Health Impacts of Air Traffic Pollution

Why freeways should not be built in or adjacent to an urban environment

Sustainable cities for a cleaner environment and clean air

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"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.

Melbourne City, a Green city

- Melbourne was voted the most liveable city for its Green spaces ¹

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- Clare Walter, PhD Honorary respiratory researcher, Royal Melbourne Hospital
- Associate Professor Sanjay Raghav, Neurologist

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 pollution$^{2,3,4,5,6}$.

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$^{7}$". 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.

2 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

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

4 http://www.nespurban.edu.au/ [accessed 11th November 2018]

5 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. 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”. 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.

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:

- 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$_{2.5}$)
- 27 per cent of all emissions of particles smaller than 10 microns (PM$_{10}$)
- 6 per cent of all sulfur dioxide (SO$_2$) emissions
- Nitrogen oxides (NOx) and volatile organic compounds (VOCs) can combine to form ozone (summer smog).

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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.

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

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

“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.

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

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Sizes of particulate matter compared to human hair and beach sand. Illustration: Eda Lu, based on US EPA “Particulate Matter (PM) Pollution” from the book “Particles in the Air,” Source accessed 22.11.2018 https://now.tufts.edu/articles/toxic-air-we-breathe

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\(^{18}\). 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:\(^{20}\) "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\(^{21}\). 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\(^{22}\). Independent of gas cooking, "*indoor NO\(_2\) 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\(_2\), and hence roadway emissions can potentially contribute a significant proportion to the indoor NO\(_2\) concentration.... Findings elsewhere suggest that a similar outdoor enhancement of traffic related NO\(_2\) (5 ppb) increases risk of lung cancer and childhood asthma (Brauer et al., 2000; Nyberg et al., 2000).*"


State Environment Protection Policy (Ambient Air Quality) Victorian Government

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”\(^23\).

“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 PM10 and PM2.5), 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:
- **PM10 concentrations** of 50 μg/m\(^3\) (24 hour average) and 20 μg/m\(^3\) (one year average)
- **PM2.5 objective** of 25 μg/m\(^3\) (24-hour average) and 8 μg/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 μg/m(^3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO</td>
<td>9 ppm</td>
<td>8-hours</td>
<td>10,400 μg/m(^3)</td>
</tr>
<tr>
<td>NO(_2)</td>
<td>0.12 ppm</td>
<td>1-hour</td>
<td>228 μg/m(^3)</td>
</tr>
<tr>
<td></td>
<td>0.03 ppm</td>
<td>1-year</td>
<td>57 μg/m(^3)</td>
</tr>
<tr>
<td>PM10</td>
<td>50 μg/m(^3)</td>
<td>24-hours</td>
<td>50 μg/m(^3)</td>
</tr>
<tr>
<td></td>
<td>20μg/m(^3)</td>
<td>1-year</td>
<td>20 μg/m(^3)</td>
</tr>
<tr>
<td>PM2.5</td>
<td>25 μg/m(^3)</td>
<td>24-hours</td>
<td>25 μg/m(^3)</td>
</tr>
<tr>
<td></td>
<td>8μg/m(^3)</td>
<td>1-year</td>
<td>8 μg/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</td>
<td>1 day a year</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 year</td>
<td>0.03 ppm</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Photochemical oxidants (as ozone) (maximum concentration)</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 (maximum concentration)</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></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$_{10}$ (maximum concentration)</td>
<td>1 day</td>
<td>50 μg/m³</td>
<td>none</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 year</td>
<td>20 μg/m³</td>
<td>none</td>
</tr>
<tr>
<td>6A</td>
<td>Particles as PM$_{2.5}$ (maximum concentration)</td>
<td>1 day</td>
<td>25 μg/m³</td>
<td>none</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 year</td>
<td>8 μg/m³</td>
<td>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 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 PM$_{2.5}$ (maximum concentration)</td>
<td>1 day</td>
<td>20 µg/m$^3$ by 2025</td>
</tr>
<tr>
<td></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. The WHO has also reviewed the evidence of air pollution and its adverse effects on human health.

