



# Health impacts associated with traffic emissions in Australia.

**Expert  
Position  
Statement**

## Endorsed by:

Australian Chronic Disease Prevention Alliance



## Acknowledgements

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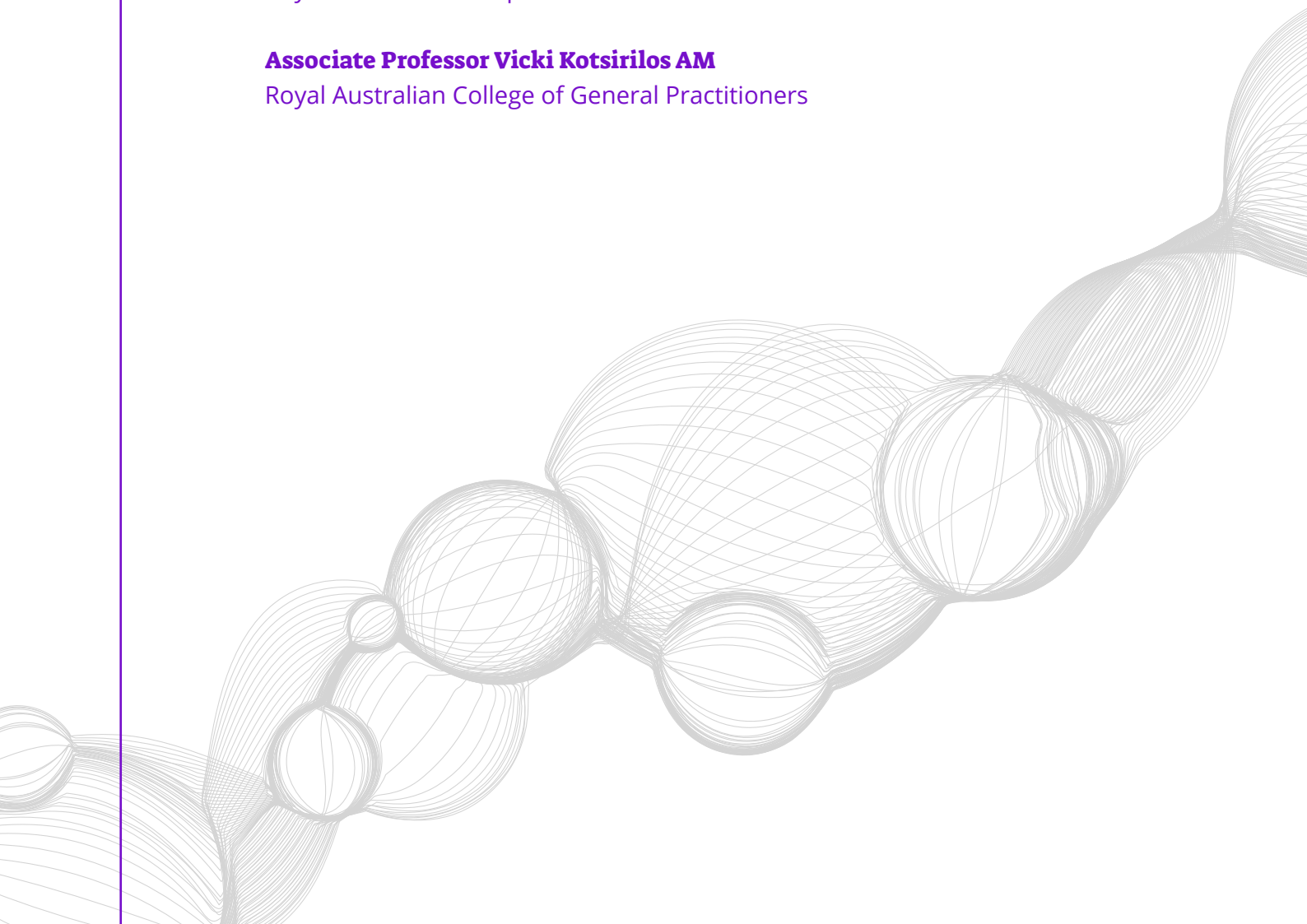
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## Background

