Review of the health effects of traffic-related air pollution

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Executive summary

In the past 10 to 15 years, research into the adverse health effects of ambient air pollution has resulted in an increasingly robust evidence base to support health impact assessments of traffic-related air pollution.

- The number of published studies on traffic-related air pollution and health has increased.
    - The 2010 Health Effects Institute report on traffic-related air pollution reviewed 167 scientific papers, whilst the 2022 Health Effects Institute report on traffic-related air pollution, some 12 years later, reviewed 353 scientific papers.
    - Across all five broad groups of health outcomes, more studies were reviewed in the 2022 Health Effects Institute report compared to the 2010 Health Effects Institute report.
- The evidence for causal associations or confidence in the evidence for associations between traffic-related air pollution and adverse health effects has strengthened.
    - Compared to the 2010 Health Effects Institute report, the 2022 Health Effects Institute report upgraded the confidence in the evidence for associations between traffic-related air pollution and adverse health outcomes for five health outcomes.
    - The 2022 Health Effects Institute report also concluded there is moderate to high confidence in the evidence for associations between traffic-related air pollution and adverse health outcomes for a further six health outcomes. These six health outcomes were not considered in the 2010 Health Effects Institute report.
    - In 2019, the United States Environmental Protection Agency upgraded the causality determination for three broad groups of health outcomes (nervous system effects, cancer, and metabolic effects) from the determination reported in 2009.
- The number of concentration-response functions available for quantification in a health impact assessment has increased.
    - Compared to 2013, 20 additional concentration-response functions for $PM_{2.5}$-outcome pairs are now available for inclusion in a health impact assessment - sub-categories of mortality (n=5), metabolic effects (n=5), respiratory effects (n=4), neurological effects (n=2), and birth outcomes (n=4).
    - Compared to 2013, 22 additional concentration-response functions for NO_2-outcome pairs are now available for inclusion in a health impact assessment - sub-categories of mortality (n=6), metabolic effects (n=5), respiratory effects (n=7), and birth outcomes (n=4).

Previous health impact assessments for road tunnels quantified limited health outcomes (5-6) with only one outcome associated with long-term exposure to air pollutants ($PM_{2.5}$ and mortality). These previous road tunnel health impact assessments would have underestimated the impact of any changes in air quality on human health. However, they could be considered to have been conducted according to the known evidence at that time. Future health impact assessments, whether conducted for road tunnels or for other major infrastructure developments that could impact air quality, or for policy purposes, should consider the additional health outcomes for which robust concentration-response functions are now available.

The scientific consensus is that there is no threshold for $PM_{2.5}$ and NO_2 below which adverse health effects do not occur (i.e., there is no “safe” level). As on-road motor vehicles account for a sizable contribution to ambient air pollution, and with the increasing number of adverse health effects attributable to air pollution, governments and regulators must continue to implement and evaluate the effectiveness of measures to reduce exposure to traffic-related air pollution thereby improving the health and well-being of the population.

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Abbreviations

ACTAQ	Advisory Committee on Tunnel Air Quality
ALRI	Acute lower respiratory infection
BC	Black carbon
BS	Black smoke
CO	Carbon monoxide
CO_2	Carbon dioxide
COMEAP	Committee on the Medical Effects of Air Pollutants
COPD	Chronic obstructive pulmonary disease
CRF	Concentration-response function
EC	Elemental carbon
HEI	Health Effects Institute
HIA	Health impact assessment
HR	Hazard ratio
HRAPIE	Health risks of air pollution in Europe project
IARC	International Agency for Research on Cancer
IHD	Ischaemic heart disease
ISA	Integrated Science Assessment
NSW	New South Wales
NO	Nitrogen oxide
NO_2	Nitrogen dioxide
NO_x	Oxide of nitrogen
O_3	Ozone
OR	Odds ratio
PAH	Polycyclic aromatic hydrocarbon
PM	Particulate matter
$PM_{2.5}$	Particulate matter ≤2.5 μm in aerodynamic diameter
PM_{10}	Particulate matter ≤10 μm in aerodynamic diameter
RR	Relative risk
SO_2	Sulphur dioxide
TRAP	Traffic related air pollution
UFP	Ultrafine particle, particulate matter ≤0.1 μm in aerodynamic diameter
US EPA	United States Environmental Protection Agency
VOC	Volatile organic compound
WHO	World Health Organization

Scope of works

The scope of works has been defined by the Advisory Committee on Tunnel Air Quality (ACTAQ) as follows:

Provide a commentary on the current evidence for the health effects of traffic-related air pollution and any implications regarding the findings of Environmental Impact Statements for recent motorway tunnel projects. A review of the primary literature is not required. The review will include a desktop review to identify relevant recent (last ten years) reviews and meta-analyses.

The review will include, but not be limited to, consideration of the following:

- *The Advisory Committee on Tunnel Air Quality Technical papers and Initial Report on Tunnel Air Quality;*
- *The Environmental Impact Statements and related technical reviews for NorthConnex, M4East, M4-M5 Link, new M5, M6 Stage 1 and Western Harbour Tunnel;*
- *Health impacts associated with traffic emissions in Australia. Expert Position Statement by The University of Melbourne; and,*
- *Health Effects Institute Panel on the Health Effects of Long-Term Exposure to Traffic-Related Air Pollution, 2022. Systematic Review and Meta-analysis of Selected Health Effects of Long-Term Exposure to Traffic-Related Air Pollution.*

This report will focus on:

1. Particulate matter less than or equal to 2.5 microns in aerodynamic diameter ($PM_{2.5}$), nitrogen dioxide (NO_2) and indirect measures (traffic intensity/density, proximity to roads) as markers of traffic-related air pollution (TRAP).

2. Health effects attributable to long-term exposure to traffic-related air pollution. Short-term exposure studies have been used to quantify associations between day-to-day changes in air pollution concentrations with day-to-day changes in numbers of health events. However, studies of the association between short-term changes in exposure and mortality provide little information about how short-term exposure relates to survival, so they cannot be used to quantify years of life lost, disability-adjusted life years or loss of life. Short-term exposure studies are also unable to account for background levels of exposure. Therefore, focusing on long-term exposure studies, where available, is preferable.

3. The 2022 report on the health effects of long-term exposure to TRAP from the Health Effects Institute (HEI) and on the Expert Position Statement from The University of Melbourne on the health effects of traffic emission in Australia.

This report will follow the practice of Boogaard et al. (Boogaard et al. 2022) and use the term relative risk (RR) to describe risk (or effect) estimates, as it is easier to communicate, even if in some of the included studies it would be technically more correct to refer to the risk as an odds ratio (OR) or hazard ratio (HR). All these three risk estimates (RR, OR and HR) are derived from epidemiological studies. However, the risk estimate is defined as a RR, HR, or an OR, depending on the statistical model. The interpretation of the risk, however, is similar. In the air pollution literature, these risk estimates are referred to as concentration-response

functions (CRFs) or exposure-response functions. In this report, the term CRF is used instead of exposure-response function.

Introduction

Ambient air pollution and health

Air pollution is one of the greatest environmental threats to human health and wellbeing. Ninety-nine percent of the world's population breathes air that exceeds World Health Organization's (WHO) air pollution guidelines for health (WHO 2022). Globally, in 2021, air pollution was the second leading risk factor for early death, second only to high blood pressure, and 8.1 million deaths were attributable to air pollution, or about 1 in 8 of all deaths (Health Effects Institute 2024).

Although air quality in Australia is relatively good, about 54% of the population lives in areas where $PM_{2.5}$ levels do not meet the national annual standard (8 $\mu g/m^3$) (https://www.stateofglobalair.org/resources/countryprofiles). The WHO guideline for annual $PM_{2.5}$ level is 5 $\mu g/m^3$ (WHO 2021) which is much lower than the national standard.

In Australia, over the 11 year period between 2006 and 2016, 2,616 premature deaths annually were attributed to anthropogenic (human-generated) $PM_{2.5}$ air pollution, at an average cost of $6.2 billion (Hanigan et al. 2020). These estimates are conservative as they do not include other health outcomes attributable to anthropogenic $PM_{2.5}$ or other important air pollutants detrimental to health.

In a health impact assessment (HIA) conducted by the New South Wales (NSW) Department of Planning and Environment in the Sydney Greater Metropolitan Region (GMR), anthropogenic sources contributed 48% (3.07 $\mu g/m^3$) to the population-weighted annual average $PM_{2.5}$ of 6.43 $\mu g/m^3$ (DPE 2023). On-road motor vehicles accounted for 17% of the population-weighted concentration from all anthropogenic sources of $PM_{2.5}$ (motor vehicle exhaust 13%; motor vehicle non-exhaust 4%). Wood heaters made the largest contribution to anthropogenic $PM_{2.5}$ (42%).

In NSW, about 600 premature deaths annually were attributed to anthropogenic sources of $PM_{2.5}$ costing $5.02 billion (in 2021 AUD). The share of on-road motor vehicles (from both exhaust and non-exhaust sources) was 110 premature deaths annually costing $913 million (in 2021 AUD) (DPE 2023).

