases in neutrophils and platelets in blood.
Ghio et al. (2000) [57]	n = 38 (divided into 4 groups), healthy adults, non-asthmatics, non-smokers, exercising.	0, 48, 107 & 207 μg/m^3 for 2 hours.	Symptoms, lung function, inflammatory mediators and cells in the lungs (bronchial lavage). No change in symptoms, FEV_1 and FVC, PEF or airway resistance. Significant increases in neutrophils and no change in IL-8 or IL-6. Significantly increased fibrinogen in blood.
Nightingale et al. (2000)[61]	n = 10, healthy adults, non-asthmatics, non-smokers, at rest.	200 μg/m^3 diesel particles (PM_{10}) for 2 hours.	Lung function, AHR, Inflammatory cells and mediators in sputum. Significant increase in neutrophils and myeloperoxidase.
Nordenhall et al. (2000)[62]	n = 15, healthy adults, non-asthmatics, non-smokers, exercising.	300 μg/m^3 diesel particles (PM_{10}) for 1 hour on two occasions.	Inflammatory cells and mediators in sputum. Significant increase in neutrophils, IL-6 and methylhistamine.
Nordenhall et al. (2001)[60]	n = 14, atopic asthmatics, with stable asthma exercising.	300 μg/m^3 diesel particles (PM_{10}) for 1 hours on two occasions.	Lung function, airway resistance and AHR. Inflammatory cells and mediators in sputum. Significant increase in airway resistance, AHR and IL-6.
Svartengren et al. (2000)[53]	n = 20 adults with mild asthma. Exposed to pollutants within a road tunnel.	30 minute exposures to NO_2 313 □g/m^3 (range 203–462). PM_{10} & $PM_{2.5}$ 170 (range 103–613) & 95 (range 61–218) μg/m^3, respectively.	Lung function, airway resistance, symptoms pre and post bronchial challenge. No effect on symptoms. Exposure to > 300 □g/m^3 NO_2 had a significantly greater early reaction, following allergen exposure, as well as lower lung function and more asthma symptoms during the late phase. Exposure to > 100 □g/m^3 $PM_{2.5}$ increased early reaction.

Salvi et al. (1999)[59] exposed 15 healthy human volunteers to air and diluted diesel exhaust under controlled conditions for 1 h with intermittent exercise. The exposures

were standardized by keeping the PM_{10} concentration at 300μg/m^3,which was associated with 1.6 ppm NO_2, 4.5 ppm NO, 7.5 ppm CO; 4.3 ppm total hydrocarbons, 0.26 mg/m^3 formaldehyde, and 4.3 x 10^6 suspended particles/cm^3. Lung function (PEFR, FEV_1, FVC) was measured before and after each exposure and did not change following diesel exposure. Blood sampling and bronchoscopy were performed 6 hrs after each exposure to obtain airway lavages and endobronchial biopsies. They found no changes in lung function but both systemic and pulmonary inflammatory responses. There was a significant increase in neutrophils and B lymphocytes in airway lavage, along with increases in histamine and fibronectin. The bronchial biopsies obtained 6 hrs after diesel exposure showed a significant increase in neutrophils, mast cells, CD4+ and CD8+ T lymphocytes along with upregulation of the endothelial adhesion molecules ICAM-1 and VCAM-1, with increases in the numbers of LFA-11 cells in the bronchial tissue. Significant increases in neutrophils and platelets were observed in peripheral blood following diesel exposure.

Nordenhall et al. (2001)[60] exposed fifteen healthy nonsmoking volunteers to 300μg/m^3 of PM_{10} and air for 1 h on two separate occasions. Analyses of sputum differential cell counts and soluble protein concentrations were performed 6 and 24 hours post exposure. Six hours after exposure to diesel exhaust, a significant increase was found in the percentage of sputum neutrophils (37.7 *versus* 26.2% p=0.002) together with increases in the concentrations of interleukin-6 (12.0 *versus* 6.3 pg.mL^{-1} , p=0.006) and methylhistamine (0.11 *versus* 0.12 μg.L-1 , p=0.024). Irrespective of exposure, a significant increase was found in the percentage of sputum neutrophils at 24 h as compared to 6 hrs, indicating that the procedure of sputum induction itself may change the composition of sputum.

Combined exposures

The impact of NO_2 and NO_2 plus combustion products on symptoms, airway responsiveness and lung function in children (n=11) and adults (n= 9) with relatively severe asthma was examined by Salome et al. (1996)[43]. There were no significant effects following 1 hour exposures to air, 0.3 and 0.6 ppm NO_2 or NO_2 in combination with other combustion products from a gas heater on lung function or symptoms. There was, however, a trend toward higher symptoms with increasing NO_2 exposure. Significant increases in airway reactivity were observed after exposure to 0.6 ppm NO_2 in ambient air, but not when combustion products were also included (Table 11).

The acute effect of motor vehicle exhaust on adults with asthma was assessed by Svartengren et al. (2000)[53]. Twenty subjects with mild asthma were placed in a car inside a road tunnel for 30 minutes during peak-hour. This tunnel had a length of ~1,500m and was used by ~35,000 vehicles per day. The levels of NO_2, PM_{10} and $PM_{2.5}$ were assessed inside the car, in the tunnel and at an urban location where control studies were performed. Four hours after tunnel or control exposures, subjects were challenged with a mild dose of inhaled allergen.

The median NO_2 concentration inside the car was 313 μg/m^3 (range 203–462) and was 28 times higher than the ambient exposure at the urban control site which was 11 μg/m^3 (range 0–51). The PM_{10} and $PM_{2.5}$ concentrations in the car were 170μg/m3 (range 103–613) and 95μg/m^3 (61–218), respectively. PM_{10} and PM_2 levels at the

control urban site were 14 times lower at 7$\mu g/m^3$ (range 2–17) and 5 μg/m3 (range 2–11), respectively.

Symptoms of noise, smell, cough irritation and self-perceived respiratory health were adversely affected by being in the tunnel. There were no differences in airway resistance or lung function during exposure to NO_2, PM_{10} and $PM_{2.5}$ in the tunnel (Table 12).

Once people left the tunnel and were challenged with allergen, their lungs were more reactive and a significant increase in airway resistance was found. Overall, the effect of NO_2 across the entire group was not significant and there was no significant relationship between the level of NO_2 and change in airway resistance. However, when the group was divided into those with exposures above 300 $\mu g/m^3$ (0.15 ppm) there was a significant increase in airway resistance and decrease in lung forced expiratory flow in those exposed to >300 $\mu g/m^3$ compared with the control (unexposed) group. The percentage reduction in FEV_1 was 8.5% for >300 $\mu g/m^3$ NO_2 versus 6.8% for the air control.

Subjects with the highest $PM_{2.5}$ exposure (>100 $\mu g/m^3$) had a marginally greater early reaction after allergen challenge, compared with the control groups (no exposure).

Tunnel exposure resulted in significantly more asthma symptoms, following allergen challenge, during the evening after the tunnel exposure than after the control exposure (p=0.016). Furthermore, the combined asthma symptoms during the evening, night and morning after the exposure tended to be worse (p=0.085). These symptoms were significantly related to NO_2 exposure. Asthma symptoms were significantly increased compared to control, when NO_2 exposure was >300 $\mu g/m^3$. The group with NO_2 exposure above 300$\mu g/m^3$ had significantly more asthma symptoms during the exposure evening compared to the control group and more symptoms during the night after allergen inhalation compared to the group with those who were exposed to less than 300 $\mu g/m^3$.

The study by Svartengren et al (2000)[53] found that exposure to $PM_{2.5}$ in the road tunnels had no effect on symptoms (Figure 11), caused a slight increased in the early phase lung asthmatic reaction, but no change in the late phase reaction (Figure 12).

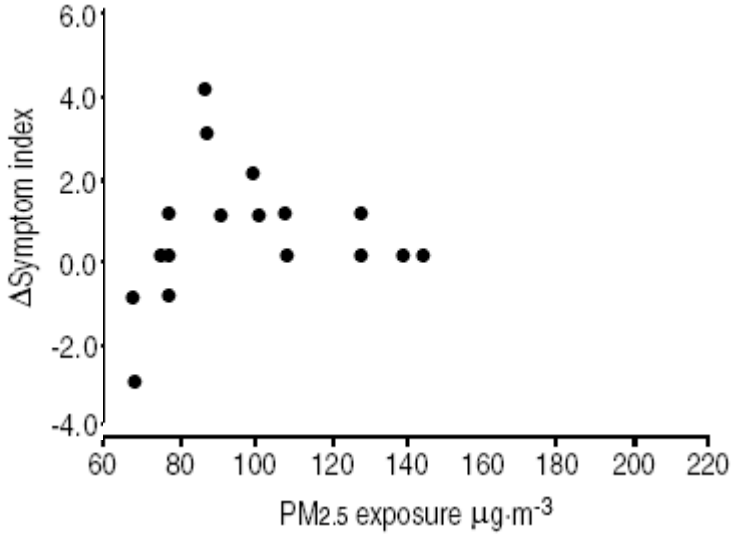

Figure 11: Effect of exposure to $PM_{2.5}$ for 30 minutes within the car in a road tunnel followed by allergen challenge on asthma symptoms index during the late phase. Changes (Δ) in symptom index during the evening, night and morning after allergen inhalation. Data are expressed as difference from the values obtained on the control day, which was exposure to air followed by allergen challenge. Negative values correspond to a greater effect on the control day compared to tunnel exposure. There was no significant relationship between individual differences between tunnel and control as a function of $PM_{2.5}$ exposure within the car (r^2=0.32, p=0.18). From Svartengren *et al*. (2000)[53].

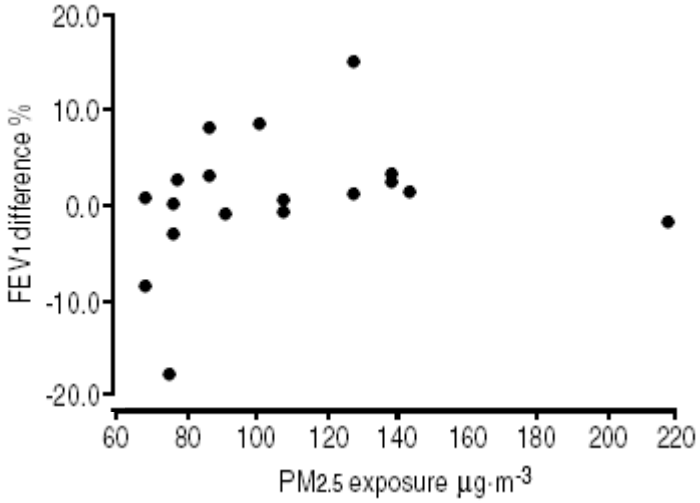

Figure 12: Asthmatic reaction during the late phase. Maximal late phase change in forced expiratory volume in one second (FEV_1). Data are expressed as the difference from the values obtained on the control day. Negative values correspond to a greater effect on the control day compared to tunnel exposure. There was no significant relationship between individual differences between tunnel and control as a function of $PM_{2.5}$ exposure within the car (r^2=0.18, p=0.46). From Svartengren *et al*. (2000)[53].

Riediker et al. (2004)[63] reported on a study of occupationally exposed young, healthy, nonsmoking, male North Carolina highway patrol troopers. Nine troopers (age 23 to 30) were monitored on 4 successive days while working a 3 pm to midnight shift. Each patrol car was equipped with air-quality monitors. Blood was drawn 14 hours after each shift, and ambulatory monitors recorded the electrocardiogram throughout the shift and until the next morning. The average in vehicle pollutant concentrations were: of $PM_{2.5}$ 24 $\mu g/m^3$ (range 4.5-54.4); CO 2.6 ppm (range 0.9-5.9) and NO_2 0.035 ppm (0.002-0.213). There were significant associations for $PM_{2.5}$, but not CO or NO_2 with inflammatory responses measured in blood and increased heart rate variability. Decreased lymphocytes (-11% per 10 $\mu g/m^3$ increase in $PM_{2.5}$) and increased red blood cell indices (1% mean corpuscular volume), neutrophils (6%), C-reactive protein (32%), von Willebrand factor (12%), next-morning heart beat cycle length (6%), next-morning heart rate variability parameters, and ectopic beats throughout the recording (20%). This small and recent study would suggest that increased exposure to $PM_{2.5}$ during a 9- hour shift does result in changes in inflammation, coagulation and cardiac rhythm.

Panel studies of PM

PM and symptoms in Australia

Jalaludin et al (2000 and 2004)[64, 65] found no association between PM_{10} and lung function, wheeze, cough or medication use, but a significant association with doctor visits for asthma. A 12 $\mu g/m^3$ increase in 24 hour PM_{10} was associated with an 11% increase in visits to the doctor for asthma, which was statistically significant ($p<0.05$) [64]. The study followed 125 children living in southwestern Sydney, aged 10, with a history of wheeze for an 11 month period in 1994 . A 0.0082 ppm increase in average daytime NO_2 was associated with an 5% increase in parent reported wet cough, which was statistically significant ($p<0.05$) [64].

PM and symptoms in Europe

In panel studies of children there were significant associations between daily PM_{10} and both lower respiratory symptoms and peak expiratory flow rate, but not cough, upper respiratory symptoms or medication use (Table 13).

Table 13: Odds ratios, beta for PEFR, and (95% confidence intervals) estimates from meta-analyses for PM_{10} and respiratory function in children. Relative risks are for a 10$\mu g/m^3$ increase in PM_{10}.

Respiratory measure	Estimate (95% CI), # of studies
Peak expiratory flow rate	***-0.085 (-0.136 - -0.033), 41 studies***
Cough	0.999 (0.987-1.011), 34 studies
Lower respiratory symptoms	***1.008(1.000-1.016), 39 studies***
Upper respiratory symptoms	0.997 (0.994-0.999), 39 studies
Medication use in symptomatic children	1.005 (0.981-1.029), 31 studies

Particle size and symptoms

Osunyana *et al* (2002)[66] followed 44 people with COPD over a 3 month period. Significant associations were found with lung function and symptoms and both the

concentration of PM_{10} and the number ultrafine particles (Figure 13). Both measures of particles were significantly associated with lung function measures, however there was no significant difference between PM_{10} and ultrafine particles. Other meta-analyses of PM_{10} versus $PM_{2.5}$ have found that there were too few studies where $PM_{2.5}$ was measured to provide reliable estimates[67].

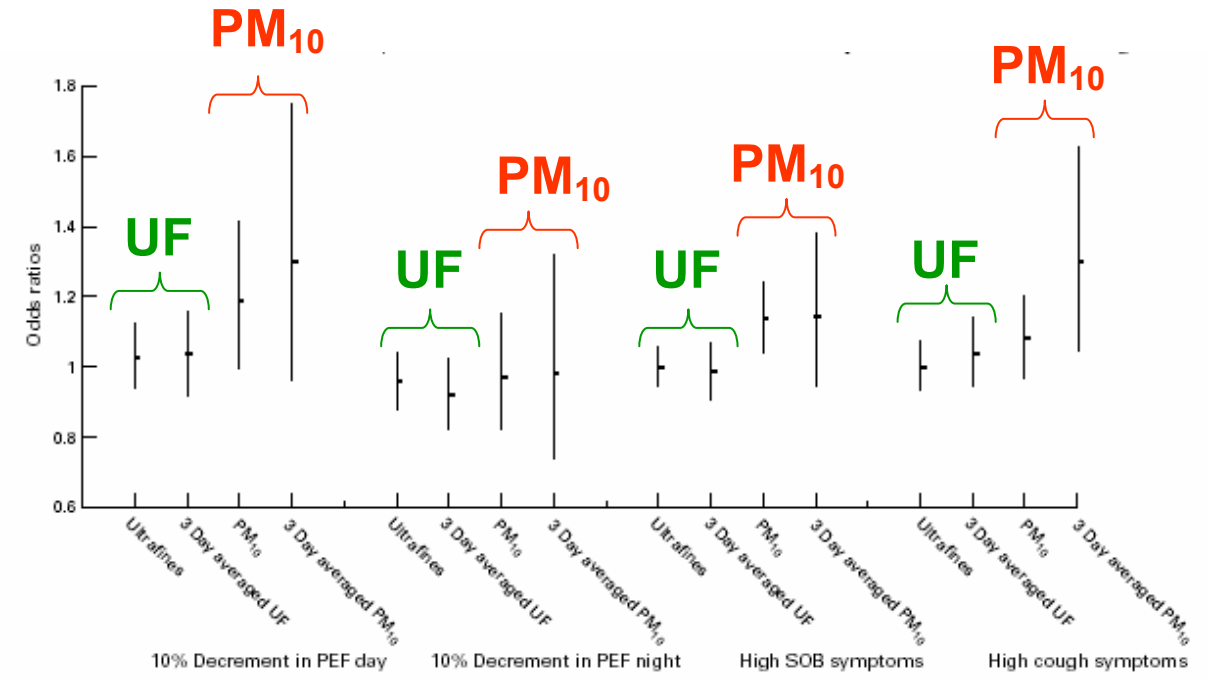

Figure 13: Effect of PM on peak flow in people with COPD. PM was measured as PM_{10} in $\mu g/m^3$ and as the number of ultrafine particles (PM lees than 0.1 μm). Source: Osunyana et al (2002)[66].

Hospital Admissions and Air Pollution

Time series studies examine associations between short term community health outcomes and fluctuations in ambient pollutants using regression models. In these studies the pollutant level is averaged over 1 to a few days and linked to a community health outcome, such as hospital admissions or mortality. In assessing the impact of pollutants on the community, it is important to recognise that communities appear to differ considerably in their response to an elevated level of pollutant. The source of this variation is unknown. It may be related to climate, geography, source of pollutants, background level of pollutant, pollutant mixes and physiological differences in the population. It is, therefore, likely that the studies that are of most relevance to assessing the health impact of changes to air pollutants in Melbourne are likely to be studies that have been performed in Melbourne and/or other Australian cities.

Dose Response Relationships for Australia

Five Australian studies have examined the acute effects of ambient pollutants on hospital admissions[68-72], while two of these studies considered Brisbane. The five

studies were published between 1997 and 2005 and the dose response relationships for each city are summarised in Tables 14-16.

The most recent study was a meta-analysis of results for Brisbane, Melbourne, Sydney and Perth[72]. The study extended from 1 January 1996 to 31 December 1999 for all four cities. Daily hospital admissions in each city and the concentration of ambient particles (light-scattering by nephelometry, bsp $10^{-4}.m^{-1}$, which is an indicator of concentrations of fine particles <2μm in diameter) nitrogen dioxide (ppb) and ozone (ppb) were recorded (Table A1, Appendix A). There were no particle matter data common to all cities in this study apart from the nephelometer data (bsp), and the relationships between bsp data and $PM_{2.5}$ or PM_{10} data are not simple. However, there were datasets for PM_{10} for Brisbane, Sydney and Melbourne and $PM_{2.5}$ datasets for Sydney, Perth and Melbourne (Table A1, Appendix A). The air pollutant data values for all pollutant variables were calculated from data provided through a network of sites across each city. Sites were selected in consultation with the relevant State environmental protection agencies to ensure that they were indicative of the region's daily ambient air quality. In determining the health effects of ambient pollutants across these Australian cities daily 24-hour averages were used for bsp, PM_{10} and $PM_{2.5}$, daily one-hour maxima for NO_2, while daily four-hour and one-hour maxima were used for O_3. The total population examined for air pollution exposure across all cities was approximately 10 million (53% of the Australian population).

The hospital admissions for each category and for each city are given in Tables A1 and A2 (Appendix A). Cardiovascular admissions were the most common form of admission reported and ranged from a daily average of 14.1 in Brisbane to 84 in Melbourne. Asthma admissions accounted for about one third of all respiratory hospitalisations. Children 0-14 years old are the most likely group in the community to be hospitalised for asthma and account for between 2.5 and 15.5 hospital admissions per day across the four cities.

CO

For carbon monoxide the air quality data from fixed-site monitoring stations reflects poorly on short-term exposures of various urban population groups, but appears to reflect better on longer averaging times, such as 8 hours [17]. CO levels are also often highly correlated with PM and NO_2, which makes it difficult to assess the effect of CO alone. In multi-pollutant models the effect of PM and NO_2 are often much greater than CO [4, 17].

Australian Studies

Of the five Australian studies on hospital admissions only two reported the impact of CO. In the meta-analysis of all four cities and the individual studies for Sydney and Brisbane, associations between CO and hospital admission were not examined[70, 72, 73].

Melbourne

In Melbourne, between 1994-1997, the mean ambient 8 hour CO was 0.92 ppm, (range 0.1– 5.68 ppm). The mean 1 hour CO was 1.51 ppm (range 0.17– 9.33 ppm) (Table A3, Appendix A).

A number of significant associations were found between ambient CO and daily hospital admissions (Table A4, Appendix A). The effect for one hour CO are summarised in Table 14.

Table 14: The relationship between a 1 ppm increase in ambient 1 hour CO and hospital admissions in Melbourne between 1994-1997. From Denison *et al*. (2001)[68]. 1ppm CO = 1.16 mg/m^3.

Type of admission	Size of effect (%)	Lag period	Lower estimate (%)	Upper estimates (%)
admissions respiratory 15-64	1.95%	3-day ave.	0.5	3.42
admissions respiratory 65+	2.10%	5-day ave.	0.59	3.63
admissions respiratory all ages	1.01%	5-day ave.	0.1	1.92
admissions asthma all	3.98%	5-day ave	2.22	5.77
admissions asthma 0-14 years	3.10%	3-day ave	1.00	5.24
admissions cardiovascular 0-64	1.18%	Same day	0.21	2.15
admissions cardiovascular 65+	2.05%	3-day ave	1.13	2.97
admissions cardiovascular all	1.73%	3-day ave	0.98	2.50
admissions ischemic heart dis. all	2.27%	3-day ave	1.07	3.48

Perth

In Perth between 1992-1997, there was no association observed between changes in daily CO concentrations and respiratory, asthma, COPD pneumonia or cardiovascular disease admissions [71]. Average 8 hour maximum CO concentration was 2.3 ppm (SD 1.3).

Overseas Studies

A number of studies have reported an association between CO and hospital admissions for cardiovascular diseases. Schwartz (1999)[74] found that a 1.75 ppm increase in ambient daily 1-hour maximum CO was associated with a 2.79% increase in cardiovascular admissions across 8 US cities between 1988 and 1990. This followed an earlier report of an 2.79% increase in cardiovascular hospital admissions for a 1.66 ppm increase in ambient daily 1-hour maximum CO in the city of Tucson, Arizona between 1988-1990 [75]. In contrast, in an earlier study Schwartz and Morris (1995) [76] reported that ambient daily 1-hour maximum CO was not associated with cardiovascular disease admissions in people aged 65 and over in Detroit, Michigan over the period 1986-1989.

Sheppard et al (1999) [77] reported that a 0.9 ppm increase in average daily ambient CO resulted in a 6% increase in hospital admissions for asthma, in people less than 65 years old living in Seattle, Washington over the period 1987-1994. The impact of CO on asthma admissions was supported by Lin et al. (2003)[78] who found a significant association between ambient daily CO and asthma admission in boys, but not girls, aged 6-12 years in Toronto between 1981 and 1993. There were 4629 boys and 2690 girls admitted for asthma over the study period. For boys a 0.5 ppm increase in daily CO increased the risk of hospital admission by up to 8%. Sunyer et al. (1991) [79] examined emergency room admissions for COPD in Barcelona during 1985-1986. A 1 mg/m^3 increase in one hour maximum CO was associated with a 0.11% increase in COPD admissions.

Nitrogen dioxide

Dose Response Relationships for Australia

The relationship between ambient NO_2 and hospital admissions has been examined in all five Australian studies. Ambient NO_2 levels are given in Tables A1 and A5 (Appendix A). Average 1-hour maximum ambient NO_2 levels measured during Australian studies were from 0-156 ppb. Average 24-hour ambient NO_2 levels were from 0-52 ppb for all year (Table A5, Appendix A).

Meta-analysis of Australian Cities.

The meta-analysis of all four Australian cities indicated significant effects of increases in NO_2 on cardiac and respiratory hospital admission (Table 15). A 1 ppb increase in the ambient 1 hour NO_2 concentration was associated with an increase in the daily number of cardiac admissions for all ages by 0.23% and respiratory admissions in people 65 years and older also increased by 0.27% (Table 15).

There were strong correlations between ambient PM and NO_2 and therefore there is uncertainty regarding whether the effect is as a result of NO_2 alone or in combination with PM. In multi-pollutant models, which included PM into the model, there were reduced effects on both cardiac and respiratory admissions, however they remained significant.

Table 15: Dose response relationships for increases in hospital admissions as a result of a 1 ppb increase in 1 hour maximum NO_2. 1ppb NO_2= 2.03 μg/m3.

City	Hospital admission	Size of effect (% increase)	Lag period	Lower estimate (% increase)	Upper estimate (% increase)
All 4 cities for 1 hour maximum NO_2 [72]	Cardiovascular all ages	0.23%	0-1 day	0.16%	0.30%
	Cardiovascular 65+	0.30%	0-1 day	0.22%	0.39%
	Ischemic heart dis. all	0.20%	0-1 day	0.10%	0.29%
	Ischemic heart dis. 65+	0.22%	0-1 day	0.11%	0.34%
	Respiratory 65+	0.27%	0-1 day	0.15%0.39	%
	Asthma &COPD 65+	0.20%	0-1 day	0.03%	0.37%
	Pneumonia & acute bronchitis 65+	0.30%	0-1 day	0.11%	0.48%
Brisbane[80]	Respiratory all ages	-0.11%	0-5 days	-0.23%	0.2%
	Cardiovascular disease all ages	-0.13%	0-5 days	-0.24	-0.2
Melbourne [68]	Respiratory all ages	0.43%	0-5 days	0.29%	0.57%
	Asthma all ages	0.59%	0-5 days	0.32%	0.87%
	Cardiovascular disease all ages	0.17%	0-5 days	0.07%	0.27%
Sydney[73]	Cardiovascular disease all ages	0.21%	0-5 days	0.13%	0.30%
	Asthma 1-14 years	0.18%	0-5 days	0.04%	0.33%
Perth[71]	Respiratory all ages	No effect	N/A	-	-
	Cardiovascular disease 65+	0.16%	N/A	0.01%	0.31%

Brisbane

There were no significant associations between ambient nitrogen dioxide and asthma, respiratory or cardiovascular admissions in any age group or for all age groups combined in single or multi-pollutant models [70] This indicates that ambient increases in 1-hr maximum nitrogen dioxide levels within the range of 0.004 to 0.156 ppm were not associated with increases in hospital admissions in Brisbane between 1987 and 1994 (Table 15 & Table A5, Appendix A).

Melbourne

In Melbourne respiratory, asthma, cardiovascular and ischemic heart disease admissions were all significantly related to ambient NO_2 (Table 15 &Table A5, Appendix A) [68]. These associations were found across almost all age groups (Table 15 & Table A4, Appendix A). The magnitude of the effect was a 0.1% -0.6% increase in hospital admissions associated with a 1 ppb (2.03$\mu g/m^3$) increase in NO_2. Using the results for all asthma admissions, a 1 ppb (2.03$\mu g/m^3$) increase in maximum 1 hour NO_2 would result in a 0.59 % increase in daily admissions to hospital for asthma. The daily hospital admission rate for asthma in Melbourne, over the same time period, was 18.5 persons, thus a 2.03 ug/m3 would result in an increase of 0.59% multiplied by 18.5, which equals 0.1 persons per day when the ambient 1 hour maximum NO_2 increased by 1 ppb.

Including PM into the model resulted in a loss of significance for most of the associations, with the exception of respiratory admissions (65+ years and cardiovascular admissions in all ages).

Sydney

In Sydney, an association between hospital admissions and ambient NO_2 has been reported. Morgan et al. (1998) [73] found an increase in the daily maximum 1-hour nitrogen dioxide concentration from 0.015 to 0.044 ppm (0.029 ppm) was associated with a 5.29% increase in asthma admissions in 1-14 year olds (Table 15 & Table A5, Appendix A). Likewise the same increase in NO_2 was associated with a 6.08% increase in heart disease admissions for all ages, 6.71% increase in the elderly (65+) and 4.79% increase in 0-64 year olds (Table 15 & Table A5, Appendix A). In multi-pollutant models these associations remained significant. This study indicated that during 1990-1994 increases in the level of ambient NO_2 from 0.015-0.044 ppm were significantly associated with increases in hospital admissions for asthma in children and heart diseases in the elderly.

Perth

In Perth cardiovascular and respiratory hospitalisations, especially in people 65+ years were reported to be significantly associated with elevated ambient NO_2 (Table 15 & Table A5, Appendix A)[71].

Overseas studies

The WHO (2003) [4] recommends against the use of regression coefficients for NO_2 for quantitative assessment of the risk from exposure to elevated levels of NO_2. This recommendation is based on the uncertainty associated with the health impact of

NO_2, given the complex relationship between NO_2, motor vehicle pollution, NOx, ozone, secondary air pollutants and particles. Since NO_2 is likely to be a marker for these pollutants, estimates will be provided in this report.

