

TP03:
**Health Effects
of Traffic-Related
Air Pollution**

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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 cancer.

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, nitrogen dioxide (NO₂) and 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}), NO₂, 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 may still have health effects at levels below current standards (eg Barnett *et al* 2006, Crouse *et al* 2012) and so reductions in exposure can still be expected to provide benefit.

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

Particulate matter

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 PM₁₀, 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 PM₁₀ 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.

Recently, the International Agency for Research on Cancer determined that PM was carcinogenic to humans (Loomis *et al* 2013).

PM_{2.5}

PM_{2.5} is generally produced by combustion. There is very good evidence that exposure to PM_{2.5} causes cardiovascular disease, respiratory disease and mortality. Associations have also been observed between PM_{2.5} exposure and reproductive and development effects such as low birth weight (Pedersen *et al* 2013).

PM₁₀

Exposure to PM₁₀ is also associated with cardiovascular disease, respiratory disease and mortality. However, because PM₁₀ 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_{10-2.5}).

UFPs

Motor vehicle exhaust is an important source of ultrafine pollution in urban settings (HEI 2013). Ultrafine particles 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, WHO 2013). Until there is clearer evidence of the concentration-effect relationship for UFPs, WHO recommends that management of PM should continue to focus on PM₁₀ and PM_{2.5}.

Ozone

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

Short term exposure to ozone 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 to support that long term exposure to ozone may affect respiratory and cardiovascular mortality, and respiratory morbidity (WHO 2013).

There is currently inconsistent evidence to indicate there is a threshold below which exposure to ozone is not associated with adverse health effects. From available evidence, if there were a threshold it would be below 45ppb (1 hour average) (WHO 2013).

Nitrogen dioxide

Nitrogen dioxide (NO₂) is produced by combustion and is a good marker of traffic-related pollution. Toxicological studies have found effects of NO₂, but at levels far exceeding those normally found in ambient air (WHO 2006). NO₂ is highly correlated with other pollutants from combustion sources, which has made it very difficult to separate the effects of ambient NO₂ from the effects of other traffic-related pollutants, especially PM. However, there is increasing evidence that indicates there are independent effects of NO₂ separate from PM (WHO 2013).

3. Health effects of proximity to traffic

Carbon monoxide

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

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

Polycyclic aromatic hydrocarbons

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 separated from the effects of particles (WHO 2013).

Volatile organic compounds

Key volatile organic compounds (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 Group 1 carcinogens to humans (IARC 2009). Motor vehicle emissions of exhaust VOCs are also important precursors to the formation of ozone (WHO 2013).

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}. Therefore, it is likely that they result from exposure to other traffic-related pollutants, either individually or in combination (WHO 2013).

The Health Effects Institute 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 to 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 500m from a major road was the most highly affected by traffic emissions (HEI 2010).

Non-exhaust emissions (brake wear, engine abrasion, tyre wear) may result in non-combustion particulate matter 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 (NSW EPA 2012). As exhaust emissions are further regulated and reduced, understanding non-exhaust emissions will increasingly become the focus to address health risks from future traffic pollution (HEI 2010, WHO 2013).

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 group found that diesel exhaust is a cause of lung cancer (sufficient evidence) and noted a positive association (limited evidence) with an increased risk of bladder cancer (Benbrahim-Tallaa *et al* 2012). Benzene and formaldehyde (VOCs linked to vehicle exhaust) have also been classified by IARC as Group 1 carcinogens (IARC 2009).

5. References

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