I, Dr Vicki Kotsirilos, am registered as a Specialist General Practitioner with the Medical Board of Australia [AHPRA] and work in Clayton, Melbourne. I reside in the suburb of Sandringham.

I am an Honorary Fellow of the Australasian College of Nutritional and Environmental Medicine. I am also an Associate Professor at the University of La Trobe and Western Sydney, and formerly held an adjunct position at Monash University Department of Epidemiology and Public Health.

I have been requested to prepare an expert witness statement related to public health effects from vehicle emission air pollution by the local residents who will be directly impacted by the construction of the freeway close to their homes, schools and work.
My expertise in relation to this matter includes:

1. I keep up to date with research and issues related to chronic diseases and public health, particularly related to lifestyle, physical activity, the environment and nutrition.

2. I treat patients from all age groups with a wide variety of medical conditions in clinical practice. These include patients suffering chronic diseases including asthma, respiratory diseases, cardiac disease and cancers that are directly impacted by road traffic air pollution.

3. I interpret and regularly publish articles based on research into lifestyle and environmental risks affecting general health and chronic diseases, in the general practice setting.

4. I have an interest and expertise in General Medical Health and Environmental Medicine. As a medical practitioner, I have knowledge of the current evidence and first-hand clinical experience of treating patients who are directly or indirectly impacted by poor air quality and pollution. These include children and adults with asthma, chronic lung diseases, heart disease and lung cancer. I directly treat patients who are impacted adversely from living or working near polluted regions.

I declare I have limitations in scientific knowledge on monitoring and how predictions are made on Concentrations of Pollutants for sensitive receptors against design limits.

However, I do regularly review the scientific literature on epidemiological and cohort studies of societies and cities based in Australia and in other countries similar to Australia, such as in Europe and the United States, in relation to how air pollution is becoming a serious widespread health related issue, and has negative impacts on human health, regardless of whether it meets State Environment Protection Policy (Ambient Air Quality) Victorian Government SEPP(AAQ) objectives. The bottom line is THERE IS NO SAFE LEVEL OF AIR POLLUTANTS.

For these reasons I support the local communities opposition of the construction of the NEL freeway being built adjacent to, or near an urban environment, and favour instead funding be diverted to improve public transport for the area to help relieve congestion of vehicles on the road. I do not object to freeways constructed outside of urban areas, but the proposed freeway will be built in one of the few remaining green spaces in Melbourne, and adjacent to an urban environment in such a way that I ascertain will cause short and long term health issues for the residents and workers of these local communities.

I have attached a review paper I have prepared of the scientific evidence that addresses my concerns of air pollution to human health to the best of my ability to support this statement,
and why freeways should not be built in urban environments. This scientific document has been peer reviewed by other medical colleagues and experts in the field, and is pending publication in a peer review journal.

Many of the residents impacted by the Project currently enjoy predominately fresh air being surrounded by local parklands and clean local suburbs. They are experiencing great fears about the health implications of building a freeway next to or near their homes, schools and workplaces, and the impact this would have on their health. Those with pre-existing health conditions such as cardiac disease, lung disease and asthma, and the more vulnerable and susceptible, such as people with pre-existing illnesses, children, elderly and pregnant women are most at risk of harm as demonstrated in studies included in my scientific report attached.

The residents have invited me to submit an expert report and have asked me to address the following questions in relation to the EES for the construction of the proposed NEL Freeway:

1. Does the NEL Freeway adequately identify and respond appropriately to the most current scientific evidence and literature in relation to health impacts from air pollution?

2. Is there evidence that the NEL Freeway constructed next to an urban residential area may impact the health of the local residents who live or work in the area?

3. How may air pollution from vehicle emissions impact the health of the local residents?

4. Have the potential health concerns to the local community been adequately addressed in the submission in view that by 2036, it is estimated that North East Link would carry up to 135,000 vehicles per day (page 9, chapter 9 Traffic & Transport NELP - EES)?

5. Do you believe the local residents have been adequately informed of the vehicle emissions and health impacts particularly those who reside or work within 250 metres of the Project?

**Relevant link:**

1. Does the NEL Freeway adequately identify and respond appropriately to the most current scientific evidence and literature in relation to health impacts from air pollution?

The NEL EES does not include any data or new insights from the most current scientific literature and publications within scientific and medical journals that demonstrate clearly that road traffic related air pollution impacts human health. For particulate matter and most pollutants from vehicle emissions, there is no level, below which negative impacts do not occur, therefore any increments in air pollutants, however small, will likely result in public health issues. Vehicle emissions are a particularly noxious sub-set of ambient particulate matter. The EES needs to fully consider all the relevant adverse health impacts due to traffic pollution.

My scientific report attached found in Appendix A summarises some of the evidence that demonstrates the health concerns from exposure to road traffic related air pollution, even at levels below what is regarded as acceptable maximum concentrations and measures as set out by the National Environment Protection (Ambient Air Quality) Measure Schedule 2 Standards and Goals¹. These Standards and Maximum concentrations are tabled below¹:

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### Table 1: Standards and Goal for Pollutants other than Particles as PM$_{2.5}$

<table>
<thead>
<tr>
<th>Item</th>
<th>Pollutant</th>
<th>Averaging period</th>
<th>Maximum concentration</th>
<th>Goal within 10 years Maximum allowable exceedences</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Carbon monoxide</td>
<td>8 hours</td>
<td>9.0 ppm</td>
<td>1 day a year</td>
</tr>
<tr>
<td>2</td>
<td>Nitrogen dioxide</td>
<td>1 hour</td>
<td>0.12 ppm</td>
<td>1 day a year</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 year</td>
<td>0.03 ppm</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>Photochemical oxidants (as ozone)</td>
<td>1 hour</td>
<td>0.10 ppm</td>
<td>1 day a year</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 hours</td>
<td>0.08 ppm</td>
<td>1 day a year</td>
</tr>
<tr>
<td>4</td>
<td>Sulfur dioxide</td>
<td>1 hour</td>
<td>0.20 ppm</td>
<td>1 day a year</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 day</td>
<td>0.08 ppm</td>
<td>1 day a year</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 year</td>
<td>0.02 ppm</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>Lead</td>
<td>1 year</td>
<td>0.50 µg/m$^3$</td>
<td>none</td>
</tr>
<tr>
<td>6</td>
<td>Particles as PM$_{10}$</td>
<td>1 day</td>
<td>50 µg/m$^3$</td>
<td>5 days a year</td>
</tr>
</tbody>
</table>

### Table 2: Advisory Reporting Standards and Goal for Particles as PM$_{2.5}$

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Averaging Period</th>
<th>Maximum Concentration</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Particles as PM$_{2.5}$</td>
<td>1 day</td>
<td>25 µg/m$^3$</td>
<td>Goal is to gather sufficient data nationally to facilitate a review of the Advisory Reporting Standards as part of the review of this Measure scheduled to commence in 2005</td>
</tr>
<tr>
<td></td>
<td>1 year</td>
<td>8 µg/m$^3$</td>
<td></td>
</tr>
</tbody>
</table>
The State Environment Protection Policy (Ambient Air Quality) SEPP(AAQ) includes objectives for 24 hour average and annual readings:

**PM10 concentrations** of 50 μg/m³ (24 hour average) and 20 μg/m³ (one year average)

**PM2.5 objective** of 25 μg/m³ (24-hour average) and 8 μg/m³ (one year average).

The Environmental Quality Objectives for PM2.5 have been further revised and lowered to 7 micrograms/cubic metre per year as tabled in the EPA website:

<table>
<thead>
<tr>
<th>Column 1</th>
<th>Column 2</th>
<th>Column 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Environmental Indicator (Pollutant)</td>
<td>Averaging period</td>
<td>Environmental quality objectives</td>
</tr>
<tr>
<td>Particles as PM₃₅ (maximum concentration)</td>
<td>1 day</td>
<td>20 μg/m³ by 2025</td>
</tr>
<tr>
<td></td>
<td>1 year</td>
<td>7 μg/m³ by 2025</td>
</tr>
</tbody>
</table>

There is now an abundance of worldwide literature including public health alerts by authorities such as the World Health Organisation, that highlight strong data and research outcomes demonstrating immediate and long term adverse effects on human health, from Particulate Matter traffic related air pollution, occurring at a greater magnitude of impact than previously thought, **now demonstrating there is no safe lower limit of exposure**.

These concerns are well described in a number of studies conducted in Europe, the United States and Australia i.e. in areas of equivalent air pollution experienced in cities and urban

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areas of Australia that experience high traffic volumes such as Melbourne are described throughout this document and in the attached scientific report.

Recent studies now demonstrate even level of PM2.5 exposure below National Environment Protection (Ambient Air Quality) Measures and SEPP (AAQ) Objectives (as described above) are considered to cause detrimental impacts on health.\(^6\)

The World Health Organisation air quality guidelines state, “there is little evidence to suggest a threshold below which no adverse health effects would be anticipated”\(^7\).

Therefore we should be aiming at ensuring the cleanest air possible.

The Environmental Protection Agency has also provided comment on the technical and scientific aspects of the NEL Bypass (Freeway) EES\(^8\). On page 10 of their submission under section 3.1 titled "Air Quality EPR AQ1" they acknowledge: "There is well established scientific evidence that traffic related air pollution, even at concentrations well below the current air quality standards, is associated with adverse health effects."

"(Air quality (operation)). Traffic is a significant contributor to air pollution in Melbourne. It generates air pollution from both exhaust emissions and non-exhaust emissions (from tyres, brakes and road). There is well established scientific evidence that traffic related air pollution, even at concentrations well below the current air quality standards, is associated with adverse health effects. As stated above in Section 2.2.7, EPA recommends the use of the SEPP (AAQ) Schedule 2 - environment air quality objectives to compare and assess air pollution levels measured near roads. EPA also recommends proponents to adopt best practice design and controls to reduce air pollution and human health impacts. For new or upgrades of major roads, the requirement of applying demonstrated best practice design and controls that reduce air pollution impacts with risk assessments using the SEPP(AAQ) objectives should be adopted. This is consistent with the regulatory approach in other jurisdictions in Australia and internationally.


The feasibility and viability exist to implement best practice design and controls to reduce air pollution impacts to meet the SEPP(AAQ) objectives.

So just to explore in more detail a few significant studies to understand these concerns:⁴ Di Qian et al NEJM 2017:

The lack of an apparent safe lower limit of exposure to PM2.5 combined with ozone has been highlighted by a remarkably large study from the USA involving almost 61 million adults followed for 12 years recently published in the New England Journal of Medicine by Di and colleagues⁴ (Di Qian et al NEJM 2017). The aim of the study was to estimate the risk of death associated with exposure to increases of 10 μg per cubic meter for PM₂.₅ and 10 parts per billion (ppb) for ozone. They demonstrated a linear increase risk of death with increases in concentration levels of exposure to PM2.5 and ozone. This study also identified that there were groups of people, who were more susceptible to the adverse effects of air pollution than the rest of the population. In this study the risk of death was higher in men, dark skinned people, and people with Medicaid eligibility.

Their results identified:

"Increases of 10 μg per cubic meter in PM₂.₅ and of 10 ppb (parts per billion) in ozone were associated with increases in all-cause mortality of 7.3% (95% confidence interval [CI], 7.1 to 7.5) and 1.1% (95% CI, 1.0 to 1.2), respectively. When the analysis was restricted to person-years with exposure to PM₂.₅ of less than 12 μg per cubic meter and ozone of less than 50 ppb, the same increases in PM₂.₅ and ozone were associated with increases in the risk of death of 13.6% (95% CI, 13.1 to 14.1) and 1.0% (95% CI, 0.9 to 1.1), respectively. For PM₂.₅, the risk of death among men, blacks, and people with Medicaid eligibility was higher than that in the rest of the population."

As illustrated in the figure below, the researchers demonstrate that the risk of harm i.e. vertical axis- "hazard ratio"= higher risk than average of all-cause mortality [death], is dose dependent and increases with greater exposure to PM 2.5 concentration (micrograms per cubic metre) and ozone emissions (parts per billion) even below accepted national levels:
Hence the researchers concluded:

"In the entire Medicare population, there was significant evidence of adverse effects related to exposure to PM$_{2.5}$ and ozone at concentrations below current national standards. This effect was most pronounced among self-identified racial minorities and people with low income."

Extrapolating these figures to the residents, recreational users and workers near the project area will be exposed to particulate matters higher than current levels and no matter what those levels are, will likely impact their health and increase morbidity and mortality, as there is no safe level of air pollutants.
Furthermore the level of air pollution will vary during the day with peak hour traffic creating the higher levels, and most likely will increase over time as the population grows leading to a rise in Total Vehicle Numbers and consequently impact air quality.

Concerning all cause mortality, studies by Lepeule 2012\(^9\) and Beelan\(^10\) 2014 are also large cohorts that give robust estimates that demonstrate an increase in mortality related to exposure to PM 2.5, **even at lower concentrations**.

The Lupeule study based in Harvard USA for instance found since 2001, average PM\(_{2.5}\) levels, for all six cities studies, were less than 18 µg/m\(^3\). Each increase in PM\(_{2.5}\) (10 µg/m\(^3\)) was associated with an adjusted increased risk of all-cause mortality (PM\(_{2.5}\) average on previous year) of 14\%, and with 26\% and 37\% increases in cardiovascular and lung-cancer mortality (PM\(_{2.5}\) average of three previous years), respectively. The concentration–response relationship was linear down to PM\(_{2.5}\) concentrations of 8 µg/m\(^3\).

**Pope et al JAMA 2002**

A study published by Pope et al JAMA 2002 demonstrated similar concerns that are well illustrated in the diagrams below which demonstrate increase mortality and morbidity with increasing level of exposure to PM\(_{2.5}\) well below **current Australian Government standards** and Objectives\(^11\):

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It is likely there will be regions along the proposed NEL project with less air pollutant levels along the proposed freeway, such as when vehicles are cruising at the same speeds but with the increase in population and consequent increase in total number of vehicles on the freeway, weather and wind influences, breaking and accelerating there is likely to be certain areas that are "hot spots" i.e. more pollutant load.

Whilst vehicles are likely to reduce emission rates per vehicle over time, there is likely to be higher levels of total emissions overall brought about by the increase in number of vehicles from the increase in population despite improvement in emissions generated by each vehicle.

Melbourne will experience a substantial growth in Total Vehicle Numbers, which will result in significant rise in total vehicle emissions in these areas especially with expected population growth.

**Hibrid and Electric cars**

Vehicle emissions are improving overall, but Australia is lagging in terms of electric vehicle take-up, now making up only just 0.2 per cent of Australian cars according to data from Australia's Electric Vehicle Council (EVC).
2. Is there evidence that the NEL Freeway constructed next to an urban residential area may impact the health of the local residents who live or work in the area?

There is clear evidence that air pollution due to vehicle emissions is a cause of a number of health problems including increased risk of mortality. Harm to human health occurs at levels below National Environment Protection (Ambient Air Quality) Measures and SEPP (AAQ) Objectives that are currently set. This is well described above and in my scientific report attached.

Traffic related air pollution, even at low levels contributes to and/or causes a number of health problems especially for individuals living and working near freeways. Studies conducted in Europe, the United States and Australia i.e. in areas of equivalent air pollution experienced in cities of Australia such as Melbourne, are described throughout this document even when considered "safe" thresholds demonstrating detrimental impacts on health\textsuperscript{12}.

None of the studies cited in the NEL EES are warning local residents, particularly susceptible groups and residents living and working near the Project who will experience an unprecedented rise in air pollution levels from the proposed freeway.

There are also a number of international studies in countries experiencing enormous growth in traffic congestion such as China. These studies demonstrate real health risks. We need better 21st century solutions to avoid Australia falling into the same situation.

**Short and long term exposure health effects to ambient PM exposure**

Below is tabled a summary of potential health effects from short and long term exposure to ambient PM exposure\textsuperscript{13}:

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\textsuperscript{12} Diana Phillips *Even 'Safe' Levels of Air Pollution Tied to Higher Mortality* -\textit{Medscape}–Dec 27, 2017. 

\textsuperscript{13} Hime, H., C. Cowie, and G. Marks, *Review of the health impacts of emission sources, types and levels of particulate matter air pollution in ambient air in NSW*, N.E.P. Authority and E.H.B. NSW Ministry of Health, Editors. 2015, Woolcock Institute of Medical Research, Centre for Air Quality and Health Research and Evaluation (CAR).
Table 2. **Summary Health effects attributed to exposure to ambient PM** (source Hime et al, 2015)

<table>
<thead>
<tr>
<th>Long-term</th>
<th>Short-term</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>All cause (non-accidental) mortality</strong></td>
<td><strong>All cause (non-accidental) mortality</strong></td>
</tr>
<tr>
<td><strong>Cardiovascular</strong></td>
<td><strong>Cardiovascular</strong></td>
</tr>
<tr>
<td>Cardiovascular-related mortality</td>
<td>Cardiovascular-related mortality</td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>Ischaemic heart disease</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>Ischaemic stroke</td>
</tr>
<tr>
<td>Complications of diabetes</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td><strong>Respiratory</strong></td>
<td><strong>Respiratory</strong></td>
</tr>
<tr>
<td>Respiratory-related mortality</td>
<td>Respiratory-related mortality</td>
</tr>
<tr>
<td>Asthma symptoms</td>
<td>Asthma symptoms</td>
</tr>
<tr>
<td>Reduced lung function in children</td>
<td>Respiratory infections</td>
</tr>
<tr>
<td>Reduced lung function in susceptible adults (elderly, people with COPD or asthma)</td>
<td>Bronchitis in children</td>
</tr>
<tr>
<td>Respiratory infections in children</td>
<td>COPD symptoms</td>
</tr>
<tr>
<td><strong>Cancer</strong></td>
<td><strong>Cancer</strong></td>
</tr>
<tr>
<td>Lung cancer mortality</td>
<td></td>
</tr>
<tr>
<td><strong>Neurological</strong></td>
<td><strong>Neurological</strong></td>
</tr>
<tr>
<td>Neurological disorders in adults</td>
<td></td>
</tr>
<tr>
<td>Impaired cognitive function</td>
<td></td>
</tr>
<tr>
<td><strong>Development</strong></td>
<td><strong>Development</strong></td>
</tr>
<tr>
<td>Lung development</td>
<td></td>
</tr>
<tr>
<td>Neurological development in children</td>
<td></td>
</tr>
<tr>
<td><strong>Reproduction</strong></td>
<td><strong>Reproduction</strong></td>
</tr>
<tr>
<td>Adverse birth outcomes</td>
<td></td>
</tr>
<tr>
<td>Sperm quality and quantity</td>
<td></td>
</tr>
<tr>
<td><strong>Allergies</strong></td>
<td><strong>Allergies</strong></td>
</tr>
<tr>
<td>Exacerbation of allergies</td>
<td></td>
</tr>
<tr>
<td>Allergic sensitization</td>
<td></td>
</tr>
</tbody>
</table>

**Vulnerable and susceptible groups**

The studies also demonstrate that there are vulnerable and susceptible groups that are likely to be more harmed by exposure to vehicle related air pollution. The following studies were
conducted in areas of low pollution considered "safe" below cut offs (similar to Australia) and demonstrated negative impacts on health in vulnerable groups:

Air pollution effects on children

Concerning the adverse respiratory impacts of traffic pollution on children, there are a number of very important studies that are not included in the EES. Children are particularly vulnerable to vehicle emissions due to a number of factors including having a higher respiratory rate, narrower airways, larger surface areas of their lungs compared to the rest of the body, and increased exposure during school hours, their commute to and from school, and spending more time outdoors with play.14

Children are also at increased risk of developing and suffering exacerbations (triggers) of asthma with high exposure to vehicle exhaust fumes. A systematic review and meta-analysis of the literature found childhood exposure to PM$_{2.5}$ and black carbon was associated with increasing risk of subsequent asthma in childhood and allergic diseases. 15

Another meta-analysis of studies based in the US demonstrated exposure to nitrogen dioxide, nitrous oxide, and carbon monoxide were positively associated with a higher prevalence of childhood asthma. Exposure to sulfur dioxide and particulate matter PM was associated with a higher risk of wheeze in children. A meta-analysis of 19 other studies of children by Gasana et al demonstrated that increased exposure to particulate matter was associated with wheeze.16

The researchers concluded “Living or attending schools near high traffic density roads exposes children to higher levels of motor vehicle air pollutants, and increases the incidence and prevalence of childhood asthma and wheeze”.