PM Dose Response Relationships for Australia

Ambient PM levels are given in Tables A1 and A7 (Appendix A). 1 hour maximum bsp was from 0.01-16.2 $bsp10^{-4}m^{-1}$, while 24 hour PM was from 0.01-5.1$bsp10^{-4}m^{-1}$(Tables A1 and A7, Appendix A). Sydney had the highest levels of PM, followed by Brisbane, Melbourne and Perth (Table A1 and A7, Appendix A).

Meta-analysis of Australian Cities.

The meta-analysis of all four Australian cities indicated strong effects of increases in bsp on cardiac and respiratory hospital admissions (Table 16 & Table A8 Appendix A). For the meta-analyses there were no particle matter data common to all cities apart from the bsp data. However, PM_{10} datasets were available for Brisbane, Sydney and Melbourne and $PM_{2.5}$ datasets were available for Sydney, Perth, and Melbourne. A meta-analysis on these sets of three cities estimated that for a 10µg/m^3 increase in PM_{10} concentration the increase in the daily number of cardiac admissions for all ages was 2.4% (1.5-3.4%) and the increase in elderly respiratory admissions also increased by 2.9% (1.3-4.4%). Also there was an increase of 5.1% (3.5-6.7%) in cardiac admissions for a 10µg/m^3 increase in $PM_{2.5}$ concentration (Table 16).

Table 16: Dose response relationships for increases in hospital admissions as a result of a 1 unit increase in bsp $10^{-4}m^{-1}$ or a 10ug/m^3 increase in 24 hour PM_{10} or $PM_{2.5}$.

City	Hospital admission	Size of effect (% increase)	Lower estimate (% increase)	Upper estimate (% increase)
All 4 cities for bsp[72]	Cardiac all ages	8.56%	6.03%	11.16%
	Respiratory 65+ years	5.52%	0.82%	10.45%
	Asthma 15-64 years	8.93%	2.4%	15.87%
All 3 cities for PM_{10}[72]	Cardiac all ages	2.4%	1.5%	3.4%
	Respiratory 65+ years	2.9%	1.3%	4.4%
All 3 cities for $PM_{2.5}$[72]	Cardiac all ages	5.1%	3.5%	6.7%
Brisbane[80]	Respiratory all ages	1.8%	0.72%	2.77%
	Cardiovascular disease all ages	No effect		
Melbourne[68]	Respiratory 65+ years	2.42%	0.14%	4.77%
	Asthma all ages	4.45%	1.90%	7.05%
	Cardiovascular disease 65+ years	1.83%	0.69%	2.99%
Sydney[73]	Cardiovascular disease 65+ years	0.97%	0.31	1.63
	Respiratory all ages	No effect		
Perth[71]	Respiratory all ages	?	?	?
	Cardiovascular disease all ages	?	?	?

Brisbane

In Brisbane, a 10 μg/m3 increase in 24 hour PM was associated with a significant 1.8% increase in all respiratory (but not asthma only or cardiovascular) admissions in single- and multi-pollutant models (Table 16 and Table A8, Appendix A) [70] .

For respiratory admissions in all ages (0-65+) a unit increase (1 x 10^{-5}/m) in 24 hr average concentration of bsp (5 day average) resulted in a 1.5% increase in hospital admission that was statistically significant ($p<0.05$). Within the 15-64 year old age group, a unit increase (1 x 10^{-5}/m) in maximum 1-hour bsp (5 day average) resulted in a statistically significant 0.5% increase in hospital admissions for respiratory conditions. There was a trend toward increased respiratory admissions in 0-4 and 65+ age groups, although this was not statistically significant (Table A8, Appendix A).

The effect was stronger for 24 hour bsp compared with 1 hour maximum bsp and in multi-pollutant models, after adjusting for high SO_2 and high ozone, respiratory admissions in all ages remained significant [70]. This study indicates that in Brisbane a 1 x 10^{-5}/m increase in 24 hour average bsp concentration across the range 0.30 – 50.8 x 10^{-5}/m resulted in a 1.5% increase in hospital admissions between 1987 and 1994 (Table A8, Appendix A).

For asthma only admissions, a subset of respiratory admissions, there were no significant increases in admissions in any of the age groups (Table A8, Appendix A).

For cardiovascular admissions, the most common reason for admission in the study (Table A8, Appendix A), there were no significant increases in admissions in any of the ages groups.

Melbourne

The effects of increased PM in Melbourne are summarised in Table 16. A 10 μg/m^3 increase in 24 hour PM_{10} resulted in statistically significant increases of 2.42%, 4.45% and 1.83% in respiratory admissions in people aged 65 years, asthma admissions in all ages and cardiovascular disease in people aged 65 years. All these effects were statistically significant ($p<0.05$). The summary estimates (Table 16) are based on more extensive analyses which are presented in Table A8 (Appendix A).

Table A8 (Appendix A) also indicates fine particles were significantly ($p<0.05$) associated with admissions for respiratory disease (15-64 years, 65+ years), asthma (0-14 years, all ages), cardiovascular disease (65+ years, all ages) and ischaemic heart disease (all ages).

For respiratory admissions in the 15-64 age group a unit ($1x10^{-4}m^{-1}$) increase in 1-hour bsp (equivalent to 15 μg/m^3 $PM_{2.5}$) was associated with a 3.8% increase in risk of admission (3-day lag). In the 65+ age group, a one unit increase in the 24- hour (equivalent to ~ 4 x SD) and maximum 1-hour (equivalent to ~ 2 x SD) concentrations of bsp was associated with a 7.5% and 4.3%, respectively, increase in risk of admission. No significant associations were found for respiratory admissions in the 0-14 years or all age groups.

For asthma, strong associations were observed across most of the lag periods and averaging times examined in both the 0-14 years and all ages groups. In the 0-14 years age group, a unit increase in same day 24-hour bsp was associated with a 14.8% increase in risk of admission (Table A8, Appendix A). In the all ages category the strongest associations were found with the 5-day cumulative average, where a unit increase in the 24-hour concentration was associated with a 13.9% increase in risk of admission for asthma.

Admissions for cardiovascular disease and ischaemic heart disease also showed strong, consistent associations with particles in Melbourne, in particular the 24-hour concentration (Table A8, Appendix A). While only marginally significant associations were found in the 0-64 years age group, admissions for cardiovascular disease in the 65+ years and all ages groups were consistently associated with bsp levels for both 1-hour maximum and 24-hour average. A unit increase in the 24-hour bsp concentration was associated with a 5.6% and 4.6% increase in risk of admission for cardiovascular disease in the 65+ years and all ages groups, respectively, per unit increase (equivalent to ~ 4 x SD) in bsp. For the maximum 1-hour particle concentration a 3.5% and 2.7% increase in risk of admission per unit increase in bsp was found for cardiovascular disease in the 65+ years age group and all ages group respectively. Similar results were obtained for ischaemic heart disease (Table A8, Appendix A).

The results of the multi-pollutant analysis showed that many of the significant associations observed between hospital admissions and fine particles in the single pollutant analysis were reduced in size and significance after controlling for the effects of other pollutants, in particular NO_2 and CO. Controlling for NO_2 in the model resulted in a reduction in the size and significance of the association between bsp and admissions for each of the outcomes. The one exception was cardiovascular admissions in the all ages group where the significance of the particle effect was retained after controlling for NO_2 (Table A8, Appendix A).

Sydney

For Sydney, a 10 $\mu g/m^3$ increase in 24 hour PM_{10} resulted in a statistically significant increase of 0.97% in cardiovascular disease in people aged 65 years and over. The lower estimate was a 0.31% increase while the upper estimate was a 1.63% increase (Table 16). The lower and upper estimates are the 95% confidence intervals. There was no effect on respiratory admissions.

An increase in 1 hour maximum particulates from 0.25 to 1.48 bscat/10^4m (~15 - 90 $\mu g/m^3$ PM_{10}) was associated with a 2.72% increase in admissions (Table A8, Appendix A). The results for 24-hour average particulates were similar (Table A8, Appendix A), with a 2.82% increase in heart disease admissions associated with an increase in particulates from 0.12-0.60 bscat/10^4m (~7 - 36 $\mu g/m^3$ PM_{10}). COPD admissions also showed a trend towards increase with particulate levels, however it was not statistically significant.

Perth

Respiratory hospitalisations in people over 65+ and especially for COPD were significantly related to the level of PM (Table A8, Appendix A). In this study a case

cross over analysis was used and the effects were large. The effects for 24 hour PM were not reported, however.

Daily Hospital Admissions and PM_{10} in the US

The WHO[4] recently provided summary estimates of the effect of particulate pollution on hospitalisations for respiratory diseases (Table 17) in the US. A 10 $\mu g/m^3$ increase in 24 hour PM_{10} was associated with a 1.5% increase in hospital admissions for COPD. The estimate was taken from the NMMAPS studies of hospital admissions which covered 10 large metropolitan areas in the United States of America with a combined population of 1,843,000 subjects over 65 years old.

Table 17: Estimated effects of air pollution on daily hospital admissions from the APHEA2 and NMMAPS. Source: WHO (2003)[4].

STUDY	Disease	Increase in disease admission per 10$\mu g/m^3$ increase in PM_{10}.
APHEA2	COPD and asthma	1.0% (0.4 – 1.5%)
Europe	Respiratory aged 65+	0.7% (0.2-1.3), 8 studies
NMMAPS	COPD	1.5% (1.0 – 1.9%)

Daily Hospital Admissions and PM_{10} in Europe

The WHO also reported on the APHEA2 hospital admission study covering a population of 38 million living in 8 European cities, which where studied for 3 to 9 years in the early-mid 1990s.

The Europe estimate (Table 17) is based on a later meta-analysis performed by the WHO that includes the APHEA2 studies [67]. The WHO recently performed a meta-analyses of time series and panel studies[67]. Using studies catalogued in bibliographic databases up to February 2003, 629 ecological time series studies and 160 individual or panel studies have been identified. 286 time-series and 124 panel studies have provided usable data. The two databases contain over 11 700 and 6400 effect estimates, respectively. In the WHO's meta-analysis for Europe, sufficient numbers of estimates (>3) of the effect of PM_{10} were available only for respiratory admissions in the 65+ age group. The relative risk for a 10$\mu g/m^3$ increase in PM_{10} was 1.007 (1.002, 1.013) and was based upon 8 studies. Six of these eight estimates were provided by the APHEA 2 project[81]. Unfortunately much of the recently published data on particles and daily admissions for respiratory disease from APHEA 2 could not be used because this study did not report all respiratory admissions in the younger age groups.

According to the WHO there were insufficient numbers of studies for other age groups or cardiovascular admissions[67]. For the age categories, ages 0–14 and 15–64 years, results were available from three studies conducted in London[82], West Midlands[83] and Rome[84]. Together these cities represent a population in excess of 10 million people. A meta-analysis of results from these three cities gave summary estimates of 1.010 (0.998, 1.021) and 1.008 (1.001, 1.015) per 10 $\mu g/m^3$ increases in PM10 for respiratory admissions, ages 0–14 and 15–64 years respectively[67].

Daily Hospital Admissions and $PM_{2.5}$ in Europe

The WHO reported that few studies recorded $PM_{2.5}$ levels[67]. For fine and coarse particles only, one study provided results for respiratory outcomes. The relative risks for $PM_{2.5}$ for each of the three age categories, 0–14, 15–64 and 65+ years were 1.091 (0.9994, 1.0391), 0.9881 (0.9633, 1.0135) and 0.9926 (0.9732, 1.0125)

respectively. There were no estimates available from the 65+ years, cardiovascular admissions group. Results for coarse particles were similar to those for fine particles.

Mortality

Dose Response Relationships for Australia
Five Australian studies have examined the relationship between air pollution and mortality. The studies were published between1997-2005 and data were collected for periods of 4-6 years between 1987 and 1999. The dose response relationships are summarised in Tables 18-19 while details are provided in Tables B1- B6 (Appendix B).

The most recent study was a meta-analysis of results for Brisbane, Melbourne, Sydney and Perth[85]. The study extended from 1 January 1996 to 31 December 1999 for all four cities. Daily mortality in each city and the concentration of ambient particles (light-scattering by nephelometry, bsp $10^{-4}.m^{-1}$, which is an indicator of concentrations of fine particles <2μm in diameter) nitrogen dioxide (ppb) and ozone (ppb) were recorded (Table B1, Appendix B). There were no particle matter data common to all cities in this study apart from the nephelometer data (bsp), and the relationships between bsp data and $PM_{2.5}$ or PM_{10} data are not simple. However, there were datasets for PM_{10} for Brisbane, Sydney and Melbourne and $PM_{2.5}$ datasets for Sydney, Perth and Melbourne (Table B1, Appendix B). The air pollutant data values for all pollutant variables were calculated from data provided through a network of sites across each city. Sites were selected in consultation with the relevant State environmental protection agencies to ensure that they were indicative of the region's daily ambient air quality. In determining the health effects of ambient pollutants across these Australian cities daily 24-hour averages were used for bsp, PM_{10} and $PM_{2.5}$, daily one-hour maxima for NO_2, while daily four-hour and one-hour maxima were used for O_3. The total population examined for air pollution exposure across all cities was approximately 10 million (53% of the Australian population).

In the meta-analysis (Table B1, Appendix B) and the earlier studies (Table B2, Appendix B) cardiovascular mortality was on average 5 times more common than respiratory mortality (Tables 18 and 19). Brisbane had the lowest daily mortalities, followed by Perth, Melbourne and Sydney. On each day an average of 5 people died of respiratory illness in Melbourne or Sydney and 23-29 died from cardiovascular diseases (Tables 18 and 19).

CO

Australian Studies

Of the five Australian studies that have examined the relationship between ambient air pollution and mortality only two have reported an association between CO and mortality.

The associations between CO and mortality were not reported in the recent meta-analysis[85] and were not examined in earlier studies for the individual cities of Sydney or Brisbane[73, 80].

Melbourne

There were 0.7% and 1.93% increases in all causes of mortality associated with a 1 ppm 91.16 mg/m^3) increase in 1 and 8 hour maximum CO, respectively. These results were based on a study from 1991-1996, where the mean ambient 1-hour CO was 1.56 ppm (range 0.1-9.4 ppm) and the mean 8- hour CO level was 0.95 ppm (range 0- 5.7 ppm)[86].

Perth

In Perth between 1992 and 1997, the average 8 hour maximum CO concentration was 2.3 ppm (SD 1.3). No associations were observed between changes in 8 hour CO concentrations and cardiovascular, respiratory or all mortality. There was, however, an unusual finding of significant association with other mortality (not cardiovascular, respiratory, accidental, poisonings or violence) where a 1 ppm increase in CO was associated with a 1.9% increase in other mortality [71].

Overseas Studies

Samet et al. (2000)[87] reported that ambient CO was not associated with mortality in an analysis of 20 cities across the US that included a population of more than 50 million people, between 1987-1994. Sunyer and Basagana (2001) [88] found no association of ambient 8-hour CO and mortality for COPD in Barcelona. However in a meta-analysis of 22 studies, Stieb et al. (2002) [89] reported an overall effect of 1.7% increase per 1.1 ppm increase in ambient 24 hour CO (Figure 14). This is similar to the effect size reported in the Melbourne study

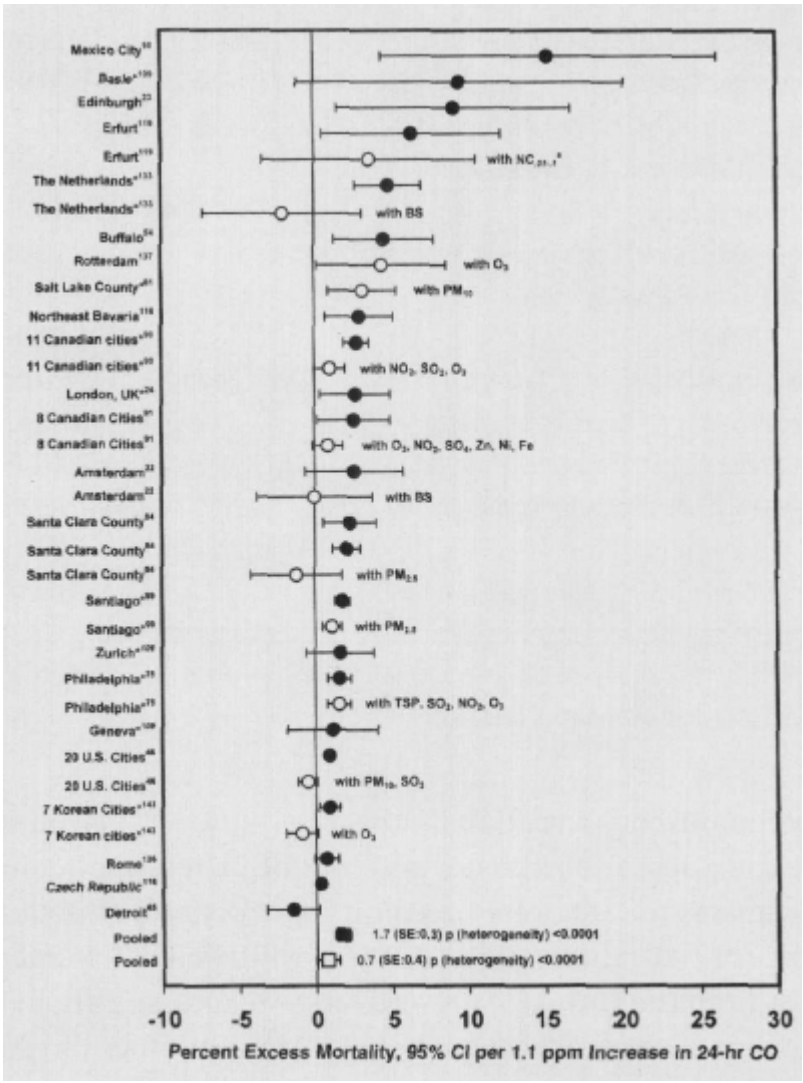

Figure 14: The effect of a 1.1 ppm increase in 24-hour ambient CO on mortality. From Stieb et al. (2002) [89].

NO_2

Dose Response Relationships for Australia

The relationship between ambient NO_2 and mortality has been examined in all five Australian studies. Ambient NO_2 levels are given in Tables B1 and B3 (Appendix A). Average 1-hour maximum ambient nitrogen dioxide levels were from 1 to 104 ppb for all year. 24-hour average ambient nitrogen dioxide levels were from 1-42 ppb (Tables B1 & B3, Appendix B).

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Meta-analysis of Australian Cities.

The meta-analysis of all four Australian cities indicated significant effects of increases in NO_2 on total, cardiovascular and respiratory mortality in people over 65 years old (Table 18). A 1 ppb increase in the ambient 1 hour NO_2 concentration was associated with an increase in the daily number of non-accidental deaths, respiratory

and cardiovascular mortality, for all ages, by 0.12%, 0.38% and 0.16%, respectively (Table 18).

There were strong correlations between ambient PM and NO_2 and therefore there is uncertainty regarding whether the effect is as a result of NO_2 alone or in combination with PM. In multi-pollutant models, which included PM into the model, it resulted in a reduced effect on both cardiac and respiratory admissions, however they remained significant.

Table 18: Dose response relationships for increases in hospital admissions as a result of a 1 ppb increase in 1 hour maximum NO_2. 1ppb NO_2= 2.03 μg/m3.

City	Hospital admission	Size of effect (% increase)	Lag period	Lower estimate (% increase)	Upper estimate (% increase)
All 4 cities for 1 hour maximum NO_2 [85]	Total mortality all ages	0.12%	1 day	0.06%	0.18%
	Cardiovascular all ages	0.16%	1 day	0.06%	0.25%%
	Respiratory all ages	0.38%	1 day	0.17%	0.58%
	Cardiovascular 65+	0.12%	1 day	0.02%	0.22%
Brisbane[80]	Total mortality all ages	No effect	N/A	-	-
	Cardiovascular all ages	No effect	N/A	-	-
	Respiratory all ages	No effect	N/A	-	-
Melbourne [86, 90]	Total mortality all ages	0.06%	1 day	0.00%	0.12%
	Total mortality 65+	0.14%	5 day ave.	0.04%	0.24%
	Respiratory all ages	No effect	N/A	-	-
	Cardiovascular disease all ages	0.15%	5 day ave.	0.00%	0.29%
	Cardiovascular disease 65+	0.20%	5 day ave.	0.00%	0.36%
Sydney[73]	Total mortality all ages	No effect	N/A	-	-
	Cardiovascular all ages	No effect	N/A	-	-
	Respiratory all ages	No effect	N/A	-	-
Perth[71]	Total mortality all ages	No effect	N/A	-	-
	Cardiovascular all ages	No effect	N/A	-	-
	Respiratory all ages	No effect	N/A	-	-

Brisbane

In Brisbane there were no significant associations between ambient NO_2 levels and total, cardiovascular or respiratory mortality (Table 18 and B4, Appendix B).

Melbourne

In Melbourne total mortality was found to correlate with ambient 1 hour maximum NO_2, while cardiovascular deaths were also related to 1 hour maximum NO_2 when averaged over 5 days, indicating a longer term trend rather than an acute effect. Respiratory deaths (all ages and 65+ years) and total mortality (all ages and 65+ years), but not cardiovascular deaths, were found to correlate with ambient 24 hour NO_2 [86, 90] in single pollutant but not multi-pollutant models (Table 18 and B4, Appendix B).

Sydney

In Sydney there were no significant associations between total mortality and ambient 1 hour maximum nitrogen dioxide concentration (Table 18 and B4, Appendix B) [73]. However, increases in 24 hour NO_2 from 0.012-0.044 ppm resulted in a 2.66%

increase in total mortality, but not cardiovascular or respiratory mortality. This association with total mortality was not significant after adjusting for particulates and ozone.

Perth

There were no significant associations between mortality and NO_2 levels in Perth (Table B4, Appendix B) [71].

Overseas studies

A meta-analysis of the health effect of NO_2 on mortality combined 49 studies, spanning a wide range of NO_2 levels. Overall, an increase in 24 hour NO_2 of 24 ppb was associated with a 2.8% increase in all cause mortality and this effect was statistically significant [89]. An indication of the spread of results across the studies is given in Figure 15.

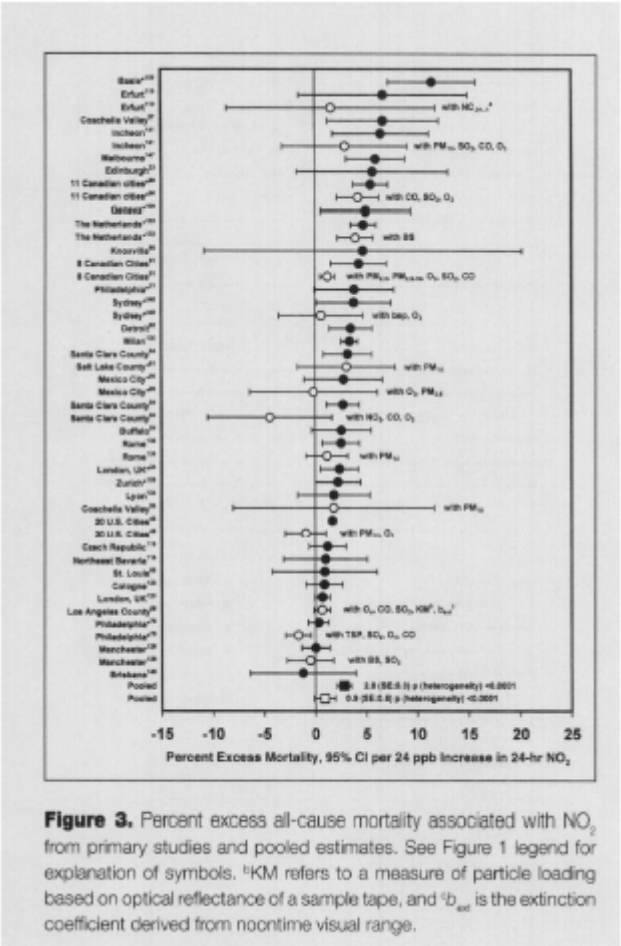

Figure 3. Percent excess all-cause mortality associated with NO_2 from primary studies and pooled estimates. See Figure 1 legend for explanation of symbols. [b]KM refers to a measure of particle loading based on optical reflectance of a sample tape, and [c]b_{ext} is the extinction coefficient derived from noontime visual range.

Figure 15: The association between mortality and ambient NO_2. From Stieb (2003)[89]

PM

Ambient PM levels are given in Tables B1 and B5 (Appendix B). 1 hour maximum bsp was from 0.01-16.2 $bsp10^{-4}m^{-1}$, while 24 hour PM was from 0.01-5.1$bsp10^{-4}m^{-}$

[1](Tables B1 and B5, Appendix B). Sydney recorded the highest levels of PM, followed by Brisbane, Melbourne and Perth (Tables B1 and B5, Appendix B).

Meta-analysis of Australian Cities.
For the meta-analysis there were no particle matter data common to all cities apart from nephelometer data, which is recorded as bsp. When bsp was used as the measure of particulate exposure significant relationships were found for all cause and cardiovascular mortality (Table 19), but not respiratory mortality.

The relationships between bsp and $PM_{2.5}$ or PM_{10} data are not simple[85] . However, there were datasets for PM_{10} for Brisbane, Sydney and Melbourne and $PM_{2.5}$ datasets for Sydney, Perth and Melbourne. The meta-analysis on these three cities estimated the increase in the daily number of deaths for all ages for a 10 µg/m3 increase in PM_{10} concentration to be 0.2% (-0.8% to 1.2%), and estimated the increase in the daily number of deaths for all ages for a 10 µg/m3 increase in $PM_{2.5}$ concentration to be 0.9% (-0.7% to 2.5%). Thus for bsp, but not PM_{10} or $PM_{2.5}$, there were significant associations between mortality and particulate air pollution in the four Australian cities.

Table 19: Dose response relationships for increases in daily mortality as a result of a 1 unit increase in 24 hour bsp $10^{-4}m^{-1}$ or a 10$\mu g/m^3$ increase in 24 hour PM_{10} or $PM_{2.5}$.

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City	Cause of mortality	Size of effect (% increase)	Lower estimate (% increase)	Upper estimate (% increase)
All 4 cities for bsp[85]	All cause	2.84%	0.15%	5.60%
	Cardiovascular	4.79%	0.76%	8.98%
All 3 cities for PM_{10}[85]	All cause	0.2%	-0.8%	1.2%
All 3 cities for $PM_{2.5}$[85]	All cause	0.9%	-0.7%	2.5%
Brisbane[80]	All cause	1.08%	0.36%	1.80%
	Cardiovascular	No effect	-	-
Sydney[73]	All cause	1.09%	0.36%	1.73%
	Cardiovascular	1.11%	0.10%	2.09%
Melbourne[90]	All cause (warm season only)	1.82%	0.70%	3.35%
	Respiratory all ages (warm season only)	6.06%	0.60%	11.9%
	Cardiovascular	No effect		
Perth[71]	All cause	No effect		
	Cardiovascular	No effect		

Brisbane
In Brisbane there were significant associations between ambient particulate levels and all cause mortality (Table 19 and B6, Appendix B). A 10 $\mu g/m^3$ increase in 24 hour PM_{10} resulted in a statistically significant increase of 1.08% in all cause mortality, which excludes accidental mortality. For all cause mortality the lower estimate was a 0.36% increase while the upper estimate was a 1.80% increase (Table 19). This association with all cause mortality was significant in single pollutant models only, while there were no significant effects on cardiovascular mortality or respiratory mortality (Table B6, Appendix B).

The effect of PM was also observed for 1 hour maximum bsp and the effect was strongest for people aged 65 and older. A 1 $x10^{-5/}$m bsp increase in 1 hour daily maximum particulates resulted in a 0.2% increase in total mortality and a 0.4% increase in cardiovascular mortality in Brisbane between 1987 and 1993[80] (Table B6, Appendix B).

Melbourne

There were no significant associations between particulate pollution in Melbourne and mortality for the entire year, however in earlier studies associations in the warm period were found (Table 19 and B6, Appendix B) [90, 91].

Sydney

A 10 $\mu g/m^3$ increase in 24 hour PM_{10} resulted in a statistically significant increase of 1.09% in all cause mortality, which excludes accidental mortality. For all cause mortality the lower estimate was a 0.36% increase while the upper estimate was a 1.73% increase (Table 19). The lower and upper estimates are the 95% confidence intervals. This association with total mortality was significant after adjusting for nitrogen dioxide and ozone. In single pollutant models there was a similar size and significant effect on cardiovascular mortality (Table 19), however no significant effect on respiratory mortality (Table B6, Appendix B).

Results for an increase in 1-hour maximum particulates from 0.23 to 1.42 bscat/10^4m (~14 - 85 $\mu g/m^3$ PM_{10}) were also significant and are given in Table B6 (Appendix B).

Perth

There were no significant associations between mortality and PM levels in Perth (Table 19 and B6, Appendix B) [71].

Overseas studies

There have been a number of summary estimates of the effect of PM on mortality in the US [32], Europe[4] and Asia.