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25 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\(^{30}\) 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\(^{31,32}\). **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**

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\(^{30}\) Australian Burden of Disease Study: impact and causes of illness and death in Australia 2011


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 inner city Melbourne, this will result in more vehicles and, consequently, greater emission of pollutants from vehicle emissions into the air.

**Adverse effects from Particulate Matters**

![Diagram of particulate matters](image)

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". The Ultrafine Particles (UFP) may penetrate and enter the body in any route including oral, skin or by inhalation.

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 highlights and reviews the science that demonstrates there is no safe lower limit of 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

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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/m³ (US standard = 12 ug/m³ Aus = 8 ug/m³).36

"Results: Since 2001, average PM$_{2.5}$ levels, for all six cities, were < 18 µg/m³. Each increase in PM$_{2.5}$ (10 µg/m³) 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/m³. 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.\(^3\) 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. \(^3\)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

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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\(^{45}\).

\(^{40}\) 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:\(^\text{39}\):

- reduced lung function in children,
- caused exacerbation and onset of asthma,
- contributed to a range of respiratory symptoms,
- impaired lung function,

---

**Figure 2**

Forest plots of the change in all-cause and cardiovascular risk associated with increases in source-specific PM\textsubscript{2.5}. The data in these forest plots are from Table 2. The reference numbers applicable to the different data are shown.
- 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 (physiological/toxicity effects includes animal studies).**

<table>
<thead>
<tr>
<th>Emission Source</th>
<th>Health Risk and Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Traffic</strong></td>
<td>exacerbation and onset of childhood asthma, respiratory symptoms, impaired lung function, all-cause mortality, cardiovascular morbidity [46]</td>
</tr>
<tr>
<td></td>
<td>myocardial infarction [49]</td>
</tr>
<tr>
<td></td>
<td>reduced lung function in children [51]</td>
</tr>
<tr>
<td></td>
<td>increased blood pressure [52]</td>
</tr>
<tr>
<td></td>
<td>allergic sensitization [53]</td>
</tr>
<tr>
<td></td>
<td>premature birth [38]</td>
</tr>
<tr>
<td><strong>Total traffic-related air pollution (TRAP)</strong></td>
<td>all-cause, respiratory and cardiovascular mortality, cardiovascular, stroke and heart failure morbidity [54,55,56,57,58,70,71,72]</td>
</tr>
<tr>
<td></td>
<td>cardiovascular toxicity and various cardiovascular effects [50,69]</td>
</tr>
<tr>
<td></td>
<td>cytotoxicity, pulmonary inflammation [62,63]</td>
</tr>
<tr>
<td><strong>Specifically traffic PM</strong></td>
<td>all-cause, cardiovascular, respiratory, ischaemic heart disease, pneumonia, lung cancer mortality [19,34,57,58,69,70,71]</td>
</tr>
<tr>
<td></td>
<td>respiratory morbidity [48,49,65,66,67,68,72]</td>
</tr>
<tr>
<td></td>
<td>cardiovascular morbidity [48,49,68]</td>
</tr>
<tr>
<td><strong>Coal-fired power stations</strong></td>
<td>respiratory mortality [53]</td>
</tr>
<tr>
<td></td>
<td>lung and oesophageal cancer mortality [84,85]</td>
</tr>
<tr>
<td></td>
<td>allergic inflammation, asthma symptoms, lung cancer [79,81,82,83]</td>
</tr>
<tr>
<td></td>
<td>cardiovascular morbidity [72,89]</td>
</tr>
<tr>
<td><strong>Diesel exhaust</strong></td>
<td>cardiovascular changes indicative of increased coronary event risk.</td>
</tr>
<tr>
<td></td>
<td>changes in lung function, nose and throat irritation [18,49,90]</td>
</tr>
<tr>
<td></td>
<td>atopy and susceptibility to infection [98,99,100]</td>
</tr>
<tr>
<td></td>
<td>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 ($\text{PM}_{2.5}$).