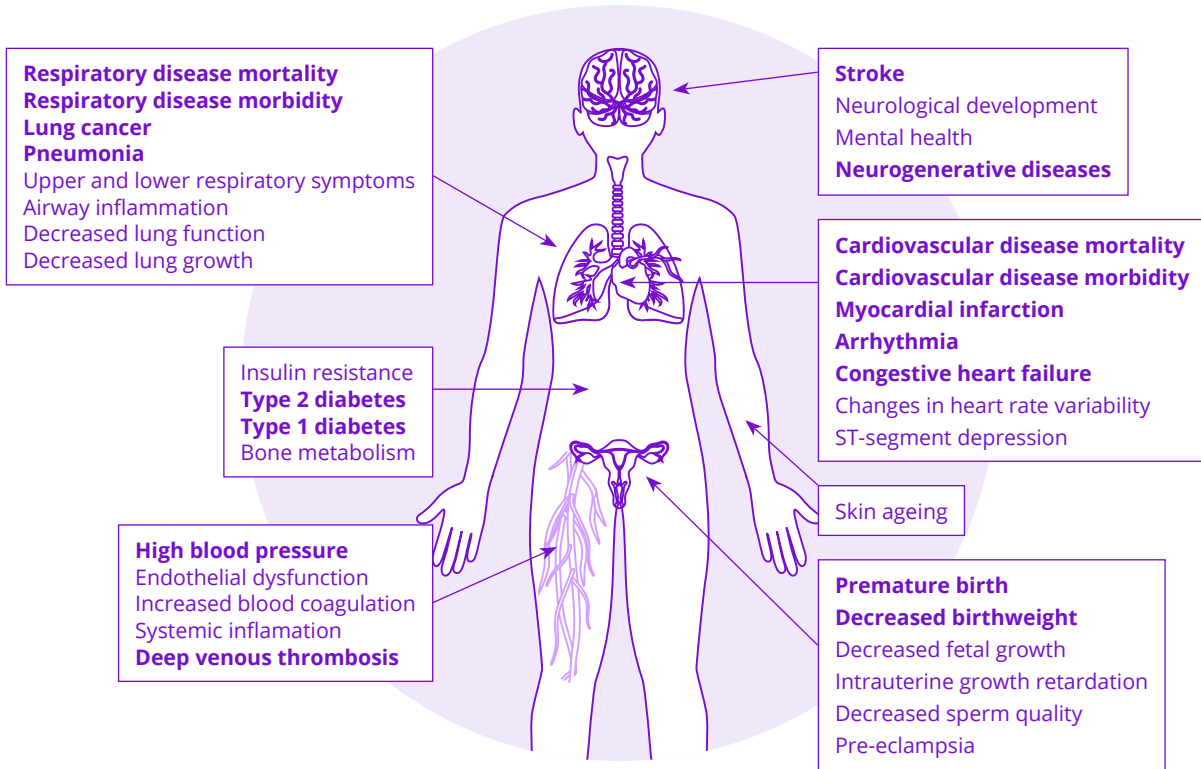
Although adverse health effects caused by traffic pollution have been known for two decades, recent evidence reveals a far greater magnitude of health impacts than previously considered. These impacts include premature deaths, a range of cardio-respiratory diseases including lung cancer, childhood asthma, adverse birth outcomes and diabetes.

In most urban areas, road traffic emissions which include dust, non-tailpipe and tailpipe emissions are the most widespread source of anthropogenic (man-made) air pollution that the public are chronically exposed to (Khreis, Nieuwenhuijsen et al. 2020). Tailpipe emissions include tiny respirable particles of black carbon and toxic gases that are released at ground level in locations where people spend much of their time, on roads, footpaths and in buildings along busy roads.

Currently, there are no robust estimates for the health impacts in Australia. Quantifying the health and economic effects of traffic emissions is critical given current federal discussions on vehicle regulations, fuel content and trajectories of decarbonising the traffic fleet. Robust estimates that align with current evidence are required to provide policymakers with the necessary data to weigh up the costs of inaction against the costs of action and the benefits that will follow.

The past decade of international evidence highlights there are two key pollutants to consider when estimating the health impacts of air pollution. Fine particulate matter (PM<sub>2.5</sub>) and nitrogen dioxide (NO<sub>2</sub>).