Daily mortality and PM_{10} in Europe

There were significant associations between PM_{10} and mortality in Europe (Table 20). The WHO meta-analysis[67] found that a 10$\mu g/m^3$ increase in 24 hour ambient PM_{10} was associated with significant increases in all-cause (0.6%), respiratory (1.0%) and cardiovascular (0.5%) mortality [67]. These estimates were largely from the Air Pollution and Health European Approach 2 (APHEA2) study. The APHEA2 mortality study covered a population of more than 43 million living in 29 European cities, which were all studied for > 5 years in the early-mid 1990s.

Daily mortality and PM_{10} in the US

A recent reanalysis of a meta-analysis of the National Morbidity and Mortality Air Pollution Study (NMMAPS)[32] reported that a 10 $\mu g/m^3$ increase of PM_{10} was associated with a 0.34% (95% CI, 0.18%, 0.51%) increase in cardiovascular-respiratory mortality and a 0.28% (95% CI, 0.16%, 0.41%) for total mortality (Table 20). The NMMAPS examined the 20 largest metropolitan areas in the US with a population of more than 50 million people over the period 1987–1994.

Table 20: Relative risk estimates (95% confidence intervals) from meta-analyses for PM_{10} and all-cause and cause-specific mortality. Relative risks are for a 10ug/m^3 increase in daily PM_{10}.

Region	Outcome and age	Summary estimate (95% CI), # of studies
Europe[67]	All-cause mortality, all ages	1.006 (1.004-1.008), 33 studies
	Respiratory mortality, all ages	1.010 (1.001-1.018), 20 studies
	Cardiovascular mortality, all ages	1.005 (1.001-1.010), 23 studies
United States[32]	All-cause mortality, all ages	1.003(1.001-1.004), 20 cities
	Cardiovascular-respiratory mortality, all ages	1.003(1.002-1.005), 20 cities

Daily mortality and $PM_{2.5}$

The WHO presented a meta-analysis for the effect of $PM_{2.5}$ on mortality[67] (Table 21). Too few studies have been performed in Europe for analysis, therefore the analysis was extended to include all studies, regardless of geographical location. For all cause mortality, 23 studies from around the World were considered, including 15 from the US and Canada. In the global meta-analysis a 10 μg/m3 increase in 24 hour $PM_{2.5}$ was associated with significant increases in daily: all cause mortality (0.9%); cardiovascular (1.3%); and respiratory mortality (1.1%) (Table 21). The effect estimates were slightly higher in the US and Canada, although not significant for respiratory mortality. Only 1-3 European studies were considered in the analyses and there were no significant effects.

Table 21: Relative risk estimates (95% confidence intervals) from meta-analyses for $PM_{2.5}$ and daily mortality. Relative risks are for a 10μg/m^3 increase in $PM_{2.5}$. Source Anderson *et al*. (2004)[67]

	All Cause (95% CI), # studies	Cardiovascular (95% CI), # studies	Respiratory (95% CI), # studies
US and Canada	1.013 (1.008-1.018), 15 studies	1.023 (1.003-1.044), 4 studies	1.016 (0.994-1.038), 4 studies
Global	1.009 (1.006-1.013), 23 studies	1.013 (1.005-1.022), 8 studies	1.011 (1.002-1.020), 8 studies
Europe	3 studies with no sig effect, RRs = 1.003, 1.006 & 0.98	1.005 (0.998-1.022), 1 study	0.994 (0.969-1.031), 1 study

Variation between Cities

While meta-analyses provide summary estimates for regions, it should be noted that considerable variation exists between cities. Figure 16 shows the individual variation in mortality associated with a 31.3 μg/m^3 increase in $PM_{10,}$ which ranged from –4 to +6%. This meta-analysis of 48 studies found a 2% increase in mortality resulting from a 31.3 μg/m^3 increase in $PM_{10.}$ Furthermore the increased relative risk for a city appears to be independent of the level of ambient PM_{10} (Figure 17)[67].

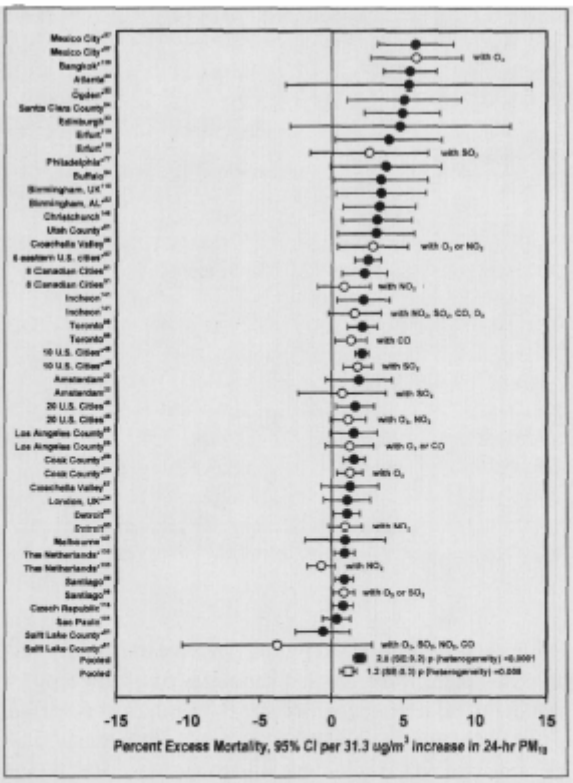

Figure 1. Percent excess all-cause mortality associated with PM_{10} from primary studies and pooled estimates. Point estimates (symbols) and 95% CI (horizontal lines) are shown. Asterisks denote estimates based on multiday average pollution concentrations. Estimates are presented in descending order of single-pollutant effect sizes (filled symbols), which are paired with corresponding estimates (where available) from multipollutant models (open symbols).

Figure 16: The association between mortality and ambient PM_{10}. Source: Stieb (2003)[89]

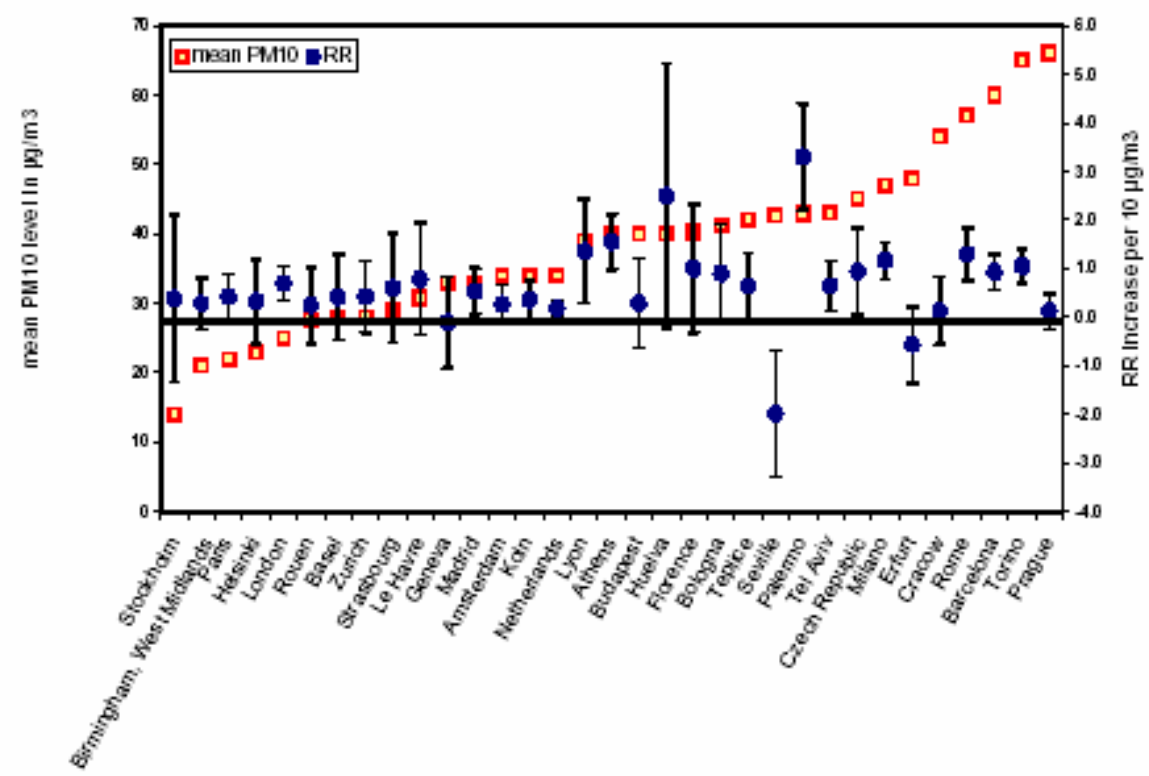

Figure 17: The association relative risk of all cause mortality for a 10µg/m^3 increase in PM_{10} and ambient annual average PM_{10}. Source: Anderson *et al*. (2004)[67].

Long term effects of ambient exposure

Elevated levels of air pollutants impact both acutely and in the longer term, on health. The acute effects are easier to quantify and therefore provide more robust estimates of likely health outcomes. The chronic effects of air pollution are inherently more difficult to interpret than those reporting immediate or short-term effects. There is potential for confounding factors to yield spurious associations between average pollutant exposures and the incidence or prevalence of chronic disease. Some of the repeated associations that have been found are effects on lung function growth in children and adult mortality.

Lung function growth

Southern Californian Children's Health Study

The Southern Californian Children's Health Study (SCCHS) examined lung function growth in children over 8 years, between 1993 and 2001. The study examined lung growth and air pollution levels across each of 12 communities in Southern California. The primary source of pollutants was motor vehicles.

In the first cohort, 3035 children were tested in 1993 and subsequently on several occasions up to 1997. For each of the 12 areas approximately 150 children in grade four, 75 in grade seven and 75 in grade 10 were selected from public schools. Over

the study period, lung function was assessed on at least 2 occasions and on average, 3.8 occasions. Three measures of lung function were made on each occasion and were: forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1), and maximal mid expiratory flow rate (MMEF). Levels of NO_2, ozone, PM_{10}, $PM_{2.5}$ and inorganic acid vapour ($HCl + HNO_3$) were measured.

NO_2

For NO_2 there was an eight-fold difference between the least and most polluted communities in the SCCHS (4.6 – 41.4 ppb annual average of daily (24hr) NO_2). In the fourth grade cohort (1,498 children) annual lung function (FEV_1) growth was from 11 to 12.2%. Lung function growth was greatest in the communities with low NO_2 and lowest in those with high NO_2 and this relationship was statistically significant ($R= -0.61$, $p =0.02$). For the seventh and 10^{th} grade cohorts higher NO_2 resulted in lower lung function growth, however in these cohorts the effect was not significant. They concluded that long-term exposure to NO_2 was associated with reduced lung function growth in children[92]. A 36.8 ppb (0.037 ppm) increase in annual 24 hour NO_2 exposure was associated with an annual decrease of 0.53% in FVC, 0.77% in FEV_1, 1.08% in MMEF and 1.37% in FEF_{75}, which were all significant ($P<0.05$). When lung function growth was examined over a 4 year period those living in the highest NO_2 exposure communities had a lung function growth that was 95-98% of those in low NO_2 communities [92].

In a second cohort of 1,678 fourth grade children these findings were confirmed for NO_2 although they were of a lesser magnitude than for the first cohort [93]. A 0.035 ppm (34.6 ppb) increase in annual 24 hour NO_2 exposure was associated with an annual decrease of 0.23% in FVC, 0.48% in FEV_1 and 1.10% in MMEF which was statistically significant for MMEF ($P<0.05$). The net result of these changes in lung function over an eight year period was that for a 0.035 ppm increase in annual average NO_2 there was a 6% increase in the number of 18 year olds who had a clinically significant lower lung function (FEV_1 less than 80% of predicted) (Figure 18). Lung function growth was significantly lower in children who spent more time outdoors in the afternoon compared to those who spent less time outdoors. Thus the impact of NO_2 was dependent upon the level of NO_2 and the amount of time spent outdoors. The WHO[4] review of these studies it concluded that the effect measured could not be attributed to NO2 exposure per se, since the relative contribution of particulate matter and NO2 on the health outcomes described could not be separated.

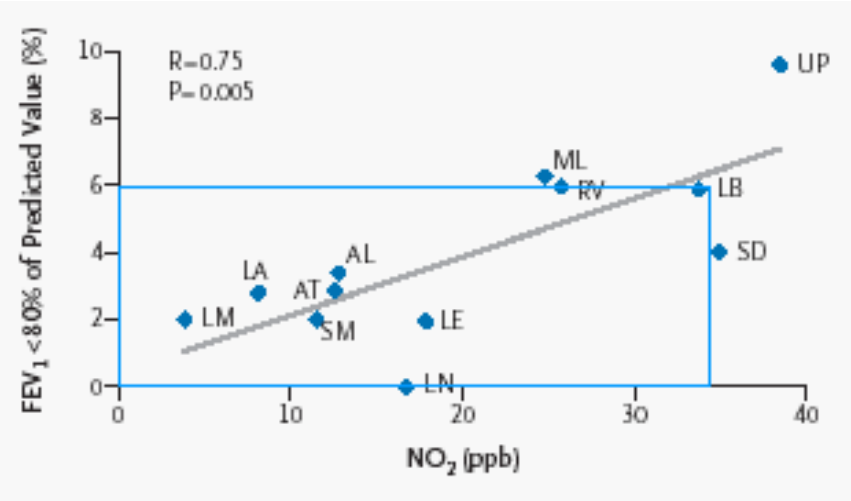

Figure 18: The association between lung function growth in children and ambient NO_2. Lung function growth is expressed as the community-specific proportion of 18-year-olds with a FEV_1 below 80 percent of the predicted value. Ambient NO_2 is the annual average level between 1994 through 2000. The communities were: AL denotes Alpine, AT Atascadero, LE Lake Elsinore, LA Lake Arrowhead, LN Lancaster, LM Lompoc, LB Long Beach, ML Mira Loma, RV Riverside, SD San Dimas, SM Santa Maria, and UP Upland. From Gauderman et al. (2004)[5].

PM

There were four-fold and five-fold differences, respectively in PM_{10} and $PM_{2.5}$ between the least and most polluted communities in the SCCHS. For PM_{10} the range in annual 24 hour average was 16.1 – 67.6 $\mu g/m^3$. In the fourth grade cohort (1,498 children) annual lung function growth was from ~ 11 to 12.2%. Lung function growth was greatest in the communities with low particulate exposure and lowest in those with high particulate exposure. For both PM_{10} and $PM_{2.5}$ these relationships were statistically significant (R= -0.57, p =0.03 and R = -0.52, p=0.05, respectively). For the seventh and 10th grade cohorts higher particulate exposure resulted in lower lung function growth, however in these cohorts the effect was not significant. A 51.5 $\mu g/m^3$ increase in annual 24 hour PM_{10} exposure was associated with an annual decrease of 0.58% in FVC, 0.85% in FEV_1, 1.32% in MMEF and 1.36% in FEF_{75}, which were all significant (P<0.05). A 29.5 $\mu g/m^3$ increased annual average two-week $PM_{2.5}$ exposure was associated with an annual decrease of 0.47% in FVC, 0.64% in FEV_1, 1.03% in MMEF and 1.31% in FEF_{75}, which was statistically significant for MMEF and FEF_{75} (P<0.05). When lung function growth was examined over a 4 year period those living in the highest PM_{10} exposure communities had a lung function growth that was 94-97.5% of those in low PM_{10} communities. Exposure to high levels of PM_{10} had a more adverse impact on lung function growth than exposure to environmental tobacco smoke [92].

The findings were confirmed for $PM_{2.5}$ in a second cohort of 1,678 fourth grade children, although they were of a lesser magnitude than for the first cohort [93]. A 22.2$\mu g/m^3$ increase in annual average $PM_{2.5}$ exposure was associated with an annual

decrease of 0.14% in FVC, 0.39% in FEV_1 and 0.94% in MMEF which was statistically significant for MMEF ($P<0.05$).

Follow-up of the first SCCHS cohort from the age of 10 to 18, has confirmed the impact of high NO_2 and $PM_{2.5}$ on lung development [5]. The significance of this study is that lung growth in females is almost completed by 18 and in males growth has slowed considerably, thus it is less likely that lung function growth after these ages will compensate for low lung function at age 18. Over the eight-year period, deficits in the growth of FEV_1 were associated with exposure to nitrogen dioxide ($P=0.005$), acid vapor ($P=0.004$), $PM_{2.5}$ ($P=0.04$) and elemental carbon ($P=0.007$) (Table 22). Associations were also observed for other lung function measures. The net result of these changes in lung function over an eight year period was that for a 51.5 $\mu g/m^3$ increase in annual average PM10 there was a 6% increase in the number of 18 year olds who had a clinically significant lower lung function (FEV_1 less than 80% of predicted) (Figure 19). These decreases in lung function growth were similar to those reported for maternal smoking [5], but smaller than those reported for the effects of personal smoking [5].

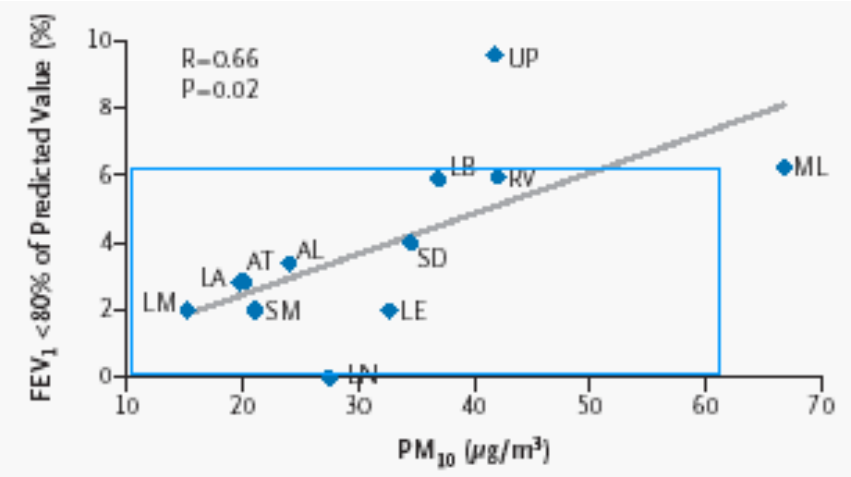

Figure 19: The association between lung function growth in children and ambient PM_{10}. Lung function growth is expressed as the community-specific proportion of 18-year-olds with a FEV_1 below 80 percent of the predicted value. Ambient PM_{10} is the annual average level between 1994 through 2000. The communities were: AL denotes Alpine, AT Atascadero, LE Lake Elsinore, LA Lake Arrowhead, LN Lancaster, LM Lompoc, LB Long Beach, ML Mira Loma, RV Riverside, SD San Dimas, SM Santa Maria, and UP Upland. From Gauderman et al. (2004)[5].

Exposure to pollutants was associated with clinically and statistically significant deficits in the FEV_1 (<80% of predicted) attained at the age of 18 years (Table 22). For example, the estimated proportion of 18-year-old subjects with a low FEV_1 (defined as a ratio of observed to expected FEV_1 of less than 80 percent) was 4.9 times as great at the highest level of exposure to $PM_{2.5}$ as at the lowest level of exposure (7.9 percent vs. 1.6 percent, $P=0.002$).

Table 22: Impact of ambient air pollution on lung function growth rate in children.

Pollutant	FVC*		FEV1*		MMEF*	
	Difference (ml)	p value	Difference (ml)	p value	Difference (ml)	p value
NO_2	-95.0	0.05	-101.4	0.005	-211.0	0.02
Acid vapour	-105.2	0.03	-105.8	0.004	-165.0	0.07
PM_{10}	-60.2	0.33	-82.1	0.08	-154.2	0.16
$PM_{2.5}$	-60.1	0.24	-79.9	0.04	-168.9	0.06
Elemental carbon	-77.7	0.08	-87.9	0.007	-165.5	0.04

* Values are the differences in the estimated rate of eight-year growth at the lowest and highest observed levels of the indicated pollutant. Differences are scaled to the range across the 12 study communities in the average level of each pollutant from 1994 through 2000 as follows: 37.5 ppb of O_3 (measured from 10 a.m. to 6 p.m.), 46.0 ppb of O_3 (the one-hour maximal level), 34.6 ppb of NO_2, 9.6 ppb of acid vapor, 51.4µg of PM_{10} per cubic meter, 22.8 µg of $PM_{2.5}$ per cubic meter, 1.2 µg of elemental carbon per cubic meter, and 10.5 µg of organic carbon per cubic meter.

Avol et al. (2001)[94] looked at the effect of relocation on lung growth in 110 children who relocated at the age of 10 and were followed up at age 15. Relocation of SCCHS children from an area with low PM_{10} to an area with 10 $\mu g/m^3$ higher PM_{10} would result in a 2.3% reduction in FEV_1 and a 5.7% reduction in MMEF. While this study demonstrated a negative effect of higher PM_{10}, it also demonstrated that the effect of PM_{10} was somewhat reversible, since in younger children who moved from a high PM_{10} exposure area to a low exposure area had an improvement in lung function.

Horak et al. (2002) followed on from earlier work by Frischer [95] and examined lung function growth in 975 Austrian school children (grades 2-3) in 8 communities with different levels of pollution [96]. An increase in 10 $\mu g/m^3$ of PM_{10} was associated with a decrease in FEV_1 growth of 84ml/yr and also a decrease in MEF_{25-75}.

Mortality and cancer

PM

There is evidence for an independent long-term PM effect on lung cancer mortality or total mortality [97].

Both the incidence and mortality for lung cancer was strongly associated with long-term concentrations of PM_{10} among males of the 6, 338 non-smoking adults participating in the Adventist Health and Smog (AHSMOG) study and followed from 1977 to 1992. In both men and women PM_{10} showed a strong association with mortality from non-malignant respiratory disease and lung cancer in males [98].

Pope et al. (2002) [99] found significant associations between long-term $PM_{2.5}$ exposure and lung cancer, cardiovascular or total mortality. This large study in the US linked mortality and air pollution data for 500,000 people across 50 States, who were followed for 18 years from 1982 to 1998. Each 10$\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 4%, 6% and 8% increase in total, cardiopulmonary and lung cancer mortality. The effects were not consistent for PM_{10}, although there was a trend

toward higher cardiopulmonary mortality with higher PM_{10}. There were no associations between long-term NO_2 levels and mortality.

Section B: Literature Review of the Health Effects of Ambient Air Toxics.

Background

The Environment protection and Heritage Council (EPHC) classify benzene, formaldehyde, toluene and xylene as air toxics. Air toxics are defined as gaseous, aerosol or particulate pollutants (other than the six criteria pollutants (see Section A), which are present in the air in low concentrations with characteristics such as toxicity or persistence so as to be a hazard to humans, plant or animal life [28]. The six criteria pollutants include particulate matter (PM_{10}), ozone, carbon monoxide, nitrogen dioxide, sulphur dioxide and lead (see Section A).

Air toxics exist at relatively low concentrations in urban air sheds, with significantly elevated levels only occurring near specific sources such as industrial sites, heavily trafficked roads and areas impacted by wood smoke (NEPC, 2003)[100]. All are considered to be carcinogenic in some animals and all are classified by the International Agency for Research on Cancer (IARC) as carcinogenic for human beings, with varying degrees of certainty. Considering ambient concentrations, however, the risk is rather low[100].

The air toxics of particular relevance to this report (that is, air toxics that result from engine exhausts) are the Volatile Organic Compounds (VOCs). The VOCs are organic compounds in the boiling range of 50-260^{o}C and include chemicals such as benzene, toluene and xylenes. VOCs are a concern because of their potential to contribute to the formation of ground level ozone and to global warming. Some of the VOCs can also have more direct effects on human health, for example, the link between benzene and leukaemia [28].

Burning fuels containing carbon (gasoline, oil, wood, coal, natural; gas), and using solvents, paints and glues releases VOCs. Motor vehicle emissions are an important source of VOCs.

Benzene

Benzene is a natural component of crude oil. Almost all benzene found at ground level comes from human activities. It is emitted from industrial sources and a range of combustion sources including motor vehicle exhaust and solid fuel combustion. Benzene is also emitted from tobacco smoke. The major outdoor source is evaporative emissions and evaporation losses from motor vehicles, and evaporation losses during the handling, distribution and storage of petrol. Workers in industries exposed to motor vehicle exhaust are at risk of exposure. Petrol vehicle emissions are the predominant source of benzene in the environment.(NICNAS, 2001) from

(NEPC 2003) In the past, benzene has been widely used as a multipurpose organic solvent, however, this use has been actively discouraged (NEPC, 2003)[100].

Benzene is naturally broken down by chemical reactions within the atmosphere. The length of time that benzene vapour remains in the air varies between a few hours and a few days depending on environmental factors, climate and the concentration of other chemicals in the air, such as nitrogen and sulphur dioxide.

Formaldehyde

Formaldehyde is a colourless gas with a strong irritant odour. Low levels of formaldehyde are produced as part of naturally occurring decomposition processes. In urban environments, formaldehyde emission sources include motor vehicle exhaust gases, domestic solid fuel and gas combustion, vapour release from goods manufactured using glues and resins containing formaldehyde and tobacco smoking (the last two sources being important indoor air quality issues). In addition, photochemical reactions involving oxidation of hydrocarbon compounds can produce formaldehyde. Formaldehyde is highly reactive and is important in photochemical smog formation[6].

Toluene

Toluene is a clear, colourless liquid with a distinctive smell. It occurs naturally in crude oil and is also generated through combustion of organic matter such as wood, coal and petroleum products. Motor vehicle emissions are the predominant source of toluene in the urban air environment, although evaporative losses from petroleum fuel storage facilities and service stations, and the use of toluene-based solvents and thinners are other contributors. Toluene is also a component of tobacco smoke. The highest concentrations of toluene usually occur in indoor air from the use of household products containing toluene (paints, thinners and adhesives) and cigarette smoke[6].

Xylene

Xylene is an aromatic hydrocarbon which exists in three isomeric forms: ortho-, meta- and para-xylene. The composition of xylene produced from petroleum is a mixture containing approximately 40 percent m-xylene and 20 percent each of o-xylene, p-xylene and ethylbenzene. Xylene occurs naturally in crude oil and is also generated through combustion of organic matter such as wood, coal and petroleum products. Motor vehicle emissions are the predominant source of xylene in the urban air environment. Evaporation from petroleum fuel storage facilities and service stations, and the use of products containing xylene-based solvents and thinners are other ways xylene enters the air environment[100].

Air quality goals/standards for air toxics

Australia

There are two sets of goals/standards that apply to ambient air toxics in the State of Queensland. These are the Australian Ambient Air Quality National Environment

Protection Measures (AAQ NEPMs) and the Queensland Environment Protection Policy (Air Quality Management) (EPP (Air) (Table 23).

Australia has recently introduced Ambient Air Quality National Environment Protection Measures (AAQ NEPMs) for Air Toxics which includes benzene, formaldehyde, toluene and xylene. The aim of the AAQ NEPMs for air toxics is to provide a framework for monitoring, assessing and reporting on ambient levels of five air toxics, benzene, formaldehyde, toluene, xylenes and PAHs, which will assist in the collection of information for the future development of national air quality standards for these pollutants. The AAQ NEPMs apply to areas where emissions from cumulative sources give rise to elevated levels of air toxics (e.g. hot-spots). Although industrial point sources may contribute to ambient levels in a specific area, the NEPM is not aimed at direct control of industrial emissions[13].

These contaminants were chosen with several others on the basis of:

- They were not primarily released from large point sources, such as factories;
- There was adequate ambient monitoring data;
- They present a significant risk to human health; and
- Based on the National Pollutant Inventory data, they had the highest mass emissions to the environment.

The AAQ NEPMs include monitoring investigation levels (MILs) and are based on the protection of human health. MILs are not compliance standards. If MILs are exceeded then some form of further investigation may be appropriate [27].

The risk of an increase in the number of people with cancer was selected as being appropriate as the basis for making AAQ NEPMs for benzene and PAHs, while for the other air toxics more acute toxicity was considered..

Table 23: National and Queensland air toxics standards and goals for ambient air [13, 27] and QLD EPA (2004)[6]

	Australian AAQ NEPMs			Qld EPP (Air)^	
Pollutant	Averaging period	Monitoring investigation level	Goal	Averaging period	Goal
Benzene	Annual average*	0.003ppm			
Benzo[*a*]pyrene (as a marker for PAHs)	Annual average	0.3ng/m^3	For all the five pollutants, the 8-year goal is to gather sufficient data nationally to facilitate development of a standard		
Formaldehyde	24 hours#	0.04ppm		30 minutes	0.07 ppm
	24 hours#			24 hours	2ppm
Toluene	Annual average	1ppm			
Xylenes (as total of ortho, meta and para isomers)	24 hours	0.1ppm			
	Annual average	0.25ppm 0.2ppm			

Nd= not determined.

*For the purposes of this Measure the annual average concentrations in are the arithmetic mean concentrations of 24-hour monitoring results. # For the purposes of this Measure monitoring over a 24 hour period is to be conducted from midnight to midnight. ^Intervention levels for Class 1, 2, and 3 indicators to be used in the assessment of local or neighborhood air monitoring data. For toluene and xylenes the Annual average and 24 hour monitoring investigation levels have been derived independently for different (chronic and acute) health endpoints. The 24 hour monitoring investigation levels in Table 23 have been derived from health based guidelines of shorter averaging periods:

- For formaldehyde the health based guideline is 0.08 ppm for a 1 hour averaging period;
- For toluene the health based guideline is 4 ppm for a 6 hour averaging period; and
- For xylene the health based guideline is 1 ppm for a 30 minute averaging period.