This data is very important because there is already a higher than average incidence of childhood asthma, and asthma admissions in the surrounding residential areas and in other regions that will result in increase traffic from the freeway. Asthma pathogenesis is a complex

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mix of genetic and environmental factors. If there is already a susceptible population in the region of the proposed NEL Freeway project, it is essential that environmental factors relevant to the development of asthma, such as air pollution, are mitigated.

Another systematic review of epidemiological studies in children whose mothers were exposed to air pollution [prenatal exposure] found the child was at increased risk of developing asthma: "The overall and subgroup risk estimates from the meta-analyses showed statistically significant associations between prenatal exposures to NO₂, SO₂, and PM₁₀ and the risk of wheezing and asthma development in childhood."

The European Study of Cohorts for Air Pollution Effects (ESCAPE) project analyzed data from birth cohort studies situated in Germany, Sweden, the Netherlands, and the United Kingdom that measured lung function at 6-8 years of age (n = 5,921). The researchers found children exposed to NO₂, NOₓ, PM2.5 absorbance, and PM2.5 from air pollution traffic was associated with reduced lung function and lung development at an early age. A second ESCAPE study in 2014 showed an increase in respiratory infections in early childhood for some components of traffic pollution.

Decreases in adverse health outcomes such as hospital admissions for asthma, the prevalence of bronchitis in children and improved lung function in children, have been observed with decreases in particulate matter following major interventions to reduce air pollution.


Systemic inflammation in children

Systemic inflammation of the body can be measured by a blood test called C-Reactive Protein. This is an inflammatory marker and an Australian, Brisbane study found high levels in children aged 8-11 years caused from exposure to the ultrafine particles smaller than 0.1 μm that penetrate deeper into the lung and circulatory system\(^\text{23}\). The implications of chronic inflammation found in children are enormous as the long term effects of chronic systemic inflammation are known to contribute to a number of health conditions.

Pregnancy exposure & Low Birthweight

Data from the ESCAPE study in 2013 demonstrated that exposure to PM 2.5 during pregnancy was associated with an increased risk of low birthweight on infants born at term, even at levels of PM 2.5 less than the current EU annual limit of 25 ug/m\(^3\)\(^\text{24}\). This study concluded that “a substantial proportion of cases of low birthweight at term babies could be prevented in Europe if urban air pollution was reduced”.

Air pollution effects on elderly

In the US Medicare population from 2000 to 2012, short-term exposures to PM2.5 and warm-season ozone found elderly to be of significant risk of mortality even at levels below current national air quality standards, suggesting that these standards may need to be reevaluated\(^\text{25}\).

Those more at risk to health impacts from traffic related air pollution are cyclists and pedestrians on trails built near the proposed Freeway, children attending nearby schools, residents living adjacent to these roads and occupational workers working in close proximity to heavy traffic causing air pollution. There is no evidence within the EES that warns these population groups: residents or workers or cyclists or pedestrians of potential harms to their health from exposure of higher concentrates of air pollution that will occur with the proposed Freeway particularly in regions of 'hot spots' and in areas of higher volume of vehicle traffic.


There is evidence that exercising in traffic pollution increases the risk of cardiovascular disease so constructing a pedestrian and bike path along the proposed NEL freeway is detrimental to users of the path. A systematic review of 18 studies calculated the health impacts based on exposure-response of cyclists to air pollution and found significant health concerns were associated with exposure to ozone, black carbon or nitrogen oxides from cycling on heavily polluted roads.  

Motorists also at risk of Vehicle related Air Pollution

Not considered by the NEL EES is also the potential harm to motorists being exposed to air pollution particularly those who rely on transport for work, whose vehicles do not have appropriate air filters, and use heavily congested roads such as freeways frequently. A study found overall, car commuters had higher exposure to all pollutants than those who commuted by bus, motorcycles, followed by a car with controlled ventilation settings and those who use public transport [ie, train, subway, or metro]. A randomised double-blind study was conducted in men with a known history of stable coronary heart disease found exposure to dilute diesel (300 micrograms/m$^3$) exhaust in patients during 1 hour of moderate exercise had a direct effect on myocardial function [ECG changes], vascular, and fibrinolytic function. The study found cardiac changes ie angina with ECG changes when breathing air with a small amount of diesel exhaust compared with breathing clean filtered air.

Specific diseases caused by Air Pollution exposure:

Lung Cancer

The EES has not incorporated any studies that demonstrate that air pollution is an important cause of lung cancer. Lung cancer incidence is rising in Australia. The Australian Government state air pollution amongst other risks as a cause of lung cancer. International Agency for Research in Cancer (IARC), a division of the World Health Organisation (WHO), raised the alarm

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in 2013\textsuperscript{30}. Also in 2013, one of the studies from the European Study of Cohorts for Air Pollution Effects (ESCAPE), by Raaschou-Nielsen et al\textsuperscript{31}, showed that exposure to PM 10 and PM 2.5 increased the risk for lung cancer. A meta-analysis of 18 studies by Hamra in 2014\textsuperscript{32} confirmed the carcinogenic nature of outdoor exposure to PM 10 and PM 2.5. A second meta-analysis by Chen et al in 2015\textsuperscript{33} included 22 studies involving occupational exposure to air pollution among professional drivers. Again exposure to fine particulate matter increased the risk of lung cancer, and the risk was greater in professional drivers than in other people. IARC currently lists diesel engine exhaust and particulate matter as class 1 carcinogens. Because of the poor survival from lung cancer in Australia (< 14% of sufferers are alive after 5 years), it is essential that preventable causes, including air pollution, are minimized.

**Cardiovascular disease**

There are a number of other significant adverse health effects of traffic pollution that have not been explored or considered in the EES. Important examples are the acute and chronic cardiovascular effects from exposure to pollution\textsuperscript{34, 35}. Dennekamp et al is a study performed in Melbourne in 2010, and confirmed studies from other cities (including Perth) showing that when the ambient concentration of PM2.5 increased, there was a corresponding increase in out-of-hospital cardiac arrests. There is now a possible biological explanation for this association, with recent data from the Framingham Heart Study showing that short term

\textsuperscript{30} IARC Monograph 109, 2013.


\textsuperscript{33} Gongbo Chen,\textsuperscript{1} Xia Wan,\textsuperscript{2} Gonghuan Yang,\textsuperscript{2} and Xiaonong Zou\textsuperscript{1} *Traffic-related air pollution and lung cancer: A meta-analysis* Thorac Cancer, 2015 May; 6(3): 307–318. Published online 2015 Apr 24. doi: 10.1111/1759-7714.12185 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4448375/


exposure to air pollution generates a systemic inflammatory response, which is linked to cardiac disease\textsuperscript{36}.

**Stroke**

A major study published by Feigin et al in 2016 looked at risk factors for stroke in 188 countries. The researchers estimate that globally, nearly one third of all strokes are attributable to air pollution\textsuperscript{37}.

**In summary the evidence clearly demonstrates air pollution has the following effects:**

1. Increases morbidity and mortality especially from fine particulate air pollution ie earlier deaths from global studies performed in cities exposed to ambient air pollution\textsuperscript{38\textsuperscript{39\textsuperscript{40\textsuperscript{41}}}
2. Increase mortality associated with long-term effect of nitrogen dioxide NO\textsubscript{2} as great as that of PM\textsubscript{2.5}\textsuperscript{42}


\textsuperscript{38} Qian Di, MS; Lingzhen Dai, ScD; Yun Wang PhD; et al Antonella Zanobetti, PhD; Christine Choirat, PhD; Joel D. Schwartz, PhD; Francesca Dominici, PhD *Association of Short-term Exposure to Air Pollution With Mortality in Older Adults* JAMA. 2017;318(24):2446-2456. doi:10.1001/jama.2017.17923 [https://jamanetwork.com/journals/jama/article-abstract/2667069](https://jamanetwork.com/journals/jama/article-abstract/2667069)


3. Increased risk of mortality, particularly for the elderly, even in areas of air pollution considered at "safe levels"43. Exposure to air pollution at levels well below current quality standards is linked to an increased risk of death. Increased risk of cardiovascular disease and deaths, such as cardiac arrests and deaths, heart failure onset and mortality associated with heart failure, deep vein thrombosis [blood clots], heart disease, and myocardial infarction (heart attacks)44,45,46,47,48.

4. Increase risk of heart failure due to a possible threshold of PM$_{2.5}$=4 µg/m$^3$ is far below the daily Australian national standard of 25 µg/m$^3$49


43 Qian Di, MS; Lingzhen Dai, ScD; Yun Wang, PhD; et al Antonella Zanobetti, PhD; Christine Choirat, PhD; Joel D. Schwartz, PhD; Francesca Dominici, PhD Association of Short-term Exposure to Air Pollution With Mortality in Older Adults JAMA. 2017;318(24):2446-2456. doi:10.1001/jama.2017.17923 https://jamanetwork.com/journals/jama/article-abstract/2667069


6. Increase risk of stroke and mortality associated with stroke.\textsuperscript{30} Pope,\textsuperscript{52,53}
7. Reduced survival from long-term exposure to traffic-related air pollution and cancer among survivors of myocardial infarction (heart attacks).\textsuperscript{54}
8. Higher risk of out-of-hospital cardiac arrest associated with elevated ambient PM2.5 and CO\textsuperscript{55}
9. Reduced lung function, increased respiratory symptoms and diseases, respiratory related deaths, chronic obstructive airways disease, cardiopulmonary disease, lung infections, and asthma.\textsuperscript{30} Pope,\textsuperscript{56,57,58,59,60,61}
10. Increase risk of asthma in children.\textsuperscript{29 Bowatte et al}


\textsuperscript{55} Lahn Straney, Judith Finn, Martine Dennekamp, Alexandra Bremner, Andrew Tonkin, Ian Jacobs. Air pollution Evaluating the impact of air pollution on the incidence of out-of-hospital cardiac arrest in the Perth Metropolitan Region: 2000–2010 https://jech.bmj.com/content/68/1/6.short


\textsuperscript{57} Benjamin D, Home, Elizabeth A Joy; Michelle G Hofmarr; Per H Gesteland; John B Cannon, Jacob S Lefler, Denita P Blagev, E. Kent Korgenski, Natalie Torosyan, Grant I Hansen, David Karchner; et al. Short-term Elevation of Fine Particulate Matter Air Pollution and Acute Lower Respiratory Infection Published Online: April 13, 2018 https://doi.org/10.1164/rccm.201709-1883OC https://www.atjournal.org/doi/10.1164/rccm.201709-1883OC


11. Systemic Inflammation from exposure to ambient NO2 in Chronic Obstructive Pulmonary Disease (COPD) patients, especially in former smokers.\(^{62}\)
12. Increase risk of inflammation and cardiovascular disease in adults.\(^{63}\)
13. Carcinogenic and lung cancer risk including in non-smokers.\(^{64,65}\) Lung cancer risk occurs due to deep penetration into the lungs of the fine particulates of 2.5 microns or less from diesel or petrol exhaust fumes.\(^{66,67}\) Air pollution shortens survival from lung cancer.\(^{68}\) PM2.5 particles or less are more toxic and carcinogenic as they penetrate deeper into the lungs and vascular system of the body.
14. Poor cognition and concentration.\(^{69}\) The researchers found that long term exposure to air pollution impacted human cognitive performance, concentration, verbal and math

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ed&uac=94815EN&spon=34&implID=1753212&faf=1](https://www.medscape.com/viewarticle/902474)


\(^{67}\) Joanne Kim, Cheryl E Peters, Victoria H Arrandale et al. Burden of lung cancer attributable to occupational diesel engine exhaust exposure in Canada. [https://dx.doi.org/10.1136/oemed-2017-104950](https://dx.doi.org/10.1136/oemed-2017-104950)


\(^{69}\) Xin Zhang, Xi Chen, Xiaobo Zhang The impact of exposure to air pollution on cognitive performance PNAS published ahead of print August 27, 2018 [https://doi.org/10.1073/pnas.1809474115](https://doi.org/10.1073/pnas.1809474115) [http://www.pnas.org/content/early/2018/08/21/1809474115](http://www.pnas.org/content/early/2018/08/21/1809474115)
skills, particularly in the elderly, potentially resulting in significant health and economic costs. Multiple studies suggest air pollution contributes to cognitive impairment\textsuperscript{70,71,72}

15. Increased mortality due to residents working or residing in industrial areas of high air pollution resulting in increase cancers, lung cancer, mortality and neurological diseases\textsuperscript{73}.

16. Increase sick leave from work\textsuperscript{74}.

17. Greater healthcare utilisation by both children and adults for respiratory infections\textsuperscript{75}.

18. Increased risk of hospitalisation due to pneumonia in children\textsuperscript{76}.

19. Increased risk of type 2 diabetes and diabetes associated mortality\textsuperscript{77,78,79}.

20. Pregnancy risks -fine particulate chemicals from air pollution impact the blood vessel barrier of the vascular system of the placenta causing low birth weight, preterm birth, preterm delivery, and preterm labour.

\textsuperscript{70} Clifford, A., et al., Exposure to air pollution and cognitive functioning across the life course – A systematic literature review. Environmental Research, 2016. 147: p. 383-398.


\textsuperscript{77} Benjamin Bowe MPH, YanXie MPH, Tingting Li MD, ProfYanYan MD, ProfHongXian PhD, ZiyadAl-Aly MD. The 2016 global and national burden of diabetes mellitus attributable to PM\textsubscript{2.5} air pollution. Volume 2, Issue 7, July 2018, Pages e301-e312 The Lancet Planetary Health https://www.sciencedirect.com/science/article/pii/S2542519618301402?via%3Dihub


21. small for gestational age, and adverse birth outcomes\textsuperscript{80,81,82} and may increase stillbirths\textsuperscript{83,84}.

22. Poor sperm quality and infertility\textsuperscript{85}.

23. Non-lung cancers: PM\textsubscript{2.5} was significantly positively associated with death from cancers of the kidney and bladder. NO\textsubscript{2} was positively associated with colorectal cancer mortality\textsuperscript{86}.


\textsuperscript{81} Dirga Kumar Lamichhane, Jong-Han Leem, Ji-Young Lee, and Hwan-Cheol Kim A \textit{meta-analysis of exposure to particulate matter and adverse birth outcomes} \textit{Environ Health Toxicol}. 2015; 30: e2015011. Published online 2015 Nov 3. doi: [10.5620/eht.e2015011] \url{https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4722965/}


\textsuperscript{84} Air Pollution 'Could Raise Stillbirth Risk' May 25, 2016 Medscape \url{https://www.medscape.com/viewarticle/863844}


4. Have the potential health concerns to the local community been adequately addressed in the submission in view that by 2036, it is estimated that North East Link would carry up to 135,000 vehicles per day (page 9, chapter 9 Traffic & Transport NELP - EES)?

No I do not believe potential health concerns are addressed adequately to the local residents inhaling pollutants generated by nearly 135,000 vehicles estimated in 2036 as predicted by the NEL Project EES.

Living near a busy road can statistically increase the risk for lung cancer. One study found living near a major road with over 4000 vehicles-km per day within 100 metres of the residence increases the risk of lung cancer with a Hazard Ratio of 1.09 (9%). Exposure of PM$_{10}$ increased the Hazard ratio [HR] to 1.22 per 10 μg/m$^3$ and for PM$_{2.5}$ the HR increased to 1.18 per 5 μg/m$^3$.

The EES report has not adequately considered the enormous growth in Total Vehicle Numbers in close proximity to urban environments for some regions along the proposed Freeway region. The growth in traffic volume is sure to increase air pollutants. The research demonstrates there are real risks to human health with even low levels of air pollution that are considered "safe".

5. Do you believe the local residents have been adequately informed of the vehicle emissions and health impacts particularly those who reside or work within 250 metres of the Project?

No, I do not believe the EES has adequately informed the local residents of the possible health impacts of vehicle emissions particularly in susceptible groups living in close proximity to the freeway. Attachment B includes Key community infrastructure relative to North East Link freeway and estimated distance information is provided by the resident Michelle Giovas as the data demonstrating proximity of residents to the freeway is not available. This is a huge omission from a major project study!