**Fine particulate matter (PM<sub>2.5</sub>)** is any airborne solid particle under 2.5 microns in diameter. Particulate matter is generated by natural (sea spray, dust, bushfires) and man-made processes (wood-heaters, industry, and motor vehicles). Particulate matter formed by combustion processes is particularly small and can enter the bloodstream leading to systemic inflammation and detrimental effects on organs throughout the body (Figure 1). The tiny size and chemical composition of vehicular exhaust particles contributes to a particularly toxic profile in terms of DNA damage and cytotoxic effects (Valavanidis, Fiotakis & Vlachogianni 2008). Figure 1 provides an overview of the health impacts currently associated with PM<sub>2.5</sub>.



**Figure 1.** Overview of diseases, conditions and biomarkers affected by outdoor air pollution. Bold type indicates conditions currently included in the Global Burden of Disease categories (Thurston, Kipen et al. 2017)

**Nitrogen dioxide (NO<sub>2</sub>)** is a gas formed from high temperature combustion, such as emissions from vehicles, power stations and industrial processes. In outdoor urban environments vehicles are the primary source of nitrogen dioxide (Emmerson KM 2021, Kuschel, Metcalf et al. 2022).

Combustion processes produce a range of pollutants making it hard to disentangle the individual effects of each pollutant. Traditionally health estimates were based on PM<sub>2.5</sub> due to availability of air quality data and a more robust body of evidence related to health impacts. However, studies with multi-pollutant models investigating general ambient or traffic related pollution have demonstrated significant effects from NO<sub>2</sub> that are independent of PM<sub>2.5</sub> (Walter, Schneider-Futschik et al. 2021).

NO<sub>2</sub> has been estimated to account for 13% of global paediatric asthma incidence (Achakulwisut, Brauer et al. 2019), and relatively small increases (2.13ppb) have been associated with a 5% increase in the risk of developing childhood asthma (Khreis, Kelly et al. 2017). The Australian Children's Health and Air Pollution Study (ACHAPs) reveal significant asthma impacts in Australia, where a 4ppb increase in NO<sub>2</sub> was associated with a 54% increased risk of asthma prevalence in children (Knibbs, de Waterman et al. 2019). Despite a reduction in tobacco smoking rates, lung cancer remains the leading cause of cancer related mortality in Australia and is also causally associated with air pollution. A 5ppb increase in NO<sub>2</sub> is associated with a 4% increase in the risk of lung cancer (Hamra, Laden et al. 2015). There is no threshold below which impacts do not occur, therefore small improvements even at low concentrations will yield significant health gains.

## International estimates

In 2010 the UK Committee on the Medical Effects of Air Pollution (COMEAP 2010) estimated an annual 29,000 premature deaths attributed to anthropogenic (man-made) PM<sub>2.5</sub>. In 2015 the UK's Department for Environment Food and Rural Affairs (Defra) estimated an annual 23,500 deaths were attributed to NO<sub>2</sub> (COMEAP 2015). After accounting for some overlap between pollutants the estimated combined effect of both pollutants was 40,000 annual premature deaths (Holgate 2017).

The New Zealand HAPINZ 3.0 study provides recent internationally peer reviewed estimates of the health and economic impacts of air pollution in New Zealand (Figure 2). Using both NO<sub>2</sub> and PM<sub>2.5</sub>, an annual 3,300 premature deaths were attributed to anthropogenic (man-made) air pollution with over two thirds (>2,200) attributed to motor vehicles, costing the New Zealand economy an annual \$15.5 billion (Kuschel, Metcalf et al. 2022). Motor vehicles were estimated to contribute 17% of the PM<sub>2.5</sub> and 100% of the NO<sub>2</sub>, making it the single largest source of anthropogenic air pollution in New Zealand (Figure 2). The HAPINZ authors described their findings as 'surprising' and 'startling', and undertook numerous additional analyses to check for bias, as well as rigorous peer-review.