International

Australia's air quality standards for air toxics are similar to overseas standards (Table 24). Overseas standards and guidelines for benzene are based on the Goodyear Pliofilm study. NICNAS have used this as the basis for their review of the Occupational Health and Safety Standards in Australia. The WHO, European Commission, UK Expert Panel on Air Quality Standards and the USEPA all use the Pliofilm study as the key study when assessing carcinogenic risk form exposure to benzene. For purposes of guideline derivation, the WHO decided to use the 1994 risk calculation of Crump (of the Pliofilm cohort) rather than to derive new estimates. The geometric mean of the range of estimates of the excess lifetime risk of leukaemia at an air concentration of 1 µg/m3 is 6 ×10^{-6}. Using this unit risk factor, the concentrations of airborne benzene associated with an excess lifetime risk of 1/10,000; 1/100,000; and 1/1,000,000 are 17, 1.7 and 0.17 µg/m^3, (5.3, 0.53, 0.053 ppm) respectively[100].

The European Commission Working Group on Benzene Position Paper (1998) stated that the Goodyear Pliofilm cohort was the most thoroughly studied group. For purposes of guideline derivation, the Working Group chose to use the 1994 risk calculation of Crump (as did WHO) rather than to derive new estimates. It was considered to result in the highest plausible estimate of risk – an excess lifetime risk of leukaemia at an air concentration of 1µg/m3 of 6 x 10^{-6}[100].

Table 24: A comparison of international goals/standards for air toxics [100]

	Benzene	Benzo[*a*]pyrene	1,3-butadiene	Toluene	Xylenes
Australia	0.003ppm (annual average)	0.3ng/m^3 (annual average)	Nd[1]	0.1ppm (annual average)	0.2ppm (annual average)
United Kingdom air quality standards	5ppb (16.25 µg/m^3) (annual average)	0.25ng/m^3 (annual average)	Nd	Nd	Nd
New Zealand ambient air quality guidelines	10ug/m^3 (annual average)	0.3ng/m^3 (annual average)	Nd	Nd	Nd
European Commission air quality standard	5ug/m^3 (annual average)	Nd	Nd	Nd	Nd
World Health Organization	Nd	Nd	Nd	0.26mg/m^3 (weekly average)	Nd

Nd=Not determined

Ambient air toxic exposure

Sources of air toxics

Air toxics are released from a variety of sources and activities. Most of the sources are anthropogenic sources (that is, related to human activities). Sources of air toxics can be divided into two main categories: point sources and diffuse sources. Point sources comprise industrial and other facilities that emit large amounts of air toxics in a localised area. Diffuse sources include mobile sources (motor vehicles and aircraft) and area based sources (solid fuel combustion, dry-cleaning, building materials, and use of paints and thinners, cigarette smoking, household chemicals, and releases from carpets and furniture)[100].

Motor vehicle emissions

Important air toxics emitted by motor vehicles are benzene, 1,3-butadiene, PAHs, formaldehyde and acetaldehyde Table 25). Most of the air toxics from cars arise from the by-products of the combustion process when fuel is burnt in the engine and then emitted via the exhaust system, and from evaporation of the fuel itself [100].

Table 25: National Pollution Index air pollutant emissions from motor vehicles and total emissions for Queensland 2004-2005[101].

Substance	Motor vehicles	Total emission
Benzene	2,200,000 76%	2,900,000
1,3-Butadiene (vinyl ethylene)	380,000 81%	470,000
Carbon monoxide	430,000,000 56%	770,000,000
Formaldehyde (methyl aldehyde)	19%	% is based on all Australia
Oxides of Nitrogen	63,000,000 18%	350,000,000
Particulate Matter 10.0 um	2,200,000 1.16%	190,000,000
Polycyclic aromatic hydrocarbons	8.1%	% is based on all Australia
Sulfur dioxide	1,900,000 0.43%	440,000,000
Toluene (methylbenzene)	3,500,000 55%	6,400,000
Total Volatile Organic Compounds	26,000,000 7.9%	330,000,000
Xylenes (individual or mixed isomers)	2,200,000 55%	4,000,000

Air Toxic Levels in Brisbane

The Queensland EPA measured ambient air toxics at either Brisbane CBD and or Springwood sites in south-east Queensland site in 2004 [6].

Table 26: Ambient air toxic concentrations for Brisbane CBD and Springwood, south-east Queensland, 2004 [6].

Monitoring location	Site	Averaging Period	Maximum (ppb)	Second highest (ppb)	Percentiles					Minimum (ppb)	Annual average (ppb)
					99 (ppb)	95 (ppb)	90 (ppb)	75 (ppb)	50 (ppb)		
Benzene	Springwood	24hr	1.3	1.2	1.2	1.0	1.0	0.9	0.7	0.4	0.8
Toluene	Brisbane CBD	24hr	16.2	15.9	15.4	9.5	7.5	5.3	3.7	1.1	4.5
	Springwood	24hr	5.9	5.6	4.7	3.4	2.9	2.3	1.8	0.8	2.0
p-Xylene	Brisbane CBD	24hr	1.9	1.5	1.5	1.3	1.2	1.0	0.9	0.4	0.9
	Springwood	24hr	1.9	1.6	1.5	1.3	1.1	0.9	0.8	0.3	0.8
Formaldehyde	Brisbane CBD	24hr	5.2	5.1	4.9	3.9	3.5	3.0	2.5	1.2	2.7
		30 min	15.4	11.3	6.1	4.8	4.2	3.4	2.6	0.0	

Ambient benzene levels in Springwood (Table 26) did not exceed the Air Toxics AAQ NEPM Monitoring Investigation Level (MIL) of 0.003 ppm in 2004 [6]. The annual average benzene recorded was 0.8 ppb, which is 0.0008 ppm or 26% of the Air Toxics AAQ NEPM MIL[6].

Toluene was monitored at Brisbane CBD and Springwood in south-east Queensland during 2004. The primary toluene emission source at the Brisbane CBD and Springwood sites was motor vehicles. Maximum 24-hour average toluene concentrations were well below both the EPP (Air) goal (2000 ppb) and Air Toxics AAQ NEPM MIL (1000ppm) at all monitoring sites. Similarly, annual average toluene concentrations were well below the Air Toxics AAQ NEPM MIL (100ppb)[6].

EPA instrumentation is currently only capable of measuring levels of the p-xylene isomer, not total xylene. However, monitoring studies conducted in urban environments around the world have shown that p-xylene consistently comprises about 20 percent of the total xylene present in the atmosphere, providing the basis for an estimate of total xylene concentrations. During 2004, p-xylene levels (and by association total xylene levels) were considerably less than24 hour (250ppb) and annual average (200ppb) Air Toxics AAQ NEPM MILS at both monitoring sites in south-east Queensland[6]. The primary p-xylene emission source at the Brisbane CBD and Springwood sites was motor vehicles[6].

During 2004, formaldehyde levels were measured at the Brisbane CBD site in south-east Queensland. Levels at this site did not exceed either the EPP (Air) goal (70 ppb over 30 minutes) or the Air Toxics AAQ NEPM MIL of 40 ppb over 24 hours, for protection of human health [6].

Quantifying the Health Effects of air Toxics

The health effects of air toxics they are divided into cancer and non-cancer for the purpose of quantifying the dose response relationships.

Carcinogenic risk assessment

Cancer risk due to air toxics is assessed using unit risk factors (URFs). It is the risk of the incidence of cancer resulting from a lifetime exposure (usually 70 years) to $1\mu g/m^3$ of a carcinogenic substance[100].

URFs are often expressed on a per million basis for comparative purposes. For example, a cancer risk of 1 in a million (that is, $1*10^{-6}$) for a particular pollutant means that individuals exposed to $1\mu g/m^3$ for 70 years have a 1 in a million chance of developing cancer. These estimates generally assume a non-threshold, low dose linearity, unless there is compelling evidence to the contrary, and are derived from occupational or animal studies.

Cancer risk can be estimated by multiplying the unit risk factor by the modelled ambient concentration to obtain a probability of cancer occurring in an exposed population.

Risk = concentration*URF

For example, if the unit risk is $1*10^6$ per $1\mu g/m^3$ for chemical “Y”, and the concentration of chemical “Y” is $5\mu g/m^3$, then the risk is calculated as:

Risk = $(1*10^{-6})*5 = 5*10^{-6}$ i.e. there is the potential for five extra cancer cases to occur over a 70-year period in a population of one million persons exposed to 5 $\mu g/m^3$ of chemical "Y".

This represents an upper estimate (usually a 95% upper confidence limit) of the potential cancer cases in a population exposed to chemical "Y" at 5$\mu g/m^3$ over a lifetime.

In cases where there is exposure to multiple carcinogens, the cancer risk of each carcinogen is summed. The assumption implicit in this is that the effect on cancer risk in the population exposed to multiple pollutants is additive.

Non-cancer risk assessment

A number of measures have been used to assess the non-cancer risks of air pollutants. These are often known as investigation levels and are levels that, if exceeded, warrant further investigation.

The Californian EPA has developed 'Reference Exposure Levels' (RELs)[102] . These are health based levels and derived from No Observed Adverse Effect Level (NOAEL) or the Lowest Observed Adverse Effect Level (LOAEL) with uncertainty factors applied.

The Agency for Toxic Substances and Drug Registry (ATSDR) in the United States, has developed 'Minimal Risk Levels' (MRLs)[100]. A MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. MRLs are intended to be used as screening levels. They have been determined for inhalation, ingestion and dermal routes of exposure.

A 'Reference Concentration' (RfC) for chronic non-cancer effects is defined as the amount of toxicant, in ug/m^3, below which long term exposure to the general population, including sensitive subgroups, is not anticipated to result in any adverse effects. A central assumption underlying RfC is that a threshold exists below which no adverse effects will occur in the general population, although such a threshold may not be observable and can only be estimated. In general RfCs are derived from animal data through the application of extrapolation and uncertainty factors to NOAEL or LOAEL.

Non-cancer health endpoints are assumed to follow the concept of a threshold for effect. It is impossible to calculate the exact concentration at which anyone in a diverse population would respond. Inter-individual differences in response and the generally limited information on air toxics preclude such a determination. To quantify potential non-cancer health impacts the *Hazard Index* (HI) approach is recommended. While this method can quantify the increased risk due to air toxics, it cannot quantify the number of people affected. The HI approach compares air pollutant concentrations with a corresponding hazard assessment benchmark such as the RfC or the REL. The Hazard Quotient (HQ) for a particular pollutant is the ratio of the concentration of the pollutant to the RfC or REL for that pollutant.

$$HQ_i = concentration_i / RfC_i$$

HQ_i Hazard Quotient for pollutant i
$concentration_i$ concentration of pollutant i
RfC_i Reference Concentration (or REL) for pollutant i

A HQ>1 raises a "red flag" with regard to the exposure to that chemical and possible health impacts. Exceedance of a HQ of one does not necessarily mean that a health impact will in fact occur. It implies that the margin of safety built into the RfC is being eroded. The higher the ratio the closer the exposure to an adverse level. Uncertainty factors are included in the calculation of the RfCs to protect sensitive members of the population.

An indicator of total non-cancer hazard can be calculated by summing the HQs for each pollutant in order to derive a total HI.

$$HI_i = \Sigma HQ_i$$

HI_i is the sum of the hazard quotients for all pollutants of interest. This measure assumes that multiple sub threshold exposures may result in an adverse health effect. Aggregate non-cancer hazards for specific target organ systems can also be evaluated by creating a separate total hazard index for several chronic non-cancer endpoints. These measures assume that, in the absence of comprehensive information, the effects of each pollutant are additive for a given organ system. The Californian EPA has developed toxicological endpoints in various target organ systems to be considered for a range of air toxics[102].

Health Effects of Ambient Air Toxics

Benzene

Benzene is a gaseous air pollutant, known as an air toxic[100]. Current understanding of the health effects of benzene are mainly derived from animal studies and human health studies in the occupational setting. The adverse health effects of benzene exposure have been assessed by numerous agencies NICNAS (2001); WHO (2000); International Program on Chemical Safety, (1993); Commission of European Communities,(1998); United Kingdom Expert Panel on Air Quality Standards, (1994); US EPA (2000); Environment Canada (1993)[100]. Benzene is known to have both acute (short-term) and chronic (long term) effects on human health.

The short term effects of benzene occur at relatively high concentrations. According to the Australian National Occupational Health and Safety Commission (NOHSC, 2005) exposure to 79,750µg/m^3 (25 ppm) of benzene by humans for 8 hours is associated with no acute adverse effects.

The most significant adverse effects of long-term exposure to benzene has been linked to an increased incidence of blood and immune system disorders, including anaemia and leukaemia and birth defects in humans and animals. Benzene is considered to be a genotoxic carcinogen for which no threshold has been established

[17, 103]. As a 'safe' or 'no effect' level cannot be identified, quantitative risk estimation is used to express the cancer risk (probability) in numerical terms[100].The mechanisms of benzene toxicity are not well understood.

Acute effects

Acute effects of benzene include skin and eye irritations, headaches, drowsiness and vomiting[15]. According to the Australian National Occupational Health and Safety Commission (NOHSC) inhalation of 25 ppm of benzene by humans is associated with no acute adverse effects. The odour threshold for benzene is 1-1.5 ppm. Concentrations in the range of 50-150 ppm produce drowsiness, dizziness and headaches, with full narcosis at 4000 ppm, while concentration of 19000-20000 ppm is considered likely to be fatal.

Although all in the population are susceptible to the adverse health effects of benzene, it is thought that at levels occurring in the ambient atmosphere, benzene does not have short-term or acute effects.

Carcinogenic effects

Benzene is carcinogenic and long term exposure can affect normal blood production and can be harmful to the immune system. The mechanisms of benzene toxicity are not well understood. The known human health effects from long term exposure to benzene are bone marrow depression and leukaemia, specifically acute non-lymphocytic leukaemia (also known as acute myeloid leukaemia). Benzene is classified as a human carcinogen[100]. It is considered to be a genotoxic carcinogen for which there appears to be a dose-response relationship without any threshold effect (NICNAS 2001, US EPA 2000, WHO 2000)[100]. Most of the human health-exposure data have been obtained from retrospective epidemiological studies relating to occupational settings. It is accepted that there are difficulties in relating these studies usually in fit, healthy adults to the population in general, which consists of all ages and various levels of health and infirmity. According to the NEPC (2003) [100] there are four key long term occupational cohort studies demonstrating an association between benzene and leukaemia for which the exposures have been assessed in detail. These are the Goodyear Pliofilm, the Chemical Manufacturers Association (CMA), Dow Chemical and the Chinese Factory Worker cohorts[100].

The Goodyear Pliofilm cohort[100]

An excess incidence of leukaemia in rubber workers at two Goodyear facilities in Ohio, USA was reported in a preliminary paper by Infante et al. (1977) and in more detail by Rinsky et al. (1981). Depending on its definition, this cohort comprises 1165-1212 male workers employed from 1936-75 in the manufacture of Pliofilm. The manufacturing process used large volumes of benzene as a solvent and there was no exposure to other known carcinogenic substances. Excluding deaths before 1950, Rinsky et al. (1987) identified 15 deaths from lymphatic and haematopoietic cancers versus 6.6 expected (Standardised Mortality Rate[34] = 2.27 (1.27- 3.76) and 9 deaths from leukaemia versus 2.7 expected (SMR = 3.37 (1.54-6.41)[100].

The Chemical Manufacturers Association (CMA) cohort study[100]

This is a study of 4602 male chemical workers who were employed for ≥6 months from 1946-75 at 7 US plants (Wong, 1987a, 1987b). Two comparison groups were used: the general US population and 3074 unexposed male workers employed at the

same plants at the same time as the cohort. The vital status of all subjects was followed until the end of 1987 and the findings compared to average and peak exposures as determined from available air monitoring data and employment records obtained from the participating companies. There were 19 deaths from cancer of the blood and lymphatic system in the exposed workers compared to 3 in the unexposed group. In the exposed group, 7 of the observed cases were diagnosed with leukaemia and the remaining 12 with lymphoma. In the unexposed workers, all 3 cases were diagnosed with lymphoma; there were no cases of leukaemia in the unexposed workers. The SMRs for all cancers of the blood and lymphatic system were 0.91, 1.47, and 1.75, and for leukaemia 0.97, 0.78 and 2.76 for cumulative exposures of less than 180, 180-719 or ≥720 ppm-months respectively, but none of the ratios was significantly different from unity. The trend for all cancers of the blood and lymphatic system was significant ($p = 0.02$), and ($p = 0.01$) for leukaemia for trend with cumulative exposure[100].

The Dow Chemical cohort[100]

This study comprised 956 male chemical workers employed at a single site in Michigan, USA, between 1940 and 1982. The workers were exposed to benzene in chlorobenzene or 30 alkylation plants which used benzene as a raw material, or in an ethyl cellulose plant where benzene was used as a solvent (Bond et al, 1986; Ott et al, 1978). Each job entry was assigned an exposure intensity level on the basis of job classification and representative personal air monitoring data. There were 6 deaths from cancer of the blood and lymphatic system against 4.8 expected, including 4 cases of myelogenous leukaemia against 0.9 expected. The excess of myelogenous leukaemia was statistically significant ($p = 0.011$; SMR and 95% CI not stated)[100].

US National Cancer Institute (NCI) and Chinese Academy of Preventive Medicine (CAPM) Chinese factory workers cohort study[100]

A follow up on a large cohort study commenced in 1982 to assess the risks of specific bone marrow disorders in relationship to occupational benzene exposure (Hayes et al, 1997). The final cohort comprises 74,828 male and female benzene-exposed workers employed from 1972 to 1987 in 672 factories in 12 cities in China and 35,805 unexposed workers. Relative risks (RRs) were determined for incident cancer of the blood and lymphatic system, non- Hodgkin's Lymphoma (NHL), leukaemia, Acute non-lymphatic leukaemia (ANLL), a diagnosis of either ANLL or Myelo Dysplastic Syndromes (MDS), and leukaemia other than ANLL, with stratification by age and sex. The exposed workers held permanent jobs in the painting, printing, footwear, rubber and chemical industries. Exposure levels were estimated from available area monitoring data, detailed production and process information, and employee records[100].

There were 58 specified cancers of the blood and lymphatic system and 18 other bone marrow disorders (2 cases of agranulocytosis, 9 of aplastic anaemia and 7 of MDS) in the cohort, compared to 13 and 0 respectively in the control group.
When the cohort was divided into three categories, according to the estimated cumulative benzene exposure level, the RR for all cancer of the blood and lymphatic system was elevated from <40 ppm-years 2.2 (1.1-4.5). The RRs for leukaemia was elevated from 40-99 ppm-years 3.1 (1.2-8.0), and ANLL/MDS from 40-99 ppm-years 6.0 (1.8-20.6)[100].

Summary of benzene non-cancer health effects[100]
The No Observed Adverse Effect Level (NOAEL) for haematotoxicity in humans was established by Tsai et al (1983) at 0.53 ppm, and by Collins et al (1997) at 0.55 ppm, from long-term worker exposure studies, with daily 8 hours exposures, 5 days per week. NICNAS (2001) also conclude NOAELs to be around the 0.5 ppm level and a LOAEL (lowest observed adverse effect level) at 7.6 ppm in a subgroup of 11 exposed workers (Rothman et al 1996)[100].

Use of health data in setting air quality guidelines and standards.
The most widely used study as the basis of overseas standards and guidelines is the Goodyear Pliofilm study[100]. NICNAS have used this as the basis for their review of the Occupational Health and Safety Standards in Australia[100]. The WHO, European Commission, UK Expert Panel on Air Quality Standards and the USEPA all use the Pliofilm study as the key study when assessing carcinogenic risk form exposure to benzene[100]. For purposes of guideline derivation, the WHO decided to use the 1994 risk calculation of Crump (of the Pliofilm cohort) rather than to derive new estimates[100].

The US EPA gives a range for the quantitative estimate of "Leukaemia" from inhalational exposure to benzene (Table 27). The cancer risk factor (URF) is 2.2 to 7.8 persons per 1 million people exposed to 1 $\mu g/m^3$ [103]. WHO [17] air quality guidelines concluded that benzene is carcinogenic to humans and no safe level of exposure can be recommended and gives the estimated excess lifetime risk of leukaemia as 6 person per 1 million people exposed to 1 $\mu g/m^3$(Table 27). The Californian EPA [102] has determined benzene is a carcinogen and identified an inhalation URF of $29*10^{-6}$ per $\mu g/m^3$ (Table 27). The health endpoint for the exposure response relationship is "All Leukaemias". It is assumed that there is no threshold for the exposure response relationship and that exposure is over a 70-year lifetime exposure.

Table 27: Cancer unit risk factors for benzene

Source	Cancer endpoint	Inhalation URF (per $\mu g/m^3$)
Californian EPA	Leukaemia in humans	$29.0*10^{-6}$
WHO	Leukemia in humans	$6.0*10^{-6}$
US EPA	Leukaemia in humans	$2.2*10^{-6}$ to $7.8*10^{-6}$
IARC	Not stated	$4.4*10^{-6}$ to $7.4*10^{-6}$

The above four reports (Table 27) present a range of cancer unit risk factors for inhalational benzene. The California EPA estimate is about an order of magnitude greater than the other two estimates and the WHO estimate falls within the range of the US EPA estimates.

Formaldehyde

The health effects of formaldehyde have been extensively reviewed, see for example NEPC (2003)[100]. Exposure to moderate levels of formaldehyde (1000-3000ppb) can result in eye, nose and upper respiratory tract irritation. Odour annoyance often

occurs at concentrations below these levels. Formaldehyde has also been classified as a probable human carcinogen, although it has not been conclusively established that typical ambient concentrations are sufficient to cause cancer[6].

Acute Irritant effects

There are numerous reports that exposure to formaldehyde causes direct irritation of the respiratory tract. In a number of clinical studies, generally mild to moderate sensory eye, nose, and throat irritation was experienced by volunteers exposed for short periods to levels of formaldehyde ranging from 0.25 to 3.0 ppm (0.30 to 3.6 mg/m^3) [100].

There is evidence of formaldehyde inducing pathological and cytogenetic changes in the nasal mucosa of humans in studies with reported mean exposures ranged from 0.02 ppm to 235 ppm, with peaks between 4.2 ppm and 15 ppm. The LOAEL for short-term exposure is 0.08ppm[100].

There is substantial variation in individual responses to formaldehyde in humans. Significant increases in signs of irritation occur at levels above 0.1 mg/m^3 in healthy subjects[17]. At concentrations above 1.2 mg/m^3, a progression of symptoms and effects occurs[17]. Lung function of healthy non-smokers and asthmatics exposed to formaldehyde at levels up to 3.7 mg/m^3 was generally unaltered[17].

The studies by Kulle et al[104, 105] are key studies used in the development of air quality guidelines and standards[100]. Kulle et al al[104, 105] (1987; 1993) exposed healthy subjects to 0, 1.0, and 2.0 40 ppm for 3-hour periods and asked them to note symptoms of eye and nose/throat irritation and to rate severity on a 0-3 scale: 0=none; 1=mild (present but not annoying); 2=moderate (annoying); and 3=severe debilitating). Ten of the subjects were also exposed to 0.5 ppm and nine were exposed to 3 ppm for 3-hour periods. The frequencies of subjects reporting eye irritation or nose/throat irritation increased with increasing exposure concentration, especially at concentrations greater than or equal to1 ppm. Under non-exposed conditions, 3 of the 19 subjects noted mild nose/throat irritation and 1 noted mild eye irritation. At 0.5 ppm, 1 of 10 subjects noted mild nose/throat irritation, but none reported eye irritation. Frequencies for subjects with mild or moderate eye irritation were 4 of 19 at 1 ppm (1 moderate), 10 of 19 at 2 ppm (4 moderate), and 9 of 9 at 3 ppm (4 moderate). The increased frequency for eye irritation (compared with controls) was statistically significant at 2.0 ppm. Frequencies for mild nose/throat irritation were l of 19 at 1 ppm, 7 of 19 at 2 ppm, and 2 of 9 at 3 ppm. Compared with control frequency for nose/throat irritation, only the response at 2 ppm was significantly elevated[100, 104].

A study by Pazdrak et al (1993)[106] which included a group of subjects sensitised to formaldehyde, is a further study that has been used in deriving air quality standards and guidelines[100]. The Pazdrak study investigated the effects of formaldehyde exposure on the severity of symptoms of nasal and eye irritation and the cellular makeup of nasal discharge in occupationally exposed patients with skin hypersensitivity to formaldehyde and unexposed controls. The study was comprised of two study groups, all non-smokers. Group 1 consisted of 7 male and 3 female volunteers, all of whom suffered from skin hypersensitivity to formaldehyde; Group 2 consisted of 11 healthy males with no history of allergic diseases, normal serum IgE

levels, and negative skin tests to common allergens. Nasal washings were performed in both groups immediately before and after a 2-hour exposure to 0 and 0.4 ppm formaldehyde and at 4 and 18 hours after completion of the exposure periods. Symptoms of were evaluated through the exposure period and through 4- and 18-hour periods after the exposure period (maximum score = 7). In both groups, placebo inhalation periods were without effects on nasal wash cellular contents or symptom score. During exposure to 0.4 ppm formaldehyde, both groups showed statistically significantly increased average symptom scores compared with average placebo scores (about 4 versus <0.5). Symptom scores were no longer elevated 18 hours after exposure. The authors concluded that the symptoms observed were the result of a nonspecific, non-allergic process in response to low-level formaldehyde vapour exposure[106].

Carcinogenic effects

There is some evidence in animals and humans that formaldehyde has carcinogenic properties. The site of cancers appears to be the upper respiratory tract. However, unlike benzene and other genotoxic carcinogens, there appears to be a two-stage mechanism for the induction of neoplastic changes induced by exposure to formaldehyde. Repeated irritation to the nasal mucosa is believed to be the precursor to cellular changes that may lead to carcinogenic effects. Therefore, protecting against the irritative effects of formaldehyde is thought to protect against the more serious carcinogenic effects

IARC (1995)[107],US EPA (1991)[108] and WHO[17] judged that there was limited evidence in humans and sufficient evidence in animals that formaldehyde was reasonably anticipated to be a human carcinogen. Reported mean exposures ranged from 0.02 to 2.4 mg/m^3, with peaks between 5 and 18 mg/m^3. Epidemiological studies suggest a causal relationship between exposure to formaldehyde and nasopharyngeal cancer, although the conclusion is tempered by the small numbers of observed and expected cases [109, 110]. There are also epidemiological observations of an association between relatively high occupational exposures to formaldehyde and sinonasal cancer[17]. IARC [107] has interpreted the available cancer data as limited evidence for the carcinogenicity of formaldehyde in humans, and classified formaldehyde as a probable human carcinogen.

The US EPA unit risk factor for formaldehyde as a carcinogen is 13 persons per million exposed to 1 μg/m^3 of formaldehyde over 70 years. While the California EPA level was 1 person per 1 million people exposed to 1 μg/m^3 of formaldehyde over 70 years.

Formaldehyde is a nasal carcinogen in rats. A highly significant incidence of nasal cancer was found in rats exposed to a level of 16.7 mg/m^3, but the dose–response curve was nonlinear, the risk being disproportionately low at low concentrations[17].

For air quality standards and guidelines based on the irritant effects of formaldehyde the studies by Kulle[104] (1993; 1987) and Pazdrak (1993) appear to be the most widely used. The lowest observable adverse effect level (LOAEL) appears to be 1 ppm and the NOAEL 0.5 ppm (Kulle 1987) which was extrapolated to a 24 hour

concentration and adjusted using an uncertainly factor to an AAQ NEPM MIL of 0.04 ppm[100].

Toluene

Health effects associated with toluene exposure primarily relate to central nervous system impairment and behavioural dysfunction (ranging from slight drowsiness and headache to mental confusion and co-ordination loss depending on the extent of exposure). There is no indication that toluene is carcinogenic. Toluene has also been linked to hormonal imbalances, which may affect reproduction and foetal developmental[6, 17]. It should be noted that these effects have only been observed in situations where toluene concentrations were much higher than ambient levels, either through occupational exposure or deliberate solvent abuse (paint sniffing)[6].

Short term exposures

Andersen et al (1983)[111] reported the effects of toluene on 16 healthy young male subjects with no previous regular exposure to organic solvents. Groups of subjects were in a chamber for 6 hours a day on 4 consecutive days. The concentration of toluene was 0, 10, 40, or 100 ppm with each group exposed to a different toluene concentration each day. After 1, 5 and 6 hours of exposure, physiological, discomfort, and performance measurements were made for the next 1.5 hours. There was a significant change in nasal mucus flow from control values during all of the toluene exposures. During the 100 ppm exposure, statistically significant increased irritation was experienced in the eyes and in the nose, but not in the throat or lower airways. There was also a statistically significant increase in the occurrence of headaches, dizziness, and feelings of intoxication during the 100 ppm exposure, but not during the other concentrations. No adverse effects were reported at the 10 and 40 ppm levels.