87 Dr Ole Raaschou-Nielsen, PhD, Zorana J Andersen, PhD, Rob Beelen, PhD, Evangelia Samoli, PhD, Massimo Stafoggia, MSC, Gudrun Weinmayr, PhD, et al. Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE) The Lancet Oncology Volume 14, ISSUE 9, P813-822, August 01, 2013 https://www.thelancet.com/journals/lanonc/article/PIIS1470-2045(13)70279-1/fulltext
A number of health authorities highlight concerns of communities living next to or near highways or freeways\textsuperscript{88}. Outdoor workers eg road workers, traffic officers, drivers, cyclists, pedestrians and children playing outdoors are most at risk. Living or working within 200 metres of a major road increases the risk of lung disease and lung cancer.

The Tasmanian Longitudinal Health study over a 5 year period assessing Traffic Related Air Pollution (TRAP) of middle aged individuals found living within 200 metres of a major road was associated with current asthma, wheeze, and lower lung function but particularly for susceptible genetic variants in individuals\textsuperscript{89}. The mechanism is not clear. The study found exposure to air pollution led to increase asthma, wheeze and poor lung function in adults:

"Over the 5-year period, higher NO2 exposures were associated with increased current asthma prevalence. Higher NO2 exposure was associated with lower forced vital capacity for carriers of the GSTT1 null genotype. TRAP exposures were associated with increased risk of asthma, wheeze and lower lung function in middle-aged adults. ..."

Another large study as part of the ‘ESCAPE project’ consisting of 17 cohort studies involving 312,944 people living in 9 European countries found those living in close proximity less than 100 metres of a major road increased the risk of lung cancer due to increase exposure to Particulate Matter (PM) from vehicle emissions\textsuperscript{90}!

\textit{The study found:}
"The meta-analyses showed a statistically significant association between risk for lung cancer and PM\textsubscript{10} (hazard ratio [HR] 1·22 [95% CI 1·03–1·45] per 10 μg/m\textsuperscript{3}). For PM\textsubscript{2·5} the HR was 1·18 (0·96–1·46) per 5 μg/m\textsuperscript{3}. The same increments of PM\textsubscript{10} and PM\textsubscript{2·5} were associated with HRs for adenocarcinomas of the lung of 1·51 (1·10–2·08) and 1·55 (1·05–2·29), respectively. An increase in road traffic of 4000 vehicle-km per day within 100 m of the residence was associated with an HR for lung cancer of 1·09 (0·99–1·21)."

An Indoor Air Quality Study of residential dwellings that was carried out in Melbourne, Australia found that dwellings in close proximity to major roads (less than 50 metres) found high levels of indoor air pollutants\textsuperscript{91}. Independent of gas cooking, "indoor NO\textsubscript{2} was elevated in Near Road dwellings relative to Far Road dwellings by approximately 4 ppb and this can be attributed to infiltration of outdoor air." They concluded "outdoor NO\textsubscript{2}, and hence roadway emissions can potentially contribute a significant proportion to the indoor NO\textsubscript{2} concentration.... Findings elsewhere suggest that a similar outdoor enhancement of traffic related NO\textsubscript{2} (~5 ppb) increases risk of lung cancer and childhood asthma (Brauer et al., 2000; Nyberg et al., 2000)."

Living in close proximity to high traffic volume is associated with a range of adverse health outcomes

Risk to drivers; National Asthma Council concerns for air pollution

Often, we underestimate the health impact of exposure to air pollution during driving on highly polluted roads. The drivers are of closest proximity to the freeway. For example people in the transport industry who require excessive transport with use of heavily congested roads. There is increased risk of air pollutants to drivers particularly if driving with windows open and with poor car air filters.

The National Asthma Council recognise the ill effects of traffic and air pollution acting as triggers for respiratory diseases, such as asthma, and even make recommendation on which vehicles contain activated carbon particulate air filters to remove toxins and particulates from the air! They note: “Pollution – Even with improving vehicle emission standards, cars and trucks produce a significant amount of pollution. You really notice this in a busy tunnel if you have

your windows down, or your air vents on fresh-air intake. Some people experience respiratory symptoms when exposed to such emissions”.92

There does not appear within the EES to warn motorists, pedestrians, cyclists, and residents living nearby [as described above] of traffic related air pollution effects on health.

It is quite possible that the project will have significant effects on local PM 2.5 concentrations – increases or decreases depending on design, traffic flow and other factors.

**Conclusion**

The proposed freeway and attendant growth in vehicle emissions will expose local residents to greater air pollution and health risks.

I am particularly concerned that the increase traffic related air pollution will affect the health of susceptible groups; the elderly, children and pregnant women cyclists and pedestrians using the freeway trail as well as workers in the local industrial region and the wider community.

**There is strong evidence** for causation or association of traffic related air pollution with multiple adverse health outcomes, being dose dependent [even below current Government standards] with different health outcomes depending on the duration of exposure:

- **Different health outcomes** depending on **duration of exposure** - eg:
  - **Day(s), hours, minutes**: Acute asthma, heart attacks, sudden death
  - **Weeks**: Low birth weight pregnancy
  - **Chronic exposure**: Cancer, cardio-vascular and chronic respiratory diseases
  - There are multiple sources of air pollution but **traffic related air pollution** is an important contributor
  - Some people are **more susceptible** than others
  - Australian research is consistent with international studies

**Worldwide efforts by health authorities highlight concerns of communities living next to or near highways or freeways, and impacts on health** 93,94,95.

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Please find attached my scientific evidence based report and relevant appendices that highlight the concerns of air pollution from vehicle emissions on heavy congested roads and its impact to human health.

Furthermore, the freeway will attract a greater number of vehicles on the road, and this exposes the local residents to greater air borne pollution when there are regions that are currently exposed to significantly less air pollution e.g. Dingley, Waterways and Aspendale Gardens. PM2.5 is particularly of concern as the smaller mass particles are likely to remain airborne longer than the higher mass particles such as PM 10 which settle quicker.

Statement of completeness

I have made all inquiries that I believe are desirable and appropriate and no matters of significance which I regard as relevant have to my knowledge been withheld from the Panel.

For the reasons outlined above, I ask our politicians to divert the funding for this proposed NEL Freeway into affordable public extensive transport system that offer ongoing work to Victorians, such as rail links for Melbourne, to help it become a sustainable green urban environment for our future generations and children to enjoy.

Appendix A Health impacts of traffic related air pollution VKotsirilos

Appendix B Key community infrastructure relative to North East Link freeway and estimated distance

Associate Professor Vicki Kotsirilos AM, MBBS, FACNEM, FASLM, Awarded Honorary Fellowship of the Royal Australian College of General Practitioners

Acknowledgements Associate Professor Louis Irving of Royal Melbourne Hospital

93 Living Near Highways and Air Pollution | American Lung Association https://www.lung.org/our-initiatives/healthy-air/outdoor/air-pollution/highways.html


Health Impacts of Vehicle Related Air Traffic Pollution

Sustainable cities for a cleaner environment and clean air

Associate Professor (Dr) Vicki Kotsirilos AM, MBBS, FACNEM, FASLM, Hon Fellow RACGP

6th March 2019

"Every human being deserves the right to breathe clean fresh air & live a healthy life"

Member (AM) in the General Division of the Order of Australia 2016
  o for 'significant service to integrative medicine, to health practitioner standards and regulations, to medical education, and to the environment'

Australia Day Environment Award, Bayside City Council 2017
  o "Recognising outstanding and long-term leadership, action, and advocacy for coastal and natural heritage protection in the City of Bayside, and generating community awareness of the value of our Bayside environment"

Awarded Honorary Fellowship of the Royal Australian College of General Practitioners

Honorary Fellow of the Australian College of Nutritional & Environmental Medicine

Winner of the Australian General Practice Accreditation Excellence award 2007

Please note this is not a comprehensive review of the literature. There are multiple studies demonstrating harm from air pollution on human health not included in this review paper.
About the author

Associate Professor (Dr) Vicki Kotsirilos is a respected Medical, Specialist General Practitioner in Clayton, Melbourne and an Associate Professor at La Trobe University and Western Sydney University.

Dr Kotsirilos has an interest in public and environmental health. She keeps up to date with research and issues related to chronic diseases and public health issues, particularly related to lifestyle, physical activity, nutrition and the environment. In her clinical practice, Dr Kotsirilos treats patients from all age groups who suffer a wide variety of medical conditions. These include patients suffering asthma, respiratory and cardiac diseases, and cancers that are impacted by air pollution.

Dr Kotsirilos interprets and publishes research on environmental risks that may impact health and contribute to chronic diseases treated in the general practice setting. She also writes regular articles for GPs in medical publications and presents lectures at conferences.

The issues of air pollution impacting on health are well recognised by the medical profession and are major health and medical issues.

Acknowledgements

My sincere gratitude to the following for editorial assistance, and/or providing references and material to assist with preparation of this document:

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- Associate Professor Louis Irving Director Respiratory and Sleep Medicine, Royal Melbourne Hospital; Visiting Physician, Peter MacCallum Cancer Centre. Professorial Fellow Physiology, Professorial Fellow Medicine, University of Melbourne
- Dr Ben Ewald, Brunker Rd General Practice, University of Newcastle
- Clare Walter, PhD Honorary respiratory researcher, Royal Melbourne Hospital
- Associate Professor Sanjay Raghav, Neurologist
- Australasian College of Nutritional & Environmental Medicine
Sustainable cities for a cleaner environment and cleaner air

Many cities worldwide are rapidly working towards improving urban planning to help create sustainable clean cities as air pollution is widely recognised as a cause of increased mortality, air-pollution related diseases and contributing to climate change. Reducing air pollution can help save lives and reduce the rate of climate change. Air pollution is recognised by authorities as a leading cause and risk of environmental health related diseases.

Improved urban planning includes provision of more energy efficient buildings, improved high quality public transport to help reduce traffic and consequently vehicle emissions, and improved walking and cycling networks to help reduce urban pollution for immediate health benefits. There are many strategies and worldwide campaigns aiming to address global pollution and reduce urban air pollution1,2,3,4,5.

The National Clean Air Agreement

The National Clean Air Agreement was established by Australia’s environment ministers "recognising the challenges facing Australia’s current and future air quality"6. The aim of the Agreement is to address priority air quality issues through four strategic approaches: standards, emission reduction measures, partnership opportunities, and better knowledge, education and awareness.

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1 Cleaner safe air needs you! Citizen scientists are key to a big data project that will identify where the bad air lurks in our cities. By Andrew Trounson, University of Melbourne; Featured: Professor Richard Sinnott, Director, eResearch; Department of Computing and Information Systems, Melbourne School of Engineering, University of Melbourne; Clare Walter, Honorary respiratory researcher, Royal Melbourne Hospital; Oncology Pharmacist https://pursuit.unimelb.edu.au/articles/cleaner-safe-air-needs-you

2 http://breathelife2030.org/about/ [accessed 11th November 2018]


4 https://now.tufts.edu/articles/toxic-air-we-breathe [accessed 11th November 2018]


Furthermore, the objectives of the Agreement was to establish clear, internationally harmonised standards for air quality and emissions, and review Australia’s national reporting standards for sulfur dioxide, nitrogen dioxide and ozone. This requires a collaborative approach of the Australian, state and territory governments to address air quality issues on a national level. Their key goal is to create “ambient air quality that allows for the adequate protection of human health and wellbeing”.

National initiatives are in harmony with the State Government initiatives and Environmental Protection Agency (EPA) to address increasing health concerns from air pollution exposure and aim for cleaner air for all Victorians. Clean Air for All Victorians – Victoria’s Air Quality Statement commenced engagement with Victorians about future air quality management with the aim to protect air quality over the coming decades, by improving understanding of where and when air pollution occurs, reducing the occurrence of air pollution, empowering communities to tackle local air pollution issues, and tackle emerging air quality challenges.

Currently Australian air quality is superior to many other countries; consequently the issue of air pollution is often overlooked when decisions are made such as proposing to build a freeway to combat an area of traffic congestion, which ultimately attracts more vehicles resulting in increases in 'Total Vehicle Count' and moves congestion to another region, contributing to further air pollution to this area also. This consequently imposes higher traffic volumes to the new region(s) within an urban environment contributing so higher levels of air pollution experienced by the residents. There is a place for freeways in rural settings to improve access between cities or towns without impacting the health of the community.

**Even relatively low levels of air pollution have significant health impacts. According to a paper published in the Australian and New Zealand Journal of Public Health (Barnett 2014) traffic pollution is the major contributor to urban air pollution in**

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Australia, and contributes to an estimated 3,000 deaths per year\textsuperscript{12}. Barnett’s analysis of literature found that, there is no safe lower limit or level beyond which impacts do not occur from air pollution, therefore, any increase in population exposure to vehicle emissions will result in an increase in health detriments. The World Health Organisation air quality guidelines state, “there is little evidence to suggest a threshold below which no adverse health effects would be anticipated”\textsuperscript{13}. Air pollution is often difficult to measure as many pollutants interact with changes in weather and wind, so measurements even at 100 metres apart can be very different. Barnett concludes that "the epidemiology of air pollution is simple: when average levels increase, the average health effects increase, and this association has been shown repeatedly around the world".

**Environmental Protection Authority (EPA)**

The Victorian EPA recognise health concerns related to motor vehicle emissions and consequently, administer and monitor for motor vehicle emissions and air quality regularly\textsuperscript{14}.

The EPA has identified motor vehicles as being a major source of urban air pollution. In 2006 the following levels of pollutants were detected in Melbourne from motor vehicle emissions (diesel and petrol emissions) negatively impacting air quality\textsuperscript{15}:

- 72 per cent of all carbon monoxide (CO) emissions
- 70 per cent of all nitrogen oxides (NOx) emissions
- 28 per cent of all volatile organic compounds (VOC) emissions
- 31 per cent of all emissions of particles smaller than 2.5 microns (PM\textsubscript{2.5})
- 27 per cent of all emissions of particles smaller than 10 microns (PM\textsubscript{10})
- 6 per cent of all sulfur dioxide (SO\textsubscript{2}) emissions
- Nitrogen oxides (NOx) and volatile organic compounds (VOCs) can combine to form ozone (summer smog).


\textsuperscript{14} EPA, 2016. Air monitoring report 2015 – Compliance with the National Environment Protection (Ambient Air Quality) Measure, Publ 1632. 1 Oct 2016

\textsuperscript{15} Vehicle emissions and air quality https://www.epa.vic.gov.au/your-environment/air/vehicle-emissions-and-air-quality
In Australia, there are multiple sources of air pollution including industrial sources. **Traffic pollution** is an important contributor, with multiple substances and constituents contributing to poor air quality including ozone, NOx and NO₂, PM10, PM2.5 contributing to health concerns.

According to the EPA, fine particulate matter can arise from fossil fuels (such as coal), organic matter (including wood and grass) and most other materials, such as rubber and plastic.¹⁶

Motor vehicles, power plant emissions and bushfires are all major sources of fine particles.

**Particulate matters (PM) are defined as**¹⁷

“The sum of all solid and liquid particles suspended in air, many of which are hazardous. This complex mixture includes both organic and inorganic particles, such as dust, pollen, soot, smoke, and liquid droplets....”

**PM10** ‘Coarse particles’ = PM 10-2.5 micrometres (μm) in diameter which is the coarse fraction of PM10

- Please note PM10 is everything that passes through a 10 micron filter, so includes PM 2-5 which is a subset.

**PM 2.5** “ ‘Fine particles’ are those with a diameter of 2.5μm (PM2.5) or less. They are 30 times smaller than the width of hair. Particles that are smaller than 0.1μm are called **ultrafine particles**.”

- UFP = ultrafine particles
- Data on PM concentration is reported on the EPA website as a mass per volume of air – micrograms per cubic metre (µg/m³) over 1 hour or in a 24 hour period.¹⁸

See illustration below to compare sizes of particulate matters to a hair and a grain of sand:

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Environmental Protection Agency EPA monitoring for air pollution

EPA air monitors measure the amount of fine particular matter by weight (mass measurement) i.e. PM 2.5 and PM 10, and the chemical composition but not the combined total chemical load. The sum of all toxins and chemicals may be more toxic than individual chemicals, and it is important to monitor and consider for all the different gases and the combined total pollutant load.

The EPA recognises the harmful effects of air pollution even at levels well below the current air quality standards: "There is well established scientific evidence that traffic related

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air pollution, even at concentrations well below the current air quality standards, is associated with adverse health effects."

EPA monitors are located in sites that may not necessarily be at sites, or nearby, or adjacent to high traffic use or freeways\textsuperscript{20}. The monitoring effort has been to measure ambient air, defined as background air away from any point source; some monitors are located in a park or green space. Occupational drivers such as transporters who are regularly using urban freeways, cyclists using heavy polluted roads, and residents who live next to busy roadways are exposed to higher levels of air pollution than what may be detected on EPA monitors, particularly in "hot spots". Both kinds of exposures matter, but the standards are set for background ambient air. Using static monitors for measurements of PM 2.5 and 10 may not necessarily provide an accurate account of public exposure to ultrafine particulates such as PM 0.1-1.0 and as there is a high degree of spatial variability in vehicle emissions, this makes it difficult to capture "hot spots" which may be influenced by building and freeway designs and changes in weather. It is likely if appropriate monitoring was held directly at busy roads, freeways or near/adjacent freeways; emissions are likely to measure well above the EPA's annual average threshold, with nearby school children, adjacent residents, car drivers and occupational workers being those most at risk of exposure to air pollutants.

An Indoor Air Quality Study of residential dwellings that was carried out in Melbourne, Australia found that dwellings in close proximity to major roads (less than 50 metres) recorded high levels of indoor air pollutants\textsuperscript{21}. Independent of gas cooking, "\textit{indoor NO}_2\text{ was elevated in Near Road dwellings relative to Far Road dwellings by approximately 4 ppb and this can be attributed to infiltration of outdoor air."} They concluded "\textit{outdoor NO}_2\text{, and hence roadway emissions can potentially contribute a significant proportion to the indoor NO}_2\text{ concentration.... Findings elsewhere suggest that a similar outdoor enhancement of traffic related NO}_2\text{ ("5 ppb) increases risk of lung cancer and childhood asthma ( Brauer et al., 2000; Nyberg et al., 2000).}"

\begin{flushleft}
\footnotesize

\end{flushleft}
The State Environment Protection Policy for Air Quality Management (SEPP (AQM)) requires “road projects to be assessed under Part D of Schedule C, which involves modelling emissions to air from proposed major new roads. The models require, as inputs, the emission rates for various contaminants, wind and other meteorological data and background (ambient) concentrations of contaminants”\(^{22}\).

