Health and air pollution in New Zealand 2016 (HAPINZ 3.0)

He rangi hauora he iwi ora

Volume 2 – Detailed methodology

Prepared for

Ministry for the Environment
Ministry of Health
Te Manatū Waka Ministry of Transport
Waka Kotahi NZ Transport Agency

March 2022

emission impossible ltd and the HAPINZ 3.0 team

Suite 2-3, 93 Dominion Rd
Mt Eden, Auckland 1024
www.emissionimpossible.co.nz

Disclaimer

This research was funded by the Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency.

Recommended Citation:

Kuschel *et al* (2022). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Volume 2 – Detailed methodology.* Report prepared by G Kuschel, J Metcalfe, S Sridhar, P Davy, K Hastings, K Mason, T Denne, J Berentson-Shaw, S Bell, S Hales, J Atkinson and A Woodward for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, March 2022.

Health and air pollution in New Zealand 2016 (HAPINZ 3.0)

He rangi hauora he iwi ora

Volume 2 – Detailed methodology

Prepared by

Gerda Kuschel, Jayne Metcalfe and Surekha Sridhar
(Emission Impossible Ltd)

Perry Davy (GNS Science)

Keith Hastings (Jacobs Ltd)

Kylie Mason (Massey University)

Tim Denne (Resource Economics Ltd)

Alistair Woodward (University of Auckland)

Simon Hales and June Atkinson (University of Otago)

Jess Berentson-Shaw and Sharon Bell (The Workshop)

March 2022

Acknowledgements

This research was funded by the Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency (**Waka Kotahi**).

The authors would like to thank staff from the following councils for enthusiastically and efficiently assisting us in sourcing relevant monitoring datasets and documents throughout the study:

- Northland Regional Council
- Auckland Council
- Waikato Regional Council
- Bay of Plenty Regional Council
- Gisborne District Council
- Hawke's Bay Regional Council
- Taranaki Regional Council
- Horizons (Manawatū-Whanganui) Regional Council
- Greater Wellington Regional Council
- Tasman District Council
- Nelson City Council
- Marlborough District Council
- Environment Canterbury
- West Coast Regional Council
- Otago Regional Council
- Southland Regional Council

We would also like to acknowledge the invaluable guidance provided by following people who acted as the HAPINZ 3.0 Steering Committee:

- Drew Bingham, Co-chair (Ministry for the Environment
- Shelley Easton and Christine Moore, Co-chairs (Waka Kotahi)
- Greg Haldane and Janet Petersen (Waka Kotahi)
- Iain McGlinchy and Daisy Cadigan (Te Manatū Waka Ministry of Transport)
- Sonja Miller (Stats NZ)
- Suz Halligan (Ministry of Health)
- Tim Mallett and Tamsin Mitchell (National Air Quality Working Group)

As well as our eminent international peer reviewers:

- Dr Xavier Querol (Institute of Environmental Assessment & Water Research, Spain)
- Dr Mike Holland (Ecometrics Research and Consulting, United Kingdom)
- Prof Bert Brunekreef (Utrecht University, The Netherlands)

The support we received from everyone was truly outstanding in the face of the challenges posed by COVID-19 over the project duration. Thanks and ngā mihi nui!

Contents

List of tables

List of figures

Executive summary

Clean healthy air contributes to New Zealand's quality of life - not only people's health, but also the natural functioning of and the "*beauty of the natural and physical environment*" (MfE 2007). New Zealand has good air quality in most locations for most of the time. However, solid fuel (wood and coal) used for domestic heating and exhaust emissions from vehicles combine to produce unacceptable air quality in some locations, particularly during winter. Despite the relatively low levels of pollution in New Zealand versus other countries, the health burden associated with air pollution is still appreciable.

Air pollution health effects in New Zealand were first comprehensively assessed in the Health and Air Pollution in New Zealand (**HAPINZ 1.0**)[1] study undertaken by Fisher *et al* (2007). In HAPINZ 1.0, health effects were evaluated for 67 urban areas based on the 2001 population and ambient monitoring data. Resulting social costs were presented in NZ$ as at June 2004.

This work was later updated by Kuschel *et al* (2012a) to incorporate population data from the 2006 census and more comprehensive monitoring being undertaken across New Zealand in response to the introduction of a national environmental standard for ambient particulate matter (**PM_{10}**) concentrations in September 2005. This update (**HAPINZ 2.0**) estimated that air pollution from all sources in New Zealand was responsible each year for approximately 2,300 premature deaths, nearly 1,200 hospitalisations and more than 2.9 million restricted activity days at a total cost of NZ$8.4 billion as at June 2010.

Since 2012, air quality monitoring has further expanded across New Zealand to include many more locations, pollutants and sources, and exposure-response functions are now available for a greater range of health endpoints. In recognition, the Ministry for the Environment and Waka Kotahi (in partnership with Te Manatū Waka Ministry of Transport and Ministry of Health) commissioned a new update – **HAPINZ 3.0** – in 2019 to better reflect the air pollution health impacts experienced by New Zealanders and to update the effects for 2016.

This report (Volume 2 – Detailed methodology) outlines the approach we developed to assess health effects associated with air pollution in New Zealand for 2016. The resulting air pollution health impacts and social costs[2] are presented in Volume 1 – Findings and implications, which is available separately (Kuschel *et al* 2022).

Recognising that air and air quality are both *taonga*[3] and a part of *kaitiakitanga*[4] for Māori, the HAPINZ 3.0 study is also named *He rangi hauora he iwi ora* which translates to healthy air means healthy people.

---

[1] This report refers to the previous studies of Fisher *et a*l (2007) and Kuschel *et al* (2012a) as HAPINZ 1.0 and HAPINZ 2.0 respectively to make it easier to differentiate between those studies and this one (HAPINZ 3.0).

[2] Costs here are referred to as social costs rather than health costs because they denote the total costs to society of the health effects, which are more than just the costs incurred by the health system.

[3] A taonga in Māori culture is a treasured thing, whether tangible or intangible.

[4] A kaitiaki is a guardian, and the process and practices of protecting and looking after the environment are referred to as kaitiakitanga.

The key features of the approach we adopted are summarised in the following table:

Key features of the HAPINZ 3.0 update

Feature	Details
Base year	2016 for population
Spatial resolution	**Calculations** undertaken using 2013 census area unit boundaries **Results** aggregated by 16 regional councils, 20 district health boards, 67 territorial authorities and 89 airsheds
Population covered	100% of 2016 population
Pollutants	**Priority pollutants** • particulate matter ($PM_{2.5}$ and PM_{10}) • nitrogen dioxide (NO_2)
Exposure assessment	**$PM_{2.5}$ and PM_{10}**: ambient monitoring data typically averaged for 2015-2017 covering the majority of urban areas in New Zealand, with proxy monitoring used in unmonitored areas **NO_2**: modelling estimates from the NZ Transport Agency NVED exposure tool
Source attributions	**$PM_{2.5}$ and PM_{10}**: using source apportionment data and assigned to domestic fires, motor vehicles, industry, windblown dust, sea spray, and secondary PM **NO_2**: no source apportionment data available but assigned to motor vehicles (estimated to contribute approximately 90% of NO_2 exposure in urban areas)
Health endpoints	**Primary health impacts** • **mortality** and years of life lost (YLL) from long-term **$PM_{2.5}$** for all adults 30+ years, all ethnicities and Māori/Pacific peoples • **cardiac admissions** from long-term **$PM_{2.5}$** for all ages, all ethnicities • **respiratory admissions** from long-term **$PM_{2.5}$** for all ages, all ethnicities • **restricted activity days** from long-term **$PM_{2.5}$** for all ages, all ethnicities • **mortality** and YLL from long-term **NO_2** for all adults 30+ years, all ethnicities • **cardiac admissions** from long-term **NO_2** for all ages, all ethnicities • **respiratory admissions** from long-term **NO_2** for all ages, all ethnicities **Secondary health impacts (for comparison with HAPINZ 2.0)** • **mortality** from long-term **PM_{10}** for all adults 30+ years, all ethnicities and for Māori • **restricted activity days** from long-term **$PM_{2.5}$** for all ages, all ethnicities (also in primary health impacts) **Childhood asthma impacts relevant to NZ** • **asthma/wheeze hospitalisations** due to long-term **NO_2** for all 0-18 years • **asthma prevalence** due to long-term **NO_2** for all 0-18 years
Social costs	**Valuation of mortality costs** • change in mortality multiplied by current NZ Value of a Statistical Life (**VoSL**) • change in total life years multiplied by a NZ Value of a Life Year (**VoLY**)

Feature	Details
Social costs (cont.)	**Valuation of morbidity costs** • cardiovascular and respiratory hospital admissions • restricted activity days • childhood asthma costs from GP visits, medication and hospitalisation **Development of a suite of NZ-specific damage costs** for consistent assessment of benefits to society in reducing harmful emissions and greenhouse gases
Key outputs	Combined **exposure/health effects model** enabling sensitivity/scenario testing and designed to be easily updateable together with a Users' Guide A **set of New Zealand-specific exposure-response functions** for assessing effects of air pollution on mortality and morbidity amongst New Zealanders A **detailed report**, suitable for a technical audience, outlining the methodology adopted and clearly stating all assumptions (Volume 2) A **summary report**, suitable for a more general audience, presenting the key findings and discussing their implications (Volume 1) A draft **messaging guide** to provide evidence-based dos and don'ts for anyone wanting to communicate the study findings through various channels together with a checklist

For further details on the development of the HAPINZ 3.0 methodology, please see:

Bell S & Berentson-Shaw J (2020). *Literature review: Framing air quality and environmental health*. Supplementary report for HAPINZ 3.0 prepared by The Workshop. Wellington, NZ, October 2020.

Davy PK & Trompetter WJ (2020). *An empirical model for attributing sources of particulate matter*. Supplementary report for HAPINZ 3.0 prepared by GNS Science, GNS Science consultancy report 2020/33, Institute of Geological and Nuclear Sciences, New Zealand. November 2020.

Kuschel G *et al* (2020). *Health and air pollution in New Zealand 2016: Approved methodology*. Prepared by G Kuschel, J Metcalfe, P Davy, K Hastings, K Mason, T Denne, J Berentson-Shaw, S Hales, J Atkinson and A Woodward for Ministry for the Environment, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency. 17 March 2020.

Sridhar S & Metcalfe J (2021). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Sources*. Excel model prepared by S Sridhar and J Metcalfe for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, February 2021.

1. Introduction

This chapter outlines background on the previous HAPINZ studies, reasons for the current update, the agencies funding and supporting this update, the purpose of the study and how the report is structured.

1.1 Background

1.1.1 The first NZ health and air pollution study in 2007 (HAPINZ 1.0)

Air pollution health effects in New Zealand were first comprehensively assessed in the original Health and Air Pollution in New Zealand (**HAPINZ 1.0**)[5] study, undertaken by Fisher *et al* (2007).

In HAPINZ 1.0, health effects were evaluated for 67 urban areas (covering 73% of the 2001 population) and ambient monitoring data. The authors estimated that air pollution from all sources in New Zealand was responsible for approximately 1,400 premature deaths per year, of which 1,100 premature deaths were attributed to *anthropogenic* (human-caused) sources. The resulting social costs for the 67 areas were estimated at approximately $1.45 billion[6] (in NZ$ as at June 2004).

The authors found the greatest effect was premature mortality associated with long-term exposure to particulate matter less than 10 micrometres (**µm**) in diameter (**PM_{10}**) from combustion sources. However, mortality effects due to carbon monoxide (**CO**) and various morbidity (non-mortality illness) effects associated with other pollutants were also identified. A separate mortality effect associated with exposure to nitrogen dioxide (**NO_2**) was not determined as the authors assumed NO_2 was strongly correlated with PM_{10} and that the exposure-response function for PM_{10} would capture mortality effects for both pollutants.

The key features of HAPINZ 1.0 are summarised in Table 1.

Table 1: Key features of the original HAPINZ study (HAPINZ 1.0)

Feature	Details
Base year	• 2001 for population
Spatial resolution	• 67 'urban' areas by 950 census area units
Population covered	• 2,803,215 covering 73% of 2001 population
Pollutants	• PM_{10} • plus CO, NO_2, benzene

[5] This report refers to the previous studies of Fisher *et al* (2007) as HAPINZ 1.0 and Kuschel *et al* (2012a) as HAPINZ 2.0 to make it easier to differentiate between those studies and the current one (HAPINZ 3.0).

[6] However, HAPINZ 1.0 used a reduced figure of $750,000 per premature death, which was significantly less than the value of statistical life used for road safety deaths of $2.725 million at that time and in later HAPINZ studies.

Feature	Details
Sources	• natural sources (sea spray and windblown dust) • domestic fires • motor vehicles • industry
Exposure assessment	• Land-based regression model developed for 'urban' areas based on ambient PM_{10} monitoring from 43 locations in 2001
Health endpoints (all for adults 30+ years)	• mortality (for PM_{10}, NO_2 combined) and mortality (for CO) • chronic bronchitis • acute respiratory admissions and acute cardiac admissions • cancer (for benzene) • restricted activity days
Social costs	• costs per case in NZ$ as at June 2004 • mortality and cancer - $750,000 (based on discounted value of Road Safety VoSL of $2.73M) • chronic bronchitis - $75,000 • cardiac admission - $3,675 • respiratory admission - $2,700 • restricted activity day - $92
Annual costs per person	• $421 per person per year from anthropogenic air pollution alone

Note: VoSL = value of statistical life

1.1.2 The first update in 2012 (HAPINZ 2.0)

Following the release of HAPINZ 1.0, data availability and the understanding of air pollution health effects improved significantly, superseding many elements and assumptions of the original methodology. Air quality monitoring was implemented in most urban locations in New Zealand – largely due to the introduction of a national environmental standard for ambient PM_{10} concentrations in 2005.

In response, an update was commissioned (**HAPINZ 2.0**) to incorporate population data from the 2006 census, utilise the more comprehensive monitoring datasets and revise the underlying assumptions (Kuschel *et al* 2012a). HAPINZ 2.0 assessed effects from PM_{10} only. Most of the health effects in New Zealand were assumed to be associated with PM_{10} and it was considered a good indicator of the sources and effects of other air pollutants. At the time, limited data existed for other pollutants such as $PM_{2.5}$ and NO_2.

Air pollution from all sources in New Zealand in 2006 was estimated to be responsible for approximately 2,300 premature deaths, nearly 1,200 hospitalisations and more than 2.9 million restricted activity days at a total cost of NZ$8.4 billion (in NZ$ as at June 2010). Approximately half of these effects and costs were associated with *anthropogenic* (human-generated) sources such as domestic fires, motor vehicles, industry and open burning. Domestic fires were identified as the largest contributor to anthropogenic effects but the authors noted that the impact of motor vehicles was likely to be under-estimated as effects due to exposure to NO_2 were not able to be quantified.

The primary outputs from this work were:

- a report summarising the methodology and presenting the findings
- a technical report with more details on the methodology
- an exposure model linked to a health effects model which enabled scenarios to be tested around changes in ambient concentrations and population.

The key features of HAPINZ 2.0 are summarised in Table 2.

Table 2: Key features of the first HAPINZ update (HAPINZ 2.0)

Feature	Details
Base year	• 2006 for population
Spatial resolution	• All of New Zealand by 1,919 census area units • plus by 16 regional councils, 71 airsheds, 74 territorial authorities and 139 urban areas
Population covered	• 4,027,902 covering 100% of 2006 population
Pollutants	• PM_{10} (as a proxy for all air pollutants)
Sources	• natural sources (sea spray and windblown dust) • domestic fires • motor vehicles • industry • open burning
Exposure assessment	• Ambient PM_{10} monitoring data for 2006-2008 covering 73% of 2006 population with proxy monitoring in remaining areas
Health endpoints	• mortality for all adults 30+ years, all ethnicities and for Māori only • mortality for babies 1 month to 1 year, all ethnicities • cardiac admissions for all ages, all ethnicities • respiratory admissions for all ages, all ethnicities and for children aged 1-4 years and children aged 5-14 years • restricted activity days for all ages, all ethnicities
Social costs	• costs per case in NZ$ as at June 2010 • mortality - $3.56 million (based on full value of Road Safety VoSL of $3.56M) • cardiac admission - $6,350 • respiratory admission - $4,535 • restricted activity day - $62
Annual costs per person	• $1,061 per person per year from anthropogenic air pollution alone

The results from HAPINZ 2.0 were constrained by limitations in the datasets and the availability of other supporting information at the time the study was undertaken. Recommendations to improve coverage and robustness in future updates included:

- incorporating assessment of NO_2 exposure
- including particulate matter less than 2.5 µm in diameter (**$PM_{2.5}$**) in addition to PM_{10}
- developing improved emission factors and activity rate data for open burning

- investigating inclusion of other transport sources, such as shipping, aviation, rail and off-road vehicles
- refining the exposure-response functions for Māori and other ethnic subgroups (such as Pacific peoples)
- reviewing the appropriateness of using a transport safety risk-based value of statistical life (**VoSL**) for an environmental risk-based VoSL in New Zealand
- estimating loss of life quality effects.

1.2 Reasons for this latest update (HAPINZ 3.0)

Following the release of HAPINZ 2.0 in 2012, the database of ambient monitoring across New Zealand expanded further to include many more locations and pollutants, with increased source apportionment analyses. In addition, exposure-response functions were reported in the literature to enable quantification of a greater range of health endpoints.

In mid-2019, the Ministry for the Environment (**MfE**) commissioned an update of HAPINZ 2.0 to provide new, robust analyses that were in line with international best practice. The objective of the update was to identify the human health effects of air pollution throughout New Zealand and link these impacts to the various sources and levels of air pollution.

A team of experienced researchers led by Emission Impossible Ltd commenced the latest update - **HAPINZ 3.0** - in July 2019. The update was funded by MfE, Ministry of Health (**MoH**), Te Manatū Waka Ministry of Transport (**MoT**) and Waka Kotahi NZ Transport Agency (**Waka Kotahi**).

1.3 Report layout

This report – Volume 2 – outlines the methodology we developed to assess health effects associated with air pollution in New Zealand for 2016, and is structured as follows:

- Chapter 2 discusses the key pollutants of concern in New Zealand, introduces the key steps typically involved in assessing air pollution health impacts and outlines the international best practice principles we followed in our study
- Chapter 3 outlines the approach we used to assess the exposure of New Zealanders to critical air pollutants
- Chapter 4 describes how we then attributed the contribution of major sources to the pollutant exposures
- Chapter 5 discusses the health impacts we assessed, including work undertaken to develop New Zealand-specific exposure-response functions for critical endpoints
- Chapter 6 describes how we estimated social costs associated with the resulting air pollution health effects
- Chapter 7 describes the development of the health effects model, its key features, updateability, treatment of uncertainty and scenario testing capability

- Chapter 8 reviews our recommendations for the most effective ways to communicate the key study messages to a broad audience
- Chapter 9 summarises the features of our methodology.

A set of technical appendices providing further detail are included at the end of this report.

Note: The results are discussed in a companion report (Volume 1 – Findings and implications), which is available separately (Kuschel *et al* 2022).

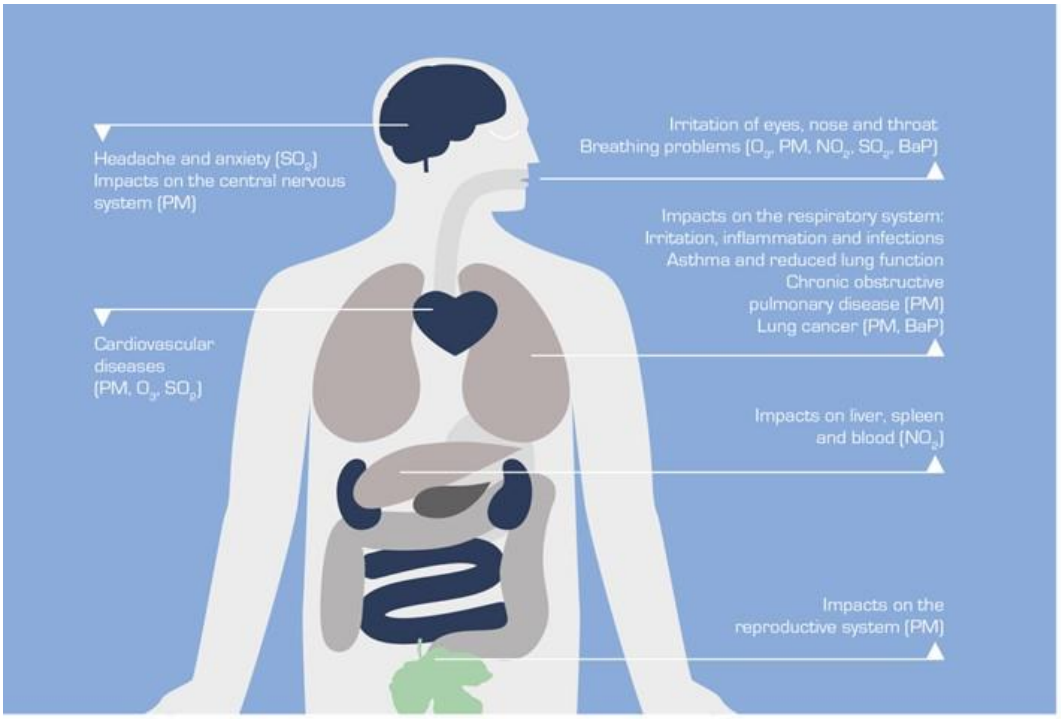
2. Assessing air pollution health impacts

This chapter discusses the nature of health effects resulting from air pollution exposure, who is most likely to be affected, the air pollutants of most concern in New Zealand, and how air pollution health impacts are typically assessed in accordance with international best practice.

2.1 Health effects of air pollution

Air pollution can cause significant health impacts ranging from increased disease and illness (*morbidity*) to premature death (*mortality*) as shown in Figure 1.

Figure 1: The impact of air pollution on the human body



Note: BaP = benzo(a)pyrene; NO_2 = nitrogen dioxide; O_3 = ozone; PM = particulate matter; SO_2 = sulphur dioxide.

Source: EEA (2014)

The resultant health effects depend on the pollutant and the length of exposure – either short-term (*acute*) or long-term (*chronic*). Short–term exposures cover minutes, hours, or days. Long-term exposures are usually over months or years.

Short-term exposure to urban air pollution can cause respiratory irritation, even in healthy people. Clinical studies have shown a range of acute cardiovascular and respiratory effects in volunteers with or without pre-existing diseases. Some short-term effects (such as heart rhythm disturbances) are completely reversible, but others can cause chronic inflammation of

the lungs and blood vessels, and eventually, following repeated exposure, lead to chronic diseases such as lung cancer and *atherosclerosis* (hardening of the arteries).

Even though short-term effects can include premature death in susceptible individuals, the major impact of air pollution exposure on life expectancy is through the gradual, cumulative effects on chronic disease. Depending on the circumstances (e.g. duration and magnitude of exposure) the health burden due to chronic exposure to air pollution may be 10 times greater than that for acute exposure, based on the relative risk ratios (WHO 2006).

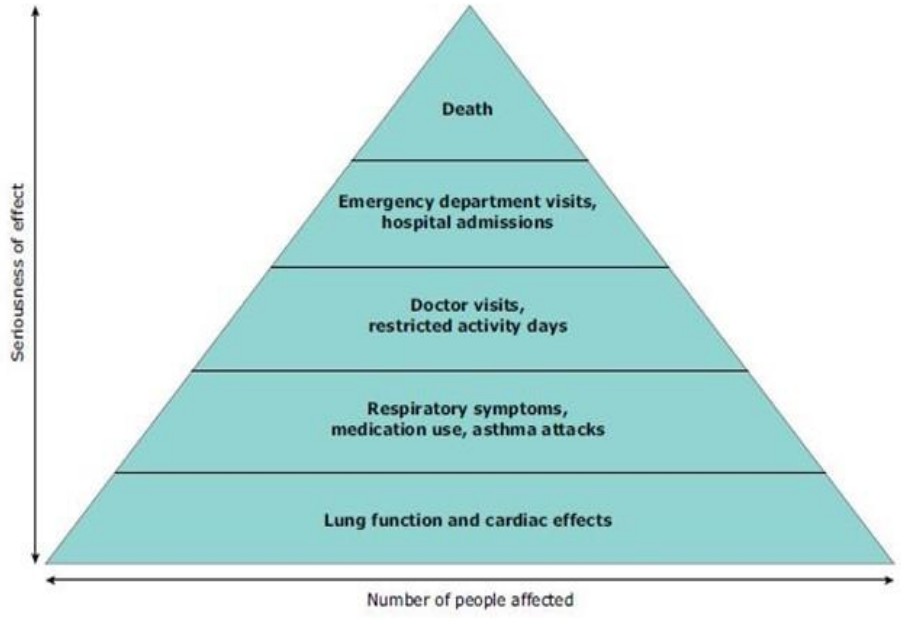
The Royal College of Physicians (**RCP**) in the United Kingdom report that:

> Research has also changed our perspective on the health risks of air pollution; an emphasis on controlling short pollution peaks from solid fuel burning has been replaced by concerns about long-term exposure to pollution from transport sources.
>
> The most-studied effects, on mortality and hospital admissions linked to short-term exposure to fine particles, capture only a small part of the range of the total health effects reported for air pollution. Population-based studies as well as modern biological science have revealed highly potent toxic effects of chronic exposure to 'modern-day pollutants', not only on the lungs but also on the heart and broader cardiovascular system. (RCP 2016)

More people are affected by less severe health effects than the proportion affected by more severe health effects (see Figure 2 for those related to PM_{10}). While there are a large number of acute/short-term cases, the fewer chronic cases incur a much greater social cost.

Note: HAPINZ 3.0 assesses health effects associated with **long-term only exposure** to air pollution in New Zealand.

Figure 2: Pyramid of PM_{10} health effects



Source: WHO (2006)

2.2 Certain people are more vulnerable to effects

Air pollution causes serious health effects. However, these impacts are not felt evenly. DANIDA (2000) identifies that people can be more vulnerable if they are:

- more **exposed** to environmental hazards
- more **sensitive** to the effects
- **less resilient** in terms of their ability to be able to anticipate, cope with or recover from the effects.

2.2.1 Exposure

For air pollution risk, the exposure is determined largely by external factors, such as the amount of time spent indoors or travelling or whether a home/office/school is located close to transport corridors or industrial facilities.

Affordable housing for low socio-economic groups is often located in areas where air quality is poor, such as near highways, in low lying valleys and in more industrialised areas. There is evidence that young children, adults and households in poverty experience increased exposure to traffic-related air pollution in particular (Barnes *et al* 2019).

Increased air pollution also makes people less likely to engage in physical activity, which of itself has wide ranging public health impacts.

2.2.2 Sensitivity

Sensitivity depends largely on internal factors such as age, health status and genetic makeup. Based on health reviews, there are groups within the population who are more affected by air pollution than others (MfE 2011a). These susceptible groups include:

- elderly people
- children (including babies, infants and unborn babies)
- people with pre-existing heart or lung disease
- people with respiratory conditions
- asthmatics
- diabetics
- pregnant women
- Māori and Pacific peoples.

Asthmatics are particularly sensitive to poor air quality. New Zealand has one of the highest prevalence of asthma in the world, with one in seven children aged 2–14 years (107,000 children) and one in nine adults aged over 15 years (389,000 adults) currently taking asthma medication (HQSC 2016). The Organisation for Economic Co-operation and Development (**OECD**) reports that New Zealand has the fourth highest hospital admission rates for asthma of OECD countries (OECD 2019).

Māori are 2.9 times and Pacific peoples are 3.7 times more likely to be hospitalised for asthma than Europeans or other New Zealanders, and people living in the most deprived areas are 3.2 times more likely to be hospitalised than those in the least deprived areas (Asthma Foundation 2016). There is also evidence of a higher prevalence of medicated asthma among Māori children (MoH 2020a).

2.2.3 Resilience

In terms of ability to cope or recover from health effects associated with air pollution, again those in low socio-economic groups are disadvantaged.

In New Zealand, both Māori and Pacific peoples have disproportionately low incomes compared to many other ethnic groups (EHINZ 2021). This means they have fewer options available to them to reduce or avoid air pollution risks.

2.3 Key air pollutants in New Zealand

Air pollution comprises a complex mixture of particles (usually referred to as particulate matter or **PM**) and gases. In New Zealand, the key pollutants of concern are PM and NO_2.

Note: HAPINZ 3.0 assesses health effects associated with **PM and NO_2 only**.

Relatively high concentrations of black carbon (**BC**), arsenic (**As**), benzo(a)pyrene (**BaP**) and lead (**Pb**) have been measured in New Zealand in the past and these pollutants were considered in the initial development of the HAPINZ 3.0 methodology. However, they were excluded from the assessment due to concerns about data availability, double-counting and robustness of exposure-response functions (Kuschel *et al* 2020).

Limited monitoring of ozone (**O_3**) and sulphur dioxide (**SO_2**) has been undertaken in New Zealand. Historically, monitored levels have been found to be low relative to other countries. Where higher levels have been recorded, these have generally been confined to localised areas. Nonetheless, due to insufficient data, these pollutants were also excluded in the HAPINZ 3.0 assessment.

2.3.1 Particulate matter

Of the common pollutants present in air pollution, the largest and best-known impacts on health (in terms of the burden on the health system and society) arise from PM_{10} and $PM_{2.5}$. Ultrafine particles (**UFP**, or particles with a size less than 0.1 µm) are of particular concern due to their ability to penetrate deep in the respiratory system and enter the bloodstream.

Sources

Particulate matter (**PM**) comes from *anthropogenic* (human-generated) sources such as burning coal, oil, wood, petrol and diesel in domestic fires, motor vehicles and industrial processes. Natural sources of PM include sea spray (marine aerosol), dust, pollens, volcanic activity and earthquakes (liquefaction dust). In most places in New Zealand, levels of PM in

the air are at their highest during winter months, due to the higher frequency of calm conditions and increased solid fuel (wood and coal) burning for home heating.

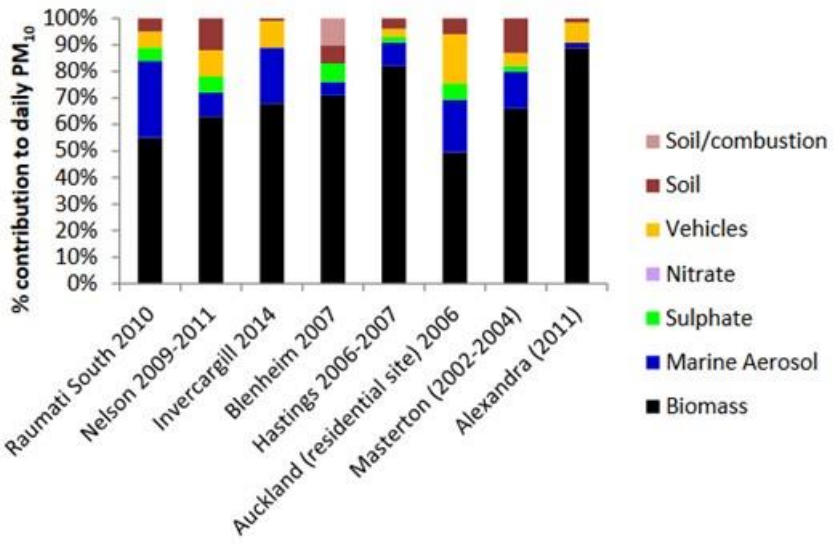
In urban atmospheres, UFP mainly arise directly from road traffic emissions (*primary* PM) but can also arise from reactions between other air pollutants (*secondary* PM), especially in high *insolation* (sunny) urban climates (Brines *et al* 2015).

New Zealand is an isolated island nation. This means that, except for rare events such as bushfires and dust storms from Australia, we generate our own pollution (MfE & Stats NZ 2018). Therefore, the composition and sources of pollution in New Zealand are quite different to other parts of the world.

In Europe, $PM_{2.5}$ urban background is usually dominated by secondary PM (Amato *et al* 2016) and the transboundary transport of air pollution is well documented. However, in New Zealand, PM speciation and source apportionment studies have found that anthropogenic PM is typically dominated by emissions from biomass burning (wood burners used for winter home heating) and motor vehicles (near busy roads and in larger cities), while natural sources of PM are dominated by marine aerosol (Davy & Trompetter 2018).

Figure 3 compares several source apportionment studies undertaken in New Zealand and shows that biomass burning typically contributes between 50% and 90% of daily wintertime PM_{10} at urban monitoring sites (Davy & Trompetter 2017). In contrast, secondary particulate typically accounts for 10 to 20% of annual particulate concentrations.

Figure 3: Comparison of wintertime source apportionment studies



Source: Davy & Trompetter (2017)

Size matters

Size matters when it comes to PM. There is no reasonable doubt that inhaling PM is harmful to human health, especially finer fractions such as PM_{10}, $PM_{2.5}$ and UFP.

Generally larger PM (between 2.5 and 10 µm) deposits in the upper airways whereas smaller PM (less than 2.5 µm) lodges in the very small airways deep in the lung. Inhaled UFP may even enter the bloodstream and reach a number of organs in the body (EFCA 2019).

Composition matters but …

Different sources emit particles with different size distributions and different chemical and biological composition. However, the mechanisms of particle toxicity are complex and still not fully understood.

In 2006, the World Health Organization (**WHO**) stated:

> While both observational and experimental findings imply that particle characteristics are determinants of toxicity, definitive links between specific characteristics and the risk of various adverse health effects have yet to be identified. (WHO 2006)

It is not yet certain which of the several classes of toxic effects observed in laboratory experiments are responsible for specific human health effects (Brook *et al* 2010). Human or animal cells exposed to particles from various sources show a range of inflammatory responses, which vary according to the source and composition of the particles. Particle characteristics such as size, concentration, metal content, potential to cause oxidation and/or immunological responses have all been shown to be important (Steenhof *et al* 2011, Degobbi *et al* 2010).

Note: Although definitive evidence for health effects associated PM composition remains inconclusive, one exception is diesel engine exhaust. In 2012, the International Agency for Research on Cancer (**IARC**), which is part of WHO, classified **diesel engine exhaust** as **carcinogenic to humans** (Group 1), based on sufficient evidence that exposure is associated with an increased risk for lung cancer (IARC 2012). They also noted a positive association (limited evidence) with an increased risk of bladder cancer (Group 1).

A year later, IARC announced that it had classified **outdoor air pollution** (irrespective of source and type) as **carcinogenic to humans** (Group 1), based on sufficient evidence that exposure to outdoor air pollution causes lung cancer (IARC 2013). As with diesel exhaust, they also noted a positive association with an increased risk of bladder cancer (Group 1). **Particulate matter**, a major component of outdoor air pollution, was evaluated separately and was also classified as **carcinogenic to humans** (Group 1).

In the latest release of the *Global Air Quality Guidelines*, WHO (2021) acknowledges this issue remains to be resolved:

> Many studies have tried to identify which sources and/or physicochemical characteristics of airborne PM contribute most greatly to toxicity. **This is a challenging area of research**, given the great heterogeneity of airborne particles, **and a definitive set of particle characteristics has yet to be identified**. However, in its 2013 review of the evidence (WHO 2013), WHO did point out that a focus on primary combustion particles, secondary inorganic aerosols and secondary organic aerosols was warranted.

WHO (2021) identifies a suite of further research needs to address uncertainties and knowledge gaps – in particular:

Study the toxicity of different sources of air pollution (e.g. tailpipe and non-tailpipe emissions, aviation and shipping emissions, specific industrial sources, wood smoke and desert dust). This includes research into the health effects of technology-driven changes in areas such as primary energy production, where mixtures of coal and biomass replace coal in places.

Note: In HAPINZ 3.0, **all PM is assumed to be equal regardless of source** in terms of the magnitude of its effect on health impacts. Nonetheless, the HAPINZ 3.0 model (see Chapter 7) has been designed to allow for differential exposure-response functions by source, should these prove warranted in future.

Health effects

The health effects of PM are predominantly respiratory and cardiovascular. The impacts range from functional changes (e.g. reduced lung function) to symptoms, impaired activities (e.g. school absenteeism, days off work), doctors' visits through to hospital admissions, reduced life expectancy and death.

2.3.2 Nitrogen dioxide

Exposure to NO_2 is an increasing concern world-wide, particularly in transport-impacted cities. Evidence of a causal relationship between short-term NO_2 and respiratory impacts has strengthened, while remaining suggestive for cardiovascular disease and mortality. New studies also point towards stronger associations of long-term exposure with mortality (respiratory, cardiovascular and all cause) as well as for lung carcinogenicity. However, much of this evidence is based on single pollutant models and the effects recorded for NO_2 may represent those for other traffic-related pollutants (such as UFP, $PM_{2.5}$, CO, BC and polycyclic aromatic hydrocarbons) (WHO 2016a).

Sources

Oxides of nitrogen (**NO_X**) primarily come from combustion sources, when fuels are burnt in the presence of air. The main components of NO_X are nitric oxide (**NO**) and NO_2. NO readily oxidises in the atmosphere to produce NO_2. Motor vehicles are the biggest source of NO_X contributing to human exposure to NO_2 in most urban areas. Other sources in New Zealand include shipping, industry, and electricity production (MfE & Stats NZ 2018).

For example, looking at emissions in the Auckland region in 2016, transport sources comprised just under 86% of all NO_X emissions across the region, with 13% from industry and just over 1% from domestic sources such as home heating (Xie *et al* 2019). However, focussing the analysis to emissions which impact urban areas, the contribution of transport sources is likely closer to 98%, with on-road motor vehicles alone at 88%. The motor vehicle contribution is dominated by diesel vehicles (64% versus 36% petrol), with heavy diesel vehicles contributing 61% of emissions while being only 4% of the fleet (Sridhar & Metcalfe 2019).

Health effects

NO_2 is a gas that causes increased susceptibility to infections and asthma. It reduces lung development in children and has been associated with increasingly more serious health effects, including reduced life expectancy (COMEAP 2015).

The effects of NO_2 are potentially significant. For example, the European Environment Agency (**EEA**) estimates that 79,900 people died prematurely across Europe in 2015 due to long-term exposure to NO_2 compared to an estimated 422,000 premature deaths due to long-term exposure to $PM_{2.5}$ (EEA 2018).

2.4 Typical assessment approach

2.4.1 Steps involved

The effects of air pollution on health are typically assessed in a stepwise process as shown in Figure 4. For each area under assessment (e.g. a census area unit, **CAU**), the health impacts are generally calculated as follows:

$$\textbf{Health Effects (cases)} = \textbf{Cases (total)} \times \textbf{PAF}$$

where:

Health effects (cases) are the number of deaths, hospital admissions or restricted activity days (depending on the health outcome being assessed) due to air pollution.

Cases (total) is the total number of health cases (deaths, hospital admissions, or for restricted activity days, population) in the area of interest.