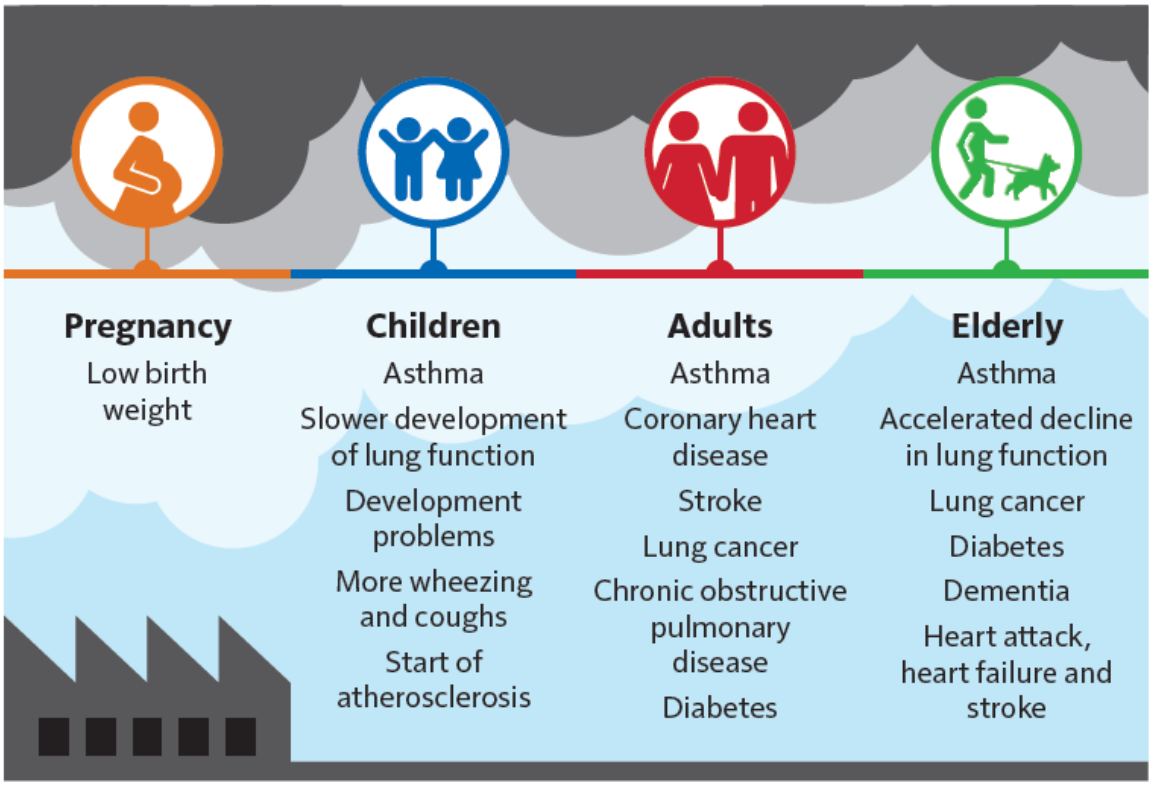
The adverse health effects of air pollution are predominantly due to deaths from, and exacerbation of, cardiovascular and respiratory diseases (Landrigan et al. 2018). However, evidence for a range of health effects beyond these traditional health metrics is well-established, e.g., for metabolic disease (particularly diabetes), neurodevelopment and neurocognitive decline, premature birth, and impaired childhood development leading to an increased risk of chronic disease later in life (Manisalidis et al. 2020). Outdoor air pollution, $PM_{2.5}$ specifically, and diesel exhaust, have been designated as carcinogenic by the International Agency for Research on Cancer (IARC) (Loomis et al. 2013, Benbrahim-Tallaa et al. 2012). Benzene and formaldehyde (constituents of vehicle exhaust) have also been classified by IARC as Group 1 carcinogens (IARC 2012).

There is now good evidence to suggest that $PM_{2.5}$ is causally linked to cardiovascular effects and mortality, and likely to be causal for respiratory effects, cancer and nervous system effects (US EPA 2024). There is moderate confidence in the evidence for an association between air pollution and type 2 diabetes (Health Effects Institute 2022). Further, $PM_{2.5}$ is regarded as a modifiable risk factor for dementia (Livingston et al. 2020) and cardiovascular

mortality and morbidity (Brook et al. 2010). This means these air pollution-related diseases are amenable to primary prevention measures. Air pollution should be considered as one of the many traditional risk factors for non-communicable diseases, e.g., cigarette smoking, obesity, poor diets, and inadequate physical activity.

Air pollution has adverse effects on health throughout life, from before birth to old age (Figure 1) (Whitty 2022). Unfavourable conditions in childhood can lead to chronic diseases later in adulthood. Improvements in ambient air quality are associated with improved lung function growth in children (Gauderman et al. 2015). In other words, the adverse effect of air pollution on lung growth in children is reversible (Dockery and Ware 2015). The reversibility of lung function growth has important ramifications as reduced lung function in childhood and later life strongly predicts both chronic respiratory and chronic cardiovascular disease in adults (Hole et al. 1996).

FIGURE 1 HEALTH EFFECTS OF AIR POLLUTION THROUGHOUT LIFE, WHITTY 2022



Previously published HIAs and health-related economic analyses will not have accounted for the recent new evidence on the adverse health outcomes attributable to air pollution. Hence, previous air pollution policy decisions would have been informed by underestimations of the health and economic burden, and the ensuing policy decisions may be inadequate in preventing harm from air pollution. Of course, policy making is complex, and the health and associated economic burden are not the only determinants for policy making. Other factors, such as, the cost of interventions to reduce air pollution, the public's perception of risks, opportunity costs, competing priorities, political ideology, and strength of lobby groups, are also important, to varying degrees, in influencing and shaping policy decisions.

In Australia, the National Environment Protection Council guidelines for setting air quality standards outline methods that go beyond the technical risk assessment process and consider the actions to meet proposed standards and the costs and benefits associated with

implementing those actions. (National Environment Protection Council 2011). It is not possible to set a standard that is completely protective of health because adverse health effects have been detected at very low levels of air pollution. Recent studies conducted in Europe and North America have shown that annual averages of $PM_{2.5}$ as low as 4 $\mu g/m^3$, or even lower, are associated with adverse health effects (Health Effects Institute 2024). The consensus is that, for $PM_{2.5}$ and NO_2, there are no safe levels of exposure.

The current approach to regulating air pollution provides no incentive for reducing exposure. It allows increases in exposure to harmful air pollutants, as long as the levels remain below the standards (Zosky et al. 2021). Standards should be complemented or replaced by an approach focused on harm minimisation by a general continual reduction in urban background air pollution levels even when air pollution levels are lower than the standards. This approach has been adopted by the European Union (European Commission 2008). Such a policy ensures that large sections of the population benefit from continually improving air quality.

Traffic-related air pollution

Motor vehicles are an important source of urban air pollution. They are also contributors to anthropogenic carbon dioxide and other greenhouse gases. TRAP is a complex mixture of gases and particles emitted from motor vehicles, including heavy-duty and light-duty vehicles, buses, passenger cars, and motorcycles. TRAP includes nitrogen oxides (NO_x), elemental carbon (EC), $PM_{2.5}$, particulate matter ≤10 µm in aerodynamic diameter (PM_{10}), ultrafine particles (UFPs, particulate matter ≤0.1 µm in aerodynamic diameter), heavy metals, polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (VOCs).

These pollutants are called *exhaust* or *tailpipe emissions* when emitted through vehicle exhaust. When emitted by other means, such as evaporative emissions from fuel, the resuspension of dust, the wear of brakes and tyres, and the abrasion of road surfaces, they are called *non-exhaust or non-tailpipe emissions* (Health Effects Institute 2022).

The rate at which vehicle emissions disperse into ambient air depends on factors that are highly variable, including wind speed, wind direction, atmospheric stability, and terrain and land use. In addition to TRAP, air pollution from other sources, such as, industry, oil, coal and wood burning, agricultural sources, and atmospheric transport of pollutants from distant sources, contributes to the overall air quality.

The concentrations of ambient air pollutants are a combination of primary emissions and the formation of secondary pollutants, such as, secondary particulate matter (PM) and ozone (O_3).

People are exposed to these air pollutants when outdoors or indoors (through the infiltration of outdoor air pollutants). Other factors, such as mobility patterns and distance from the source, also determine human exposure.

As TRAP is a complex mixture of particles, aerosols and gases, epidemiological health studies use direct and indirect proxy measures or markers of exposure to TRAP. Direct measures or markers include $PM_{2.5}$, NO_2, EC, and black smoke (BS), whilst indirect proxy measures of exposure to TRAP include exposure to traffic, e.g., distance to roads and traffic density. The chain of events linking TRAP to health effects is shown in Figure 2 (Khreis et al. 2020).

FIGURE 2 THE FULL CHAIN OF EVENTS LINKING TRAFFIC-RELATED AIR POLLUTION TO HEALTH EFFECTS, KHREIS 2020

Figure 1.1. The full chain of events linking TRAP to health effects. Source: Center for Advancing Research in Transportation Emissions, Energy and Health (CARTEEH), available from https://www.carteeh.org/.

In general, the health effects of TRAP are very similar to the health effects of ambient air pollution. This is not surprising as TRAP is one of many sources of ambient air pollution. TRAP may contribute varying amounts to the total ambient air pollution, depending on the air pollutant.

For example, in the Sydney GMR, anthropogenic sources contributed 40% of $PM_{2.5}$ and 99% of NO_2 to the population-weighted annual averages (DPIE 2020). Wood heaters, industry, on-road motor vehicles and power stations, respectively, accounted for 31%, 26%, 19% and 17%, of the total anthropogenic source contribution to annual average $PM_{2.5}$ levels.

In contrast, on-road motor vehicles, non-road diesel and marine, other human-generated sources, and power stations, respectively, accounted for 63%, 15%, 9% and 7% of the total anthropogenic source contribution to annual average NO_2 levels (DPIE 2020) (Figure 3).

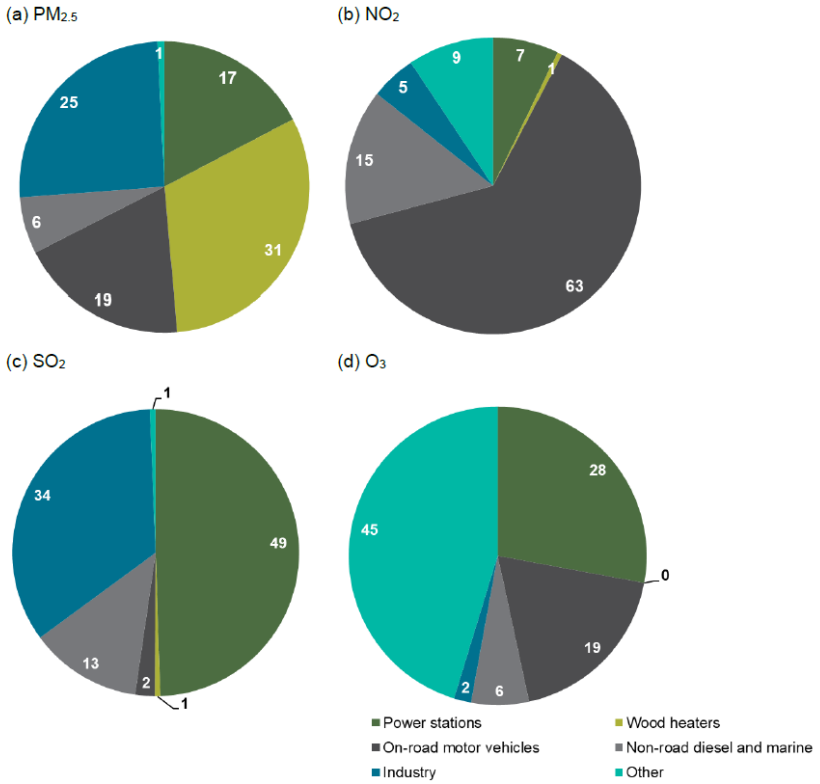
Non-exhaust emissions are also a source of on-road particle emissions. As exhaust emissions are further regulated and reduced, the health impacts of non-exhaust emissions will assume greater importance (Health Effects Institute 2010).

The differences in health effects from air pollution stemming from different sources (e.g., TRAP, bushfire smoke, industry) may be explained by differential toxicity (Khreis et al. 2020).

Some of the factors that might be responsible for differential toxicity include the different chemical composition of the TRAP mixture as well as the different physical characteristics of the particles, such as, size, surface area, and number of the smallest particles that are inhaled and reach the target organs (Khreis et al. 2020).

Although the current view is that all PM, regardless of the source, are equally toxic (Hime, Marks and Cowie 2018), there is some evidence that the increased risk of cardiovascular diseases is due to $PM_{2.5}$ from coal combustion and diesel exhaust (Thurston et al. 2021).