“The State Environment Protection Policy (Ambient Air Quality) lists environmental quality objectives for CO, NO\(_2\), photochemical oxidants (as ozone), sulphur dioxide (SO\(_2\)), lead and particles (as PM\(_{10}\) and PM\(_{2.5}\)), together with an additional objective for visibility reducing particles. The SEPP(AAQ) objectives apply to air quality within a region or sub-region considered to be representative of exposure of the general population in Victoria”.

The SEPP(AAQ) includes objectives for 24 hour average and annual readings:

- **PM\(_{10}\) concentrations** of 50 \(\mu\)g/m\(^3\) (24 hour average) and 20 \(\mu\)g/m\(^3\) (one year average)
- **PM\(_{2.5}\) objective** of 25 \(\mu\)g/m\(^3\) (24-hour average) and 8 \(\mu\)g/m\(^3\) (one year average).

These objectives are listed in Table 4-3 of the gazette:

<table>
<thead>
<tr>
<th>Contaminant</th>
<th>Objective</th>
<th>Averaging Period</th>
<th>Conversion to (\mu)g/m(^3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO</td>
<td>9 ppm</td>
<td>8-hours</td>
<td>10,400 (\mu)g/m(^3)</td>
</tr>
<tr>
<td>NO(_2)</td>
<td>0.12 ppm</td>
<td>1-hour</td>
<td>228 (\mu)g/m(^3)</td>
</tr>
<tr>
<td></td>
<td>0.03 ppm</td>
<td>1-year</td>
<td>57 (\mu)g/m(^3)</td>
</tr>
<tr>
<td>PM(_{10})</td>
<td>50 (\mu)g/m(^3)</td>
<td>24-hours</td>
<td>50 (\mu)g/m(^3)</td>
</tr>
<tr>
<td></td>
<td>20 (\mu)g/m(^3)</td>
<td>1-year</td>
<td>20 (\mu)g/m(^3)</td>
</tr>
<tr>
<td>PM(_{2.5})</td>
<td>25 (\mu)g/m(^3)</td>
<td>24-hours</td>
<td>25 (\mu)g/m(^3)</td>
</tr>
<tr>
<td></td>
<td>8 (\mu)g/m(^3)</td>
<td>1-year</td>
<td>8 (\mu)g/m(^3)</td>
</tr>
</tbody>
</table>

The Environmental Quality Objectives and Goals by 2025 is tabled below located within the EPA site:

**SCHEDULE 2 – ENVIRONMENTAL QUALITY OBJECTIVES AND GOAL**

Table 1: Environmental quality objectives for environmental indicators

<table>
<thead>
<tr>
<th>Column 1 Item</th>
<th>Column 2 Environmental Indicator (Pollutant)</th>
<th>Column 3 Averaging period</th>
<th>Column 4 Environmental quality objectives</th>
<th>Column 5 Maximum allowable exceedances</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Carbon monoxide (maximum concentration)</td>
<td>8 hours</td>
<td>9.0 ppm</td>
<td>1 day a year</td>
</tr>
<tr>
<td>2</td>
<td>Nitrogen dioxide (maximum concentration)</td>
<td>1 hour</td>
<td>0.12 ppm (0.03 ppm)</td>
<td>1 day a year (none)</td>
</tr>
<tr>
<td>3</td>
<td>Photochemical oxidants (as ozone) (maximum concentration)</td>
<td>1 hour 4 hours</td>
<td>0.10 ppm (0.08 ppm)</td>
<td>1 day a year (1 day a year)</td>
</tr>
<tr>
<td>4</td>
<td>Sulfur dioxide (maximum concentration)</td>
<td>1 hour 1 day 1 year</td>
<td>0.20 ppm (0.08 ppm 0.02 ppm)</td>
<td>1 day a year (1 day a year none)</td>
</tr>
<tr>
<td>5</td>
<td>Lead (maximum concentration)</td>
<td>1 year</td>
<td>0.50 µg/m³</td>
<td>none</td>
</tr>
<tr>
<td>6</td>
<td>Particles as PM&lt;sub&gt;10&lt;/sub&gt; (maximum concentration)</td>
<td>1 day 1 year</td>
<td>50 µg/m³ (20 µg/m³)</td>
<td>none (none)</td>
</tr>
<tr>
<td>6A</td>
<td>Particles as PM&lt;sub&gt;2.5&lt;/sub&gt; (maximum concentration)</td>
<td>1 day 1 year</td>
<td>25 µg/m³ (8 µg/m³)</td>
<td>none (none)</td>
</tr>
<tr>
<td>7</td>
<td>Visibility reducing particles (minimum visual distance)</td>
<td>1 hour</td>
<td>20 km</td>
<td>3 days a year</td>
</tr>
</tbody>
</table>

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The Environmental quality objectives for PM2.5 have been further revised and lowered to 7micrograms/cubic metre per year as tabled in the EPA website:

**Table 2: Environmental quality objectives for particles as PM$_{2.5}$ by 2025**

<table>
<thead>
<tr>
<th>Column 1</th>
<th>Column 2 Averaging period</th>
<th>Column 3 Environmental quality objectives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Environmental Indicator (Pollutant)</td>
<td>1 day</td>
<td>20 µg/m$^3$ by 2025</td>
</tr>
<tr>
<td>Particles as PM$_{2.5}$ (maximum concentration)</td>
<td>1 year</td>
<td>7 µg/m$^3$ by 2025</td>
</tr>
</tbody>
</table>

Recent studies demonstrate even levels below National Environment Protection (Ambient Air Quality) Measures and SEPP thresholds (as described above) are considered to cause detrimental impacts on health.

**Health impacts of air pollution**

**World Health Organisation**

The World Health Organisation (WHO) has produced a number of documents expressing grave concerns of the impact of air pollution on human health in urban environments, particularly in the face of a growing population worldwide$^{24,25,26,27}$. The WHO has also reviewed the evidence of air pollution and its adverse effects on human health.$^{28}$

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$^{24}$ WHO | Health costs due to road traffic-related air pollution. An impact assessment project of Austria, France and Switzerland [http://www.who.int/hia/examples/trspt_comms/whohia065/en/](http://www.who.int/hia/examples/trspt_comms/whohia065/en/)


In summary the WHO note:

"A wide range of adverse effects of ambient air pollution on health has been well documented by studies conducted in various parts of the world. There is significant inequality in exposure to air pollution and related health risks: air pollution combines with other aspects of the social and physical environment to create a disproportionate disease burden in less affluent parts of society."

According to WHO data, outdoor air pollution is contributing to around 3 million deaths a year worldwide. In Australia, the Australian Institute of Health and Welfare has estimated over 3000 Australian deaths each year are due to urban air pollution. These are alarmingly high number of deaths from a single preventable cause.

Social and health inequality

People residing next to freeways may suffer social and health inequality. For example, housing prices may be less as they are situated next to a freeway and exposure to daily car emissions increases health risks. Building freeways adjacent to houses that never experienced this level of pollution [emissions or noise] may be a violation of human rights if the residents had a history of breathing fresh unpolluted air and by no choice a freeway is then built next to their homes exposing their families to more background noise and vehicle related air pollution potentially causing harm to health. Air pollution is a threat to Human Rights. "Air pollution and climate change are very closely related. Taking steps to address to air pollution, if it’s done right, can also address climate change"


29 Australian Burden of Disease Study: impact and causes of illness and death in Australia 2011


There are numerous studies demonstrating noise pollution harms health not included in this review.

Annoyance caused by air pollution and noise can result in emotional and mental health problems also. Planning to build freeways adjacent to people's homes creates fear and anxiety, and this too is an important aspect of harm to mental health, not just the risk to physical health. *Every person deserves to breathe fresh air and in the latter part of this document, it explores why alternative methods of commuting must be considered by Authorities at every level of decision making, particularly at sites of green open spaces. Has the current State Government considered constructing Public transport as a priority first to address traffic congestion hot spots before planning and constructing freeways?*

As Australia's population growth occurs around cities, this will result in more vehicles and, consequently, greater emission of pollutants from vehicle emissions into the air.

**Adverse effects from Particulate Matters**
The image above demonstrates how finer particulate matters are particularly dangerous in that they penetrate deeper into the lung tissue and hence blood stream contributing to wider systemic health effects [Source of image: Dr Ben Ewald, Brunker Rd General Practice, University of Newcastle 2018].

Fine particle matters can also be emitted from various sources other than motor vehicles such as power plants and industrial processes. The smaller the particulates, the more likely they will penetrate deeper into the lungs and consequently into the circulation. These finer particulates are also transported further and persist for longer in the atmosphere.

The size of particulate matters will selectively deposit at different places in the respiratory tract system. In contrast to the larger sized particles, the smaller, ultrafine particles or fractions of PM2.5 have the ability to penetrate deeper into the lungs and enter the lymphatic system and "access the blood circulation by different transfer routes and mechanisms, resulting in distribution throughout the body, including the brain, with potential neurotoxic consequences"33. The Ultrafine Particles (UFP) may penetrate and enter the body in any route including oral, skin or by inhalation34.

Chemical composition also matters. For example, PM 2.5 from diesel exhaust is likely more toxic than PM2.5 that is silica or other benign matters.

**No safe level of air pollution**

Particulate matters are one of the main contributors to health concerns. Air pollution consisting of the finer particulate matters [less than 2.5 micrometres PM$_{2.5}$ in diameter] emitted from vehicle emissions is more likely to be associated with death and diseases. The research for this statement will be discussed throughout this document. There is no safe level of exposure or safe cut off. If we assume a baseline of no or very little exposure to particulates containing toxic matter, even small minor incremental concentrations depositing in the lungs and circulation can be harmful to the human body. Barnett's paper 12(ANZJPH 2014) highlights and reviews the science that demonstrates there is no safe lower limit of

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exposure to particulates from vehicle emissions on human health. There is now further strong data demonstrating the immediate and long term adverse effects of traffic related air pollution, highlighting a greater magnitude of impact than previously thought demonstrating there is no safe lower limit of exposure. Vehicle emissions are particularly noxious sub-set of ambient particulate matter, so no level is safe beyond which negative impacts start to occur. Any reduction in pollutants results in public health benefits.

A significant study assessing the health impacts of PM2.5 concentration at six Harvard cities in the USA with a range of air pollution involving 8096 white adults (aged 25-74 years) with acceptable pulmonary function were followed prospectively for 35 years from 1974. Potential co-variants were also accounted for in the study such as sex, education, smoking, age, Body Mass Index, diabetes, hypertension, and socio-economic factors. The first results were reported in 1993 (Dockery et al NEJM 1993) that demonstrated linear increases in all-cause mortality, and cardiovascular and lung cancer mortality. Further follow-up found as air quality levels improved the concentration-response relationship continued to demonstrate linear increases in mortality down to annual average PM 2.5 exposure of 8 ug/m$^3$ (US standard = 12 ug/m$^3$ Aus = 8 ug/m$^3$).

"Results: Since 2001, average PM$_{2.5}$ levels, for all six cities, were < 18 µg/m$^3$. Each increase in PM$_{2.5}$ (10 µg/m$^3$) was associated with an adjusted increased risk of all-cause mortality (PM$_{2.5}$ average on previous year) of 14% [95% confidence interval (CI): 7, 22], and with 26% (95% CI: 14, 40) and 37% (95% CI: 7, 75) increases in cardiovascular and lung-cancer mortality (PM$_{2.5}$ average of three previous years), respectively. The concentration–response relationship was linear down to PM$_{2.5}$ concentrations of 8 µg/m$^3$. Mortality rate ratios for PM$_{2.5}$ fluctuated over time, but without clear trends despite a substantial drop in the sulfate fraction. ...

Conclusions: These results suggest that further public policy efforts that reduce fine particulate matter air pollution are likely to have continuing public health benefits."
However despite the improvement in mean PM2.5 levels during this time in each city, there still demonstrated raised mortality with lower level of exposure.

The researchers noted in their Discussions:

"Including more recent observations with PM$_{2.5}$ exposures down to 8 µg/m$^3$, we continued to find a statistically significant association between chronic exposure to PM$_{2.5}$ and all-cause and cardiovascular mortality. Furthermore, in the present extended follow-up, PM$_{2.5}$ exposure was also statistically significantly associated with lung-cancer mortality....

... Our results indicated a statistically significant 14% increase in all-cause mortality for a 10-µg/m$^3$ annual increase in PM$_{2.5}$, which is similar to the results of the previous follow-ups (Dockery et al. 1993; Laden et al. 2006).

.....our current results are consistent with those from the ACS cohort (Pope et al. 2002), the Nurses’ Health Study (Puett et al. 2009), and the Medicare cohort (Eftim et al. 2008), which indicated mortality increases ranging from 3–26% per 10-µg/m$^3$ increase in PM$_{2.5}$.

The 26% increase in cardiovascular mortality for each 10-µg/m$^3$ increase in PM$_{2.5}$ exposure during the previous 3 years estimated in this extended follow-up is similar to the previous estimate (Laden et al. 2006)."

Di Q, Wang Y et al$^{35}$ (Di Qian, Wang Y et al. N Engl J Med 2017) study is an open cohort of all Medicare beneficiaries (60,925,443 persons) in the continental United States from the years 2000 through 2012, with 460,310,521 person-years of follow-up. It measured “annual averages of fine particulate matter (particles with a mass median aerodynamic diameter of less than 2.5 µm [PM$_{2.5}$]) and ozone were estimated according to the ZIP Code of residence for each enrollee with the use of previously validated prediction models”. The aim of the study was to estimate the risk of death associated with exposure to increases of 10 µg per cubic meter for PM$_{2.5}$ and 10 parts per billion (ppb) for ozone. 35 (Di Qian, Wang Y et al. N Engl J Med 2017).

They demonstrated increase risk of death with increases in concentration levels of exposure to PM2.5 and ozone. As illustrated in the figure below, it demonstrates that the risk of harm i.e. vertical column - "hazard ratio"= all-cause mortality [death], is dose dependent and increases with greater exposure to PM 2.5 concentration (micrograms per cubic metre) and ozone emissions (parts per billion) even below accepted national levels:
Figure 3 Concentration–Response Function of the Joint Effects of Exposure to PM$_{2.5}$ and Ozone on All-Cause Mortality A log-linear model with a thin-plate spline was fit for both PM$_{2.5}$ and ozone.\textsuperscript{35} Source https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5766848/figure/F3/?report=objectonly

These figures demonstrate increasing risk of death and harm with increasing level of exposure to PM 2.5 concentration, even at levels below the current Quality Air standards, and the Australian Government Objectives for Particles as PM$_{2.5}$ levels of 7 and 8 micrograms/cubic metre per year.\textsuperscript{23,24,37}

The researchers demonstrated that for:

"Increases of 10 μg per cubic meter in PM$_{2.5}$ and of 10 ppb (parts per billion) in ozone were associated with increases in all-cause mortality of 7.3% (95% confidence interval [CI], 7.1 to 7.5) and 1.1% (95% CI, 1.0 to 1.2), respectively. When the analysis was restricted to person-years with exposure to PM$_{2.5}$ of less than 12 μg per cubic meter and ozone of less than 50 ppb, the same increases in PM$_{2.5}$ and ozone were associated with increases in the risk of death of 13.6% (95% CI, 13.1 to 14.1) and 1.0% (95% CI, 0.9 to 1.1), respectively. For PM$_{2.5}$, the risk of death among men, blacks, and people with Medicaid eligibility was higher than that in the rest of the population."

The researchers concluded:

"In the entire Medicare population, there was significant evidence of adverse effects related to exposure to PM$_{2.5}$ and ozone at concentrations below current national standards. This effect was most pronounced among self-identified racial minorities and people with low income. (Supported by the Health Effects Institute and others.)"

Similar findings were demonstrated in an analysis of 30 cohort studies which found an overall risk estimates of cancer mortality for 10 μg/m$^3$ per increase of particulate matter (PM)$_{2.5}$, PM$_{10}$, and NO$_2$ were 1.17 (95% confidence interval (CI): 1.11–1.24), 1.09 (95% CI: 1.04–1.14), and 1.06 (95% CI: 1.02–1.10), respectively$^{38}$.  

Another significant large study gathered data from 22 European cohort studies, which created a total study population of 367,251 participants$^{39}$. They assessed residential exposure to air pollutants as annual average concentrations of particulate matter (PM) with diameters of less than 2.5 μm (PM2.5), less than 10 μm (PM10), and between 10 μm and 2.5 μm (PMcoarse), PM2.5 absorbance, and annual average concentrations of nitrogen


oxides (NO2 and NOx). They found a significant increased risk [hazard ratio (HR)] for PM2.5 of 1.07 (95% CI 1.02-1.13) per 5 μg/m(3) was recorded, even for participants exposed to pollutant concentrations lower than the European annual mean limit value of 25 μg/m(3) (HR 1.06, 95% CI 1.00-1.12) or below 20 μg/m(3) (1.07, 1.01-1.13).

They concluded:

"Long-term exposure to fine particulate air pollution was associated with natural-cause mortality, even within concentration ranges well below the present European annual mean limit value."

Therefore we should be aiming at ensuring the cleanest air possible as there is no safe level of air pollution. The guidelines need to be reviewed based on recent research.

Health impacts of Traffic related air pollution

There is strong evidence for causation or association of traffic related air pollution with multiple adverse health outcomes, being dose dependent [even below current Government standards] with different health outcomes depending on the duration of exposure.

- Cancer, cardio-vascular and chronic respiratory disease is associated with chronic exposure of traffic related air pollution
- Low birth weight pregnancy – with weeks of exposure to traffic related air pollution
- Acute asthma, heart attacks, sudden death – within day(s), hours, minutes of exposure to traffic related air pollution

• There are multiple sources of air pollution but traffic pollution is an important contributor
• Some people are more susceptible than others
• Australian research is consistent with international studies
Worldwide efforts by health authorities highlight concerns of communities living next to or near highways or freeways, and impacts on health.\(^{40,41,42}\)

The State of Environment Australian Government body have summarised the research of health impacts from air pollution ambient air quality\(^{43}\). They note the aim of "monitoring and reducing air pollution is to reduce its adverse impacts on human health. Other aims are to prevent loss of amenity—for example, because of poor visibility or offensive odour, damage to vegetation, and corrosion of buildings and other infrastructure."

The report findings were based on a number of epidemiological and exposure studies of Australia’s population that demonstrates a statistically significant relationship between air pollution impacting human health, morbidity, and mortality. Coronary heart disease, stroke, lung cancer and chronic obstructive airways disease contribute to the highest levels of mortality associated with air pollution.

