Technical Paper

TP03: Health Effects of Traffic-related Air Pollution

Advisory Committee on Tunnel Air Quality

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Key Points

- Motor vehicles are a major source of air pollution in Sydney and other urban centres.
- Exposure to traffic-related air pollution is linked to a range of adverse health outcomes.
- Reducing exposure to traffic-related air pollution will provide public health benefits, including improved cardiovascular and respiratory health and reduced rates of some cancers.

Contents

1. Introduction

Outdoor air pollution is a complex mixture of substances with differing physical and chemical properties. Important air pollutants, particularly in the context of traffic, are particulate matter (PM), ozone (O_3), nitrogen dioxide (NO_2), carbon monoxide (CO), polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs). In urban environments, motor vehicles are a significant source of particulate matter smaller than 2.5 micrometres in diameter ($PM_{2.5}$), NO_2, PAHs and VOCs.

Air quality in Australia is very good when compared to countries at similar levels of economic development. To control air quality, each state and territory is required to comply with a set of standards specified in the National Environment Protection (Ambient Air Quality) Measure.

These standards have been designed to provide adequate protection to human health; however, air pollution will still have health effects at levels below current standards[1]. As such, reductions in levels of air pollution below current standards can be expected to provide some benefits.

This paper summarises what is known about the health effects of air pollution related to traffic pollutants.

2. Key Air Pollutants Linked to Traffic Pollution

2.1 Particulate matter

PM is a term used to describe airborne microscopic solid or liquid particles. PM is generally classified according to the size of the particles. Particles less than 10 micrometres in diameter are called PM_{10}, particles less than 2.5 micrometres in diameter are $PM_{2.5}$ and particles less than 0.1 micrometres in diameter are called ultrafine particles (UFPs). It is important to note that PM_{10} includes both $PM_{2.5}$ and UFPs.

A key feature of PM is that no threshold has been identified below which exposure is not associated with adverse health effects, and reductions in ambient concentrations will provide public health benefits.

In 2013, the International Agency for Research on Cancer classified PM as carcinogenic to humans.[2]

$PM_{2.5}$

$PM_{2.5}$ is an emission of combustion and there is very good evidence that increased exposure to $PM_{2.5}$ is associated with increases in cardiovascular disease symptoms, respiratory disease symptoms and mortality. Associations have also been observed between $PM_{2.5}$ exposure and adverse reproductive and developmental outcomes such as low birth weight.[3]

PM_{10}

Exposure to PM_{10} is also associated with similar health effects observed for $PM_{2.5}$. However, because PM_{10} includes $PM_{2.5}$, there is some uncertainty about how much of the observed effect is due to $PM_{2.5}$ and how much is due to the larger particle fraction ($PM_{10\text{-}2.5}$).

Ultrafine particles

Motor vehicle exhaust is an important source of ultrafine pollution in urban settings.[4] UFPs are thought to play a role in the adverse health impacts seen in association with exposure to particulate pollution, although the epidemiological evidence of their effects is limited (HEI 2013).

1 Barnett AG, et al (2006). The Effects of Air Pollution on Hospitalisations for Cardiovascular Disease in Elderly People in Australian and New Zealand Cities; and Crouse DL, et al (2012) Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study.
2 Loomis D, et al. (2013). The carcinogenicity of outdoor air pollution.
3 Pedersen M, et al (2013). Ambient Air Pollution and Low Birthweight: A European Cohort Study.
4 HEI Panel on Ultrafine Particles. (2013).

2.2 Ozone

O_3 is formed when precursor compounds (VOC and NOx) photo-chemically react in the presence of sunlight. O_3 pollution can reach high levels on hot, still days and builds over a day, reaching its peak in late afternoon.

Short-term exposure to O_3 can result in reduced lung function, exacerbation of asthma and chronic respiratory diseases, irritation and inflammation of eyes, nose, throat and lower airways. There is a growing body of evidence that long-term exposure to O_3 may be associated with respiratory and cardiovascular mortality, as well as respiratory morbidity (WHO 2013).

2.3 Nitrogen dioxide

NO_2 is produced by combustion and is a good marker of traffic-related air pollution. Toxicological studies have found adverse health effects of NO_2, but at levels far exceeding those normally found in ambient air.[5] NO_2 is highly correlated with other pollutants from combustion sources, making it very difficult to separate the effects of ambient NO_2 from the effects of other traffic-related pollutants, especially PM. However, there is increasing evidence indicating there are effects associated with NO_2 that are in addition to those associated with PM (WHO 2013).