FIGURE 3 MAJOR SOURCE GROUPS CONTRIBUTIONS (%) TO TOTAL HUMAN-MADE SOURCES CONTRIBUTION TO POPULATION-WEIGHTED: (A) ANNUAL AVERAGE $PM_{2.5}$, (B) ANNUAL AVERAGE NO_2, (C) ANNUAL AVERAGE SULPHUR DIOXIDE (SO_2) AND (D) HOURLY MAXIMUM O_3 FOR THE GREATER METROPOLITAN REGION , DPIE 2020



Review of selected relevant reports

Expert Position Statement, Melbourne Climate Futures, The University of Melbourne, 2023

The Expert Position Statement on the "Health Impacts Associated with Traffic Emissions in Australia" from Melbourne Climate Futures, The University of Melbourne, (herein known as the Expert Position Statement or EPS) (Walter and Say 2023) aims to provide robust estimates of the health and economic impacts of motor vehicle emissions in Australia and inform policy decisions. It was published in early 2023.

The EPS scales up the results from the HAPINZ 3.0 study, that was conducted in New Zealand (Kuschel et al. 2022a, Kuschel et al. 2022b), to the Australian population to estimate the health impacts from motor vehicle emissions for Australia. The HAPINZ 3.0 study assessed the impacts of long-term exposure to $PM_{2.5}$ and NO_2 (considered surrogate measures of TRAP) from two-pollutant models, as these air pollutants contribute most to air pollution adverse health effects in New Zealand.

The HAPINZ 3.0 study determined health impacts for the following sources of air pollution:

- Anthropogenic air pollution sources:
    - motor vehicles (exhaust and brake/tyre wear from on-road vehicles);
    - domestic fires (wood and coal burning for home heating);
    - windblown dust (construction, land use activities, road dust, etc.); and,

    - industry.
- natural air pollution sources:
    - sea spray (sea salt); and,
    - secondary particulate matter (atmospheric gases reacting to form particles).

The health outcomes included in the HIA were mortality, restricted activity days, cardiovascular disease hospitalisation, respiratory disease hospitalisation, asthma/wheeze hospitalisation (age 0-18 years), and asthma prevalence (age 0-18 years). All CRFs for the HIA were derived from an associated New Zealand-wide study (Hales et al. 2021) except for the CRF for asthma prevalence in children which was derived from Khreis et al. (Khreis et al. 2017). In the HAPINZ 3.0 study, motor vehicles were estimated to contribute 17% of the $PM_{2.5}$ and 100% of the NO_2 to ambient air pollution.

The EPS presented the annual health impact of motor vehicle emissions in Australia for four health outcomes:

- 11,105 premature deaths in people aged ≥30 years;
- 12,210 cardiovascular hospitalisations in people of all ages;
- 6,840 respiratory hospitalisations in people of all ages; and,
- 66,000 young people (0-18 years of age) with asthma (asthma prevalence).

The EPS concludes that the health impacts are magnitudes greater than previously published estimates and that Australians are exposed to a much larger health burden attributed to TRAP than currently recognised.

There are many reasons why the EPS health impacts may be magnitudes greater than previously published estimates:

1. Previous Australian HIAs of long-term exposure to air pollution have focused only on $PM_{2.5}$ and have not included NO_2 associated adverse health effects. This was because either the objective of the study was related to $PM_{2.5}$, e.g., shipping (Broome et al. 2016), biomass smoke (Borchers-Arriagada et al. 2020), source-specific $PM_{2.5}$ (Broome et al. 2020), bushfire smoke (Johnston et al. 2021) or wood heater smoke (Borchers-Arriagada et al. 2024) or because there was limited evidence that health effects were causally related to NO_2.

   The US EPA has determined that the association between NO_2 and mortality is *likely to be a causally related* rather than *causally related* (US EPA 2024). The recent 2022 HEI report on TRAP (Health Effects Institute 2022) upgraded the causality determination for NO_2 and mortality from *suggestive but not sufficient evidence to infer a causal association* to *high confidence in the evidence for an association between NO_2 and mortality*.

2. The CRFs for long-term exposure to $PM_{2.5}$ and hospital admission used in the EPS (derived from the HAPINZ 3.0 study) were 1.115, 95%CI: 1.084-1.146 per 10 $\mu g/m^3$ for cardiovascular hospitalisation and 1.070, 95%CI: 1.021-1.122 per 10 $\mu g/m^3$ for respiratory hospitalisation. Previous Australian HIAs only included the short-term exposure adverse health effects (Borchers-Arriagada et al. 2020, Johnston et al. 2021).

Two Australian studies have investigated long-term exposure to $PM_{2.5}$ and respiratory hospitalisation – one from Sydney (Salimi et al. 2018) and the other from Perth (Salimi et al. 2022). Both studies produced non-significant associations – RR 0.82, 95%CI: 0.44-1.51 per 10 μg/m^3 for Sydney and RR 1.05, 95%CI: 0.82-1.35 per 10 μg/m^3 for Perth. Both these estimates are smaller than that used in the EPS (RR 1.115, 95%CI: 1.084-1.146 per 10 μg/m^3). There are no Australian studies on long-term exposure to $PM_{2.5}$ and cardiovascular disease hospitalisation.

There are no recommended CRFs for long-term exposure to $PM_{2.5}$ and hospital hospitalisation from WHO, the United Kingdom's Committee on the Medical Effects of Air Pollutants (COMEAP), US EPA or the HEI.

3. The CRFs for long-term exposure to NO_2 and hospital admission used in the EPS was 1.047, 95%CI: 1.031-1.064 per 10 μg/m^3 for cardiovascular hospitalisation and 1.130, 95%CI: 1.102-1.159 per 10 μg/m^3 for respiratory hospitalisation. Previous Australian HIAs included only the short-term health effects of exposure to NO_2 (Borchers-Arriagada et al. 2020, Johnston et al. 2021).

   Two Australian studies investigated long-term exposure to NO_2 and respiratory hospitalisation – one from Sydney (Salimi et al. 2018) and the other from Perth (Salimi et al. 2022). Both studies produced non-significant associations – RR 0.98, 95%CI: 0.90-1.06 per 10 μg/m^3 for Sydney and RR 1.00, 95%CI: 0.90-1.11 per 10 μg/m^3 for Perth. Both these estimates are smaller that used in the EPS (RR 1.130, 95%CI: 1.102-1.159 per 10 μg/m^3). There are no Australian studies on long-term exposure to NO_2 and cardiovascular hospitalisation.

   There are no recommended CRFs for long-term exposure to NO_2 and hospital hospitalisation from WHO, COMEAP, US EPA or HEI.

4. The CRF for $PM_{2.5}$ and mortality (RR 1.105, 95%CI:1.065-1.145) developed for the HAPINZ 3.0 study, and subsequently used in the EPS, is substantially larger than that used in previous Australian HIAs. Recent Australian HIAs (Borchers-Arriagada et al. 2020, Borchers-Arriagada et al. 2024, Broome et al. 2016, Broome et al. 2020, Johnston et al. 2021, Hanigan et al. 2020) have all used the $PM_{2.5}$-mortality CRF (RR 1.062, 95%CI: 1.040-1.083) from Hoek et al. (Hoek et al. 2013). Borchers et al. (Borchers-Arriagada et al. 2024) also used a larger CRF (RR 1.08, 95%CI: 1.06-1.09) (Chen and Hoek 2020) in a sensitivity analysis.

   Meta-analytic $PM_{2.5}$-mortality CRFs have been reported by WHO (RR 1.08, 95%CI: 1.06-1.09) per 10 μg/m^3 (Chen and Hoek 2020, WHO 2021), COMEAP (RR 1.08, 95%CI: 1.06-1.09) per 10 μg/m^3 (COMEAP 2022) and HEI (RR 1.06, 95%CI: 1.02-1.10) per 10 μg/m^3 (Health Effects Institute 2022, Boogaard et al. 2023). These CRFs are smaller than those used in the EPS.

   Two Australian studies investigated long-term exposure to $PM_{2.5}$ and all-cause mortality. The Sydney study (Hanigan et al. 2019) found a RR of 1.63, 95%CI: 0.84-3.18 per 10 μg/m^3 and a study conducted in Perth (Dirgawati et al. 2019) reported a RR of 1.10, 95%CI: 0. 93-1.31 per 10 μg/m^3. Both these CRFs, although similar or larger in magnitude to that used in the EPS, are statistically non-significant. Therefore, one should be cautious when applying a CRF from a study that is not conducted in Australia to the Australian population.

5. The CRF for NO_2 and mortality (RR 1.097, 95%CI: 1.074-1.120) used in the EPS is also larger than those previously published. For example, much lower meta-analytic NO_2-mortality CRFs are reported by WHO (RR 1.02, 95%CI: 1.01-1.04) (Huangfu and Atkinson 2020, WHO 2021), HEI (RR 1.04, 95%CI: 1.01-1.06) (Boogaard et al. 2023, Health Effects Institute 2022), and COMEAP (RR 1.023, 95%CI: 1.008-1.037 attributable to NO_2 and TRAP; RR between 1.006 and 1.013 attributable to NO_2 alone) (COMEAP 2022).

   Two Australian studies have investigated NO_2 and all-cause mortality. A Sydney study (Hanigan et al. 2019) found a RR of 1.06 95%CI: 0.99-1.14 per 10 μg/m^3 and a study conducted in Perth (Dirgawati et al. 2019) reported a RR of 1.06 95%CI: 1.00-1.12 per 10 μg/m^3. These CRFs are statistically non-significant and smaller than that used in the EPS.

In summary, the EPS used CRFs developed for the New Zealand population for the HAPINZ 3.0 study and scaled up the results of the HAPINZ 3.0 study to the Australian population. The HAPINZ 3.0 CRFs are higher than CRFs reported by the HEI (Health Effects Institute 2022) and WHO (WHO 2021).

Ideally, HIAs in Australian populations should use CRFs derived from large, well-conducted, multi-city Australian studies. If these are not available, then the CRFs should be selected from internationally conducted systematic reviews and meta-analyses.

At the very least, sensitivity analyses should be conducted using internationally accepted CRFs to demonstrate the upper and lower ranges of the disease burden, in this case, mortality, hospitalisations and asthma prevalence.

The EPS has not used CRFs from Australian studies or systematic reviews and meta-analyses, nor presented results from sensitivity analyses to demonstrate the size of the uncertainty in the estimates associated with their choice of CRFs.

HEI reports on traffic-related air pollution and health effects

Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects (Special Report 17), 2010

This Special Report 17 from the HEI summarises and synthesises information linking emissions from, exposures to, and health effects of traffic sources (i.e., motor vehicles) (Health Effects Institute 2010).