Hime et al, (2015) is a comprehensive rigorous review of the literature and provides evidence demonstrating health impacts of vehicle related particulate emissions relevant to Australia\(^{44}\). Hime et al (2018) went further to compare and review the evidence of health effects associated with exposure to particulate matter (PM) air pollution comparing five common outdoor emission sources: traffic, coal-fired power stations, diesel exhaust, domestic wood combustion heaters, and crustal dust\(^{45}\).

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\(^{40}\) Living Near Highways and Air Pollution | American Lung Association https://www.lung.org/our-initiatives/healthy-air/outdoor/air-pollution/highways.html

\(^{41}\) Health Effects Institute https://www.healtheffects.org/ & https://www.healtheffects.org/air-pollution/traffic-related-air-pollution


\(^{44}\) Hime, H., C. Cowie, and G. Marks, *Review of the health impacts of emission sources, types and levels of particulate matter air pollution in ambient air in NSW*, N.E.P. Authority and E.H.B. NSW Ministry of Health, Editors. 2015, Woolcock Institute of Medical Research, Centre for Air Quality and Health Research and Evaluation (CAR).

The researchers found investigation of traffic alone published in hundreds of publications on the health effects of PM from vehicle emission sources and on comparison found "there is no clear hierarchy in the impact that PM$_{2.5}$ from different emission sources has on mortality risk, there is a suggestion that PM$_{2.5}$ from traffic and coal-fired power stations have a greater mortality impact, especially in relation to cardiovascular diseases." These findings are well illustrated in the Forest Plot below which demonstrate exposure to PM$_{2.5}$ increases the risk of all-cause and cardiovascular mortality with increase exposure$^{45}$ (Hime et al, 2018) (next page):
The researchers found exposure to traffic related air pollution\textsuperscript{39}:

- reduced lung function in children,
- caused exacerbation and onset of asthma,
- contributed to a range of respiratory symptoms,
- impaired lung function,
- increased risk of all-cause mortality and cardiovascular morbidity, myocardial infarction, hypertension,
- contributed to allergic sensitisation, and
- premature birth.

Below is extracted from Table 3 summarising the evidence from Total traffic-related air pollution, specifically traffic PM (Particulate Matter) emissions, and Diesel exhaust:

### Summary of PM emission sources and reported health and physiological/toxicity effects

<table>
<thead>
<tr>
<th>Emission Source</th>
<th>Health Risk and Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total traffic-related air pollution (TRAP)</td>
<td>all-cause, respiratory and cardiovascular mortality, cardiovascular, stroke and heart failure morbidity [54, 55, 56, 57, 58, 70, 71, 72] cardiovascular toxicity and various cardiovascular effects [50, 60] cytotoxicity, pulmonary inflammation [62, 63]</td>
</tr>
<tr>
<td>Coal-fired power stations</td>
<td>respiratory mortality [53] lung and oesophageal cancer mortality [84, 85] allergic inflammation, asthma symptoms, lung cancer [79, 81, 82, 83] cardiovascular morbidity [72, 89] cardiovascular changes indicative of increased coronary event risk, changes in lung function, nose and throat irritation [18, 49, 90] atopy and susceptibility to infection [98, 99, 100] effects on offspring from exposure during pregnancy [101, 102, 103]</td>
</tr>
</tbody>
</table>
Motor vehicles are one of the largest contributors to urban air pollution, emitting significant quantities of toxins that include carbon dioxide, carbon monoxide, hydrocarbons, nitrogen oxides, and particulate matters. The health concerns on humans resulting from vehicle emissions have been extensively studied with well in excess of 100 published papers in Australia and Internationally with equivalent exposure to vehicle air pollution as Australians\textsuperscript{46,47,48,49}.

Vulnerable and susceptible groups

The elderly, pregnant women, the unborn child, children and unwell individuals with pre-existing lung diseases (e.g. asthma or chronic obstructive airways or lung disease) or heart diseases are particularly susceptible to urban air pollution and the ill-effects of daily exposure to increased levels of fine particulate matter (PM$_{2.5}$).

The following studies were conducted in areas of pollution levels similar to Australia and demonstrated negative health impacts particularly on vulnerable groups:

Air pollution effects on elderly

In the US Medicare population from 2000 to 2012, short-term exposures to PM2.5 and warm-season ozone found elderly to be of significant risk of mortality even at levels below current national air quality standards\textsuperscript{50}.


\textsuperscript{48} ISEE Young 2018, Early Career Researchers Conference on Environmental Epidemiology – Together for a Healthy Environment, 19–20 March 2018, Freising, Germany Table of contents | Occupational & Environmental Medicine \url{http://oem.bmj.com/content/75/Suppl_1}


Air pollution effects on children

Children are particularly vulnerable to vehicle emissions due to a number of factors that include having a higher respiratory rate, narrower airways, larger surface areas of their lungs compared to the rest of the body, increased exposure during school hours especially if the school is located near high traffic density roads, their commute to and from school, and spending more time outdoors with play (12 Barrett),51.

Asthma in children

Children are also at increased risk of developing and suffering exacerbations or triggers of asthma with exposure to vehicle exhaust fumes. A systematic review and meta-analysis of the literature found childhood exposure to PM$_{2.5}$ and black carbon was associated with increasing risk of subsequent childhood asthma and allergic diseases52.

Another meta-analysis of studies based in the US demonstrated exposure to nitrogen dioxide, nitrous oxide, and carbon monoxide were positively associated with a higher prevalence of childhood asthma. Exposure to sulfur dioxide and particulate matter PM was associated with a higher risk of wheeze in children. The researchers concluded “Living or attending schools near high traffic density roads exposes children to higher levels of motor vehicle air pollutants, and increases the incidence and prevalence of childhood asthma and wheeze” 53.

An Australian study found exposure to NO2 increased the risk of asthma in children:54

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The study findings:

Most studies of long-term air pollution exposure and children’s respiratory health have been performed in urban locations with moderate pollution levels. We assessed the effect of outdoor nitrogen dioxide (NO$_2$), as a proxy for urban air pollution, on current asthma and lung function in Australia, a low-pollution setting. We undertook a national population-based cross-sectional study of children aged 7-11 years living in 12 Australian cities. We collected information on asthma symptoms from parents via questionnaire and measured children’s lung function (forced expiratory volume in 1 s [FEV$_1$], forced vital capacity [FVC]) and fractional exhaled nitric oxide [FeNO]). We estimated recent NO$_2$ exposure (last 12 months) using monitors near each child’s school, and used a satellite-based land-use regression (LUR) model to estimate NO$_2$ at each child’s school and home. Our analysis comprised 2630 children, among whom the prevalence of current asthma was 14.9%. Mean (±SD) NO$_2$ exposure was 8.8 ppb (±3.2) and 8.8 ppb (±2.3) for monitor- and LUR-based estimates, respectively. Mean percent predicted post-bronchodilator FEV$_1$ and FVC were 101.7% (±10.5) and 98.8% (±10.5), respectively. The geometric mean FeNO concentration was 9.4 ppb (±7.1). An IQR increase in NO$_2$ (4.0 ppb) was significantly associated with increased odds of having current asthma; odds ratios (ORs) were 1.24 (95% CI: 1.08, 1.43) and 1.54 (95% CI: 1.26, 1.87) for monitor- and LUR-based estimates, respectively. Increased NO$_2$ exposure was significantly associated with decreased percent predicted FEV$_1$ (-1.35 percentage points [95% CI: -2.21, -0.49]) and FVC (-1.19 percentage points [95% CI: -2.04, -0.35], and an increase in FeNO of 71% (95% CI: 38%, 112%). Exposure to outdoor NO$_2$ was associated with adverse respiratory health effects in this population-based sample of Australian children. The relatively low NO$_2$ levels at which these effects were observed highlight the potential benefits of continuous exposure reduction.

Prenatal exposure

Another systematic review of epidemiological studies in children whose mothers were exposed to air pollution ie prenatal exposure, found these children were at higher risk of developing asthma\textsuperscript{55}. The researchers concluded:

"The overall and subgroup risk estimates from the meta-analyses showed statistically significant associations between prenatal exposures to NO\textsubscript{2}, SO\textsubscript{2}, and PM\textsubscript{10} and the risk of wheezing and asthma development in childhood."

Reduced lung function in children exposed to air pollution

A study extracted from the European Study of Cohorts for Air Pollution Effects (ESCAPE project) from cohort studies situated in Germany, Sweden, the Netherlands, and the United Kingdom measured lung function in 5,921 children at 6-8 years of age and found children exposed to NO\textsubscript{2}, NO\textsubscript{x} and PM\textsubscript{2.5} from air pollution traffic was associated with reduced lung function\textsuperscript{56}.

Mitigation of air pollution improves lung function

On a positive note, children's lungs can heal. Community efforts to improve air quality and reduce air pollution interventions can reduce prevalence of lung disease, bronchitis and respiratory hospital admissions in children\textsuperscript{57,58}. According to the Organisation for Economic

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\textsuperscript{58} Heinrich J. (2003). Nonallergic respiratory morbidity improved along with a decline of traditional air pollution levels: A review. European Respiratory Journal 21(Supplement 40): 64S-69s. https://pdfs.semanticscholar.org/381c/dc4f6e305a8b3459c5eccdce05a842410b1a.pdf
Co-operation and Development (OECD) mitigation of air pollution is the pillar to reducing the impacts of traffic pollution and is associated with significant health improvements\(^59\).

The most notable study to date demonstrated significant improvement in lung function in children \((n= 2120)\) over 3 cohort study periods in Southern California corresponding to the time periods 1994 – 1998, 1997 – 2001 and 2007- 2011\(^60\). Declining levels of nitrogen dioxide and PM due to a range of mitigation strategies were associated with significant improvements in lung function. Southern California experienced high levels of air pollution due to large motor-vehicle fleet, numerous industries, the largest seaport complex in the United States, and a natural landscape that traps polluted air over the Los Angeles basin. Due to the growing body of scientific evidence demonstrating the adverse health effects of air pollution, aggressive pollution-reduction policies were implemented. These strategies included control pollution from mobile and stationary sources, as well as fuel and consumer-product reformulations. These positive changes correlated with improvement in children’s health. The proportion of children with clinically low lung function \(\text{(FEV1)}\) declined from 7.9% to 6.3% to 3.6% across the three time periods as air quality improved during the study periods \((p = 0.001)\). The benefits of improved lung development in children extended throughout their lives. These findings are encouraging.

**Systemic inflammation in children**

A study based in metropolitan region of Brisbane, Australia, researchers demonstrated children aged 8-11 years of age have raised systemic inflammation of the body including raised C-Reactive Protein \([\text{an inflammatory marker found on pathology blood testing}]\) caused from chronic ambient exposure to air pollution thought to be due to the UFPs \((\text{ultrafine particles})\) smaller than 0.1 \(\mu\text{m}\) that penetrate


deeper into the lung and circulatory system\textsuperscript{61}. The implications of chronic inflammation found in children are enormous as the long term effects of chronic systemic inflammation are known to contribute to a number of health conditions including cardiovascular disease.

**Pregnancy and low birthweight in newborns**

The European Study of Cohorts for Air Pollution Effects (ESCAPE) project collated and analyzed data from birth cohort studies involving 14 cohorts in 12 European countries. The study population included 74,178 women who had singleton deliveries between 1994 and 2011\textsuperscript{62}. The regions assessed in the ESCAPE project are similar to Australian regions with equivalent levels of exposure by inhabitants to air pollution. **The researchers found exposure to air pollution increased the risk of low birthweight.** They demonstrated an adjusted Odds Ratio (OR) of 1.18 (1.06-1.33) per 5ug PM2.5 for birth weight less than 2500g in full term babies born after 37 weeks in the full cohort. The risk however was higher at OR 1.41 (1.20-1.65) in the subset with exposure less than the European standard at the time of 20ug/m\textsuperscript{3}. Please note Australian exposure values are similar eg less than 20 ug/m\textsuperscript{3}.

The study found:

"A 5 μg/m\textsuperscript{3} increase in concentration of PM2·5 during pregnancy was associated with an increased risk of low birthweight at term (adjusted odds ratio [OR] 1·18, 95% CI 1·06-1·33). An increased risk was also recorded for pregnancy concentrations lower than the present European Union annual PM2·5 limit of 25 μg/m(3) (OR for 5 μg/m(3) increase in participants exposed to concentrations of less than 20 μg/m(3) 1·41, 95% CI 1·20-1·65). PM10 (OR for 10 μg/m(3) increase 1·16, 95% CI 1·00-1·35), NO2 (OR for 10 μg/m(3) increase 1·09, 1·00-1·19), and traffic density on nearest street (OR for increase of 5000 vehicles per day 1·06, 1·01-1·11) were also associated with increased risk of low birthweight at term. The population attributable risk estimated for a reduction in PM2·5 concentration to 10 μg/m(3) during pregnancy corresponded to a decrease of 22% (95% CI 8-33%) in cases of low birthweight at term."


Interpretation: Exposure to ambient air pollutants and traffic during pregnancy is associated with restricted fetal growth. A substantial proportion of cases of low birthweight at term could be prevented in Europe if urban air pollution was reduced."

Reducing air pollution, increases birth weight

Low Birth Weight is a good measure to assess the benefits of mitigating and reducing air pollution exposure, as the benefits are demonstrable within months, while for cardiovascular disease and deaths for reducing air pollution may be delayed by years. The most convincing immediate evidence demonstrating benefits of reducing air pollution on fetal growth and improved birth weight occurred in China during the Beijing Olympics in 2008. Beijing is a polluted city. Motor vehicles were heavily restricted, and many industries including four coal fired power stations were closed for the duration of the Olympics. Comparison of the weights of babies born during the clean air weeks, compared to the same weeks of high air pollution in 2007 and 2009 showed an average 23 grams of birth weight increase. There was no other plausible explanation(s) for differences in birth weight other than differences in levels of air pollution exposure of pregnant mothers and prenatal exposure.

Disease risk from air pollution

All-cause mortality

The American Thoracic Society recognises the harmful effects and resulting increased mortality associated with exposure to air pollution since 1996.

The Society note there is increased risk of mortality and morbidity with higher exposure to air pollution:"Combined effects were estimated as percent increase in comparable measures of mortality and morbidity, associated with each 10 micrograms/m3 increase in daily mean PM10 exposure ..). While total mortality increased by 1% for each 10 micrograms/m3 increase in PM10, respiratory mortality increased by 3.4% and cardiovascular mortality increased by 1.4%. Hospital admissions and emergency


department visits increased approximately 1% for all respiratory complaints, and 2% to 3% for asthma. Exacerbation of asthma increased by about 3%, as did lower respiratory symptoms. Small decreases in lung function, approximately 0.1%, have also been observed. This review suggests that the epidemiologic studies of adverse morbidity measures are coherent with the mortality studies showing quantitatively similar adverse effects of acute exposures to particulate pollution."

One of the earliest and biggest cohort studies over a 6 year period published and organised by the American Cancer Society enrolled a cohort of 1.2 million people in 1982. Of the cohort assessed, 400,000 people were living in cities with well measured air. This study led to further cohort studies with the aim to estimate the risk of air pollution still used today. The studies made every effort to exclude confounding factors such as smoking, employment, race, alcohol, diet etc through a carefully worded questionnaire to exclude other causes of variable factors contributing to mortality. The study explored long term exposure to combustion related particulate matter and found it was associated with increased risk mortality, lung cancer and cardiopulmonary mortality:

"Fine particulate and sulfur oxide--related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10-microg/m³ elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality."

The findings of this study are well illustrated in these diagrams which demonstrate increase mortality and morbidity with exposure to PMs well below accepted “safe” levels. Pope et al JAMA 2002

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How to read these graphs: Each chart has a solid line which is the estimate of the effect on the disease outcome (Vertical axis) from each amount of PM2.5 air pollution (Horizontal axis). The dashed lines are the 95% confidence intervals showing the degree of statistical certainty. A flat line shows no relationship between the exposure and mortality, while a line that slopes up to the right shows increasing deaths with increasing air pollution.

A European study of 71,362 residents living in the area of Civitavecchia, Italy were followed up from 1996 till 2013 and found an association of long term exposure to NO\textsubscript{x} from traffic emissions increased the incidence from all cancers (HR=1.13, 95% CI 1.01 to 1.26) and neurological diseases (HR=1.50, 95% CI 1.01 to 2.20).

The authors concluded

"Estimated exposures to different pollution sources in this area were independently associated with several mortality outcomes while adjusting for occupation and socioeconomic status\textsuperscript{66}."

\textsuperscript{66} Lisa Bauleo, Simone Bucci, Chiara Antonucci, Roberto Sozzi, Marina Davoli, Francesco Forastiere, Carla Ancona. \textit{Long-term exposure to air pollutants from multiple sources and mortality in an industrial area: a cohort study} Occup Environ Med
Cancers from air pollution

This large international study demonstrated from an analysis of 30 cohort studies an overall risk estimates of cancer mortality for 10 µg/m³ per increase of particulate matter (PM)_{2.5}, PM_{10}, and NO₂ - 1.17 (95% confidence interval (CI): 1.11–1.24), 1.09 (95% CI: 1.04–1.14), and 1.06 (95% CI: 1.02–1.10), respectively.⁶⁷

“With respect to the type of cancer, significant hazardous influences of PM_{2.5} were noticed for lung cancer mortality and non-lung cancer mortality including liver cancer, colorectal cancer, bladder cancer, and kidney cancer, respectively, while PM_{10} had harmful effects on mortality from lung cancer, pancreas cancer, and larynx cancer. Our meta-analysis of cohort studies indicates that exposure to the main air pollutants is associated with increased mortality from all cancers.”

Cardiovascular disease

There are a number of studies linking air pollution to increase risk of cardiovascular disease. A systematic and meta-analysis of the literature identified 15 studies and found exposure to PM_{10}, PM_{2.5}, NO₂ and O₃ →↑ risk of Outer Hospital Cardiac Arrest risk (OHCA)⁶⁸. The individual risks were: PM_{10} 1.021, 95%CI: 1.006-1.037; PM_{2.5} 1.041, 95%CI: 1.012-1.071; NO₂ 1.015, 95%CI: 1.001-1.030; O₃ 1.016, 95%CI: 1.008-1.024). Population attributable fractions for PM_{10}, PM_{2.5}, and O₃ were 2.1%, 3.9% and 1.6%, respectively.

The researchers concluded that the associations between short-term exposure to PM_{2.5}, PM_{10} and ozone increased the risk of OHCA with the strongest association being observed for PM_{2.5}.