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 $\text{PM}_{2.5}$ 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 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 (13 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.


Prenatal exposure

Another systematic review of epidemiological studies in children whose mothers were exposed to air pollution (prenatal exposure), found these children were at higher risk of developing asthma. The researchers concluded:

"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."

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 NO₂, NOₓ and PM2.5 from air pollution traffic was associated with reduced lung function.

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. According to the Organisation for Economic

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57 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/dc4b6e305a8b2345c5ecdce05a842410b1a.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\textsuperscript{58}.

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\textsuperscript{59}. 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 (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 [an inflammatory marker found on pathology blood testing] caused from chronic ambient exposure to air pollution thought to be due to the UFPs (ultrafine particles) smaller than 0.1 \(\mu\)m that penetrate


deeper into the lung and circulatory system\textsuperscript{60}. 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{61}. 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(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

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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/m$^3$ 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: 64 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 NOx 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." 

65 Lisa Bauleo, Simone Bucci, Chiara Antonucci, Roberto Sozzi, Marina Davoli, Francesco Forastiere, Carla Ancona. 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/m$^3$ per increase of particulate matter (PM)$_{2.5}$, PM$_{10}$, and NO$_2$ - 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$^{66}$.

“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$_{2.5}$, NO$_2$ and O$_3$ → ↑ risk of Outer Hospital Cardiac Arrest risk (OHCA)$^{67}$. 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; NO$_2$ 1.015, 95%CI: 1.001-1.030; O$_3$  1.016, 95%CI: 1.008-1.024).

Population attributable fractions for PM$_{10}$, PM$_{2.5}$, and O$_3$ 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.

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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\(^{68}\). Oxidative stress has emerged 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\(^{69}\). 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/m\(^3\)) exhaust during 1 hour of moderate exercise had a direct effect on myocardial function [ECG changes], vascular, and fibrinolytic function\(^{70}\). 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

Interesting a 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. 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. 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. 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 sup-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. 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.

*See Forest Plot 1. Relative risks for lung cancer associated with a 10µg/m³ increase of PM2.5 (A) and PM10 (B).*

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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.\textsuperscript{74}

"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 \(\mu g/m^3\)). For PM\textsubscript{2.5} the HR was 1.18 (0.96–1.46) per 5 \(\mu g/m^3\). 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)."

**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]).\textsuperscript{75}

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.

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.

\textsuperscript{74} 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

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.\textsuperscript{76}

**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: \textit{“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”}\textsuperscript{77}.

**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 such as Melbourne are described throughout this document even when considered "safe" thresholds demonstrating detrimental impacts on health\textsuperscript{78}. 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.


\textsuperscript{77} Car air filters - National Asthma Council Australia \url{https://www.nationalasthma.org.au/living-with-asthma/resources/patients-carers/factsheets/car-air-filters}