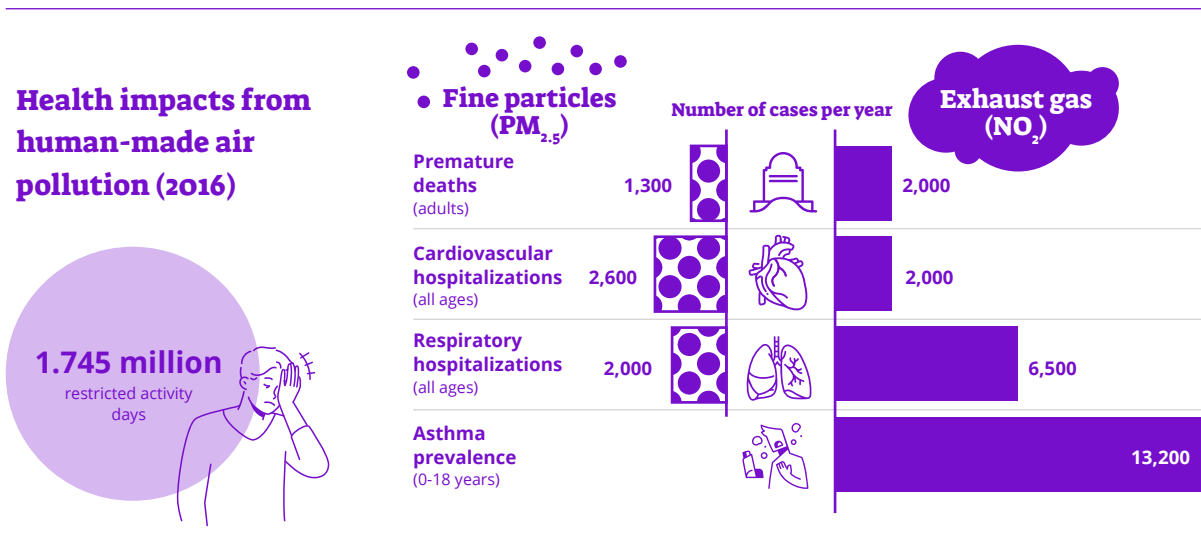


Figure 2. Key findings of the HAPINZ 3.0 Study

## Australian context

To date there are no robust estimates of the health and economic impacts of vehicle emissions in Australia. Estimates have been based on desktop extrapolations of the contribution vehicle exhaust makes to overall PM<sub>2.5</sub> and do not include NO<sub>2</sub>. Due to urbanisation demographics, fuel content, traffic fleet composition and similar underlying asthma prevalence, it's reasonable to anticipate health impacts are the same if not worse per capita compared to New Zealand. Scaling New Zealand results for PM<sub>2.5</sub> and NO<sub>2</sub> from motor vehicles, up to the Australian population produces the following figures for motor vehicle emissions:

Premature deaths (adults)	<b>11,105</b>
Cardiovascular hospitalisations (all ages)	<b>12,210</b>
Respiratory hospitalisations (all ages)	<b>6,840</b>
Asthma prevalence (0 – 18 years)	<b>66,000</b>

These figures are magnitudes greater than estimates used to inform policy decisions, highlighting Australians are exposed to a much larger health burden attributed to traffic related air pollution than currently recognised. It is important policy decisions are informed by the most recent evidence, and these figures highlight the need for an urgent preventative focus. There are few education or preventative measures in place to reduce population exposure to vehicle exhaust.

## Vulnerable groups

Air pollution impacts are not evenly distributed across the population. A range of intrinsic (biological) and extrinsic factors, contribute to higher risks in unborn children, children, elderly, Indigenous Australians, people with underlying disease and disadvantaged populations. Of these groups, children are the most impacted by chronic exposure to traffic pollution (Walter, Schneider-Futschik et al. 2021).

**Unborn children** have a high frequency of vulnerable points where the development and growth of organs, hormone and immune systems can be damaged. In-utero exposures are associated with still birth, low birth weight, premature birth and organ damage (Makri and Stilianakis 2008). Emerging evidence also indicates impaired respiratory and immune development and adverse impacts on neurodevelopment including a reduced IQ (Suades-González, Gascon et al. 2015). Combusted PM<sub>2.5</sub> particles are small enough to cross the placenta and cause in-utero DNA damage leading to adverse effects later in life including epigenetic effects (inherited by subsequent generations) (Pedersen, Wichmann et al. 2009).

**Children** have a much higher respiratory rate, larger surface area of lungs relative to the rest of their bodies, and immature defence systems that allow greater penetration of air pollutants (US EPA, 2011). Extrinsic factors include longer periods of physical activity outdoors and closer proximity to exhaust pipes due to their lower height or being pushed in a pram. Traffic emissions are causally associated with the incidence and prevalence of asthma and respiratory infections in children (HEI, 2022). Chronic exposure has been associated with reduced lung development that is carried through into adulthood (Gauderman, Urman et al. 2015). More recently vehicle exhaust exposure in the school environment has been linked to systemic inflammation (Clifford, Mazaheri et al. 2018), impaired cognitive development (Sunyer, Esnaola et al. 2015) and behavioural problems in schoolchildren (Forns, Davvand et al. 2016).