The term ‘traffic-related exposure’ is used in this HEI report to refer to exposure to primary emissions from motor vehicles, not to the more broadly dispersed secondary pollutants, e.g., ozone (O_3), which is derived from motor vehicle emissions. The report focuses on scenarios with a high proportion of motor vehicles and people, i.e., urban settings and residences near busy roadways.

Two broad categories of TRAP surrogates were used in this report to estimate traffic exposure:

- measured or modelled (e.g., geostatistical interpolation, land-use regression, dispersion models) concentrations of TRAP surrogates (e.g., NO_2, EC, BC, BS, PM); and,
- measures of traffic (such as proximity, or distance, of the residence to the nearest road and traffic volume within geographical buffers).

This report critically reviewed 167 published papers. Meta-analyses were not conducted. The main reason for not conducting meta-analyses was the wide variations in the exposures and populations being studied resulting in considerable heterogeneity (variability) among the findings. The heterogeneity raises questions about the validity and interpretation of the meta-analytic summaries. Therefore, the report relied on qualitative summaries of the available data.

The 2010 HEI report found there was sufficient evidence to infer a causal relationship between TRAP and exacerbation of asthma in children.

There was also suggestive evidence to infer a causal relationship with the onset of childhood asthma, non-asthma respiratory symptoms in adults, impaired lung function in children and adults, all-cause and cardiovascular mortality, and cardiovascular morbidity.

There was only limited evidence to infer a causal relationship for several other health outcomes as the data were either inadequate or insufficient to draw firm conclusions.

Systematic Review and Meta-analysis of Selected Health Effects of Long-Term Exposure to Traffic-Related Air Pollution (Special Report 23), 2022

This HEI Special Report 23 was published in 2022 and updated in April 2023 (Health Effects Institute 2022). The overall objective was to systematically evaluate the epidemiological evidence regarding the associations between long-term exposure to TRAP and selected health outcomes.

Three hundred and fifty-three studies were included in the review. Results were quantitatively combined in meta-analyses to evaluate the strength of the evidence, where appropriate. All studies (whether included in the meta-analysis or not) were used to evaluate the level of confidence in the evidence for an association between TRAP and health outcomes. This report has produced two publications (Boogaard et al. 2022, Boogaard et al. 2023).

Health outcomes for systematic review were selected based on whether they were causally related to TRAP or likely to be causally related to TRAP. The selected health outcomes included birth outcomes (e.g., term low birth weight), respiratory outcomes (e.g., asthma onset), cardiometabolic outcomes (e.g., ischaemic heart disease [IHD], diabetes) and all-cause and cause-specific (e.g., circulatory, respiratory) mortality.

Literature reviews were also conducted for neurodevelopmental outcomes in children and dementia-related outcomes and Parkinson disease in adults as these conditions are important emerging areas. For these outcomes no meta-analyses were conducted, there was no evaluation of the confidence in the quality of the body of evidence, and there was no formal risk of bias assessment of individual studies.

Epidemiological studies focusing on exposure contrasts at the local and neighbourhood scale offered the greatest potential in determining exposure to TRAP emissions, i.e., exposure to NO_2, EC (including related metrics such as BC, BS, and PM absorbance), carbon monoxide (CO), UFPs, and indirect traffic measures (distance to roads and traffic density. Studies that evaluated exposure to $PM_{2.5}$ and PM_{10} were only included if the exposure contrasts were likely due to variations in traffic emissions.

An extensive search of literature published between January 1980 and July 2019 was conducted. Effect estimates from single-pollutant models were selected as the effect estimates for the meta-analysis as the aim was to assess the TRAP mixture, not individual components of TRAP.

A random-effects meta-analysis was performed when at least three studies were available for a specific exposure–outcome pair. Risk of bias was assessed for all exposure–outcome associations included in the meta-analyses.

Meta-analyses of indirect traffic measures (proximity to roads, traffic density) were not conducted for several reasons, including the limited number of available studies and variability in the definitions of indirect traffic measures.

Overall, there was high or moderate-to-high level of confidence in the evidence for associations between long-term exposure to TRAP and all-cause, circulatory, IHD, and lung cancer mortality; asthma onset in both children and adults; and acute lower respiratory infection (ALRI) in children.

Confidence in the evidence for associations with TRAP was considered moderate, low, or very low for the other selected outcomes.

The complete set of results, with associated CRFs and confidence assessment, is presented in Figure 4.

FIGURE 4 FULL SET OF RESULTS FROM THE 2022 HEI REPORT

Executive Summary Table. Overall Confidence Assessment and Meta-analytical Summary Estimates of Associations Between Long-Term Exposure to the Most Common Traffic-Related Air Pollutants (NO_2, EC, $PM_{2.5}$) and Health Outcomes (NOTE: the individual pollutants are considered indicators of TRAP)

Health Outcome	Overall Confidence Assessment	NO_2 per 10-μg/m³		EC per 1-μg/m³		$PM_{2.5}$ per 5-μg/m³	
		N	Relative Risk (95% CI)	*N*	Relative Risk (95% CI)	*N*	Relative Risk (95% CI)
Birth Outcomes							
Term low birth weight	Moderate	12	1.01 (0.99–1.03)	5	1.01 (0.99–1.04)	7	1.11 (1.03–1.20)
Term birth weight	Low	8	−3.2 (−11.0 to 4.6)[a]	4	−2.6 (−6.1 to 0.9)[a]	6	−17.3 (−33.2 to −1.5)[a]
Small for gestational age	Moderate	11	1.00 (0.98–1.02)	3	1.02 (0.92–1.14)	4	1.09 (1.04–1.14)
Preterm birth	Low	14	1.00 (0.96–1.04)	5	1.02 (0.97–1.07)	4	0.99 (0.90–1.09)
Respiratory Outcomes—Children							
Asthma onset[b]	Moderate to high	12	1.05 (0.99–1.12)	5	1.11 (0.94–1.31)	5	1.33 (0.90–1.98)
Asthma ever[c]	Moderate	21	1.09 (1.01–1.18)	3	1.30 (0.56–3.04)	3	1.29 (0.58–2.87)
Active asthma[c]	Moderate	12	1.12 (1.02–1.23)	3	1.25 (0.98–1.59)	<3	NA
ALRI[b]	Moderate to high	11	1.09 (1.03–1.16)	4	1.30 (0.78–2.18)	<3	NA
Respiratory Outcomes—Adults							
Asthma onset[b]	Moderate to high	7	1.10 (1.01–1.21)	<3	NA	<3	NA
ALRI[b]	Very low to low	3	1.07 (0.71–1.61)	<3	NA	<3	NA
COPD[b]	Low	7	1.03 (0.94–1.13)	<3	NA	4	0.91 (0.62–1.36)
Cardiometabolic Outcomes							
IHD events[b]	Moderate	5	0.99 (0.94–1.05)	5	1.01 (0.99–1.03)	4	1.09 (0.86–1.39)
Coronary events[b]	Low	7	1.03 (0.95–1.11)	<3	NA	<3	NA
Stroke events[b]	Low to moderate	7	0.98 (0.92–1.05)	6	1.03 (0.98–1.09)	4	1.08 (0.89–1.32)
Diabetes[b]	Moderate	7	1.04 (0.96–1.13)	3	1.16 (0.57–2.36)	4	1.05 (0.96–1.15)
Diabetes[c]		7	1.09 (1.02–1.17)	<3	NA	3	1.08 (0.70–1.67)
Mortality							
All-cause	High	11	1.04 (1.01–1.06)	11	1.02 (1.00–1.04)	12	1.03 (1.01–1.05)
Circulatory	High	10	1.04 (1.00–1.09)	9	1.02 (1.00–1.04)	11	1.04 (1.01–1.08)
Respiratory	Moderate	8	1.05 (1.00–1.09)	8	1.01 (0.98–1.05)	7	1.03 (0.97–1.10)
Lung cancer	Moderate to high	5	1.04 (1.01–1.07)	3	1.02 (0.88–1.19)	6	1.06 (0.99–1.13)
IHD	High	6	1.05 (1.03–1.08)	6	1.05 (0.99–1.11)	7	1.07 (1.04–1.10)
Stroke	Low to moderate	6	1.01 (0.98–1.04)	<3	NA	3	1.04 (1.01–1.07)
COPD	Low	3	1.03 (1.00–1.05)	<3	NA	<3	NA

95% CI = 95% confidence interval; ALRI = acute lower respiratory infection; COPD = chronic obstructive pulmonary disease; IHD = ischemic heart disease; NA = not applicable.

[a] Mean difference in grams.

[b] Incidence.

[c] Prevalence.

Summary of COMEAP recommendations for the quantification of health effects associated with air pollutants, 2022

This COMEAP report (COMEAP 2022) provides recommendations for quantifying the health effects of air pollutants, e.g., CRFs for individual pollutant-outcome pairs, and general principles for conducting HIAs.

It also includes information on uncertainties relevant to the recommendations, e.g., limited evidence base, inconsistent association, uncertain underlying causality, and lack of specificity to the air pollutant. This information can be used to inform decisions regarding whether to include a pollutant-outcome pair in the main health impact assessment or in sensitivity analyses.

The relevant meta-analytical CRFs for long-term exposure to $PM_{2.5}$ and NO_2 and health outcomes from single-pollutant models are presented in Table 1.

TABLE 1 CONCENTRATION-RESPONSE FUNCTIONS FOR LONG-TERM EXPOSURE TO $PM_{2.5}$ AND NO_2, COMEAP 2022

Health outcome	$PM_{2.5}$ RR (95%CI) per 10 µg/m^3	NO_2 RR (95%CI) per 10 µg/m^3
All-cause mortality	1.08 (1.06, 1.09) (Chen and Hoek 2020)	1.023 (1.008, 1.037) (effects attributable to NO_2 and corresponding reductions in other TRAP) between 1.006 and 1.013 (effects attributable to NO_2 alone[1]; not possible to calculate CIs)
Ischaemic heart disease incidence	1.07 (0.99, 1.16) (COMEAP 2021)	n/a[2]
Cerebrovascular disease (stroke) incidence	1.11 (0.99, 1.25) (COMEAP 2021)	n/a
Cognitive decline and dementia	Recommendation for quantification not developed	n/a
Respiratory morbidity in children (respiratory symptoms [including bronchitic symptoms in asthmatic children], indices of lung function, asthma)	n/a	Recommendation against quantification
Asthma	Recommendation for quantification not developed	Recommendation for quantification not developed

[1]The CRF for NO_2 effects alone is lower than the CRF for the combined effects of NO_2 and TRAP. This decision was reached by expert judgement. [2]Not available.

WHO Global Air Quality Guidelines, 2021

The WHO Global Air Quality Guidelines (AQG) published in 2021 (WHO 2021) updated the WHO AQGs published in 2006 (WHO 2006). The updated global guidelines aim to provide quantitative health-based recommendations to governments to inform legislation and policy for air pollution control. Exceedances of the AQGs are expected to increase the risk to health of populations. Guidelines are provided for $PM_{2.5}$, PM_{10}, O_3, NO_2, SO_2 and CO linked to averaging periods.