\textsuperscript{78} Diana Phillips \textit{Even 'Safe' Levels of Air Pollution Tied to Higher Mortality} -\textit{Medscape}–Dec 27, 2017. \url{https://www.medscape.com/viewarticle/890636}
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><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>
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<tr>
<td>Ischaemic heart disease</td>
<td>Ischaemic stroke</td>
</tr>
<tr>
<td>Complications of diabetes</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td></td>
<td>Congestive heart failure</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</td>
<td>Bronchitis in children</td>
</tr>
<tr>
<td>(elderly, people with COPD or asthma)</td>
<td>COPD symptoms</td>
</tr>
<tr>
<td>Respiratory infections in children</td>
<td></td>
</tr>
<tr>
<td><strong>Cancer</strong></td>
<td><strong>Cancer</strong></td>
</tr>
<tr>
<td>Lung cancer mortality</td>
<td>Lung cancer mortality</td>
</tr>
<tr>
<td><strong>Neurological</strong></td>
<td><strong>Neurological</strong></td>
</tr>
<tr>
<td>Neurological disorders in adults</td>
<td>Neurological disorders in adults</td>
</tr>
<tr>
<td>Impaired cognitive function</td>
<td>Impaired cognitive function</td>
</tr>
<tr>
<td><strong>Development</strong></td>
<td><strong>Development</strong></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><strong>Reproduction</strong></td>
<td><strong>Reproduction</strong></td>
</tr>
<tr>
<td>Adverse birth outcomes</td>
<td>Adverse birth outcomes</td>
</tr>
<tr>
<td>Sperm quality and quantity</td>
<td>Sperm quality and quantity</td>
</tr>
<tr>
<td><strong>Allergies</strong></td>
<td><strong>Allergies</strong></td>
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<td>Exacerbation of allergies</td>
<td>Exacerbation of allergies</td>
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<tr>
<td>Allergic sensitization</td>
<td>Allergic sensitization</td>
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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. 

![Pie chart showing the proportion of deaths attributed to exposure of urban air pollution in Australia in 2003.](chart.png)

Source: Begg et al.

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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 pollution\(^{80,81,82,83}\)

2. Increase mortality associated with long-term effect of nitrogen dioxide NO\(_2\) as great as that of PM2.5\(^{84}\)

3. Increased risk of mortality, particularly for the elderly, even in areas of air pollution considered at "safe levels"\(^{85}\). 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\(^{86,87}\) and deaths, heart

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\(^{80}\) 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]


\(^{85}\) 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]


failure onset and mortality associated with heart failure, deep vein thrombosis [blood clots], heart disease, and myocardial infarction (heart attacks)\textsuperscript{88,89,90,91}.

4. Increase risk of heart failure due to a possible threshold of PM\textsubscript{2.5}=4 \mu g/m^{3} is far below the daily Australian national standard of 25 \mu g/m^{3}\textsuperscript{92}

5. Higher risk of developing hypertension [high blood pressure]\textsuperscript{93,94}.

6. Increase risk of stroke and mortality associated with stroke\textsuperscript{30} Pope,95,96

7. Reduced survival from long-term exposure to traffic-related air pollution and cancer among survivors of myocardial infarction (heart attacks)\textsuperscript{97}.

8. Higher risk of out-of-hospital cardiac arrest associated with elevated ambient PM\textsubscript{2.5} and CO\textsuperscript{98}


https://bmjopen.bmj.com/content/8/5/e021798


9. Reduced lung function, increase respiratory symptoms and diseases, respiratory related deaths, chronic obstructive Airways disease, cardiopulmonary disease, lung infections, and asthma. Pope,99,100,101,102,103,104

10. Increase risk of asthma in children. (29 Bow et al)

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.106

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\textsuperscript{109,110}. Air pollution shortens survival from lung cancer\textsuperscript{111}. 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{112}. 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{113}.

15. Increase sick leave from work\textsuperscript{114}.

16. Greater healthcare utilisation by both children and adults for respiratory infections\textsuperscript{115}.

17. Increased risk of hospitalisation due to pneumonia in children\textsuperscript{116}.


\textsuperscript{110} Joanne Kim, Cheryl E Peters, Victoria H Arrandale et al. Burden of lung cancer attributable to occupational diesel engine exhaust exposure in Canada \url{http://dx.doi.org/10.1136/oemed-2017-104950} \url{https://oem.bmj.com/content/75/9/617?etoc}


\textsuperscript{112} 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 http://www.pnas.org/content/early/2018/08/21/1809474115


18. Increased risk of diabetes and diabetes associated mortality\textsuperscript{117,118}.
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 adverse birth outcomes\textsuperscript{119,120,121} and may increase stillbirths\textsuperscript{122,123}.
20. Poor sperm quality and infertility\textsuperscript{124}.
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{125}.