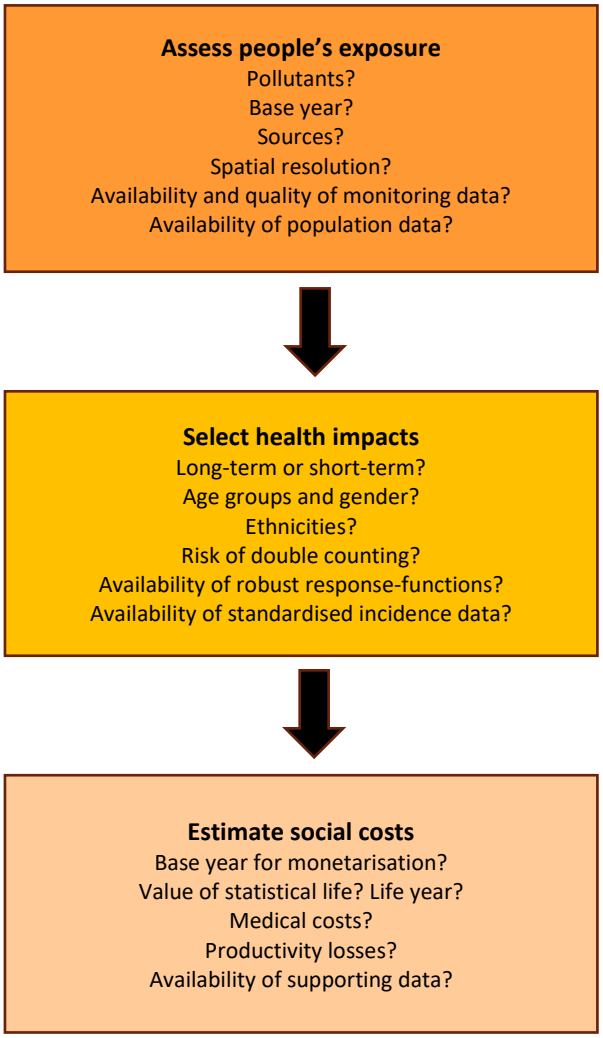
PAF (population attributable fraction) is the estimated percentage of total health cases that are attributable to the air pollution exposure.

The PAF is calculated using the **exposure–response function** (the relative increase in the health effect for every increment of air pollution[7], e.g. 1.11 for every 10 µg/m^3 of annual average PM_{10}) and the **exposure** (the average pollution concentration in the area of interest, e.g. an annual average PM_{10} concentration of 15 µg/m^3).

This approach estimates the health effects that would be prevented if exposure to the pollutant (e.g. PM_{10}) was at the minimum risk level possible, recognising that there is no safe threshold for most air pollutants.

[7] A relative risk of 1.11 means the risk increases by 11% per pollution increment, in this case per 10 µg/m^3 of annual average PM_{10}.

Figure 4: Typical steps involved in an assessment of air pollution health effects



The social costs of air pollution are then calculated as follows:

$$\textbf{Social Costs} = \textbf{Health Effects (cases)} \times \textbf{Cost per case}$$

In simple terms, we combine the health effects cases estimated as per the previous formula (e.g. the number of premature deaths) with published health-cost data (e.g. the latest value of a statistical life) to estimate costs.

Results can be aggregated and reported for larger urban areas (such as towns and cities) or management areas (such as regions or airsheds) depending on physical and political boundaries.

The information is used in cost-benefit analyses for a range of applications, such as:

- weighing the benefits of health improvements against the costs of (various) air pollution reduction initiatives
- evaluating the effectiveness of existing policy initiatives (backcasting)

- assessing the likely effects of current population and business as usual trends (forecasting)
- developing targeted strategies for reducing air pollution exposure of particularly vulnerable groups in the population.

Health impact assessments combine information on exposure of the population concerned (often based on air pollution monitoring) with exposure-response functions to provide an estimate of the effect. In practice, because air pollution exposures are complex, it is necessary to simplify the assessment of air pollution effects by using summary indicators of exposure.

For example, air pollution contains a complex mixture of gases and particles. Consequently, the adverse health impacts observed in epidemiological studies, which are attributed to an *individual* air pollutant, may actually be due to other pollutants in the mixture. This means that the air pollutants investigated in epidemiological studies may be *proxies* for the air pollutant mixture (WHO 2016a).

The uncertainties involved in extrapolating health effects from one population to another are minimised when the exposures are estimated, and summarised, using comparable methods.

2.4.1 Best practice guidelines

The WHO recommends air pollution health risk assessments be undertaken in accordance with the following principles (WHO 2014):

- Health effects assessment should address an area of uncertainty and an unmet need for information (particularly with respect to social costs).
- The assessment reflects the core WHO value of the “right to health”.
- The process of undertaking an assessment is explicit and transparent so the end user can see how health impacts and social costs were selected and calculated.
- The process of undertaking an assessment is multidisciplinary and includes all relevant expertise and perspectives, including input from stakeholders.
- The evidence used to develop the assessment is publicly available.
- Assessment outputs (in the form of exposure and effects models) can be implemented in, and adapted to, local settings and contexts.
- Assessment communication products should be tailored to the general public.

Note: The following chapters describe in detail the methodology HAPINZ 3.0 follows for each of the steps shown in Figure 4 and how the assessment gives effect to the WHO best practice principles above.

3. Evaluating exposure

Understanding exposure is critical to understanding potential health impacts. This is because the length of time people are exposed to air pollutants, the concentration of the pollutants and the sensitivity of individuals exposed combine to determine the likelihood and magnitude of resultant health effects.[8]

This chapter summarises the methodology used for assessing exposure in the HAPINZ 2.0 study, reviews developments that have occurred since in data availability and presents the approach we developed for the HAPINZ 3.0 update.

3.1 Approach used in HAPINZ 2.0

HAPINZ 2.0 utilised the following approach to assess exposure to air pollution in New Zealand:

- The **base year** was selected to be 2006. This year aligned with the 2006 Census so population data could be matched with associated ambient monitoring and morbidity/mortality datasets.
- **Particulate matter (PM_{10})** was used as the best available indicator of air pollution exposure due to the wealth of monitoring data available (covering 73% of the population), and peer-reviewed, published exposure-response functions developed from New Zealand and international epidemiological studies.
- **Actual monitoring data** were used in preference to modelling estimates and **averaged for 2006 to 2008** to reduce the influence of year-to-year variability in meteorology.
- For areas with no ambient air quality monitoring, annual concentrations were estimated using comparisons with monitored areas with the same urban/rural classification[9] and land use regression techniques (i.e. domestic and industry contributions, if applicable, based on housing density and available industrial emissions inventory data).
- **Results were estimated by census area units (CAU)** as at 2006 (1,919 in total) but were able to be aggregated by airshed (71), by urban area (139), by territorial authority (**TA**) such as a district or city council (74)[10], by regional council (16), or nationally.

[8] For pollutants where causality has been established

[9] As defined by Stats NZ. Definition available on request from info@stats.govt.nz

[10] Note the number of TAs is based on those that were in existence for the 2006 census. 2010 saw an amalgamation of the eight Auckland councils so the number of TAs reduced to 67 (excluding the Chatham Islands).

3.2 Developments since HAPINZ 2.0

Since HAPINZ 2.0, significantly more ambient monitoring has been undertaken (especially of $PM_{2.5}$) and new exposure models have been developed to address knowledge gaps (e.g. road dust and NO_2).

3.2.1 PM_{10} and $PM_{2.5}$ monitoring

Air quality in New Zealand is managed by **airsheds**, which are areas delineated by regional councils for the purpose of managing air quality. The term is analogous to catchments or watersheds used in the management of freshwater. Regional councils are required to monitor air quality in their airsheds and compare the results against national environmental standards (**NESAQ**) for air quality (MfE 2011b).

New Zealand has good representative ambient PM_{10} monitoring data relative to other countries as demonstrated in summary data from the WHO's *Global Burden of Disease* (**GBD**) 2016 study presented in Table 3 (Ostro *et al* 2018; GBD 2017). New Zealand monitors more locations than Sweden and Australia; countries with similar air quality but more than two and four times our population, respectively.

Table 3: Number of monitoring locations per country used in Global Burden of Disease 2016

Country	#	Country	#	Country	#
Afghanistan	2	Hungary	17	Oman	1
Albania	1	Iceland	3	Pakistan	5
Andorra	1	India	122	Panama	1
Argentina	1	Indonesia	1	Paraguay	1
Australia	**37**	Iran	25	Peru	1
Austria	60	Iraq	1	Philippines	8
Bahrain	5	Ireland	8	Poland	154
Bangladesh	8	Israel	40	Portugal	12
Belgium	43	Italy	236	Qatar	2
Bhutan	1	Jamaica	6	Republic of Korea	16
Bolivia	2	Japan	15	Romania	42
Bosnia & Herzegovina	2	Jordan	4	Russia	1
Brazil	45	Kenya	1	Saudi Arabia	7
Bulgaria	25	Kuwait	11	Senegal	1
Cameroon	3	Latvia	4	Serbia	3
Canada	126	Lebanon	4	Singapore	1
Chile	23	Liberia	2	Slovakia	21
China	210	Lithuania	9	Slovenia	13
Colombia	18	Lux	3	South Africa	13
Costa Rica	7	Madagascar	1	Spain	225
Croatia	5	Malaysia	6	Sri Lanka	1
Cyprus	5	Maldives	1	**Sweden**	**19**
Czech	49	Malta	4	Switzerland	9
Denmark	5	Mauritius	4	Thailand	26
Ecuador	9	Mexico	9	Yugoslavia-Macedonia	4

Country	#	Country	#	Country	#
Egypt	2	Monaco	1	Tunisia	4
El Salvador	1	Mongolia	1	Turkey	81
Estonia	4	Montenegro	5	Uganda	1
Finland	24	Morocco	7	Tanzania	1
France	315	Myanmar	14	United Arab Emirates	5
Georgia	2	Nepal	1	United Kingdom	51
Germany	161	Netherlands	24	United States of America	372
Greece	10	**New Zealand**	**40**	Uruguay	1
Guatemala	1	Nigeria	12	Venezuela	1
Honduras	1	Norway	12		

Source: Ostro *et al* (2018)

Ambient PM_{10} air quality data, collected using approved regulatory methods for the purposes of the NESAQ, are available for 67 out of New Zealand's 89 airsheds[11] for the last 13 years (i.e. 2006 to 2018). Between 2015 and 2017, 49 monitoring sites around the country had three full years of data and 59 monitoring sites had at least one full year of PM_{10} monitoring data.

Monitoring for $PM_{2.5}$ has progressively increased since 2010 and is now undertaken in 22 airsheds covering nine regions: Northland, Auckland, Waikato, Bay of Plenty, Hawke's Bay, Wellington, Nelson, Marlborough and Canterbury. Between 2006 and 2018, 37 monitoring sites around the country had at least one full year of $PM_{2.5}$ monitoring data. Whilst not mandated in the regulations, regional councils also collect $PM_{2.5}$ data using approved regulatory methods.

The $PM_{2.5}$ monitoring sites are all co-located with PM_{10} monitoring. Source apportionment analysis has also been undertaken at a number of these sites. This means that there is a considerable amount of data for development of $PM_{2.5}$ to PM_{10} ratios. These allow for the indirect estimation of $PM_{2.5}$ concentrations in airsheds with PM_{10} monitoring. $PM_{2.5}$ / PM_{10} ratios are used internationally to fill in gaps in monitoring records, recognising that widespread $PM_{2.5}$ monitoring is a more recent development (WHO 2016b).

3.2.2 NO_2 monitoring

Health effects of NO_2 were not assessed in HAPINZ 2.0 because insufficient ambient air quality monitoring data were available to establish exposure.

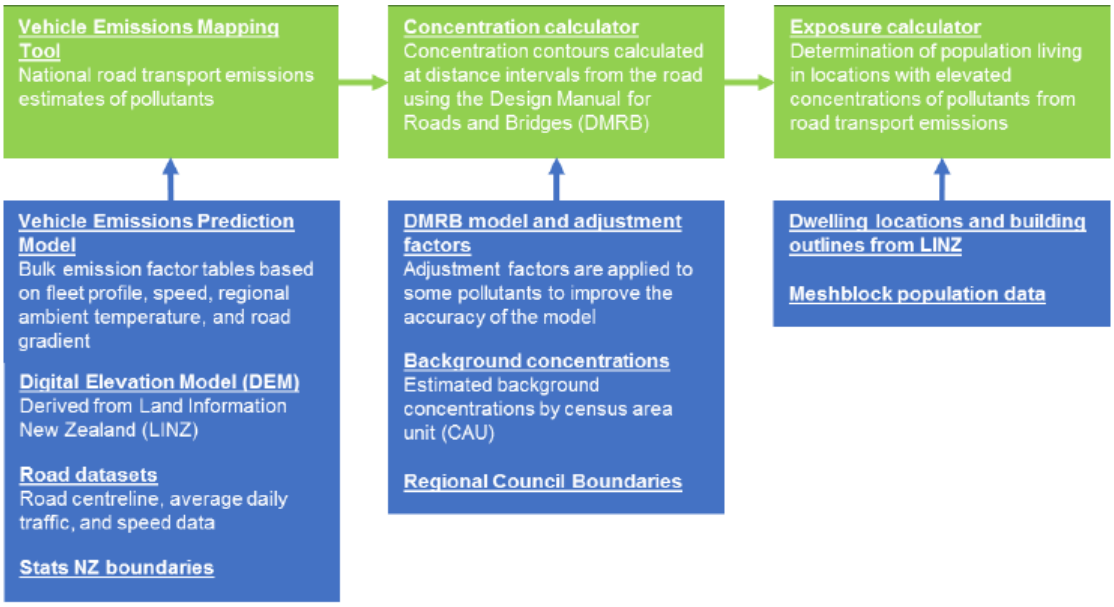
Since HAPINZ 2.0, NO_2 monitoring has increased considerably. Regional Councils monitor NO_2 continuously, using regulatory methods, at 15 sites around New Zealand. In addition, Waka Kotahi operates a network of passive monitoring sites at 129 locations as at end 2016 (NZTA 2020). This monitoring dataset has enabled development of high resolution NO_2 exposure assessments which are incorporated into the National Vehicle Emissions Dataset (**NVED**) exposure tool.

[11] This includes 73 gazetted airsheds and 16 regional council airsheds.

3.2.3 National vehicle emissions mapping and exposure tools

Waka Kotahi has developed a suite of tools to provide for assessment of emissions and exposure to traffic pollution (as shown in Figure 5).

Figure 5: Inputs and outputs of the VEMT and the NVED concentration and exposure calculators



Source: T&T (2021)

VEMT

The Vehicle Emissions Mapping Tool (**VEMT**)[12] automates calculation of both harmful air pollutants and greenhouse gas emissions resulting from motor vehicles and can be applied to all public roads throughout New Zealand.

The tool is housed in a geographical information system (**GIS**) framework so that data can readily be presented as maps. The tool extracts road and activity data from Waka Kotahi's information technology systems to build a detailed set of input variables for the emission calculations. A matrix of vehicle emission factors, extracted from the New Zealand Vehicle Emission Prediction Model (**VEPM**), is used with the input variables to calculate the mass of pollutant per length of roadway.

Maps can be produced that allow users to explore how vehicle emissions vary at a range of spatial scales from national to local. The tool has flexibility to be used for analyses including:

- Developing inventories of harmful air pollutant and greenhouse gas emissions
- Reporting trends in emissions over time
- Supporting investigations into the health effects of exposure to vehicle emissions.

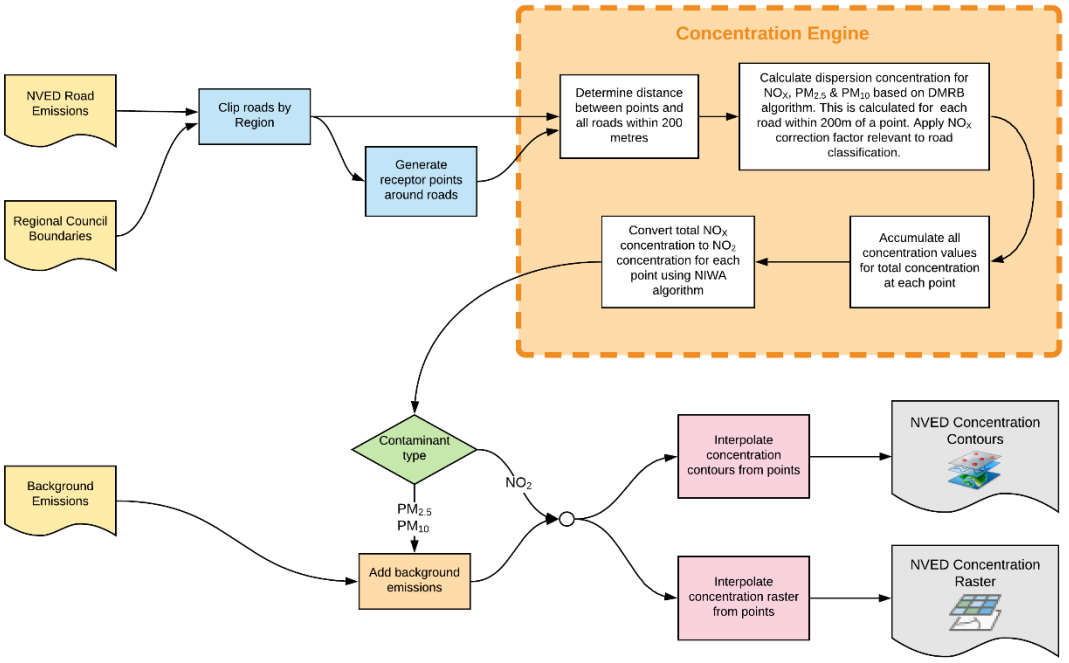
[12] This tool has been developed for internal NZTA use but may become available for the public in future. For a description see https://www.nzta.govt.nz/roads-and-rail/highways-information-portal/technical-disciplines/air-quality-climate/planning-and-assessment/vehicle-emissions-mapping-tool/

Validation of the tool has involved comparing output data with local authority air pollutant emissions inventories. The results demonstrate that the tool can be used as a reliable and consistent means of generating vehicle emission datasets at various geographical scales throughout New Zealand (Jacobs 2016).

NVED outputs

Output from VEMT is used to generate annual National Vehicle Emissions Datasets (**NVED**) (Jacobs 2018) and as input to the NVED Concentration and NVED Exposure calculators (see Figure 6).

Figure 6: Stages involved in the NVED concentration calculator



Source: T&T (2021)

The NVED Concentration calculator converts emissions from VEMT to **roadside concentrations** using the algorithm developed as part of the *Design Manual for Roads and Bridges* (**DMRB**) (The Highways Agency 2007). The DMRB algorithm calculates pollutant concentrations based on the distance from the centre of the road. The contribution from all roads within 200m are summed to give the roadside concentrations at each location. The algorithm is used to calculate annual NO_X, $PM_{2.5}$ and PM_{10}. Total NO_X at each receptor location is adjusted depending on the adjacent road classification. The NO_X concentration is then converted to NO_2 using the NIWA NO_X/NO_2 conversion method (Longley & Sommervell 2019).[13]

[13] The NIWA conversion method is based on a review of all NO_2 monitoring data – including passive sampler data which has not been adjusted to a reference method equivalent.

Representative annual **background concentrations** are assumed in all locations where predicted roadside contributions from the DMRB are lower than the representative background concentration.

The representative annual background NO_2 concentrations were developed from monitoring data, where available, and supplemented with:

- a modelled dataset based on the NIWA Traffic Impact Model (the NIWA TIM model) (Longley 2020)
- default representative concentrations based on urban or rural classification.

This information is used to generate contour-based GIS datasets of concentrations for annual average daily mean NO_2, PM_{10} and $PM_{2.5}$ within 200m of roadways, which can be overlaid with dwelling locations and population data in the NVED Exposure calculator. The NVED Exposure calculator is essentially a land use regression model (using traffic emissions density).

3.2.4 Road dust exposure model

In 2015, Waka Kotahi commissioned a preliminary investigation into the impacts that dust emissions from unsealed roads in Northland had on people and the possible efficacy of dust mitigation measures (Bluett *et al* 2016).

Waka Kotahi is currently developing a tool to estimate exposure to dust from unsealed roads and is broadening the investigations to a comprehensive assessment of the health effects of road dust[14].

Note: The impact of road dust was not able to be included in HAPINZ 3.0 as the results of the Waka Kotahi project were still pending at time of writing. High concentrations have been recorded in localised areas previously but there is insufficient information to undertake a New Zealand-wide assessment.

3.2.5 Other exposure assessment methods

In HAPINZ 2.0, actual monitoring data were used to estimate exposure to PM_{10} in preference to modelling estimates.

A review by Health Canada concluded that ambient measurements are a reasonable proxy for exposure (Health Canada 2016). However, other methods are available for assessment of exposure as shown in Table 4 (Hoek 2017).

Hoek (2017) discusses the merits of the "monitoring" method where monitoring data from typically one or a few monitors are used to estimate exposure. These include low cost, consistency of monitoring methods, and often a long period of monitoring. **The use of monitoring further avoids the problems of models with limited or uncertain validity. Hoek states that the main limitation of this approach is the lack of characterisation of intra-urban contrasts related to traffic emissions and other local sources.**

[14] Assessing the impact of road dust and other forms of non-exhaust PM will remain important as the New Zealand fleet decarbonises as electric vehicles will still generate road dust.

Table 4: Methods to assess long-term average outdoor air pollution exposure studies

Method	Principle	Comment
Monitoring	Measured value from surface-monitoring stations directly assigned to participants	Nearest station (within a certain distance) or average of all stations in a city
Interpolation	Assign interpolations of measured values from monitoring stations, using ordinary kriging, inverse distance weighing or other geo-statistical methods.	Applied for ozone and $PM_{2.5}$, pollutants with limited local variation
Satellite monitoring	Surface $PM_{2.5}$ and NO_2 concentrations obtained by combining measured column concentration and vertical distribution of a chemical transport model (CTM).	Combines remote sensing and CTM for vertical gradient; often supplemented with additional land use and traffic data
Indicators of exposure	Traffic intensity nearest to the road, distance to a major road	Not a quantitative pollution estimate
Land use regression modelling	Fixed site and more recently mobile monitoring to develop empirical models using traffic, population and land use predictor variables	Spatial and spatiotemporal models; increase in predictor variables such as satellite and dispersion/chemical transport models
Dispersion/chemical transport modelling	Modelling of dispersion of emissions from source to receptors using deterministic models	Recently on a finer spatial scale.

Source: Hoek (2017)

Intra-urban contrasts - PM

Monitoring of $PM_{2.5}$ in Auckland (New Zealand's largest urban area) records similar concentrations of $PM_{2.5}$ at comparable locations. For example, Figure 7 presents monthly $PM_{2.5}$ concentrations for two background sites and three urban (traffic-influenced) sites (Talbot *et al* 2017).

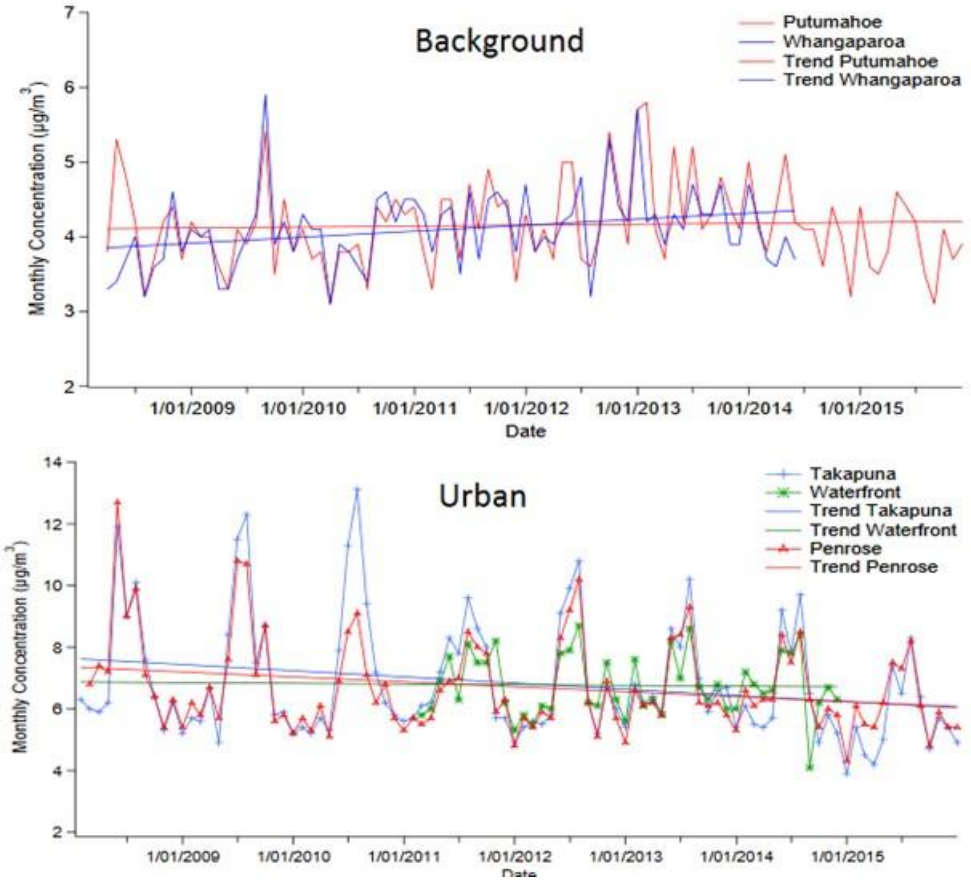
These results support the assumption that $PM_{2.5}$ monitoring data from a single site (or the average of multiple sites) provide a *reasonable* estimate of background $PM_{2.5}$ across airsheds. There are some limitations in this assumption, for example:

- PM_{10} and $PM_{2.5}$ concentrations will be elevated in the vicinity of local sources of air pollution.
- There are localised variations in the concentration of air pollution in some locations due to complex topography and meteorology.

Intra-urban contrasts introduce some uncertainty in the exposure assessment. Even if population exposure is well-estimated, individual exposures can vary substantially due to differences in concentrations in different places and individual activity patterns. To accurately assess population exposure, personal monitoring would be required.

Ideally, the most appropriate method and resolution for exposure assessment reflects the methods and resolution used in the original epidemiological research.

Figure 7: Monthly $PM_{2.5}$ concentrations at Auckland background and urban monitoring sites



Source: Talbot *et al* (2017)

Intra-urban contrasts – NO_2

In urban areas, NO_2 concentrations are very dependent on proximity to roads. This means that high-resolution exposure estimates are needed to make meaningful assessment of health effects.

As mentioned for PM, the exposure assessment should ideally use the same spatial scale and resolution as the original epidemiological research. However, authors of a recent review of methodologies for NO_2 exposure assessment in the EU concluded that the spatial scale of epidemiological studies is not always clear to start with or is based on meta-analysis combining different studies (Bino *et al* 2017).

Considerable uncertainty exists in exposure-response functions for NO_2 (see Chapter 5). Detailed sensitivity analysis of assessment methodologies found that the exposure-response functions are the dominant source of uncertainty in assessing the effects of NO_2 (Bino *et al* 2017). They also found that spatial scale is a significant source of uncertainty for a resolution of less than 1 km and recommend 100 m for an EU-wide assessment. However, the exposure-

response functions we report here, based on cohort studies carried out in HAPINZ 3.0, are themselves affected by misclassification of exposure.

3.3 What we did in HAPINZ 3.0

For the HAPINZ 3.0 update, we relied primarily on the results of monitoring to estimate exposure to PM_{10} and $PM_{2.5}$. For NO_2, we utilised concentration estimates from the Waka Kotahi NVED exposure tool.

Note: In HAPINZ 2.0, regression analysis was used in some locations (including Auckland) to estimate PM_{10} concentration by CAU based on emissions density. For HAPINZ 3.0, we repeated this analysis based on estimated 2013 data to see whether this approach might still be useful. However, we found that emissions density by CAU no longer correlated well with measured PM_{10} (see Appendix A) so we changed our approach to that indicated above.

3.3.1 Base year

The base year for the HAPINZ 3.0 exposure assessments was 2016 (with data typically averaged over 2015-2017). Due to the need to align the various population, health and monitoring datasets, we collated and analysed all data by CAU based on the 2013 Census boundaries (**CAU2013**).

Note: 2016 is not a census year, unlike the base years in HAPINZ 1.0 and HAPINZ 2.0.

For the source attribution method, we extended the dataset to 2018 (i.e. 2015-2018) to gather sufficient monthly data to compare with source apportionment results in order to refine the contributions of the different sources (see Chapter 4). More extensive monitoring records (2004-2018) were utilised in the detailed cohort study undertaken to derive New Zealand-specific exposure-response functions (see Chapter 5).

We used the full datasets to assess long-term trends, examine relationships and inform expert judgements to estimate exposures for the entire New Zealand population.

3.3.2 Population data

All population data were sourced from Stats NZ and is the **estimated resident population** as at 30 June 2016, based on 2018 Census results and using the geographic boundaries of census area units as at 1 January 2013 (CAU2013). Data for the following age groups were provided by Stats NZ:

- All ages
- 0-14 years
- 0-18 years
- 30+ years.

Stats NZ provided the estimated resident population counts for total response ethnic groups (for all ages and 30+ years) for 2013 and 2018 (as part of a customised data request).

We used linear interpolation to derive the estimated resident population as at 30 June 2016, for the following ethnic groups:

- Māori 30+ years
- Pacific peoples 30+ years.

Note: The population data provided by CAU had been rounded to the nearest 5 or 10 (when population totals were less than 2,000). For this reason, **the individual figures used in HAPINZ 3.0 do not sum exactly to official published totals**. For example, the total estimated resident population for New Zealand (as at 30 June 2016) in HAPINZ 3.0 is 4,713,415 people versus the official Stats NZ figure of 4,714,100. Nonetheless, this discrepancy is very minor as the HAPINZ 3.0 total is 99.985% of the Stats NZ published total.

While there are 2,012 CAU2013s for 2016, only 1,900 have non-zero populations and are in within designated airsheds. Regardless, the HAPINZ 3.0 model includes all CAU2013s.

3.3.3 PM_{10} exposure

We collated annual ambient PM_{10} data, measured using approved regulatory methods for the purposes of the NESAQ, for 101 monitoring sites over the last 13 years (i.e. 2006 to 2018). We only considered sites with more than 75% valid data for each year to ensure that annual PM_{10} monitoring averages were robust. The dataset is provided in the *HAPINZ 3.0 Health Effects Model - PM10 data* worksheet.

Our 13-year dataset included annual PM_{10} monitoring data for 68 out of New Zealand's 89 airsheds.[15] This provided robust annual exposure estimates, for at least one year, for airsheds representing 84% of New Zealand's population.

The approaches we took to assess PM_{10} exposure depended on data availability as follows.

Annual PM_{10} in CAUs and airsheds with monitoring data

Monitoring data were used to estimate exposure for areas with available data as follows:

- The monitored concentration was applied to the CAU where monitoring was undertaken.
- Where the monitoring was undertaken within an airshed, the monitored concentration was generally applied to the entire airshed.
- For airsheds with more than one monitor, the most representative site(s) were selected as shown in Table 5.

The base year for this national assessment was 2016. We estimated the annual average PM_{10} for this base year using monitoring data collected over the three-year period 2015–2017 (i.e. an average of the base year with a year either side). Between 2015 and 2017, 49 monitoring sites around the country recorded three full years of data. **The three-year datasets were used to provide robust estimates of annual PM_{10} exposure for approximately 65% of New Zealand's population**.

[15] Comprising 73 gazetted airsheds and 16 regional airsheds

Table 5: Monitoring site(s) selected in airsheds with more than one monitor

CAUs in gazetted airshed	Monitoring site(s) selected to represent the CAUs
All regions Monitored CAUs	Monitoring site in the CAU
Auckland: CBD (Auckland Central, East and West CAUs)	Queen St
Auckland: Harbourside	Waterfront
Auckland: Newmarket, Epsom North, Grafton East & West, Newton, Eden Terrace, Arch Hill	Khyber Pass
Auckland: All other unmonitored CAUs in urban airshed	Average of Penrose, Takapuna, Henderson, Pakuranga, Glen Eden & Botany Downs
All unmonitored CAUs in the **Christchurch Airshed**	St Albans
All unmonitored CAUs in the **Hamilton Airshed**	Average of Claudelands and Bloodbank
All unmonitored CAUs in the **Tauranga Airshed**	Otumoetai
All unmonitored CAUs in the **Masterton Airshed**	Average of Masterton West and Masterton East

Where monitoring **data were available, but not covering the full 2015–2017 three-year period**, we estimated annual PM_{10} exposure for 2016 in the following in order of preference:

- Used **only two or fewer years of available monitoring data** (e.g. Te Awamutu is the average of 2015 and 2016).
- Used **2018 monitoring data** to represent 2015–2017 period (e.g. Whakatane has monitoring available for 2018 only).
- Used **historical monitoring data to benchmark with another airshed** in the same region. The PM_{10} concentration in the airshed with historical monitoring results was then assumed to be the same as the PM_{10} concentration in the benchmarked airshed for 2016.

  For example, the PM_{10} concentration in Pukekohe was 15.2µg/m^3 in 2007-2008 (two-year average), which was similar to the average across representative Auckland urban airshed monitoring sites in 2007-2008 of 15.3µg/m^3. Monitoring results from the Auckland Urban Airshed have shown a downward trend since 2008, with an average across representative sites of 13.2µg/m^3 for 2015-2017. Assuming it is likely that a similar downward trend has occurred in Pukekohe, the Auckland Urban Airshed monitoring results from 2015-2017 were used for the Pukekohe airshed.
- Used **older available monitoring data** to represent 2015–2017 period. We considered it was better to use site-specific data, where available, even if that information was dated. However, this approach was only used if trends suggested there had been no significant change in annual average PM_{10} between the actual period monitored and the 2015–2017 period. For example, Lawrence is based on monitoring undertaken in 2011 because concentrations in other small Otago towns (Arrowtown, Alexandra and Mosgiel) show no consistent trend since 2011.

The above approaches using **partial datasets were used to estimate annual PM_{10} exposure for 19% of New Zealand's population**.

Annual PM_{10} in airsheds with no monitoring data

For unmonitored airsheds, the PM_{10} concentration was **based on monitoring from other airsheds in the same region** as follows:

- Tuakau, Paeroa, Te Aroha, Matamata and Otorohonga were assigned the value from the Morrinsville site (11.6 µg/m^3). This is comparable to the average concentration across all monitoring sites in Waikato (11.9 µg/m^3), and with the measured concentration in Te Awamutu (11.8 µg/m^3), Cambridge (11 µg/m^3) and Hamilton (11.3 µg/m^3 at Claudelands and 12.5 µg/m^3 at Bloodbank monitoring sites). Morrinsville appears to be representative of urban areas in Waikato.
- Towns in Otago are grouped into four airshed categories. Unmonitored towns in Otago airsheds were assigned the average of monitoring values across each of the airshed groups, with the exception of Dunedin. For example, unmonitored Otago 3 airshed towns were assigned the average of all monitoring results across Otago 3 airsheds (excluding Dunedin). All Dunedin CAUs within an airshed were assigned the monitoring value for Dunedin.
- All other airsheds were assigned the lowest monitoring value from comparable monitored locations in their region. This included Whitianga and Whangamata (assigned 8.5 µg/m^3 from Thames), Keri Keri and Dargaville (assigned 13.7 µg/m^3 from Whangarei), Wellsford, Snells Beach, Riverhead, and Maraetai (assigned 11.6 µg/m^3 from Botany Downs), Whirinaki (assigned 13.2 µg/m^3 from Napier), and Kapiti Coast (assigned 10.4 µg/m^3 from Upper Hutt).

Annual PM_{10} in areas outside gazetted airsheds

Areas outside airsheds were assigned a monitoring value aligned to Stats NZ urban/rural classifications (Stats NZ 2017) as shown in the examples listed in Table 6.

Table 6: Example monitoring sites corresponding to Stats NZ urban/rural classifications

HAPINZ 3.0 classification	StatNZ description	Population	Example monitoring site
Urban	Main urban areas	30,000 or more residents	Takapuna, Auckland
Urban	Secondary urban areas	10,000-29,999 residents	Tokoroa, Waikato
Urban	Minor urban areas	1000 to 9,999 residents	Alexandra, Otago
Rural	Rural centre	300 to 999 residents	Wallacetown, Southland
Rural	Other*		Pongakawa, Bay of Plenty

Note: Other* includes all areas that are not urban – including: rural (including some off-shore islands), inland water not in urban, inlet, and oceanic areas.

PM_{10} in urban areas outside airsheds

For urban areas outside gazetted airsheds, we estimated annual PM_{10} exposure **based on the lowest measured value from an urban area in the region**, with the following exceptions:

- In Waikato, the 2nd lowest PM_{10} concentration of 9.9 µg/m^3 from Huntly was used. The lowest concentration of 8.5 µg/m^3 from Thames was unusually low (being the 3rd lowest concentration measured across New Zealand) and was considered unlikely to be representative of urban areas in Waikato.

- No monitoring data are available for Taranaki for the period of interest. However, historical monitoring in New Plymouth showed similar concentrations to Hamilton so the PM_{10} concentration for Hamilton was used for all urban areas in Taranaki.
- Tasman only has one monitoring site located in Richmond. The PM_{10} concentration was relatively high (17.7 $\mu g/m^3$) and was considered unlikely to be representative of other urban areas outside the Tasman airshed. Therefore, the lowest PM_{10} concentration across all Nelson and Tasman airsheds was assumed (11 $\mu g/m^3$ from Nelson Stoke).
- In Southland, the PM_{10} concentration from Edendale was used (10.3 $\mu g/m^3$). This is higher than concentrations in Te Anau (5.9 $\mu g/m^3$) and Bluff (7.9 $\mu g/m^3$), which were the lowest and 2nd lowest measured concentrations in New Zealand respectively, but these were considered unlikely to be representative of urban areas in Southland.

This approach assumes that air quality outside designated airsheds is better than air quality inside airsheds and is consistent with the approach taken in HAPINZ 2.0.

PM_{10} in rural areas outside airsheds

Only two rural monitoring sites across New Zealand have 3-year average PM_{10} data representative for 2016 - Marsden Point and Patumahoe. However, Marsden Point is an industrial location (home to New Zealand's only oil refinery and North Port as well as other heavy industry) so was not considered representative of other rural locations.

Annual average PM_{10} in Patumahoe has not significantly changed over the 13 years ending 2018. Another rural monitoring site at Pongakawa ceased monitoring in 2006. Based on the stability in the Patumahoe results, we assumed that Pongakawa had also not significantly changed since 2006.

In the Auckland Region, all rural areas outside gazetted airsheds were assigned 11.3 $\mu g/m^3$ from Patumahoe (2015-2017 average). For the rest of New Zealand, rural areas outside gazetted airsheds (including rural centres and other areas) were assigned 9.2 $\mu g/m^3$ from Pongakawa (2004-2006 average), which is the lowest measured annual average concentration in a rural area. This is the same value that was assumed in HAPINZ 2.0.

3.3.4 $PM_{2.5}$ exposure

We collated annual ambient $PM_{2.5}$ data, measured using approved regulatory methods for the purposes of the NESAQ, for 36 monitoring sites over the last 13 years (i.e. 2006 to 2018). We only considered sites with more than 75% valid data for each year to ensure that annual $PM_{2.5}$ monitoring averages were robust. The dataset is provided in the *HAPINZ 3.0 Health Effects Model - PM2.5 data* worksheet.

The approaches we took to assess $PM_{2.5}$ exposure depended on data availability as follows.