This WHO report also provides interim targets to guide reduction efforts to meet the AQGs for countries that substantially exceed the AQGs. It also provides good practice guidelines to manage some other types of PM (e.g., BC, EC, UFPs, and particles originating from sand

and dust storms) for which there is insufficient information to propose AQGs but the available evidence indicates risk to human health.

The AQGs were formulated by following a rigorous process. The steps in the development of the AQG levels included:

- a determination of the scope of the guidelines and formulation of systematic review questions;
- a systematic review of the evidence and meta-analyses of quantitative effect estimates to inform updating of the AQG levels;
- an assessment of the level of certainty of the bodies of evidence resulting from systematic reviews for the air pollutants; and,
- the identification of AQG levels, that is, the lowest levels of exposure for which there is evidence of adverse health effects.

The scientific understanding of the health effects of air pollution has increased since the 2005 WHO report:

1. More long-term exposure studies have been reported, especially from Asia and Oceania. These studies have found relationships between air pollutants and ill-health that are qualitatively similar to those in high-income countries. However, the CRFs are sometimes quantitatively different, with steeper relationships at low concentrations compared to high concentrations of air pollutants, i.e., the effects are larger at lower levels of air pollution and the magnitude of the effects decrease as air pollution levels increase.

2. Air pollution has been implicated in the initiation or exacerbation of several health conditions that have not been previously investigated. These include, among others, asthma, diabetes, reproductive outcomes, and several neurocognitive outcomes.

3. Many studies have tried identifying the sources and/or physicochemical characteristics of PM that contribute most to toxicity.

4. Large collaborative studies of long-term effects have been conducted, e.g., the European Study of Cohorts for Air Pollution Effects (ESCAPE), which includes data from 36 different cohorts; the Global Exposure Mortality Model (GEMM), which includes data from 41 cohorts from 16 countries worldwide; and studies on the long-term health effects of exposure to low levels of air pollution in Europe, Canada and the United States.

For long-term exposure to both $PM_{2.5}$ and NO_2, mortality was the only outcome considered for systematic review and meta-analysis. The systematic review and meta-analysis were published (Chen and Hoek 2020, Huangfu and Atkinson 2020).

The relevant meta-analytical single-pollutant model CRFs for long-term exposure to $PM_{2.5}$ and NO_2 and mortality outcomes are presented in Table 2.

TABLE 2 CONCENTRATION-RESPONSE FUNCTIONS FOR LONG-TERM EXPOSURE TO $PM_{2.5}$ AND NO_2 AND MORTALITY, WHO 2021

Mortality	$PM_{2.5}$* RR (95%CI) per 10 µg/m³	NO_2 per 10 µg/m³** RR (95%CI) per 10 µg/m³
Natural cause	1.08 (1.06, 1.09)	1.02 (1.01-1.04)
Circulatory	1.11 (1.09, 1.14)	n/a***
Ischaemic heart disease	1.16 (1.10, 1.21)	n/a
Stroke	1.11 (1.04, 1.18)	n/a
Respiratory	1.10 (1.03, 1.18)	1.03 (1.00-1.05)
Chronic obstructive pulmonary disease	1.11 (1.05, 1.17)	1.03 (1.01-1.04)
Acute lower respiratory infection	1.16 (1.01, 1.34)	1.06 (1.02-1.10)
Lung cancer	1.12 (1.07, 1.16)	n/a

*High certainty in the evidence for associations between all $PM_{2.5}$ and all mortality outcomes except for respiratory mortality where the evidence was of moderate certainty. **Certainty in the evidence for associations with mortality was rated low to moderate for all NO_2 and mortality outcomes except for chronic obstructive pulmonary disease mortality where the evidence was of high certainty. ***Not available.

HRAPIE project: recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide, 2013

Three reports were published by the WHO Regional Office for Europe in 2013 to provide the European Commission with evidence-based advice on the health aspects of air pollution. The advice was based on a review of the latest scientific evidence on the health effects of air pollutants conducted by a panel of invited experts from eminent institutions worldwide.

The three reports were:

- The "HRAPIE project: recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide" (WHO 2013a);
- "Review of evidence on health aspects of air pollution – REVIHAAP" (WHO 2013c); and,
- "HRAPIE project: New emerging risks to health from air pollution – results from the survey of experts" (WHO 2013b).

The HRAPIE project that recommends CRFs for cost–benefit analysis of PM, O_3 and NO_2 (WHO 2013a) is now just over 10 years old. However, the recommended CRFs continue to be used in quantifying the burden of disease attributable to air pollution.

For long-term exposure, one $PM_{2.5}$-outcome CRF ($PM_{2.5}$-mortality) and two NO_2-outcome CRFs (NO_2-mortality and NO_2-prevalence of bronchitic symptoms in asthmatic children) are recommended.

The relevant CRFs for long-term exposure to $PM_{2.5}$ and NO_2 are presented later in this report (Tables 9 and 10) for comparison with more recent recommended CRFs.

What has changed in the past 10 to 15 years

This section summarises changes in the air pollution and health scientific literature over the past 10 to 15 years.

Number of air pollution studies

In this section, the 2010 HEI study and the 2022 HEI study on TRAP and health effects will be compared. The 2006 WHO AQG report and the 2021 WHO AQG report on ambient air pollution and health effects will also be compared.

Comparing HEI reports

The 2010 HEI report reviewed 167 scientific papers whilst the 2022 HEI report reviewed 353 scientific papers.

Table 3 presents the number of studies included in the systematic reviews, narrative reviews and meta-analyses in the two HEI reports. Meta-analyses were not conducted for the 2010 HEI report. Meta-analyses were not conducted for indirect measures of TRAP in the 2022 HEI report. Where meta-analyses were not performed, evidence was presented as narrative reviews. Not all studies included in the systematic reviews were subsequently included in the meta-analyses.

Across all five broad groups of outcomes, considerably more studies were reviewed in the 2022 HEI report than in the 2010 HEI report, reflecting the increase in published air pollution research. The large increase in the number of studies on NO_2/NO_x in the HEI 2022 report compared to the 2010 HEI report is noteworthy.

TABLE 3 NUMBER OF STUDIES IN SYSTEMATIC REVIEWS, NARRATIVE REVIEWS AND META-ANALYSES, HEI 2010 AND HEI 2022 REPORTS

Health outcome	Year HEI report published	Number of studies in systematic review	Number of studies in meta-analyses/narrative review[1]			
			NO_2/NO_x	$PM_{2.5}$	EC	Distance to main road/traffic density
Birth Outcomes	2010	4	2	3	0	3
	2022	86	43	5	10	13
Respiratory outcomes - children	2010	32	14	0	0	23
	2022	118	53	8	11	42
Respiratory outcomes - adults	2010	3	1	0	0	3
	2022	50	16	4	<3	7
Cardiometabolic outcomes	2010	4[2]	1	0	0	3
	2022	57	39	10	10	18
Mortality	2010	9	1	0	1 (BS)	8
	2022	48	22	16	12	14

[1]Meta-analyses were not conducted for the HEI 2010 report. Meta-analyses were not conducted for indirect measures of TRAP in the HEI 2022 report. Narrative reviews were conducted when meta-analyses were not performed. Not all papers in the systematic reviews were included in meta-analyses. [2]None for diabetes.

Table 4 presents the number of studies included in the systematic reviews, narrative reviews, and meta-analyses in the two HEI reports by sub-categories of health outcomes. Compared to the 2010 HEI report, the 2022 HEI report reviewed more studies and on a

greater range of health outcomes for each of the TRAP measures. The 2010 HEI report reviewed none or very few studies on $PM_{2.5}$, EC, and distance/traffic density measures for most of the outcome sub-categories. Many more pollutant-outcome pairs were reviewed in the 2022 HEI 2022 report compared to the 2010 HEI report.

Unlike the 2022 HEI report, the 2010 HEI report did not consider acute lower respiratory infection (pneumonia, acute bronchitis and bronchiolitis) for systematic review. The 2020 HEI report did conduct systematic reviews for respiratory symptoms (e.g., morning and night cough, phlegm) other than wheeze. These studies are not reported in Table 4.

TABLE 4 NUMBER OF STUDIES INCLUDED IN THE NARRATIVE REVIEWS OR META-ANALYSIS IN THE 2010 HEI AND 2022 HEI REPORTS FOR SUB-CATEGORIES OF HEALTH OUTCOMES

		2010 HEI report[1]				2022 HEI report			
		NO_2	$PM_{2.5}$	EC	Distance/traffic density measures	NO_2	$PM_{2.5}$	EC	Distance/traffic density measures
Health Outcome	**Sub-category**								
Birth Outcomes	Term low birth weight	1	1	0	1	12	7	5	13
	Term birth weight	0	0	0	2	8	6	4	4
	Small for gestational age	1	1	0	1	11	4	3	11
	Preterm birth	0	0	0	2	4	4	5	17
Respiratory Outcomes—Children	Asthma onset[a]	6	0	0	4	12	5	5	11
	Asthma ever[b]	4	0	0	8	21	3	3	23
	Active asthma[b,2]	10	0	0	12	12	<3	3	19
	Acute lower respiratory infection[a,3]	0	0	0	0	11	4	<3	10
Respiratory Outcomes—Adults	Asthma onset[a]	0	0	0	1	7	<3	<3	6
	Acute lower respiratory infection[a,3]	0	0	0	0	3	<3	<3	1
	Chronic obstructive pulmonary disease[a]	0	0	0	2	7	4	<3	4
Cardio-metabolic outcomes	Ischaemic heart disease[a]	1	0	0	2	5	4	5 M	4
	Coronary heart disease[a]	0	0	0	0	7	<3	<3	8
	Stroke[a]	0	0	0	0	7	4	6	6
	Diabetes[a]	0	0	0	0	7	4	3	5
	Diabetes[b]	0	0	0	0	7	3	<3	3

		2010 HEI report[1]				2022 HEI report			
		NO_2	$PM_{2.5}$	EC	Distance/traffic density measures	NO_2	$PM_{2.5}$	EC	Distance/traffic density measures
Mortality	All-cause	0	0	1 (BS)	6	11	12	11	11
	Circulatory	2	0	1 (BS)	6	10	11	9	11
	Respiratory	0	0	0	0	8	7	8	5
	Lung cancer	0	0	0	0	5	6	3	5
	Ischaemic heart disease	0	0	0	0	6	7	6	n/a[4]
	Stroke	0	0	0	0	6	3	<3	n/a[4]
	Chronic obstructive pulmonary disease	0	0	0	0	3	<3	<3	n/a[5]

[1]Meta-analyses were not conducted for the HEI 2010 report. Meta-analyses were not conducted for indirect measures of TRAP in the HEI 2022 report. Narrative reviews were conducted when meta-analyses were not performed. Not all papers in the systematic reviews were included in meta-analyses. [2]Only wheeze used as a marker of active asthma in 2020 HEI report. [3]Not reported in 2020 HEI report. [4]Included with circulatory mortality. [5]Included with respiratory mortality.
[a]Incidence [b]Prevalence

Comparing WHO AQG reports

The 2006 and 2021 WHO AQG reports (WHO 2006, WHO 2021) relied on air pollution and mortality studies to provide evidence for guideline setting.