The NSW in-tunnel guideline limit of 0.5 ppm NO_2 is based on a review of studies of the health effects of exposure to NO_2 for up to 30 minutes.[6] A more recent review of the health effects of exposure to NO_2 for up to 60 minutes[7] supports the use of this guideline value for up to 60 minutes of exposure.

2.4 Carbon monoxide

CO is produced during incomplete combustion of carbon-containing fuels such as petrol (WHO 2006).

CO can cause harmful health effects by reducing the amount of oxygen reaching the body's organs (such as the heart and brain) and tissues. At extremely high levels, CO can cause death (carbon monoxide poisoning).

2.5 Polycyclic aromatic hydrocarbons

PAHs comprise over 100 different compounds. Some PAHs are carcinogens, for example benzo(a)pyrene. PAHs are often transported in the atmosphere attached to $PM_{2.5}$, which means their effects cannot be easily separated from the effects of particles (WHO 2013).

2.6 Volatile organic compounds

Key VOCs from vehicle exhaust include benzene and formaldehyde (HEI 2010). These are typically present in low concentrations in the air but have toxic characteristics that may result in health effects from exposure even at low levels. Benzene and formaldehyde are classified as carcinogenic to humans.[8] Motor vehicle exhaust emissions of VOCs are important precursors to the formation of O_3 (WHO 2013).

5 WHO Regional Office for Europe (2006). Air quality guideline global update 2005: particulate matter, ozone, nitrogen dioxide and sulphur dioxide.
6 Jalaludin B (2015) Review of experimental studies of exposure to nitrogen dioxide.
7 Environmental Risk Science Pty Ltd (2017) Literature Review and Risk Characterisation of Nitrogen Dioxide: Long and Heavily Trafficked Road Tunnels.
8 IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. (2009).

3. Health Effects of Proximity to Traffic

Adverse health effects have been observed in association with proximity to roads. These effects persist after adjustment for noise and socioeconomic status and are only partly explained by exposure to $PM_{2.5}$. As such, it is likely that observed health effects result from exposure to a number of traffic-related pollutants, either individually or cumulatively (WHO 2013).

The Health Effects Institute (HEI) report *Traffic-related air pollution: a critical review of literature on emissions, exposure and health effects* synthesised research on traffic-related air pollution exposure and health outcomes (HEI 2010).

The HEI review panel considered that there was sufficient evidence to conclude that exposure to traffic-related air pollution causes exacerbation of asthma. The evidence linking exposure from traffic-related air pollution to several other health outcomes was weaker, but considered suggestive of a causal relationship. These outcomes were onset of childhood asthma, non-asthma respiratory symptoms, impaired lung function, total and cardiovascular mortality and cardiovascular morbidity.

The HEI report also identified that an exposure zone extending up to 300 to 500 metres from a major road was the zone most highly affected by traffic emissions (HEI 2010).

Non-exhaust traffic emissions (brake wear, engine abrasion, tyre wear) result in non-combustion PM pollution. Toxicological studies have demonstrated that these non-exhaust emissions contribute to the health impact from exposure to traffic-related pollution (WHO 2013). Non-exhaust emissions are a significant source of on-road particle emissions in Sydney (*Technical Paper No.1 – Trends in fuel quality and motor vehicle emissions*).[9]

As exhaust emissions are further regulated and reduced, understanding the health impacts of non-exhaust emissions will increasingly become the focus to address health risks from future traffic pollution (HEI 2010, WHO 2013).

9 NSW EPA (2012) 2008 calendar year air emissions inventory for the Greater Metropolitan Region of NSW.

4. Carcinogenicity of Air Pollution and its Constituents

The International Agency for Research on Cancer (IARC) has classified outdoor air pollution as carcinogenic to humans (IARC Group 1) (Loomis et al 2013).

The IARC has also concluded that diesel engine exhaust is carcinogenic to humans (Group 1). In coming to this conclusion, the IARC identified diesel exhaust as a cause of lung cancer and noted limited evidence of a positive association with an increased risk of bladder cancer.[10] Benzene and formaldehyde (VOCs linked to vehicle exhaust) have also been classified by IARC as Group 1 carcinogens (IARC 2009).

10 Benbrahim-Tallaa L, et al. (2012). Carcinogenicity of diesel-engine and gasoline-engine exhausts and some nitroarenes.

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