Annual $PM_{2.5}$ in CAUs and airsheds with monitoring data

Monitoring data were used to estimate exposure for areas with available data. The methodology adopted was consistent with that used for assigning PM_{10} monitoring values to monitored areas (see section 3.3.3) as follows:

- The monitored concentration was applied to the CAU where monitoring was undertaken.
- Where the monitoring was undertaken within an airshed, the monitored concentration was generally applied to the entire airshed.
- For airsheds with more than one monitor, the most representative site(s) were selected as shown in Table 5 (for PM_{10}). The exception was the Auckland Urban Airshed where the $PM_{2.5}$ concentration was calculated from the Auckland Urban Airshed PM_{10} concentration multiplied by the average $PM_{2.5}$ / PM_{10} ratio from Penrose, Takapuna, Kingsland and Whangaparaoa.

Between 2015 and 2017, 10 monitoring sites around the country recorded three full years of data. **The three-year datasets were used to provide robust estimates of annual $PM_{2.5}$ exposure for approximately 43% of the population.**

Where monitoring **data were available, but not covering the full 2015–2017 three-year period**, we estimated annual $PM_{2.5}$ exposure for 2016 in the following in order of preference:

- Used **only two or fewer years of available monitoring data** (e.g. Lyttelton is the average from 2016 only).
- Used **more recent available monitoring data to supplement** 2015–2017 period (e.g. Tokoroa Airshed is the average of 2017 and 2018).
- Used **2018 monitoring data** to represent 2015–2017 period (e.g. Napier Airshed is the average for 2018 only).
- Used **site-specific $PM_{2.5}$ / PM_{10} ratio**s (based on monitoring undertaken prior to 2015) to generate annual $PM_{2.5}$ from available PM_{10} data (e.g. Khyber Pass uses $PM_{2.5}$ / PM_{10} ratio generated from PM_{10} and $PM_{2.5}$ data collected at the site between 2013 and 2014). This approach was only used in the Auckland Region.

The above approaches using **partial datasets were used to estimate annual $PM_{2.5}$ exposure for approximately 10% of New Zealand's population.**

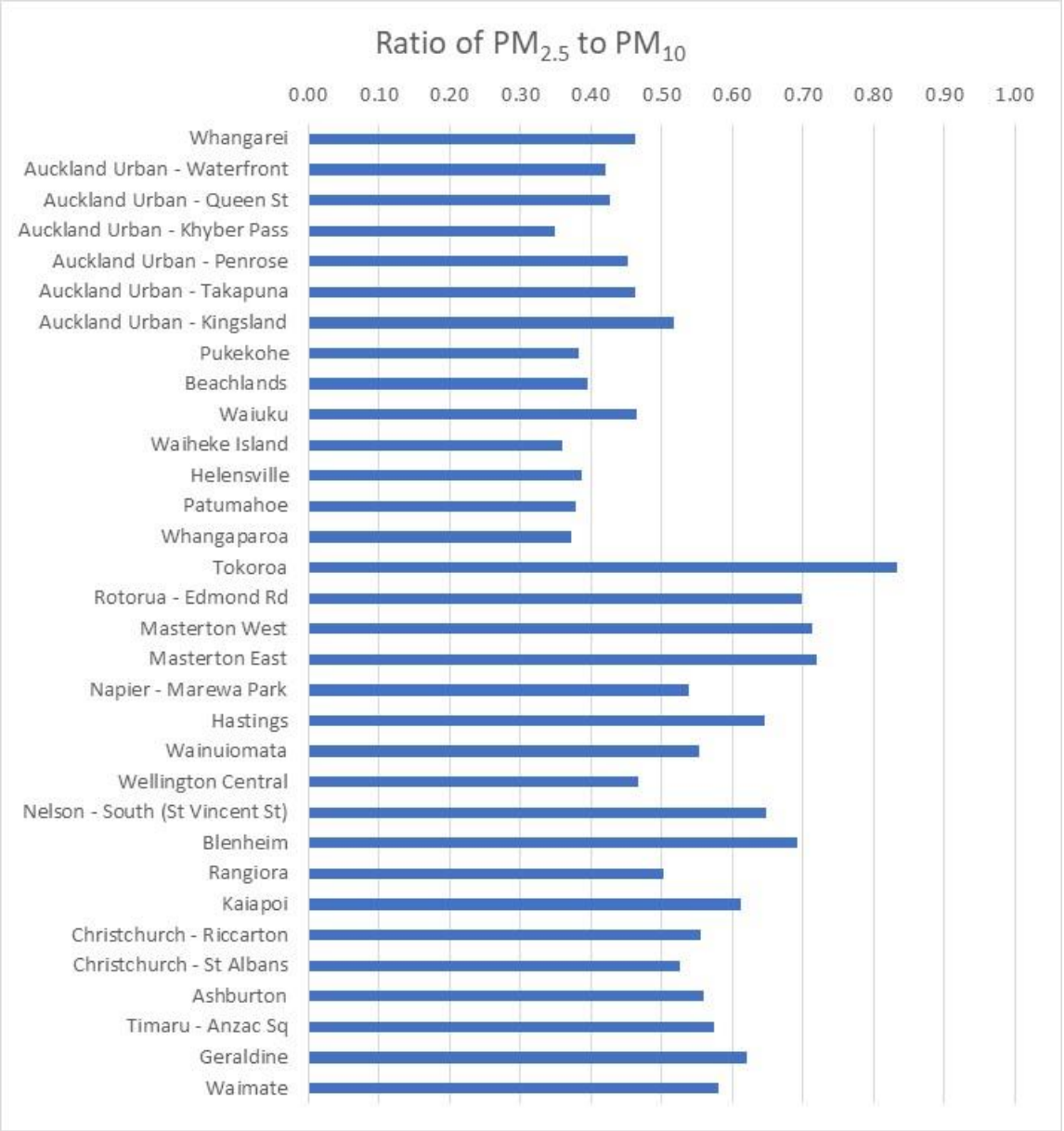
Annual $PM_{2.5}$ in airsheds with no $PM_{2.5}$ monitoring data

Estimating $PM_{2.5}$ / PM_{10} ratio from PM_{10} monitoring results

Figure 8 shows the $PM_{2.5}$ / PM_{10} ratio in all locations where PM_{10} and $PM_{2.5}$ were simultaneously monitored (excluding industrial sites). These ratios are based on monitoring undertaken between 2015-2018, where available. The additional year of data was used to provide more ratios than were offered from the base 2015-2017 dataset. In some Auckland locations, the ratios were based on monitoring undertaken prior to 2015. The average $PM_{2.5}$ / PM_{10} ratio across New Zealand (excluding industrial sites) was 0.53.

The $PM_{2.5}$ / PM_{10} ratio tends to be lower in Northland and Auckland (average ratio 0.42) compared with the rest of New Zealand (average ratio 0.61). The results also show that the ratio tends to be higher in locations where we know that domestic heating contributes to high levels of air pollution, e.g. Tokoroa, Rotorua, and Masterton in the North Island. A higher ratio means that a higher proportion of the PM_{10} is in the smaller size fraction ($PM_{2.5}$).

Figure 8: $PM_{2.5}$ / PM_{10} ratios at all locations where both were simultaneously monitored (excluding industrial monitoring sites)

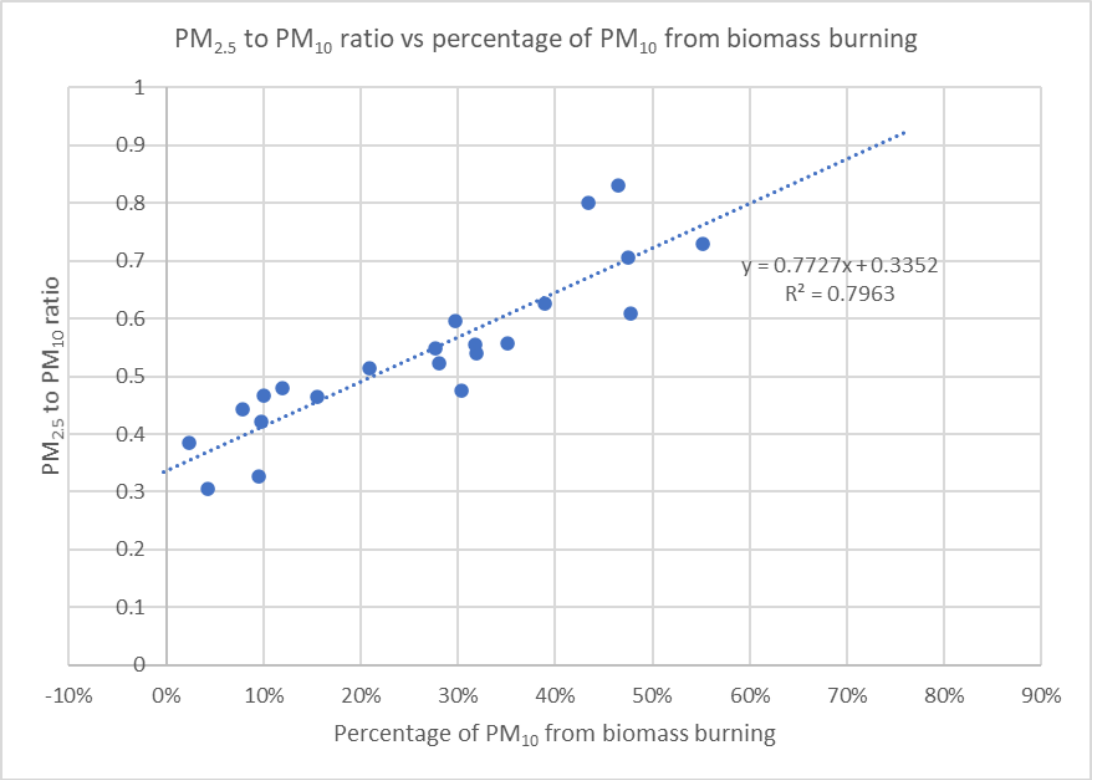


We investigated several methods to estimate the $PM_{2.5}$ / PM_{10} ratio in **locations with only PM_{10} monitoring**. We found no clear correlation between:

- PM_{10} concentration and $PM_{2.5}$ / PM_{10} ratio, or
- Home heating emissions density and $PM_{2.5}$ / PM_{10} ratio.

However, we found a good correlation between the $PM_{2.5}$ / PM_{10} ratio and the estimated percentage of PM_{10} that is from biomass burning, as shown in Figure 9.

Figure 9: Correlation between $PM_{2.5}$ / PM_{10} ratio and percentage of PM_{10} from biomass burning at all locations where PM_{10} monitoring was available



We estimated the percentage contribution of biomass burning to PM_{10} in all locations where suitable monitoring data was available, based on analysis of monthly PM_{10} monitoring results as described in section 4.3.1. These estimates were then used to estimate the $PM_{2.5}$ / PM_{10} ratio using this formula:

$$\boldsymbol{PM_{2.5}} \,/\, \boldsymbol{PM_{10}} = \%\boldsymbol{PM_{10}}\ \textbf{biomass} \times \mathbf{0.77} + \mathbf{0.34}$$

Where:

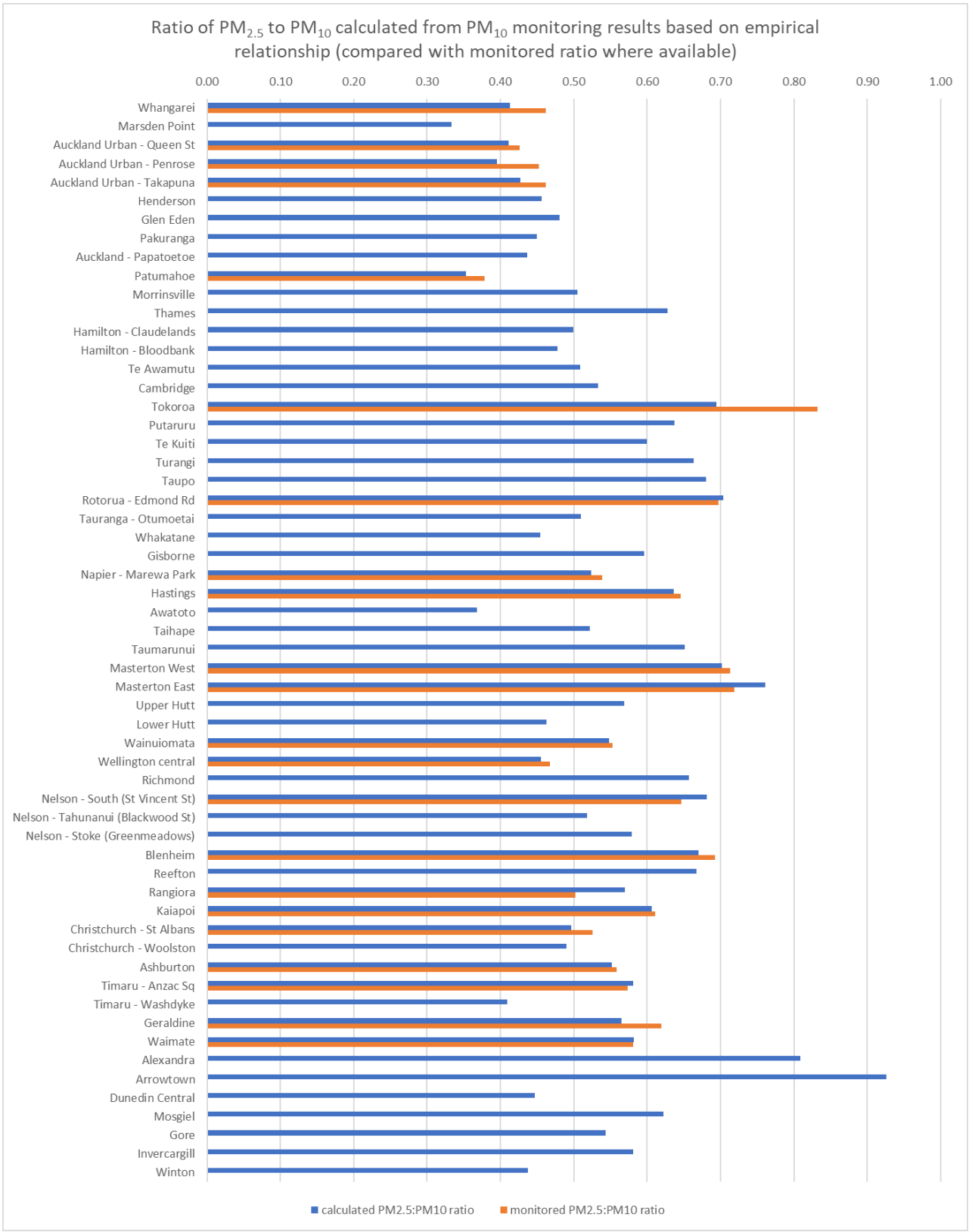
$PM_{2.5}$ / PM_{10} = the ratio of $PM_{2.5}$ to PM_{10} which is used to estimate $PM_{2.5}$ concentration indirectly from PM_{10}

% PM_{10} biomass = the percentage of PM_{10} from biomass burning. This was estimated from monthly PM_{10} monitoring results (see section 4.3.1)

The above approach using PM_{10} monitoring to estimate a $PM_{2.5}$/PM_{10} ratio was used to estimate **annual $PM_{2.5}$ exposure for 16% of New Zealand's population.**

Figure 10 presents the estimated $PM_{2.5}$ / PM_{10} ratio for all locations where suitable monthly PM_{10} data for 2015 and 2018 were available. The measured $PM_{2.5}$ / PM_{10} ratio from sites where $PM_{2.5}$ monitoring results were available is also shown for comparison.

Figure 10: $PM_{2.5}$ / PM_{10} ratio estimated from the biomass burning empirical relationship at all sites with suitable monitoring data



The estimated $PM_{2.5}$ / PM_{10} ratios are provided in the *HAPINZ 3.0 Health Effects Model – PM Ratios* worksheet.

Annual $PM_{2.5}$ in airsheds with PM_{10} monitoring data

In areas with no $PM_{2.5}$ monitoring data, we estimated annual $PM_{2.5}$ exposure for 2016 in the following order of preference:

- Used the **estimated $PM_{2.5}$ / PM_{10} ratio based on biomass burning**, where available (using the empirical relationship described in the previous section) to estimate $PM_{2.5}$ from the monitored PM_{10} concentration. Where more than one estimated ratio was available in the airshed, we made the following assumptions:
    - Hamilton was assigned the average estimated ratio from Claudelands and Bloodbank
    - Otago 1 airsheds were assigned the estimated ratio for Alexandra
- Assigned a **$PM_{2.5}$ / PM_{10} ratio from a comparable location** in the same region, where possible (either measured or estimated using the empirical relationship described in the previous section). This method was used in locations where a PM_{10} monitoring value was available, but the data were not suitable for estimating an empirical $PM_{2.5}$ / PM_{10} ratio. For urban areas, the lowest $PM_{2.5}$ / PM_{10} ratio from other urban areas in the region was assigned, with the following exceptions:
    - Wellsford, Warkworth, Snells Beach, Kumeu, and Riverhead were assigned a ratio of 0.38, based on the average of ratios from other small Auckland airsheds (Helensville, Waiheke, Beachlands, Pukekohe).
    - Waikato airsheds were assigned a ratio of 0.51, based on the estimated ratio for Morrinsville, which is considered likely to be representative of Waikato towns (see the analysis of PM_{10} discussed in previous section).
    - Porirua was assigned a ratio of 0.55, based on monitoring at Wainuiomata. This is the same ratio as that calculated for Upper Hutt and is higher than the monitored ratio for Wellington Central (0.45) and the estimated ratio for Lower Hutt (0.46). The Wellington and Lower Hutt ratios are low compared with small towns in the lower North Island and were considered unlikely to be representative of Porirua.
    - Otago 3 and Otago 4 airsheds were assigned a ratio of 0.81, which is the ratio for Otago 1 airsheds (estimated for Alexandra). This was higher than the ratio estimated for Dunedin (0.45), which was considered unlikely to be representative of small towns in Otago, and Mosgiel (0.62) which may have an industrial influence at the monitoring site.

Annual $PM_{2.5}$ in airsheds with no $PM_{2.5}$ or PM_{10} monitoring data

In areas with no $PM_{2.5}$ or PM_{10} monitoring data, we **assigned a $PM_{2.5}$ value from a comparable location in the same region**. This approach was consistent with the approach for assigning PM_{10} values to unmonitored airsheds. The assigned value was based on the lowest measured or estimated $PM_{2.5}$ concentration from monitored airsheds in the same region, with the following exceptions:

- Tuakau, Paeroa, Waihi, Huntly, Te Aroha, Ngaruawahia, Matamata and Otorohanga were assigned the value for Morrinsville (5.9 $\mu g/m^3$ based on the estimated $PM_{2.5}$ / PM_{10} ratio). This is consistent with the PM_{10} assumption for these locations.
- Whirinaki was based on the value for Napier (7.0 $\mu g/m^3$), which is the 2nd lowest $PM_{2.5}$ value in Hawke's Bay after Awatoto (which is an industrial site).

Annual $PM_{2.5}$ in areas outside gazetted airsheds

$PM_{2.5}$ in urban areas outside airsheds

For urban areas outside gazetted airsheds, we estimated annual $PM_{2.5}$ exposure based on the **lowest measured or estimated value** from an urban area in the region, with the following exceptions:

- In Waikato, the 2nd lowest $PM_{2.5}$ concentration of 5.8 µg/m^3 was used. This is consistent with the assumption for PM_{10} in Waikato.
- In Hawkes Bay, the 2nd lowest $PM_{2.5}$ concentration was used (excluding Awatoto which is influenced by industry).
- In Manawatū-Whanganui, the $PM_{2.5}$ concentration was estimated based on the assumed PM_{10} concentration of 11.6 µg/m^3 (the monitored value from Taumaranui). A $PM_{2.5}$ / PM_{10} ratio of 0.52 was assumed. This was the lowest ratio estimated for the region based on PM_{10} monitoring at Taihape.
- In Taranaki, the $PM_{2.5}$ concentration was assumed to be the same as Hamilton. This is consistent with the assumption for PM_{10}.
- In Marlborough, the $PM_{2.5}$ concentration was estimated based on the assumed PM_{10} concentration of 9.7 µg/m^3 (the monitored value from Picton). A $PM_{2.5}$ / PM_{10} ratio of 0.58 was assumed. This is the 2nd lowest ratio estimated across the Nelson, Marlborough and Tasman Regions based on PM_{10} monitoring at Nelson Stoke (the 2nd lowest after Tahunanui which is influenced by industry).
- In Tasman, the 2nd lowest $PM_{2.5}$ concentration from across Tasman and Nelson regions was used (excluding Tahunanui which is influenced by industry).
- In Southland, the $PM_{2.5}$ concentration was estimated based on the assumed PM_{10} concentration of 10.3 µg/m^3. A $PM_{2.5}$ / PM_{10} ratio of 0.54 was assumed. This is the lowest ratio estimated for Southland based on PM_{10} monitoring at Gore.

$PM_{2.5}$ in rural areas outside airsheds

Patumahoe in Auckland is the only rural $PM_{2.5}$ monitoring site in New Zealand. The average concentration measured from 2009-2018 (4.4 µg/m^3) was applied to all rural areas across New Zealand.

Annual PM_{10} and $PM_{2.5}$ in industrial areas

There were nine monitoring sites with PM_{10} monitoring data and five sites with $PM_{2.5}$ monitoring data in industrial areas for 2016. The monitoring data is summarised in Table 7.

We identified other significant industrial and port areas based on our knowledge and examination of satellite imagery. A full list of census area units that were identified as predominantly industrial or port is provided in the *HAPINZ 3.0 Health Effects Model – Industry Sites* worksheet.

These areas were assigned annual average PM_{10} values based on monitoring results from other industrial locations as follows:

- Glenbrook and Tiwai Point census area units were assigned a PM_{10} value of 12.3 µg/m^3 based on the Marsden Point monitoring result.
- All other industrial or port census area units were assigned a PM_{10} value of 18.2 µg/m^3. This is the average of all industrial area monitoring results shown in Table 7, excluding Marsden Point.

Table 7: PM monitoring data for industrial areas

Site	Airshed	PM_{10} µg/m^3	Averaging period	$PM_{2.5}$ µg/m^3	Averaging period
Penrose	Auckland urban	14.7	2015-2017	6.6	2015-2017
Ngapuna	Rotorua	13.9	2013		
Mt Maunganui	Mt Maunganui	20.0	2018		
Hawkes Bay – Awatoto	Awatoto	18.6	2015-2017	6.2	2017-2018
Nelson – Tahunanui	Nelson B1	18.3	2015-2017		
Woolston	Christchurch	20.0	2015-2017	8.1	2015-2017
Timaru – Washdyke	Washdyke	18.0	2015-2017	5.3	2016-2017
Lyttelton	Canterbury region	22.0	2016	9.0	2016
Marsden Point	Marsden Point	12.3	2015-2017		

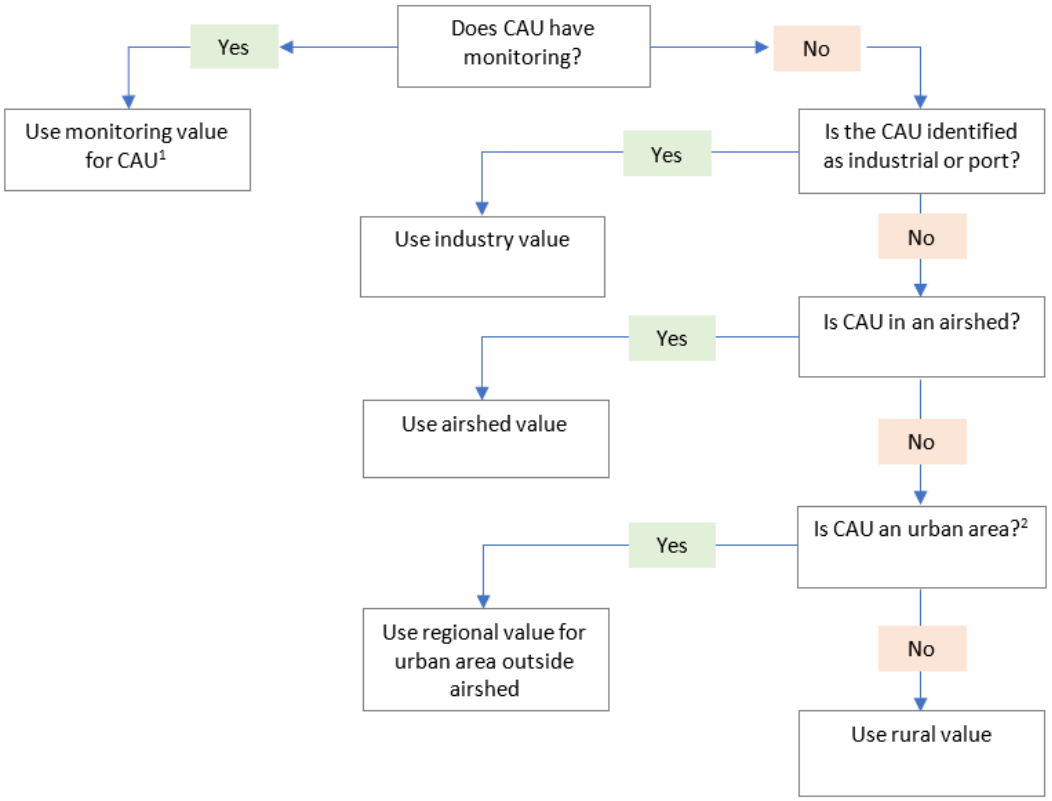
For industrial areas without $PM_{2.5}$ monitoring, the annual average concentration was estimated from PM_{10} based on an average $PM_{2.5}$ / PM_{10} ratio of 0.36. This is the average of ratios from Woolston, Washdyke and Awatoto (shown in Table 7). Industrial areas without PM_{10} or $PM_{2.5}$ monitoring were assigned a value of 6.5 µg/m^3 which is the assigned PM_{10} value of 18.2 µg/m^3 multiplied by the assigned ratio of 0.36.

3.3.5 Decision tree for assigning PM_{10} and $PM_{2.5}$ exposure

The PM_{10} and $PM_{2.5}$ exposure values assigned to each airshed, and other areas, are collated in the *HAPINZ 3.0 Health Effects Model – PM Values* worksheet. These values were developed based on the methodology described in previous sections.

The *HAPINZ 3.0 Health Effects Model* assigns a monitoring value to every CAU in New Zealand based on these values as shown in Figure 11.

Figure 11: Decision tree for assigning PM_{10} and $PM_{2.5}$ exposure values to every CAU



Notes:

1. Exceptions include:
   - Auckland CBD CAUs (Auckland Central East and West) assigned Queen Street monitoring value.
   - Auckland waterfront CAU (Harbourside) assigned Waterfront monitoring value from 2013.
   - Central Auckland CAUs (Arch Hill, Newton, Eden Terrace, Grafton East and West, Newmarket, Epsom North) assigned Khyber Pass value.
2. Main, secondary or minor urban area as defined by StatNZ (2017) as described in Table 6

3.3.6 NO_2 exposure

Despite more NO_2 monitoring data being available than in the past, the coverage is still insufficient to undertake a robust assessment of national exposure using actual data only. Consequently, we relied on estimates of NO_2 exposure in 2016 which were generated from the Waka Kotahi VEMT and NVED tools (described in section 3.2.4). Population-weighted annual average NO_2 concentration were generated for each meshblock and CAU, based on a map of estimated concentrations at 50m resolution.

Dataset for the health effects model

The *HAPINZ 3.0 Health Effects Model* (see section 7.3) is based on **population-weighted annual average NO_2 concentration by CAU**. These values were provided by Jacobs and were calculated as follows:

- All dwellings within 200 metres of a road were assigned a concentration from the NVED concentration tool. Each dwelling has an assigned population based on the CAU population that the dwelling resides in and the number of dwellings in that

CAU. The concentration for each dwelling is multiplied by its population, and these are summed for each CAU.

- The remaining (not roadside dwelling) population in the CAU has the representative annual background concentration for the CAU applied to it. The summed dwelling concentrations and the summed (not roadside dwelling) population concentrations are added together and then divided by the total CAU population, producing the final population weighted average concentration for each CAU.

As mentioned in section 3.2.3, the NO_2 concentration estimates include two components:

- the **roadside** component (within 200m of roads)
- the **background** component (further than 200m from a road).

The results provided show the total population weighted NO_2 estimates (used for our exposure assessment) are dominated by the background values with very minor contributions coming from the roadside, e.g. for Newton the total annual average NO_2 is 19.76 µg/m^3 versus a background value of 19.00 µg/m^3.

A full description of the methods used in the VEMT and the NVED concentration tool is outlined in T&T (2021). This report also includes discussion of the DMRB validation work and NO_X adjustments made to correct the NO_2 values for different road types.

Dataset for the cohort study

The *HAPINZ 3.0 Cohort Study* (see section 5.3.1) required a finer resolution in the data to differentiate potential changes in health impacts so Jacobs provided NO_2 concentration estimates at a 50m resolution directly from the Waka Kotahi NVED tool (Figure 12).

This dataset was then used to derive annual average NO_2 concentrations **by meshblock** as shown in Figure 13. We tried population-weighting NO_2 at meshblock scale but found that there was virtually no difference from the unweighted average, and so **we used the unweighted average in the cohort study.**

Figure 14 shows that the concentrations for some meshblocks were composited from a small number (1-3) of 50 m resolution "pixels" whilst others were made up of up to 20 "pixels".

Note: We repeated the cohort analysis using population-weighted annual average NO_2 concentrations by CAU (as in the *HAPINZ 3.0 Health Effects Model*) and found that the results were not altered substantially compared with the analysis based on meshblock data.

Figure 12: NO_2 concentrations at 50m resolution for Auckland in 2016 (provided by Jacobs)

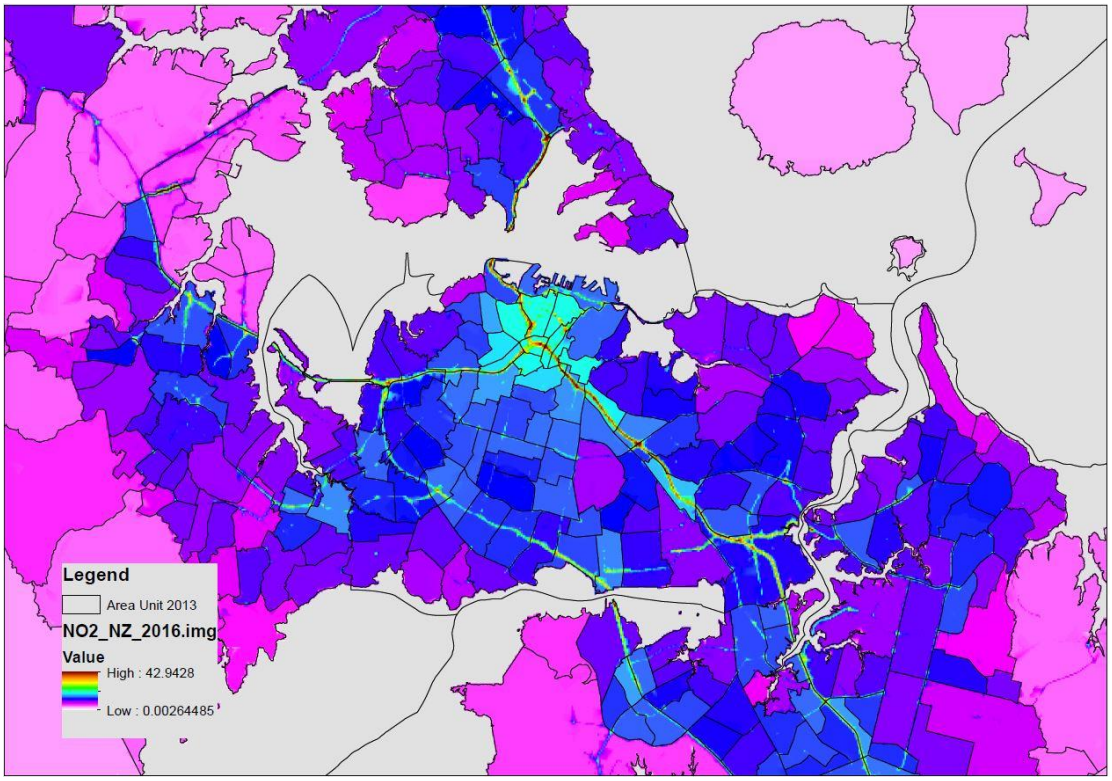


Figure 13: NO_2 concentrations at meshblock for Auckland in 2016 (used in the cohort study)

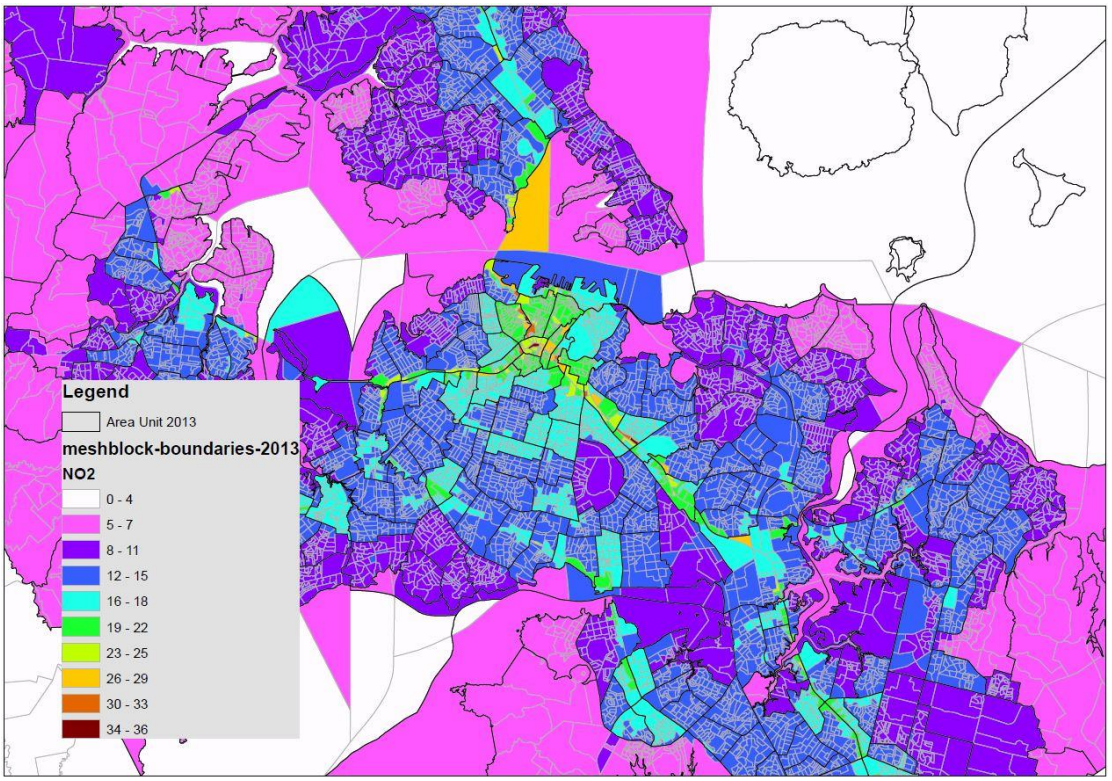
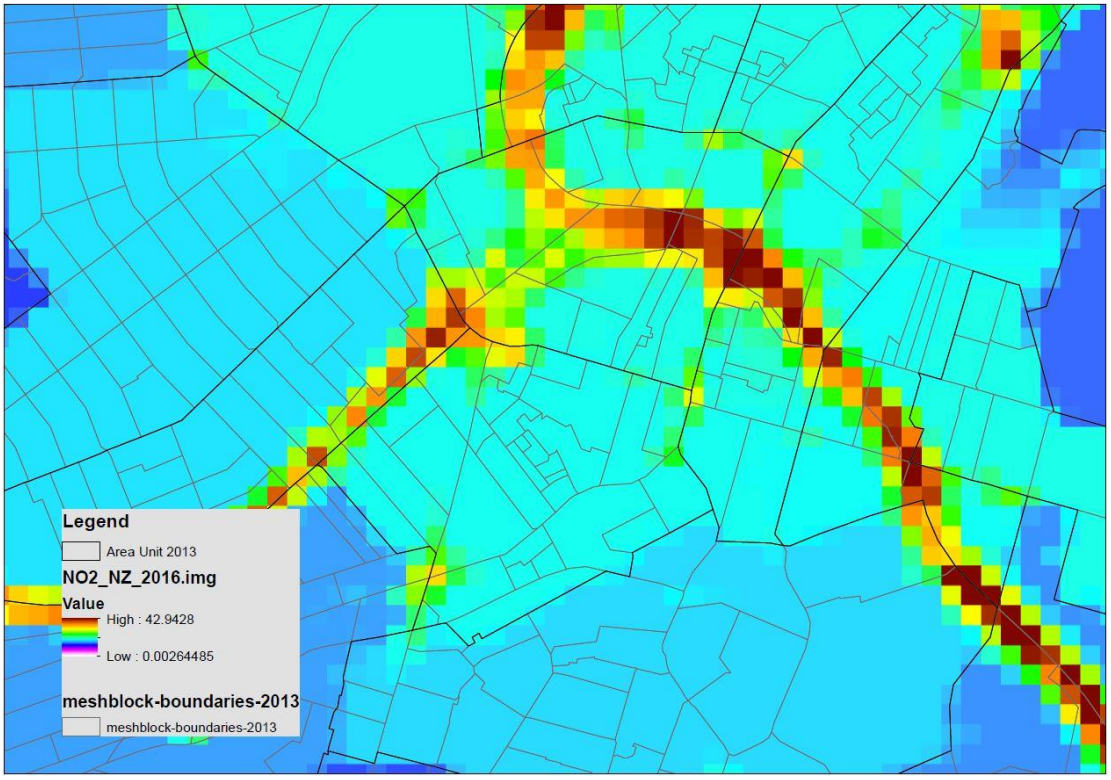


Figure 14: Comparison of 50m versus meshblock resolution for NO_2 concentrations



3.3.7 Other pollutant exposures

We did not assess exposure to O_3 or SO_2 in HAPINZ 3.0.

Concentrations of O_3 in New Zealand are very low and O_3 is currently only monitored at two locations - Musick Point, Auckland and Wellington Central.

Elevated levels of SO_2 tend to be localised and there are insufficient data available to assess impacts at a national level. However, the effects of secondary sulphate particulate are captured in the assessment of exposure to PM.

3.3.8 Uncertainty

As discussed in section 3.2.5, intra-urban contrasts (localised variations in concentration) introduce some uncertainty in the exposure assessment.

Roadside PM concentrations

We investigated whether outputs from the Waka Kotahi VEMT could be used to characterise intra-urban variation in concentrations of PM_{10} and $PM_{2.5}$ in the vicinity of roads. Tonkin and Taylor (**T&T**) recently undertook to validate the modelled roadside $PM_{2.5}$ concentrations from the Waka Kotahi VEMT (T&T 2020). They concluded that, due to the very small number of roadside $PM_{2.5}$ monitoring sites and an absence of reliable background monitoring sites, it is not currently possible to reliably confirm the road contribution.

Therefore, we could not use VEMT to reliably account for elevated PM exposure in the vicinity of roads so the PM exposure assessment for HAPINZ 3.0 is based on the results of ambient monitoring. These monitoring results reflect total emissions, including those from vehicles and other sources, but cannot differentiate background and roadside exposures separately. Adding predicted roadside contributions from VEMT risks double-counting.

We recommend further investigation to determine whether source apportionment results (which estimate the contribution of motor vehicles to measured concentrations) could be used to validate roadside concentration estimates and account for the road component of monitoring results in future.