In the 2005 WHO AQG report, meta-analyses were not performed. The recommended CRFs for long-term exposure to $PM_{2.5}$ and all-cause mortality and cardiorespiratory mortality were derived from the 2002 Pope study (Pope Iii et al. 2002). No CRFs were recommended for NO_2 or for other sub-categories of mortality associated with $PM_{2.5}$.

On the other hand, meta-analyses were conducted for all-cause mortality and seven sub-categories of mortality for $PM_{2.5}$ (Chen and Hoek 2020), and for all-cause mortality and three respiratory sub-categories of mortality for NO_2 (Huangfu and Atkinson 2020) for the 2021 WHO AQG report (Table 2).

More studies and health outcomes were reviewed in the 2021 WHO AQG report compared to the 2006 WHO AQG report.

Determination of causality

The causality determination of a number of pollutant-outcome pairs has been upgraded as the evidence base has expanded.

Comparing HEI reports

The 2010 HEI report and the 2022 HEI report used different methodologies to assess evidence for causality inference and associations between TRAP and health outcomes.

The 2010 HEI report evaluated the strength of the evidence to infer causality, ranked from A (Sufficient evidence to infer a causal association) to D (Evidence is suggestive of no causal association) (Table 5).

In contrast, the 2022 HEI report evaluated the confidence in the evidence for associations between TRAP and health outcomes, ranked from High (High confidence in the association

between exposure and the outcome) to Very Low (Very low confidence in the association between exposure and the outcome) (Table 5).

Although it is not possible to make direct comparisons between the two methods of assessing the strength of the evidence, it can be surmised that the two ranking systems are equivalent, i.e., 2010 HEI rank 'A' is equivalent to 2022 HEI rank 'High (H)'.

The 2022 HEI report makes such direct comparisons, e.g., on page 491:

> *"The overall judgment of high confidence in an association between TRAP and all-cause, circulatory, IHD, and lung cancer mortality represents a significant increase in confidence compared with that reported in the 2010 HEI Traffic Review (HEI 2010). Although the methodologies of the two reviews differed, the Panel judged that the main reason for the increased confidence is the larger number of studies published since the 2010 review."*

As the HEI reports were focused on TRAP and health effects, traffic-specific criteria were used in both reports to ensure that the health outcomes could be attributed to TRAP.

TABLE 5 DESCRIPTORS OF STRENGTH OF THE EVIDENCE FOR CAUSAL INFERENCE (2010 HEI REPORT) AND CONFIDENCE IN THE EVIDENCE FOR AN ASSOCIATION BETWEEN EXPOSURE AND OUTCOME (2022 HEI REPORT)

2010 HEI report	Strength of the evidence for causal inference
	A: *Sufficient evidence to infer a causal association*
	B: *Suggestive but not sufficient evidence to infer a causal association*
	C: *Inadequate and insufficient evidence to infer the presence or absence of a causal association*
	D: *Evidence is suggestive of no causal association*
2022 HEI report	**Confidence in the evidence for an association between exposure and the outcome**
	High (H): *High confidence in the association between exposure and the outcome.*
	Moderate (M): *Moderate confidence in the association between exposure and the outcome.*
	Low (L): *Low confidence in the association between exposure and the outcome.*
	Very Low (VL): *Very low confidence in the association between exposure and the outcome.*

The strength of the evidence to infer causality for TRAP and health outcomes from the 2010 HEI report and the confidence in the evidence for associations between TRAP and health outcomes from the 2022 HEI report are presented in Table 6.

In the 2022 HEI report compared to the 2010 HEI report, there was an increase in confidence in the evidence for an association with TRAP for five health outcomes.

Eleven health outcomes were not considered in the 2010 HEI report compared to the 2022 HEI report. There was moderate or high confidence in the evidence for an association with TRAP for six of these 11 health outcomes in the 2022 HEI report.

Three health outcomes were downgraded (decrease in the level of confidence in the evidence for an association with TRAP) in the HEI 2022 report (Active asthma and Asthma ever in children and Ischaemic heart disease in adults).

TABLE 6 STRENGTH OF THE EVIDENCE TO INFER CAUSAL ASSOCIATION BETWEEN TRAP AND HEALTH OUTCOMES (2010 HEI REPORT) OR CONFIDENCE IN THE EVIDENCE FOR ASSOCIATION BETWEEN TRAP AND HEALTH OUTCOMES (2022 HEI REPORT)

		2010 HEI report	2022 HEI report	Change in evidence from 2010 HEI report to 2022 HEI report[2]
		Strength of evidence to infer causal association[1]	Confidence in the evidence for an association[1]	
Health Outcome	**Sub-category**			
Birth Outcomes	Term low birth weight	C	M	↑
	Term birth weight	C	L	↔
	Small for gestational age	C	M	↑
	Preterm birth	C	L	↔
Respiratory Outcomes—Children	Asthma onset[a]	A/B	M/H	↔
	Asthma ever[b]	A/B	M	↓
	Active asthma[b]	A	M	↓
	Acute lower respiratory infection[a]	n/a[3]	M/H	↑
Respiratory Outcomes—Adults	Asthma onset[a]	C	M/H	↑
	Acute lower respiratory infection[a]	n/a	VL/L	↑
	Chronic obstructive pulmonary disease[a]	C	L	↔
Cardiometabolic outcomes	Ischemic heart disease[a]	A/B	M	↓
	Coronary heart disease[a]	n/a	L	↑
	Stroke[a]	n/a	L/M	↑
	Diabetes[a]	n/a	M	↑
	Diabetes[b]	n/a	M	↑

			2010 HEI report Strength of evidence to infer causal association[1]	2022 HEI report Confidence in the evidence for an association[1]	Change in evidence from 2010 HEI report to 2022 HEI report[2]
Mortality	-	All-cause	B	H	↑
		Circulatory	B	H	↑
		Respiratory	n/a	M/H	↑
		Lung cancer	n/a	M	↑
		Ischemic heart disease	n/a	H	↑
		Stroke	n/a	L/M	↑
		Chronic obstructive pulmonary disease	n/a	L	↑

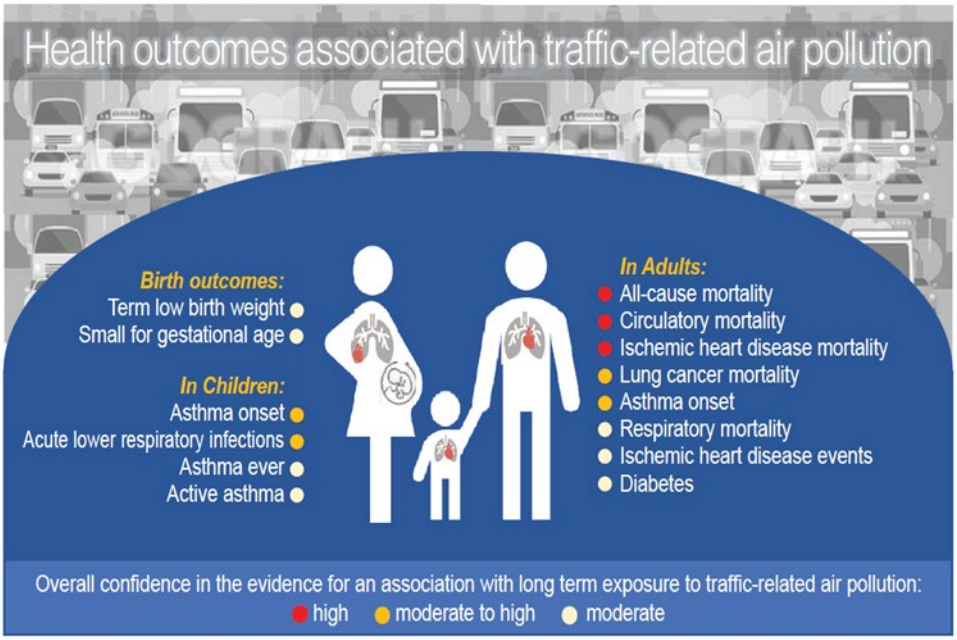
[1]See Table 5 for descriptors of the strength of the evidence to infer causality (2010 HEI report) and confidence in the evidence for associations between TRAP and health outcomes (2022 HEI report).

[2] ↑ = Increase in evidence ↓ = Decrease in evidence ⟷ = No change in evidence

[3]Not available [a]Incidence [b]Prevalence

Health outcomes for which the 2022 HEI report deemed to have moderate to high confidence in the evidence for associations with TRAP are depicted in Figure 5.

FIGURE 5 HEALTH OUTCOMES WITH MODERATE TO HIGH CONFIDENCE IN THE EVIDENCE FOR AN ASSOCIATION WITH TRAFFIC-RELATED AIR POLLUTION, 2022 HEI REPORT



Executive Summary Figure. Overall confidence in the evidence for an association between long-term exposure to TRAP and selected health outcomes. Health outcomes for which the overall confidence in the evidence was low to moderate, low, or very low are not in the figure.

Comparing US EPA reports

The most recent causality determinations from the US EPA Integrated Science Assessments (ISAs) for $PM_{2.5}$ (US EPA 2019, US EPA 2022) and NO_2 (US EPA 2016) are presented in Table 7 (adapted from Forastiere et al. (Forastiere et al. 2024)). $PM_{2.5}$ adverse health effects are more often causal or likely to be causal compared to NO_2 adverse health effects.

TABLE 7 CAUSALITY DETERMINATION[1] OF LONG-TERM EXPOSURE TO $PM_{2.5}$ AND NO_2 FROM US EPA INTEGRATED SCIENCE ASSESSMENTS

Health outcome	$PM_{2.5}$	NO_2
Reproductive and developmental effects	Suggestive of, but not sufficient to infer causality	Suggestive of, but not sufficient to infer causality
Respiratory effects	Likely to be causal	Likely to be causal
Cardiovascular diseases	Causal	Suggestive of, but not sufficient to infer causality
Metabolic effects	Suggestive of, but not sufficient to infer causality	Suggestive of, but not sufficient to infer causality
Neurological effects	Likely to be causal	n/a[2]
Lung cancer	Likely to be causal	Suggestive of, but not sufficient to infer causality
Mortality	Causal	Suggestive of, but not sufficient to infer causality

[1]The US EPA determines causality as follows (US EPA 2016): 1. Causal relationship; 2. Likely to be a causal relationship; 3. Suggestive of, but not sufficient to infer, a causal relationship; 4. Inadequate to infer a causal relationship; 5. Not likely to be a causal relationship. [2]Not available.