More studies are listed below from page 41 and in Appendix 1.

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Diesel exhaust – cardiovascular disease & cancer risk

Diesel exhaust inhalation promotes oxidative stress that can be directly associated with adverse cardiovascular effects and is a known carcinogen. Oxidative stress has emerged as the likely plausible mechanism by which inhalation of diesel exhaust PM leads to multiple facets of cardiovascular dysfunction. Diesel vehicle emissions contain much higher number of particles compared with petrol emissions and are also recognised as a major risk factor for health concerns. Diesel is a known group 1 carcinogen.

Diesel exhaust inhalation in men during exercise

Deep breathing occurs with exercise and this leads to deeper penetration of PMs and toxins whilst being exposed to vehicle emissions. Inhalation during exercise of Diesel combustion fumes can increase the risk of myocardial ischaemia. A randomised double-blind study was conducted in men with a known history of stable ischaemic coronary heart disease to determine if controlled exposure to dilute diesel (300 micrograms/m³) exhaust during 1 hour of moderate exercise had a direct effect on myocardial function [ECG changes], vascular, and fibrinolytic function. The study found cardiac changes when breathing air contaminated with even small amounts of diesel exhaust compared with breathing clean filtered air resulted in ST segment changes on ECG trace implying the heart muscle is suffering greater stress on its oxygen supply. This experiment supports the epidemiological evidence of increased heart attacks on poor quality air days. It also demonstrates toxicity from diesel exhaust fumes.

---


The top trace is participant's heart rate, which rises from 60 at baseline to about 90 while exercising. The bottom trace is the changes in the ST segment on their ECG trace.

The Mills study concludes:

"Brief exposure to dilute diesel exhaust promotes myocardial ischemia and inhibits endogenous fibrinolytic capacity in men with stable coronary heart disease. Our findings point to ischemic and thrombotic mechanisms that may explain in part the observation that exposure to combustion-derived air pollution is associated with adverse cardiovascular events."

Asthma and impaired lung function in adults

Studies have demonstrated that short and long term exposure to outdoor particulate air pollution impacts lung function in healthy adults\(^\text{72}\).

\(^{72}\) Stefan Edginton, Dylan E O'Sullivan, Will King, M Diane Lougheed, *Effect of outdoor particulate air pollution on FEV\(_1\) in healthy adults: a systematic review and meta-analysis* http://dx.doi.org/10.1136/oemed-2018-105420
The study findings:

"The effect of acute and long-term exposures to outdoor particulate air pollution on lung function in healthy adults is not well established. The objective of this study was to conduct a systematic literature review and meta-analysis of studies that assessed the relationship of outdoor particulate air pollution and lung function in healthy adults. Studies that contained data on outdoor air particulate matter levels (PM$_{10}$ or PM$_{2.5}$) and forced expiratory volume in 1 s (FEV$_1$) in healthy adults were eligible for inclusion. Effect estimates, in relation to long-term and acute exposures, were quantified separately using random effects models. A total of 27 effect estimates from 23 studies were included in this review. Acute exposures were typically assessed with PM$_{2.5}$, while long-term exposures were predominantly represented by PM$_{10}$. A 10 µg/m$^3$ increase in short-term PM$_{2.5}$ exposure (days) was associated with a $-7.02$ mL (95% CI $-11.75$ to $-2.29$) change in FEV$_1$. A 10 µg/m$^3$ difference in long-term PM$_{10}$ exposure was associated with a $-8.72$ mL (95% CI $-15.39$ to $-2.07$) annual change in FEV$_1$ and an absolute difference in FEV$_1$ of $-71.36$ mL (95% CI $-134.47$ to $-8.24$). This study provides evidence that acute and long-term exposure to outdoor particulate air pollution are associated with decreased FEV$_1$ in healthy adults. Residual confounding from other risk factors, such as smoking, may explain some of the effect for long-term exposures. More studies are required to determine the relationship of long-term exposure to PM$_{2.5}$ and short-term exposure to PM$_{10}$, which may have different biologic mechanisms."

Interesting the Tasmanian Longitudinal Health study over a 5 year period assessing Traffic Related Air Pollution (TRAP) of middle aged individuals found living within 200 metres of a major road was associated with current asthma, wheeze, and lower lung function but particularly for susceptible genetic variants in individuals$^{73}$. The mechanism is not clear. The study found exposure to air pollution led to increase asthma, wheeze and poor lung function in adults:

"Over the 5-year period, higher NO$_2$ exposures were associated with increased current asthma prevalence. Higher NO$_2$ exposure was associated with lower forced vital capacity for carriers of the GSTT1 null genotype. TRAP exposures were associated with increased risk of asthma, wheeze and lower lung function in middle-aged adults. The interaction with

the GSTT1 genotype suggests that deficient antioxidant mechanisms may play a role in these adverse health effects.

Lung cancer risk from air pollution exposure

According to research, Australian urban air pollution contributes to increased risk of lung cancer with a lag time from exposure to presentation of disease of about 15-30 years\textsuperscript{74}.

A systematic review gave a quantitative summary of the relationship between outdoor PM 2.5 and PM10 and lung cancer. It found a relative increase in life-time risk for lung cancer higher in smokers, and specific sub-type of lung cancer Adenocarcinoma risk associated with PM2.5 and PM10 were 1.40 (95% CI: 1.07, 1.83) and 1.29 (95% CI: 1.02, 1.63), respectively.\textsuperscript{75}

To put this simply, this is significant finding and equates to a 40% increased risk (relative to the current risk) of developing lung adenocarcinoma over the course of 60 years! The authors classify PM and outdoor air pollution as a Class 1 cause of lung cancer.

\textit{See Forest Plot 1. Relative risks for lung cancer associated with a 10µg/m\textsuperscript{3} increase of PM2.5 (A) and PM10 (B).}\textsuperscript{68} (Hamra et al 2014)


### A

#### Study by region

<table>
<thead>
<tr>
<th>Country</th>
<th>RR (95% CI)</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>North America</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>McDonell et al. 2000</td>
<td>1.39 (0.79, 2.46)</td>
<td>0.86</td>
</tr>
<tr>
<td>Krewski et al. 2009</td>
<td>1.09 (1.05, 1.13)</td>
<td>21.19</td>
</tr>
<tr>
<td>Hart et al. 2011</td>
<td>1.18 (0.95, 1.48)</td>
<td>3.77</td>
</tr>
<tr>
<td>Lipsett et al. 2011</td>
<td>0.95 (0.70, 1.28)</td>
<td>2.22</td>
</tr>
<tr>
<td>Lupeule et al. 2012</td>
<td>1.37 (1.07, 1.75)</td>
<td>3.20</td>
</tr>
<tr>
<td>Hystad et al. 2013</td>
<td>1.29 (0.95, 1.76)</td>
<td>2.14</td>
</tr>
<tr>
<td>Jerrett et al. 2013*</td>
<td>1.12 (0.91, 1.37)</td>
<td>—</td>
</tr>
<tr>
<td>Puett et al. 2014</td>
<td>1.06 (0.90, 1.24)</td>
<td>6.48</td>
</tr>
<tr>
<td><strong>Subtotal (I^2 = 0.0%, p = 0.490)</strong></td>
<td>1.11 (1.05, 1.16)</td>
<td>39.67</td>
</tr>
<tr>
<td><strong>Europe</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beelen et al. 2008</td>
<td>0.81 (0.63, 1.04)</td>
<td>3.11</td>
</tr>
<tr>
<td>Carey et al. 2013</td>
<td>1.11 (0.86, 1.43)</td>
<td>3.07</td>
</tr>
<tr>
<td>Cesaroni et al. 2013</td>
<td>1.05 (1.01, 1.10)</td>
<td>20.21</td>
</tr>
<tr>
<td>Raaschou-Nielsen et al. 2013</td>
<td>1.39 (0.91, 2.13)</td>
<td>1.17</td>
</tr>
<tr>
<td><strong>Subtotal (I^2 = 50.0%, p = 0.112)</strong></td>
<td>1.03 (0.89, 1.20)</td>
<td>27.56</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cao et al. 2011</td>
<td>1.03 (1.00, 1.07)</td>
<td>21.25</td>
</tr>
<tr>
<td>Katanoda et al. 2011</td>
<td>1.24 (1.12, 1.37)</td>
<td>11.52</td>
</tr>
<tr>
<td><strong>Subtotal (I^2 = 91.0%, p = 0.001)</strong></td>
<td>1.13 (0.94, 1.34)</td>
<td>32.77</td>
</tr>
<tr>
<td><strong>Overall (I^2 = 53.0%, p = 0.010)</strong></td>
<td>1.09 (1.04, 1.14)</td>
<td>100.00</td>
</tr>
</tbody>
</table>

### B

#### Study by region

<table>
<thead>
<tr>
<th>Country</th>
<th>RR (95% CI)</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>North America</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beeson et al. 1998</td>
<td>1.16 (1.02, 1.32)</td>
<td>12.18</td>
</tr>
<tr>
<td>Pope et al. 2002</td>
<td>0.98 (0.95, 1.01)</td>
<td>17.84</td>
</tr>
<tr>
<td>Hart et al. 2011</td>
<td>1.08 (0.91, 1.30)</td>
<td>9.12</td>
</tr>
<tr>
<td>Lipsett et al. 2011</td>
<td>0.93 (0.81, 1.07)</td>
<td>11.44</td>
</tr>
<tr>
<td>Puett et al. 2014</td>
<td>1.04 (0.95, 1.14)</td>
<td>14.55</td>
</tr>
<tr>
<td><strong>Subtotal (I^2 = 57.7%, p = 0.051)</strong></td>
<td>1.02 (0.98, 1.05)</td>
<td>65.12</td>
</tr>
<tr>
<td><strong>Europe</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carey et al. 2013</td>
<td>1.03 (0.88, 1.21)</td>
<td>10.16</td>
</tr>
<tr>
<td>Heinrich et al. 2013</td>
<td>2.39 (1.35, 4.22)</td>
<td>1.66</td>
</tr>
<tr>
<td>Raaschou-Nielsen et al. 2013</td>
<td>1.22 (1.03, 1.45)</td>
<td>9.60</td>
</tr>
<tr>
<td><strong>Subtotal (I^2 = 76.5%, p = 0.014)</strong></td>
<td>1.27 (0.96, 1.68)</td>
<td>21.42</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hales et al. 2012</td>
<td>1.16 (1.04, 1.29)</td>
<td>13.45</td>
</tr>
<tr>
<td><strong>Overall (I^2 = 74.6%, p = 0.000)</strong></td>
<td>1.08 (1.00, 1.17)</td>
<td>100.00</td>
</tr>
</tbody>
</table>
Proximity to high traffic air pollution

Living in close proximity to high traffic volume is associated with a range of adverse health outcomes

The European Study of Cohorts for Air Pollution is a meta-analysis of data from 17 cohort studies consisting of 312,944 people from nine European countries and found increase exposure to Particulate Matter (PM) from vehicle emissions causes a significant risk of lung cancer among people living within 100 metres of a major road.76

"The meta-analyses showed a statistically significant association between risk for lung cancer and PM$_{10}$ (hazard ratio [HR] 1·22 [95% CI 1·03–1·45] per 10 μg/m$^3$). For PM$_{2.5}$ the HR was 1·18 (0·96–1·46) per 5 μg/m$^3$. The same increments of PM$_{10}$ and PM$_{2.5}$ were associated with HRs for adenocarcinomas of the lung of 1·51 (1·10–2·08) and 1·55 (1·05–2·29), respectively. An increase in road traffic of 4000 vehicle-km per day within 100 m of the residence was associated with an HR for lung cancer of 1·09 (0·99–1·21)."

Active commuters eg pedestrians and cyclists exposed to air pollution

A systematic review of 39 studies compared exposure to carbon monoxide, black carbon, nitrogen dioxide, and fine and coarse particles in active commuters (pedestrians or cyclist) and commuters using motorised transport (car, motorcycle, bus, or massive motorised transport [MMT-ie, train, subway, or metro]).77

The study found overall, car commuters had higher exposure to all pollutants than did active commuters, followed by those who commuted by bus, then motorcycle, followed by a car with controlled ventilation settings and by MMT.

76 Dr Ole Raaschou-Nielsen, PhD, Zorana J Andersen, PhD, Rob Beelen, PhD, Evangelia Samoli, PhD, Massimo Stafoggia, MSc, Gudrun Weinmayr, PhD, et al. Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE) The Lancet Oncology Volume 14, ISSUE 9, P813-822, August 01, 2013 Published:July 10, 2013DOI: https://doi.org/10.1016/S1470-2045(13)70279-1
https://www.thelancet.com/journals/lanonc/article/PIIS1470-2045(13)70279-1/fulltext

However, active commuters (pedestrian and cyclists) had higher inhalation doses of pollutants than other forms of commuters using motorised transport, car, motorcycle, MMT, and bus. It is likely that with exercise increase respiratory rate would promote deeper penetration of fine particulate matter into the lungs and vascular system.

A systematic review of 18 studies calculated the health impacts based on exposure-response of cyclists to air pollution found larger health concerns were associated with exposure to ozone, black carbon or nitrogen oxides.78

Risk to drivers; National Asthma Council concerns for air pollution

Often, we underestimate the health impact of exposure to air pollution during driving on highly polluted roads. For example people in the transport industry who require excessive transport with use of heavily congested roads. There is increased risk of air pollutants to drivers particularly if driving with windows open and with poor car air filters.

The National Asthma Council recognise the ill effects of traffic and air pollution acting as triggers for respiratory diseases, such as asthma, and even make recommendation on which vehicles contain activated carbon particulate air filters to remove toxins and particulates from the air! They note: “Pollution – Even with improving vehicle emission standards, cars and trucks produce a significant amount of pollution. You really notice this in a busy tunnel if you have your windows down, or your air vents on fresh-air intake. Some people experience respiratory symptoms when exposed to such emissions”79.

Summary evidence and causal association of health problems related to air pollution

There is clear evidence that air pollution due to vehicle emissions is a cause of a number of health problems including increased risk of mortality. Harm to human health occurs at levels below what is accepted as “safe” cut off levels. Air pollution, even at low levels contributes to and/or causes a number of health problems especially for individuals living and working near freeways. Studies conducted in Europe, the United States and Australia ie in areas of equivalent air pollution experienced in cities of Australia are described throughout this document even when considered "safe" thresholds demonstrating detrimental impacts on


Recent studies demonstrate even levels below National Environment Protection (Ambient Air Quality) Measures and SEPP thresholds (as described above) are considered to cause detrimental impacts on health.

There are also a number of international studies highlighting the risks of rising air pollution in more congested cities than Australian cities, which Australia may face one day with increasing population and the number of vehicles on the road.

Below is a summary of potential health effects from short and long term exposure to ambient PM exposure.

Table 2. Summary Health effects attributed to exposure to ambient PM (source Hime et al, 2015)

<table>
<thead>
<tr>
<th>Long-term</th>
<th>Short-term</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>All cause (non-accidental) mortality</strong></td>
<td><strong>All cause (non-accidental) mortality</strong></td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Cardiovascular</td>
</tr>
<tr>
<td>Cardiovascular-related mortality</td>
<td>Cardiovascular-related mortality</td>
</tr>
<tr>
<td>Atherosclerosis</td>
<td>Ischaemic heart disease</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>Ischaemic stroke</td>
</tr>
<tr>
<td>Complications of diabetes</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>Respiratory-related mortality</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Respiratory</td>
</tr>
<tr>
<td>Asthma symptoms</td>
<td>Asthma symptoms</td>
</tr>
<tr>
<td>Reduced lung function in children</td>
<td>Respiratory infections</td>
</tr>
<tr>
<td>Reduced lung function in susceptible adults (elderly, people with COPD or asthma)</td>
<td>Bronchitis in children</td>
</tr>
<tr>
<td>Respiratory infections in children</td>
<td>COPD symptoms</td>
</tr>
<tr>
<td>Cancer</td>
<td>Cancer</td>
</tr>
<tr>
<td>Lung cancer mortality</td>
<td>Lung cancer mortality</td>
</tr>
<tr>
<td>Neurological</td>
<td>Neurological</td>
</tr>
<tr>
<td>Neurological disorders in adults</td>
<td>disorders in adults</td>
</tr>
<tr>
<td>Impaired cognitive function</td>
<td>Impaired cognitive function</td>
</tr>
<tr>
<td>Development</td>
<td>Development</td>
</tr>
<tr>
<td>Lung development</td>
<td>Lung development</td>
</tr>
<tr>
<td>Neurological development in children</td>
<td>Neurological development in children</td>
</tr>
<tr>
<td>Reproduction</td>
<td>Reproduction</td>
</tr>
</tbody>
</table>

Adverse birth outcomes
Sperm quality and quantity

<table>
<thead>
<tr>
<th>Allergies</th>
<th>Allergies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exacerbation of allergies</td>
<td>Exacerbation of allergies</td>
</tr>
<tr>
<td>Allergic sensitization</td>
<td></td>
</tr>
</tbody>
</table>

Studies demonstrate air pollution is a Public health issue.

The proportion of deaths attributed to exposure of urban air pollution in Australia in 2003 are demonstrated in the table below, majority are from heart disease, followed by stroke, lung cancer and chronic lung disease. The type and concentration of pollutant, the duration of exposure, and susceptibility of the individual are factors that determine risk:\(^81\):

In summary the evidence demonstrates air pollution

1. Increases morbidity and mortality especially from fine particulate air pollution ie earlier deaths from global studies performed in cities exposed to ambient air pollution82,83,84,85

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82 Qian Di, MS; Lingzhen Dai, ScD; Yun Wang PhD; et al. Antonella Zanobetti, PhD; Christine Choirat, PhD; Joel D. Schwartz, PhD; Francesca Dominici, PhD. Association of Short-term Exposure to Air Pollution With Mortality in Older Adults. JAMA. 2017;318(24):2446-2456. doi:10.1001/jama.2017.17923. https://jamanetwork.com/journals/jama/article-abstract/2667069
2. Increase mortality associated with long-term effect of nitrogen dioxide NO2 as great as that of PM2.586

3. Increased risk of mortality, particularly for the elderly, even in areas of air pollution considered at "safe levels"87. Exposure to air pollution at levels well below current quality standards is linked to an increased risk of death. Increased risk of cardiovascular disease and deaths, such as cardiac arrests88,89 and deaths, heart failure onset and mortality associated with heart failure, deep vein thrombosis [blood clots], heart disease, and myocardial infarction (heart attacks)90,91,92,93.