Other localised sources

We cannot accurately account for elevated exposure in the vicinity of other localised sources, such as large industry. These effects are managed by regional councils on a case-by-case basis.

For HAPINZ 3.0, we identified significant industrial areas and assigned a typical concentration to these areas. Further work, including development of a comprehensive national inventory of industrial emissions and dispersion modelling, would be required to improve estimates of exposure to industrial emissions and the subsequent effects at a national level.

Note: Ultimately, the most appropriate method and resolution for exposure assessment reflects the methods and resolution used in the original epidemiological research. For this study, we developed and used New Zealand specific exposure-response functions for PM_{10}, $PM_{2.5}$ and NO_2. This means that uncertainty in the exposure assessment is captured (to some extent) in the uncertainty of the exposure-response functions and is therefore captured in the overall uncertainty estimates quoted in the results.

3.3.9 Summary of our approach

Particulate matter (PM_{10} and $PM_{2.5}$) was used as the primary indicator of air pollution exposure based on the wealth of monitoring data available and the links to existing exposure-response functions developed from New Zealand and international epidemiological studies.

Nitrogen dioxide (NO_2) was also included based on increased epidemiological evidence of effects, and improved information about exposure to NO_2 from road transport in New Zealand.

Exposure to PM relied on actual monitoring data in preference to modelling estimates, with data **averaged for 2015 to 2017** (where possible) to reduce the influence of interannual variability in meteorological conditions.

In general, **the monitored PM concentration was applied to the entire airshed**. However, some judgement was required, for example:

- For airsheds with more than one monitor, the most representative site(s) was selected.
- For unmonitored areas, annual concentrations were based on comparisons with monitored areas that had similar characteristics (including urban/rural classification, topography, meteorology and emissions).
- To estimate $PM_{2.5}$ in areas where only PM_{10} is monitored, ratios of $PM_{2.5}$ to PM_{10} were derived using a new empirical relationship based on PM_{10} monitoring data. This new empirical method was specifically developed for HAPINZ 3.0.

Exposure to NO_2 was estimated for 2016 based on the Waka Kotahi VEMT and NVED tools. Population-weighted annual average NO_2 concentration were generated for each meshblock and CAU, based on a map of estimated concentrations at 50m resolution covering both roadside and background contributions.

4. Attributing source contributions

Understanding the contributions that individual sources make to air pollution concentrations or exposure and the associated health effects is important. Management strategies can then be tailored to reduce the health burden associated with air pollution.

This chapter summarises the methodology used for attributing exposures and effects to different sources in the HAPINZ 2.0 study, reviews developments that have occurred since in emissions inventories and source apportionment data and presents the approach we developed for the HAPINZ 3.0 update.

4.1 Approach used in HAPINZ 2.0

HAPINZ 2.0 attributed effects to the following air pollution sources:

- **domestic fires** used for home heating (primarily wood burners, coal burners and open fires etc but some gas-fired appliances were reported in the regional inventories)
- **motor vehicles** (on road only transport such as petrol and diesel cars, vans, trucks and buses)
- **industry** (stationary facilities for manufacturing products or generating energy that release process or combustion emissions)
- **open burning** (burning of biomass such as tree trimmings or waste outdoors)
- **natural sources** (primarily sea spray and windblown dust).

Disaggregation by source was undertaken using a combination of approaches:

- The **natural** source contribution was estimated based on the results of source apportionment studies. These studies estimate source contributions based on particulate matter composition analysis and receptor modelling.
- The **industrial** tall stack contribution was based on a national industrial emissions inventory (Wilton *et al* 2008) with assumptions to estimate ground level concentrations based on available stack emission characteristics and dispersion.
- The contribution of **other anthropogenic sources** to concentrations was assumed to be proportional to the proportion of emissions from that source.

Emission estimates for other sources (such as aviation, shipping, rail, as well as off-road construction, farming and agriculture vehicles) were not reported consistently in emission inventories across New Zealand so were not included. However, in most areas, the first four sources – domestic fires, motor vehicles, industry and open burning – were considered likely to represent at least 95% of all possible anthropogenic PM_{10} emissions.

4.2 Developments since HAPINZ 2.0

This section reviews the developments in the availability of source apportionment data and emissions inventories for New Zealand since HAPINZ 2.0.

4.2.1 Contribution of sources to concentration

For all anthropogenic sources, except tall stacks, the HAPINZ 2.0 study assumed that the contribution of each source to the PM_{10} concentration was proportional to the estimated quantity PM_{10} *emissions* from each source. The emissions from each source were estimated based on regional and national emissions inventories available at the time.

Air emissions inventories are a critical component of managing air quality and are undertaken by regional councils regularly to gauge how emissions are tracking in the region. The inventories cover key emission sources and pollutants in the region, sometimes covering all major sources/pollutants or just focussing on one source/pollutant. Emission inventories rely on emission factors (e.g. the amount of pollution produced per km travelled by petrol cars) and activity data (e.g. the annual km travelled by petrol cars).

Updated emissions inventories are available at a national and regional level. However, these are subject to the following limitations:

- The National Air Emissions Inventory 2015 (Metcalfe & Sridhar 2018) uses primarily top-down methods to provide total estimated emissions at a national level only.
- The industrial emissions inventory, which was used to estimate tall stack contributions in HAPINZ 2.0, has not been updated since 2008.

Regional inventories across New Zealand are not always consistent and directly comparable (Sridhar & Kuschel 2018). They typically vary in terms of the emissions sources covered, the pollutants assessed, the spatial and temporal resolution and the base year.

Even with a robust emissions inventory, the relative contributions of sources to concentrations and subsequent exposures are not necessarily proportional to their emissions. Inventories do not reflect dispersion and the proximity of receptors to the emission sources (which can be much reduced for exposure to road traffic than for other sources in urban areas). In addition, it is challenging to ensure emission factors reflect real-world emissions.

Substantial work has been undertaken in New Zealand to directly measure the contribution of sources to particulate pollution. Source apportionment uses elemental analysis of real-world monitoring results to identify the relative contributing sources as well as the contribution of secondary PM.

Particulate matter has been collected and analysed from approximately 40 sites across New Zealand, with some urban areas including multiple sites. For example, in Auckland, PM samples have been collected at Takapuna, Henderson, Kingsland, Newmarket, Auckland CBD (Queen Street), Penrose and Patumahoe (40 km southwest of the CBD). At each location, the PM samples have been analysed for BC and multi-elemental speciation (hydrogen, sodium to uranium) with the accompanying receptor modelling (source apportionment) and reporting.

In addition to the urban speciation monitoring locations, several studies have targeted source specific PM. These include motor vehicle tunnels (Ancelet *et al* 2011; Davy *et al* 2011) and wood burner emissions (Davy *et al* 2009; Ancelet *et al* 2010; Ancelet *et al* 2011b) in order to better understand emission source characteristics and composition. Further details of the PM speciation sampling are provided in Davy & Trompetter (2020).

Figure 15 and Table 8 show the locations across New Zealand where PM speciation sampling has been undertaken to date (Davy & Trompetter 2018).

Figure 15: Particulate matter speciation sampling locations in New Zealand

Source: Davy & Trompetter (2018)

Table 8: Particulate matter speciation sampling locations in New Zealand

Location	Sites	Time period	Frequency
Northland	Whangarei	2004-2012	1 day-in-6
Wellington Region	Masterton	2002-2004	1 day-in-3,
	Masterton (2 sites)	Winter 2010	Hourly
	Upper Hutt	2000-2002	Variable
	Wainuiomata	2006-2008, 2011-2014	1 day-in-3
	Wainuiomata	2014-onwards	6-hourly continuous
	Seaview	2002-2004, 2005-2007	1 day-in-3
	Wairarapa (Masterton, Carterton, Featherston)	Winter 2009	Daily (screening)
	Mt Victoria Tunnel	Summer 2009	
	Baring Head	1996-1998	
	Raumati	Winter 2010	12-hourly
	7 Wellington sites indoor/outdoor	Winter 2017	2-hourly
	Masterton East	2018	1-day-in-3
Auckland Region	Kingsland	2004-2007	1 day-in-3
	Takapuna	2007-2016	1 day-in-3
	Takapuna	2006-onwards	1 day-in-3
	Takapuna (3 sites)	Winter 2012	Hourly
	Queen Street	2006-2016	1 day-in-3
	Queen Street	2006-onwards	Daily
	Penrose	2006-2016	1 day-in-3
	Khyber Pass Road	2006-2015	1 day-in-3
	Henderson	2006-onwards	1 day-in-3
	Patumahoe	2010	Daily
	Auckland (4 sites)	Winter 2018	Daily
	Johnstone Hills tunnel	Jun-10	3-hourly
Nelson	Tahunanui	2008-2009	1 day-in-3
	Nelson City	2006-2012	1 day-in-6,
	Nelson City (3 sites)	Winter 2011	Hourly
Marlborough	Blenheim	2007	1 day-in-3
Otago	Dunedin	2010	1 day-in-3
	Alexandra (3 sites)	Winter 2011	Hourly
Canterbury	Christchurch	2001-2002	Daily
	Timaru	2006-2007	1 day-in-3
	Woolston	2013-2014	2-hourly
	Christchurch (Coles Place)	2013-2015	1 day-in-3
	Christchurch (Coles Place, Woolston, Riccarton) h	Winter 2014	2-hourly
Hawkes Bay	Hastings	2006-2007	1 day-in-3
	Meanee Rd	2006+2008	1 day-in-2 (screening survey)
	Napier	2008-2009	1 day-in-3
	Awatoto	2016-2017	1 day-in-3
	Marewa Park	2017-2018	1 day-in-3
Southland	Invercargill	Winter 2014	Hourly
Waikato	Tokoroa	Winter 2014	Daily
	Tokoroa	October 2015- October 2016	Daily
Bay of Plenty	Rotorua (Whakarewarewa Village)	October 2014 -onwards	1 day-in-3
Tasman	Richmond	2013 - 2016	1 day-in-3
	Richmond	2015 - 2016	Daily

Source: Davy & Trompetter (2018)

4.2.2 Contribution of sources to health effects

HAPINZ 2.0 attributed health effects associated with different sources of PM_{10} (natural, domestic fires, motor vehicles, industry and open burning) based solely on the estimated contribution of each source to the concentration of PM_{10}. This was based on the assumption that all PM_{10} is treated as equally toxic, irrespective of source. This, in turn was consistent with the approach recommended by WHO at that time:

> **The mass-based standards that have been proposed inherently assume that all airborne PM has the same potential to cause adverse health effects, regardless of chemical composition or physical characteristics.** While both observational and experimental findings imply that particle characteristics are determinants of toxicity, definitive links between specific characteristics and the risk of various adverse health effects have yet to be identified. (WHO 2006)

Despite a further 15 years of research, WHO acknowledges this issue remains to be resolved:

> Many studies have tried to identify which sources and/or physicochemical characteristics of airborne PM contribute most greatly to toxicity. **This is a challenging area of research**, given the great heterogeneity of airborne particles, **and a definitive set of particle characteristics has yet to be identified**. (WHO 2021).

Particles from different sources (e.g. domestic fire emissions as opposed to sea spray) will have quite different chemical compositions, different physical characteristics and therefore potentially quite different toxicities. However, **the evidence from epidemiological studies to support different exposure-response functions for different PM sources remains inconclusive**. This is discussed further in section 5.2.1.

The contribution of sea salt and, to a lesser extent, other 'natural sources' to health effects of air pollution in New Zealand has been controversial.

In the 2013 review of evidence on health aspects of air pollution (**REVIHAAP**), the **WHO concluded that there was little epidemiological evidence of the harmfulness of sea spray** (WHO, 2013). **However**, health effects are estimated based on exposure-response functions derived from epidemiological studies. These **epidemiological studies are based on total measured PM concentration, including any contribution of sea salt**.

We cannot assess the relative contributions of different sources to PM health effects currently due to insufficient evidence. However, we have designed the *HAPINZ 3.0 Health Effects Model* so that risk response functions can be specified by source if better information becomes available at a later date**.** This is discussed in more detail in section 7.3.

To some extent this concern has been addressed in HAPINZ 3.0 by focussing the assessment on the effects of $PM_{2.5}$. The relative contributions of sea salt and crustal material (referred to as windblown dust in HAPINZ 2.0) to $PM_{2.5}$ concentrations are much lower than their relative contributions to PM_{10} concentrations.

4.3 What we did in HAPINZ 3.0

In HAPINZ 3.0, we used two different approaches to attribute source contributions depending on the pollutant.

For PM, with the wealth of data now available, we attributed contributions to sources based on already identified source apportionment "fingerprints" [16] as follows:

- **domestic fires** used for home heating – based on a *biomass burning* fingerprint (where available) or an empirical method based on $PM_{2.5}$
- **motor vehicles** encompassing exhaust, brake/tyre wear and re-suspended road dust – based on a *motor vehicle* fingerprint
- **industry** – based on a *local industry* fingerprint (where available)
- **windblown dust** from sources such as construction, land use, industry and the movement of motor vehicles (e.g. road abrasion, suspension and re-suspension of surface material) – based on a *crustal material* fingerprint
- **sea spray** – based on a *marine aerosol* fingerprint
- **secondary PM** resulting from gases emitted from natural and anthropogenic sources reacting in the atmosphere to form particles – based on a *secondary sulphate* fingerprint.

We started with assigning preliminary values to individual source contributions in monitored areas (see section 4.3.1), then made assumptions based on that information to assign preliminary values for individual sources in unmonitored areas (see section 4.3.2). Once preliminary values were available for all relevant individual sources, the estimated totals were compared with the values from the actual monitoring from Chapter 3 and minor adjustments made to ensure the totals matched (see section 4.3.3). The analysis is provided in the *HAPINZ 3.0 Sources* workbook. [17]

In contrast, the only source considered for NO_2 was motor vehicles and the approach taken is outlined in section 4.3.5.

4.3.1 Preliminary PM_{10} and $PM_{2.5}$ source attributions in areas with suitable monitoring data

The contribution of sources to ambient PM_{10} and $PM_{2.5}$ at locations with suitable monitoring were estimated based on the methodology described in the following sections. The overall results of source attribution at sites with suitable monitoring are summarised in the *Site values* worksheet of the *HAPINZ 3.0 Sources* workbook.

[16] HAPINZ 3.0 derives sources of PM from the compositional analysis of samples collected at the same monitoring stations that PM_{10} or $PM_{2.5}$ ambient concentrations are measured. Each source is identified by specific groupings of elements (fingerprints).

[17] This is separate to the *HAPINZ 3.0 Health Effects Model.*

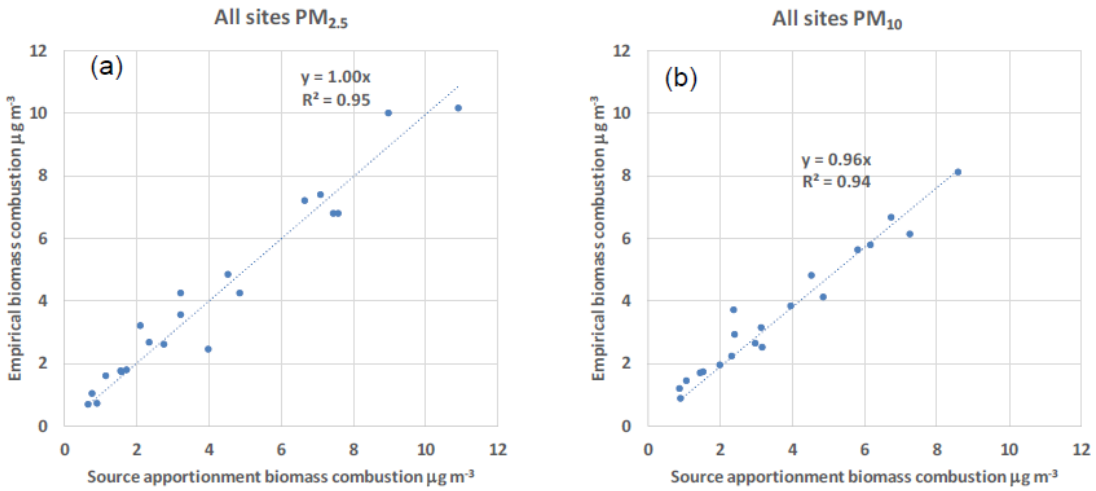
Domestic fire PM

Domestic fire PM was attributed based on a *biomass burning* fingerprint (where available) or an empirical method based on $PM_{2.5}$.and assigned to **anthropogenic**.

Biomass burning PM is primarily associated with residential wood burning for heating. While source apportionment data are available for biomass burning in various New Zealand locations, this information covers different time periods (often wintertime only) and different base years so was not available in all locations.

As part of HAPINZ 3.0, GNS Science developed an empirical model to estimate the contribution of biomass burning to both $PM_{2.5}$ and PM_{10} in locations without source apportionment data but where monthly ambient data were available. The method yielded strong correlations for biomass contributions based on $PM_{2.5}$ (R^2=0.95) and based on PM_{10} (R^2=0.94) as shown in Figure 16 (Davy & Trompetter 2020). This enabled a consistent approach to be adopted for all locations being assessed across New Zealand.

Figure 16: Comparison of the empirical model versus source apportionment data for average biomass combustion source contributions to (a) $PM_{2.5}$ and (b) PM_{10} at all NZ sites



Source: Davy & Trompeter (2020)

All available monthly PM_{10} and $PM_{2.5}$ monitoring data between 2015 and 2018 were obtained from Regional Councils and collated. The additional year of data was used to provide more monthly averages than were available from the base 2015-2017 dataset. We then applied the GNS empirical model to estimate the contribution of biomass burning to PM_{10} and $PM_{2.5}$ concentrations in each location.

For airsheds with more than one monitor, the following assumptions were made:

- Auckland Urban airshed was based on the average from Penrose, Takapuna, Henderson, Glen Eden, Pakuranga and Papatoetoe.
- Hamilton was based on the average from Claudelands and Bloodbank.
- Masterton was based on the average from Masterton West and Masterton East.
- Otago 1 was based on the average from Alexandra and Arrowtown.

- Christchurch was based on St Albans.

To estimate $PM_{2.5}$, the following assumptions were made:

- In locations with both PM_{10} and $PM_{2.5}$ monitoring data, the biomass contribution to both PM_{10} and $PM_{2.5}$ was assumed to be the average of the two.
- In locations with only PM_{10} data, all biomass PM_{10} was assumed to be $PM_{2.5}$. This is consistent with the results of source apportionment (Davy & Trompetter 2020).

The estimated contribution of biomass burning to PM_{10} and $PM_{2.5}$ in all locations with available monthly monitoring data between 2015 and 2018 is summarised in the *Site values* worksheet of the *HAPINZ 3.0 Sources* workbook.

Open burning in urban areas

Open burning of waste is now banned, or severely restricted, in most urban areas and source apportionment studies have not identified it as a significant source of pollution in urban areas. Nonetheless, there are still a few urban areas across New Zealand where open burning is allowed.

Table 9 presents estimates of the likely contribution open burning makes to total PM_{10} emissions from biomass burning (both domestic heating and open burning) in urban areas where it is allowed (Wilton *et al* 2015). This shows that open burning may account for up to 12% of total biomass burning PM_{10} in these areas (but more typically no more than 8%)

In the few urban areas, where it is still allowed, we approximated the likely contribution to PM exposure based on emissions inventory data and assigned it to domestic fires.

Table 9: Estimated contribution of open burning to PM_{10} from total biomass burning (due to domestic heating and open burning) in urban areas where open burning is allowed

Urban area	Open burning PM_{10} as a percentage of total biomass burning PM_{10}
Whangarei	9%
Hamilton	2%
Gisborne	5%
Taupo	4%
Whanganui	7%
New Plymouth	8%
Palmerston North	8%
Masterton	6%
Lower Hutt	8%
Wellington	12%
Invercargill	1%

Note: These percentage figures are calculated from open burning and domestic heating emission estimates in the 2013 national inventory (Wilton *et al* 2015). The 2013 inventory included open burning emissions in Tauranga, Rotorua, Hastings, Napier, Blenheim, Dunedin and Queenstown. However, open burning in these airsheds is now banned or significantly restricted so they are not included here

Open burning in rural areas

Burning of crop residues in rural areas is a significant source of PM emissions in some rural locations. However, we do not have monitoring data, emissions are intermittent, and very few people are exposed.

Consequently, **we did not attempt to estimate the contribution of open burning in rural areas**.

Motor vehicle PM

Motor vehicle PM was attributed using a *motor vehicle* fingerprint and assigned to **anthropogenic**.

The contribution of motor vehicles to ambient PM is influenced by proximity to roadways, traffic volumes and density of the local urban road network (Davy & Trompetter 2020). The estimated motor vehicle contribution to PM based on source apportionment includes all PM associated with the source. This will include exhaust emissions, brake wear, tyre wear and particles from road wear and other mechanical abrasion processes. [18]

Average motor vehicle source contributions to PM from source apportionment (receptor modelling) studies are summarised in Table 3.3 of Davy & Trompetter (2020).

To estimate the contribution of motor vehicles to ambient PM concentrations in urban areas, we compared these source apportionment estimates with the motor vehicle emissions density in the CAU where the monitoring site was located as shown in Figure 17 for PM_{10} and Figure 18 for $PM_{2.5}$. Motor vehicle emissions density by CAU information was obtained from the 2013 National Air Emissions Inventory (Wilton *et al* 2015).

We estimated **the contribution of motor vehicles to PM_{10}** using this formula:

$$\boldsymbol{Motor\ Vehicle\ PM_{10}} = \textbf{emissions density} \times \mathbf{0.31} + \mathbf{1.57}$$

Where:

Motor Vehicle PM_{10} = the estimated contribution of motor vehicles to urban PM_{10} concentration ($\mu g/m^3$)

Emissions density = the population weighted motor vehicle emissions density, calculated for each airshed based on population and PM_{10} emissions density data by CAU from the 2013 National Air Emissions Inventory (Wilton *et al* 2015)

[18] Note some of the re-suspended dust may also be present in the crustal material fingerprint.

Figure 17: Correlation between motor vehicle PM_{10} concentration from source apportionment and motor vehicle PM_{10} emissions density

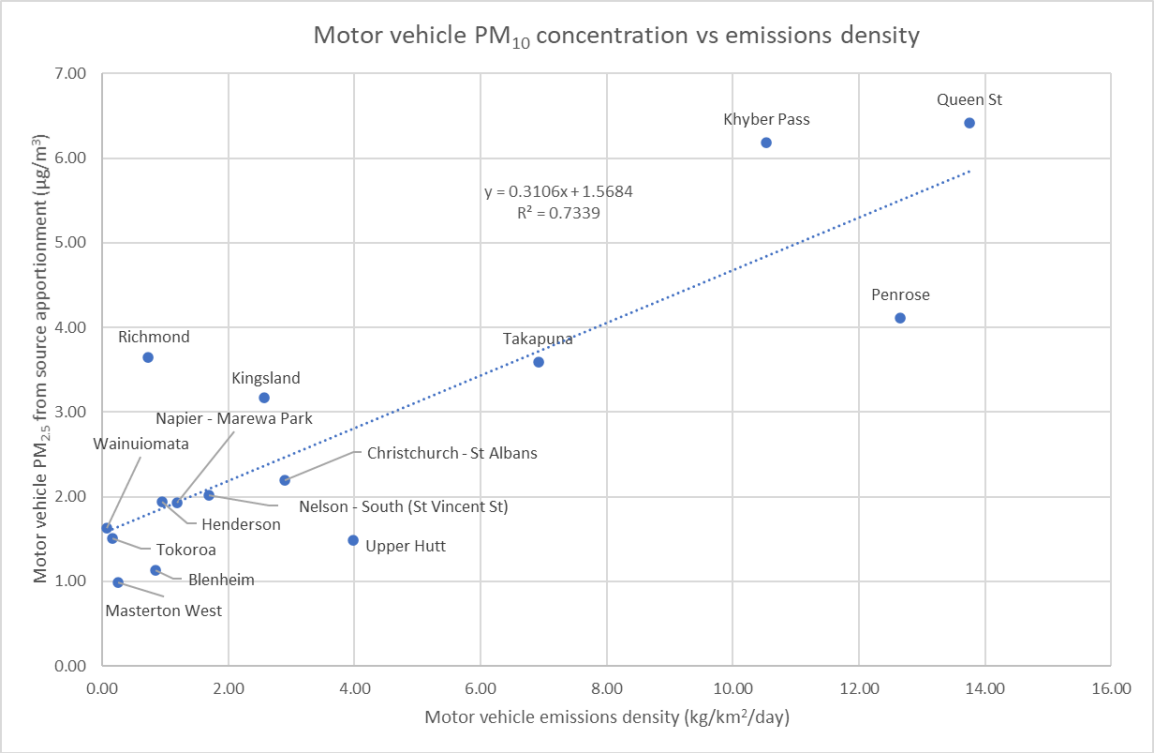
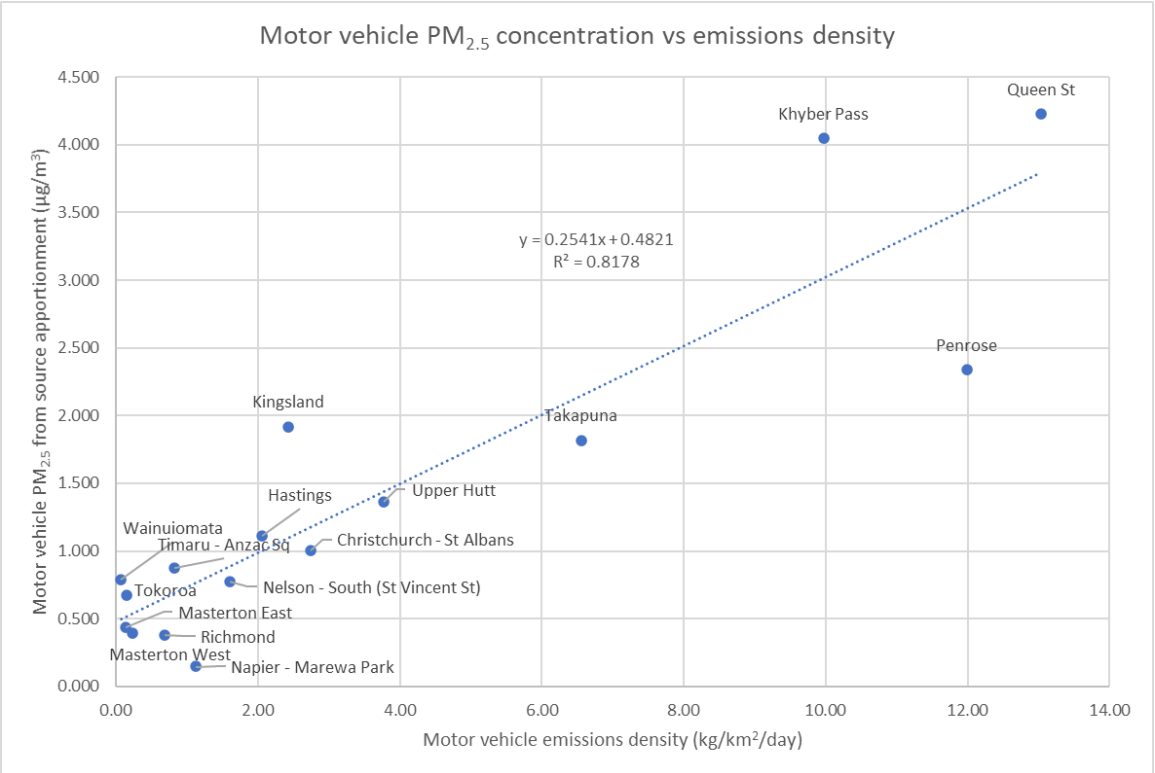


Figure 18: Correlation between motor vehicle $PM_{2.5}$ concentration from source apportionment and motor vehicle $PM_{2.5}$ emissions density



We estimated **the contribution of motor vehicles to $PM_{2.5}$** using this formula:

$$\boldsymbol{Motor\ Vehicle\ PM_{2.5} = \text{emissions density} \times 0.25 + 0.48}$$

Where:

Motor Vehicle $PM_{2.5}$ = the estimated contribution of motor vehicles to urban $PM_{2.5}$ concentration ($\mu g/m^3$)

Emissions density = the population weighted motor vehicle emissions density calculated for each airshed based on population and $PM_{2.5}$ emissions density data by CAU from the 2013 National Air Emissions Inventory (Wilton *et al* 2015).

The estimated contribution of motor vehicles to PM_{10} and $PM_{2.5}$ in all airsheds where a biomass burning estimate was also available, are summarised in the *Site values* worksheet of the *HAPINZ 3.0 Sources* workbook.

Other transport sources PM

Emission estimates for other anthropogenic PM sources – especially other modes of transport such as aviation, shipping, and rail – are not reported consistently in emission inventories across New Zealand. The HAPINZ 2.0 report concluded that domestic fires, motor vehicles, industry and open burning are likely to represent at least 95% of anthropogenic emissions. This conclusion is supported by source apportionment studies, which have typically quantified sources accounting for 95% of total PM concentrations.

Fortuitously, another study – the Domestic Transport Costs and Charges (**DTCC**) Study – was being undertaken while HAPINZ 3.0 was being finalised.

One of the components of the DTCC Study involved estimating air emissions from a range of transport modes in urban and rural areas of New Zealand for 2018/19 (Kuschel *et al* 2021). In the DTCC work, *urban* was assumed to be an area with an estimated resident population of more than 1,000 people, which is consistent with the approach taken in HAPINZ 3.0. Table 10 shows the relative contribution of road, rail, shipping and aviation to 2018/19 emissions of PM, NO_X and SO_2 in urban and rural areas of New Zealand, as well as overall.

Table 10: Relative contributions of road, rail, shipping and aviation to 2018/19 emissions of PM, NO_X and SO_2 in urban areas, rural areas and nationally

Transport mode	$PM_{2.5}$ exh t/yr	PM_{10} B&T t/yr	PM_{10} RD t/yr	Total PM_{10} t/yr	Total NO_X t/yr	Total SO_2 t/yr	PM10 %
Urban areas							
Roads	707	374	168	1,250	17,477	18	94%
Rail	-	-	-	12	550	0.2	1%
Shipping	-	-	-	59	932	417	4%
Aviation	-	-	-	3.0	824	58	0%
Total urban	**707**	**374**	**168**	**1,324**	**19,783**	**494**	**100%**

Transport mode	$PM_{2.5}$ exh t/yr	PM_{10} B&T t/yr	PM_{10} RD t/yr	Total PM_{10} t/yr	Total NO_X t/yr	Total SO_2 t/yr	PM10 %
Rural areas							
Roads	1,308	647	930	2,886	32,303	30	98%
Rail	-	-	-	38	2,007	0.6	1%
Shipping	-	-	-	5.7	55	50	0%
Aviation	-	-	-	0.8	229	16	0%
Total rural	**1,308**	**647**	**930**	**2,931**	**34,593**	**97**	**100%**
Nationally							
Roads	2,016	1,021	1,099	4,136	49,780	48	97%
Rail	-	-	-	50	2,556	0.8	1%
Shipping	-	-	-	64	986	467	2%
Aviation	-	-	-	3.8	1,053	75	0%
Total national	**2,016**	**1,021**	**1,099**	**4,254**	**54,376**	**591**	**100%**

Source: Kuschel *et al* (2021)

Note: exh=exhaust, B&T=brake & tyre wear and RD=road dust (both sealed & unsealed)

Although for a slightly different base year than HAPINZ 3.0, the DTCC estimates confirm that road transport is responsible for the bulk of transport-related emissions of PM_{10} (97% overall nationally) and NO_X (92% overall nationally), with shipping generating most of the transport-related SO_2 emissions (79% overall nationally).

While emissions do not translate directly to concentrations, the association is still strong. Therefore, the contribution of transport sources other than roads to PM concentrations in the locations assessed in HAPINZ 3.0 is likely to be minimal.

For SO_2, at least some of the emissions released by shipping likely appear in the secondary particulate source apportionment fingerprint but are not attributed specifically as shipping.

In addition, the road network is ubiquitous across New Zealand – with very few CAUs not being impacted by roads. By comparison, only 11 major seaports and 18 major airports were identified in the DTCC work so while impacts of these activities will be localised, they will not be widespread.

Shipping has been identified as a separate source in one source apportionment study to date (Davy *et al* 2017). Monitoring undertaken at Queen Street, Auckland found shipping contributed 0.5 µg/m^3 or 3% of PM_{10} and 0.4 µg/m^3 or 5% of $PM_{2.5}$. However, the spatial extent of this effect at Auckland or at other ports is not known. This is an area for further research.

We did not estimate the contribution of other sources of PM. However, we consider that these are likely to account for less than 5% of exposure at a national level.

Industry PM

Industry PM was attributed using a *local industry* fingerprint (where available) and assigned to **anthropogenic**.

PM_{10} source apportionment results, provided in Table 3.4 of Davy & Trompetter (2020), show that industrial sources are not specifically identified in source apportionment studies in most urban locations. At monitoring sites where industrial sources are identified, the estimated contribution of industrial emissions to PM is very location-specific (depending on the industries in the vicinity of the site). For example, the following specific sources were identified at Auckland monitoring sites:

- Cement dust from a nearby concrete batching plant at Takapuna
- Construction at Queen Street and Khyber Pass Road sites
- An unidentified zinc source at Henderson
- Hot dip galvanising, concrete batching and aggregate handling at Penrose (an industrial area).

The individual sources influencing these monitoring sites are likely to have localised effects and are unlikely to influence PM concentration across an entire CAU (except in Penrose, which is an industrial area with multiple sources).

Industrial areas were identified (as described in section 3.3.4). We did not attempt to quantify the contribution of industry to PM in other areas. Although some industries are located in these areas, their effects are highly location- and industry-specific. Industries are licensed by councils and potential adverse effects are assessed on a case-by-case basis under the Resource Management Act.

The results of PM_{10} source apportionment studies undertaken at urban industrial monitoring sites are summarised in Table 11, taken from Table 3.4 of Davy & Trompetter (2020).

Table 11: PM_{10} source apportionment results for urban industrial areas

Site	Total PM_{10} µg/m³	Biomass burning µg/m³	Motor vehicles µg/m³	Secondary sulphate µg/m³	Marine aerosol µg/m³	Crustal material µg/m³	Industry µg/m³
Auckland -Penrose	16.5	2.4	4.1	1.1	6.6	0.5	1.0
Hawkes Bay – Awatoto	13.6	1.5	0.9	1.4	9.1	2.8	1.1
Wellington – Seaview	16.6	1.0	2.2	2.6	6.4	3.2	0.5
Nelson – Tahunanui	19.9	7.3	2.3	1.3	4.2	3.5	2.5
Christchurch -Woolston	23.6	6.2	4.9	0.6	3.8	4.5	0.0
Average	**18.0**	**3.7**	**2.9**	**1.4**	**6.0**	**2.9**	**1.0**

Source: Davy & Trompetter (2020)

Note: The average source contributions to PM_{10} shown above are derived from *receptor modelling* analyses and when summed may not equal the total PM_{10}, which is the *measured* concentration.

The results of $PM_{2.5}$ source apportionment studies undertaken at urban industrial monitoring sites are summarised in Table 12, taken from Table 3.3 of Davy & Trompetter (2020).

Table 12: $PM_{2.5}$ source apportionment results for urban industrial areas

Site	Total $PM_{2.5}$ µg/m³	Biomass burning µg/m³	Motor vehicles µg/m³	Secondary sulphate µg/m³	Marine aerosol µg/m³	Crustal material µg/m³	Industry µg/m³
Auckland -Penrose	7.1	1.8	2.3	0.8	0.6	0.4	0.2
Hawkes Bay – Awatoto	3.7	0.7	0.2	0.8	1.2	0.1	0.3
Wellington – Seaview	5.1	1.2	0.6	1.2	1.1	0.4	0.5
Christchurch -Woolston	10.3	3.9	2.6	1.2	0.4	0.3	0.0
Average	**6.6**	**1.9**	**1.4**	**1.0**	**0.8**	**0.3**	**0.3**

Source: Davy & Trompetter (2020)

Note: The average source contributions to $PM_{2.5}$ shown above are derived from *receptor modelling* analyses and when summed may not equal the total $PM_{2.5}$, which is the *measured* concentration.

We estimated the contribution of industrial sources in industrial airsheds and CAUs with suitable monitoring, based on the results of source apportionment as follows:

- Awatoto and Penrose industrial contributions were based on the results of PM_{10} and $PM_{2.5}$ source apportionment at these sites.
- Mt Maunganui and Lyttleton industrial contributions were assumed to be 2.5 µg/m³ PM_{10} and 0.5 µg/m³ $PM_{2.5}$. These are the maximum estimated industry contributions from source apportionment studies at industrial sites.
- The Tahunanui industrial contribution for PM_{10} was based on source apportionment undertaken at the site. $PM_{2.5}$ was assumed to be 0.5 µg/m³, which is the maximum estimated industry contribution from source apportionment at other industrial sites.
- The Marsden Point, Ngapuna, Woolston and Washdyke industrial contributions were assumed to be 1 µg/m³ PM_{10} and 0.3 µg/m³ $PM_{2.5}$. These are the average estimated industry contributions from source apportionment at industrial sites.

We estimated the contribution of industrial sources to PM in industrial areas without suitable monitoring, as described in section 4.3.2.

Windblown dust PM

Windblown dust was attributed using a *crustal material* fingerprint and assigned to **anthropogenic**.

Analysis of source apportionment datasets shows that urban concentrations of crustal material are lower on weekends compared with weekdays indicating that the generation of airborne crustal material in urban areas is largely due to human activities (including construction, demolition, earthworks, roadworks and movement of vehicles on roads and unpaved areas).

PM_{10} source apportionment results, provided in Table 3.4 of Davy & Trompetter (2020), show that the average estimated concentration of PM_{10} crustal material varies considerably, typically ranging from 0.5 to 3 µg/m³ in urban areas. Estimated concentrations are typically higher in industrial areas (3.2 µg/m³ at Seaview, 3.5 µg/m³ at Tahunanui, 4.5 µg/m³ at Woolston and 2.8 µg/m³ at Awatoto). The Dunedin monitoring site, which was influenced by

construction during the monitoring period, recorded the highest estimated concentration of 7.5 µg/m^3 crustal material PM_{10}.

To estimate the concentration of crustal material PM_{10} we subtracted all other individual source contributions (estimated by the methodology outlined in previous sections) from the total PM_{10} concentration. The remainder was assigned to crustal material PM_{10}.

$PM_{2.5}$ source apportionment results, provided in Table 3.3 of Davy & Trompetter (2020), show that the average estimated concentration of $PM_{2.5}$ crustal material ranges from 0.1 µg/m^3 to 0.8 µg/m^3. There does not appear to be a significant regional influence or any significant difference between urban and industrial sites.

We assigned the average crustal $PM_{2.5}$ concentration of 0.3 µg/m^3 (based on all monitoring sites) to all locations.

Davy & Trompetter (2020) report crustal material to actually be largely anthropogenic in origin, arising from activities such as motor vehicles (causing road abrasion, suspension and re-suspension of surface material), construction and industry. Consequently, the crustal material contribution is assigned to **anthropogenic.**

Sea spray PM

Sea spray PM was attributed using a *marine aerosol* fingerprint and assigned to **natural**.

Sea spray (largely sea salt) is the primary source of natural PM present in New Zealand urban atmospheres.