Table 8 presents change in causality determination (**in bold text**) for $PM_{2.5}$ from the 2009 ISA (US EPA 2009) to the 2019 ISA (US EPA 2019) (adapted from (US EPA 2024)). Since the 2009 ISA determination, the level of causality of three broad groups of health outcomes (cancer, nervous system effects and metabolic effects) have been upgraded.

TABLE 8 CHANGES IN CAUSALITY DETERMINATION[1] FOR LONG-TERM $PM_{2.5}$-RELATED HEALTH EFFECTS, 2009 AND 2019 US EPA INTEGRATED SCIENCE ASSESSMENTS

Health Outcome	2009 US EPA Integrated Science Assessment	2019 US EPA Integrated Science Assessment
Mortality	Causal	Causal
Cardiovascular effects	Causal	Causal
Respiratory effects	Likely to be causal	Likely to be causal
Nervous system effects	**None[2]**	**Likely to be causal**
Cancer	**Suggestive of, but not sufficient to infer causality**	**Likely to be causal**
Metabolic effects	**None**	**Suggestive of, but not sufficient to infer causality**
Male and female reproduction and fertility	Suggestive of, but not sufficient to infer causality	Suggestive of, but not sufficient to infer causality
Pregnancy and birth outcomes	Suggestive of, but not sufficient to infer causality	Suggestive of, but not sufficient to infer causality

[1]The US EPA ranks causality as follows (US EPA 2016): 1. Causal relationship; 2. Likely to be a causal relationship; 3. Suggestive of, but not sufficient to infer, a causal relationship; 4. Inadequate to infer a causal relationship; and, 5. Not likely to be a causal relationship. [2]Changes in causality are **bolded**.

Number of concentration-response functions

In the past 10 to 15 years, the increase in air pollution and health research and the expanding evidence base have resulted in many more health outcomes that are eligible for inclusion in an HIA.

Tables 9 and 10, respectively, present CRFs for long-term exposure to $PM_{2.5}$ and NO_2.

The CRFs have been collated from two recent reports – the 2022 HEI report (Health Effects Institute 2022) and the 2022 COMEAP report (COMEAP 2022) - and a review of systematic reviews by Forastiere et al. (Forastiere et al. 2024).

CRFs from the 2013 WHO HRAPIE report (WHO 2013a) are presented as a baseline to highlight changes in pollutant-outcome pairs eligible for inclusion in an HIA or changes in the magnitude of the CRFs.

Compared to the 2013 WHO HRAPIE report, additional CRFs for $PM_{2.5}$-outcome pairs are now available for sub-categories of mortality (n=5), metabolic effects (n=5), respiratory effects (n=4), neurological effects (n=2) and birth outcomes (n=4) (Table 9). Some of the CRFs are not statistically significant, e.g., respiratory mortality in the 2022 HEI report.

TABLE 9 CONCENTRATION-RESPONSE FUNCTIONS FOR LONG-TERM EXPOSURE TO $PM_{2.5}$ AND HEALTH OUTCOMES

	2013 WHO HRAPIE report RR (95%CI) per 10 µg/m³	2022 COMEAP report RR (95%CI) per 10 µg/m³	2022 HEI report RR (95%CI) per 10 µg/m³	Forastiere 2024[1] RR (95%CI) per 10 µg/m³
Birth outcomes				
Term low birth weight	n/a[2]	n/a	1.23 (1.06-1.44)	n/a
Term birth weight (in grams)	n/a	n/a	-34.6[3] (-66.3 to -2.9)	n/a
Small for gestational age	n/a	n/a	1.19 (1.08-1.30)	n/a
Preterm birth	n/a	n/a	0.98 (0.81-1.19)	n/a
Respiratory outcomes - children				
Asthma onset[a]	n/a	No CRF recommended	1.77 (0.80-3.89)	1.34 (1.10, 1.63) (Khreis et al. 2017)
Asthma ever[b]	n/a	n/a	1.66 (0.34-8.23)	n/a
Active asthma[b]	n/a	n/a	n/a	n/a
Acute lower respiratory infection[a]	n/a	n/a	n/a	n/a

	2013 WHO HRAPIE report RR (95%CI) per 10 µg/m^3	2022 COMEAP report RR (95%CI) per 10 µg/m^3	2022 HEI report RR (95%CI) per 10 µg/m^3	Forastiere 2024[1] RR (95%CI) per 10 µg/m^3
Respiratory outcomes - adults				
Asthma onset[a]	n/a	n/a	n/a	n/a
Acute lower respiratory infection[a]	n/a	n/a	n/a	n/a
Chronic obstructive pulmonary disease[a]	n/a	n/a	0.83 (0.38-1.82)	1.18 (1.13, 1.23) (Park et al. 2021)
Bronchitis[b]	n/a	n/a	n/a	n/a
Lung cancer[a]	n/a	n/a	n/a	1.16 (1.10, 1.23) (Yu et al. 2021)
Neurological outcomes				
Cognitive decline and dementia[a]	n/a	No CRF recommended	n/a	1.46 (1.20, 1.78) (Cheng et al. 2022)
Autism spectrum disorders[a]	n/a	n/a	n/a	1.66 (1.23, 2.25) (Lin et al. 2022)
Cardiometabolic outcomes				
Ischaemic heart disease[a]	n/a	1.07 (0.99-1.16)	1.18 (0.74-1.92)	1.13 (1.05, 1.22) (Zhu et al. 2021)
Coronary heart disease[a]	n/a	n/a	n/a	n/a
Cerebrovascular disease/stroke[a]	n/a	1.11 (0.99-1.25)	1.17 (0.79-1.73)	1.16 (1.12, 1.20) (Yuan et al. 2019)
Diabetes[a]	n/a	n/a	1.10 (0.92-1.32)	1.10 (1.03, 1.18) (Yang et al. 2020)
Diabetes[b]	n/a	n/a	1.17 (0.49-2.78)	1.08 (1.04-1.12)[4] (Yang et al. 2020)
Hypertension[a]	n/a	n/a	n/a	1.17 (1.05, 1.30) (Qin et al. 2021)

	2013 WHO HRAPIE report RR (95%CI) per 10 µg/m³	2022 COMEAP report RR (95%CI) per 10 µg/m³	2022 HEI report RR (95%CI) per 10 µg/m³	Forastiere 2024[1] RR (95%CI) per 10 µg/m³
Mortality				
All-cause mortality	1.062 (1.040–1.083) (Hoek et al. 2013)	1.08 (1.06-1.09) (Chen and Hoek 2020)	1.06 (1.02-1.10)	n/a
Circulatory	n/a	n/a	1.08 (1.01-1.16)	n/a
Respiratory	n/a	n/a	1.06 (0.94-1.20)	n/a
Lung cancer	n/a	n/a	1.12 (0.98-1.28)	n/a
Ischaemic heart disease	n/a	n/a	1.14 (1.00-1.31)	n/a
Stroke	n/a	n/a	1.08 (1.02-1.15)	n/a
Chronic obstructive pulmonary disease	n/a	n/a	n/a	n/a
Infant (0-12 months of age)	1.04 (1.02, 1.07) (Woodruff, Grillo and Schoendorf 1997)	n/a	n/a	n/a

[1]Forestiere et al. only reported incident morbidity outcomes that the US EPA determined were either causally or likely to be causally related to air pollution. [2]Not available. [3]Mean difference in grams. [4]CRF for diabetes prevalence was not reported by Forestiere et al. (2024). The CRF is from Yang et al. (2020). [a]Incidence [b]Prevalence

Compared to the 2013 HRAPIE report, additional CRFs for NO_2-outcome pairs are now available for sub-categories of mortality (n=6), metabolic effects (n=5), respiratory effects (n=7), and birth outcomes (n=4) (Table 10). Some of the CRFs are not statistically significant, e.g., stroke mortality in the 2022 HEI report.

TABLE 10 CONCENTRATION-RESPONSE FUNCTIONS FOR LONG-TERM EXPOSURE TO NO_2 AND HEALTH OUTCOMES

	2013 WHO HRAPIE report RR (95%CI) per 10 µg/m³	2022 COMEAP report RR (95%CI) per 10 µg/m³	2022 HEI report RR (95%CI) per 10 µg/m³	Forastiere 2024[1] RR (95%CI) per 10 µg/m³
Birth outcomes				
Term low birth weight	n/a[2]	n/a	1.01 (0.96-1.03)	n/a
Term birth weight (in grams)	n/a	n/a	-3.2[3] (-11.0 to 4.6)	n/a
Small for gestational age	n/a	n/a	1.00 (0.96-1.02)	n/a
Preterm birth	n/a	n/a	1.00 (0.96-1.04)	n/a

	2013 WHO HRAPIE report RR (95%CI) per 10 µg/m^3	2022 COMEAP report RR (95%CI) per 10 µg/m^3	2022 HEI report RR (95%CI) per 10 µg/m^3	Forastiere 2024[1] RR (95%CI) per 10 µg/m^3
Respiratory outcomes - children				
Asthma onset[a]	n/a	No CRF recommended	1.05 (0.96-1.12)	1.10 (1.05, 1.18) (Khreis et al. 2017)
Asthma ever[b]	n/a	n/a	1.09 (1.01-1.18)	n/a
Active asthma[b]	n/a	n/a	1.12 (1.02-1.23)	n/a
Acute lower respiratory infection[a]	n/a	No CRF recommended[4]	1.09 (1.03-1.16)	1.09 (1.03-1.16) (Health Effects Institute 2022)
Bronchitis[b]	1.023 (0.875–1.732) (McConnell et al. 2003)	n/a	n/a	n/a
Respiratory outcomes - adults				
Asthma onset[a]	n/a	No CRF recommended	1.10 (1.01-1.21)	1.10 (1.01, 1.21) (Health Effects Institute 2022)
Acute lower respiratory infection[a]	n/a	n/a	1.07 (0.71-1.81)	n/a
Chronic obstructive pulmonary disease[a]	n/a	n/a	1.03 (0.94-1.13)	n/a
Bronchitis[b]	n/a	n/a	n/a	n/a
Lung cancer[a]	n/a	n/a	n/a	n/a
Neurological outcomes				
Cognitive decline and dementia[a]	n/a	n/a	n/a	n/a
Autism spectrum disorders[a]	n/a	n/a	n/a	n/a
Cardiometabolic outcomes				
Ischaemic heart disease[a]	n/a	1.07 (0.99-1.16)	0.99 (0.94-1.05)	n/a
Coronary heart disease[a]	n/a	n/a	1.03 (0.95-1.11)	n/a
Cerebrovascular disease/stroke[a]	n/a	1.11 (0.99-1.25)	0.98 (0.93-1.05)	n/a
Diabetes[a]	n/a	n/a	1.04 (0.96-1.13)	n/a
Diabetes[b]	n/a	n/a	1.09 (0.92-1.17)	n/a
Hypertension[a]	n/a	n/a	n/a	n/a