\textsuperscript{117} 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


Melbourne projected population growth

The Melbourne population is projected to grow from the current estimate of 4.5 million [2017] -4.9 million [2018] people to 8 million people in 2050\(^{126}\).

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. 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.

Like many cities in Australia, Melbourne's increasing population and urban growth will result in more vehicles on freeways, contributing to further air pollution\(^{127}\).

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\(^{128}\).

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.”


\(^{128}\) Transport Security Outlook to 2025. Department of Infrastructure and Regional Development 2014 Australian Government, Canberra
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.

Tullamarine Freeway during peak hour traffic and now there are plans to widen it further to meet increase vehicle demand\(^\text{129}\).

Source: [Accessed 28th November 2018] https://www.google.com.au/search?hl=en&tbm=isch&source=hp&ei=BUX-W_WuN8XorQGAqIdOqA&q=tullamarine+freeway&oq=tullamarine+freeway&gs_i=mobile-gws-wiz-img.1.0.0.2414.1729.9512..12786...0.0...0.288.4508.0j3j16......0........1........2...41j0i8i30.vTTVctiZrY#imgrc=EuaYAKn3gp6gjM:

When constructed over 20 years ago Monash, Tullamarine and Westgate Freeways promised to offer better access of vehicles across Melbourne. The first five-to ten years demonstrated benefits, but with the growth of the population and number of vehicles, these freeways are now 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 Melbourne. Judging from our past experience with

Monash, Tullamarine and Westgate Freeways, such freeways 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 Melbourne will continue to deteriorate over time if we do not address air pollution now and limit vehicles into Melbourne city. Melbourne will suffer an increase in air pollution if we continue to widen roads for more vehicles to enter our city 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 Melbourne 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\(^\text{130,131}\). Studies and research demonstrate that planting more trees, encouraging leafy suburbs, green spaces, parklands and urban forests improve the general health of communities\(^\text{132,133}\).

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. 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.\textsuperscript{134} 

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.\textsuperscript{135,136} Certain trees also trap and filter pollens on windy days. 

3. Develop urban forests to help mitigate air pollution and improve air quality.\textsuperscript{137} 

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{138} 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{139} 


\textsuperscript{138} 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

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.

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.

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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\(^{143}\). However residents are still exposed to pollution depending on direction of wind and changing weather patterns as can be viewed in the following illustration:

Figure 1. Description of flow and pollutant dispersion patterns in a street canyon and open road with and without different type of vegetation\(^{143}\) Abhijith et al.

\(^{143}\) K.V.Abhijith, PrashantKumar, John Gallagher et al. Air pollution abatement performances of green infrastructure in open road and built-up street canyon environments – A review *Atmospheric Environment* Volume 162, August 2017, Pages 71-86

Alternative solutions to Freeways and Public transport

Alternative and affordable options to building freeways, such as extensive and improved public transport, have already been considered by the State Government and need to be seriously explored further as priorities before considering freeways to help cater for our growing population, and mitigating air pollution impacts from vehicle emissions on human health. For instance, new train stations and a rail loop are excellent proposals for reducing road traffic and providing more work for our population.\(^{144}\)

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\textsuperscript{145,146,147} for Melbourne to interconnect outer suburbs, and 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 queuing\textsuperscript{[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.

\textsuperscript{145} https://www.weforum.org/agenda/2018/06/estonia-is-making-public-transport-free/
\textsuperscript{146} https://www.weforum.org/agenda/2014/07/seven-ways-cities-around-world-tackling-traffic/
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.[148,149,150]

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.

Thank you!

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

References:

[148] 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 https://pursuit.unimelb.edu.au/articles/cleaner-safe-air-needs-you


[150] 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 https://pursuit.unimelb.edu.au/articles/cleaner-safe-air-needs-you
Appendix 1
- further references on air pollution impact on health

Appendix 2
A. Prof Louis Irving health concerns West Gate Tunnel Project LHRC report - relevant report re traffic related air pollution expressing the same concerns from research studies