Research has shown that marine aerosol concentrations are relatively uniform across regions, with the primary influence on regional concentrations being the relative exposure to prevailing oceanic winds and the sheltering effect of mountain and hill ranges (Davy & Trompetter 2020).

We assigned a value for marine aerosol PM_{10} for each airshed based on the results of source apportionment undertaken in the airshed, or in the same region. The source apportionment results are summarised in Table 3.4 of Davy & Trompetter (2020).

Where results were not available for a region, we assigned results from a similar region (geographically). The PM_{10} marine aerosol concentration values assigned to each region or airshed are shown in Table 13.

Table 13: Marine aerosol PM_{10} values assigned to each region or airshed

Region or airshed	Marine aerosol concentration assumed	Based on source apportionment results from site(s)
All of Northland and Auckland	6.6	Average of all Auckland sites
All of Waikato, Rotorua, all of Manawatū-Whanganui	3.9	Tokoroa
Awatoto	9.1	Awatoto
Remainder of Hawkes Bay (excl Awatoto), all of Taranaki, remainder of Bay of Plenty (excl Rotorua)	4.5	Napier (Marewa Park)

Region or airshed	Marine aerosol concentration assumed	Based on source apportionment results from site(s)
Wellington Region	4.3	Average of Masterton, Wainuiomata & Upper Hutt
Nelson and Tasman Regions	3.6	Average of Tahunanui, Nelson & Richmond
Marlborough Region	2.0	Blenheim
Canterbury and West Coast Regions	6.9	Christchurch
Otago and Southland regions	4.9	Dunedin

$PM_{2.5}$ source apportionment results from Table 3.3 of Davy & Trompetter (2020) suggest there is no significant regional influence on the concentration of $PM_{2.5}$ marine aerosol. Concentrations typically range between 0.4 and 1.8 µg/m^3 for all regions.

We assigned the average $PM_{2.5}$ marine aerosol concentration of 1.2 µg/m^3 (based on all monitoring sites) to all locations.

Secondary PM

Secondary PM was attributed using a *secondary sulphate* fingerprint and assigned to **natural**.

Urban background PM_{10} and $PM_{2.5}$ includes secondary PM generated from atmospheric reactions of precursor gases, including VOCs, SO_2, NO_X and ammonia (**NH_3**).

Source apportionment studies show that secondary PM from gas-to-particle atmospheric reactions is generally dominated by secondary sulphate in New Zealand. Our oceanic location, narrow land mass, low industrial emissions, and minimal use of coal burning means that New Zealand does not have the secondary PM dominance seen in continental air masses.

Anthropogenic sources of sulphate include combustion of automotive fuels, shipping emissions, industrial and domestic combustion of coal and diesel. Natural sources include dimethyl sulphide production from marine organisms and volcanic emissions.

Davy & Trompetter (2020) suggest 1.0 µg/m^3 of annual average $PM_{2.5}$ as a reasonable estimate for the general population exposure to secondary sulphate aerosol across New Zealand. This value (1.0 µg/m^3) corresponds to the average of secondary sulphate $PM_{2.5}$ concentrations derived from source apportionment results across all New Zealand monitoring sites, as shown in Table 3.3 of Davy & Trompetter (2020). For secondary sulphate PM_{10}, the average derived from source apportionment (as shown in Table 3.4) is 1.45 µg/m^3.

We assigned an average secondary sulphate particulate concentration of 1.45 µg/m^3 for PM_{10} and 1.0 µg/m^3 for $PM_{2.5}$ (based on all monitoring sites) to all locations.

Source apportionment has shown that there are natural and anthropogenic sources of secondary sulphate aerosol in New Zealand. In some locations, particularly close to ports, the anthropogenic component of sulphate is likely to be significant. However, in most locations, **secondary sulphate is likely to be dominated by natural sources**. Secondary sulphate is therefore assigned to the **natural** component of PM_{10} and $PM_{2.5}$.

While sulphate is the principal component of secondary PM in New Zealand, other types of secondary particulate are likely to be present in urban PM.

We did not quantify other types of secondary PM. However, $PM_{2.5}$ mass closure analysis suggests that the mass contribution of unquantified secondary particulate is likely to be somewhat less than 5%.

4.3.2 Preliminary PM_{10} and $PM_{2.5}$ source attributions in areas without suitable monitoring data

This section describes the methodology for assigning source apportionment values in areas where the GNS empirical biomass relationship (described in section 4.3.1) was not applied because suitable monthly monitoring data were not available.

Unmonitored airsheds and urban areas outside airsheds

For **airsheds and urban areas outside airsheds without monitoring data**, the methodology was as follows:

- The **motor vehicle contribution** was calculated for all **airsheds** based on the method described in section 4.3.1. For **urban areas outside airsheds**, the motor vehicle contribution was assigned the lowest value from an urban airshed in the same region.
- Source attribution values for **biomass burning, industry, marine aerosol and secondary PM** were based on values from another area:
    - In locations where a PM_{10} or $PM_{2.5}$ monitoring value or a $PM_{2.5}$ / PM_{10} ratio was assigned from another area (as described in section 3.3), the source attribution values from the same area were also applied.
    - In locations where a PM_{10} or $PM_{2.5}$ monitoring value was assigned (as described in section 3.3), but suitable 2015-2018 monthly data were not available for source attribution, source attribution values were assigned from a similar location in the same region. We made assumptions to account for total concentration differences between airsheds:
        - For example, the average PM_{10} concentration in Kaitaia (16.0 µg/m^3) is higher than Whangarei (13.7 µg/m^3). From a review of the locations, we assumed the difference was likely due to higher levels of biomass burning in Kaitaia. On this basis, all source attribution values in Kaitaia were based on Whangarei except biomass burning, which was assigned a value 2.3 µg/m^3 above that for Whangarei.
        - The same approach was taken in all other locations (Kaitaia, Waihi, Porirua, Picton, Nelson City, Otago 1 and Otago 2 airsheds) except Waihi, where we assumed that the 1 µg/m^3 difference between Waihi and Morrinsville was likely due to an industrial source in Waihi.
- Source attribution values for crustal material were then estimated for all areas based on the method described in section 4.3.1.

Unmonitored rural areas

All **unmonitored rural areas** across New Zealand were assigned a total PM_{10} concentration of 9.2 µg/m^3 and a total $PM_{2.5}$ concentration of 4.4 µg/m^3 (as described in sections 3.3.3 and 3.3.4). For consistency with this approach, we developed universal source attribution profiles to be applied to all unmonitored rural areas across New Zealand.

An estimate of source attributions was made, based on the following assumptions:

- Biomass burning was assigned PM_{10} and $PM_{2.5}$ values of 0.9 µg/m^3 (for both), using the source apportionment results for Patumahoe.
- Motor vehicles were assigned PM_{10} and $PM_{2.5}$ values of 0 µg/m^3 (for both).
- Marine aerosol was assigned a PM_{10} value of 5.3 µg/m^3 and a $PM_{2.5}$ value of 1.2 µg/m^3, which are the averages of all source apportionment results across New Zealand.
- Secondary particulate was assigned a PM_{10} value of 1.5 µg/m^3 and a $PM_{2.5}$ value of 1.0 µg/m^3, which are the same values assigned to all other areas.
- Industry was assigned PM_{10} and $PM_{2.5}$ values of 0 µg/m^3 (for both).
- Crustal PM_{10} was assigned a value after subtracting all other individual source contributions (estimated as above) from the total PM_{10} concentration of 9.2 µg/m^3.
- Crustal $PM_{2.5}$ was assigned a value of 0.3 µg/m^3, which is the same value assigned to all other areas.

Unmonitored urban industrial areas

All **unmonitored urban industrial areas** across New Zealand were assigned a total PM_{10} concentration of 18.2 µg/m^3 and a total $PM_{2.5}$ concentration of 6.7 µg/m^3 (as described in section 3.3.4). For consistency, we developed universal source attribution profiles to be applied to all unmonitored urban industrial areas across New Zealand.

A preliminary estimate of PM_{10} source attributions was made, based on the following assumptions:

- Biomass burning was assigned a PM_{10} value that matched the $PM_{2.5}$ (as described in the following section).
- Motor vehicles were assigned a PM_{10} value of 2.9 µg/m^3, which is the average of source apportionment results for urban industrial areas. This approach recognises that the motor vehicle contribution is likely to be higher in industrial areas (compared to non-industrial areas) due to a higher proportion of heavy vehicle movements and more unsealed surfaces.
- Marine aerosol was assigned a PM_{10} value of 5.3 µg/m^3, which is the average of all source apportionment results across New Zealand.
- Secondary particulate was assigned a PM_{10} value of 1.45 µg/m^3, which is the same as the value assigned to all other locations.
- Industry was assigned a PM_{10} value of 1.0 µg/m^3, which is the average of source apportionment results for urban industrial areas.

- Crustal material PM_{10} was assigned a value after subtracting all other individual source contributions (estimated as above) from the total PM_{10} concentration of 18.2 µg/m^3.

A preliminary estimate of $PM_{2.5}$ source attributions was made, based on the following assumptions:

- Biomass burning was assigned a $PM_{2.5}$ value of 1.9 µg/m^3, which is the average of source apportionment results for urban industrial areas. This approach recognises that the biomass burning contribution is likely to be lower in industrial areas (compared to urban areas) because biomass burning is dominated by domestic heating emissions.
- Motor vehicles was assigned a $PM_{2.5}$ value of 1.4 µg/m^3, which is the average of source apportionment results for urban industrial areas. This is consistent with the approach for motor vehicle PM_{10}.
- Marine aerosol was assigned a $PM_{2.5}$ value of 1.2 µg/m^3, which is the same as the value assigned to all other locations.
- Secondary particulate was assigned a $PM_{2.5}$ value of 1.0 µg/m^3, which is the same as the value assigned to all other locations.
- Industry was assigned a $PM_{2.5}$ value of 0.3 µg/m^3, which is the average of source apportionment results for urban industrial areas.
- Crustal material was assigned a $PM_{2.5}$ value of 0.3 µg/m^3, which is the same as the value assigned to all other locations.

Unmonitored rural industrial areas

Source contributions in unmonitored rural industrial areas were based on the assumed source attribution for Marsden Point, which was based on the methodology described for areas with suitable monitoring data.

4.3.3 Adjusted PM_{10} and $PM_{2.5}$ source attributions overall

On average, the estimated source contributions described in previous sections accounted for 101% of the total measured PM_{10} concentration and 94% of the total $PM_{2.5}$ measured concentration. However, adjustments were required in some locations to better align the estimates with measured data. The methodology we developed to adjust the source attribution results for all locations, is described as follows:

- Individual source contributions were assumed to account for **100% of the total $PM_{2.5}$ concentration** (i.e. each source contribution was scaled so that the sum of the source contributions equalled the assigned total $PM_{2.5}$ concentration).
- **Biomass burning PM_{10}** was adjusted to equal biomass burning $PM_{2.5}$.
- **Crustal PM_{10}** was re-calculated based on the method described in section 4.3.1 (by subtracting all other individual source contributions from the total PM_{10} concentration). For consistency with the $PM_{2.5}$ source attribution, it was assumed that the minimum crustal PM_{10} contribution was equal to the $PM_{2.5}$ contribution at the same location.

In some locations, the sum of all individual sources was higher than the total PM_{10} concentration, even when crustal PM_{10} was set to equal crustal $PM_{2.5}$. In these locations:

- The contributions of marine aerosol, secondary sulphate and motor vehicle PM_{10} were then reduced proportionally so that the sum of individual sources was equal to the total PM_{10} concentration.
- This adjustment was applied in Thames, Whitianga, Whangamata, Tokoroa, Turangi, Masterton, Reefton, and Otago 1, Otago 3 and Otago 4 airsheds. The overall contribution of marine aerosol, secondary sulphate and motor vehicle PM_{10} was reduced by a maximum of 1.8 µg/m^3 and an average of 1.0 µg/m^3 across these airsheds. Adjusted contributions were within the range of source apportionment monitoring results from other locations and were considered realistic.

Summary of overall adjusted results

The overall adjusted results of the PM source attribution are shown in Appendix B and are also available in the *Source values* worksheet of the *HAPINZ 3.0 Sources* workbook.

4.3.4 PM source attribution in HAPINZ 3.0 versus HAPINZ 2.0

The wealth of source apportionment data provides an updated, robust evidential basis to better attribute the contribution of various sources to the exposure. However, the source descriptions and categories now available differ significantly from those used in HAPINZ 2.0 and the results will not be directly comparable as discussed below:

- HAPINZ 2.0 was based on primarily on emissions estimates for anthropogenic sources supplemented by source apportionment for natural sources. Consequently, this study relied on the source categories from emissions inventories which tend to be discrete (e.g. motor vehicle exhaust *versus* domestic heating).
- In contrast, the comprehensive source apportionment dataset used for HAPINZ 3.0 derives sources of PM from the compositional analysis of samples collected at the same monitoring stations that PM_{10} or $PM_{2.5}$ ambient concentrations are measured. Each source is identified by specific groupings of elements (fingerprints) and the contribution of each of those sources to total PM concentrations is calculated.

Table 14 compares the source descriptions and methods used in HAPINZ 2.0 with those now available for HAPINZ 3.0. **The key differences are largely in windblown dust now being considered in the anthropogenic category and secondary PM being considered in the natural category** – these affect the allocation of the subsequent health effects.

The source apportionment data and how they have been used to derive the HAPINZ 3.0 PM source attributions are available as a separate deliverable as follows:

Sridhar S & Metcalfe J (2021). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Sources*. Excel model prepared by S Sridhar and J Metcalfe for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, February 2021.

Table 14: Comparison of source attribution methods used in HAPINZ 2.0 and HAPINZ 3.0

Source description	HAPINZ 2.0 source attribution method	HAPINZ 3.0 source attribution method
Domestic fires	The contribution of domestic fires to anthropogenic PM_{10} in HAPINZ 2.0 is estimated from emissions inventory data. The domestic fires contribution is assigned to **anthropogenic**.	The contribution of domestic fires in HAPINZ 3.0 is estimated from source apportionment data (2001-2018) using the biomass burning fingerprint or an empirical method using $PM_{2.5}$ concentrations. Davy & Trompetter (2020) report the biomass burning signal is dominated by domestic fires in urban areas of New Zealand. The domestic fires contribution is assigned to **anthropogenic.**
Open burning in urban areas	The contribution of urban open burning to anthropogenic PM_{10} in HAPINZ 2.0 is estimated from emissions inventory data. Open burning was included as a separate source in HAPINZ 2.0 because emission inventories identified it as a key source of PM_{10} in urban areas. The urban open burning contribution is assigned to **anthropogenic.**	The contribution of rural open burning to PM is **not assessed** in HAPINZ 3.0 due to insufficient data. Open burning of waste is now banned in most polluted urban areas and source apportionment studies have not identified it as significant in these areas. We identified urban areas where open burning still occurs and assessed its likely contribution to be typically no more than 8% of biomass burning PM and incorporated it in the domestic fires source (see section 4.2.1).
Open burning in rural areas	The contribution of rural open burning to anthropogenic PM_{10} in HAPINZ 2.0 is estimated from emissions inventory data (as above for open burning in urban areas). The rural open burning contribution is assigned to **anthropogenic.**	The contribution of rural open burning to PM is **not assessed** in HAPINZ 3.0 due to insufficient data. Burning of crop residues is a significant source of PM emissions in some rural locations in New Zealand. However, we do not have monitoring data, emissions are intermittent, and very few people are exposed.
Industry	The contribution of industry to anthropogenic PM_{10} in HAPINZ 2.0 is estimated separately for industries with tall stacks and those without, using emissions inventory data. The industry contribution is assigned to **anthropogenic**.	The contribution of industry in industrial areas in HAPINZ 3.0 is estimated from source apportionment data (2001-2018) where available but this is an approximation only. Better data on the contribution of industrial emissions to exposure are not readily available. The industry contribution is assigned to **anthropogenic**.
Windblown dust	The concentration of windblown dust/soil in HAPINZ 2.0 was estimated from source apportionment data (2001-2009). The windblown dust contribution is assigned to **natural**.	The concentration of windblown dust in HAPINZ 3.0 is estimated from source apportionment data (2001-2018) using the crustal material fingerprint. Davy & Trompetter (2020) report crustal material to be largely anthropogenic in origin, arising from activities such as motor vehicles (causing road abrasion, suspension and re-suspension of surface material), construction and industry. However, the specific sources vary between locations. Consequently, the windblown dust contribution is now assigned to **anthropogenic**.
Sea spray	The concentration of sea spray in HAPINZ 2.0 is estimated from source apportionment data (2001-2009). The sea spray contribution is assigned to **natural**.	The concentration of sea spray in HAPINZ 3.0 is estimated from source apportionment data (2001-2018) using the marine aerosol fingerprint. The sea spray contribution is assigned to **natural**.

Source description	HAPINZ 2.0 source attribution method	HAPINZ 3.0 source attribution method
Secondary PM from all sources	The contribution of secondary PM to PM_{10} is **not assessed** in HAPINZ 2.0 because data were not available at the time from source apportionment or emissions inventories (which estimate primary emissions).	The contribution of secondary PM In HAPINZ 3.0 is estimated from source apportionment data (2001-2018) using the secondary sulphate fingerprint. Secondary sulphate can arise from both natural and anthropogenic sources (Davy & Trompetter 2020). However, with significant reductions in fuel sulphur it is more likely to be dominated by the former (except around ports with ships using bunker or residual oil as fuels). The secondary PM contribution is assigned to **natural**

4.3.5 Motor vehicle NO_2

We assessed the effects of exposure to NO_2 using exposure estimates from the Waka Kotahi NVED tool. These estimates are based on estimated *background* concentrations combined with estimated *roadside* concentrations. The NVED tool focusses on assessing exposure due to road transport emissions but the estimated background concentrations are based on the results of monitoring, which may be influenced by other sources of NO_2 for which we have little data in the way of source breakdowns.

While **we defaulted to assigning all effects of NO_2 to motor vehicles**, we considered this assumption was reasonable given motor vehicles are likely to be responsible for approximately 90% of all NO_2 exposure in urban areas.

4.3.6 Uncertainty

We have developed a nationally consistent source apportionment methodology based on best available data. At a national level, we estimate the uncertainty in the HAPINZ 3.0 source apportionment allocations for domestic fires to be less than +/-10%. This is a qualitative assessment based on the team's expert judgement.

For other sources, the uncertainty in source apportionment allocations is likely to be higher. We consider our methodology delivers reasonable approximations of the relative source contributions for the other key categories (sea spray, motor vehicles, secondary PM and windblown dust) at a national level. However, the estimates may not be realistic for individual airsheds.

For motor vehicles, the empirical relationships between emissions density and the contribution of motor vehicles to ambient PM are based on limited data measured over different time periods. However, in the absence of better information, we consider these relationships provide a reasonable estimate of the likely contribution of motor vehicles to PM_{10} and $PM_{2.5}$, at least in urban areas.

As shown in Figures 17 and 18, the best fit relationships between motor vehicle PM and motor vehicle emission density by CAU have intercepts of 1.6 $\mu g/m^3$ for PM_{10} and 0.5 $\mu g/m^3$ for $PM_{2.5}$. In theory, areas with no motor vehicle emissions should have no motor vehicle PM (i.e. the graphs should have an intercept of zero). However, urban air quality is influenced by

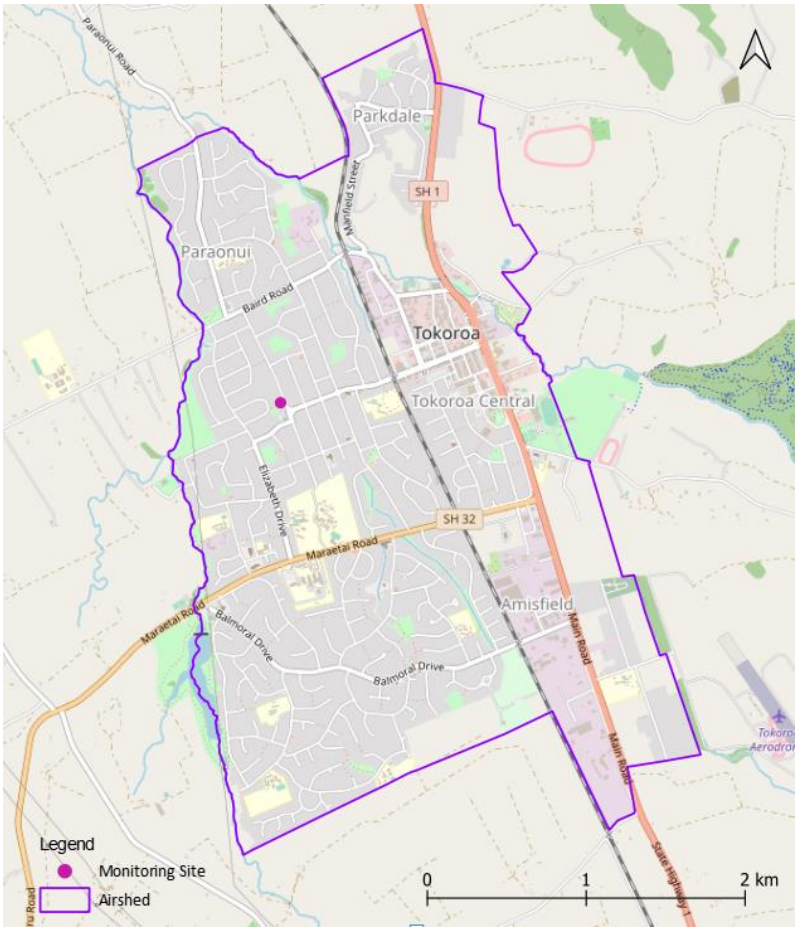
emissions across the entire urban area, not just emissions within the immediate CAU. By way of example, the Tokoroa monitoring site is in a CAU with very low traffic density (see Figure 19). However, ambient air quality at the site will be influenced by State Highway 1 and State Highway 32, both of which pass through the airshed.

The empirical relationship effectively establishes an urban background component of motor vehicle PM. This may overestimate the contribution of motor vehicle emissions in some locations, including small airsheds without state highways.

As discussed in section 3.3.8, development of exposure models for transport is an area of ongoing research. We recommend further investigation to determine whether source apportionment results (which estimate the contribution of motor vehicles to measured concentrations) could be used to validate roadside concentration estimates and account for the road component of monitoring results.

GNS Science (Davy & Trompetter 2020) suggest that it may also be possible to extract further source information from routine monitoring data with more sophisticated data processing (such as a cognitive computing/machine learning approach) trained by their observational knowledge of diurnal, weekly and seasonal temporal patterns exhibited by different source categories, particularly for the influence of motor vehicle emissions on urban air quality.

Figure 19: The location of the Billah St (Tokoroa) monitoring site relative to state highways



Based on the current level of understanding, we estimate the uncertainty in our source apportionment allocations for domestic fires in HAPINZ 3.0 is likely to be less than +/-10%. The uncertainty for other sources is likely to be higher.

4.3.7 Summary of our approach

Disaggregation of **$PM_{2.5}$ and PM_{10} by source** was based on actual monitoring (source apportionment) where available or use of proxy data where no monitoring was undertaken. PM was assigned to the following key categories:

- domestic fires used for home heating - based on a biomass burning fingerprint (where available) or an empirical method based on $PM_{2.5}$
- motor vehicles encompassing exhaust, brake/tyre wear and re-suspended road dust - based on a motor vehicle fingerprint
- industry - based on a local industry fingerprint (where available)
- windblown dust from sources such as construction, land use, industry and the movement of motor vehicles (e.g. road abrasion, suspension and re-suspension of surface material) - based on a crustal material fingerprint
- sea spray - based on a marine aerosol fingerprint
- secondary PM resulting from gases emitted from natural and anthropogenic sources reacting in the atmosphere to form particles - based on a secondary sulphate fingerprint.

We used source apportionment to approximate the likely contribution of industry to PM exposure in areas that were identified as industrial. We were not able to quantify the contribution of industry to PM in other areas.

We identified airsheds where open burning occurs and assessed its likely contribution to be typically no more than 8% of biomass burning PM in urban areas (based on emissions inventory data). Urban open burning was captured in the domestic fires source. We were not able to assess rural open burning.

We were unable to assess the impact of rail, aviation and shipping due to lack of suitable data but estimate that these sources are unlikely to contribute more than 5% to urban exposures.

In the absence of source apportionment data, we assigned all effects of NO_2 to motor vehicles. We estimate that motor vehicles are likely to be responsible for approximately 90% of all NO_2 exposure in urban areas.

5. Selecting health impacts and calculating the health burden

Exposure to air pollution may damage human health, depending on the pollutant, the duration of exposure and the susceptibility of the exposed population (which is often related to age). Adverse effects include morbidity (increased illness and disease) or, in some instances, premature mortality (death).

This chapter describes the health impacts selected for the HAPINZ 2.0 study, reviews developments in epidemiology that have occurred since that time and their relevance to New Zealand, and presents the health impacts we selected for HAPINZ 3.0. We describe also the methods used to analyse health data and calculate the burden of ill-health caused by air pollution in New Zealand in 2016.

Critically important in assessments of this kind is the exposure-response functions (**ERF**), the quantitative relation between exposure to air pollution and the frequency of morbidity or mortality. As part of HAPINZ 3.0, we conducted a detailed cohort study to develop New Zealand-specific ERFs for selected mortality and morbidity impacts. How this was done and the key findings are discussed in this chapter.

5.1 Approach used in HAPINZ 2.0

HAPINZ 2.0 assessed the following primary health impacts using the ERFs shown below (expressed as the increase in risk for a specified increase in the concentration of the pollutant in outdoor air):

- **Premature mortality** from long-term exposure (defined by the PM_{10} annual mean)
  - adults, aged 30 years and over (Hales *et al* 2012): 1.07 (95% CI: 1.03, 1.10) [19] per 10 µg/m^3
  - infants, aged 1 month to 1 year (Lacasaña *et al* 2005): 1.05 (95% CI: 1.02, 1.08) per 10 µg/m^3
- **Hospital admissions** from short-term exposure (PM_{10} daily mean)
  - cardiac hospital admissions, all ages (APHEIS 2004): 1.006 (95% CI: 1.003, 1.009) per 10 µg/m^3
  - respiratory hospital admissions, all ages (APHEIS 2004): 1.1 (95% CI: 1.06, 1.017) per 10 µg/m^3

[19] A relative risk of 1.07 means the risk increases by 7% per pollution increment. The bracketed numbers are the 95% confidence intervals, which mean there is a 95% probability that the true risk increase ranges between 3% and 10%.

- **Restricted activity days** from long-term exposure ($PM_{2.5}$ annual mean[20])
    - restricted activity days, all ages (ALA 1995 based on Ostro 1987): 0.9 (lower/upper bounds: 0.5, 1.7) per 10 µg/m^3

The following **population sub-group impacts** were also assessed:

- Premature mortality for Māori adults, aged 30 years and over (Hales *et al* 2012): 1.20 (95% CI: 1.07, 1.33) per 10 µg/m^3
- Respiratory hospital admissions for children aged 1 to 4 years (Barnett *et al* 2005): 1.01 (95% CI: 1.006 to 1.017) per 10 µg/m^3 (PM_{10} daily mean)
- Respiratory hospital admissions for children aged 5 to 14 years (Barnett *et al* 2005): 1.03 (95% CI: 1.0, 1.05) per 10 µg/m^3 (PM_{10} daily mean)

Sensitivity analyses were conducted using the extreme values in 95% confidence intervals (**CI**) or lower/upper bounds of selected exposure-response functions.

As a **cross check**, a comparison was also undertaken for one health outcome – mortality for all adults aged 30 years and over – using an indicative exposure-response function for $PM_{2.5}$ based on the results of international studies as there were no data available at this time for New Zealand:

- All adults, aged 30 years and over: 1.09 per 10 µg/m^3 ($PM_{2.5}$ annual mean)

5.2 Developments since HAPINZ 2.0

This section reviews the developments in the literature on health impacts of long-term air pollution exposure since HAPINZ 2.0. We considered robustness of exposure-response functions by pollutant, the availability of exposure and health outcome data, and the likely public health significance of the exposure pathway in order to estimate the most relevant health effects in New Zealand.

5.2.1 PM and constituents health endpoints

Particulate matter in ambient air is a complex and variable mixture of inorganic and organic chemicals as well as biological material. Both short- and long-term exposure to PM are associated with health endpoints, including premature mortality and a range of cardiovascular and respiratory diseases.

There is growing evidence of effects of PM exposure on adverse birth outcomes (Laurent *et al* 2016; Huang *et al* 2018; Seeni *et al* 2019), childhood respiratory diseases (Guarniera & Balmes 2014; Bowatte *et al* 2015; Burte *et al* 2016; Hehua *et al* 2017; Khreis *et al* 2017; Muñoz *et al* 2019; Williams *et al* 2019), and diabetes and dementia (WHO 2013; Peters *et al* 2019). Disruptions of the immune system contribute to toxicity of inhaled pollutants, especially in the case of respiratory diseases (Falcon-Rodriguez *et al* 2016).

Considerable scientific effort has been directed to identifying features of PM that are primarily responsible for health impacts. Size of particles matters; there are stronger and more

[20] HAPINZ 2.0 assumed that 60% of annual PM_{10} in urban areas and 40% of annual PM_{10} in rural is $PM_{2.5}$.

consistent associations between health impacts and $PM_{2.5}$ than for PM_{10}. Otherwise, there is not, at present, a clear picture of the particular constituents that are responsible for the bulk of the health effects.

Sulphur content may be important, according to some research. A recent study reported that the sulphur content of PM was most strongly associated with mortality (Beelen *et al* 2015).

Another study concluded that:

> …the association between PM in air pollution and lung cancer can be attributed to various PM components and sources. PM containing S and Ni might be particularly important. (Raaschou-Neilsen *et al* 2016)

Ostro *et al* (2015) reported that $PM_{2.5}$ from high sulphur fuel combustion was associated with mortality in teachers. However, there were no significant associations with $PM_{2.5}$ from petrol or diesel for land-based transport, wood smoke or meat cooking. Thurston *et al* (2016) investigated associations between $PM_{2.5}$ from different sources and ischaemic heart disease mortality. Sources of $PM_{2.5}$ (soil, motor vehicle traffic, steel industry, non-ferrous metals industry, coal combustion, oil combustion, salt particles, and biomass burning) were estimated based on measurement of elemental tracers. In models adjusted for 42 individual level variables, but without random effects or contextual variables, $PM_{2.5}$ mass and several elements including S, Se, As, Cl, Pb and Fe were statistically significant predictors. In fully adjusted models, only total $PM_{2.5}$ from coal combustion and from non-ferrous metals industry sources were significant; the strongest association was for coal combustion.

A report on the effects of black carbon (**BC**) concluded that:

> Cohort studies provide sufficient evidence of associations of all-cause and cardio-pulmonary mortality with long-term average BC exposure. Studies of short-term health effects suggest that BC is a better indicator of harmful particulate substances from combustion sources (especially traffic) than undifferentiated particulate matter (PM) mass, but the evidence for the relative strength of association from long-term studies is inconclusive. … BC may not be a major directly toxic component of fine PM, but it may operate as a universal carrier of a wide variety of chemicals of varying toxicity to the lungs, the body's major defence cells and possibly the systemic blood circulation. (WHO 2012)

A recent systematic review of studies reporting effect estimates for the association of ambient BC, or elemental carbon, (**EC**) and $PM_{2.5}$, with cardiovascular diseases (**CVD**) concluded:

> The evidence was not sufficient to determine if associations with BC were distinct, or stronger, than associations with $PM_{2.5}$. (Kirrane *et al* 2019)

Health impacts from exposure to BC could be estimated based on exposure-response functions from WHO (2012) or from a more recent cohort study undertaken in Europe (Strak *et al* 2021). The latter includes a hazard ratio (HR)[21] for natural-cause mortality of 1.08 (95% CI: 1.07, 1.10) per 0.5 x 10^{-5}/m BC.

[21] The hazard ratio is the measure of association in survival analyses (it is the ratio of the rate of events in an exposed group to that in an un-exposed group). It is comparable to a risk ratio for present purposes.

In summary, the life shortening effect of PM is well established, but it is not possible to identify specific components of PM that are primarily responsible for this effect. There is emerging evidence of higher toxicity of fossil fuel combustion sources of PM, especially PM with high sulphur content, than PM from other sources (such as sea spray or dust).

5.2.2 NO_2 health endpoints

A 2013 review reported statistically significant associations between NO_2 and mortality, but with substantial *heterogeneity*[22] of the effect size (Hoek *et al* 2013) while a meta-analysis of short- and long-term effects reported more consistent associations between long-term exposure to NO_2 and mortality (Faustini *et al* 2014). In the meta-analysis, the associations were broadly similar in strength to those of $PM_{2.5}$, and in four studies, the NO_2 results were not substantially altered following adjustment for $PM_{2.5}$.

Because of difficulties in apportioning health impacts of a complex mixture of pollutants to individual components, the UK Committee on the Medical Effects of Air Pollutants (**COMEAP**) suggested that the choice of effect size for NO_2

> …will depend on whether the aim is to assess the effects of reductions in concentrations of NO_2 itself, the effects of reductions in NO_2 as well as of other co-varying pollutants, or to estimate the burden of the air pollution mixture as a whole. (COMEAP 2018)

Concerning effects of long-term exposure on mortality, several members of the committee recommended using an adjusted coefficient of 1.006 to 1.013 per 10 µg/m^3 of NO_2, although other members of the committee considered that the evidence was not strong enough to justify this. Regardless, all members agreed that:

> To assess the health benefits of interventions that reduce a mixture of traffic-related pollutants it is recommended that the unadjusted NO_2 coefficient of 1.023 (95% CI: 1.008, 1.037) per 10 µg/m^3 annual average NO_2 is used, taking NO_2 as a marker for the mixture, to calculate the benefits of changes in the mixture. (COMEAP 2018)

The committee also noted that there was stronger evidence of effects of short-term NO_2 exposure on respiratory diseases.

There is increasing evidence that traffic-related air pollution is associated with the development of asthma (Bowatte *et al* 2015; Burte *et al* 2016; Khreis *et al* 2017; Khreis & Nieuwenhuijsen 2017), and we note that exposure to NO_2 is frequently used as a metric for traffic-related air pollution in epidemiological studies. A recent meta-analysis reported a risk estimate for onset of childhood asthma of 1.05 (95% CI: 1.02, 1.07) per 4 µg/m^3 NO_2 (Khreis *et al* 2017). Another meta-analysis reported associations between air pollution exposure and asthma exacerbations (Orellano *et al* 2017). In that study, statistically significant associations were reported for NO_2, SO_2 and $PM_{2.5}$ exposure and episodes of asthma in both children and adults. A recent study estimated the global burden of childhood asthma attributable to NO_2

[22] Heterogeneity in meta-analysis refers to the variation in study outcomes between studies. It is normal when a number of different studies (which are likely to cover different exposure ranges and different population groups) are combined and analysed.

exposure (Achakulwisut *et al* 2019). Exposure estimates at 100m resolution were used in this global study.

The United States Environmental Protection Agency (**US EPA**) has concluded that there is sufficient evidence of a causal effect of both long- and short-term NO_2 exposure on respiratory diseases, but that

> …evidence is suggestive of, but not sufficient to infer, a causal relationship with cardiovascular effects and diabetes, total mortality, birth outcomes, and cancer. (US EPA 2016)

Double counting

The WHO recommends quantification of the health impacts of air pollution based on three pollutants – PM (PM_{10} and $PM_{2.5}$), NO_2 and O_3 – and states that:

> …for any particular health outcome and exposure period (long-term or short-term exposure), estimated impacts of the three pollutants should not be added without recognising that this will, in most practical circumstances, lead to some overestimation of the true impact. Impacts estimated for one pollutant only will, on the other hand, underestimate the true impact of the pollution mixture, if other pollutants affect that same health outcome independently. (WHO 2013)

Note: King's College (2015) recommends reducing the effects of NO_2 by 30% to account for the possible overlap in effects when using exposure-response functions developed from *single* pollutant models. However, HAPINZ 3.0 uses functions derived from *multi*-pollutant models[23] so total mortality from long-term exposure to $PM_{2.5}$ and NO_2 is calculated by simply adding together the effects of $PM_{2.5}$ and NO_2.

5.2.3 Sensitive populations

People with poor diet or chronic disease, children, the elderly, pregnant women and certain ethnic groups may be particularly susceptible to the health impacts of air pollution (Wang *et al* 2016; Tibuakuu *et al* 2018).

> … multiple lifestyle related factors may play a role in the stronger effects observed in less-educated subjects. These may include dietary factors such as lower fruit and antioxidant intake … higher risk of obesity or other pre-existing diseases, higher actual exposures than assumed in the studies, lack of air conditioning and possibly interaction with other risk factors such as poorer housing conditions. (Hoek *et al* 2013)

[23] Multi-pollutant models assume that more than one pollutant may be contributing to the effects and generate exposure-response functions based on the combination. For example, a single pollutant model for $PM_{2.5}$ seeks to explain the change in adverse effects based on $PM_{2.5}$ concentrations alone and will likely have a high relative risk factor, e.g. 1.205 per 10 µg/m^3 $PM_{2.5}$ with wide 95% confidence intervals. However, if part of the observed effect is actually due to NO_2 then a two pollutant model covering $PM_{2.5}$ and NO_2 is better able to explain the changes and will likely reduce the relative risk factor for $PM_{2.5}$, e.g. 1.105 per 10 µg/m^3 $PM_{2.5}$ with narrower confidence intervals. Because the model is based on both pollutants, the effects of each are additive.

The US EPA concluded that there is adequate evidence

> … that children are at increased risk of a $PM_{2.5}$-related health effects... [and that] non-white people are at increased risk ... (US EPA 2018)

There was also "suggestive evidence" that

> … populations with pre-existing cardiovascular and respiratory disease, that are overweight or obese, with genetic variants in genes in the glutathione pathway and oxidant metabolism, or that are of low socio-economic status are at increased risk … (US EPA 2018)

A previous cohort study of mortality in relation to PM_{10} exposure in New Zealand reported suggestive, but inconclusive, evidence that Māori are particularly susceptible to the life shortening effect of PM (Hales *et al* 2012).

5.3 What we did in HAPINZ 3.0

For HAPINZ 3.0, we started with the set of health impacts selected in HAPINZ 2.0 (which were based largely on internationally-derived ERFs) then investigated New Zealand-specific ERFs using a national cohort study (section 5.3.1). Given the strength of the associations we found in the cohort study, we undertook a suite of sensitivity analyses to explore the robustness of the results (section 5.3.2) before confirming which metrics should be used in HAPINZ 3.0 (section 5.3.3). The final selection of ERFs also included appropriate indicators to enable assessment of childhood asthma in New Zealand.

Note: Based on developments in the literature, the availability of monitoring data in New Zealand, the project scope and the relative impact of effects, **the health impacts we assessed in HAPINZ 3.0 were based on long-term exposure** (defined by annual average concentrations).

As discussed in section 2.2, the dominant social cost is from the reduction in life expectancy through the gradual, cumulative effects of air pollution on chronic disease, with the health burden due to chronic exposure potentially 10 times greater than that for acute exposure (WHO 2006).

5.3.1 Development of NZ-specific ERFs – the cohort study

One of the key objectives of the HAPINZ 3.0 programme of work was to develop a suite of New Zealand-specific ERFs for critical mortality and morbidity impacts relevant to New Zealand.