Baelum et al (1985)[112] reported a LOAEL of 100 ppm for neurological effects in humans. In this study, occupationally exposed subjects were exposed to either clean air or air containing 100 ppm toluene for 6.5 hours in a climate chamber. A battery of ten tests of visuo-motor coordination, visual performance, and cortical function were administered during the 6.5 hour period. For toluene exposed subjects, there were complaints of air quality, irritation of the nasal passages, and increased feelings of fatigue and sleepiness. Subjects also complained of headaches and dizziness. Toluene exposure decreased performance on four of the neuro-behavioural tests; three on visual perseverance, one of visuo-motor function. Baelum et al. (1990)[113] evaluated effects of toluene at 0 or 100 ppm, or to varying exposures with peaks up to 300 ppm (with TWA = 102 ppm), for 7 hours. Volunteers exercised on an ergometer cycle during the exposure. Exposed subjects (with and without peak exposures) reported a significant increase over non-exposed subjects in nose and lower respiratory irritation, feelings of intoxication, dizziness, increased coughing, and headaches. No significant differences were found in the performances between the exposed and control groups in a battery of tests for performance, visual attention, and reaction times. Echeverria et al. (1991) reported a LOAEL of 75 ppm for neurological effects in humans. In this study, two groups of 42 students were exposed to 0, 75, and 150 ppm toluene for a 7 hour period. A complete battery of 12 tests was administered before and at the end of each exposure. Toluene caused a dose-related impairment of function on digit span pattern recognition, the one hole

test, and pattern memory at the 150 ppm level only. Test results for visual perception differed from control values for both exposure levels.

Longer term exposures

The lowest level of chronic occupational toluene exposure unequivocally associated with neurobehavioural functional decrements is 332 mg/m^3 (88 ppm)[114]. Studies of workers repeatedly exposed to toluene in workplace air at concentrations ranging from about 30 to 150 ppm have found evidence for increased incidence of self-reported neurological symptoms, performance deficits in neurobehavioural tests, hearing loss, changes in visual-evoked brainstem potential and colour vision impairment[100].

Women occupationally exposed to toluene at an average concentration of 332 mg/m^3 (88 ppm) incurred higher spontaneous abortion rates and menstrual function disturbances. The interpretation of these observations was hampered, however, by confounding factors. Men occupationally exposed to toluene at 5–25 ppm have also been shown to exhibit hormonal changes[114].

Toluene air quality guidelines and standards

The short term human exposure studies by Andersen et al (1983)[111] and Baelum et al (1985, 1990)[112, 113] have been used by various agencies to base their short-term exposure goals/standards[100]. In setting the AAQ NEPM MIL for toluene the NEPC converted the Anderson et al NOAEL of 40ppm concentration to a 24 hour concentration of 20ppm and then applied an uncertainly factor of 10, resulting in a goal of 2ppm from 24 hours.

Xylene

The adverse health effects of xylene exposure have been assessed by various agencies (see NEPC 2003)[100]. Health effects associated with xylene exposure primarily relate to eye, nose and throat irritation and neurological effects such as impaired reaction time, impaired short-term memory and changes in equilibrium and body balance. There is no indication that xylene is carcinogenic. Animal studies have suggested that xylene may affect foetal development. It should be noted that the above effects have only been observed in situations where xylene concentrations were much higher than levels typically experienced in ambient air[6].

The first signs of adverse effects of xylenes on humans are irritation of the nose, throat and eyes. The irritation has been chosen as the critical end point because it occurs at a low level after short exposures[100].

Short-term exposures

Carpenter et al (1975)[115] evaluated eye irritation in 6 human volunteers exposed for 15 minutes to 460, 1000, 2000, or 3000 mg/m^3. One volunteer noted mild throat discomfort at 460 mg/m^3, but not at 2000 mg/m^3. Four subjects reported eye irritation after exposure to 2000 or 3000 mg/m^3 (460 or 690 ppm) xylene for 15 min while one subject reported eye irritation at 1000 mg/m^3 (230 ppm) and none at 478 mg/m^3 (110 ppm) [100]. Hastings et al (1984)[116] exposed 50 healthy individuals to 100, 200, or 400 ppm mixed xylenes for 30 minutes to evaluate eye, nose, and throat irritation. The percent of subjects reporting eye irritation was 56% for controls (clean air), 60% at 100 ppm, 70% at 200 ppm, and 90% at 400 ppm. The authors concluded there was

no effect on eye irritation at 100 ppm because the incidence of irritation was as low as the control group[100].

Longer term exposures

Information on the toxicity of xylenes to humans is almost exclusively limited to case reports of acute exposures and studies of occupational exposures in which persons often inhaled a mixture of hydrocarbon solvents 8 hours per day, 5-6 days per week. These studies often have incomplete information on the airborne concentrations of xylene and other hydrocarbons. Uchida et al. (1993)[117], surveyed production workers exposed to a geometric mean of 14.2 ± 2.6 ppm xylene over 7 years. Exposure was to geometric means of 1.2 ppm o-xylene, 7.3 ppm m-xylene, 3.8 ppm p-xylene, 3.4 ppm ethyl benzene, and 1.2 ppm toluene. Analysis of data from the health examinations found no statistically significant difference between blood examinations for xylene-exposed and unexposed populations. Results of the survey on subjective symptoms found differences in symptoms occurring during work and during a similar analysis over the proceeding three-month period apparently related to effects on the functions of the central nervous system and to local effects on the eyes, nose and throat in the exposed workers. Dose dependency appeared to exist for 3 subjective symptoms noted during work: irritation in the eyes, sore throat and floating sensations[100].

Xylene air quality guidelines and standards

The short term human exposure studies by Hastings et al (1984)[116], and supported by Carpenter et al (1975)[115] have been used by various agencies to base their short-term exposure goals/standards[100]. In setting the AAQ NEPM MIL for xylene the NEPC converted the Hastings et al (1984)[116] NOAEL of 100ppm over 30 minutes to a 24 hour concentration of 2ppm and then applied an uncertainly factor of 10, resulting in a goal of 0.2ppm from 24 hours.

Section C: Health Effects Resulting from the proposed Airport Link: Changes to Ambient Benzene, CO, Formaldehyde, NO_2, PM_{10}, $PM_{2.5}$, Toluene and Xylene.

Approach

Areas considered

Assessment of the health effects as a result of the Airport Link was based on the modelled changes in pollutants at specific ground level receptors as provided by Holmes Air Sciences (HAS).

Our approach has been to use the forecast changes in pollutants at the ground level receptors, provided by HAS, to estimate the health impacts as a result of the changes in these pollutants. Consistent with the HAS working paper, we have considered the health effects resulting from changes to pollutants at two levels:

1) At ground level sites the maximum increases in CO, NO_2, PM_{10} and $PM_{2.5}$ were modelled to occur from vehicle emissions on roads associated with the proposed Airport Link. The CALINE 4 dispersion model was used to predict short-term concentrations of pollutants at various distances (10m, 30m & 50m) from 11 major roads in:

- 2012
- 2016
- 2026

2) At ground level sites in the vicinity of tunnel vents. HAS used the CALPUFF model to predict the maximum increases in benzene, CO, formaldehyde,NO_2, PM_{10}, $PM_{2.5}$, toluene and xylene resulting from predicted cumulative emissions from the portal ventilation emissions and surrounding roadways. Modelling was undertaken for:

- 2012
- 2016
- 2026

Ozone and sulphur dioxide are also known ambient air pollutants, however they are not considered relevant for health effect modelling of infrastructure projects such as Airport Link[16]. Ozone is not a primary pollutant, i.e. it does not come out of the tail pipe of motor vehicles, but is produced photochemically over a period of hours. Local changes in ozone as a result of the proposed Airport Link are therefore not

expected to occur as ozone is produced and dispersed across the airshed rather than locally. Sulphur dioxide is produced locally; however sulphur dioxide levels are very low across all Australian cities, perhaps due to low sulphur content fuels. Ambient sulphur dioxide levels are only of concern in Australia around metal smelters such as in Mt Isa and Port Pirie.

Estimating the health impacts of the forecast changes in ambient air pollutants was based on established peer reviewed publications of the relationships between community health outcomes and changes in these pollutants in Brisbane. Where sufficient information was not available for Brisbane non-peer reviewed reports were used as well as peer reviewed publications from other Australian or overseas cities.

A three tiered approach was used to encompass a wide range of potential health outcomes:

1) Acute effects on hospital admissions and mortality. Simpson *et al*. (2005a & 2005b) examined the effect of changes in CO, NO_2, PM_{10} and $PM_{2.5}$ on hospital admissions[72] and mortality[85]. Descriptions of these two studies are provided earlier in this report (Section A) and the results are provided in Appendices A and B. Where the results from the Simpson *et al*. (2005a & 2005b)[72, 85] studies differ from other Australian cities or meta-analyses, the worst case effect estimates were used.

The forecast changes in hospital admissions and mortality from epidemiological models such as Simpson *et al*. (2005a & 2005b)[72, 85] are likely to significantly over estimate the impact on mortality and hospital admissions as a result of the proposed Airport Link, since the proposed Airport Link is confined to a smaller area than considered in the epidemiological models (all Brisbane). To account for this, the effect is estimated per 100,000 people exposed to the increase as a result of the ventilation outlets or nearby roads.

2) Acute effects on symptoms. In addition to the effect on hospital admissions and mortality the effect on symptoms and medication use in children with asthma, a sensitive sub-group was also estimated. Again peer reviewed and published estimates of the relationship between ambient pollutants and health outcomes were used. Jalaludin *et al*. (2000 and 2004) investigated the impact of changes in ambient air pollutants on lung function and symptoms in asthmatic children in western and south western Sydney[64, 65]. Where the results from Jalaludin et al's studies differ from other meta-analyses, the worst case effect estimates were used. The WHO recently conducted a meta-analysis of the effects of PM_{10} on cough and medication use in children and adults with asthma [67]. Around 30 estimates were available for PM_{10} and cough or medication in children. The same meta-analyses also examined respiratory symptoms and lung function in children with chronic respiratory conditions.

3) Long term effects on health. The long term health effects of pollutants are less well understood than the short term effects, however, there have been two studies that are widely accepted. Gauderman et al. (2004)[5] demonstrated a long term effect on lung growth in children; while Pope et al. (2002)[99] found an effect on mortality.

There are other adverse health outcomes reported in the literature which have not been used in this report, for example, restricted activity days. The reason they have

not been considered is that there is less certainty about both their occurrence and the magnitude of any effect. The major undisputed health outcomes have been included in this report and provide both clarity and breadth regarding the potential health effects.

Health outcomes examined

The adverse health effects as a result of ambient pollutant exposure may range from the relatively mild sub-clinical effects such as throat irritation, clinical effects of reduction in lung function or increased medication usage, through to seeking medical attention from a GP, emergency department attendances, hospital admission and premature mortality. Figure 20 illustrates the relationship between the frequency of an adverse health outcome and its severity. Mortality and hospital admissions are often studied in relation to ambient air pollutants, since they are a clearly defined health outcome that has a measurable impact on the community. However, within a small community death is not a frequent event and therefore may be a less sensitive marker of a health impact than, for example, changes in respiratory symptoms. This is important in relation to assessing the health effect of changes in ambient air pollutants over relatively small areas, where fewer people may be exposed.

The forecast increases in pollutants resulting from emissions from the major roads and ventilation system or major roads connecting to the proposed Airport Link are likely to affect a relatively small number of people and therefore less severe but more frequent events such as respiratory symptoms may be better indicators of health effects, rather than rare events such as asthma deaths.

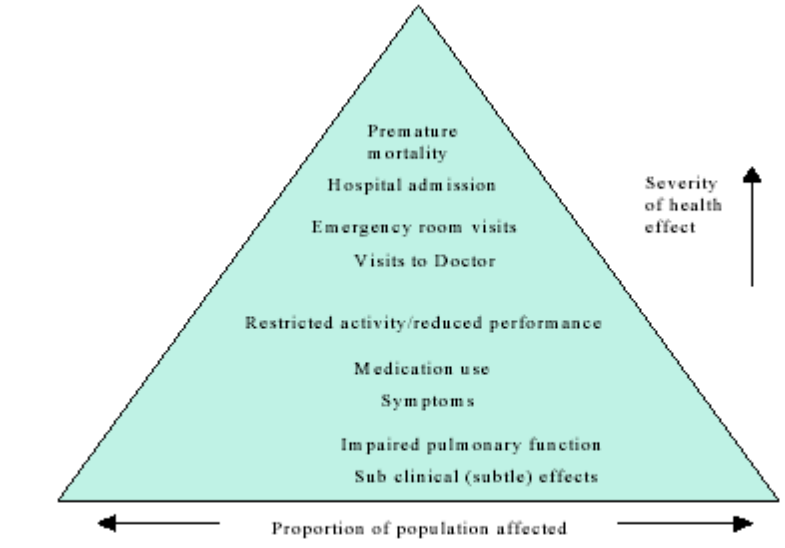

Figure 20: The severity and frequency of an adverse health impact as a result of ambient air pollution exposure. From WHO (2001)[118].

Section C1: Health Effects resulting from Ventilation Outlet and Surface Road Emissions associated with the proposed Airport Link

Holmes Air Sciences (HAS) provided the forecast increases in pollutants for ground level receptors at Albert Bishop Park, Bowen Hills, Kalinga Park and Kedron (Table 28). HAS used CALPUFF modeling to simulate the air quality impacts of the project over an area 20 km by 20km. The proposed Airport Link was located approximately in the centre of the modeled area. The forecast levels of pollutants associated with the proposed Airport Link were compared with no Airport Link for 2004, 2012, 2016 and 2026.

The results presented in Tables 28 and 29 (below) are the net incremental increase in ambient pollutants as a result of the increased emissions from the proposed Airport Link and do not include background levels of pollutants or pollutants from the proposed Northern Busway. This enables an assessment of the health effects on the community as a result of the increases in pollutants associated with the proposed Airport Link.

For most of the pollutants and at most locations, even under worst case conditions there is forecast to be either an improvement in air quality or no effect (Tables 28 and 29). The worst case increase in pollutants represent from 0.001% to 3.7% of the AAQ NEPMs. The forecast net ground level concentrations 8-hr maximum CO, 1-hr maximum NO_2, annual average NO_2, 24-hr maximum PM_{10} and 24-hr maximum $PM_{2.5}$ as a result of the Airport Link emissions are either negative or zero for Bowen Hills. At Kedron, Airport Link is forecast under worst case conditions to result in a small elevation in 1 hour maximum NO_2, annual average NO_2 and annual average PM_{10} (Table 28). At Kalinga Park, the only forecast increase is in 2026 for 8-hour CO which is forecast to increase by 0.1 mg/m^3, for all other pollutants and at all other times the pollutants are forecast either not to change or decrease (Table 28). At Albert Bishop Park small increases in each pollutant are forecast at some time periods (Table 28).

For the air toxics, the worst case net changes in acetaldehyde, 1,3 butadiene, benzene, Benzo(a)pyrene, formaldehyde, toluene and xylene are forecast to be negative at Bowen Hills, that is a very small improvement in air quality (Table 29). At Kedron small increases or no change are forecast (Table 29). For Eagle Farm small increases are forecast for all the air toxics. For acetaldehyde, formaldehyde, toluene and 24 hour xylene small increases are forecast (Table 29). Across all sites the worst case changes in air toxics are between 0.001 and 0.7% of the AAQ NEPM Monitoring Investigational Levels (Table 29).

In all cases where a net increase is forecast and added to the existing background level of pollutants, the resultant concentration is well below the AAQ NEPMs (Tables 28 and 29 compared with Tables 1 and 23) which are designed to protect the community. There is, however, no lower limit below which an adverse health impact will not occur, therefore an incremental increase in a pollutant can have an impact on health even though it is below the AAQNEPM. It should also be noted, that for all the

pollutants: the worst case maximum forecast increases as result of the proposed Airport Link are well below the maximum levels currently recorded in these regions (Tables 28 and 29 compared with Tables 3 -6 and 26).

Table 28: HAS's predictions for CO, NO_2 and PM at Albert Bishop Park, Kalinga Park, the Bowen Hills and Kedron air quality monitoring locations due to surface roads and ventilation outlets. From HAS Report, Table 14.

	2004	2012			2016			2026			
SITE	DM	DM	DS	DS-DM	DM	DS	DS-DM	DM	DS	DS-DM	Goal
Bowen Hills monitoring site											
Max. 8-hr ave. CO (mg/m³)	2.2	2.3	2.3	**0**	2.4	2.3	**-0.1**	2.4	2.4	**0**	10
Max. 1-hr ave. NO_2 (µg/m³)	169	174	168	**-6**	174	168	**-6**	174	169	**-5**	246
Annual ave. NO_2 (ug/m³)	34	35	34	**-1**	35	34	**-1**	35	34	**-1**	62
Max. 24-hr ave. PM_{10} (µg/m³)*	4.2	4.2	3.9	**-0.3**	4	3.6	**-0.4**	3.7	3.3	**-0.4**	50
Annual ave. PM_{10} (µg/m³)*	1.1	1.1	1	**-0.1**	1	0.9	**-0.1**	0.9	0.8	**-0.1**	30
Kedron monitoring site											
Max. 8-hr ave. CO (mg/m³)	2.1	2.1	2.1	**0**	2.1	2.1	**0**	2.1	2.2	**0.1**	10
Max. 1-hr ave. NO_2 (µg/m³)	146	150	156	**6**	151	158	**7**	153	162	**9**	246
Annual ave. NO_2 (ug/m³)	30	30	31	**1**	30	31	**1**	30	31	**1**	62
Max. 24-hr ave. PM_{10} (µg/m³)*	2.3	2.2	2.1	**-0.1**	2.1	2	**-0.1**	1.8	1.8	**0**	50
Annual ave. PM_{10} (µg/m³)*	0.4	0.4	0.5	**0.1**	0.4	0.5	**0.1**	0.3	0.4	**0.1**	30
Kalinga Park (location 505603 mE, 6968186 mN)											
Max. 8-hr ave. CO (mg/m³)	2.1	2.1	2.1	**0**	2.1	2.1	**0**	2.1	2.2	**0.1**	10
Max. 1-hr ave. NO_2 (µg/m³)	144	147	147	**0**	148	148	**0**	151	151	**0**	246
Annual ave. NO_2 (ug/m³)	30	30	30	**0**	30	30	**0**	30	30	**0**	62
Max. 24-hr ave. PM_{10} (µg/m³)*	1.7	1.7	1.6	**-0.1**	1.6	1.5	**-0.1**	1.4	1.4	**0**	50
Annual ave. PM_{10} (µg/m³)*	0.4	0.4	0.4	**0**	0.4	0.4	**0**	0.4	0.4	**0**	30
Albert Bishop Park (location 506853 mE, 6968486 mN)											
Max. 8-hr ave. CO (mg/m³)	2.1	2.1	2.2	**0.1**	2.1	2.2	**0.1**	2.2	2.2	**0**	10
Max. 1-hr ave. NO_2 (µg/m³)	148	150	150	**0**	152	153	**1**	155	156	**1**	246
Annual ave. NO_2 (ug/m³)	30	30	31	**1**	31	31	**0**	31	31	**0**	62
Max. 24-hr ave. PM_{10} (µg/m³)*	1.7	1.6	1.7	**0.1**	1.6	1.7	**0.1**	1.5	1.5	**0**	50
Annual ave. PM_{10} (µg/m³)*	0.5	0.4	0.5	**0.1**	0.4	0.5	**0.1**	0.4	0.4	**0**	30

* Predictions due to modelled roads and outlets only. DM refers to "Do Minimal" or "No tunnel case". DS refers to "Do Something" or "Tunnel case".

Table 29: HAS's predictions in 2012 for air toxics and current levels in 2004 at the Bowen Hills, Eagle Farm and Kedron air quality monitoring locations due to surface roads and ventilation outlets. Predictions for 2012 are also expressed as change (DS-DM), change as a percentage of 2004 concentrations and change as a percentage of the Ambient Air Quality National Environmental Protection Measure Monitoring Investigation Levels (AAQ NEPM MIL).

Air Toxic (mg/m^3)	2004	2012 DM	2012 DS	2012 Change (DS-DM)	AAQ NEPM MIL	2012 change % 2004	2012 change % AAQ NEPM MIL
Bowen Hills air quality monitoring site							
Annual ave. 1,3 Butadiene	2.54E-05	3.44E-05	3.15E-05	-2.90E-06	-	-11.4%	
Annual ave. Acetaldehyde	5.51E-05	7.48E-05	6.84E-05	-6.40E-06	-	-11.6%	
Annual ave. Benzene	2.57E-04	3.48E-04	3.19E-04	-2.90E-05	9.35E-03	-11.3%	-0.3%
Annual ave. Benzo(a)pyrene	1.80E-08	2.44E-08	2.23E-08	-2.10E-09	3.00E-07	-11.7%	-0.7%
Annual average Formaldehyde	8.06E-05	1.09E-04	1.00E-04	-9.00E-06	-	-11.2%	
Annual average Toluene	4.09E-04	5.55E-04	5.08E-04	-4.70E-05	3.84E-01	-11.5%	0.0%
Annual average Xylene	2.96E-04	4.02E-04	3.68E-04	-3.40E-05	8.44E-01	-11.5%	0.0%
Maximum 24-hr. ave. Toluene	1.85E-03	2.44E-03	2.21E-03	-2.30E-04	3.84E+00	-12.4%	0.0%
Maximum 24-hr. ave. Xylene	1.34E-03	1.76E-03	1.60E-03	-1.60E-04	1.06E+00	-11.9%	0.0%
Eagle Farm air quality monitoring site							
Annual ave. 1,3 Butadiene	1.41E-05	1.65E-05	1.66E-05	1.00E-07	-	0.7%	
Annual ave. Acetaldehyde	3.05E-05	3.58E-05	3.60E-05	2.00E-07	-	0.7%	
Annual ave. Benzene	1.42E-04	1.67E-04	1.68E-04	1.00E-06	9.35E-03	0.7%	0.0%
Annual ave. Benzo(a)pyrene	9.96E-09	1.17E-08	1.17E-08	0.00E+00	3.00E-07	0.0%	0.0%
Annual average Formaldehyde	4.47E-05	5.24E-05	5.27E-05	3.00E-07	-	0.7%	
Annual average Toluene	2.27E-04	2.66E-04	2.68E-04	2.00E-06	3.84E-01	0.9%	0.0%
Annual average Xylene	1.64E-04	1.93E-04	1.94E-04	1.00E-06	8.44E-01	0.6%	0.0%
Maximum 24-hr. ave. Toluene	9.99E-04	1.18E-03	1.22E-03	4.00E-05	3.84E+00	4.0%	0.0%
Maximum 24-hr. ave. Xylene	7.23E-04	8.57E-04	8.84E-04	2.70E-05	1.06E+00	3.7%	0.0%
Kedron air quality monitoring site							
Annual ave. 1,3 Butadiene	1.22E-05	1.50E-05	1.80E-05	3.00E-06	-	24.6%	
Annual ave. Acetaldehyde	2.64E-05	3.27E-05	3.90E-05	6.30E-06	-	23.9%	
Annual ave. Benzene	1.23E-04	1.52E-04	1.82E-04	3.00E-05	9.35E-03	24.4%	0.3%
Annual ave. Benzo(a)pyrene	8.61E-09	1.06E-08	1.27E-08	2.10E-09	3.00E-07	24.4%	0.7%
Annual average Formaldehyde	3.87E-05	4.78E-05	5.71E-05	9.30E-06	-	24.0%	
Annual average Toluene	1.96E-04	2.43E-04	2.90E-04	4.70E-05	3.84E-01	24.0%	0.0%
Annual average Xylene	1.42E-04	1.75E-04	2.10E-04	3.50E-05	8.44E-01	24.6%	0.0%
Maximum 24-hr. ave. Toluene	1.04E-03	1.26E-03	1.24E-03	-2.00E-05	3.84E+00	-1.9%	0.0%
Maximum 24-hr. ave. Xylene	7.50E-04	9.08E-04	8.94E-04	-1.40E-05	1.06E+00	-1.9%	0.0%

Benzene

Across the three ground level receptors, that were selected by HAS for modelling, increases in annual average benzene concentration were forecast in 2012 for Eagle Farm and Kedron (Table 29). The highest forecast increase was at Kedron and was 3×10^{-5} mg/m^3 or 0.03 $\mu g/m^3$. This represents a 24.4% increase above the background benzene concentration in 2004 and is equivalent to 0.3% of the AAQ NEPM MIL for benzene.

Benzene is known to have both short-term and long-term effects on human health. Acute effects of benzene include skin and eye irritations, headaches, drowsiness and vomiting (US EPA, 2002). According to the Australian National Occupational Health and Safety Commission (NOHSC), inhalation of 79,750µg/m^3 (25 ppm) of benzene by humans is associated with no acute adverse effects. The odour threshold for benzene is 3,190-4,785 µg/m^3 (1-1.5 ppm). Concentrations in the range of 159,500-478,500 µg/m^3 (50-150) ppm produce drowsiness, dizziness and headaches, with full narcosis at 12,800,000µg/m^3 (4,000) ppm. A concentration of 61,000,000 µg/m^3 (9,000-20,000 ppm) is considered to be likely to be fatal (NOHSC, 2005). HAS did not model the short-term increase in benzene resulting from emissions associated with the proposed Airport Link, however based on no change in annual average benzene, it seems extremely likley that the acute increases would be several thousand fold lower than the NOHSC no effect level.

Long term health effects

For the purposes of quantifying the chronic health effects of exposure to benzene the main health endpoint used is leukaemia, for which no lower threshold has been established[100]. A 1µg/m^3 increase in benzene sustained over a 70 year period would result in approximately 8 additional leukaemia cases per 1 million people exposed over a 70 year period[100].

The forecast worst case increase in ambient benzene is (0.03µg/m^3) (Table 29), if sustained over a 70 year period this would be expected to result in approximately 0.24 additional leukaemia cases per 1 million people exposed over a 70 year period[103]. Therefore the additional risk of developing leukaemia as a result of 70 years of exposure to the 0.03 µg/m^3 increase in benzene is 0.000024%. This is a negligible increase in leukaemia risk. By comparison in Queensland in 2002 the cumulative rate for all types of leukemia by age 70 was 0.35%[119]. In 2002 the total number of people with leukaemia aged 70 years and older in Queensland was 1086 persons and the incidence was 173 new cases for 2001-2002[119].

CO

For Bowen Hills no increases in 8 hour ambient CO in 2012, 2016 and 2026 are predicted (Table 28). For the other three sites the maximum increase was 0.1 mg/m^3 which is equivalent to 1.8% of the highest and 20.8% of the median level of CO level recorded at the EPAs Woolloongabba monitoring site in 2004 (Table 3). The worst case increase in 8-hour average CO level resulting from the proposed Airport Link is equal to 1% of the AAQ NEPM of 10 mg/m^3 and when combined with highest levels in 2004, results in a total ambient level of 2.2 mg/m^3, which is 22% of the AAQ NEPM.

Acute effects on hospital admissions and mortality

In assessing the worst case health effects on the community resulting from CO emissions from major roads and ventilation outlets associated with the proposed Airport Link, the maximum increase in 8-hour CO was used, which was 0.1mg/m^3 (Table 28). The recent analysis of all four Australian cities by Simpson *et al*. (2005)[72, 85] did not present data for CO. There have been no peer reviewed studies that have

published an association between CO and hospital admissions in Brisbane. The Victorian EPA study[68], predicts that a 0.1 mg/m^3 increase in 8-hour CO would result in 0.08%, 0.54% and 0.23% increases in the background risk for hospital admissions for respiratory diseases, asthma and cardiovascular diseases, respectively, in all ages on each day when this maximum increase in ambient 8-hour maximum CO occurred (Table 30).

The background daily risk for hospital admissions for respiratory diseases, asthma and cardiovascular diseases in Brisbane was reported to be 3.26 persons/100,000 people, 0.72 persons/100,000 people and 2.19 persons/100,000 people, respectively (Table 28). The incremental increase in hospital admissions for all respiratory diseases, asthma and cardiovascular diseases as a result of the maximum increase in CO associated with the proposed Airport Link is therefore 0.003 (0.08% x 3.26), 0.004 (0.54% x 0.72) and 0.005 (0.23% x 2.19) persons/100,000 exposed population on each day when the maximum CO level occurs (Table 30). This is a negligible increase in health risk

The forecast maximum change in CO would result in a 0.17 % increase in all causes of mortality on each day when the maximum CO level occurs (Table 30). The background risk for daily mortality is 0.92 persons/100,000 people. Therefore on the days when the maximum increase in CO occurred it would result in an incremental increase in mortality of 0.0015 (0.17% x 0.92) persons/100,000 people (Table 30). It is unlikely that this small effect could be measured.

Table 30: Potential increases in the background daily rate of health events as a result of the largest forecast increase in regional CO associated with the proposed Airport Link.