87 Qian Di, MS; Lingzhen Dai, ScD; Yun Wang, PhD; et al Antonella Zanobetti, PhD; Christine Choirat, PhD; Joel D. Schwartz, PhD; Francesca Dominici, PhD Association of Short-term Exposure to Air Pollution With Mortality in Older Adults JAMA. 2017;318(24):2446-2456. doi:10.1001/jama.2017.17923 https://jamanetwork.com/journals/jama/article-abstract/2667069


4. Increase risk of heart failure due to a possible threshold of PM$_{2.5}=4$ µg/m$^3$ is far below the daily Australian national standard of 25 µg/m$^3$. 94
5. Higher risk of developing hypertension [high blood pressure] 95, 96.
6. Increase risk of stroke and mortality associated with stroke 97, 98.
7. Reduced survival from long-term exposure to traffic-related air pollution and cancer among survivors of myocardial infarction (heart attacks) 99.
8. Higher risk of out-of-hospital cardiac arrest associated with elevated ambient PM2.5 and CO 100.
9. Reduced lung function, increase respiratory symptoms and diseases, respiratory related deaths, chronic obstructive airways disease, cardiopulmonary disease, lung infections, and asthma 30 Pope, 101, 102, 103, 104, 105, 106.


100 Lahn Straney, Judith Finn, Martine Dennekamp, Alexandra Bremner, Andrew Tonkin, Ian Jacobs. Air pollution Evaluating the impact of air pollution on the incidence of out-of-hospital cardiac arrest in the Perth Metropolitan Region: 2000–2010 https://jehc.bmj.com/content/68/1/6.short

10. Increase risk of asthma in children (S2 Bowatte et al Allergy Cochrane Library. 2015. p. 245-256)

11. Systemic inflammation from exposure to ambient NO2 in Chronic Obstructive Pulmonary Disease (COPD) patients, especially in former smokers. Increase risk of inflammation and cardiovascular disease in adults.

12. Carcinogenic and lung cancer risk in non-smokers. Lung cancer risk due to deep penetration into the lungs of the fine particulates of 2.5 microns or less from diesel or petrol exhaust fumes. Air pollution shortens survival from lung cancer.

102 Benjamin D. Home, Elizabeth A. Joy, Michelle G. Hoffmann; Per H. Gesteland; John B. Cannon, Jacob S. Lefer, Deritza P. Blagev; E. Kent Korgenski; Natalie Torsayan, Grant I. Hansen, David Kartchner; et al. Short-term Elevation of Fine Particulate Matter Air Pollution and Acute Lower Respiratory Infection. Published Online: April 13, 2018. https://doi.org/10.1164/rcrm.201709-1883OC


cancer\textsuperscript{113}. PM2.5 particles or less are more toxic and carcinogenic as they penetrate deeper into the lungs and vascular system of the body.

13. Poor cognition and concentration\textsuperscript{114}. The researchers found that long term exposure to air pollution impacted human cognitive performance, concentration, verbal and math skills, particularly in the elderly, potentially resulting in significant health and economic costs.

14. Increased mortality due to residents working or residing in industrial areas of high air pollution resulting in increase cancers, lung cancer, mortality and neurological diseases\textsuperscript{115}.

15. Increase sick leave from work\textsuperscript{116}.

16. Greater healthcare utilisation by both children and adults for respiratory infections\textsuperscript{117}.

17. Increased risk of hospitalisation due to pneumonia in children\textsuperscript{118}.

18. Increased risk of diabetes and diabetes associated mortality\textsuperscript{119,120}.

19. Pregnancy risks - fine particulate chemicals from air pollution impact the blood vessel barrier of the vascular system of the placenta causing low birth weight, preterm birth, small for gestational age, and preterm delivery\textsuperscript{111}.

\begin{itemize}
  \item \textsuperscript{114} Xin Zhang, Xi Chen, Xiaobo Zhang \textit{The impact of exposure to air pollution on cognitive performance} PNAS published ahead of print August 27, 2018 \url{https://doi.org/10.1073/pnas.1809474115} \url{http://www.pnas.org/content/early/2018/08/21/1809474115}
  \item \textsuperscript{116} \textit{European Respiratory Society (ERS) International Congress 2018}: Abstract OA5182. Presented September 19, 2018. \url{http://www.rhine.nu/}
  \item \textsuperscript{117} Benjamin D. Horne, Elizabeth A. Joy, Michelle G. Hofmann; Per H. Gesteland; John B. Cannon; Jacob S. Leffer; Deniz C. Blagey; E. Kent Korgenski, Natalie Torosyan, Grant I. Hansen, David Karchner; et al. \textit{Short-term Elevation of Fine Particulate Matter Air Pollution and Acute Lower Respiratory Infection} \url{https://doi.org/10.1164/rccm.201709-1883OC} PubMed: 29652174 \url{https://www.atsjournals.org/doi/10.1164/rccm.201709-1883OC}
  \item \textsuperscript{119} Benjamin Bowe MPH, YanXie MPH, Tingting Li MD, ProfYanYan MD, ProfHongXian PhD, ZiyadAl-Aly MD. The 2016 global and national burden of diabetes mellitus attributable to PM\textsubscript{2.5} air pollution. \textit{Volume 2, Issue 7}, July 2018, Pages e301-e312 \textit{The Lancet Planetary Health} \url{https://www.sciencedirect.com/science/article/pii/S2542519618301402?via%3Dihub}
\end{itemize}
small for gestational age, and adverse birth outcomes\textsuperscript{121,122,123} and may increase risk of autism especially with exposure during the 3rd trimester\textsuperscript{124,125} and stillbirths\textsuperscript{126,127}.

20. Poor sperm quality and infertility; Air pollution is found to affect marker of female fertility in real-life study: Decline in ovarian reserve related to particulate matter and nitrogen dioxide in atmosphere-- ScienceDaily \textsuperscript{128,129}

21. Non-lung cancers: PM\textsubscript{2.5} was significantly positively associated with death from cancers of the kidney and bladder. NO\textsubscript{2} was positively associated with colorectal cancer mortality\textsuperscript{130}.

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\textsuperscript{122} Dirga Kumar Lamichhane, Jong-Han Leem, Ji-Young Lee, and Hwan-Cheol Kim. \textit{A meta-analysis of exposure to particulate matter and adverse birth outcomes}. Environ Health Toxicol. 2015; 30: e2015011. Published online 2015 Nov 3. doi: [10.5620/eh.t.e2015011] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4722965/


\textsuperscript{124} \textit{Autism Spectrum Disorder and Particulate Matter Air Pollution before, during, and after Pregnancy: A Nested Case-Control Analysis within the Nurses’ Health Study II Cohort} | Environmental Health Perspectives | Vol. 123, No. 3 https://ehp.niehs.nih.gov/doi/full/10.1289/ehp.1408133

\textsuperscript{125} \textit{Perinatal Air Pollutant Exposures and Autism Spectrum Disorder in the Children of Nurses’ Health Study II Participants} | Environmental Health Perspectives | Vol. 121, No. 8 https://ehp.niehs.nih.gov/doi/full/10.1289/ehp.1206187


https://www.sciencedaily.com/releases/2019/06/190625181939.htm
22. Psychosis: Increase risk of psychosis in teenagers\textsuperscript{131}. Air pollution exposure—particularly NO\textsubscript{2} and NO\textsubscript{x}—was associated with increased odds of adolescent psychotic experiences, which may partly explain the association between urban residency and adolescent psychotic experiences.

23. Liver impairment in adults. Long term exposure to PM2.5 causes increase levels of liver enzymes\textsuperscript{132}.

Population growth

Based on current trends, Australia’s population is projected to reach 30 million people by 2033, according to the latest figures released by the Australian Bureau of Statistics (ABS) and Australia’s estimated resident population at 30 June 2017 of 24.6 million people is projected to increase to between 37.4 and 49.2 million people by 2066\textsuperscript{133}.

This means as the population grows and with urban sprawl there will also be more motor vehicles on the road causing greater air pollution from petrol and diesel exhaust fumes. Electric cars will help reduce the level of emissions, but it may take a generation or two for the Australian population to fully embrace these alternatives to fossil fuel vehicles.


\textsuperscript{131} Joanne B. Newbury, PhD\textsuperscript{1}; Louise Arseneault, PhD\textsuperscript{1}; Sean Beevers, PhD\textsuperscript{2}; et al Nutthida Kitwiroon, PhD\textsuperscript{2}; Susanna Roberts, PhD\textsuperscript{2}; Carmine M. Pariante, PhD\textsuperscript{4}; Frank J. Kelly, PhD\textsuperscript{2}; Helen L. Fisher, PhD\textsuperscript{1}. Association of Air Pollution Exposure With Psychotic Experiences During Adolescence \textit{Original Investigation March 27, 2019}. \textit{https://jamanetwork.com/journals/jamapsychiatry/fullarticle/2729441}


\textsuperscript{133} Australian Bureau of Statistics Latest ISSUE Released at 11:30 AM (CANBERRA TIME) 22/11/2018 3222.0 - 
Economics, politics and sourcing earth elements such as lithium which are limited are potential barriers. The concern is the effects of vehicle fossil fuel emissions to the current generation of the population, and possibly the next generation, particularly the vulnerable such as children and the elderly, and those with pre-existing respiratory illnesses are most susceptible to harm.

With increasing population and urban growth this will result in more vehicles on freeways and in cities, contributing to further air pollution\textsuperscript{134}.

The National Clean Air Agreement state there will be an increase in Transport and Energy Demands and estimates currently “Over 70 per cent of all domestic travel occurs via roads. By 2030, road and rail freight are expected to grow by 80 and 90 per cent, respectively. National public transport is projected to grow by 30 per cent to 2030. The Australian transport sector is expected to rely heavily on oil over the next 20 years”\textsuperscript{135}.

Such challenges could erode the successes in air quality achieved to date and lead to poorer air quality outcomes for current and future generations. Given the evidence at hand and the challenges impacting on our air quality, Environment Ministers have recognised it is timely to map out a path for a clean air future for Australia.”

Working towards reducing urban air pollution in cities by implementing a number of strategies will ultimately help reduce health care costs. Alternative cheap (even free) and accessible public transport would better help facilitate and address the needs of our growing population.

One generation ago, families were lucky to own one vehicle per family. Now it is common for every child to have their own car resulting in four-to-five vehicles per family. It is not unusual for most cars to carry only one person (the driver) per vehicle. Apart from parents dropping off their children at school, most vehicles commuting on our roads now contain only the one driver.


\textsuperscript{135} Transport Security Outlook to 2025. Department of Infrastructure and Regional Development 2014 Australian Government, Canberra

Freeways are now often congested, particularly during peak hour travel despite widening of these freeways! The technical name for this is "Induced Demand". This is what would be expected with any proposed Freeways, especially with the population growth and urban development into outer suburbs of cities. Judging from our past experience with Freeways, they are not long term sustainable solutions and further widening of these freeways are often required with the growth in the number of vehicles on the road. None of these freeways have pedestrian and cycling trails alongside them, as they are not conducive to such activities, particularly the air and noise pollution impacting on humans. Monitoring for air pollution and air quality at or near these sites would yield alarming results. Air quality in cities will continue to deteriorate over time if we do not address vehicle related air pollution now and limit vehicles into cities. Cities will suffer an increase in air pollution if we continue to widen roads for more vehicles to enter them or build more freeways next to residential areas and/or within urban environments that increase the number of vehicles in close proximity to the growing population placing them at risk and harm.
Conclusion

The National Environment Protection (Ambient Air Quality) in Australia

Precautionary Principle

The National Environment Protection (Ambient Air Quality) in Australia states “In the risk management phase, regulators consider the results of the risk assessment stages ..., apply the precautionary principle and take into account social and economic factors”.

Planning new freeways, and widening freeways to accommodate the growing number of vehicles into cities should raise alarm bells based on the growing scientific evidence. Precautionary principal is not being observed particularly for children with chronic exposure to increasing levels of air pollution.

Every effort should be considered and thoroughly explored in determining how public exposure to vehicle emissions is associated with potential harm and every effort should be made to reduce harm by first considering alternative options to freeways first. Successful utilisation of the precautionary principle would involve applying conservatism (towards over-estimating health impacts) at every point and then addressing every possible way in which these impacts may be reduced.

National Environmental Protection Measures legislation discusses ‘equivalent protection’ and the goal that “all Australian’s enjoy the benefit of equivalent protection from air”. The ultrafine particles and air toxics which largely comprise vehicle emissions have high variability across the air-shed. Busy urban roads inevitably create air pollution ‘hot spots’. Most people cannot easily change the location of their homes, workplaces, schools or childcare centres, so when large road projects such as freeways being built in or next to urban environments have the ability to significantly alter a community’s air pollution exposure every possible mitigation strategy should be comprehensively considered in terms of the net benefit to the community and associated health savings and then weighed up against the practicalities and costs of implementing a freeway or busy roadway.

The health of the population and community must come as a priority to decision making.
Preserving outdoor spaces and natural environments

There are worldwide efforts and strategies to improve public transport including offering free public transport in busy cities, develop urban forests and retain green spaces within an urban environment to help with air purification and climate regulation, recognising the health benefits these strategies have on our community. Studies and research demonstrate that planting more trees, encouraging leafy suburbs, green spaces, parklands and urban forests improve the general health of communities.

Strategies include:

1. Reducing air pollutants and improving quality of air by avoiding more construction of freeways and aim to retain as much green space available and for cities to develop into urban forests to reduce air pollution in our environment. For example, A City of Melbourne paper cites a New York study that found an urban forest removed 1,821 metric tonnes of air pollution at an estimated value to society of $9.3 million per year.

2. Planting more trees to improve air quality, consequently improving the health of the population and reducing the incidence of respiratory diseases, asthma and allergies. Certain trees also trap and filter pollens on windy days.

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138 Urban Forest Strategy; Making a great city greener 2012-2032 City of Melbourne melbourne.vic.gov.au/urbanforest


3. Develop urban forests to help mitigate air pollution and improve air quality\textsuperscript{143}.
4. Plant and retain more trees to provide shade and cool our cities, helping us to keep us cooler over summer, reducing sun exposure and hence protecting us from skin damage and cancer effects.
5. Plant and retain trees to remove carbon dioxide from the air that traps heat in the atmosphere, thereby reducing climate change, and reducing the risk of heat impacts on vulnerable groups such as elderly and children.
6. Build and retain parkland to improve the psychological well being of the individual. Parklands and green spaces help people to exercise more and relax in nature\textsuperscript{144}. A study performed in the Netherlands found communities who lived within a 1 km radius of a nature reserve suffered fewer mental illnesses\textsuperscript{53}. The psychological benefits of green spaces and biodiversity are well known\textsuperscript{145}.
7. Fostering more green spaces to improve air quality for exercise in nature; thereby breathing fresh air rather than polluted air. Encouraging outdoor activity, offers greater opportunities for exercise, nature and bird watching for relaxation and community cohesion.

**Lifestyle related chronic diseases**

In hospitals we are witnessing more and more lifestyle related chronic diseases such as obesity, diabetes, cardiovascular diseases, pulmonary diseases, and cancers. Outdoor exercise in a clean environment offers a long term solution to address hospitalisation related illnesses and chronic diseases, with significant cost savings to our community.


\textsuperscript{144} Kwan Hui Lim, Dave Kendal, Kate Lee Tweet all about it – people in parks feel more positive May 8, 2018 https://theconversation.com/tweet-all-about-it-people-in-parks-feel-more-positive-95290

Peaceful parks encourage exercise and relaxation. **Traffic noise and environmental noise pollution are also major issues of freeways and negatively impacts cardiovascular and mental health**. Furthermore research demonstrates more travel time in vehicles compared with walk time increases the risk of obesity, now a national health concern.

Parks and nature reserves are precious in urban environments and should be protected and cared for with the utmost sensitivity—we owe it to our children to leave our planet and environment clean and healthy.

**Barrier walls and vegetation barriers lining heavy traffic roads**

Thick dense vegetation barriers along traffic roads and freeways are considerably helpful in reducing pollutant load, and mitigating urban air pollution. However residents are still exposed to pollution depending on direction of wind and changing weather patterns as can be viewed in the following illustration:

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Solutions and alternatives to Freeways:

- Protect existing open spaces and nature reserves, and develop more areas of urban forests.
- Plant more trees for oxygen and reduction of carbon dioxide and pollutants.
- Create an extensive affordable and even consider a free public transport plan as is occurring in many cities worldwide\(^{150,151,152}\) to interconnect outer suburbs, and

150 https://www.weforum.org/agenda/2018/06/estonia-is-making-public-transport-free/
consider an extensive underground train system like Sydney where appropriate. This helps mobilise the community, encourages more walking and cycling, and will help combat preventable rising chronic diseases.

- Divert tax public funding from freeways to public transport and create cycling and walking paths. This will provide more work for the community through an extensive public transport system.
- Less motor vehicles on the road means less pollution i.e. air, noise and visual pollution. This will result in a healthier community and help reduce healthcare costs and hospitalisations.
- Induced demand will create more vehicles on freeways and with time, as the number of vehicles increase on freeways, there will continue to be more stop-start ques [see image of Tullamarine Freeway above] that will create more vehicle emissions and cause more air pollution.
- Less car travel and freeways - reduces risk of crashes, obesity, sedentary travel, air pollution, loss of amenity.
- Create micro-cities to ensure that work/schools/retail/home are geographically close to reduce travel time on the road, and as a better solution to addressing traffic congestion than building bigger roads.

The strongest argument against freeways is the induced demand- i.e. once there is a freeway people build their lives around the freeway until congestion of traffic occurs.

Congestion is a cultural phenomenon. People will keep queuing up on the road until travel times pass their tolerance threshold, then they will divert to other times of day or other destinations. If you shorten the trip time, more people will queue up until the threshold is reached again.