The *HAPINZ 3.0 Cohort Study* built on the initial work of Hales *et al* (2012), which was used in HAPINZ 2.0 to estimate premature mortality in adults with PM_{10} as a proxy for all air pollution, and was undertaken to:

- Assess associations with exposure to both $PM_{2.5}$ (chosen as the principal exposure metric for mortality) and NO_2 (highlighted as a knowledge gap due to data limitations in HAPINZ 2.0)

- Investigate the susceptibility of Māori to air pollution (indicated as a potential issue in HAPINZ 2.0) and extend the analysis to other ethnicity subgroups, such as Pacific peoples
- Analyse for morbidity impacts (such as hospitalisations) as well as mortality impacts
- Explore the development of a robust ERF for childhood asthma.

We also assessed PM_{10} exposure to compare with the ERFs used in HAPINZ 2.0.

The following sections summarise the methodology followed in the *HAPINZ 3.0 Cohort Study* and our findings. A detailed description may be found in Hales *et al* (2021).

Note: We report the *HAPINZ 3.0 Cohort Study* ERFs to three decimal places in this report but reduce them to two decimal places when comparing our results with those of other researchers (who typically publish ERFs to only two decimal places).

Data sources

Detailed data are now available on national mortality rates and hospital admissions for up to the past 20 years. The Stats NZ Integrated Data Infrastructure (**IDI**) has made the analysis of linked environmental, social and health outcome data much more practical than in the past.

The cohort study used a national dataset covering the entire New Zealand population (4.2 million people in 2013) with 2013 Census data linked to the New Zealand Mortality Collection data (MoH 2021a) and the National Minimum Dataset of publicly funded hospital discharges (MoH 2021b). Participants were spatially referenced at the *meshblock*[24] and CAU level.

For annual $PM_{2.5}$ and PM_{10} exposures in 2016, we used the dataset by CAU developed from ambient monitoring data (see section 3.3.4). For 2006, sufficient monitoring data were available to develop concentration estimates by CAU in accordance with the HAPINZ 3.0 PM exposure methodology. Initial models were analysed in relation to the estimated exposure at the place of *usual residence* at the time of the 2013 Census.

With NO_2, it is necessary to assess exposure at sub-kilometre level, due to the strong exposure gradients near to roads, given the importance of motor vehicle traffic as a source and the relatively rapid decay of NO_2. For the same reason, use of address of usual residence as a defining factor is a more serious limitation for NO_2 than it is for PM.

For NO_2, we carried out the analysis at meshblock scale. For annual NO_2 exposure in 2016, we used the concentration estimates at a 50m resolution provided from the Waka Kotahi VEMT/NVED tool averaged with population weighting to 2013 meshblock boundaries to get meshblock average estimates (see section 3.3.6). The meshblock scale estimates were averaged at CAU scale with population weighting for sensitivity analyses. For annual NO_2 exposure in 2006, a method was developed for scaling the 2016 VEMT/NVED dataset using passive sampling and continuous monitoring data from earlier years (see section 7.3.3 for details on the scaling method).

[24] A meshblock is the smallest administrative unit, containing approximately 100 people whereas a CAU can contain between several hundred and several thousand people.

Note: The original objective of the HAPINZ 3.0 study was to undertake a *preliminary* only investigation of NO_2 impacts. However, with the evolution of the datasets and emissions models over the period of our research, we were able to undertake more detailed analyses. HAPINZ 3.0 represents the first attempt to comprehensively assess the impacts of NO_2 exposure for New Zealand.

Statistical analyses – mortality

We fitted Poisson regression models of mortality in adults (aged 30 and above) for all causes, all non-external causes and by sub-group of major cause: cardiovascular diseases, ischaemic heart disease, stroke, respiratory diseases, asthma and lung cancer.

We adjusted for confounding by ambient temperature, age, sex, ethnicity, personal income, education and smoking status in the models.

As discussed earlier, air pollution has both short-term and long-term health impacts. The long-term effects should ideally be assessed with reference to lifetime (including *in utero*) exposures, but this information was not available for HAPINZ 3.0.

Where possible, we linked historical addresses to derive more inclusive estimates of past exposure. We analysed health outcome data based on average exposures in 2006 and 2016, for both $PM_{2.5}$ and NO_2, restricting the analyses to people with the same address of usual residence in 2013 and in 2008.

Mortality in adults

We followed a step-wise process in analysing the mortality risks.

Initially, the effects of all pollutants were assessed in *single pollutant* models with adjustment for age (in 5-year groups), sex and ethnicity (step 1). Next, we added individual income, education and smoking history to the model (step 2). This step is necessary to reveal the true effect of air pollution, while removing possible confounding effects of social factors affecting mortality risk.

Additional pollutants were added to the single pollutant models to create *two pollutant* models (step 3). For the $PM_{2.5}$ or PM_{10} single pollutant models, the additional pollutant was NO_2. For the NO_2 single pollutant model, the additional pollutant was $PM_{2.5}$. The purpose here was to investigate which components of air pollution are most important in causing health impacts. Finally, we adjusted for annual mean temperature (step 4).

Table 15 shows how risks of mortality from natural causes in adults changed with each adjustment step.

In step 2, after including income, education and smoking, the change in mortality for every 10 $\mu g/m^3$ increase in concentration was reduced somewhat. The inclusion of social factors in the model controls for independent effects on the underlying risk of death and provides a more accurate assessment of the true effects of air pollution.

Mortality risks for PM_{10} and $PM_{2.5}$ were reduced further when NO_2 and temperature were considered in the final step; however, those for NO_2 were largely unchanged. This suggests

that the association between PM and mortality is confounded by that of temperature. It is therefore necessary to include temperature in the model in order to estimate the true association.

What this means is that **while PM_{10} based on a single pollutant model is a good measure (proxy) for the effects of all air pollution, much of its association with mortality can be explained by $PM_{2.5}$ and NO_2**. Consequently, we opted for the two pollutant ($PM_{2.5}$ and NO_2) model mortality risks shown in the final line of Table 15.

Table 15: Change in central estimate of mortality risk from single and two pollutant models

Adjustment step	PM_{10} (per 10 µg/m³)	$PM_{2.5}$ (per 10 µg/m³)	NO_2 (per 10 µg/m³)
Single pollutant models			
1. Adjusted for age, sex, ethnicity	1.168	1.288	1.100
2. Adjusted for age, sex, ethnicity, income, education, smoking	1.117	1.205	1.092
Two pollutant (PM and NO_2) models			
3. Adjusted for age, sex, ethnicity, income, education, smoking	n/a	1.184	1.070
4. Adjusted for age, sex, ethnicity, income, education, smoking, temperature	**1.024**	**1.105**	**1.097**

Mortality and $PM_{2.5}$

Our results for $PM_{2.5}$ are comparable with the findings from a recent meta-analysis: 1.08 (95% CI: 1.06, 1.09) per 10 µg/m³ in single pollutant models (Chen & Hoek 2020). However, those authors noted that a subset of five studies that ran two pollutant models with $PM_{2.5}$ and NO_2 also observed a reduction in $PM_{2.5}$ effect to 1.02 (95% CI: 1.00, 1.04). The authors concluded:

> Two pollutant models can be difficult to interpret when the correlation between pollutants is high or exposure for pollutants is assessed with different methods or at a different spatial resolution. (Chen & Hoek 2020).

In models adjusted for NO_2, a European-wide cohort study reported a hazard ratio (**HR**) of 1.08 (1.05, 1.11) per 5 µg/m³ $PM_{2.5}$ (Strak *et al* 2021).

Mortality and NO_2

For the associations between **long-term exposure to NO_2 and mortality, our results are substantially higher than recently reported in several international studies** (Table 16).

A 2020 meta-analysis reported a pooled estimate of 1.02 (95% CI: 1.01, 1.04) per 10 µg/m³ NO_2 (Huangfu & Atkinson 2020). However, three of the individual studies in that analysis reported effect sizes of greater than 1.10 and are therefore comparable with our findings. Another recent study examined the effect of using different methods of exposure assessment in the Netherlands. In that study, results for exposure based on a dispersion model, the most comparable approach to that used in the present study, were: 1.015 (95% CI: 1.005, 1.024)

per 10 µg/m^3 NO_2 (Klompmaker *et al* 2021). In models adjusted for $PM_{2.5}$, a recent European-wide cohort study reported an HR of 1.05 (1.03, 1.07) per 10 µg/m^3 NO_2 (Strak *et al* 2021).

One reason for the stronger association in our study may be the relatively low levels of exposure in New Zealand, compared to other countries. The reason is that at low pollution levels, the slope of the exposure-response curve may be steeper than in more highly polluted circumstances, such as in the United States or Europe (Strak *et al* 2021). In all epidemiological studies of air pollution and health impacts that rely on place of residence for exposure assessment, the true association is likely to be weakened by misclassification of exposure since people may change address, and do not spend all of their time at home.

Table 16: Exposure-response functions for long-term exposure to NO_2 and mortality (per 10 µg/m^3)

Mortality	Author	Year	Central estimate	Lower 95% CI	Upper 95% CI	No of studies
Natural causes	Strak	2021	1.09	1.07	1.10	8
	Stieb	2020	1.03	1.02	1.05	39
	Stieb	2020a[1]	1.03	1.01	1.04	32
	Huangfu	2020	1.02	1.01	1.04	24
	Atkinson	2018	1.02	1.01	1.03	20
	Faustini	2014	1.04	1.02	1.07	12
	Hoek	2013	1.06	1.03	1.08	12
	HAPINZ 3.0	**2022**	**1.10**	**1.07**	**1.12**	**1**
Cardiovascular	Strak	2021	1.09	1.06	1.12	8
	Stieb	2020	1.07	1.00	1.16	29
	Stieb	2020a	1.03	1.01	1.05	23
	Atkinson	2018	1.03	1.02	1.05	15
	Faustini	2014	1.14	1.09	1.19	16
	HAPINZ 3.0	**2022**	**1.09**	**1.05**	**1.13**	**1**
Respiratory	Strak	2021	1.10	1.04	1.17	8
	Stieb	2020	1.09	1.01	1.19	29
	Stieb	2020a	1.03	1.02	1.05	24
	Huangfu	2020	1.03	1.01	1.05	15
	Atkinson	2018	1.03	1.01	1.05	13
	Faustini	2014	1.02	1.02	1.03	8
	HAPINZ 3.0	**2022**	**1.14**	**1.06**	**1.23**	**1**
Lung cancer	Stieb	2020	1.04	1.02	1.07	28
	Stieb	2020a	1.04	1.02	1.07	23
	Atkinson	2018	1.05	1.02	1.08	16
	Hamra	2015	1.04	1.01	1.08	15
	HAPINZ 3.0	**2022**	**1.03**	**0.95**	**1.13**	**1**

1 Steib 2020a omits studies judged to have higher risk of bias. Based on Steib *et al* (2021)

Statistical analyses – morbidity

Long-term air pollution exposure leads to chronic diseases as well as mortality.

Given the availability of detailed data on hospital admissions, we repeated the analyses using data on public hospital discharges for the same disease classifications as for mortality. These include cardiovascular diseases, ischaemic heart disease, stroke, respiratory diseases, lung cancer and asthma in adults (30 years and above) and asthma in children (0-14 years inclusive).

For the analysis of children, educational and smoking status data were not available and we substituted equivalised household income for personal income.

Hospital admissions in adults

Effects of PM_{10} on hospital admissions were weaker than those for $PM_{2.5}$ and NO_2. In two pollutant models, there were statistically significant associations between $PM_{2.5}$ and NO_2 and hospital admissions for both cardiovascular disease (all causes) and respiratory disease (all causes). The strongest associations were for $PM_{2.5}$ and ischaemic heart disease and for NO_2 and asthma (Table 17).

Table 17: Results for hospital admissions in adults, two pollutant models

$PM_{2.5}$ (per 10 µg/m³), annual exposure	Central estimate)	Lower 95% CI	Upper 95% CI
All cardiovascular disease	**1.115**	**1.084**	**1.146**
Ischaemic heart disease	1.289	1.227	1.353
Stroke	1.128	1.064	1.197
All respiratory disease	**1.070**	**1.021**	**1.122**
Lung cancer	0.989	0.860	1.137
Asthma	0.954	0.810	1.123
NO_2 (per 10 µg/m³), annual exposure	**Central estimate)**	**Lower 95% CI**	**Upper 95% CI**
All cardiovascular disease	**1.047**	**1.031**	**1.064**
Ischaemic heart disease	0.972	0.944	1.001
Stroke	1.041	1.006	1.077
All respiratory disease	**1.130**	**1.102**	**1.159**
Lung cancer	1.011	0.930	1.098
Asthma	1.169	1.075	1.271

The associations shown are stronger than those reported in previous studies of long-term exposure. For example, Kloog *et al* (2012) reported a 4.22% increase in respiratory admissions per 10 µg/m³ $PM_{2.5}$ long-term exposure. Yitshak-Sade *et al* (2018) reported a 6.58% increase in cardiovascular admissions for a 2.3 µg/m³ (interquartile range) increase in $PM_{2.5}$ long-term exposure. Both studies adjusted for temperature, and short-term changes in air pollution, but did not have access to individual data on income, education or smoking history.

Hospital admissions for asthma

We ran a Poisson regression model including $PM_{2.5}$ and NO_2 exposure, and asthma hospital admissions for children aged 0-18 years. For $PM_{2.5}$ the risk was elevated but not significant: RR 1.10 (0.95, 1.28) per 10 µg/m^3. However, **for NO_2, the risk was significantly increased**: RR 1.18 (95% CI: 1.09, 1.28) per 10 µg/m^3, which is similar to our finding for morbidity in adults.

Table 18 compares our findings for hospital admissions for asthma in both children and adults with those for asthma incidence published in other studies.

Table 18: Exposure-response functions for long-term exposure to NO_2 and hospital admissions for asthma (per 10 µg/m^3)

Asthma hospital admissions	Author	Year	Central estimate	Lower 95% CI	Upper 95% CI	No of studies
Adults	Liu	2020	1.17	1.10	1.25	1
	HAPINZ 3.0	**2022**	**1.17**	**1.08**	**1.27**	**1**
Children (0-18yrs)	Khreis	2017	1.13	1.05	1.18	41
	HAPINZ 3.0	**2022**	**1.18**	**1.09**	**1.28**	**1**

A recent study reported an RR of 1.17 (95% CI 1.10, 1.25) per 10 µg/m^3 for asthma incidence in adults (Liu *et al* 2020). A meta-analysis of 41 epidemiological studies of asthma incidence in children reported an RR of 1.05 (1.02, 1.07) per 4 µg/m^3 increase in ambient NO_2, which is approximately equivalent to 1.13 (1.05, 1.18) per 10 µg/m^3 (Khreis *et al* 2017).

Statistical analyses – ethnicity

Potential effect modification by ethnicity was assessed in subgroup analyses. **We found no robust differences in any of the ERFs in the two pollutant model when we limited the analyses to specific ethnic groups.**

The central estimates for some exposure-response functions were higher for different ethnic groups. However, when combined with the confidence intervals, there was no statistically significant difference between the results. This is similar to what we found in HAPINZ 2.0.

5.3.2 Sensitivity analyses

After the cohort analyses were completed, the HAPINZ 3.0 draft reports and models were sent to three peer reviewers, particularly to get feedback on the NO_2 associations which were considerably higher than those reported in the literature at the time (early 2021).

Two international experts were commissioned by the HAPINZ 3.0 team to peer review the draft outputs as follows:

- Dr Xavier Querol (Institute of Environmental Assessment & Water Research, Spain) who focussed his feedback on emissions, exposure and source apportionment
- Dr Mike Holland (Ecometrics Research and Consulting, United Kingdom) who focussed his feedback on health endpoints/ERFs, social costs and messaging.

Given the significance of the New Zealand-specific ERFs to the HAPINZ 3.0 overall results, the HAPINZ 3.0 Steering Committee also engaged a specialist epidemiologist:

- Prof Bert Brunekreef (Utrecht University, The Netherlands)

Prof Brunekreef is one of the lead researchers for the ELAPSE study – looking at the impact of low-level air pollution in Europe – which is especially relevant to the low levels we experience in New Zealand.

All three reviewers were also part of the international team preparing the *WHO Global Air Quality Guidelines* (WHO 2021).

Given the strength of the NO_2 ERF (in particular), the reviewers recommended we undertake a number of sensitivity analyses to confirm the robustness of our results. The findings of these analyses are discussed in the following sections with further details reported in Appendix C.

Risk of bias assessment

The World Health Organization notes that:

> Assessment of potential bias lies at the core of all epidemiology when trying to understand the relationship between an exposure and an outcome. (WHO 2020)

They have recently published guidance (WHO 2020) on how to assess the risk for the six key categories of bias:

1. Confounding
2. Selection bias
3. Exposure assessment
4. Outcome measurement
5. Missing data
6. Selective reporting.

We assessed the risk of bias in the *HAPINZ 3.0 Cohort Study* using the WHO guidance (see Appendix C.1) and found that the risk was *low* in all categories except *confounding* which was assessed as *low to moderate*.

Under confounding, WHO recommends controlling for body mass index **(BMI**) which we were unable to do. However, we found no significant difference in the effects of NO_2 by ethnicity. Including ethnicity in the models would be expected to control for BMI indirectly, since BMI is associated with ethnicity in NZ. Therefore, lack of control for BMI is unlikely to have affected the results of HAPINZ 3.0.

Mortality model selection

The two statistical models most often used for assessing *mortality* events are the Cox proportional hazards model and the Poisson regression model. We opted for the latter – Poisson regression – in the *HAPINZ 3.0 Cohort Study* because we also wanted to use the same model to assess *morbidity* impacts and Poisson enables us to consider multiple events (i.e. multiple hospitalisations) for the same affected person.

We re-ran the mortality analyses undertaken for the two pollutant modelling using the Cox proportional hazards model.

We found that the results of the main models for non-external causes of mortality were virtually identical using Cox proportional hazards or Poisson regression (see Appendix C.2). Therefore, the selection of model had little influence on our findings.

Exposure assessment time period

We estimated the long-term average concentrations of NO_2 and $PM_{2.5}$ over the decade 2006 to 2016 to see whether trends in air pollution over time had biased the results. We ran the two pollutant analyses using the decadal average concentrations by averaging the estimates for 2006 and 2016.

Results of the main models for non-external causes of death were not substantially altered (see Appendix C.3). This means that trends in air pollution concentrations since 2006 are unlikely to have biased the results.

In a single pollutant model, there was evidence of a stronger association between $PM_{2.5}$ and mortality among Pacific peoples. However, there was no significant difference by ethnicity in the associations with either NO_2 or $PM_{2.5}$ in the model using 2016 pollutant concentrations

Three pollutant modelling

Given the strength of the NO_2 associations, we investigated whether NO_2 might be acting as a proxy for other traffic-related pollutants that are causally related to impacts. Road dust, brake wear and tyre wear contribute to traffic emissions and are typically in the coarse PM size fraction covering particles 2.5 to 10 µm in diameter ($PM_{10\text{-}2.5}$) as opposed to exhaust PM which is in the smaller $PM_{2.5}$ fraction.

As we had PM_{10} exposure estimates already, we were able to create a $PM_{10\text{-}2.5}$ dataset by subtracting $PM_{2.5}$ values from those of PM_{10} for all CAUs. We then included $PM_{2.5}$, $PM_{2.5\text{-}10}$ and NO_2 concentrations as explanatory variables in a three pollutant model. We ran the model at the national level and then re-ran analyses restricted to Auckland urban areas, which we considered might have the most robust estimates of air pollution exposure.

In national models including $PM_{2.5}$ $PM_{10\text{-}2.5}$ and NO_2 concentrations, associations between $PM_{2.5}$, NO_2 and mortality were strengthened (see Appendix C.4). Similar results were obtained when this model was run for Auckland urban area alone. In analyses restricted to specific ethnic groups, associations with $PM_{2.5}$ were significantly stronger among Māori and Pacific peoples compared to other ethnicities. However, $PM_{10\text{-}2.5}$ had an apparent negative association with mortality.

Interpretation of the three pollutant models is unclear. The apparent negative association between $PM_{10\text{-}2.5}$ and mortality is unlikely to be causal. Measurements of $PM_{2.5}$ and/or PM_{10} concentrations were unavailable for some CAUs, and not available at finer geographic scale. In many areas, estimates of $PM_{10\text{-}2.5}$ concentrations relied on extrapolated $PM_{2.5}$ to PM_{10} ratios. For these reasons, it is possible that the results of models including coarse PM were affected by exposure misclassification. Further work is needed to investigate this.

Given the uncertainty regarding the coarse PM results, we used the ERFs developed in the two pollutant modelling in the *HAPINZ 3.0 Health Effects Model*. **Most importantly, the NO_2 associations remained strong in either the two or three pollutant models indicating that we cannot attribute the effect (at this stage) to the influence of coarse PM.**

Shape of the ERF at low concentrations

There is emerging evidence that the exposure-response relationships for air pollution exposure may be higher at low doses (Papadogeorgou *et al* 2019; Chen & Hoek 2020; Yazdi *et al* 2021). For example, Chen & Hoek (2020) noted for annual $PM_{2.5}$ that:

> The combined effect estimate is 1.17 (95% CI 1.12, 1.23) for the five studies with a mean concentration below 10 μg/m^3.

This contrasted with a combined risk ratio of 1.08 (95% CI 1.06, 1,09) for annual $PM_{2.5}$ and natural-cause mortality across all studies regardless of concentration.

A supra-linear dose response (i.e. greatest effects at the lowest levels of exposure) is a possible explanation for the higher effect sizes reported in HAPINZ 3.0, compared to international studies. In HAPINZ 3.0, the population-weighted 2016 annual average for $PM_{2.5}$ is 6.5 μg/m^3 and for NO_2 is 7.8 μg/m^3. For example, in the ELAPSE study, the pooled cohort from 15 European countries was exposed to an average of 15 μg/m^3 $PM_{2.5}$ and 25 μg/m^3 NO_2 (Brunekreef *et al* 2021).

We explored the shape of the concentration response in stages:

1. by fitting quintiles of $PM_{2.5}$ and NO_2 concentrations by CAU, in single pollutant and two pollutant models. This involved ranking the concentrations for each CAU in order and analysing the ERFs for each fifth of the observations in categories 1 (lowest 20%) to 5 (highest 20%). The ERF for each quintile represents the risk ratio with respect to the lowest quintile (for which is the ERF set to 1.0).

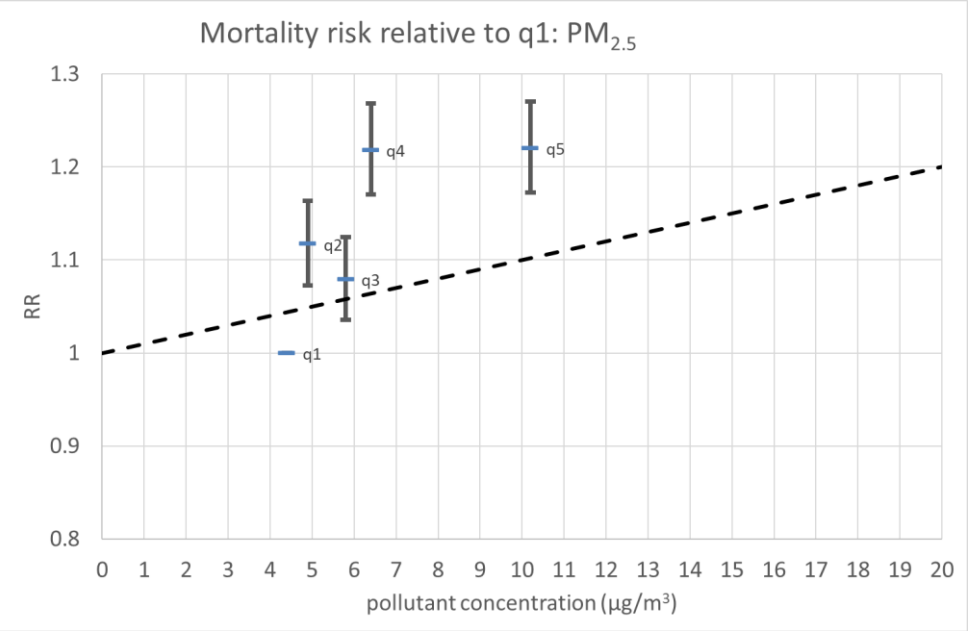
2. by fitting linear models restricted to CAUs with estimated pollutant concentrations below or equal to the median (in single pollutant and two pollutant models).

When pollutants were classified in quintiles, mortality from non-external causes was positively associated with each pollutant (though not significantly so for quintile 3 of $PM_{2.5}$). The shape of the ERFs appeared supra-linear (Figure 20): that is, the slope of the ERF is steeper at lower concentrations. However, this impression is based on a small number of data points and consequently should be treated with caution.

Consistent with a supra-linear association between mortality and NO_2, in models restricted to pollutant concentrations below or equal to the median the association between mortality and NO_2 was substantially strengthened (see Appendix C.5).

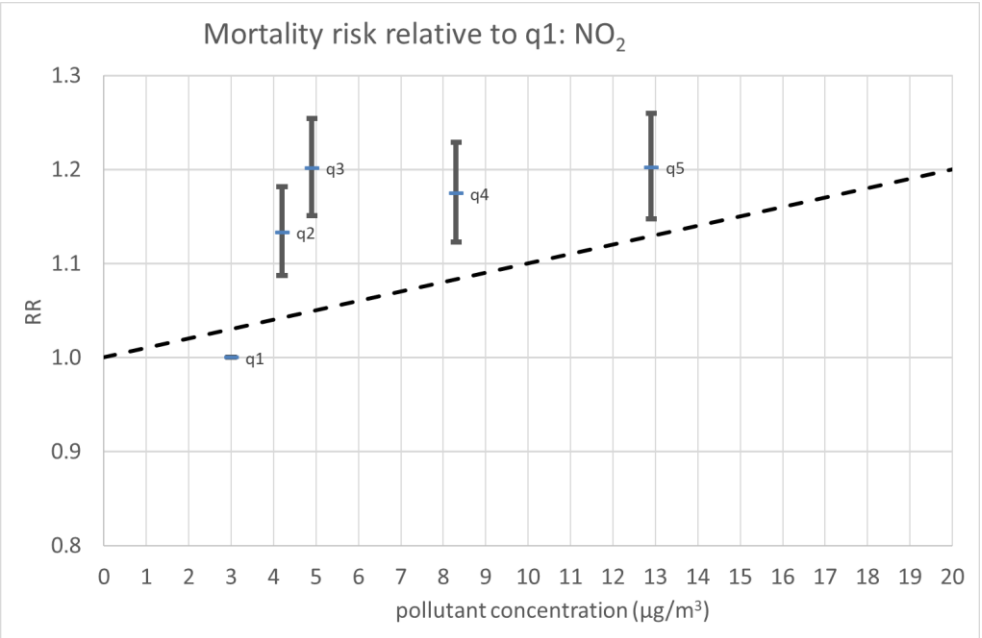
Figure 20: Shape of ERFs at low concentrations for the single pollutant models.

(a) $PM_{2.5}$ mortality



Note: The x-axis represents the quintiles of the pollutant concentration while the y-axis (X axis) shows the risk ratio for each quintile, plus 95% confidence intervals, compared to the lower quintile. For comparison, the dashed line indicates a linear ERF with no threshold and RR 1.10 µg/m³.

(b) NO_2 mortality



Note: The x-axis represents the quintiles of the pollutant concentration while the y-axis (X axis) shows the risk ratio for each quintile, plus 95% confidence intervals, compared to the lower quintile. For comparison, the dashed line indicates a linear ERF with no threshold and RR 1.10 µg/m³.

Conclusions

The final models included $PM_{2.5}$ and NO_2 at the place of usual residence at the time of the 2013 Census, with adjustment for confounding by age, sex, ethnicity, personal income, education, smoking status and ambient temperature. The two pollutant model was robust to sensitivity analyses.

The main results were not substantially altered using Cox proportional hazards regression or using estimated decadal average concentrations of $PM_{2.5}$ and NO_2 (2006 to 2016).

In models restricted to specific ethnic groups, we found no robust differences in any of the associations. Given the number of results generated in this study, some apparently significant results may nevertheless be chance findings. As well as considering the statistical significance of individual results, it is important to also consider overall consistency and plausibility.

In models including $PM_{2.5}$, $PM_{10\text{-}2.5}$ and NO_2 concentrations, associations between $PM_{2.5}$, NO_2 and mortality were strengthened, and there were statistically significant differences by ethnicity, with apparently higher ERFs among Māori and Pacific people than for other ethnicities. However, interpretation of the three pollutant models is unclear. The apparent negative (protective) association between $PM_{10\text{-}2.5}$ and mortality is unlikely to be causal. It is possible that the results of models including $PM_{10\text{-}2.5}$ were affected by exposure misclassification and further work (outside the scope of the current HAPINZ) is needed to investigate this.

In two pollutant models, there was evidence for a stronger association between NO_2 and mortality at low concentrations (<7.5 μg/m^3) and a significant association between NO_2 and mortality at very low concentrations (<4.5 μg/m^3). This implies that if there is a threshold for this association, it is very low. On the other hand, there was no evidence of a stronger association between $PM_{2.5}$ and mortality in equivalent models restricted to CAUs with concentrations of $PM_{2.5}$ below the median.

The main findings of the *HAPINZ 3.0 Cohort Study* have been published (Hales *et al* 2021). The authors concluded that the statistically significant findings in two pollutant models were likely to be causal, meaning the health effects can be attributed to both $PM_{2.5}$ and NO_2. This interpretation is consistent with recent international evidence (Brunekreef *et al* 2021; Forastiere & Peters 2021).

In addition, Hales *et al* (2021) noted that:

> The strength of the association of NO_2 with childhood asthma stands out for local policy and clinical practice, given that the prevalence of this condition in New Zealand is high by international standards. (Hales *et al* 2021).

In all sensitivity analyses undertaken the associations identified in the *HAPINZ 3.0 Cohort Study* were robust. Consequently, we used the ERFs developed in the two pollutant modelling in the *HAPINZ 3.0 Health Effects Model*.

5.3.3 Selected exposure-response functions for health impacts

The exposure-response functions we adopted fell into two categories:

- Those from the *HAPINZ 3.0 Cohort Study* carried out as part of the overall project – HAPINZ 3.0 – for which the effects of different pollutants are largely additive.
- Those which enable backcasting of health effects to 2006 to compare the health burden estimated using the HAPINZ 3.0 methodology with that in HAPINZ 2.0.

Primary exposure-response functions

We assessed the following pollutant-outcome pairs:

- **Premature mortality and YLL** from long-term exposure ($PM_{2.5}$ annual mean, no threshold)
  - All adults, aged 30 years and over (HAPINZ 3.0 NZ cohort study): 1.105 (95% CI: 1.065, 1.145) per 10 µg/m^3
  - All Māori adults, aged 30 years and over and All Pacific adults, aged 30 years and over (the same ERF as above for All adults)

Note: We found no statistically significant difference in effects by ethnicity. Therefore, we report $PM_{2.5}$ mortality for all adults and for all Māori and Pacific adults using the same exposure-response function but with the population and incidence data for those subgroups.

- **Premature mortality and YLL** from long-term exposure (NO_2 annual mean, no threshold)
  - All adults, aged 30 years and over (HAPINZ 3.0 NZ cohort study): 1.097 (95% CI: 1.074, 1.120) per 10 µg/m^3
- **Hospital admissions** from long-term exposure (based on $PM_{2.5}$ annual mean)
  - CVD (including stroke), all ages (HAPINZ 3.0 NZ cohort study): 1.115 (95% CI: 1.084,1.146) per 10 µg/m^3
  - Respiratory diseases, all ages (HAPINZ 3.0 NZ cohort study): 1.070 (95% CI: 1.021, 1.122) per 10 µg/m^3
- **Hospital admissions** from long-term exposure (based on NO_2 annual mean)
  - CVD (including stroke), all ages (HAPINZ 3.0 NZ cohort study): 1.047 (95% CI: 1.031, 1.064) per 10 µg/m^3
  - Respiratory diseases, all ages (HAPINZ 3.0 NZ cohort study): 1.130 (95% CI: 1.102, 1.159) per 10 µg/m^3
- **Restricted activity days** from long-term exposure ($PM_{2.5}$ annual mean)
  - Restricted activity days, all ages (ALA 1995 based on Ostro 1987): 0.9 (lower/upper bounds: 0.5, 1.7) per 10 µg/m^3

Secondary exposure-response functions (for backcasting)

We also assessed the following pollutant-outcome pairs used in the HAPINZ 2.0 report, with updated exposure-response functions from the HAPINZ 3.0 cohort study where available, to allow comparison with the previous results:

- **Premature mortality** from long-term exposure (PM_{10} annual mean)
  - All adults, aged 30 years and over (HAPINZ 3.0 NZ cohort study): 1.117 (95% CI: 1.093,1.142) per 10 µg/m^3
  - Māori adults, aged 30 years and over (the same ERF as above for All adults)
- **Restricted activity days** from long-term exposure ($PM_{2.5}$ annual mean)
  - Restricted activity days, all ages (ALA 1995 based on Ostro 1987): 0.9 (lower/upper bounds: 0.5, 1.7) per 10 µg/m^3

Note: Restricted activity days were used as a primary outcome as well as for backcasting to 2006. While the exposure-response function was the same in HAPINZ 2.0 and HAPINZ 3.0, the method for assessing $PM_{2.5}$ exposure was more sophisticated in HAPINZ 3.0 (see section 3.3.4) with a greater reliance on actual monitoring data rather than generic ratios.

Childhood asthma

We also assessed the following pollutant-outcome pairs relating to childhood asthma:

- **Asthma/wheeze hospitalisations** due to long-term exposure (NO_2 annual mean)
  - All children, 0-18 years (HAPINZ 3.0): 1.182 (95% CI: 1.094, 1.276) per 10 µg/m^3
- **Prevalence** of childhood asthma due to long-term exposure (NO_2 annual mean)
  - All children, 0-18 years (Khreis *et al* 2017): 1.05 (95% CI: 1.02, 1.07) per 4 µg/m^3

5.3.4 Analysis of health and population datasets

For the *HAPINZ 3.0 Health Effects* Model, health data were analysed at the level of a small spatial unit (census area unit 2013, CAU2013). To ensure consistency, we aligned the years of analysis for health data, population data and exposure data to be 2015-2017 (with a mid-point of 2016). As for HAPINZ 2.0, we averaged health data across three years to reduce inter-annual variability.

For the cohort studies, we used health outcome data for 2006 to 2016.

Mortality

The mortality analyses required for HAPINZ 3.0 were:

- premature mortality, all adults aged 30 years and over
- premature mortality, Māori adults aged 30 years and over
- premature mortality, Pacific adults aged 30 years and over.

We analysed confidentialised unit record data from the New Zealand Mortality Collection (MoH 2021a), extracted by the Ministry of Health in August 2021. The Mortality Collection includes date of birth, date of death, underlying cause of death (using the International Classification of Diseases 10th revision – Australian Modification, **ICD-10 AM**), ethnic groups,

and domicile code (which relates to census area unit). We used data for deaths registered in New Zealand in the three-year period 2015–2017.

Mortality included all non-external causes of death (i.e. the analysis excluded deaths due to external causes, defined as ICD codes V00-Y98). Non-residents were excluded from the analysis. The number of deaths in each CAU2013 was summed for 2015–2017, for all adults aged 30 years and over, as well as for Māori and Pacific adults. Total response ethnic groups were used, whereby every person recorded as being of Māori ethnicity was included in the Māori ethnic group, and every person recorded as being of Pacific ethnicity was included in the Pacific ethnic group.

Years of life lost (YLLs)

Years of life lost were calculated using the mortality data (above) and life tables, to estimate the number of years of life lost for each individual.

We used the New Zealand abridged period life tables for 2015–2017 by sex (Stats NZ 2018), as full life tables were unavailable. The abridged period life tables present data for five-year age bands (0, 1–4, 5–9, … 90+ years), for males and females; these were applied to the mortality outputs as appropriate, based on age at death and sex of each individual.

We chose to use life tables relating specifically to the New Zealand population (*endogenous* life tables), rather than hypothetical life tables referring to the 'best possible' life expectancy (*exogenous* life tables). Endogenous life tables are more appropriate when trying to estimate the number of life years lost in a particular country (Anand & Reddy 2019), but it is not possible to make direct comparisons of the impact of air pollution with other countries that have used the 'best possible' life expectancy approach.

Hospital admissions

The hospital admissions analyses required for HAPINZ 3.0 included:

- cardiovascular hospital (**CVHA**) admissions (ICD-10 AM chapter I[25]), all ages
- respiratory hospital (**RHA**) admissions (ICD-10 AM chapter J), all ages
- asthma hospital admissions (ICD-10 AM codes J45, J46), 0–18 years
- asthma or wheeze hospital admissions (ICD-10 AM codes J45, J46, R062), 0–18 years

We analysed anonymised unit record data from the National Minimum Dataset (**NMDS**) (MoH 2021b), extracted by the Ministry of Health in August 2021. The NMDS Collection includes date of birth, date of hospital admission and discharge, primary diagnosis (ICD-10 AM code), ethnic groups and domicile code.

The hospital admissions analyses used principal diagnosis, and only included acute and arranged (within 7 days) hospital admissions. The analyses excluded non-residents and transfers between medical wards and/or hospitals (i.e. only the initial hospital admission

[25] Each type of hospital admission or health outcome is assigned an ICD-10 AM code to enable global comparison.

information was included in the analysis). Deaths were excluded from hospital admissions data to avoid double-counting.

Data were analysed for the three-year period 2015–2017. For this purpose, the year referred to the year at discharge, to ensure that the dataset was complete for the full year. We then took the total hospital bed nights for each endpoint and divided by the total number of admissions to get the average nights per stay which came to 6.80 per CVHA, 3.32 per RHA, and 0.92 for asthma or wheeze admissions.

The analysis of children's asthma hospital admissions included the principal diagnosis of wheeze, as there is evidence that paediatricians in New Zealand tend to describe suspected asthma as 'wheeze' in younger children (HQSC 2016; Simpson *et al* 2017).

Spatial units for health data

For the mortality, YLL and hospital admissions analyses, counts were output at the CAU2013 level, using domicile code. National health data collections use domicile codes to represent a person's usual residential address. Domicile codes relate one-to-one to CAUs, although they lag behind the census by about two years (Table 19). For example, domicile codes relating to 2006 CAU boundaries were assigned to health records from mid-2008 until mid-2015. After mid-2015, the domicile codes relate one-to-one to 2013 CAUs. There is not yet any way to translate health domicile codes to the new Census geographic units of Statistical Areas 1 or 2 (SA1s and SA2s).

Table 19: Concordance from domicile codes to CAU

Years of health data	Census area unit (CAU) boundaries that domicile codes relate to
1 July 2003 to 30 June 2008	2001 CAU
1 July 2008 to 30 June 2015	2006 CAU
1 July 2015 to current	2013 CAU

To analyse health data from the first half of 2015, some recoding needed to occur, as these records were coded to 2006 CAUs. Most 2006 CAUs can be directly assigned to a 2013 CAU; however, some 2006 CAUs had split into multiple 2013 CAUs. In these specific cases, we needed to reassign the 2006 CAU data to these new (multiple) 2013 CAUs.

We reassigned the data proportional to the population living in the 2013 CAUs, with the population used depending on the analysis of interest:

- all ages: cardiovascular hospital admission; respiratory hospital admissions
- 30 years and over: premature mortality
- 0–18 years: asthma hospital admissions
- Māori total population: premature mortality (30+ years)
- Pacific total population: premature mortality (30+ years).