Largest potential maximal increase in pollutant 8-hour CO (mg/m^3)	Adverse health event	Most conservative estimate of percentage increase in health effect on maximum CO pollution day	Background daily event rate	Projected increment in rate on maximum CO pollution day
0.1	Respiratory admission (all ages)	0.08%[1]	3.26 persons/100,000 people[2]	0.003 person/100,000 people exposed to the worst case increase
0.1	Asthma admission (all ages)	0.54%[1]	0.72 persons/100,000 people[2]	0.004 person/100,000 people exposed to the worst case increase.
0.1	Cardiovascular Admissions (all ages)	0.23%[1]	2.19 persons/100,000 people[3]	0.005 persons/100,000 people exposed to the worst case increase.
0.1	Cardiovascular mortality (all ages)	0.17%[1]	0.92/100,000 people[3]	0.0015 persons/100,000 people exposed to the worst case increase.

[1] Epidemiological studies of the effect of CO on health in Melbourne from the EPA Studies [68, 86]
[2] Epidemiological studies of the effect of air pollution on health in Brisbane[70]
[3] Epidemiological studies of the effect of air pollution on health in Brisbane[72, 85]

Acute effect on symptoms

The impact of small increases in CO on symptoms, respiratory or cardiovascular function has not been extensively researched and there are no Australian panel studies or meta-analysis of this. Chamber studies of acute exposure to CO were used to set the WHO thresholds for CO, which are designed to maintain COHb below 2.5% (Table 8). The WHO 8 hour guideline of 10 ppm (11.6 mg/m^3) is well above the forecast 0.1 mg/m^3 increase in CO reported in Table 28, therefore acute clinical effects of CO exposure from regional increases in CO associated with the proposed Airport Link are not expected.

Formaldehyde

Across the three ground level receptors, that were selected by HAS for modelling, increases in maximum annual average formaldehyde concentration in 2012 were forecast for Eagle Farm but not Bowen Hills or Kedron. The forecast increases in annual average formaldehyde concentration in 2012 is 3×10^{-5} mg/m^3, which is very small and represents an increase equivalent to 0.68% of the current level recorded at Eagle Farm.

The US EPA unit risk factor for formaldehyde as a carcinogen is 13 persons per 1 million exposed to 1 $\mu g/m^3$ of formaldehyde over 70 years. The modeled maximum annual average formaldehyde concentrations of 3×10^{-5} mg/m^3 (Table 29), is equivalent to 3% of the unit risk factor and therefore would be predicted to result in 0.39 additional cancer cases per 1 million people exposed to this increase over a 70 year period and therefore no increase in cancer risk.

NO_2

Acute effects on hospital admissions and mortality

For Bowen Hills 1 hour maximum NO_2 is predicted to decrease a result of the proposed Airport Link (Table 28). No increase is forecast for Kalinga Park and 1 $\mu g/m^3$ increase is predicted for Albert Bishop Park. The predicted maximum increase of 9 $\mu g/m^3$ for 1-hour maximum NO_2 at Kedron at in 2026 is equivalent to 5.9% of the highest 1 hour maximum recorded at the EPA's Brisbane CBD monitoring station in 2004 (Table 4) and 29% of the highest median 1 hour maximum recorded at the South Brisbane monitoring station.

Previous research in Brisbane has found associations between changes in 1-hour maximum NO_2 concentration and both hospital admissions and mortality [70, 91]. The meta-analyses of 4 Australian cities, including Brisbane found that an increase in ambient NO_2 was significantly associated with increases in total, cardiovascular and respiratory mortality (Table 18) [72]. Hospital admissions for cardiovascular and respiratory diseases were also significantly associated with increases in 1 hour maximum ambient NO_2 across the 4 cities (Table 15) [85]. An earlier study in Melbourne reported a significant association between NO_2 and asthma [68].

The maximum increase in regional 1-hour maximum in NO_2 as a result of emissions from vents and major roads associated with the proposed Airport Link is forecast to be 9$\mu g/m^3$ (Table 28), which using the Australian 4-cities meta-analysis data [72], is forecast increase in mortality of 0.53% (Table 31). Hospital admissions for cardiovascular disease in all ages, respiratory disease in people aged 65 and over and asthma in all ages, are forecast to increase by 0.56%, 0.66% and 2.62%, respectively (Table 31)[68, 85]. These predictions are supported by separate epidemiological studies conducted in Sydney and Perth which have also shown positive associations between NO_2 and hospital admissions for respiratory and cardiovascular diseases and mortality[69, 71, 73].

The forecast incremental increase in hospital admissions for cardiovascular diseases, respiratory admissions in people aged 65 and over and asthma are forecast to be 0.022 (1.01% x 2.19), 0.010 (1.19% x 0.88) and 0.019 (2.62% x 0.72) persons per 100,000 population exposed to the maximum increase per day on the days when the maximum increases in NO_2 occurs (Table 31). The incremental increase in mortality is forecast to be 0.010 person/100,000 people exposed to the worst case increase in NO_2.

Table 31: Potential increases in the background daily rate of health events as a result of the largest forecast increase in regional NO_2 exposure from emissions from vents and major roads associated with the proposed Airport Link.

Largest potential maximal increase 1-hour NO_2 ($\mu g/m^3$)	Adverse health event	Most conservative estimate of percentage increase on maximum NO_2 pollution day	Background daily event rate	Projected increment in rate on maximum NO_2 pollution day
9	Cardiovascular Admissions (all ages)	1.01%[1]	2.19 persons/100,000 population[1]	0.022 persons/100,000 people exposed to the worst case increase.
9	Asthma admission (all ages)	2.62%[2]	0.72 persons/100,000 population[3]	0.019 person /100,000 people exposed to the worst case increase.
9	Respiratory Admissions (65+ years)	1.19%[1]	0.88 persons/100,000 population[1]	0.010 persons/100,000 people exposed to the worst case increase.
9	Mortality (all ages)	0.53%[1]	1.99/ persons/100,000 people[1]	0.010 persons/100,000 people exposed to the worst case increase.

[1] Epidemiological studies of the effect of air pollution on health in Brisbane [72, 85]
[2] Melbourne estimates as published by Denison *et al*. (2001)[68]
[3] Petroechevsky *et al*. (2001) for Brisbane [70]

Acute effect on symptoms

Less severe and more common health effects are changes in lung function, symptoms or medication use. Jalaludin *et al* (2000 and 2004)[64, 65] found no association between NO_2 and lung function, wheeze, dry cough, medication use, doctor visits for asthma, but a significant association with wet cough. This study was conducted in western and southwestern Sydney. The study followed 125 children, aged 10, with a history of wheeze for an 11 month period in 1994. A 0.0082 ppm increase in average daytime NO_2 was associated with a 5% increase in parent reported wet cough, which was statistically significant ($p<0.05$) [64]. Unfortunately NO_2 was measured as average daytime NO_2, not maximum 1 hour NO_2. The relationship between symptoms and an increase in 1 hour maximum NO_2, as forecast to result from emission from the proposed Airport Link, is unknown.

Long term effect on lung function growth

A long term impact of elevated levels of NO_2 was found in the Southern California Children's Health Study (SCCHS)[5]. A 34.6 ppb (71.5 $\mu g/m^3$) increase in annual 24 hour NO_2, over an eight year period, resulted in a 6% increase in the number of 18 year olds who had a clinically significant lower lung function (FEV_1 less than 80% of predicted).

The forecast worst case change in annual NO_2 as a result of emissions from the proposed Airport Link was forecast to be 1 $\mu g/m^3$ at Kedron (Table 28), which is 1.4% of the difference observed in the SCCHS. Based on HAS's forecast annual average level of NO_2 and the published SCCHS studies [5], the impact on lung function growth in adolescents is likely to be very small.

Long term effect on mortality

Pope et al. (2002) [99] did not find a relationship between long term exposure to elevated levels of NO_2 and increases in total, cardiopulmonary and lung cancer mortality. On the basis of Pope's work [99] there is not forecast to be an increase in long term mortality as a result of NO_2 from emission associated with the proposed Airport Link.

PM_{10}

The forecast changes in ambient 24 hour and annual average PM_{10} concentrations resulting from proposed Airport Link are either negative, i.e. an improvement in air quality or zero (Table 28), with the exception of Albert Bishop Park in 2012 and 2016 where a 0.1$\mu g/m^3$ increase is forecast (Table 28).

Acute effects on hospital admissions and mortality

The maximum forecast increase in PM_{10} is predicted to result in negligible increases in hospital admissions for all respiratory diseases (0.03%), cardiovascular diseases (0.02%) and respiratory diseases in people over 65 (0.03%) and a 0.02% increase in total mortality [70, 72, 80, 85] on the days when this maximum increase actually occurs (Table 32). The background daily rate of these health events is small, therefore only very small increases in these events are forecast (0.0003-0.0005 per 100,000 people) (Table 32).

Table 32: Potential increases in the background daily rate of health events as a result of the largest forecast increase in regional PM_{10} resulting from the proposed Airport Link.

Largest potential maximal increase in 24-hour PM_{10} ($\mu g/m^3$)	Adverse health event	Most conservative estimate of percentage increase on maximum PM_{10} pollution day[1,2]	Background daily event rate	Projected increment in rate on maximum PM_{10} pollution day
0.1	Cardiovascular Admissions (all ages)	0.02%[1]	2.19 persons/100,000 people[1]	0.0005 persons/ 100,000 people exposed to the worst case increase.
0.1	Respiratory Admissions (65+ years)	0.03%[1]	0.88 persons/100,000 people[1]	0.0003 persons/ 100,000 people exposed to the worst case increase.
0.1	Respiratory Admissions (all ages	0.03%[4]	3.26 persons/100,000 people[4]	0.0002 persons/ 100,000 people exposed to the worst case increase.
0.1	Total mortality (all ages)	0.02%[5]	1.99 persons/100,000 people[1]	0.0004 persons/ 100,000 people exposed to the worst case increase.
0.1	Visits to doctor for asthma	0.09%[2]	47/100,000 people	0.04 visits/100,000 people exposed to the worst case increase.
0.1	Lower respiratory symptoms in children with chronic respiratory conditions[3]	0.01%	N/A	
0.1	Cough in adults[3]	0.04%	N/A	

[1] As published for the 4 cities meta-analysis by Simpson *et al* (2005a, 2005b) [72, 85].
[2]Sydney estimates as published by Jalaludin *et al.* (2004) [64].
[3] As published by WHO 2004[67].
[4] Petroechevsky *et al*. (2001) for Brisbane [70]
[5] Simpson *et al.* (1997) for Brisbane[80]

Acute effects on symptoms

Jalaludin *et al* (2000 and 2004)[64, 65] found no association between PM_{10} and lung function, wheeze, cough or medication use, but a significant association with doctor visits for asthma. A 12 $\mu g/m^3$ increase in 24 hour PM_{10} was associated with an 11% increase in visits to the doctor for asthma, which was statistically significant ($p<0.05$) [64]. Based on the results of Jalaludin *et al*. (2004) [64] and the worst case forecast increase in PM_{10} of 0.1 $\mu g/m^3$, as a result of emissions from the vents and roadways associated with the proposed Airport Link, a 0.09% increase in doctor attendances for asthma in children exposed to the worst case increase in PM_{10} may occur (Table 32). The daily rate of GP attendances for asthma is around 47 per 100,000 people. The increased risk for GP attendance for asthma as a result of the worst case increase in 24 hour PM_{10} from the proposed Airport Link is therefore 0.05 (0.09% x 47) visits/100,000 people exposed on each day when the maximum level of PM_{10} occurred, which is negligible.

The WHO recently conducted a meta-analysis of the effects of PM_{10} on cough and medication use in children and adults with chronic respiratory conditions [67]. Around 30 estimates were available for PM_{10} and cough or medication in children. The pooled odds ratio estimate is close to 1.0 for both cough and medication use with 95% confidence limits indicating non-statistical significance. In adults, there were three estimates of PM_{10} and cough or medication use. A 10 $\mu g/m^3$ increase in PM_{10} resulted in a 4.3% worsening of cough which was statistically significant (OR=1.043, 95% CI =1.005, 1.084) but was not significant for medication use [67]. The same meta-analyses also examined respiratory symptoms and lung function in children with chronic respiratory conditions. A 10$\mu g/m^3$ increase in ambient PM_{10} was associated with a 0.8% increase in lower respiratory symptoms. Based on the results of Anderson *et al.* (2004) [67] and the worst case forecast of 0.1 $\mu g/m^3$ increase in PM_{10} negligible increases in symptoms are predicted. Emissions from the vents and roadways associated with the proposed Airport Link are forecast to result in a 0.04% increase in cough for adults and 0.01% increase in lower respiratory symptoms in children with chronic respiratory conditions exposed to the worst case increase in PM_{10} (Table 32).

Long term effect on lung function growth

The forecast worst case increase in annual average PM_{10}, at Albert Bishop Park and Kedron, are not likely to have an effect on lung function growth in children. Gauderman *et al.* (2004) [5] reported that a 51.5 $\mu g/m^3$ increase in annual average PM_{10} exposure over an 8 year period was associated with an increased proportion of children with clinically low lung function. By age 18, this resulted in 6% more children having a lung function that was clinically significantly below predicted. The forecast increase in regional PM_{10} resulting from the proposed Airport Link vents and roads is 0.1 $\mu g/m^3$, which represents 0.2% of the increment recorded in the Gauderman *et al.* (2004) [5] study.

Long term effect on mortality

The forecast increase in annual average PM_{10} from the proposed Airport Link is not likely to have an effect on long term mortality, lung cancer mortality or cardiopulmonary mortality.

Pope *et al*. (2002)[99] reported a 10$\mu g/m^3$ increase in $PM_{2.5,}$ but not PM_{10}, resulted in a 4, 6 and 8% increase in total, cardiopulmonary and lung cancer mortality. The forecast increase in PM_{10} as a result of emissions from the vents and major roadways associated with the proposed Airport Link is 0.1 $\mu g/m^3$ (Table 28) is therefore not expected to have an impact on long term mortality. Even if all the forecast increase in PM_{10} was due to $PM_{2.5}$, this still equates to 1% of the increase recorded by Pope *et al*. (2002)[99].

$PM_{2.5}$

Assuming all the PM_{10} is $PM_{2.5}$ and given that HAS predict very small increases in PM_{10}, negligible health effects are predicted to occur at Albert Bishop Park, Bowen Hills, Kalinga Park or Kedron or as a result of $PM_{2.5}$ from the proposed Airport Link.

The health effects of $PM_{2.5}$ are less well understood than the effects of PM_{10}, since equipment of monitoring $PM_{2.5}$ and $PM_{2.5}$ standards have only recently been introduced into Australia. The recent studies of Simpson *et al.* (2005)[72, 85] were the first Australian epidemiological studies to quantify the relationship between $PM_{2.5}$ and health outcomes, while earlier studies inferred the concentration of $PM_{2.5}$ from nephelomoter data. Since $PM_{2.5}$ is a subset of PM_{10} it is likely that earlier studies have captured some of the effect of $PM_{2.5}$ in their estimates of the health effects of PM_{10}.

There is currently no AAQNEPM for $PM_{2.5}$, the advisory standard for 24-hour $PM_{2.5}$ is 25 $\mu g/m^3$. The maximum worst case levels of $PM_{2.5}$ resulting from portal and roadway emissions associated with the proposed Airport Link is 0.1 $\mu g/m^3$, thus the increase as a result of emissions from the vents and major roadways associated with the proposed Airport Link represents 0.4% of the $PM_{2.5}$ advisory standard.

Acute effects on hospital admissions and mortality

Simpson *et al*. (2005)[72] reported a 10 $\mu g/m^3$ increase in $PM_{2.5}$ to result in a 5.1% increase in cardiovascular admission in all ages, while there was no significant effect on total mortality. These results were from a meta-analysis of 3 Australian cities Melbourne, Sydney and Perth, however no data was available for Brisbane [72, 85]. An earlier study in Melbourne inferred the levels of $PM_{2.5}$ from measurement of bsp. Based on conversion of 1 x 10^{-4}/m bsp to 15 $\mu g/m^3$ $PM_{2.5}$, Denison *et al.* (2001)[68] reported an approximate 15 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 2.4% and 13.9% increase in all respiratory and asthma admissions, respectively.

The maximum forecast increase in $PM_{2.5}$ from emissions from the vents and roadways associated with the proposed Airport Link is predicted to result in a 0.05% increase in hospital admissions for cardiovascular diseases, a 0.09% increase in asthma and 0.02% increase in all respiratory admissions (Table 33) [68, 72]. The background daily rate of cardiovascular admissions in Brisbane is 2.19 persons/100,000, therefore it is forecast that there would be an additional 0.001 persons per day admitted to hospital for cardiovascular diseases per 100,000 people exposed to the worst case increase in $PM_{2.5}$ on the days when this worst case occurs (Table 33).

Table 33: Potential increases in the background daily rate of health events as a result of the largest forecast increase in $PM_{2.5}$.

Largest potential maximal increase in 24-hour $PM_{2.5}$ ($\mu g/m^3$)	Adverse health event	Most conservative estimate of percentage increase on maximum PM_{10} pollution day[1,2]	Background daily event rate	Projected increment in rate on maximum PM_{10} pollution day
0.1	Cardiovascular Admissions (all ages)	0.05%[1]	2.19 persons/100,000 people	0.001 persons/ 100,000 people exposed to the worst case increase.
0.1	Asthma admission (all ages)	0.09%[2]	0.72 persons/100,000 people	0.001 persons/ 100,000 people exposed to the worst case increase.
0.1	Respiratory Admissions (all ages)	0.02%[2]	3.26 persons/100,000 people	0.001 persons/ 100,000 people exposed to the worst case increase.

[1] Estimates from meta-analysis of Melbourne, Perth and Sydney[72, 85].
[2] As published for Melbourne [68].

Acute effects on symptoms

There have been no Australian studies on the acute effect of $PM_{2.5}$ on symptoms.

Long term effect on mortality

The forecast increase in annual average $PM_{2.5}$ as a result of emission from the vents and roadways associated with the proposed Airport Link is unlikely to have an effect on long term mortality, lung cancer mortality or cardiopulmonary mortality.

Pope et al. (2002)[99] reported a 10$\mu g/m^3$ increase in $PM_{2.5,}$ but not PM_{10}, resulted in a 4, 6 and 8% increase in total, cardiopulmonary and lung cancer mortality. The forecast increase $PM_{2.5}$ as a result of emissions from the vents and major roadways associated with the proposed Airport Link equates to 1% of the increase recorded by Pope et al. (2002)[99]. The 0.1 $\mu g/m^3$ forecast increase in $PM_{2.5}$ (Table 28) is therefore not expected to have an impact on long term mortality.

Toluene

At Bowen Hills and Kedron both maximum 24-hour and annual average toluene concentrations in 2012 are forecast to decrease as a result of the proposed Airport Link (Table 29). At Eagle Farm 4 x 10^{-5} mg/m^3 increase in ambient 24 hour toluene is forecast (Table 29), which is equivalent to 0.001% of the 24-hour AAQ NEPM Monitoring Investigational Levels. At Kedron a 4.7x 10^{-5} mg/m^3 increase in annual average toluene is predicted (Table 29), which is equal to 0.012% of the annual average toluene AAQ NEPM Monitoring Investigational Levels. The forecast increases in ambient maximum 24-hour and annual average toluene concentrations are equivalent to 4% and 24% of the respective toluene concentrations currently recorded at Eagle Farm.

The NEPC[100] reported that a concentration of 100ppm (375 mg/m^3) of toluene was the lowest concentration where observable impaired reaction time, symptoms of headache, dizziness, a feeling of slight intoxication (CNS depressant effects), eye and nose irritation were reported. At 40 ppm (150 mg/m^3) both irritant effects and CNS effects were not observed after 6 hours of exposure[100]. The no–effect level of 40 ppm (150 mg/m^3), was converted to a 24-hour concentration of 20 ppm (75 mg/m^3) and an uncertainly factor of 10 applied resulting in the AAQ NEPM MIL of 2 ppm (7.5 mg/m^3) which is 190,000 times higher than the worst case forecast increase from the proposed Airport Link, suggesting that toluene emissions form Airport Link are unlikely to have a known impact on health.

Xylene

At Bowen Hills both maximum 24-hour and annual average xylene concentrations in 2012 are forecast to decrease as a result of the proposed Airport Link (Table 29). The largest increase in 24-hour xylene is at Eagle Farm and is 2.7 x 10^{-5} mg/m^3, which is 0.003% of the NEPM Monitoring Investigational Level (MIL). At Kedron a 3.5 x 10^{-5} mg/m^3 increase in annual average xylene is predicted, which is the largest forecast increase and equivalent to 0.004% of the AAQ NEPM MIL. The forecast increases in ambient maximum 24-hour xylene concentration is equal to 3.7% of the concentration currently recorded at Eagle Farm.

The NEPC[100] reported that exposure to 100ppm (43 mg/m^3) for 30 minutes resulted in no evidence of respiratory or eye irritation. The no–effect level of 100 ppm (43 mg/m^3) was converted to a 24-hour concentration of 2ppm (8.68 mg/m^3) and an uncertainly factor of 10 applied resulting in the AAQ NEPM MIL of 0.2 ppm (0.87 mg/m^3) which is 32,000 times higher than the worst case forecast increase from the proposed Airport Link, suggesting that xylene emissions form Airport Link are unlikely to have a known impact on health.

Section C2: Health Effects Resulting from Major Roads associated with the proposed Airport Link

HAS provided the forecast levels of emissions from vehicles travelling on the major roads associated with the proposed Airport Link (see HAS Report, Figures 51-61). The forecast increases were provided for ground level receptors located nearby 11 major roads (Table 34) for 2012, 2016 and 2026. The results presented are the incremental increase in ambient pollutants as a result of the increased emissions from traffic using the roads impacted upon by the proposed Airport Link and do not include background levels of pollutants or pollutants from other sources such as the proposed Northern Busway. This enables an assessment of the health effects as a result of the increases in pollutants from major roads associated with the proposed Airport Link.

In all cases the increase in ambient air pollutants resulting from the major roads, associated with the proposed Airport Link, are well below the AAQ NEPMs (Table 34), which are designed to protect the community. There is, however, no lower limit

below which an adverse health impact will not occur; therefore an incremental increase in a pollutant can have an impact on health even though it is below the AAQNEPM. It should be noted, however, that for all the pollutants assessed: CO, NO_2, PM_{10} and $PM_{2.5}$ the maximum roadside levels that are forecast to occur as result of the proposed Airport Link are well below the maximum levels currently recorded around Brisbane (Table 34 compared with Tables 3 -7). Under worst case conditions five of the eleven locations are forecast to record reductions in ambient roadside CO and NO_2 and $PM_{10}/PM_{2.5}$.

Table 34: Maximum predicted roadside concentrations of CO, NO_2, PM_{10} and $PM_{2.5}$ for major roads associated with the Airport Link. From HAS.

	Road (modeled distance from road)	Year(s) in which worst case is forecast to occur year(s)	CO 8-hr mg/m3	NO_2 1-hr max μg/m3	NO_2 annual ave. μg/m3	$PM_{10}/PM_{2.5}$ 24 hr max. μg/m3	$PM_{10}/PM_{2.5}$ annual ave. μg/m3
Air Quality Goals (AAQ NEPM)			**10**	**246**	**60**	**50/25**	**-/8**
	Forecast worst case changes in roadside ambient air pollutants. From HAS Report.						
03	Bowen Br Road (10m)	2012/2016/2026	**-0.14**	**-15.24**	**-3.80**	**-1.19**	**-0.37**
04	Lutwyche & Gympie Rds Interchange (30m)	2026	**0.15**	**14.36**	**3.69**	**1.29**	**0.48**
05	Gympie Road (10m)	2026	0.21	15.32	5.80	1.72	0.72
11	Sandgate Road S (10m)	2012	**-0.14**	**-12.59**	**-3.37**	**-0.97**	**-0.32**
12	Sandgate Road N (10m)	2012//2026	**-0.08**	**-6.23**	**-1.62**	**-0.58**	**-0.18**
38	Stafford Road E(10m)	2026	0.15	11.73	3.76	1.22	0.46
44	Newmarket Road(10m)	2012/2016	**-0.09**	**-6.63**	**-2.31**	**-0.61**	**-0.24**
50	Gateway Motorway N(30m)	2012/2016	0.02	3.54	0.80	0.15	0.07
52	East-west Arterial(10m)	2012/2016/2026	0.15	9.30	2.64	0.75	0.26
53	Airport Drive(10m)	2016/2026	0.03	1.36	0.55	1.72	0.60
54	Lutwyche Road N. Maygar (10m)	2122	-0.25	-20.28	-5.67	-1.32	-0.38
# sites where the worst case is a decrease			5 of 11	5 of 11	5 of 11	5 of 11	5 of 11
Net change across all modeled roads			0.00	-5.35	0.47	2.18	1.10

Numerous studies have demonstrated that living by busy roads has an adverse impact on health [120-126].

The changes in air pollutant concentrations reported in Table 34 are at distances close to the main roads (10-30m), however in assessment of the health effects the location of any sensitive receptors such as childcare centres, schools, nursing homes or aged care facilities was also considered (Table 35).

Table 35: Distances of sensitive receptors from the main roads associated with Airport Link. Source: Personal communication from SKM, 2006.

Facility	Use	Road	Distance (m)
ABC Developmental Learning Centre (Nundah)	CHILD CARE CENTRE	Sandgate Road	128
ABC Developmental Learning Centre (Hendra)	CHILD CARE CENTRE	Sandgate Road	1100
Aberleigh Child Care	CHILD CARE CENTRE	Lutwyche Road	818
Albion Peace Centre	AGED CARE	Sandgate Road	228
Allambe Mercy Centre	AGED CARE	Lutwyche Road	750
Amarina Nursing Home and Support Centre	AGED CARE	Lutwyche Road	41
Arthritis Foundation	AGED CARE	Lutwyche Road	10
Ascot Hendra Child Care and Nursery Centre	CHILD CARE CENTRE	Sandgate Road	1216
C & K's Kelvin Grove Community Childcare Centre	CHILD CARE CENTRE	Lutwyche Road	1026
Clayfield Capers Child Care Centre	CHILD CARE CENTRE	Sandgate Road	400
Clayfield Childhood Development	CHILD CARE CENTRE	Sandgate Road	370
Clayfield College	SCHOOL	Sandgate Road	10
Clayfield Early Learning Centre	CHILD CARE CENTRE	Sandgate Road	700
Clayfield Kindergarten and Preschool	CHILD CARE CENTRE	Sandgate Road	550
Clifford House Aged Care	AGED CARE	Sandgate Road	780
Denmora/ River Breeze Manor Nursing Home	AGED CARE	Lutwyche Road	1200
Eagle Junction School	SCHOOL	Sandgate Road	380
Fagan Childcare	CHILD CARE CENTRE	Lutwyche Road	830
Gordon Park Day Respite Centre	AGED CARE	Lutwyche Road	430
Hendra College	SCHOOL	East West Arterial	135
Herston First Steps Child Care Centre	CHILD CARE CENTRE	Lutwyche Road	817
Holy Cross Primary School	SCHOOL	Lutwyche Road	590
Holy Rosary School	SCHOOL	Lutwyche Road	24
Hutchinsons Early Child Care	CHILD CARE CENTRE	Gympie Road	230
Kedron Heights Pre-School	CHILD CARE CENTRE	Gympie Road	520
Kedron Park Rd Child and Development Centre	CHILD CARE CENTRE	Lutwyche Road	390
Kedron State High	SCHOOL	Gympie Road	226
Kedron State School	SCHOOL	Gympie Road	260
Loosends Outside School Hours Care	CHILD CARE CENTRE	Sandgate Road	80
Nundah Kindergarten	CHILD CARE CENTRE	Sandgate Road	320
Nundah State School	SCHOOL	Sandgate Road	95
Prentice Park Kindergarten	CHILD CARE CENTRE	Lutwyche Road	110
Regis Crana Respite Centre	AGED CARE	Lutwyche Road	1070
Rosemount Hospital	HOSPITAL	Lutwyche Road	10
Royal Brisbane Hospital	HOSPITAL	Bowen Bridge Road	10
Sisters of Mercy Complex	AGED CARE	Lutwyche Road	610
St James Nursing Home	AGED CARE	Sandgate Road	710
St Paul's Lutheran Child Care Centre	CHILD CARE CENTRE	Sandgate Road	208

Wagner Rd Early Childhood Centre	CHILD CARE CENTRE	Sandgate Road	107
Windsor Neighbourhood Child Care	CHILD CARE CENTRE	Lutwyche Road	155
Windsor Primary School	SCHOOL	Lutwyche Road	10
Wooloowin Nursing Home	AGED CARE	Lutwyche Road	270
Wooloowin Primary School	SCHOOL	Lutwyche & Gympie Road interchange	25
YMCA Child Care Centre	CHILD CARE CENTRE	Lutwyche Road	10
Zion Lutheran Nursing Home	AGED CARE	Sandgate Road	158

CO

Five of the 11 ground level roadside receptors that were selected by HAS for modelling are predicted to record an increase in CO (Table 34). The highest 8-hour level of CO is forecast to be 0.21 mg/m^3 in 2026 at a distance of 10m from Gympie Road (Table 34). This is equivalent to 4.6% of the highest and 53% of the median level of CO level recorded at the EPAs Woolloongabba monitoring site in 2004 (Table 3). The worst case increase in 8-hour average CO level resulting from the Airport Link roadways is equal to 2.1% of the AAQ NEPM of 10 mg/m^{3}and when combined with background ambient level in 2004 of 0.59 mg/m^3 (HAS Report), results in a total ambient level of 0.80 mg/m^3, which is 8 % of the AAQ NEPM.