	2013 WHO HRAPIE report RR (95%CI) per 10 µg/m^3	2022 COMEAP report RR (95%CI) per 10 µg/m^3	2022 HEI report RR (95%CI) per 10 µg/m^3	Forastiere 2024[1] RR (95%CI) per 10 µg/m^3
Mortality				
All-cause mortality	1.055 (1.031–1.080) (Hoek et al. 2013)	1.023 (1.008-1.037) (for NO_2 and TRAP combined[5]) between 1.006 and 1.013 (for NO_2 alone[6])	1.04 (1.01-1.06)	n/a
Circulatory	n/a	n/a	1.04 (1.00-1.09)	n/a
Respiratory	n/a	n/a	1.05 (1.00-1.06)	n/a
Lung cancer	n/a	n/a	1.04 (1.01-1.07)	n/a
Ischaemic heart disease	n/a	n/a	1.05 (1.03-1.08)	n/a
Stroke	n/a	n/a	1.01 (0.98-1.04)	n/a
Chronic obstructive pulmonary disease	n/a	n/a	1.03 (1.00-1.05)	n/a
Infant (0-12 months of age)	n/a	n/a	n/a	n/a

[1]Forestiere et al. only considered incident morbidity outcomes that the US EPA reported were either causally or likely to be causally related to air pollution. [2]Not available. [3]Mean difference in grams. [4]Unable to disentangle NO_2 effects from the effects of other air pollutants. [5]Effects attributable to reductions in NO_2 and corresponding reductions in other TRAP. [6]Effects attributable to NO_2 reductions alone, the CRF reduced using expert judgement, unable to calculate confidence intervals.
[a]Incidence [b]Prevalence

Implications for past and future motorway tunnel health impact assessments

Six Environmental Impact Statements have been conducted for road tunnels in NSW in the past decade. These are for the following road tunnels: NorthConnex, M4East, M4-M5 Link, New M5, M6 Stage 1 and Western Harbour.

All six Environmental Impact Statements contain an HIA to quantify potential adverse health impacts from the construction and operation of the road tunnels. All six HIAs quantified impacts of $PM_{2.5}$ and NO_2. These six HIAs were published within an eight-year period, from 2013 to 2020. Five of the HIAs were conducted by Environmental Risk Sciences (Environmental Risk Sciences 2014, Environmental Risk Sciences 2015a, Environmental Risk Sciences 2015b, Environmental Risk Sciences 2017, Environmental Risk Sciences 2020) and one by AECOM (AECOM 2018).

For $PM_{2.5}$, all six HIAs evaluated the same three primary health outcomes (all-cause mortality for long-term exposure, and cardiovascular and respiratory hospitalisations for short-term exposures) (see Table 11). The HIAs also evaluated four to five secondary health outcomes (long-term exposure and cardiopulmonary mortality; and, short-term exposure and all-cause mortality, cardiovascular mortality, respiratory mortality, emergency department visits for asthma).

For NO_2, five of the six HIAs evaluated the same three health outcomes (all-cause mortality, respiratory mortality, and emergency department visits for asthma). These three outcomes were related to short-term exposures (Table 11). The NorthConnex HIA did not conduct quantitative assessments for exposure to NO_2.

All six HIAs used the same

All six HIAs used the same CRFs to quantify the $PM_{2.5}$ health impacts. Only one long-term exposure health outcome (all-cause mortality) was considered for the $PM_{2.5}$ primary analysis and the CRF was derived from Krewski et al. 2009 (Krewski et al. 2009). CRFs for short-term exposure to $PM_{2.5}$ and cardiovascular and respiratory hospitalisations were derived from Bell et al. 2008 and Bell 2012 (Bell 2012, Bell et al. 2008).

For NO_2, no health outcomes associated with long-term exposure were considered for the primary analysis. All three health outcomes quantified were related to short-term exposure. All six HIAs used the same CRFs to quantify the health impacts. The CRFs for all-cause mortality and respiratory mortality were derived from reports by the EPHC and Golder Associates (EPHC 2010, Golder Associates 2013). The CRF for emergency department visits for asthma was derived from Jalaludin et al. and the report by Golder Associates (Golder Associates 2013, Jalaludin et al. 2008).

These six HIAs were conducted before the publication of the recent reports included in Tables 9 and 10, e.g., the 2022 HEI report on TRAP and health effects (Health Effects Institute 2022) and the 2022 COMEAP report (COMEAP 2022). Therefore, these HIAs could be considered to have been conducted according to known evidence at that time.

Independent advice provided to ACTAQ regarding tunnel ventilation stack emissions is that "*nearby residents will experience little, if any, increase in exposure to vehicle emissions*" (Longley 2018).

However, as the scientific consensus is that there is no threshold for $PM_{2.5}$ and NO_2 below which adverse health effects do not occur (i.e., there is no "safe" level) (Zosky et al. 2021), that on-road motor vehicles account for a moderate to a large proportion of the population-weighted exposure to $PM_{2.5}$ and NO_2 (DPIE 2020), and the increasing number of adverse health effects attributable to $PM_{2.5}$ and NO_2, it is imperative that governments and regulators continue to implement and evaluate the effectiveness of measures to reduce exposure to TRAP thereby improving the health and well-being of the population.

TABLE 11 PRIMARY HEALTH OUTCOMES AND CONCENTRATION-RESPONSE FUNCTIONS[1] IN ROAD TUNNEL HEALTH IMPACT ASSESSMENTS

	$PM_{2.5}$ RR (95%CI) per 10 µg/m³			NO_2[2] RR per 10 µg/m³		
	All-cause mortality (long-term exposure, age ≥30 years)	**Cardiovascular hospitalisation (short-term exposure, all ages)**	**Respiratory hospitalisation (short-term exposure, age ≥30 years)**	**All-cause mortality (short-term exposure, all ages)**	**Respiratory mortality (short-term exposure, all ages)**	**Asthma emergency department visits (short-term exposure, age 1-14 years)**
NorthConnex (Environmental Risk Sciences 2014)	1.06 (1.04-1.08)	1.008 (1.0059-1.011)	1.0041 (1.0009-1.0074)	n/a[3]	n/a	n/a
M4 East (Environmental Risk Sciences 2015a)	1.06 (1.04-1.08)	1.008 (1.0059-1.011)	1.0041 (1.0009-1.0074)	1.0190	1.0435	1.0116
New M5 (Environmental Risk Sciences 2015b)	1.06 (1.04-1.08)	1.008 (1.0059-1.011)	1.0041 (1.0009-1.0074)	1.0190	1.0435	1.0116
M4-M5 Link (Environmental Risk Sciences 2017)	1.06 (1.04-1.08)	1.008 (1.0059-1.011)	1.0041 (1.0009-1.0074)	1.0190	1.0435	1.0116
M6 Stage 1 (AECOM 2018)	1.06, (1.04-1.08)	1.008 (1.0059-1.011)	1.0041 (1.0059-1.011)	1.0190	1.0435	1.0116
West Harbour Tunnel (Environmental Risk Sciences 2020)	1.06 (1.04-1.08)	1.008 (1.0059-1.011)	1.0041 (1.0009-1.0074)	1.0190	1.0435	1.0116

[1]The source reference for the CRFs are presented in the text. [2]Beta-coefficient converted to relative risks to enable comparison with other CRFs in this report. It was not possible to calculate confidence intervals as standard errors were not reported in the source documents. [3]Not available as no quantitative assessment was conducted.

Over the past 10 to 15 years, the evidence base for exposure to TRAP and its adverse health effects has increased. Therefore, future HIAs for road tunnels should consider the following:

1. The number of studies and health outcomes linked to air pollution has increased (Tables 3 and 4). For many of these outcomes, there is moderate to high confidence in the evidence for associations between TRAP and health outcomes. Where current evidence for inclusion of health outcomes in HIAs is insufficient, the evidence should be re-evaluated when future HIAs are conducted. For example, the 2022 HEI report (Health Effects Institute 2022) conducted narrative reviews for neurodevelopmental outcomes in children, and dementia-related outcomes and Parkinson disease in

adults, as the expert review panel thought these were important emerging areas that should be highlighted, while awaiting more robust evidence.

2. The strength of the evidence base for inferring causality and confidence in the associations between TRAP and health outcomes is expanding. There are now more pollutant-outcome pairs for which there is sufficient evidence to infer causality or confidence in the evidence for associations between TRAP and adverse health effects. The number of pollutant-outcome pairs that can be classed as causal or likely to be causal (or moderate to high confidence in the evidence for associations) has increased (see Tables 6 and 8). Moderate to high confidence in the evidence for associations with health outcomes are more common for $PM_{2.5}$ than NO_2.

3. Most importantly, the number of available CRFs for inclusion in an HIA has increased (Tables 9 and 10). For example, the WHO HRAPIE 2013 report (WHO 2013a) presented two statistically significant CRFs for long-term exposure to $PM_{2.5}$ and one statistically significant CRF for long-term exposure to NO_2. In contrast, about 10 years on, in the two reports (COMEAP 2022, Health Effects Institute 2022) and one review of reviews (Forastiere et al. 2024), there are 19 statistically significant CRFs each for $PM_{2.5}$ and NO_2. CRFs are also available for health outcomes not presented in Tables 9 and 10. The US EPA has published CRFs for long-term exposure to $PM_{2.5}$ and Alzheimer disease and Parkinson disease in older adults and allergic rhinitis in children (US EPA 2024).

Summary

In the past 10 to 15 years there has been continuing research into the health effects of ambient air pollution, resulting in an increasingly robust evidence base. New adverse health effects continue to be identified, the evidence for robust associations or causality continues to strengthen, and the number of CRFs available for quantification in an HIA has increased. New information about the adverse impact of air pollution will better inform policies to control TRAP.

There are no safe levels for $PM_{2.5}$ and NO_2, with demonstrated adverse health effects at very low levels of $PM_{2.5}$. Whole populations (including foetuses) are exposed to air pollution. Therefore, measures to control air pollution should aim to continually reduce air pollution levels and to levels below the existing national standards.

Previous HIAs for road tunnels quantified a limited number of health outcomes (5-6 outcomes) with only one outcome associated with long-term exposure to air pollutants ($PM_{2.5}$ and mortality). These HIAs would have underestimated the adverse impact of any changes in air quality on human health. However, they could be considered to have been conducted according to known knowledge at that time. Future HIAs, whether conducted for road tunnels or for other major infrastructure developments that could impact air quality, or for policy purposes, should consider the additional health outcomes for which robust CRFs are now available.

Independent advice provided to ACTAQ is that tunnel ventilation stack emissions will have minimal impact on local communities. However, as the scientific consensus is that there is no threshold for $PM_{2.5}$ and NO_2 below which adverse health effects do not occur (i.e., there is no “safe” level), that on-road motor vehicles account for a sizable proportion of the exposure to ambient air pollution(DPIE 2020), and the increasing number of adverse health effects

attributable to air pollution, governments and regulators must continue to implement and evaluate the effectiveness of measures to reduce exposure to TRAP thereby improving the health and well-being of the population.

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