Restricted activity days

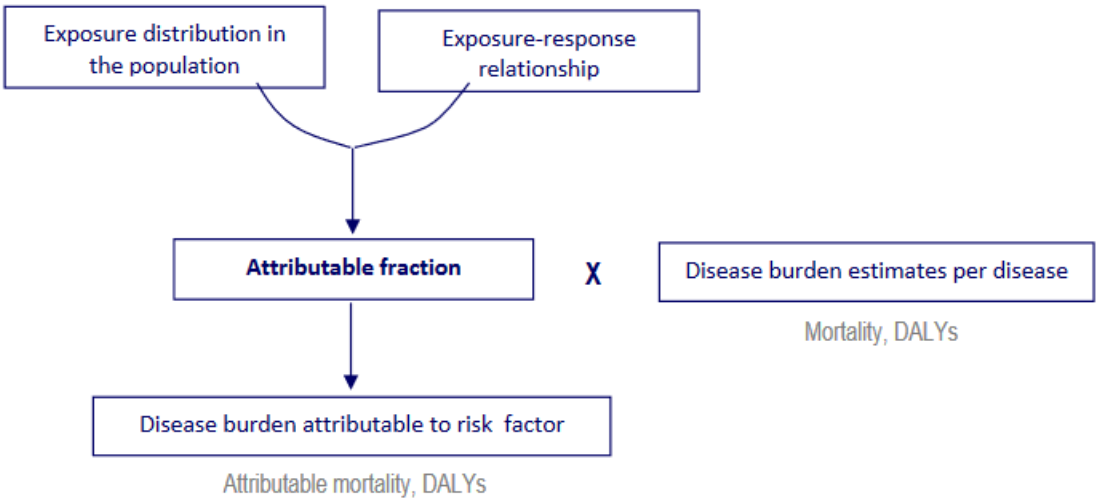
The analysis for restricted activity days was based on the estimated resident population. Population estimates were provided by Stats NZ, for the year 2016. These population estimates were given for each 2013 census area unit (CAU2013) and were based on 2018 Census results. Population estimates were rounded to the nearest 5 or 10 people, depending on the size of the area.

5.3.5 Calculation of the health burden

Our approach to estimating the health burden attributable to air pollution followed the method used in the Environmental Burden of Disease Study (WHO 2018; Prüss-Üstün *et al* 2003) and is summarised in Figure 21. It is also consistent with the approach used in previous HAPINZ studies.

This method uses the **population attributable fraction** or **PAF (**the proportion of the health burden attributable to a specified risk factor) to estimate the **attributable burden** (the disease burden attributable to a specified risk factor).

Figure 21: Method for estimating burden of disease



Source: WHO (2018), Burden of Disease methods for ambient air pollution

Note: DALYs= disability-adjusted life years

Premature mortality and hospital admissions

For the estimated health burden of PM and NO_2, we used the following formula for the population attributable fraction (PAF), adapted from Prüss-Üstün *et al* (2003):

$$\mathbf{PAF} = \frac{(\mathbf{RR} - \mathbf{1}) \times \mathbf{E}}{[(\mathbf{RR} - \mathbf{1}) \times \mathbf{E}] + \mathbf{1}}$$

In this formula:

RR (relative risk, also referred to as the exposure-response function) shows the change in risk for a particular health outcome (e.g. premature death) per unit change in concentration of a particular air pollutant (e.g. 1.11 per 10 µg/m^3 of PM_{10}), based on epidemiological evidence

E (exposure) is the concentration of pollutant in the area of interest (e.g. annual average PM_{10} concentration in a particular census area unit), in terms of the units of relative risk

PAF is the population attributable fraction, which can be interpreted as the estimated percentage of total health cases that are attributable to the exposure (i.e. air pollution).

The PAF can then be used, along with the total number of health cases, to estimate the health effects attributable to air pollution:

$$\mathbf{Health\ effects_{(Cases)}} = \mathbf{PAF} \times \mathbf{Cases_{(Total)}}$$

where:

Health effects$_{(Cases)}$ = the *number* of health cases attributable to air pollution

PAF = population attributable fraction calculated above, based on the exposure and the exposure-response function

Cases$_{(Total)}$ = the total number of health cases in the area of interest.

The health cases can include any type of health burden, including deaths, hospital admissions and YLLs.

We calculated the PAF and attributable burden for each CAU, then summed the attributable burden across the whole country and by region. In this approach, we assumed that in a CAU everybody was exposed to the same level of air pollution.

This approach gives the same results as the formula used in HAPINZ 2.0, to calculate the health effects due to air pollution (Health effects$_{Cases}$ or Cases$_{AP}$):

$$\mathbf{Health\ effects_{Cases}} = \mathbf{Cases_{AP}} = \frac{\mathbf{Cases_{Total}}}{\left(\mathbf{1} + \left(\frac{\mathbf{1}}{(\mathbf{RR} - \mathbf{1}) \times \mathbf{E}}\right)\right)}$$

Restricted activity days

The number of restricted activity days (RADs) due to air pollution was calculated by CAU as follows:

$$\mathbf{RAD} = \mathbf{Population} \times \mathbf{RF} \times \mathbf{E}$$

where*:*

RF (risk factor) is 0.9 (lower/upper bounds: 0.5, 1.7) days per person per year per 10 µg/m^3 annual $PM_{2.5}$ (ALA 1995 based on Ostro 1987)

E (exposure) is the annual $PM_{2.5}$ average in $\mu g/m^3$ figure per CAU divided by 10

Population is the estimated 2016 population for all ages, all ethnicities, for the CAU (Stats NZ data).

Note: RADs are not calculated relative to a baseline incidence; hence a risk factor (RF) of 0.9 is used.

Childhood asthma prevalence

For the assessment of the prevalence of childhood asthma due to NO_2 exposure, asthma prevalence data were not available at the CAU level. As a result, **we developed a proxy indicator of childhood asthma prevalence using linked health datasets, which provided CAU-level data on the estimated number of children with asthma.**

This proxy indicator was developed using linked data from the following the PHO register, National Minimum Dataset, and Pharmaceutical Collection (Ministry of Health). Based on the methods of Donovan *et al* (2018), our indicator included children aged 0–18 years who had either one or more hospitalisations for asthma (primary or secondary diagnosis), and/or at least four pharmaceutical dispensings for anti-asthma drugs between 1 January 2014 and 31 December 2018. We used sensitivity analyses to determine the most appropriate number of dispensings, so we could match the estimated number of children with medicated asthma from the New Zealand Health Survey 2014-2017. [26]

Note: During external validation checks, we compared the number of children with asthma with the New Zealand Health Survey estimates, from the 20 District Health Boards in New Zealand. While our measure gave good agreement in a number of DHBs, it identified more children with asthma in the three Auckland DHBs than the New Zealand Health Survey, while in the Canterbury DHB, our measure identified fewer children. Several possible reasons exist for these differences in prevalence estimates, including measurement issues with our proxy indicator (such as prescriptions being used for other health conditions), issues with the New Zealand Health Survey data (such as under-reporting due to English as a second language, and health literacy levels), and differences in definitions (such as inclusion of asthma hospitalisations in one definition but not the other). Further work could investigate these regional differences.

We also carried out a **cross-check of this method, by using an indirect method** to estimate the national childhood asthma burden due to NO_2. Instead of calculating a PAF for each CAU, this approach calculates an overall PAF for New Zealand, using the NO_2 exposure and the population count in each CAU.

In the indirect method, the PAF can be calculated using the following formula (Prüss-Üstün *et al* 2003, p45), which combines the relative risk and proportion of the population exposed across many different levels of exposure*:*

$$\mathbf{PAF} = \frac{\sum_{\mathbf{i}}^{\mathbf{n}} \mathbf{P_i RR_i} - \mathbf{1}}{\sum_{\mathbf{i}}^{\mathbf{n}} \mathbf{P_i RR_i}}$$

[26] Data provided by Ministry of Health on 20 October 2020 (from Mel Duncan via email).

where:

PAF = population attributable fraction

i = different levels of exposure, up to a total of **n** levels of exposure

$\mathbf{P_i}$ = proportion of the population in exposure category i (P_i adds to 1 across all categories of i)

$\mathbf{RR_i}$ = relative risk at exposure category i compared to the reference level.

We used this formula to calculate the PAF across all the CAUs in New Zealand (so in the formula, **n**= number of CAUs). We used an overall relative risk of 1.05 per 4 µg/m^3 from the literature for the prevalence of childhood asthma due to long-term NO_2 exposure (Khreis *et al* 2017). To calculate the PAF, we used the modelled NO_2 level at each CAU, and then calculated the relative risk (RR_i) for each CAU based on the NO_2 exposure in that CAU. Using population estimates for 2016 for children aged 0-18 years from Stats NZ, we calculated the percentage of children in each CAU, among the total children in NZ. We then calculated the P_iRR_i for each CAU and summed them to give the PAF for New Zealand. The PAF was then applied to national-level data on the estimated number of children and young people aged 2-18 years with medicated asthma from the New Zealand Health Survey 2014-2017.

Using the proxy indicator gave very similar results as the indirect method for the attributable asthma burden due to air pollution, using an earlier version of the model. For this reason, the proxy indicator has been used in the main HAPINZ 3.0 results. However, the indirect method could be used in future updates if the more complex analysis required for the proxy indicator is not possible.

5.3.6 Uncertainty

Statistical uncertainty in the exposure-response functions themselves is indicated by the 95% confidence intervals. These intervals can be used to undertake sensitivity analyses on the health burden calculated from these estimates.

Other sources of uncertainty include misclassification of exposures and health impacts, misspecification of the statistical model, and presence of residual confounding in the models. These factors are more difficult to assess than statistical uncertainty.

In estimating the exposure-response functions, misclassification of exposure is likely to be the most important of the above factors in the present study. Ideally, we would use estimates of exposure calculated at individual level over several decades, however this is impractical based on the administrative data available in New Zealand. The fact that exposures for PM and NO_2 were assessed with different methods and at a different spatial resolution introduces uncertainty about their *relative* contributions. However, it does not influence estimates of the *effects* of individual pollutants.

The final models included $PM_{2.5}$ and NO_2 at the place of usual residence at the time of the 2013 Census, with adjustment for confounding by ambient temperature, age, sex, ethnicity, personal income, education and smoking status.

Given the importance of the ERFs to the health burden, we undertook both a risk of bias assessment and a suite of sensitivity analyses to confirm the robustness of *HAPINZ 3.0 Cohort Study* findings (discussed in section 5.3.2.).

Using the WHO risk of bias assessment guidance (WHO 2020), we found that the risk was *low* in all categories except *confounding* which was assessed as *low to moderate* (due to lack of available data on BMI).

In all sensitivity analyses undertaken the associations identified in the *HAPINZ 3.0 Cohort Study* were robust. Consequently, we used the ERFs developed in the two pollutant modelling in the *HAPINZ 3.0 Health Effects Model*.

5.3.7 Summary of our approach

The primary health impacts of the assessment include:

- **Premature mortality and YLL** from long-term exposure ($PM_{2.5}$ annual mean, no threshold)
  - All adults, aged 30 years and over (HAPINZ 3.0 NZ cohort study): 1.105 (95% CI: 1.065, 1.145) per 10 µg/m^3
  - All Māori adults and All Pacific adults, aged 30 years and over (the same ERF as above for All adults)
- **Premature mortality and YLL** from long-term exposure (NO_2 annual mean, no threshold)
  - All adults, aged 30 years and over (HAPINZ 3.0 NZ cohort study): 1.097 (95% CI: 1.074, 1.120) per 10 µg/m^3
- **Hospital admissions** from long-term exposure ($PM_{2.5}$ annual mean)
  - CVD (including stroke) hospital admissions, all ages (HAPINZ 3.0 NZ cohort study): 1.115 (95% CI: 1.084, 1.146) per 10 µg/m^3
  - Respiratory disease hospital admissions, all ages (HAPINZ 3.0 NZ cohort study): 1.070 (95% CI: 1.021, 1.122) per 10 µg/m^3
- **Hospital admissions** from long-term exposure (NO_2 annual mean)
  - CVD (including stroke) hospital admissions, all ages (HAPINZ 3.0 NZ cohort study): 1.047 (95% CI: 1.031, 1.064) per 10 µg/m^3
  - Respiratory disease hospital admissions, all ages (HAPINZ 3.0 NZ cohort study): 1.130 (95% CI: 1.102, 1.159) per 10 µg/m^3
- **Restricted activity days** from long-term exposure ($PM_{2.5}$ annual mean)
  - Restricted activity days, all ages (ALA 1995 based on Ostro 1987): 0.9 (lower/upper bounds: 0.5, 1.7) per 10 µg/m^3

The secondary health impacts to allow for comparison with HAPINZ 2.0 include:

- **Premature mortality** from long-term exposure (PM_{10} annual mean)
  - All adults, aged 30 years and over (HAPINZ 3.0 NZ cohort study): 1.111 (95% CI: 1.089, 1.133) per 10 µg/m^3
  - All Māori adults, aged 30 years and over (the same ERF as above for All adults)
- **Restricted activity days** from long-term exposure ($PM_{2.5}$ annual mean)
  - Restricted activity days, all ages (ALA 1995 based on Ostro 1987): 0.9 (lower/upper bounds: 0.5, 1.7) per 10 µg/m^3

Childhood asthma impacts of relevance to New Zealand include:

- **Asthma/wheeze hospitalisations** due to long-term exposure (NO_2 annual mean)
  - All children, 0-18 years (HAPINZ 3.0 NZ cohort study): 1.182 (95% CI: 1.094, 1.276) per 10 µg/m^3
- **Prevalence of childhood asthma** due to long-term exposure (NO_2 annual mean)
  - All children, 0-18 years (Khreis *et al* 2017): 1.05 (95% CI: 1.02, 1.07) per 4 µg/m^3

6. Estimating social costs

This chapter summarises the methodology used for estimating air pollution social costs in the HAPINZ 2.0 study, reviews alternative approaches and developments that have occurred since and outlines our methodology for the HAPINZ 3.0 update.

It reviews relevant theory on the analysis of social costs, i.e. the estimation of the costs of air pollution to society as a whole and of approaches used elsewhere. The focus is on identifying costs per case, where cases are the indicators for various mortality and morbidity effects. Because of the dominance of mortality impacts in the assessment and the availability currently of a published 2019 value of statistical life, we provide all values in 2019 dollars.

6.1 Approach used in HAPINZ 2.0

HAPINZ 2.0 estimated social costs using a two-step process. First the aggregate health effects (number of cases) were estimated from (a) the exposure of an estimated population to air pollution and (b) exposure-response functions. Second, the number of cases were then multiplied by estimated costs per case (Table 20).

Table 20: Costs per case of health effects assumed in HAPINZ 2.0 (2010 NZ$ values)

Health effect	Cost per case	Sensitivity analysis
Premature mortality (all)	$3.56 million	$7.12 million
Hospital admission (cardiovascular)	$6,350	$356,000
Hospital admission (respiratory)	$4,535	$356,000
Restricted activity day (RAD)	$62	$34-$87

Source: Kuschel *et al* (2012b)

The costs per case were derived as follows:

- **Premature mortality** was valued using the value of statistical life (**VoSL**) based on studies of willingness to pay (**WTP**) for improvements in road safety (MoT 2010). This was multiplied by the number of cases defined as numbers of premature lives lost, or more appropriately, premature *statistical* lives lost (see section 6.2.2).
- The costs of **hospital admissions** (for cardiovascular and respiratory problems) included medical costs and the loss of economic output during hospitalisation but did not include loss of life quality due to prolonged pain and suffering. These were derived from the costs of hospitalisations for road accidents, adjusted for the numbers of days in hospital, plus assessment of losses of economic output while people are in hospital using average income as a proxy for the value of output.
- The costs of **restricted activity days** (RADs) were based on the average loss of output per day (irrespective of a working or non-working day), as for hospitalisations.

Sensitivity analyses were conducted using a VoSL twice as high to reflect higher international values for air pollution risk and using a range of likely loss of life quality and medical costs for the morbidity effects.

6.2 Developments since HAPINZ 2.0

This section reviews developments in the literature on social cost estimation, which prompted changes in the way we estimated these costs in HAPINZ 3.0. The following sections discuss:

- The relative significance of health effects.
- The valuation of mortality impacts – in particular:
    - Use of value of life years lost (**VoLY**) as an alternative measure to VoSL.
    - A cessation lag adjustment to account for the delay to the full achievement of benefits when the effects are dominated by chronic health impacts.
- The valuation of morbidity impacts – in particular, the addition of quality of life impacts to the other morbidity effects reported in HAPINZ 2.0.
- The inclusion of values relevant to policy studies in addition to estimates of the total costs of air pollution.
- The development of damage costs to value changes in emissions to air – including the cost of carbon – for comparing benefits to society of a change in policy/operation with costs of implementing the change.

6.2.1 Relative significance of health effects

The adverse effects of air pollution include:

- human health effects
- reduced visibility and discolouration of air
- nuisance and amenity effects, including dust, smoke, materials damage and odour.

A number of studies in the late 1990s and early 2000s, particularly in Europe, estimated the relative costs of the different effects, concluding that the most significant impacts are those on human health (e.g. studies undertaken for ExternE by Rabl *et al* (2005) and Hohmeyer (1998)). More recently this has been confirmed by other studies, including Ricardo-AEA (2014) and Amann *et al (*2017), although Holland *et al* (2013) note the possible importance of unquantified impacts.

In New Zealand, MoT examined the full range of external effects of transport in its land transport pricing study in the mid-90s, suggesting that the damage costs of air pollution were dominated by health effects, especially mortality caused by particulates (MoT 1996). Jakob *et al* (2006) compared health costs of air pollution in Auckland to those of damage to agriculture and forests, concluding that these other costs were only 0.002% of the total air pollution costs. Other studies internationally and in New Zealand, have not questioned this hierarchy of effects but have concentrated on health effects.

6.2.2 Valuing mortality impacts

Several issues are relevant to the valuation of mortality impacts:

- Characterisation of the mortality effect, e.g. premature deaths vs life years lost
- Terminology used to describe impacts on mortality

- Methods for estimating VoSL
- Methods for estimating VoLY
- The relationship between value and the age of those affected
- Marginal effects and lagged benefits– the implications for policy analysis of health effects being dominated by chronic mortality, with full benefits only emerging after some time.

Characterisation of the mortality effect

Understanding the nature of the mortality effect provides a background to the discussion of whether to use VoSL or VoLY (or both). The ERFs used to estimate numbers of cases are derived from statistical analysis of the relationship between pollution concentrations and death rates in individual age categories (see section 5.3). For a given change in pollution level, the product of existing death rates, the change in pollutant concentration and the ERF is used to estimate the change in the number of deaths in individual age categories. The result is often presented as a change in the number of premature deaths, but this is a simplification using a statistical artefact.

The observed change in death rates used to develop the exposure-response functions can result from (1) a relatively small number of individuals dying in younger age categories who would otherwise have died in old age; (2) by all people dying a little earlier than they would otherwise, such that all deaths are squeezed into fewer age categories; or in practice, (3) an infinite number of changes to every individual's survival function (Nielsen *et al* 2010). Summarising the impact as numbers of premature deaths is using the first characterisation and is misleading. Using the terminology of premature *statistical* deaths better reflects the way they are derived, however, increasing numbers of analysts and government agencies are estimating the change in total life years to summarise the impact, and this indicator is relevant to all potential ways in which air pollution affects individuals and populations.

Most deaths, even with no air pollution, could be considered premature. People tend to die of something, such that air pollution only changes the degree of prematurity of a death. Alberini *et al* (2004) suggest that

> the majority of statistical lives saved by environmental programs appear to be the lives of older people and people with chronically impaired health

and Brunekreef *et al (*2007) suggest the effects

> occur primarily in a subgroup with serious pre-existing (though possibly undiagnosed) cardio-respiratory disease, and who therefore have a life expectancy far less than others of the same age.

Because the impacts are contributory, individual deaths cannot be attributed directly to air pollution, even in retrospect (Rabl 2003); only changes in age-specific all-cause death rates can be observed and age-specific all-cause death rates are observed to increase under higher levels of air pollution.

Air pollution does not result in more deaths, in the long-term; it changes the timing. This is true of all causes of death, whether they be motor vehicle trauma, heart attacks or COVID-19.

Increased mortality risk means life is shortened by the effects of air pollution; numbers of deaths and death rates increase amongst younger age groups and, because all people die, numbers of deaths in this particular cohort decrease in older age groups because there are fewer who will have survived to die in old age. Epidemiological studies that analyse changes in death rates have separated out the age groups to examine the effects – for example, Hales *et al* (2012) developed ERFs by analysing data for those aged under 75.

Some studies estimate the impacts of air pollution on average life expectancy (or life years) for the whole population. These are generally in the region of a six months gain or loss per 10 µg/m^3 change in $PM_{2.5}$ (Table 21). Analysts are beginning to develop relationships directly between concentrations and life expectancy to project life expectancy outcomes, rather than via life tables (e.g. Kolasa-Więcek & Suszanowicz 2019). This is consistent with characterising the effect as a *shortening of everyone's life by a small amount*.

Table 21: Estimated impact of change in PM concentration on average life expectancy

Location	Change in life expectancy	Source
USA (51 metropolitan areas)	0.61 years (per 10 µg/m^3 $PM_{2.5}$)	Pope *et al* (2009)
Austria, France, and Switzerland	c.6 months (per 10 µg/m^3 PM_{10})	Sommer *et* al (1999) in Künzli *et al* (2000)
UK	6 months (for a 8.97 µg/m^3 reduction in $PM_{2.5}$)	COMEAP (2010)
USA	0.35 years (per 10 µg/m^3 $PM_{2.5}$)	Correia *et al* (2013)

Addressing whether to present the results as premature mortality or life years lost, COMEAP notes there is a trade-off between full accuracy and accessibility and that the metrics can be

> valid representations of population aggregate or average effects, but they can misleading when interpreted as reflecting the experience of individuals. (COMEAP 2010, p84)

They suggest that

> total population survival time (life years gained or lost) is … the most accurate and complete way of capturing the mortality effects of air pollution reductions … [and] by far the single most relevant metric for policy analysis.

COMEAP also notes that air pollution mostly affects older people.[27] This means it cannot be compared simply with the effects of road traffic accidents, suicide, or HIV/AIDS, which typically affect younger people. They suggest that implicit in any communication about deaths is some understanding of age at death or, equivalently, the loss of life implied by death at various ages. This is best captured explicitly – which, in effect, means discussion in terms of total population survival time (or of life years gained or lost).

[27] We find this result through the simple assumption that the percentage impact is the same at all ages and there is a higher initial death rate amongst older people.

International practice

In the United Kingdom (**UK**), the benefits of policy measures targeted at reducing levels of particulates have been expressed in terms of 'total life years' rather than reductions in numbers of deaths – for example, in the economic analysis to inform the Air Quality Strategy (DEFRA *et al* 2007). More recently, Birchby *et al* (2019) use life years in their report on damage costs to the Department of Environment, Food and Rural Affairs (**DEFRA**).

In the European Union (**EU**), a 2005 CBA of the Clean Air for Europe (**CAFE**) programme recommends

> years of life lost as the most relevant metric for valuation. (AEA Technology Environment 2005).

However, they also include estimates of the number of deaths per year attributable to long-term exposure to ambient $PM_{2.5}$ because, although it over-estimates the impact, it is easy to understand.

Similarly, CBAs in the EU tend to use both VoSL and VoLY, without expressing a preference (Amman *et al* 2017; Åström *et al* 2018; DEFRA *et al* 2007; Holland *et al* 2005; Holland 2019; Torfs *et al* 2007).

Analysis in the United States (**US**) has tended towards the use of premature deaths rather than life years lost (e.g. Industrial Economics 2006). However, the government's 2003 guidance on regulatory impact analysis suggests that it is appropriate to consider using estimates of both, recognising the developing state of knowledge (US Office of Management & Budget 2003). Consistent with this, the 2011 CBA of the Clean Air Act Amendments included results in terms of avoided premature mortality, life years lost and changes in life expectancy (US EPA 2011).

Internationally, both life years lost and premature statistical deaths are currently used to quantify the monetary impacts of air quality impacts.

Terminology for the impacts on death rates and life years

The value of statistical life (VoSL) is the term often used when quantifying the benefits of reductions in the risks of fatalities (Viscusi 2005) and it is widely used in New Zealand.

In the UK, the preferred term is the value of preventing a fatality (**VPF**) (Glover & Henderson 2010) and this is the term preferred by Clough *et al* (2018) also. The VoSL or VPF is not used to estimate the value of the life of any individual but is the value to society of reducing the risk of fatalities as follows:

> if the average person would be willing to pay $500 to reduce the probability of dying by one in ten thousand, then a population of ten thousand individuals would be willing to pay $5 million to prevent one member of that population from dying prematurely. The $5 million figure is the V(o)SL. Because it is not possible ex ante to identify the person whose life will be saved, this prevented mortality is considered a statistical life (Aldy & Viscusi 2007, p 243).

Dockins *et al* (2018) note that VoSL is well-understood by economists (as the marginal rate of substitution between mortality risk and money), but to many others, including decision-makers and media professionals, the term resembles "*obfuscated jargon bordering on the immoral*." They examined several alternative terms, suggesting that value of reduced

mortality risk (**VRMR**) was the most effective and readily understood alternative. It is similar to the more generic value of risk reductions (**VRR**) as used by Rizzi and Ortúzar (2006a) and others or value of a fatal risk reduction (**VFRR**) (González *et al* 2018).

Cameron (2010) outlined the misinterpretation and confusion caused by VoSL and WTP. She prefers willingness to swap (**WTS**) other goods and services or WTP for a microrisk reduction, which is a 1/1,000,000 risk reduction (also called a 'micromort' by Howard (1984)). WTS addresses the objections of some to the idea of monetisation in the context of human lives and the idea of a microrisk reduction reflects the size of the effect at an individual level. While noting the possibly greater suitability of some of these other terms, we have continued the use of VoSL in this report.

Methods for estimating VoSL

The VoSL used in HAPINZ 2.0 was taken from MoT (2010) and was derived from a survey of the WTP for a reduction in the risk of fatal road accidents, updated to 2010 dollar values. Road deaths can occur suddenly and unexpectedly to people at any stage of life, and the average age of people dying in road crashes in New Zealand is approximately 43 years old.[28] In contrast, the increased risk of a fatality from air pollution applies particularly to older people so this raises the question of whether the road crash-based value is valid.

Alberini and Ščasný (2013) found from a survey in Milan that estimates of WTP to reduce the risk of death differed significantly with the source of risk, with higher values for deaths from cancer or respiratory disease than from road accidents. We do not have alternative values for New Zealand so are limited to the value derived from road accidents.

As noted above, the VoSL expressed as a value per life, might be better expressed as a small reduction in the risk of death. Examples using the most recent VoSL are shown in Table 22.

Table 22: Current estimated VoSL and value of microrisk reductions (2019 NZ$ values)

Indicator	2019 value
VoSL	$4,527,300
WTP for 1/10,000 reduction in risk of death	$452.73
WTP for 1/million reduction in risk of death	$4.53

Source: VoSL from MoT (2020)

Note: At the time of finalising HAPINZ 3.0, a study is underway (commissioned by Waka Kotahi) to update the original 2001 WTP study which informs the current New Zealand VoSL and links to the *Monetised Benefits and Costs Manual* (NZTA 2021). The results of the study are not available to be incorporated into HAPINZ 3.0 but we expect updated values to be available later in 2022.

[28] The average age for 2016-19 is 43 and the median age is 41. Data from the Crash Analysis System from Waka Kotahi (as at July 2020).

Methods for estimating VoLY

Because impacts are expressed as life years lost as well as reductions in the risk of death, the value of a life year (VoLY) is required in addition to VoSL.

VoSL-based VoLY

The simplest way to estimate VoLY is to convert the VoSL into a discounted stream of annual life year values over the remaining lifetime of the person as shown below from Aldy and Viscusi (2008):

$$\mathbf{VoLY} = \frac{\mathbf{r}.\mathbf{VoSL}}{\mathbf{1} - (\mathbf{1} + \mathbf{r})^{-L}}$$

where: **VoSL** = value of statistical life

r = the discount rate

L = years of life expectancy

Telfar-Barnard and Zhang (2019) used this approach in estimating the costs of respiratory disease in New Zealand; the 2015 VoSL ($4.06 million) was used with a 3% discount rate and a life expectancy of 40 years to estimate a VoLY of $176,000. To estimate the mortality costs of respiratory disease, they multiply this by the years of life lost based on average life expectancy at age of death.

To update this, the current average age of death for traffic accidents is 43, suggesting a weighted average life expectancy of approximately 40 years.[29] To complete the equation, this leaves the appropriate discount rate still to estimate.

Although Robinson *et al* (2018) suggest people may discount life years differently from consumption or money, several researchers have identified time preferences for reducing mortality of people of different ages approximates the real discount rate for money (Johannesson & Johansson 1997; Cropper *et al* 1994; Horowitz & Carson 1990). The question of the appropriate discount rate for money is another issue, but because the costs of air pollution (and of changes to pollution levels), are being estimated for public policy reasons, our interest is in the public discount rate (see Box 1). As our main assumption we have used the NZ Treasury recommended rate for policy analysis[30], which is currently 5%, with sensitivity analysis at 2% (as used by Treasury in their CBAx model).

Using the VoSL-based approach, an assumed 40 years of lifetime remaining and a 5% real discount rate, produces a VoLY of $263,843.

$$\mathbf{VoLY} = \frac{\mathbf{0.05} \times \$\mathbf{4,527,300}}{\mathbf{1} - (\mathbf{1} + \mathbf{0.05})^{-\mathbf{40}}} = \$\mathbf{263,843}$$

[29] Based on Stats NZ New Zealand Period Life Tables: 2012–14.

[30] https://www.treasury.govt.nz/publications/guidance/planning/costbenefitanalysis/currentdiscountrates

Box 1: Social discount rate

There are two main methodologies for deriving a discount rate for public policy purposes.

- The social rate of time preference (**SRTP**) measures time preferences directly – how much people prefer to consume now rather than later.
- The social opportunity cost of capital (**SOC**) examines returns on investment in which investing money, which might otherwise have been used to pay for consumption goods now, obtains a return enabling greater future consumption.

The NZ Treasury generally uses the SOC as the basis for setting discount rates for use in public policy (NZ Treasury 2015), currently recommending use of a 5% default rate.[31] However, for sensitivity analysis they use a 2% rate in their CBAx tool, which is a spreadsheet model that contains a database of values to help agencies measure impacts and undertake CBAs (NZ Treasury 2021). These discount rates are in real terms, i.e. they apply to monetary values using current dollars, so at 2% inflation they are equivalent to rates of approximately 7% and 4% in nominal terms.

Empirical studies

Other approaches to defining VoLY have used survey-based approaches. Swedish researchers Johannesson and Johansson used a telephone survey of adults between 18 and 69 years old and asked the following question:

> The chance for a man/woman of your age to become at least 75 years old is x percent. On average, a 75-year-old lives for another 10 years. Assume that if you survive to the age of 75 years you are given the possibility to undergo a medical treatment. The treatment is expected to increase your expected remaining length of life to 11 years. Would you choose to buy this treatment if it costs y and has to be paid for this year?" (Johannesson & Johansson 1997).

The resulting VoLY averages approximately US$2,700 (in 19"5$ values).[32] These values are estimates of the present value of some future benefit. Dolan *et al* (2008) reviewed other studies with similarly low values, e.g., £242-£508/VoLY in a 2004 UK study and a Swedish study that found a low WTP for cigarettes with lower health risks that would extend life.

The CBA for the UK's Air Quality Strategy used VoLYs from Chilton *et al* (2004). They derived values of £6,040 to £27,630 in 2002 prices based on a survey which measured people's WTP to live longer (life extension at the end of their life). More recently, surveys in a number of European countries indicated an EU-wide VoLY of €40,000 in 2010, but with the value varying with income across the EU (Desaiguesa *et al* 2011). Based on updated (inflated) values from the original Chilton *et al* (2004) study, Birchby *et al* (2019) use a VoLY of £42,800 within a range of £32,000 – £53,300 (2017 prices) equivalent to NZ$58,000 to NZ$97,000.[33]

[31] https://treasury.govt.nz/information-and-services/state-sector-leadership/guidance/financial-reporting-policies-and-guidance/discount-rates

[32] This was the average for those expressing a positive WTP. Half had a WTP of zero, which was ignored.

[33] Using average daily GBP:NZD exchange rate for 2017 (Reserve Bank of NZ - EXR.DS11.D04).

QALY-based values

Dolan *et al* (2008) suggest that VoLYs can be estimated from using a ratio to the WTP for a quality-adjusted life year (**QALY**), essentially a life year in perfect health. They suggest a ratio of the value of 1 QALY:1.2 VoLYs.[34] In contrast, the UK Treasury (HM Treasury 2018) recommends the same value for VoLY and the value of a QALY (£60,000 in 2018).

However, the QALY values used in New Zealand are the average *costs* of health sector expenditure to achieve a QALY, rather than the *value* (or benefit) of achieving it. NZ Treasury (2020) uses a value per QALY of $33,306 (2020 values) based on a cost effectiveness of 37 QALYs per $1 million net cost to the health sector in 2016/17[35] inflated to 2020[36]. The input values change significantly over time, however. For example, since this analysis, the number of QALYs achieved per $1 million of expenditure has varied between 238 in 2017/18 and 31 in 2019/20 (PHARMAC 2021) suggesting values per QALY varying between $4,200 and $32,600. The average cost per QALY varies significantly over time, e.g. in 2018/19, the equivalent value would be $8,475 per QALY.[37]

Values (rather than costs) of a QALY have been derived using a threshold of the maximum spend to achieve a QALY, e.g. using GDP per capita.[38] This assumes that affordability sets the maximum amount to spend on saving a life, but this is only ever an average affordability and does not provide information on marginal WTP. However, it might set a useful minimum for the value of a VoLY. GDP per capita in New Zealand was approximately $62,000 in the year to June 2019,[39] suggesting a VoLY of $51,600 - $62,000, depending on whether a ratio of 1.2 or 1 VoLY per QALY is used.

Summary and suggested approach

The estimate of VoLY varies with the assumptions.

- The VoSL-based approach assumes the VoSL represents the present value of VoLYs over the remaining expected lifetime. The road crash-derived VoSL is combined with the life expectancy of the average-aged crash victim.
- The empirical analyses have been based on WTP for life extension, generally involving hypothetical scenarios in which people's life is extended some years in the future. The difference from the VoSL-based values is consistent with discounting. For example, taking a VoSL-based VoLY of $263,843 (based on a 5% discount rate) and assuming that the life extension by one year occurs in 40 years' time, the discounted value now is $37,478.

---

[34] A QALY is worth more than a VoLY because it is in better health.

[35] Rounded up from 36.8 per $1 million in PHARMAC (2017).

[36] The current QALY is $36,363 in 2022 values. https://www.treasury.govt.nz/publications/guide/cbax-spreadsheet-model

[37] This is based on 118 QALYs achieved per $1 million spent for funded proposals (PHARMAC 2019). Gyrd-Hansen (2005) also notes the difficulty of identifying a single value per QALY.

[38] For example, see Webber-Foster *et al* (2014); Bertram *et al* (2016).

[39] Statis NZ. Nominal per capita GDP, annual to June 2019 (Table ref: SNE131AA) = $62,187.

- The average cost of a QALY approach has no theoretical justification as it is not based on marginal costs. Using GDP per capita is still using an average rather than identifying a marginal WTP.

Assuming the valuation (of the change in the risk of death) occurs at the time of the observed change in death rate (and of the number of deaths in an age class) then the VoSL-based approach would produce results consistent with the empirical results which value changes in future death rates.

Relationship between value and the age of those affected

Age-related VoSL

The question of whether to use VoSL or VoLY to value changes to fatality risks raises the question of the relationship between VoSL and age. If, as assumed above for the VoSL-based VoLY, VoSL is equal to the present value of the future VoLYs, then the VoSL would decline with the age of a person and the expected number of years of life lost (Chilton *et al* 2020). The cost of increased risk of death is greater for a young person than an old person.

HAPINZ 2.0 did not use VoLY and it assumed VoSL did not vary with age, applying a constant value to all estimates of the costs of premature deaths. The constant VoSL assumption in HAPINZ 2.0 was based on:

- the authors finding no clear evidence in the literature of VoSL declining with age
- an increasing level of trauma with the onset of heart disease or cancer, with the implication that, because these diseases occur mostly in old age, there is additional benefit from saving the lives of the elderly that offsets the smaller number of life years saved
- the recommendations of the OECD.[40]

In Appendix D, we review the literature relevant to the age-VoSL relationship in more detail. It suggests the argument is less clear cut, that there is a strong theoretical argument for VoSL declining with age, and that empirical studies which find no relationship may use an inappropriate valuation stance (private rather than public) or reflect market failures (lack of knowledge of what the future holds). This would argue for use of life years lost and VoLY in analysis.

Age-related VoLY

Using a VoLY to estimate the costs of increased risks of death is consistent with an assumption of declining VoSL with age. Most simply this is done with an assumption of a constant VoLY across all age groups. Some analysts have raised objections to a constant VoLY (Krupnick 2007; Jones-Lee *et al* 2015; Robinson *et al* 2018; Chilton *et al* 2020). However, Abelson (2008) provides a useful and pragmatic discussion and note:

[40] Kuschel *et al* (2012c) cited OECD (2010), but the same assertion of no adjustment for age because of inconclusive evidence is made in more recently in OECD (2012).

If V(o)LY is constant, as is often assumed, V(o)SL declines significantly with age. On the other hand, if V(o)SL is constant with age, V(o)LY rises with age. This would imply that an increase in a given number of years (and any given improvement in health status) is more valuable to an old person than to a young one. If we have to choose between a constant V(o)LY and a constant V(o)SL, a constant V(o)LY seems more attractive. This implies that, other things such as health status being equal, saving more life years is better than saving fewer years. It also means that the value of an improvement in health status is the same at all ages.

Note: We do not have sufficient data to develop age-related VoLY for New Zealand and this remains an emerging field of research. The approach taken here is to use a constant VoSL and a constant VoLY with age. As Chilton *et al* (2020) note, the former over-values older people, while the latter over-values younger people. However, these simplifying assumptions are used to provide a range in the results.

Marginal effects and lagged benefits

The health effects of air pollution may result from short run changes in concentrations (acute effects) but most studies suggest the more significant effects are from long-term exposure to elevated concentrations, particularly of small particulates. Long-term exposure increases a person's susceptibility to death or disease because they become more "frail", although the event of death may be from air pollution or some other cause (Seethaler *et al* 2003). If the effects are from long-term exposure, the benefits of reducing emissions will not result immediately; they will only occur after a person has lived for some time under lower concentrations.

The delay issues have been recognised in international policy studies for some time. In the US, the UK and elsewhere in Europe, studies of the costs and benefits of air pollution policy use lagged benefits. This reduces the PV of benefits because of the impacts of discounting.