Acute effects on hospital admissions and mortality

In assessing the worst case health effects on the community resulting from CO emissions from major roads associated with the proposed Airport Link, the maximum increase in 8-hour CO was used, which was 0.21 mg/m^3 (Table 34). The recent analysis of all four Australian cities by Simpson *et al.* (2005) [72, 85] did not present data for CO. There have been no peer reviewed studies that have published an association between CO and hospital admissions in Brisbane. The Victorian EPA study, predicts that a 0.21 mg/m^3 increase in 8-hour CO would result in 0.16%,1.13% and 0.49% increases in the background risk for hospital admission for respiratory diseases, asthma and cardiovascular diseases, respectively, in all ages on each day when this maximum increase in ambient 8-hour maximum CO occurred (Table 36).

The background daily risk for hospital admissions for respiratory diseases, asthma and cardiovascular diseases in Brisbane was reported to be 3.26 persons/100,000 people, 0.72 persons/100,000 people and 2.19 persons/100,000 people, respectively (Table 36). The incremental increase in hospital admissions for all respiratory diseases, asthma and cardiovascular diseases as a result of the maximum CO emitted from the Airport Link tunnel ventilation system is therefore 0.005 (0.16% x 3.26), 0.008 (1.13% x 0.72) and 0.011 (0.49% x 2.19) persons/100,000 exposed population on each day when the maximum CO level occurs (Table 36). These small effects are negligible increases in health risk.

The forecast maximum change in CO would result in a 0.35 % increase in cardiovascular mortality on each day when the maximum CO level occurs (Table 36). The background risk for daily mortality is 0.92 persons/100,000 people. Therefore, on the days when the maximum increase in CO occurred, it would result in an

incremental increase in mortality of 0.0032 (0.35% x 0.92) persons/100,000 people (Table 36), which is a negligible increase in health risk.

Table 36: Potential increases in the background daily rate of health events as a result of the largest forecast increase in CO as a result of the roadside emissions from the Airport Link.

Largest potential maximal increase in pollutant 8-hour CO (mg/m^3)	Adverse health event	Most conservative estimate of percentage increase in health effect on maximum CO pollution day	Background daily event rate	Projected increment in rate on maximum CO pollution day
0.21	Respiratory admission (all ages)	0.19%[1]	3.26 persons/100,000 people[2]	0.005 persons/100,000 people exposed to the worst case increase
0.21	Asthma admission (all ages)	1.13%[1]	0.72 persons/100,000 people[2]	0.008 persons/100,000 people exposed to the worst case increase.
0.21	Cardiovascular Admissions (all ages)	0.49%[1]	2.19 persons/100,000 people[3]	0.011persons/100,000 people exposed to the worst case increase.
0.21	Cardiovascular mortality (all ages)	0.35%[1]	0.92/100,000 people[3]	0.003 persons/100,000 people exposed to the worst case increase.

[1] Epidemiological studies of the effect of CO on health in Melbourne from the EPA Studies [68, 86]
[2] Epidemiological studies of the effect of air pollution on health in Brisbane[70]
[3] Epidemiological studies of the effect of air pollution on health in Brisbane[72, 85]

Acute effect on symptoms

The impact of small increases in CO on symptoms or respiratory and cardiovascular function has not been extensively researched and there are no Australian panel studies or meta-analysis of these effects of CO on the above. Chamber studies of acute exposure to CO were used to set the WHO thresholds for CO, which are designed to maintain COHb below 2.5% (Table 8). The WHO 8 hour guideline of 10 ppm (11.6 mg/m^3) is well above the forecast concentration of 0.8 mg/m^3 (background plus 0.21 mg/m^3 increase) in CO reported in Table 34. Therefore acute clinical effects of roadside CO exposure on major roads associated with the Airport Link are not expected.

NO_2

Acute effects on hospital admissions and mortality

The predicted maximum increase of 15.32 $\mu g/m^3$ for 1-hour maximum NO_2 10 m from Gympie Road is equivalent to 11% of the highest 1 hour maximum recorded at the EPA's Brisbane CBD monitoring station in 2004 (Table 4) and 50% of the highest median 1 hour maximum recorded at the South Brisbane monitoring station.

Previous research in Brisbane has found associations between changes in 1-hour maximum NO_2 concentration and both hospital admissions and mortality [70, 91]. The meta-analyses of 4 Australian cities, including Brisbane found that an increase in ambient NO_2 was significantly associated with increases in total, cardiovascular and respiratory mortality (Table 18) [72]. Hospital admissions for cardiovascular and respiratory diseases were also significantly associated with increases in 1 hour maximum ambient NO_2 across the 4 cities (Table 15) [85]. An earlier study in Melbourne reported a significant association between NO_2 and asthma [68].

The maximum predicted increase in 1-hour maximum in NO_2 as a result of emissions from major roads associated with the Airport Link is forecast to be 15.32$\mu g/m^3$ (Table 34), which using the Australian 4-cities meta-analysis data [72], is forecast increase mortality by 0.9% on the days when the actual worst case occurs (Table 37). Hospital admissions for cardiovascular disease in all ages, respiratory disease in people aged 65 and over and asthma in all ages, are forecast to increase by 1.7%, 2.0% and 4.5%, respectively (Table 37) [68, 85]. These predictions are supported by separate epidemiological studies conducted in Sydney and Perth which have also shown positive associations between NO_2 and hospital admissions for respiratory and cardiovascular diseases and mortality [69, 71, 73].

The forecast incremental increase in hospital admissions for cardiovascular diseases, respiratory admissions in people aged 65 and over and asthma are negligible and equal to 0.038 (1.7% x 2.19), 0.018 (2.20% x 0.88) and 0.032 (4.5% x 0.72) persons per 100,000 people exposed to the maximum increase per day on the days when the maximum increases in NO_2 occurs (Table 37). The incremental increase in mortality is forecast to be 0.018 person/100,000 people exposed to the worst case increase in NO_2, which is a negligible increase in health risk.

Table 37: Potential increases in the background daily rate of health events as a result of the largest forecast increase in NO_2 exposure from emissions form major roads associated with the Airport Link.

Largest potential maximal increase 1-hour NO_2 (mg/m^3)	Adverse health event	Most conservative estimate of percentage increase on maximum NO_2 pollution day	Background daily event rate	Projected increment in rate on maximum NO_2 pollution day
15.32	Cardiovascular Admissions (all ages)	1.7%[2]	2.19 persons/100,000 population[2]	0.038 persons/100,000 people exposed to the worst case increase.
15.32	Asthma admission (all ages)	4.5%[1]	0.72 persons/100,000 population[3]	0.032 person /100,000 people exposed to the worst case increase.
15.32	Respiratory Admissions (65+ years)	2.0%[2]	0.88 persons/100,000 population[2]	0.018 persons/100,000 people exposed to the worst case increase.
15.32	Mortality (all ages)	0.9%[2]	1.99/ persons/100,000 people[2]	0.018 persons/100,000 people exposed to the worst case increase.

[1] Melbourne estimates as published by Denison *et al*. (2001)[68]
[2] Brisbane estimates [72, 85]
[3] Brisbane estimate [70].

Acute effect on symptoms

Less severe and more common health effects are changes in lung function, symptoms or medication use. Jalaludin *et al* (2000 and 2004)[64, 65] found no association between NO_2 and lung function, wheeze, dry cough, medication use, doctor visits for asthma, but a significant association with wet cough. This study was conducted in western and southwestern Sydney. The study followed 125 children, aged 10, with a history of wheeze for an 11 month period in 1994. A 0.0082 ppm increase in average daytime NO_2 was associated with an 5% increase in parent reported wet cough, which was statistically significant ($p<0.05$) [64]. Unfortunately NO_2 was measured as average daytime NO_2, not maximum 1 hour NO_2. The relationship between symptoms and an increase in 1 hour maximum NO_2, as forecast to result from emission from major roads associated with the proposed Airport Link, is unknown.

Long term effect on lung function growth

A long term impact of elevated levels of NO_2 was found in the Southern California Children's Health Study (SCCHS) [5]. A 71.5 $\mu g/m^3$ (34.6 ppb) increase in annual 24 hour NO_2, over an eight year period, resulted in a 6% increase in the number of 18 year olds who had a clinically significant lower lung function (FEV_1 less than 80% of predicted). The location of child care centres and schools is therefore an important

consideration (Table 37) in assessing the potential long term impact of changes to roadside annual NO_2 associated with roads surrounding the proposed Airport Link.

The worst case maximum increase in roadside annual NO_2 as a result of the major roads associated with the proposed Airport Link was forecast to occur 10m from Gympie Road and was 5.80 $\mu g/m^3$ (~3 ppb) (Table 34), which is approximately 8% of the increment recorded in the SCCHS. While the forecast worst case increase in ambient roadside annual average NO_2 from Airport Link represents a small increase in comparison to the SCCHS[5], it may be of concern for children who are living for a number of years within 10m of Gympie Road or close to other roads where increases in annual average NO_2 are forecast.

The health effect of the forecast increase in annual average NO_2 from Airport Link is difficult to predict, since the relationship between long term lung function growth and increases in ambient NO_2 is confounded by other pollutants[4] and is not sufficiently precise to accurately predict the effect of a small change in NO_2. In the SCCHS where there was a 71.5 $\mu g/m^3$ difference between the lowest and highest communities only 56% of the variation in lung function growth over the 8 year period was accounted for by the concentration of NO_2, the effect was however statistically significant.

A major concern regarding long term effects on children's health is the proximity of schools and child care centres to roads where a worsening in long term air pollution is forecast. There are no schools or childcare centres close to Gympie Road, the nearest being more than 200m from this road (Table 35). Wooloowin Primary School is 25m from the Lutwyche Road and Gympie Road interchange and forecast to have an increase in annual average NO_2 of approximately 3.69 $\mu g/m^3$ (1.8 ppb), which is 5% of the increment recorded in the SCCHS. Other schools and childcare centres that are close (10-30m) to major roadways associated with Airport Link are forecast to have an improvement in air quality, which may translate to improvements in lung function growth of children.

Long term effect on mortality

Pope et al. (2002) [99] did not find a relationship between long term exposure to elevated levels of NO_2 and increase in total, cardiopulmonary and lung cancer mortality. On the basis of Pope's work [99] there is not forecast to be an increase in long term mortality as a result of changes to roadside ambient NO_2 on roads associated with the proposed Airport Link.

PM_{10}

The maximum predicted increase in roadside ambient PM_{10} concentration resulting on the major roads associated with the Airport Link is 1.72 $\mu g/m^3$ 10m from Gympie Road (Table 34). The AAQNEPM for 24-hour PM_{10} is 50 $\mu g/m^3$, thus the increase as a result of the Airport Link represents 3.4% of the PM_{10} standard.

Acute effects on hospital admissions and mortality

The maximum forecast increase in PM_{10} is predicted to result in a small increase in hospital admissions for all respiratory diseases (0.51%), cardiovascular diseases (0.41%) and respiratory diseases in people over 65 (0.49%) and a 0.31% increase in total mortality [70, 72, 85] on the days when this maximum increase actually occurs (Table 38). The background daily rate of these health events is small, therefore only very small increases in these events are forecast (0.004-0.009 per 100,000 people) (Table 38).

Table 38: Potential increases in the background daily rate of health events as a result of the largest forecast increase in PM_{10}.

Largest potential maximal increase in 24-hour PM_{10} ($\mu g/m^3$)	Adverse health event	Most conservative estimate of percentage increase on maximum PM_{10} pollution day[1,2]	Background daily event rate	Projected increment in rate on maximum PM_{10} pollution day
1.72	Cardiovascular Admissions (all ages)	0.41%[1]	2.19 persons/100,000 people[1]	0.009 persons/ 100,000 people exposed to the worst case increase.
1.72	Respiratory Admissions (65+ years)	0.49%[1]	0.88 persons/100,000 people[1]	0.004 persons/ 100,000 people exposed to the worst case increase.
1.72	Respiratory Admissions (all ages	0.51%[4]	3.26 persons/100,000 people[4]	0.004 persons/ 100,000 people exposed to the worst case increase.
1.72	Total mortality (all ages)	0.31%[5]	1.99[1] persons/100,000 people	0.006 persons/ 100,000 people exposed to the worst case increase.
1.72	Visits to doctor for asthma	1.50%[2]	47/100,000 people	0.71 visits/100,000 people exposed to the worst case increase.
1.72	Lower respiratory symptoms in symptomatic children	0.14%	N/A	
1.72	Cough in adults	0.73%	N/A	

[1] As published for the 4 cities meta-analysis by Simpson et al. Simpson et al (2005a, 2005b) [72, 85].
[2]Sydney estimates as published by Jalaludin et al. (2004)[64].
[3] As published by WHO (2004)[67].
[4] Petroechevsky et al (2001) for Brisbane[70]
[5] Simpson et al (1997)for Brisbane [80]

Acute effects on symptoms

Jalaludin *et al.* (2000 and 2004)[64, 65] found no association between PM_{10} and lung function, wheeze, cough or medication use, but a significant association with doctor visits for asthma. A 12 $\mu g/m^3$ increase in 24 hour PM_{10} was associated with an 11% increase in visits to the doctor for asthma, which was statistically significant ($p<0.05$) [64]. Based on the results of Jalaludin *et al.* (2004) [64] and the worst case forecast 1.72

$\mu g/m^3$ increase in ambient roadside PM_{10} on Gympie Road would be expected to result in a 1.50% increase in doctor attendances for asthma in children exposed to the worst case increase in PM_{10} (Table 38). The daily rate of GP attendances for asthma is around 47 per 100,000 people. Therefore, the increased risk for GP attendance for asthma as a result of the worst case increase in 24 hour PM_{10} from the roadway emission associated with the Airport Link is 0.71 (1.50% x 47) visits/100,000 people exposed on each day when the maximum level of PM_{10} occurred.

The WHO recently conducted a meta-analysis of the effects of PM_{10} on cough and medication use in children and adults with asthma [67]. Around 30 estimates were available for PM_{10} and cough or medication in children. The pooled odds ratio estimate is close to 1.0 for both cough and medication use with 95% confidence limits indicating non-statistical significance. In adults, there were three estimates of PM_{10} and cough or medication use. A 10 $\mu g/m^3$ increase in PM_{10} resulted in a 4.3% worsening of cough which was statistically significant (OR=1.043, 95% CI =1.005, 1.084) but was not significant for medication use [67]. The same meta-analyses also examined respiratory symptoms and lung function in children with chronic respiratory conditions. A 10$\mu g/m^3$ increase in ambient PM_{10} was associated with a 0.8% increase in lower respiratory symptoms. Based on the results of Anderson et al. (2004) [67] and the worst case forecast increase in roadside ambient 24 hour PM_{10} of 1.72 $\mu g/m^3$ at a distance of 10 m from Gympie Road, a 0.73% increase in cough for adults and a 0.14% increase in lower respiratory symptoms in children with chronic respiratory conditions is forecast (Table 38). These small increases in symptoms are not likely to be clinically significant.

Long term health effects

The forecast worst case increase in annual average PM_{10} for 2026 10m from Gympie Road as a result of the proposed Airport Link is 0.72 $\mu g/m3$ (Table 34) and is not expected to have an effect on lung function growth in children. Gauderman *et al*. (2004) [5] reported that a 51.5 $\mu g/m^3$ increase in annual average PM_{10} exposure over an 8 year period was associated with an increased proportion of children with clinically low lung function. By age 18, this resulted in 6% more children having a lung function that was clinically significantly below predicted. The forecast increase of 0.72 $\mu g/m^3$ in PM_{10} for resulting from the proposed Airport Link represents 0.9% of the increment recorded in the Gauderman *et al*. (2004) [5] study. Based on Gauderman's results for Southern California and HAS worst case predicted PM_{10}, the percentage of children living within 10m of Gympie Road that would be forecast to have clinically reduced lung function as a result of the modelled increase in annual PM_{10} from emission from the proposed Airport Link is likely to be very small.

Long term effect on mortality

The forecast increase in PM_{10} for 2036 as a result of emission from the proposed Airport Link is not likely to have an effect on long term mortality, lung cancer mortality or cardiopulmonary mortality.

Pope et al. (2002)[99] reported a 10$\mu g/m^3$ increase in $PM_{2.5,}$ but not PM_{10}, resulted in a 4, 6 and 8% increase in total, cardiopulmonary and lung cancer mortality. The

forecast increase in PM_{10} for 2035 as a result of emission from the proposed Airport Link is 0.72 $\mu g/m^3$ (Table 34) is therefore not expected to have an impact on long term mortality. Even if all the forecast increase in PM_{10} was due to $PM_{2.5}$, this still equates to 7.2% of the increase recorded by Pope et al. (2002)[99].

$PM_{2.5}$

The health effects of $PM_{2.5}$ are less well understood than the effects of PM_{10}, since equipment of monitoring $PM_{2.5}$ and $PM_{2.5}$ standards have only recently been introduced into Australia. The recent studies of Simpson *et al.* (2005)[72, 85] \were the first Australian epidemiological studies to quantify the relationship between $PM_{2.5}$ and health outcomes, while earlier studies inferred the concentration of $PM_{2.5}$ from nephelomoter data. Since $PM_{2.5}$ is a subset of PM_{10} it is likely that earlier studies have captured some of the effect of $PM_{2.5}$ in their estimates of the health effects of PM_{10}.

There is currently no AAQNEPM for $PM_{2.5.}$, The advisory standard for 24-hour $PM_{2.5}$ is 25 $\mu g/m^{3.}$

HAS did not model the ambient roadside concentration of $PM_{2.5}$ resulting from the proposed Airport Link, however consistent with our conservative approach it can be assumed that all PM_{10} is as a result of $PM_{2.5}$. The maximum increase in $PM_{2.5}$ is forecast to be 1.72 $\mu g/m^3$ (Table 34) at a distance of 10 m from Gympie Road. The highest forecast increase in $PM_{2.5}$ on the major roads associated with the Proposed Airport Link represents 6.9% of the $PM_{2.5}$ AAQ NEPM advisory standard.

Acute effects on hospital admissions and mortality

Simpson *et al.* (2005) reported a 10 $\mu g/m^3$ increase in $PM_{2.5}$ to result in a 5.1% increase in cardiovascular admissions in all ages, while there was no significant effect on total mortality. These results were from a meta-analysis of 3 Australian cities Melbourne, Sydney and Perth, however no data was available for Brisbane [72, 85]. An earlier study in Melbourne inferred the levels of $PM_{2.5}$ from measurement of bsp. Based on conversion of 1 x 10^{-4}/m bsp to 15 $\mu g/m^3$ $PM_{2.5}$, Denison *et al.* (2001) reported an approximate 15 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 2.4% and 13.9% increase in all respiratory and asthma admissions, respectively.

The maximum forecast increase in ambient roadside $PM_{2.5}$ around roadways associated with Airport Link is predicted to result in a 0.86% increase in hospital admissions for cardiovascular diseases, a 1.51% increase in asthma and 0.27% increase in all respiratory admissions (Table 39) [68, 72]. The background daily rate of cardiovascular admissions in Brisbane is 2.19 persons/100,000, therefore it is forecast that there would be an additional 0.019 persons per day admitted to hospital for cardiovascular diseases per 100,000 people exposed to the worst case increase in $PM_{2.5}$ on the days when this worst case occurs (Table 39).

Table 39: Potential increases in the background daily rate of health events as a result of the largest forecast increase in $PM_{2.5}$.

Largest potential maximal increase in 24-hour $PM_{2.5}$ ($\mu g/m^3$)	Adverse health event	Most conservative estimate of percentage increase on maximum PM_{10} pollution day[1,2]	Background daily event rate	Projected increment in rate on maximum PM_{10} pollution day
1.72	Cardiovascular Admissions (all ages)	0.86%[1]	2.19 persons/100,000 people[1]	0.019 persons/ 100,000 people exposed to the worst case increase.
1.72	Asthma admission (all ages)	1.51%[2]	0.72 persons/100,000 people[3]	0.011 persons/ 100,000 people exposed to the worst case increase.
1.72	Respiratory Admissions (all ages)	0.27%[2]	3.26 persons/100,000 people[3]	0.009 persons/ 100,000 people exposed to the worst case increase.

[1] Melbourne, Perth and Sydney estimates from Simpson *et al.* (2005a, 2005b) [72, 85].
[2] Melbourne estimates as published by Denison *et al*. (2001)[68],
[3] Brisbane estimate as published by Petroeschevsky *et al.* (2001)[70]

Acute effects on symptoms

There have been no Australian studies on the acute effect of $PM_{2.5}$ on symptoms and very few international studies[67].

Long term effect on mortality

The forecast worst case increase in ambient roadside annual average $PM_{2.5}$ for 2026 at a distance of 10m from Gympie Road is 0.72$\mu g/m^3$. Pope et al. (2002)[99] reported a 10$\mu g/m^3$ increase in annual average $PM_{2.5}$, resulted in a 4, 6 and 8% increase in total, cardiopulmonary and lung cancer mortality over an 18 year period. The forecast worst case increase in roadside $PM_{2.5}$ in 2026 as a result of the proposed Airport Link is therefore likely to have a small impact on long term mortality, this equates to 7.2% of the increase recorded by Pope et al. (2002)[99]. The resultant increase in long term mortality is equivalent to 0.28% for the people living within 10m of Gympie Road.

Appendix A

Table A1: Summary statistics for daily hospital admissions and particulate air pollutant concentrations for the meta-analysis (years 1996-99)[72].

	Brisbane Mean (range)	Sydney Mean (range)	Melbourne Mean (range)	Perth Mean (range)
Daily hospital admissions				
Cardiac				
All ages	17.7 (1-44)	68.8 (37-113)	55.7 (26-91)	24.6 (8-43)
≥65 years	12.6 (0-34)	44.8 (21-77)	37.1 (15-60)	17.1 (3-32)
15-64 years	5.1 (0-17)	23.7 (9-45)	18.5 (4-38)	7.3 (0-17)
IHD				
All ages	9.6 (0-24)	33.8 (15-62)	30.2 (14-55)	12.9 (2-27)
≥65 years	6.6 (0-20)	20.8 (6-44)	18.9 (6-36)	8.5 (1-20)
Stroke				
≥65 years	3.1 (0-15)	12.2 (2-31)	11.0 (3-24)	4.4 (0-12)
Total respiratory				
≥65 years	7.1 (0-28)	31.3 (9-88)	24.9 (4-66)	10.5 (1-32)
Asthma				
15-64 years	2.2 (0-15)	8.6 (0-29)	6.3 (0-24)	2.6 (0-13)
Asthma + COPD				
≥65 years	3.1 (0-15)	15.1 (1-41)	10.2 (0-30)	5.2 (0-18)
Pneumonia + acute bronchitis				
≥65 years	2.6 (0-12)	10.3 (0-33)	10.1 (0-30)	3.1 (0-12)
Pollutant levels				
CO (ppm) 8 hr	0.74[c] (0.02-4.17)	0.95 (0.02-4.62)	1.0 (0.07-6.95)	1.19 (0.10-4.25)
NO_2 (ppb) 1 hr max.	21.43(2.05-63.31)	23.66(6.54-59.38)	23.65(4.41-66.70)	16.33(1.87-41.0)
bsp ($10^{-4}m^{-1}$) 24 hr	0.26(0.02-2.49)	0.25(0.04-1.58)	0.26(0.03-2.23)	0.25(0.07-1.76)
PM_{10}($\mu g/m^3$) 24 hr	16.5(2.6-57.6)	16.3(3.7-75.5)	18.2(3.3-51.9)	
$PM_{2.5}$($\mu g/m^3$) 24 hr	7.5[b](1.9-19.7)	9.0[a](2.4-35.3)	9.3[a](2.7-35.1)	9.0(2.8-37.3)
O_3 (ppb) 1 hr	30.95(2.85-111.50)	29.55(3.15-110.97)	24.35(1.62-96.0)	33.78(13.0-105.0)

Notes:
(a) More than 25% missing data.
(b) More than 40% missing data.
(c) Only one monitor and more than 10% missing data.

Table A2: Daily hospitalisations – mean and range for the earlier studies on individual cities[68-71].

Outcome	ICD 9	Mean	Range
Brisbane			
Asthma 0-14 years	493	2.6	0-18
Asthma – All	493	4.5	0-23
Respiratory – 65+ years	460-519	3.1	0-21
Respiratory – All	460-519	11.5	1-35
Cardiovascular - All	390-459	14.1	2-36
Sydney			
Asthma 0-14 years	493	15.5	1-76
Asthma – 15-64 years	493	9.0	0-27
COPD – 65+ years	490-492, 494, 496	9.7	0-33
Cardiovascular - All	410, 413, 427, 428	47.2	17-89
Melbourne			
Asthma 0-14 years	493	9.65	0-29
Asthma – All	493	18.47	3-52
Respiratory – 65+ years	460-519	24.08	8-68
Respiratory – All	460-519	65.87	21-132
Cardiovascular - All	390-459	84.04	47-129
Perth			
Asthma 0-14 years	493	5.6	2-10 (10^{th}-90^{th} percentiles)
Asthma – All	493	8.8	4-15 (10^{th}-90^{th} percentiles)
Other COPD	490-492, 494, 496	3.3	1-6 (10^{th}-90^{th} percentiles)
Respiratory – All	460-519	25.3	14-38 (10^{th}-90^{th} percentiles)
Cardiovascular - All	390-459	26.5	19-35 (10^{th}-90^{th} percentiles)

Table A3: Mean daily pollutant concentrations (network average), Melbourne, July 1994 – December 1997

	Whole study period				Cool season[a]				Warm season[b]			
	Mean	SD	Min	Max	Mean	SD	Min	Max	Mean	SD	Min	Max
O_3 (ppb)												
8 hour	21.79	8.89	0.99	77.57	19.57	6.07	0.99	56.14	25.07	11.13	11.14	77.57
4 hour	24.65	9.99	2.01	87.86	22.28	6.10	2.01	63.00	28.18	13.13	11.57	87.86
1 hour	26.35	11.11	2.00	97.57	23.66	6.07	2.00	67.57	30.35	15.02	10.73	97.57
Particles, bsp ($10^{-4}m^{-1}$)												
24 hour	0.24	0.23	0.03	2.00	0.27	0.27	0.03	2.00	0.19	0.13	0.03	1.25
1 hour	0.55	0.48	0.07	3.26	0.66	0.54	0.07	3.26	0.40	0.31	0.08	2.73
NO_2 (ppb)												
24 hour	11.35	4.62	2.47	27.29	13.03	4.34	2.87	27.29	8.85	3.83	2.47	24.67
1 hour	22.90	8.39	5.17	64.29	25.12	7.28	6.50	64.29	19.6	8.83	5.17	52.15
CO (ppm)												
8 hour	0.92	0.75	0.10	5.68	1.15	0.86	0.10	5.68	0.58	0.32	0.10	2.35
1 hour	1.51	1.19	0.17	9.33	1.88	1.33	0.20	9.33	0.95	0.60	0.17	3.57

[a]April-October
[b]November-March

Table A4: Relative risk of admission per 1 ppm increase in CO in Melbourne, with 95% confidence intervals.

Admissions category	Averaging Time	Pollutant lag	Relative Risk	95% CI
Respiratory 0-14 year	8-hour	lag 1	1.0082	0.9916-1.0252
	1-hour	lag 1	1.0056	0.9950-1.0163
Respiratory 15-64 year	8-hour	3-day av	1.0328	1.0098-1.0564
	1-hour	3-day av	1.0195	1.0050-1.0342
Respiratory 65+ year	8-hour	5-day av	1.0305	1.0069-1.0546
	1-hour	5-day av	1.0210	1.0059-1.0363
Respiratory all ages	8-hour	lag 1	1.0090	0.9992-1.0190
	1-hour	5-day av	1.0101	1.0010-1.0192
Asthma 0-14 years	8-hour	3-day av	1.0606	1.0274-1.0948
	1-hour	3-day av	1.0310	1.0100-1.0524
Asthma all ages	8-hour	5-day av	1.0639	1.0363-1.0922
	1-hour	5-day av	1.0398	1.0222-1.0577
Cardiovascular 0-64 years	8-hour	3-day av	1.0248	1.0043-1.0457
	1-hour	same day	1.0118	1.0021-1.0215
Cardiovascular 65+ years	8-hour	3-day av	1.0329	1.0185-1.0476
	1-hour	3-day av	1.0205	1.0113-1.0297
Cardiovascular all ages	8-hour	3-day av	1.0272	1.0154-1.0391
	1-hour	3-day av	1.0173	1.0098-1.0250
Ischaemic heart disease	8-hour	3-day av	1.0368	1.0180-1.0558
	1-hour	3-day av	1.0227	1.0107-1.0348

Table A5: Nitrogen dioxide levels from time series hospitalisation studies conducted in four Australian cities

Location (study period)	Time period of study	Averaging period	NO_2 levels (ppm)^ Mean (range)#		
			Whole study period	Cool season	Warm season
Brisbane (Petroeschevsky (2001) [70]	1987-1994	1-h max	0.028 (0.004-0.156)	0.035 (0.001-0.045)	0.021 (0.004-0.058)
		24-h	0.014 (0.001-0.050)	0.018 (0.004-0.081)	0.010 (0.002-0.033)
Sydney (Morgan 1998)[69]	1990-1994	1-h max	0.029 (0.0-0.139)	N/a*	N/a
		24-h	0.015 (0.0-0.052)	N/a*	N/a
Melbourne (Simpson, 2000) [68, 90]	1994-1997	1-h max	0.023 (0.005-0.064)	0.025 (0.007-0.064)	0.02 (0.005-0.052)
		24-h	0.01 (0.002.-0.027)	0.013 (0.003-0.027)	0.09 (0.003-0.025)
Perth (WA Dep. of Envir, 2003)[71]	1992-1997	1-h max	0.025 (0.013-0.038)	0.025 (0.014-0.036)	0.025 (0.012-0.039)
		24-h	0.01 (0.004-0.017)	0.011 (0.005-0.018.0)	0.01 (0.004-0.0156)

^ To convert to $\mu g/m^3$ multiply by 2050.
*Not available
10-90th centile for Perth
Brisbane: Cool=June-August, Warm=December-February; Melbourne: Cool=April-October, Warm=November-March; Perth: Cool= May-October, Warm = November-April.