## Mitigation strategies

International policies that focus on protecting children from vehicle emissions have demonstrated some success in reducing children's exposure to vehicle emissions (Gauderman, Urman et al. 2015, Abhijith, Kukadia et al. 2022) which have been associated with measurable health improvements (Gauderman, Urman et al. 2015). Examples of some of these strategies include:

- Active transport initiatives
- Anti-idling legislation
- Replacement of diesel school buses
- Pollution barriers around schools
- Low emission zones 'school streets'
- Buffer zones between schools and major road
- Banning diesel vehicles.

Australia has yet to widely implement any of these mitigation strategies and continues down the path of car-centric urban design, forcing further reliance on individual motor vehicles. Current trajectories of diesel uptake and population growth indicate increasing health and economic impacts (ABS,2021)

## Raising public awareness

Raising public awareness is a critical first step. In much the same way it is no longer socially acceptable to smoke tobacco in the vicinity of children, exposing children to vehicle exhaust (for example idling cars outside of schools and childcare centres) would no longer be tolerated. Successful decarbonisation of transport will require a new cultural paradigm. A widespread public awareness of the profound impacts of vehicle exhaust on public health will help drive the necessary changes, including the levers required to promote public and active transport, reduce our reliance on car culture, and electrify the remaining vehicle fleet.

## A car without an exhaust pipe is good, a bicycle is even better

Reducing population exposure to vehicle emissions requires the adoption of multiple strategies, including the promoting and incentivising of active transport. There are significant health co-benefits associated with active transport including, reductions in obesity, type 2 diabetes, and cardiovascular disease (Jarrett, Woodcock et al. 2012, Penn, Bartington et al. 2022). Broader benefits include the facilitation of urban greening which in turn have further mental health benefits (Schebella, Weber et al. 2019) and help reduce urban heat island effects (Aram, Garcia et al. 2019).

## Recommendations

1. Update vehicle and fuel standards to bring Australia into line with international markets by 2030. This includes incorporating new vehicle carbon dioxide standards; Australia is currently the only OECD country yet to do this. As Europe is about to implement Euro 7 standards for fuel quality and vehicle emissions, Australia is currently a decade behind with the Euro 5 standards, a lag that needs urgent redress.
2. Registration checks should include exhaust quality, to pick up poorly maintained vehicles, or those with tampered pollution controls. This would help target the small number of highly polluting vehicles (both cars and trucks) that contribute an outsized share of the problem.
3. Adopt interim mitigation strategies that focus on protecting the most vulnerable members of society. These may include pollution barriers, low emission zones and anti-idling policies. Where possible, sensitive use facilities such as schools and childcare centres should not be located on or close to major traffic routes. When this does occur, specific factors should be considered in the design, such as aerodynamics, direction of windows and other ventilation intakes, air purifiers with HEPA filtration and location of outdoor play areas.
4. Update Australian estimates of mortality and morbidity impacts attributed to vehicle emissions to reflect the most recent evidence. Estimates should account for the full range of causal associations, including diabetes and adverse birth outcomes and in alignment with the precautionary principle, consideration should also be given to probable associations. Concentration response functions (CRFs) should be used for both PM<sub>2.5</sub> AND NO<sub>2</sub>, and where possible sensitivity analyses should incorporate available Australian CRFs. In addition, the analysis should account



for fleet emission factors that are specific to the Australian context, including fleet and fuel composition. Whilst this analysis is needed, policy decisions should not be delayed, the precautionary principle should be observed, and the recent international evidence should be used to guide policy decisions.

5. Undertake an economic analysis of the social costs that result from these anthropogenic PM<sub>2.5</sub> and NO<sub>2</sub> emissions and the influence of transport electrification pathways on these costs.
6. Undertake a campaign to raise public awareness regarding the impacts of vehicle emissions on public health. The bushfire smoke crisis highlighted a need for community education and guidance around air quality, leading to Asthma Australia's Air Smart program. Expanding this to incorporate roadside NO<sub>2</sub> monitoring linked to maps would build awareness and provide the public with information that can be used to protect themselves when considering transport routes.
7. Focus infrastructure spending on active and public transport and develop policies that meaningfully incentivise these modes.

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