The only solution is to build public transport that is faster than driving. Or cycle ways that are faster than driving.
Our state Government, DELWP and leading universities and researchers have already highlighted concerns with pollution and created policies and strategies to help create a beautiful healthy sustainable city\footnote{Cleaner safe air needs you! Citizen scientists are key to a big data project that will identify where the bad air lurks in our cities, By Andrew Trounson, University of Melbourne \url{https://pursuit.unimelb.edu.au/articles/cleaner-safe-air-needs-you}}\footnote{Urban Forest Strategy 2014 URBAN FOREST STRATEGY Making a great city greener 2012-2032 \url{https://www.vicparktrees.com/urban-forest-strategy} & \url{https://www.melbourne.vic.gov.au/SiteCollectionDocuments/urban-forest-strategy.pdf}}\footnote{Professor Richard Sinnott, Director, eResearch; Department of Computing and Information Systems, Melbourne School of Engineering, University of Melbourne; Clare Walter, Honorary respiratory researcher, Royal Melbourne Hospital; Oncology Pharmacist. Cleaner safe air needs you! | Pursuit by The University of Melbourne \url{https://pursuit.unimelb.edu.au/articles/cleaner-safe-air-needs-you}}.

Please, let’s adopt the findings and recommendations by Authorities based on a number of position papers that aim to reduce the pollutant load on humans and the environment, and work towards a green sustainable city to protect our environment to ensure it remains clean for future generations to enjoy and live a healthy life.

A/Professor (Dr) Vicki Kotsirilos AM, MBBS, FACNEM, FASLM, Awarded Honorary Fellowship of the Royal Australian College of General Practitioners

Appendix 1 further references demonstrating vehicle related air pollution impacting health
Appendix B  Key community infrastructure relative to North East Link

Note: the reported distances have been established in good faith using:
1. Maps provided in this NEL EES I Social assessment,
2. NEL MAP books,
3. NearMAPs and
4. Melways online.

This is in accordance with the advice and instructions received from Sallyanne Everett, Partner, Clayton Utz, acting for NELP via email on 3rd July 2019

<table>
<thead>
<tr>
<th>Suburb</th>
<th>Map code</th>
<th>Facility name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bundoora</td>
<td>1</td>
<td>Hughes Circuit Reserve <em>within 100m of construction zone and finished project</em></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Maroondah Aqueduct</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>Garvey Oval Parade College  (1850 secondary school students)- Omitted from GHD</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Abuts the construction zone and within 200m finished project</em></td>
</tr>
<tr>
<td>Watsonia North</td>
<td>4</td>
<td>Gillingham Reserve <em>Playground taken up by construction works, then shared bike path on completion</em></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>Trist Street Reserve <em>taken up construction zone, becomes shared bike pathway</em></td>
</tr>
<tr>
<td>Greensborough</td>
<td>8</td>
<td>Abacus Child Care Centre – <em>within 100m construction zone and completed project</em></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>Greensborough Road Surgery – <em>abutting construction surface works, within 100m of finished project</em></td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>St Mary's Church – <em>Less than 100m from construction zone and finished project</em></td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>St Mary’s School – abutting <em>construction zone and finished project</em></td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>Greensborough College - <em>within 120 m of construction zone and 200m finished project</em></td>
</tr>
<tr>
<td></td>
<td>17</td>
<td>Kalparrin Gardens <em>within 310 m of construction zone and finished project</em></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>Fell Reserve <em>within 310 m of construction zone and finished project</em></td>
</tr>
<tr>
<td></td>
<td>22</td>
<td>St Mary's Reserve <em>taken up by the construction zone then shared path</em></td>
</tr>
<tr>
<td></td>
<td>23</td>
<td>Sarah's Reserve (Banfield Terrace Reserve) <em>taken up by the construction zone then shared path</em></td>
</tr>
<tr>
<td>Watsonia</td>
<td>26</td>
<td>Watsonia Occasional Child Care Centre - <em>abuts construction zone, 150m from finished project</em></td>
</tr>
<tr>
<td></td>
<td>27</td>
<td>MS Respite Services – <em>50m from construction zone and finished project</em></td>
</tr>
<tr>
<td></td>
<td>31</td>
<td>Watsonia Library - <em>abuts construction zone &amp; finished project</em></td>
</tr>
<tr>
<td></td>
<td>Location</td>
<td>Notes</td>
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<tr>
<td>32</td>
<td>Holy Spirit Anglican Church</td>
<td><strong>abuts construction zone &amp; finished project</strong></td>
</tr>
<tr>
<td>33</td>
<td>Grace Baptist Church</td>
<td><strong>abuts construction zone &amp; finished project</strong></td>
</tr>
<tr>
<td>36</td>
<td>Watsonia Primary School</td>
<td><strong>abuts construction zone &amp; finished project</strong></td>
</tr>
<tr>
<td>37</td>
<td>AK Lines Reserve</td>
<td><strong>abuts construction zone &amp; finished project, partly lost</strong></td>
</tr>
<tr>
<td>39</td>
<td>West Mayling Reserve</td>
<td><strong>abuts construction zone, 30m from finished trench</strong></td>
</tr>
<tr>
<td>40</td>
<td>Gabonia Avenue Reserve</td>
<td><strong>inside construction zone</strong></td>
</tr>
<tr>
<td>41</td>
<td>Watsonia Station Carpark Reserve</td>
<td><strong>abuts construction zone &amp; finished project</strong></td>
</tr>
<tr>
<td>42</td>
<td>Watsonia Road Reserve</td>
<td><strong>Taken up as construction Zone becomes surface road</strong></td>
</tr>
<tr>
<td>Loyola College (1350 secondary school students)</td>
<td>Omitted from GHD</td>
<td>Report for North East Link Project – North East Link Environment Effects Statement, 1 Social assessment within 200 m of construction zone and finished project</td>
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<td>43</td>
<td>Greensborough Road Early Learning and Kinder</td>
<td><strong>abuts construction zone &amp; finished project</strong></td>
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<td>44</td>
<td>Macleod Recreation and Fitness &amp; CC</td>
<td><strong>abuts construction zone &amp; finished project- rail</strong></td>
</tr>
<tr>
<td>45</td>
<td>Macleod Preschool</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>46</td>
<td>Baptcare Strathalan Macleod</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>47</td>
<td>Regis Macleod</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
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<tr>
<td>49</td>
<td>IDV</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>50</td>
<td>YMCA Recreation and Fitness Centre</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>51</td>
<td>Nets Stadium Banyule</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>52</td>
<td>Macleod Organic Community Garden</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>53</td>
<td>Winsor Reserve</td>
<td><strong>taken up by construction Zone then abuts project trench</strong></td>
</tr>
<tr>
<td>54</td>
<td>Macleod College Oval</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>55</td>
<td>Harry Pottage Reserve</td>
<td><strong>within 50m of construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>56</td>
<td>Macleod Park</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>57</td>
<td>Macleod Tennis Club</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>58</td>
<td>Junior Football Club and Macleod Cricket</td>
<td><strong>abuts construction zone &amp; finished project rail</strong></td>
</tr>
<tr>
<td>59</td>
<td>Simpson Barracks</td>
<td>13 hectares <strong>within project construction zone and completed project, site of proposed ventilation structure</strong></td>
</tr>
<tr>
<td>Page</td>
<td>Location</td>
<td>Details</td>
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</tr>
<tr>
<td>60</td>
<td>Rosanna</td>
<td>Coleen Reserve</td>
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<tr>
<td>61</td>
<td>Rosanna</td>
<td>Borlase Reserve – <em>Destroyed by construction, site of Lower Plenty Road interchange ramps</em></td>
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<td>65</td>
<td>Rosanna</td>
<td>Japara Rosanna Views Nursing Home - <em>150 m away from construction zone and finished project</em></td>
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<td>66</td>
<td>Rosanna</td>
<td>St Martin of Tours Catholic Church - <em>abuts construction zone &amp; finished project</em></td>
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<td>67</td>
<td>Rosanna</td>
<td>St Martin of Tours Catholic Primary School -- <em>abuts construction zone &amp; finished project</em></td>
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<td>68</td>
<td>Rosanna</td>
<td>Banyule Primary School – <em>250m away from construction zone and finished project</em></td>
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<td>69</td>
<td>Rosanna</td>
<td>Maleela Grove, Rosanna - <em>in potential tunnel area</em></td>
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<td>70</td>
<td>Rosanna</td>
<td>Mercedes Court Reserve - <em>in potential tunnel area</em></td>
</tr>
<tr>
<td>71</td>
<td>Rosanna</td>
<td>River Gum Walk -- <em>abuts construction zone &amp; finished project, beneath tunnel</em></td>
</tr>
<tr>
<td>72</td>
<td>Rosanna</td>
<td>Creekbend Reserve <em>in potential tunnel area</em></td>
</tr>
<tr>
<td>74</td>
<td>Viewbank</td>
<td>Viewbank College - <em>in potential tunnel area</em></td>
</tr>
<tr>
<td>75</td>
<td>Viewbank</td>
<td>Viewbank family medical group <em>in potential tunnel area</em></td>
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<td>Viewbank</td>
<td>Viewbank podiatry <em>in potential tunnel area</em></td>
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<td>Viewbank</td>
<td>Simla Close Reserve <em>in potential tunnel area</em></td>
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<td>Heidelberg</td>
<td>Banyule Flats <em>likely above tunnel</em></td>
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<td>Heidelberg</td>
<td>River Gum Walk <em>in potential tunnel area</em></td>
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<td>80</td>
<td>Heidelberg</td>
<td>Warringal Parklands <em>in potential tunnel area</em></td>
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<td>81</td>
<td>Heidelberg</td>
<td>Banyule Tennis Club <em>in potential tunnel area</em></td>
</tr>
<tr>
<td>82</td>
<td>Heidelberg</td>
<td>Heidelberg Park and Heidelberg cricket ground <em>in potential tunnel area</em></td>
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<tr>
<td>83</td>
<td>Heidelberg</td>
<td>Banyule Theatre Complex <em>in potential tunnel area</em></td>
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<tr>
<td>84</td>
<td>Heidelberg</td>
<td>Heidelberg Police Station <em>300m from construction zone</em></td>
</tr>
<tr>
<td>87</td>
<td>Eaglemont</td>
<td>Yarra Flats Park – <em>within construction zone and abuts finished project surface works</em></td>
</tr>
</tbody>
</table>
Manningham
Appendix B. 1 Key community infrastructure relative to North East Link for Manningham & Boroondara

Note: the reported distances have been established in good faith using:

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<tbody>
<tr>
<td>Bulleen</td>
<td>1</td>
<td>Creative Play Early Learning Centre (abuts) construction zone &amp; finished project, looks like some land loss</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Kalker Montessori Centre (Less than 70m from) construction zone &amp; finished project</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>Veneto Club (abuts) construction zone &amp; finished project, part land loss</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>Heide Museum of Modern Art (abuts) construction zone &amp; finished project, partly lost, likely to be above tunnel alignment</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>Bulleen Art and Garden - (within) construction zone &amp; finished project acquired and likely loss</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>Marcellin College (abuts) construction zone &amp; finished project, partial land loss</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>Trinity Grammar School Sporting Complex (abuts) construction zone &amp; finished project, partly lost</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>Bulleen Swim Centre (within) construction zone &amp; finished project, acquired and likely loss</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>Bolin Bolin Billabong - (abuts) construction zone &amp; finished project</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>Carey Grammar Sports Complex (abuts) construction zone &amp; finished project, partly lost</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>Yarra Valley parklands – Banksia Park (abuts) construction zone &amp; finished project, partly lost likely to be above tunnel alignment</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>Sandra Street Reserve (abuts) construction zone &amp; 75m from finished project</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>Bulleen Park Oval No 1 (within) construction zone &amp; finished project, site of south tunnel exit lost</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>Koonung Reserve (within) construction zone &amp; some loss from finished project busway and shared paths</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>Koonung Creek Reserve (within) construction zone &amp; finished project, substantial permanent loss</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>Willow Bend Reserve (50m) construction zone &amp; 75m finished project</td>
</tr>
<tr>
<td>No.</td>
<td>Location</td>
<td>Distance &amp; Impact</td>
</tr>
<tr>
<td>-----</td>
<td>------------------------------------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>17</td>
<td>Pipe Line Reserve</td>
<td>Unsure of location</td>
</tr>
<tr>
<td>18</td>
<td>Yarra Valley Country Club</td>
<td>Likely to be above tunnel alignment</td>
</tr>
<tr>
<td>19</td>
<td>Bulleen Golf Driving Range</td>
<td>Greater than 300m away</td>
</tr>
<tr>
<td>20</td>
<td>Wonderland Childcare and Kinder</td>
<td>Within construction zone &amp; finished project, permanent loss of land and holes</td>
</tr>
<tr>
<td>21</td>
<td>Applewood Retirement Village</td>
<td>Abuts construction zone, 120 m from finished project</td>
</tr>
<tr>
<td>22</td>
<td>Tende Beck Scout Hall</td>
<td>Abuts construction zone, 50 m from finished project</td>
</tr>
<tr>
<td>23</td>
<td>North Eastern Jewish Centre-Yeshurun Congregation</td>
<td>Abuts construction zone, 50 m from finished project</td>
</tr>
<tr>
<td>24</td>
<td>Birrallee Primary School</td>
<td>50m construction zone &amp; 100m finished project</td>
</tr>
<tr>
<td>25</td>
<td>Wilsons Road Reserve</td>
<td>40m construction zone &amp; 100m finished project</td>
</tr>
<tr>
<td>26</td>
<td>Katrina Street Reserve</td>
<td>Site of proposed construction compound, abuts finished project</td>
</tr>
<tr>
<td>27</td>
<td>Davis Street Reserve</td>
<td>Greater than 2km from project works</td>
</tr>
<tr>
<td>28</td>
<td>Tram Road Reserve</td>
<td>Section within construction zone, abuts finished project, some land loss</td>
</tr>
<tr>
<td>29</td>
<td>Koonung Creek Linear Park</td>
<td>Section of Koonung Creek Linear Park stretching East to Wetherby Rd entirely taken up by construction with permanent land loss. Abuts finished project</td>
</tr>
<tr>
<td>30</td>
<td>Stanton Street Reserve</td>
<td>Abuts construction zone, &amp; finished project</td>
</tr>
<tr>
<td>31</td>
<td>Doncaster Park and Ride</td>
<td>Within construction zone, planned multi story carpark part of finished project,</td>
</tr>
<tr>
<td>32</td>
<td>Tino Ceberano Martial Arts Academy</td>
<td></td>
</tr>
<tr>
<td>33</td>
<td>Greythorn Bowling Club</td>
<td>Abuts construction zone &amp; finished project</td>
</tr>
<tr>
<td>34</td>
<td>Manningham Park Reserve</td>
<td>Partly within construction zone and abuts finished project.</td>
</tr>
<tr>
<td>35</td>
<td>Koonung Creek Reserve</td>
<td>Partly within construction zone &amp; finished project</td>
</tr>
<tr>
<td>36</td>
<td>Boronia Grove Reserve</td>
<td>Partly within construction zone &amp; finished project</td>
</tr>
<tr>
<td>37</td>
<td>Beverley Hills Primary School</td>
<td>300m construction zone &amp; 350m from finished project</td>
</tr>
<tr>
<td>38</td>
<td>Japara Sydney Williams Apartments</td>
<td>25m construction zone &amp; 75m from finished project</td>
</tr>
<tr>
<td>39</td>
<td>Heatherwood School</td>
<td>50m construction zone &amp; 200m from finished project</td>
</tr>
<tr>
<td>40</td>
<td>State Vision Resource Centre</td>
<td>-150m construction zone &amp; 250m from finished project</td>
</tr>
<tr>
<td>41</td>
<td>Koonung Creek Linear Park</td>
<td>Partly within construction zone and abuts finished project.</td>
</tr>
<tr>
<td>42</td>
<td>Donvale Primary School</td>
<td>200m construction zone &amp; 250m from finished project</td>
</tr>
<tr>
<td>43</td>
<td>Aranga Reserve</td>
<td>25m construction zone &amp; 100m from finished project</td>
</tr>
<tr>
<td>44</td>
<td>Oxford Street Reserve</td>
<td>25m construction zone &amp; 200m from finished project, abuts East Link</td>
</tr>
</tbody>
</table>
## Key community infrastructure relative to North East Link in the Boroondara from Table 6.4

<table>
<thead>
<tr>
<th>Suburb</th>
<th>Map code</th>
<th>Facility name</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Balwyn North</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>Belle Vue Primary School - <em>abuts major construction zone &amp; finished project interchange</em></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Boroondara Tennis Centre <em>taken up by construction zone becomes Bulleen Park and Ride</em></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>Musca Street Reserve <em>taken up by construction zone abuts finished project</em></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>Columba Street Reserve <em>inside construction zone abuts finished project</em></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>Leonis Avenue Reserve <em>inside construction zone abuts finished project</em></td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>Koonung Creek Reserve <em>taken up by construction with permanent land loss. Abuts finished project</em></td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>Yarra Flats Reserve -- <em>abuts construction zone &amp; finished project, potential construction compound</em></td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>Freeway Public Golf Course <em>abuts construction zone &amp; finished project, loss of land/ course</em></td>
</tr>
<tr>
<td><strong>Kew East</strong></td>
<td>11</td>
<td>Kew Golf Club <em>abuts construction zone &amp; finished project,</em></td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>Hays Paddock Reserve <em>abuts construction zone &amp; finished project, appears to result in loss of land</em></td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>Hyde Park - <em>10m construction zone &amp; 50m from finished project</em></td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>Burke Rd Billabong Reserve <em>abuts construction zone &amp; finished project</em></td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>Green Acres Golf Club <em>100m construction zone &amp; 150m from finished project</em></td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>Willsmere-Chandler Park <em>abuts construction zone &amp; finished project, appears to result in loss of land during construction</em></td>
</tr>
<tr>
<td><strong>Kew</strong></td>
<td>17</td>
<td>Guide Dogs Victoria - <em>abuts construction zone &amp; finished project, appears to result in loss of land during construction</em></td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>Royal Talbot Rehabilitation Centre <em>abuts construction zone &amp; finished project</em></td>
</tr>
<tr>
<td></td>
<td>19</td>
<td>Yarra Bend Park <em>abuts construction zone &amp; finished project, appears to result in loss of land during construction</em></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>Kate Campbell Reserve <em>abuts construction zone &amp; finished project, appears to result in loss of land during construction</em></td>
</tr>
<tr>
<td></td>
<td>21</td>
<td>Jack O’Toole Reserve <em>abuts construction zone &amp; finished project, results in loss of land during construction</em></td>
</tr>
<tr>
<td></td>
<td>22</td>
<td>North Kew Tennis Club <em>abuts construction zone &amp; finished project, results in loss of land during construction</em></td>
</tr>
<tr>
<td></td>
<td>23</td>
<td>1st Kew Scout Group <em>abuts construction zone &amp; finished project, results in loss of land during construction</em></td>
</tr>
</tbody>
</table>