Table A6: Summary of Australian hospitalisation studies and ambient NO_2

Author, year and city	Type of Hospital Admission	Age	Averaging Period	RR	Unit Increase	Ambient NO_2 mean(SD)	Adjusted confounders
Petroeschevsky	Asthma	0-14	1 hr max.	0.975(0.947-1.004)	0.01ppm	0.028 ppm for 1-hr max	Season, flu, day, long term,
1987-1994	Asthma	15-64	1 hr max.	0.983(0.949-1.018)		(range: 0.004-0.016)	,holiday, temp, humidity,
Brisbane	Asthma	All	1 hr max.	0.962(0.936-0.989)		0.014 pphm for 24 hr	rainfall, age, year
	Respiratory	0-4	1 hr max.	1.015(0.996-1.035)		(range:0.001-0.05)	, lag 0-5 days.
	Respiratory	5-14	1 hr max.	0.985(0.950-1.021)			
	Respiratory	15-64	24 hr.	1.027(0.984-1.071)			
	Respiratory	65+	24 hr.	0.903(0.851-0.959)			Single pollutant model for NO_2.
	Respiratory	All	1 hr max.	0.989(0.977-1.002)			
	Cardiovascular	15-64	1 hr max.	0.986(0.968-1.005)			
	Cardiovascular	65+	1 hr max.	0.990(0.977-1.003)			No significant effects
	Cardiovascular	All	1 hr max.	0.987(0.976-0.998)			for multi-pollutant models
Morgan	Asthma	1-14	1 hr max.	1.0529(1.0107-1.0968)*	0.015-0.044	Daily 1-h max.: 0.029	Weather, season,
1990-1994	Asthma	15-64	1 hr max.	1.0318(0.9847-1.0811)	ppm	(0.013) ppm	long term trends, day,
Sydney	COPD	65+	1 hr max.	1.046(0.9983-1.0961)			holidays.
	Heart Disease	All	1 hr max.	1.0608(1.0363-1.0859)*			
	Heart Disease	65+	1 hr max.	1.0671(1.0425-1.0925)*			Single pollutant.
	Heart Disease	<64	1 hr max.	1.0479(1.0118-1.0853)*			
	Asthma	1-14	24 hr.	1.0328(0.9818-1.0854)	0.006-0.023	0.015 (0.006) ppm for 24 hr	
	Asthma	15-64	24 hr.	1.0229(0.9703-1.0783)	ppm		
	COPD	65+	24 hr.	1.043(0.9925-1.0961)			
	Heart Disease	All	24 hr.	1.0725(1.0521-1.0988)*			
	Heart Disease	65+	24 hr.	1.0839(1.0541-1.1146)*			
	Heart Disease	<64	24 hr.	1.0581(1.0163-1.1017)*			
	Asthma	1-14	1 hr max.	1.0595(1.0111-1.1102)*			Above plus:
	COPD	65+	1 hr max.	1.0370(0.9897-1.0866)			particulates and ozone
	Heart Disease	65+	1 hr max.	1.0668(1.0361-1.0984)*			
EPA Victoria	Respiratory	0-14	24 hr.	1.0079 (1.0038-1.0121)*	1ppb	11.35 (4.62) ppb for 24 hour (all year)	Weather, season, day,
1994-1997	Respiratory	0-14	1 hr max.	1.0025 (0.9999-1.0051)		22.90(8.39) ppb for 1hour (all year)	holidays, long term trends.
Melbourne	Respiratory	15-64	24 hr.	1.0084 (1.0043-1.0126)*			
	Respiratory	15-64	1 hr max.	1.0045 (1.0020-1.0071)*			

	Respiratory	65+	24 hr.	1.0110 (1.0070-1.0149)*			Single pollutant model
	Respiratory	65+	1 hr max.	1.0048 (1.0025-1.0072)*			
	Respiratory	All	24 hr.	1.0078(1.0051-1.0102)*			
	Respiratory	All	1 hr max.	1.0043(1.0029-1.0057)*			
	Asthma	0-14	24 hr.	1.0118 (1.0058-1.0177)*			
	Asthma	0-14	1 hr max.	1.0048(1.0013-1.0084)*			
	Asthma	All	24 hr.	1.0145(1.0099-1.0191)*			
	Asthma	All	1 hr max.	1.0059(1.0032-1.0087)*			
	Cardiovascular	0-64	24 hr.	1.0036 (1.0011-1.0062)*			
	Cardiovascular	0-64	1 hr max.	1.0013 (0.9999-1.0027)			
	Cardiovascular	65+	24 hr.	1.0045(1.0023-1.0067)*			
	Cardiovascular	65+	1 hr max.	1.0020(1.0008-1.0032)*			
	Cardiovascular	All	24 hr.	1.0040(1.0022-1.0058)*			
	Cardiovascular	All	1 hr max.	1.0017(1.0007-1.0027)*			
	Ischaemic heart disease	All	24 hr.	1.0037(1.0013-1.0061)*			
	Ischaemic heart disease	All	1 hr max.	1.0012 (1.000-1.0025)*			
WA Dept of Envir	Cardiovascular	All	24 hr.	1.0029(1.0002-1.0056)*	1ppb	24.8 (13.3-37.5) ppb	Case cross-over analyses.
1992-1997	Cardiovascular	65+	24 hr.	1.0047(1.0013-1.0081)*		10.3 (4.4-17.1) ppb	
Perth	Cardiovascular	65+	1 hr max.	1.0016(1.0001-1.0031)*			Current day with
	Respiratory	65+	24 hr.	1.0058(1.0003-1.0113)*			1 week before and 1 week after. Adjusted for humidity, temperature, day and holidays.

*= p <0.05

Table A7: **Particulate levels from the earlier time series hospitalisation studies conducted in four Australian cities**

Location (study period)	Time period of study	Averaging period	Particulate pollution levels[1] Mean (range)^		
			Whole study period	Cool season	Warm season
Brisbane (Petroeschevsky (2001)[70]	1987-1994	1-h max	7.01 (0.78-162.4) bsp 10^{-5}/m1	8.60 (1.10-162.4)# bsp 10^{-5}/m	5.28 (0.78-81.0) bsp 10^{-5}/m
		24-h	2.74 (0.30-50.8) bsp 10^{-5}/m	3.32 (0.47-50.8)# bsp 10^{-5}/m	2.08 (0.42-15.9) bsp 10^{-5}/m
Sydney (Morgan 1998)[69]	1990-1994	1-h max	0.76 (0.01-7.86) bscat/10^4m	N/a*	N/a*
		24-h	0.32 (0.01-3.72) bscat/10^4m	N/a*	N/a*
Melbourne (Simpson, 2000)[68, 90]	1994-1997	1-h max	0.55 (0.07-3.26) bsp $10^{-4}m^{-1}$	0.66 (0.07-3.26) bsp $10^{-4}m^{-1}$	0.40 (0.08-2.73) bsp $10^{-4}m^{-1}$
		24-h	0.24 (0.03-2.00) bsp $10^{-4}m^{-1}$	0.27 (0.03-2.00) bsp $10^{-4}m^{-1}$	0.19 (0.03-1.25) bsp $10^{-4}m^{-1}$
Perth (WA Dep. of Envir, 2003)[71]	1992-1997	1-h max	1.2 (0.3-2.6) bscat/10^4m	1.61 (0.39-3.73) bscat/10^4m	0.74 (0.25-1.39) bscat/10^4m
		24-h	0.25 (0.1-0.47) bscat/10^4m	0.3 (0.12-0.57) bscat/10^4m	0.2 (0.09-0.33) bscat/10^4m

*Not available

Brisbane: Cool= Spring (September-November) since levels in Spring were slightly above Winter (June-August), Warm=December-February.
Melbourne: Cool=April-October, Warm=November-March; Perth: Cool= May-October, Warm = November-April
^ 10-90th Percentile for Perth.

[1] PM are reported as black smoke, bsp, bscat, PM_{10} and $PM_{2.5}$. The conversion factors for these are dependent on the city. To convert to ug/m^3 the conversion factors are dependent on the city. For Sydney the conversion factor is: $PM_{2.5}$ = 30 x bscat/10^4m, PM_{10} = 2 x $PM_{2.5}$[69, 73], therefore to convert PM_{10} in $\mu g/m^3$ to bscat/10^4m divide by 60. For Brisbane the conversion factor is: 1 x 10^{-5}/m bsp = 0.3 x $PM_{2.5}$ and $PM_{2.5}$ ~ 0.4 x PM_{10} [70, 80].

Table A8: **Australian hospitalisation studies on the health effects of particulate pollution.**

Author, year and city	Type of Hospital Admission	Age	Averaging Period	RR	Unit Increase	Ambient particulate level mean(SD)	Adjusted confounders
Simpson	Cardiac	All	24 hr	1.0856 (1.0603-1.1116)	1 bsp$10^{-4}m^{-1}$	0.3 (0.0-2.5)	season, flu ,day ,long term, holiday, temp, humidity,
1996-1999	Cardiac	15-64	24 hr	1.0446 (1.0021-1.0889)			Single pollutant models for bsp and significant for multi-pollutant models for cardiac all ages
Brisbane, Melbourne, Sydney and Perth	Cardiac	>65	24 hr	1.1013 (1.0701-1.1334)			
	Ischemic Heart D	All	24 hr	1.0872 (1.0516-1.1240)			
	Ischemic Heart D	>65	24 hr	1.1029 (1.0591-1.1486)			
	Respiratory	>65	24 hr	1.0552 (1.0082-1.1045)			
	Asthma	15-64	24 hr	1.0893 (1.0240-1.1587)			
	Asthma and COPD	>65	24 hr	1.0713 (1.0179-1.1276)			
	Pneumonia and acute bronchitis	>65	24 hr	1.0769 (1.0046-1.1544)			
Petroeschevsky	Asthma	0-14	1 hr max.	0.995(0.990-0.999)	1 x 10^{-5}/m	7.01 for 1-hr max	season, flu ,day ,long term, holiday, temp, humidity,
1987-1994	Asthma	15-64	24 hr	1.016(0.994-1.038)		(0.78-162.4) 10^{-5}/m	Rainfall, age, year, , lag 0-5 days
Brisbane	Asthma	All	1 hr max.	0.999(0.995-1.002)		2.74 for 24 hr (0.30-50.8) 10^{-5}/m	Single pollutant models for bsp only
	Respiratory	0-4	1 hr max.	1.002(0.999-1.006)			
	Respiratory	5-14	1 hr max.	0.999(0.995-1.005)			
	Respiratory	15-64	1 hr max.	1.005(1.001-1.010)*			
	Respiratory	65+	1 hr max	1.002(0.999-1.005)			
	Respiratory	All	24 hr	1.015(1.006-1.023)*			
	Cardiovascular	15-64	24 hr	0.995(0.911-1.087)			
	Cardiovascular	65+	1 hr max.	0.998(0.996-0.999)			
	Cardiovascular	All	1 hr max.	0.998(0.997-0.999)			
	Respiratory	15-64	1 hr max.	1.002(0.995-1.008)			Not significant in a multi-pollutant model controlling for high ozone
	Respiratory	All	24 hr	1.015(1.004-1.026)*			Significant in a multi-pollutant model controlling for high ozone
	Respiratory	All	24 hr	1.015(1.007-1.024)*			Significant in a multi-pollutant model controlling for high SO_2
Morgan	Asthma	1-14	1 hr max.	1.008(0.9726-1.0449)	0.25-1.48 bscat/10^4m	0.76 for 1 hr max (0.01-7.86) bscat/10^4m	weather, season, long term trends, day,
1990-1994	Asthma	15-64	1 hr max.	1.0225 (0.9864-1.0598)			holidays, single pollutant
Sydney	COPD	65+	1 hr max.	1.031(0.9962-1.0652)			

	Heart Disease	All	1 hr max.	1.0192(1.0047-1.0339)*			
	Heart Disease	65+	1 hr max.	1.0272(1.0077-1.047)*			
	Heart Disease	0-64	1 hr max.	1.0028(0.9762-1.0295)			
	Asthma	1-14	24 hr	0.9913(0.9537-1.0302)	0.12-0.6 bscat/10^{4}m	0.32 for 24 hr average (0.01-3.72) bscat/10^{4}m	weather, season, long term trends, day,
	Asthma	15-64	24 hr	1.0121(0.9764-1.0510)			holidays, single pollutant
	COPD	65+	24 hr	1.0241(0.9910-1.0584)			
	Heart Disease	All	24 hr	1.0223(1.0061-1.0388)			
	Heart Disease	65+	24 hr	1.0282(1.009-1.0477)*			
	Heart Disease	0-64	24 hr	1.0102(0.983-1.0381)*			
	Heart Disease	65+	24 hr	1.0017(0.982-1.0236)			Not significant in multi-pollutant models (above plus ozone and NO_2)
EPA Victoria	Respiratory	15-64	1 hr max.	1.0383 (1.0063-1.0714)*	1x $10^{-4}m^{-1}$	0.55 (0.48) x $10^{-4}m^{-1}$for 1 hr max. (all year)	weather, season, long term trends, day,
1994-1997	Respiratory	65+	1 hr max.	1.043 (1.0088-1.0783)*		0.24 (0.23) x $10^{-4}m^{-1}$for 24 hr (all year)	holidays, single pollutant
Melbourne	Respiratory	All	1 hr max.	1.0119 (0.9967-1.0273)			
	Asthma	0-14	1 hr max.	1.0592 (1.0197-1.1002)*			
	Asthma	All	1 hr max.	1.0766 (1.0374-1.1172)*			
	Cardiovascular	65+	1 hr max.	1.0352 (1.0143-1.0565)*			
	Cardiovascular	All	1 hr max.	1.0274 (1.0104-1.0446)			
	Ischaemic heart disease		1hr max.	1.0297 (1.0090-1.0509)			
	Respiratory	15-64	24 hr	1.0784 (1.0121-1.1491)*			
	Respiratory	65+	24 hr	1.0745 (1.0041-1.1499)*			
	Respiratory	All	24 hr	1.0239(0.9927-1.0561)			
	Asthma	0-14	24 hr	1.1481 (1.0628-1.2403)*			
	Asthma	All	24 hr	1.1394 (1.0582-1.2268)*			
	Cardiovascular	65+	24 hr	1.0560 (1.0208-1.0924)*			
	Cardiovascular	All	24 hr	1.0461 (1.0174-1.0757)			
	Ischaemic heart disease		24 hr.	1.0631 (1.0188-1.1093)*			
	Respiratory	65+	1 hr max.	1.0041 (1.0014-1.0069)*			bsp and NO_2 in multi-pollutant models
	Respiratory	65+	1 hr max.	1.0017 (1.0003-1.0031)*			bsp and O_3 in multi-pollutant models
	Cardiovascular	65+	1 hr max.	1.0188(1.0016-1.0362)*			bsp and CO in multi-pollutant models
	Cardiovascular	65+	24 hr	1.0021(1.005-1.0037)*			bsp and NO_2 in multi-pollutant models

	Cardiovascular	65+	24 hr	1.0172(1.0045-1.0301)*			bsp and CO in multi-pollutant models
WA Dept of Envir	Respiratory	65+	1 hr max.	1.0196(1.0048-1.0347)*	1x $10^{-4}m^{-1}$ bsp	1.2 (0.3-2.6) bscat/10^4m	Case cross-over analyses. Current day with
1992-1997	COPD	All	1 hr max.	1.0347(1.0125-1.0573)*		0.25 (0.1-0.47) bscat/10^4m	1 week before and 1 week after. Adjusted for humidity,
Perth	COPD	65+	1 hr max.	1.0492(1.0239-1.0752)*			temperature, day and holidays.
	COPD	65+	24 hr	1.2431(1.0377-1.4892)*			

*= p <0.05

Appendix B

Table B1: Descriptive data for the meta-analysis of four Australian cities for the period 1996-99[85].

	Brisbane Mean (range)	Sydney Mean (range)	Melbourne Mean (range)	Perth Mean (range)
Mortality outcome				
Total all-cause (all ages)	16.03 (5-33)	56.83 (31-103)	56.10 (30-90)	20.26 (5-40)
Respiratory (all ages)	1.51 (0-8)	5.43 (0-25)	4.92 (0-17)	1.96 (0-11)
Cardiovascular (all ages)	7.42 (1-18)	25.49 (10-56)	23.19 (8-41)	8.20 (0-20)
Cardiovascular (≥65 years)	6.78 (0-18)	22.78 (8-54)	20.91 (7-38)	7.38 (0-18)
Pollutant levels				
CO (ppm) 8 hr	0.74[c] (0.02-4.17)	0.95 (0.02-4.62)	1.0 (0.07-6.95)	1.19 (0.10-4.25)
NO_2 (ppb) 1 hr max.	21.43(2.05-63.31)	23.66(6.54-59.38)	23.65(4.41-66.70)	16.33(1.87-41.0)
bsp ($10^{-4}m^{-1}$) 24 hr	0.26(0.02-2.49)	0.25(0.04-1.58)	0.26(0.03-2.23)	0.25(0.07-1.76)
PM_{10}($\mu g/m^3$) 24 hr	16.5(2.6-57.6)	16.3(3.7-75.5)	18.2(3.3-51.9)	
$PM_{2.5}$($\mu g/m^3$) 24 hr	7.5[b](1.9-19.7)	9.0[a](2.4-35.3)	9.3[a](2.7-35.1)	9.0(2.8-37.3)
O_3 (ppb) 1 hr	30.95(2.85-111.50)	29.55(3.15-110.97)	24.35(1.62-96.0)	33.78(13.0-105.0)

Table B2: Daily mortality – mean and range for earlier studies on individual cities[71, 73, 80, 86].

Outcome	ICD 9	Mean	Range
Sydney			
Asthma - All	N/a		
Respiratory - All	460-519	4.7	0-17
Cardiovascular - All	390-459	28.8	9-60
Brisbane			
Asthma - All	N/a		
Respiratory - All	460-519	1	N/a
Cardiovascular - All	393-399, 402, 404, 410-416, 420, 429	5	N/a
Melbourne			
Asthma - All	N/a		
Respiratory - All	460-519	4.5	0-16
Cardiovascular - All	390-459	24.3	8-43
Perth			
Asthma - All	N/a		
Respiratory - All	460-529	2.4	0-5 (10-90 Percentiles)
Cardiovascular - All	390-459	11.6	7-17 (10-90 Percentiles)

N/a=not available

Table B3: Nitrogen dioxide levels from time series mortality studies conducted in four Australian cities

Location (study period)	Time period of study	Averaging period	NO_2 levels (ppb)[1] Mean (range)		
			Whole study period	Cool season	Warm season
Brisbane (Simpson, 1997)[80]	1987-1993	1-hr max.	28 (4-82)	33 (4-81)	24 (4-82)
		24-h	14 (1-42)	16 (1-42)	12 (2-37)
Sydney (Morgan 1998)[73]	1989-1993	1-h	26 (0-104)	N/a	N/a
		24-h	13(0-39)	N/a	N/a
Melbourne (Simpson, 2000) [86]	1991-1996	1-h	24 (5-81)	26 (5-81)	20 (5-71)
		24-h	12 (1-34)	13 (2-34)	9 (1-30)
Perth (WA Dep. of Envir, 2003)[71]	1992-1997	1-h max	24.8 (13.3-37.5)	24.9 (14.4-35.7)	24.7(12.4-39.2)
		24-h	10.3 (4.4-17.1)	11.1 (4.8-18.0)	9.6 (4.3-15.7)

N/a = Not available
Brisbane: Cool=June-August, Warm=December-February; Melbourne: Cool=April-October, Warm=November-March; Perth: Cool= May-October, Warm = November-April.
1 For conversions see page 13.

Table B4: Summary of the Australian studies of ambient nitrogen dioxide and mortality.

Author, year and city	Type of Mortality	Age	Averaging Period	RR	Unit Increase	Adjusted confounders
Simpson	Total	All	1 hr max.	1.003(0.991-1.015)	10ppb	Weather, long term trends, day,
1987-1993	Total	All	24 hr	0.995(0.973-1.017)		season, holidays, influenza.
Brisbane	Cardiovascular	All	1 hr max.	1.000(0.980-1.020)		Single pollutant model only,
	Cardiovascular	All	24 hr	0.985(0.945-1.025)		
	Respiratory	All	1 hr max.	1.010(0.972-1.048)		
	Respiratory	All	24 hr	0.958(0.880-1.036)		
Morgan	Total	All	1 hr max.	1.0128 (0.9940-1.032)	0.012-0.044 ppm 1 hr-max.	Weather, , long term trends, day,
1989-1994	Cardiovascular	All	1 hr max.	1.0457(0.9823-1.1131)		holidays, influenza epidemic,
Sydney	Respiratory	All	1 hr max.	1.0096(0.9847-1.0352)		
	Total	All	24 hr	1.0266(1.0004-1.0535)*	0.005-0.023 ppm 24 hr.	
	Cardiovascular	All	24 hr	1.02(0.9853-1.0569)		
	Respiratory	All	24 hr	1.0771(0.9966-1.1640)		
Simpson	Total	All	24 hr	1.0024(1.0012-1.0036)	1ug/m3	Weather, long term trends, day,
1991-1996	Total	65+	24 hr	1.0019(1.0007-1.0032)		season, holidays, influenza
Melbourne	Respiratory	All	24 hr	1.0045(1.0003-1.0087)		Single model only, no sig. effect in muti-models
	Respiratory	65+	24 hr	1.0049(1.0005-1.0093)		
	Cardiovascular			no sig. effect		
WA Dept of Envir	No significant associations for mortality				1ppb	Case cross-over analyses. Current day with 1 week before and 1 week after. Adjust. for humidity, temp., day and holidays.
1992-1997						
Perth						

Table B5: Particulate pollution levels from time series mortality on earlier studies conducted in four individual Australian cities.

Location (study period)	Time period of study	Averaging period	Particulate pollution levels[1] Mean (range)#		
			Whole study period	Cool season	Warm season
Brisbane (Simpson, 1997)[80]	1987-1993	1-hr max.	6.68 (0.78-77.3) bsp10^5/m	7.70 (0.95-77.73) bsp10^5/m	4.89 (0.78-50.88) bsp10^5/m
		24-h	2.59 (0.3-15.16) bsp10^5/m	2.93 (0.3-15.16) bsp10^5/m	2.27 (0.42-14.17) bsp10^5/m
Sydney (Morgan 1998)[73]	1989-1993	1-h max.	0.70 (0.08-6.16) bscat/10^4m	N/a	N/a
		24-h	0.30 (0.03-1.85) bscat/10^4m	N/a	N/a
Melbourne (Simpson, 2000) [86]	1991-1996	1-h max.	0.60 (0.08-4.98) bscat/10^4m	0.70 (0.1-4.36) bscat/10^4m	0.46 (0.08-4.98) bscat/10^4m
		24-h	0.26 (0.04-2.52) bscat/10^4m	0.29 (0.04-2.52) bscat/10^4m	0.22 (0.04-2.07) bscat/10^4m
Perth (WA Dep. of Envir, 2003)[71]	1992-1997	1-h max	1.2 (0.3-2.6) bscat/10^4m	1.61 (0.39-3.73) bscat/10^4m	0.74 (0.25-1.39) bscat/10^4m
		24-h	0.25 (0.1-0.47) bscat/10^4m	0.3 (0.12-0.57) bscat/10^4m	0.2 (0.09-0.33) bscat/10^4m

N/a = Not available
Brisbane: Cool=June-August, Warm=December-February; Melbourne: Cool=April-October, Warm=November-March; Perth: Cool= May-October, Warm = November-April.
10-90th centile in Perth
[1] PM are reported as black smoke, bsp, bscat, PM_{10} and $PM_{2.5}$. The conversion factors for these are dependent on the city. To convert to ug/m^3 the conversion factors are dependent on the city. For Sydney the conversion factor is: $PM_{2.5}$ = 30 x bscat/10^4m, PM_{10} = 2 x $PM_{2.5}$ [69, 73], therefore to convert PM_{10} in $\mu g/m^3$ to bscat/10^4m divide by 60. For Brisbane the conversion factor is: 1 x 10^{-5}/m bsp = 0.3 x $PM_{2.5}$ and $PM_{2.5}$ ~ 0.4 x PM_{10} [70, 80].

Table B6: Summary of the Australian studies of ambient particulate pollution and mortality.

Author	Type of Mortality	Age	Averaging Period	RR	Unit Increase	Adjusted confounders
Simpson	Total	All	24 hr	**1.0284** (1.0015-1.0560)	1 bsp$10^{-4}m^{-1}$	weather, long term trends, day,
1996-1999	Respiratory	All	24 hr	1.0690 (0.9814-1.1645)		season, holidays, influenza
Brisbane, Melbourne, Sydney and Perth	Cardiovascular	All	24 hr	**1.0479** (1.0076-1.0898)		single pollutant model only, not significant when NO_2 was included in the model
	Cardiovascular	>65	24 hr	1.0337 (0.9918-1.0774)		
Simpson	Total	All	1 hr max.	1.002(1.000-1.004)*	1 x 10^{-5}/m	weather, long term trends, day,
1987-1993	Total	>65	1 hr max	1.002(1.000-1.004)*		season, holidays, influenza
Brisbane	Cardiovascular	All	1 hr max.	1.004(1.001-1.008)*		single pollutant model only,
	Cardiovascular	>65	1 hr max	1.005(1.001-1.009)*		
	Respiratory	All	1 hr max.	1.001(0.997-1.005)		
	Total	All	24 hr	1.009(1.003-1.015)*	1 x 10^{-5}/m	
	Total	>65	24 hr	1.010(1.002-1.018)*		
	Cardiovascular	All	24 hr	1.010(0.998-1.02)		
	Cardiovascular	>65	24 hr	1.011(0.999-1.023)		
Morgan	Total	All	1 hr max.	1.0253 (1.0087-1.042)*	0.23-1.42 bscat/10^4m	weather, , long term trends, day,
1989-1994	Cardiovascular	All	1 hr max.	1.0296(1.0082-1.0514)*		holidays, influenza epidemic,
Sydney	Respiratory	All	1 hr max.	1.041(0.991-1.0930)		
	Total	All	24 hr	1.0263((1.0087-1.044)*	0.10-0.5 bscat/10^4m	
	Cardiovascular	All	24 hr	1.0268(1.0025-1.0516)*		
	Respiratory	All	24 hr	1.0334(0.9787-1.0911)		
	Total	All	24 hr	1.0229(1.002-1.044)*		in multi-pollutant models (NO2 and ozone)
	Cardiovascular	All	24 hr	1.021(0.9925-1.0497)		
	Respiratory	All	24 hr	1.007(0.9446-1.0747)		
Simpson	Total	All	1 hr max. or 24 hr	no sig effect (all year)	1ug/m3	weather, long term trends, day,
1991-1996	Respiratory	All	1 hr max. or 24 hr	no sig effect (all year)		season, holidays, influenza
Melbourne	Cardiovascular	All	1 hr max. or 24 hr	no sig effect (all year)		single model only, no sig. effect in muti-models
	Total	All	24 hr $PM_{2.5}$	1.0038(1.0006-1.007)*		warm season
	Respiratory	All	24 hr $PM_{2.5}$	1.0118 (1.0003-1.0232)*		
	Respiratory	65+	24 hr $PM_{2.5}$	1.0127 (1.0009-1.0246)*		
	Total	All	24 hr PM_{10}	1.0018(1.0007-1.0033)*		warm season
	Respiratory	All	24 hr PM_{10}	1.0059 (1.0006-1.0113)		

	Respiratory	65+	24 hr PM_{10}	1.0065 (1.0012-1.0119)
WA Dept of Envir	No significant associations with mortality			Case cross-over analyses. Current day with
1992-1997				
Perth				

*= P<0.05.

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