United States of America

In the US, prior to 2004, the US EPA and the Health Effects Subcommittee used a weighted five-year benefit profile in which 25% of the PM-related mortality benefits of policy were assumed to occur in the first and second years, and 16.7% were assumed to occur in each of the remaining three years (US EPA 2004a). Subsequently, following a suggestion from the US EPA (US EPA 2004b), the Science Advisory Board noted that considerable uncertainty remained but recommended that a lag structure was used in which 30% of the mortality reductions occur in the first year, 50% was distributed equally (12.5% per year) in years 2 through 5 and the remaining 20% was distributed equally over years 6 through 20 (Cameron & Ostro 2004).

This approach is still used as the primary assumption, although in recognition of the uncertainty, a number of alternative lag structures have been used also (Lepeule *et al* 2012; US EPA 2011): a 5-year distributed lag (20% per year over 5 years) and an exponential decay model based on analysis by Röösli *et al* (2005).

European Union

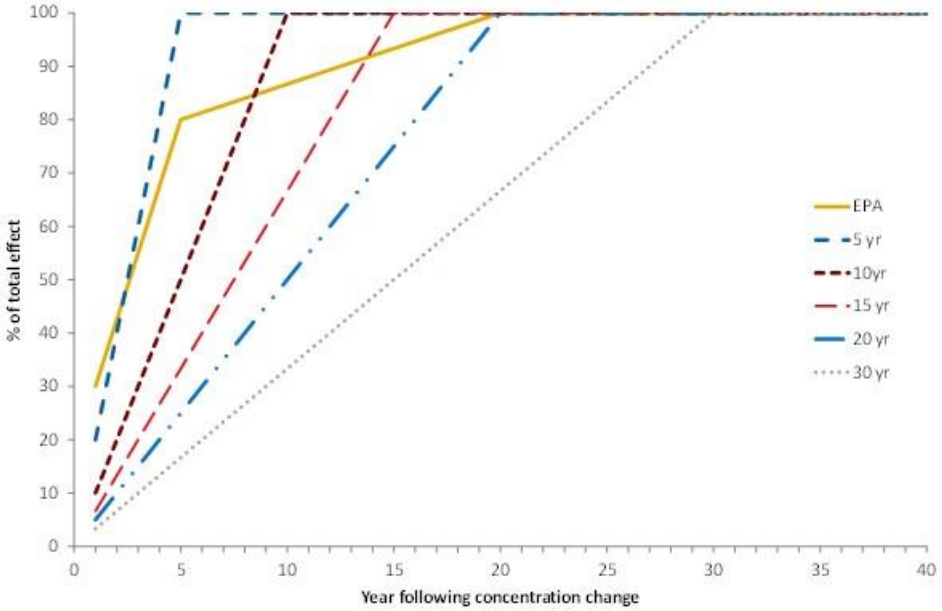
Work for the European Commission has examined the effects associated with a one-year pulse change (i.e. a sudden reduction in pollution for one year) as a way to understand the marginal effects (AEA Technology Environment 2005). Here, in contrast to a simple immediate 6% increase in mortality for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentrations, they spread the 6% over 11 years, assuming a 2.4% increase in year 1, followed by 0.36% increases in years 2 to 11, followed by reversion to the original mortality rate.

An analysis relating to the National Emissions Ceiling Directive adopted the US EPA's lag structure (Miller *et al* 2011).

United Kingdom

In the UK, damage costs were initially developed using a lag range for all chronic mortality effects between 0 and 40 years based on the advice of COMEAP (DoH 2001 in Birchby *et al* 2019). Subsequently COMEAP used lag options that included no lag and five, ten, 20 and 30 year phased-in lags in addition to the US EPA suggested lag structure based on advice from Walton (2010) as shown in Figure 22.

Figure 22: Selection of lag structures examined by Walton (2010)



Source: Walton (2010)

Table 23 shows the implications of these different lag structures on damage estimates in relative terms, using different discount rates. At a 5% discount rate, recommended for public policy analysis in New Zealand, the US EPA approach reduces the impact to 86% of what it would be with no lag and a 30-year lag reduces the impact to 54%.

In the absence of studies that have quantified the effect statistically over the long run, analysts using lagged benefits are making assumptions about the duration and shape of the lag curve. However, a zero lag with an instantaneous response to reductions in emissions over-estimates the measured impact.

Table 23: Implications of lag structures for impact estimates (Index: no lag = 100, <100 means a reduced impact)

Discount rate	No lag	EPA	5 yr	10 yr	15 yr	20 yr	30 yr
0%	100	100	100	100	100	100	100
1%	100	97	98	96	93	91	87
3%	100	91	94	88	82	77	67
5%	**100**	**86**	**91**	**81**	**73**	**65**	**54**
8%	100	80	86	72	62	53	41

Suggested approach for policy studies

The US EPA lag formula appears to be the most widely used approach, including most recently by Birchby *et al* (2019) in advice to DEFRA in the UK. Within the identified range of possible lag structures, it is conservative (i.e. it does not reduce the measured impact significantly). We suggest using the US EPA lag structure as the primary assumption for measuring the marginal impacts of changes in concentrations, consistent with international practice.

There is a question whether the lagged benefits approach is relevant to estimating total costs. This would be if populations are not stable so that some of the current population has only been exposed to elevated concentrations for a short space of time. Hales *et al* (2012) addressed this issue to some extent by limiting the analysis (in sensitivity analysis) to those residents who were in the same location five years previously. However, in general, data limitations will preclude this analysis.

6.2.3 Valuing morbidity impacts

In addition to the additional risks to life, air pollution has various ill-health impacts on those who continue to live.

Internationally, morbidity impacts are estimated to typically cost in the order of 8-10% of the mortality impacts (WHO 2015), although HAPINZ 2.0 estimated costs totalling only 2.2% of total social costs.[41]

Cardiac and respiratory hospital admissions

The social costs of hospital admissions include the financial costs of hospitalisation, productivity losses from time off work or school for those hospitalised, family and friends, and recovery costs after discharge from hospital including any long-term disability.

HAPINZ 2.0 estimated medical costs and loss of output (during hospitalisation, but not afterwards) summing to $6,350 and $4,535 per hospitalisation (June 2010 prices) for cardiovascular and respiratory hospital admissions (CVHA and RHA) respectively. It did not include any wider wellbeing losses.

[41] Morbidity costs were estimated at $187 million and total social costs at $8,429 million (Table 6.1 in Kuschel *et al* 2012b).

Hospitalisation

Following NZIER (2009), hospitalisation costs (in HAPINZ 2.0) were assumed to be the same as those for serious traffic injuries. The costs were taken from MoT based on a mid-1990s study of hospital costs in Dunedin and Waikato,[42] updated using a Producer Price Index (**PPI**) for Health Care and Social Assistance.[43]

The costs include a fixed (emergency) cost and a per day cost, estimated as the MoT hospital costs divided by the days hospitalised for a serious injury, and then multiplied by the estimated days in hospital for CVHAs and RHAs.

Lost income

The hospitalisation costs in HAPINZ 2.0 include costs of lost income, which are used as a measure of the loss of economic output. This assumes that, in a competitive economy, the gross income paid to a worker represents the value of the output produced. Exposure-response functions used to estimate hospitalisation rates are defined for people of all ages, so the hospitalised person is a random individual in the population who may be of working age or not. To estimate lost economic output requires an *average daily income for the whole population*. In addition, because the hospitalised day is random within a week, this needs to be an *average weekly income spread over seven days*.

The average weekly pre-tax income (from wages & salaries and self-employment)[44] for the working age population (those 15 years and over, including those working and not working) and the number of people of working age is from the Household Labour Force Survey;[45] the product of these is the total national weekly income. Dividing this by the total estimated resident population[46] provides an average weekly income; this is divided by 7 to estimate an average daily income. Using this approach, the estimate for 2010 is $62/day and for 2019 is $89/day (Table 24). [47]

Table 24: Average daily income (2019 NZ$ values)

Average weekly income (working age population)*	Number of people of working age	Total national weekly income ($m)	Total estimated population	Average weekly income (all people)	Average daily income (all people)
$796	3,902.00	$3,107	4,972.30	$624.87	$89.27

Source: Stats NZ (NZ.Stat) Income by sex, region, ethnic groups and income source; Stats NZ (Infoshare) Estimated Resident Population (Table Ref: DPE059AA)

Note: * Excluding Government transfer income

[42] As described in MoT (2020).

[43] Stats NZ Table ref: PPI019AA

[44] Income from Government transfers (e.g. benefits and superannuation) is not included as they do not represent compensation for activity that produces economic output.

[45] Stats NZ (NZ.Stat) Income by sex, region, ethnic groups and income source

[46] Stats NZ (Infoshare) Estimated Resident Population (Table Ref: DPE059AA)

[47] This is lower than the $126.80 used by MoT (2020), which is the estimated lost income for a sub-set of the population, based on the age and gender profiles of 2016-2018 crash data.

These numbers are combined in Table 25 to provide an updated estimate of hospitalisation costs using the HAPINZ 2.0 approach.

Table 25: Hospitalisation costs using HAPINZ 2.0 methodology (2019 NZ$ values)

Category	Serious injury (2019)	Cost per day	CVHA days/cost	RHA days/cost
Days in hospital	10.8		6.80	3.32
Hospital/medical	$9,600	$889	$6,044	$2,951
Emergency/pre-hospital (fixed)	$1,200	$1,200	$1,200	$1,200
Follow-on	$4,700	$435	$2,959	$1,445
Lost income		$89	$605	$295
Total costs	**$15,500**	**$2,613**	**$10,809**	**$5,891**

Source: MoT (2020) for serious injury costs and days for 2019; days in hospital for CVHA and RHA from analysis of unit record data from the National Minimum Dataset (NMDS), provided by MoH (see section 5.3.4)

Alternative hospitalisation costs

There are alternative sources to MoT for hospitalisation costs, based on more recent analysis than the MoT mid-1990s hospital cost survey.

- PHARMAC (2018) estimates an average cost per hospital day of $1,200 per day in a medical ward and $5,500 in intensive care (in 2017 NZ$ values); these costs would inflate to $1,252 and $5,739 respectively in 2019 prices using PPI as above.
- Telfar-Bernard and Zhang (2019) estimate a total of 80,999 RHAs in 2015[48] and costs from RHAs of $333.5 million in 2015, averaging approximately $4,117 per RHA. This inflates to $4,423/RHA in 2019 NZ$ values. They do not report on the length of stay for hospitalisations, but assuming 3.32 (from Table 25), would mean a cost of $1,332/day.
- The National Health Committee, an independent statutory body advising the New Zealand Minister of Health, estimated costs for different types of CVHA (Table 26). The weighted average cost (for the 2011/12 financial year) was $9,122/CVHA and a weighted average of 3.9 days ($2,334/day).[49] Using PPI, this would inflate to $10,418 in 2019 or $2,671/day.

Table 26: Cardiovascular disease impacts and costs (2012 NZ$ values)

	Hospital-isations	Individuals	Average days	$/hospital-isation	$/day	Deaths	DALYs
IHD	30,745	21764	4.4	$10,500	$2,386	6,027	8,900
Stroke	10,370	9,000	4.9	$7,400	$1,510	2,700	37,688
HTN	120	111	5.7	$6,200	$1,088	>280	3,300
RHD	487	392	9.7	$15,000	$1,546	>100	2,800

[48] This is based on a respiratory hospitalisation rate of 1762.5 per 100,000 (Raw number for 2015 in Table A100 on p145) and a 2015 NZ population of 4,595,703 (p28).

[49] The weighted averages are calculated using number of hospitalisations under each disease category.

	Hospital-isations	Individuals	Average days	$/hospital-isation	$/day	Deaths	DALYs
NRVHD	2,250	1,573	6.3	$23,800	$3,778	~470	6,730
AA	1,100	889	6.6	$27,800	$4,212	400	5,500
AF	9,600	7,700	2.0	$3,800	$1,900	189	4,385
CM	826	683	6.7	$12,100	$1,806	175	4,824
IHD	523	419	8.4	$16,000	$1,905	43	1,351
PVD	2,409	1,859	1.7	$14,100	$8,294	>100	1,300
VTE	2,779	2,500	3.4	$5,800	$1,706	44	1,084
Other	10,880	9,536	2.4	$5,600	$2,333	34	670
Total/ average	**72,089**	**56,426**	**3.9**	**$9,122**	**$2,334**	**10,562**	**78,532**

Source: National Health Committee (2013)

Notes: DALYs = Disability-adjusted life years, where one DALY represents the loss of one year lived in full health, estimated as years of life lost (YLL) plus years lived with disability (YLD) adjusted for severity. HTN = Hypertension; RHD = Rheumatic Heart Disease; NRVHD = Non-Rheumatic Valvular Heart Disease; AA = Aortic aneurysm; AF = Atrial Fibrillation; CM = Cardiomyopathies; IHD = Inflammatory Heart Disease; PVD = Peripheral Vascular Disease; VTE = Pulmonary Embolism and Venous Thrombo-Embolism

These alternative sources suggest the hospitalisation costs are higher than those estimated using MoT numbers. The difference is less stark if we include the fixed costs in calculating an average from the MoT numbers in Table 25, i.e. ($2,951+$1,200)/3.32 = $1,250/day, which closely matches the PHARMAC cost per day of $1,252.

Reduced life quality

HAPINZ 2.0 included values for morbidity effects that are more easily monetarised. Less-easily monetarised impacts are those in the form of a lower quality of life. In sensitivity analysis, a loss of life quality after hospitalisation was added based on 10% of the VoSL, which is the valuation approach used by MoT for the costs of a serious injury, and assuming a permanent disability (MoT 2020). HAPINZ 2.0 also noted that these life quality costs were relevant to the choice of VoSL for measuring impacts, because even if fewer years of life are saved, if reducing the risks of death of the elderly, there is a greater saving of trauma also. However, as Abelson (2008) suggests, separating mortality impacts from pain, suffering and trauma in life (morbidity), is a more transparent approach in analysis.

Health indicators have been developed that incorporate both the quality and the length of life into a common currency. The main two are QALY and disability-adjusted life year (**DALY**) (Hammitt 2013). A DALY is the sum of years of life lost (YLLs) and years lived with disability (**YLD**s). A YLD is calculated as the number of cases in a period multiplied by the average duration of the disease or disability, i.e. the prevalence (incidence times duration) and the severity of a health state based on a disability weight (**DW**) (MoH 2020b). So, the adverse health status impacts of air pollution might be measured using the YLD component of DALYs and the positive effects of air pollution reductions using the equivalent for QALYs.[50]

[50] See further discussion in PHARMAC (2015).

Several studies have estimated YLDs from the number of people affected (cases) and a DW, which is an estimate of the severity of the health effect from 0 (perfect health) to 1 (loss equivalent to death) (Salomon *et al* 2015). Telfar-Bernard and Zhang (2019) used 2006 estimated YLDs for respiratory diseases and multiplied these by 20% of a VoLY estimated at $176,000[51] to yield a cost of $35,200 per YLD in 2015 values, equivalent to $37,815 per YLD in 2019.[52] They also estimated a total of 39,456 YLDs associated with 80,999 RHAs in 2015, i.e. an average of approximately 0.49 YLDs per hospitalisation.

Restricted activity days

The loss of output during hospitalisation can be used to estimate the loss of income per restricted activity day (**RAD**). This was the approach taken in HAPINZ 2.0 and resulted in a cost of $62 per day (irrespective of a working or non-working day), assuming that the loss per RAD applied to the whole day on average.

A range was used in sensitivity analysis.

- The low end was based on a previous cost benefit analysis (MfE 2004) which assumed that each RAD would amount to a loss of 55% work time on average (based on 90% of RADs causing minor restrictions and the remaining 10% causing major loss of work). It assumed as cost per RAD of $62 x 0.55 = $34.
- The high end assumed a caregiver or parent has to take time off from work to support the person affected by the RAD, particularly in cases involving children or the elderly. This is equivalent to assuming the RAD is a working day rather than a random day that may or may not require time off work, i.e. $87/RAD was assumed which is $62 x (7/5).

Childhood asthma

The social costs of childhood asthma were not evaluated in HAPINZ 2.0. Asthma has impacts that include reductions in life quality, the need for medication, hospitalisation for more severe cases and deaths. Some of these costs are included in impacts already counted. The deaths from air pollution include those that are asthma-related and the YLDs estimated above are for all respiratory diseases, including asthma. The costs of asthma considered here are those resulting from GP visits, medication and hospitalisations.

GP visits and medication

The costs of additional GP visits and medication were assessed by Telfar-Barnard and Zhang (2019). For children, because private costs are zero, the GP visit costs were estimated using the level of General Medical Services subsidy per visit. From July 2015, the rate is $31.11 (GST

[51] The multiplication of the VoLY by 20% was described as arbitrary, using the same methodology as Holt & Beasley (2002).

[52] Producer Price Index (PPI) for Health Care and Social Assistance (Stats NZ Ref: PPI019AA), inflated from 2015Q2 (1050) to 2019Q2 (1128).

exclusive);[53] this rate still applies currently, although we assume that it reflected costs in 2015 and we inflate it to $33.42/visit in 2019$ using PPI.[54]

Pharmaceutical costs were estimated by Telfar-Barnard and Zhang using analysis of household level costs; the methodology is outlined in Telfar-Barnard *et al* (2011). They estimated total costs of pharmaceuticals for asthma treatment in 2015 at $35.6 million, a cost of $89/asthma case[55] or $95/case in 2019$. These are average costs for all asthma cases and not specifically for children, but we assume they apply to children.

Assuming costs for one GP visit and the average costs for pharmaceuticals, the total costs are estimated at $128/case.

Hospitalisations

An exercise by students at Otago University estimated the childhood costs of asthma in New Zealand. Carswell *et al* (2015) estimated costs including parents' expenses, time off work and intangible costs, such as stress and anxiety. They estimate these costs using expenditure and willingness-to-pay surveys of parents of hospitalised children.

The estimated median non-healthcare costs were estimated at $380.74 per night on top of the hospitalisation costs of $1,397 per day ($424 and $1,557 respectively in 2019 values). On average, asthma hospitalisations are estimated as averaging 0.92 days (discussed in section 5.3.4) resulting in an estimated cost per case of $1,822 (Table 27).

Table 27: Costs of childhood asthma hospitalisation (2014 & 2019 NZ$ values)

	2014	2019
Hospitalisation ($/per day)	$1,397	$1,557
Non-healthcare costs ($/per hospitalised day)	$380	$424
Total ($ per day)	$1,778	$1,981
For 0.92 days	$1,635	$1,822

Source: Carswell *et al* (2015); inflated to 2019 using PPI

6.2.4 Policy studies in New Zealand

Although HAPINZ 2.0 assessed the *total* costs of air pollution, the results have been used in policy studies which are based on *marginal* cost analysis (i.e. a measure of the change in total costs resulting from a small change in concentrations or emissions of pollutants). However, marginal costs may be significantly different from *average* costs because the most important health effects are *cumulative* and the benefits depend on repair to damaged health.

[53] https://www.health.govt.nz/our-work/primary-health-care/primary-health-care-subsidies-and-services/general-medical-services

[54] Producer Price Index for Health Care and Social Assistance (Stats NZ Table ref: PPI019AA)

[55] Based on 401,000 asthma cases in New Zealand in 2014/15. https://minhealthnz.shinyapps.io/nz-health-survey-2018-19-annual-data-explorer/_w_38bb6a53/#!/explore-topics

In contrast to emerging practice internationally, New Zealand policy studies have largely assumed that the benefits are instantaneous following emission reductions, with the long-run exposure-response relationship being used to predict the immediate effects. Below we note the major studies to highlight the more significant assumptions used.

Ministry for the Environment (2004)

The Ministry for the Environment (**MfE**) analysed the costs and benefits of proposed national environmental standards for air pollution in 2004 (MfE 2004).

Estimated reductions in premature deaths were multiplied by a VoSL which was adjusted downwards by 25% to reflect the older age of those dying prematurely using an assumption that VoSL reduces with age. (The relationship between VoSL and age is discussed later).

NZ Institute of Economic Research (2009)

The NZ Institute of Economic Research (**NZIER**) undertook an update of the 2004 cost benefit analysis (**CBA**) (NZIER 2009) and highlighted several shortcomings of the original analysis:

- There is no empirical basis for assuming either that elderly people are most affected or that the VoSL differs with age (see below). NZIER used the same VoSL as for traffic accidents and did not vary it with age.
- The costs of loss of life quality for those suffering from chronic ill-health were not assessed.
- No explicit allowance was made for medical costs saved by reducing "bad air" days.

The approach retained the structure of the 2004 analysis, but some economic input assumptions were updated including VoSL and the discount rate.

Covec/Tonkin + Taylor (2015)

A 2015 study for MoT addressed the costs and benefits of introducing Low Emission Zones (**LEZs**), regional emissions testing and road pricing in Auckland (Denne & Atkins 2015). It discussed developments in international CBAs, including those that took account of cessation lag[56] and the use of VoLYs rather than or in addition to VoSL.

Using life tables for analysis, alongside the exposure-response functions from HAPINZ 2.0, estimates were made of the life years gained from air quality policy options in addition to reductions in premature deaths. The analysis showed a significant difference between the net benefits using a VoLY and a VoSL-based analysis. However, the VoLY used ($25,000) was low (based on UK empirical studies).

[56] This recognised that the major effects are on chronic mortality and that repairs to health will not happen instantaneously with reductions in concentrations, but rather will emerge over time after living in lower concentrations for several years.

Market Economics (2019)

A 2019 study (Akehurst *et al* 2019) updated the 2004 MfE CBA and added an assessment of costs using VoLY in addition to VoSL, consistent with emerging international practice.

However, the Market Economics (**ME**) approach significantly over-estimated the number of YLLs and consequently the policy benefit in their VoLY analysis. This is because they distributed the total estimated number of reduced premature mortalities across age cohorts in proportion to the current *population* in those cohorts, rather than in proportion to current *deaths* in these cohorts (via a change in age-specific all-cause mortality rates). Thus, their analysis is not consistent with the expectation that most of the estimated premature deaths from air pollution are of old people (with few life years remaining) because this is where most of the deaths are in the absence of air pollution. In addition, they also adopted inconsistent and unexpected discount rates in analysis.[57]

Implications for HAPINZ 3.0

Policy analysis approaches in New Zealand have changed over time, taking account of improvements in understanding of the marginal effects and international practice. This includes the calculation of cessation lag, inclusion of loss of life quality costs and the use of VoLY alongside VoSL.

6.2.5 Damage costs

Damage costs are a way to value changes in emissions to air to compare the benefits to society of a change in policy/operation with the cost of implementing the change. They can be used to capture benefits of emission reductions of both harmful pollutants (e.g. PM_{10}) and greenhouse gases (e.g. CO_2) and for comparing options to identify which will produce the best overall outcome. Many international government agencies publish relevant values to be used in the assessment of costs and benefits of policy options in their jurisdictions (e.g. Powell *et al* 2019 for DEFRA).

Harmful emissions

In New Zealand, the application of damage costs has largely been in transport projects, such as assessing environmental impacts of roading infrastructure projects (NZTA 2021) or comparing the environmental performance of different bus fleets (Kuschel *et al* 2017). These damage costs have been developed using HAPINZ 2.0 estimates for urban areas (such as Auckland) with robust air emissions inventories (assuming emissions approximate exposure) and reviewing overseas values as a crosscheck.

Unit costs of harmful and CO_2 emissions are included in the *Monetised Benefits and Costs Manual* (**MBCM**) published by Waka Kotahi (NZTA 2021) based on values taken from Austroads (2012). These are shown in Table 28.

[57] Their estimate of VoLY was calculated using a starting value (inflated from 2012 to 2015) estimated by O'Dea and Wren (2012) that used a 3.5% discount rate, but ME use an 8% discount rate as their baseline assumption (and sensitivity analysis at 6% and 4%) in the remainder of their analysis, although the NZ Treasury recommended discount rate for public policy was 6% in 2019 (and is currently 5%).

Table 28: Damage costs for use in project evaluations in $/tonne (2016 NZ$ values)

Pollutant	Costs in NZ$/tonne	Value Base Date
PM_{10}	$460,012	2016
NO_X	$16,347	2016
VOC	$1,310	2016
CO_2e	$86.58	2016
CO	$4.13	2016

Source: NZTA (2021)

Greenhouse gases and the social cost of carbon

While determining damage costs to society resulting from harmful emissions is relatively straightforward, establishing comparable costs for GHG emissions is more difficult because the effects are global rather than purely local. The social cost of carbon (**SCC**) is the estimated total cost to society that results from the emission of a unit of a greenhouse gas (GHG), measured as its CO_2-equivalent (**CO_2e**). The SCC has been used in policy analysis of the benefits of policies that reduce GHGs, both in New Zealand and elsewhere.[58]

Often the SCC has been calculated from an estimate of marginal social damage costs of emissions at a global level.[59] This recognises that GHGs (and CO_2 in particular) are very long lived and mix thoroughly in the atmosphere so emissions from any one country result in damaging effects across the whole planet. However, measuring costs that fall outside of New Zealand is different from how social costs are usually measured and may not be an appropriate basis for the SCC. The reasons are:

- Social costs are a means to a desired end, not an end in themselves. They are used in analysis or pricing of emissions so that an optimal (socially desired) outcome can be achieved. The desired outcome would not be achieved using an SCC based on global damage costs because, unlike for purely local pollutants, this is not how New Zealand has defined its GHG objectives. Targets for GHG emissions are consistent with international commitments that reflect a precautionary principle and which include developed nations taking greater initial action.
- Measuring global costs is inconsistent with how CBA is used for policy. Using global damage cost as the basis for SCC would include the wellbeing impacts on people outside New Zealand. If this is valid for climate policy, arguably it should be used consistently across all policy issues, e.g. by pricing imported goods on the basis of external costs of production in other countries or measuring consumer surplus impacts for tourists rather than just the retained benefits of their expenditure.
- Defining global damage costs has high uncertainty.[60] Pindyck (2015) notes arbitrary inputs with large effects on the results (e.g. internationally agreed discount rates), uncertainty over climate sensitivity, lack of theory or data to develop damage

[58] For example, it is the basis for the costs of GHG emissions in the *Monetised Benefits and Costs Manual* (NZTA 2021).

[59] For example, see Nordhaus (2016).

[60] For example, see Pezzey (2019), Pindyck (2013), Pindyck (2015) and Scovronick *et al* 2019.

functions and the potentially significant but uncertain impacts of catastrophic climate change.

Instead, a preferred approach to identifying the SCC for New Zealand is using the marginal cost of abatement to achieve national targets consistent with international commitments.

Uncertainty of global damage costs

Using marginal abatement costs for the SCC is suggested by other researchers[61] and is used in the UK for policy analysis (Department of Business, Energy & Industrial Strategy 2019). The UK Government originally used a global damage cost-based approach for the SCC in public policy analyses, but following the introduction of limits on emissions, the approach shifted to one based on estimates of the marginal abatement costs to meet specific emissions reduction targets. Now, "short-term traded carbon values" are used to value the impact of government policies on emissions from sectors covered by the EU ETS (Department of Business, Energy & Industrial Strategy 2018). Short-term traded values are estimated from the average daily settlement prices of end of year EU Allowance futures contracts of 2018 and 2019 vintages, averaged over a period of three months.

Using the same approach in New Zealand, the current and projected future NZU price would be used. However, this might not be the best price. The cost to New Zealand of another kg of CO_2 is the marginal cost of coming back into compliance. This is the maximum unit cost of reducing emissions amongst the full set of actions taken to limit emissions, assuming the Government pursues a least cost emission reduction strategy. The NZU price is limited in this regard because the ETS does not include all sources (notably agricultural emissions are currently excluded) and includes some price controls.

A better approach would be to use a modelled estimate of the marginal cost of emission reduction to meet current and proposed future emission limits. NZ Treasury suggests values for GHG reductions based on estimates of future costs of emissions reductions (abatement) required to reach New Zealand's emissions targets (NZ Treasury 2020; 2021). A summary of values is shown in Table 29. Taking these values and interpolating to 2019 yields a central estimate of $88 with low and high estimates of $59 and $116 respectively.

Table 29: NZ Treasury recommended shadow emission values (2021 NZ$ values)[1]

	Central	Low	High
2020	$90	$60	$119
2025	$99	$67	$132
2030	$145	$97	$192
2035	$173	$116	$230
2040	$201	$135	$268
2045	$230	$154	$306
2050	$258	$173	$343

Source: 2020 values are in 2020$ (from NZ Treasury 2020); all other values are from 2021 (from NZ Treasury 2021)

[61] For example, see Morgan *et al* (2017) and Pezzey (2019).

6.3 What we did in HAPINZ 3.0

The approach used in HAPINZ 2.0 was updated for HAPINZ 3.0 to better reflect the development and application of social cost analysis in other countries (discussed in section 6.2). Major changes included:

- Estimation of VoLY as well as VoSL for mortality impacts.
- Inclusion of quality of life impacts in morbidity effects.
- Inclusion of separate values relevant to policy studies (adjusted for cessation lag) to account for the delay to the full achievement of benefits when the effects are dominated by chronic health impacts.
- Provision of a suite of New Zealand-specific damage costs.

The methodologies and values used are described in the following sections.

6.3.1 Values for mortality impacts

Two approaches were used for valuing mortality impacts, with the values summarised in Table 30 for our base case, and with low and high estimates for sensitivity analysis; we also show the effects of using a low (2%) discount rate.

Table 30: Central and alternative values for VoSL and VoLY (2019 NZ$ values)

	Central	Low	High
VoSL	$4,527,300	$4,050,742	$5,242,137
VoLY @5%	$263,843	$62,187	$305,502
VoLY @2%	$165,499		

Under the VoSL approach, the estimated change in the number of premature deaths[62] was multiplied by the current New Zealand-based VoSL for road crash deaths, as was done in HAPINZ 2.0. The published VoSL, at June 2019 prices, is $4,527,300 per fatality (MoT 2020).

Sensitivity analysis was undertaken on the VoSL using the 95% confidence intervals from the original study which established the VoSL for New Zealand (Miller & Guria 1991). That study produced a survey value of $1.9 million with 95% confidence intervals of $1.7 - $2.2 million, i.e. -10.5% to +15.8%.

Under the VoLY approach, the change in total life years was multiplied by VoLY. The VoLY was estimated from the VoSL, discounted over 40 years according to the formula of Aldy and Viscusi (2008) in section 6.2.3. A base case discount rate of 5% was chosen to be consistent with current (2022) Treasury advice. The resulting estimate is $263,843 (June 2019 prices).

Sensitivity analysis for VoLY used GDP per capita ($62,187 in 2019) to define the low value and the high VoSL value as input to analysis for the high value. Alternative values are also shown using a lower (2%) discount rate as adopted by NZ Treasury.

[62] This value would also apply to the costs of cancer cases due to air pollution if they had been included in HAPINZ 3.0.

6.3.2 Values for morbidity impacts

Cardiac and respiratory hospital admissions

The social costs of hospital admissions include the financial costs of hospitalisation, productivity losses from time off work or school for those hospitalised, family and friends, and recovery costs after discharge from hospital including any long-term disability.

For all hospitalisation costs, except quality of life, we used the MoT numbers (from Table 25), inflated to current values.

For quality of life, we used the Telfar-Bernard and Zhang (2019) methodology described previously to estimate a YLD, based on 20% of VoLY (0.2 x $263,843 = $52,769). The quality of life cost per hospitalisation was then assumed to be 0.49 of this amount (i.e. 0.49 x $52,769 = $25,857), which we applied to RHAs and CVHAs.

Table 31 summarises the base case assumptions and, for sensitivity analysis:

- a low cost option assuming zero quality of life impacts
- a high cost option assuming an average $2,500 per day for hospitalisation (with no fixed costs), plus quality of life impacts based on 10% of VoSL consistent with the MoT approach to valuing lost life quality from a serious traffic injury.

Table 31: Hospitalisation cost assumptions per admission (2019 NZ$ values)

Category	CVHA (base)	RHA (base)	CVHA (low)	RHA (low)	CVHA (high)	RHA (high)
Days in hospital	6.80	3.32	6.80	3.32	6.80	3.32
Hospital/medical	$6,044	$2,951	$6,044	$2,951	$17,000	$8,300
Emergency/pre-hospital (fixed)	$1,200	$1,200	$1,200	$1,200	$0	$0
Follow-on	$2,959	$1,445	$2,959	$1,445	$2,959	$1,445
Lost income	$605	$295	$605	$295	$605	$295
Quality of life impacts	$25,857	$25,857	$0	$0	$452,730	$452,730
Total costs (base assumption)	**$36,666**	**$31,748**	**$10,809**	**$5,891**	**$473,294**	**$462,770**

Source: Table 25 for base assumptions

Restricted activity days

For the social costs of RADs, we used the same approach as in HAPINZ 2.0 with an updated value of lost income.

We used an updated value of lost average income per day ($89/day from Table 24), with low and high values for sensitivity analysis of $49 ($89 x 0.55) and $125 ($89 x 7/5) respectively.

Childhood asthma

Childhood asthma costs included here are those resulting from GP visits, medication and hospitalisations.

GP visit and pharmaceutical costs were estimated at $128/case. Hospitalisation costs were based on Carswell *et al* (2015) (see Table 27). These are for costs of $1,981 per hospitalised day or $1,822 per case (in 2019 dollars), assuming 0.92 days in hospital on average.

For sensitivity analysis, we arbitrarily increased or decreased the costs by 50%.

6.3.3 Costs per case used in our assessment

The costs per case for analysis of total costs in HAPINZ 3.0 are summarised in Table 32.

Table 32: Costs per case of health effects (2019 NZ$ values)

Category	Central value	Low value	High value
Premature mortality ($/premature death)	$4,527,300	$4,050,742	$5,242,137
Premature mortality ($/life year lost)	$263,843	$62,187	$305,502
Hospital admission (cardiovascular) ($/admission)	$36,666	$10,809	$473,294
Hospital admission (respiratory) ($/admission)	$31,748	$5,891	$462,770
Restricted activity day ($/RAD)	$89	$49	$125
Childhood asthma ($/case)	$128	$64	$192
Childhood asthma hospitalisation ($/case)	$1,822	$911	$2,733

6.3.4 Recommended values for policy analysis

As discussed in section 6.2.1, policy analysis should modify the *mortality* costs using cessation lag assumptions as discussed above. Morbidity costs are assumed to stay unchanged.

Lag structure should include, as a central assumption, the US EPA values, i.e. following the introduction of policy to reduce emissions: 30% of the mortality reduction benefits occur in the first year, 50% is distributed equally (12.5% per year) in years 2 through 5 and the remaining 20% is distributed equally over years 6 through 20. Other lag structures might be used in sensitivity analysis. The suggested values are shown in Table 33; a central set of values should be based on the US EPA lag formula with sensitivity using no lag and 30 years.

Table 33: Costs per case of health effects for policy analysis (2019 NZ$ values)

Category	No lag	US EPA lag formula	30-year lag
Premature mortality ($/premature death)	$4,527,300	$4,102,005	$3,046,640
Premature mortality ($/life year lost)	$263,843	$239,057	$177,553
Hospital admission (cardiovascular) ($/admission)	$36,666	$36,666	$36,666
Hospital admission (respiratory) ($/admission)	$31,748	$31,748	$31,748
Restricted activity day ($/RAD)	$89	$89	$89
Childhood asthma ($/case)	$128	$128	$128
Childhood asthma hospitalisation ($/case)	$1,822	1,822	1,822

6.3.5 Damage costs

The HAPINZ 3.0 study provides the opportunity to consider costs more holistically by developing a suite of damage costs that can be applied consistently across New Zealand to assess benefits of reducing exposure to harmful emissions *and* GHGs resulting from emissions reduction strategies and other policy interventions.

We used the *HAPINZ 3.0 Health Effects Model* to develop New Zealand-specific damage costs for $PM_{2.5}$ and NO_X, for urban and rural areas to reflect significantly different population densities. Emissions and costs for $PM_{2.5}$ and NO_X were estimated at the CAU level and then assigned to urban or rural using the Stats NZ *Urban Area 2015* protocol[63] for each CAU as follows:

1. Main urban area (minimum population of 30,000)
2. Secondary urban area (population 10,0000 to 29,999)
3. Minor urban area (population 1,000 to 9,999)
4. Rural centre (populations 300 to 999)
5. Other (population less than 300).

CAUs in categories 1-3 were considered *urban* and those in categories 4-5 *rural*.

Damage costs for other pollutants were developed from other sources as outlined in the following sections.

Estimation of $PM_{2.5}$ and NO_X emissions

Emissions of $PM_{2.5}$ and NO_X were estimated for the two major anthropogenic sources – home heating (biomass burning) and motor vehicles. These sources represent the majority of anthropogenic $PM_{2.5}$ and NO_X emissions and information was readily available from published air emissions inventories covering New Zealand.

Home heating emissions were estimated for 2016 based on the 2013 national inventory (Wilton *et al* 2015). 2016 emissions were estimated assuming that:

- emissions changed in proportion to the change in the total number of households burning solid fuel (wood, wood pellets, or coal)
- the rate of change was linear between the 2013 and 2018 census.

On this basis, we estimated that 2016 home heating emissions were 93% of 2013 home heating emissions.

Motor vehicle emissions were based on those estimated for road transport and buses for a 2018/19 base year in the DTCC study (Kuschel *et al* 2021). NO_X emissions were taken directly from the DTCC estimates. Total $PM_{2.5}$ emissions were estimated as the sum of:

- **Exhaust** emissions. These are reported as PM_{10} in the DTCC study. However, we assumed that exhaust PM is essentially all $PM_{2.5}$.

[63] https://datafinder.stats.govt.nz/layer/105772-urban-area-2015-generalised/metadata/

- **Brake and tyre wear** $PM_{2.5}$ emissions. These are reported as PM_{10} in the DTCC study. We estimated $PM_{2.5}$ brake and tyre emissions based on the ratio of $PM_{2.5}$ to PM_{10} brake and tyre wear emissions factors for the default 2019 fleet in VEPM. The ratio at 80 km/hour (59%) was assumed for rural roads and the ratio at 40 km/hour (53%) was assumed for urban roads.
- **Road surface wear** (sealed roads) and unsealed road dust $PM_{2.5}$ emissions.

Estimation of damage costs

$PM_{2.5}$ and NO_X

For damage costs for $PM_{2.5}$ and NO_X, we matched emissions from the previous step with social costs from the *HAPINZ 3.0 Health Effects Model* as follows.

$PM_{2.5}$ costs were calculated based on the social costs due to $PM_{2.5}$ exposure from biomass burning and motor vehicle emissions. Social costs included premature mortality for adults 30+, cardiovascular hospitalisations, respiratory hospitalisations and restricted activity days.

NO_X costs were calculated based on the social costs of exposure to NO_2. Social costs included premature mortality for adults aged 30+ years, cardiovascular hospitalisations and respiratory hospitalisations, as well as asthma hospitalisations for children aged 0-18 years. Social costs of increased asthma prevalence for children aged 0-18 years due to annual NO_2 exposure were also included in the New Zealand total damage costs.

Other harmful pollutants

For other pollutants, in the absence of other information, we started with the damage costs outlined in the MBCM (NZTA 2021), assuming these values were applicable in urban areas, then calculated corresponding rural values using the urban/rural damage cost ratio for $PM_{2.5}$.

Note: The MBCM costs were updated from the previous Economic Evaluation Manual (**EEM**) costs which were based on the best available data at the time – which was largely Austroads (2012). The PM damage costs in the EEM were originally checked against estimates derived from HAPINZ 2.0 which established that the PM costs were reasonable. However, the desire was always to develop a suite of damage costs using New Zealand-specific data – hence this being identified as a key output for HAPINZ 3.0.

The damage costs developed in HAPINZ 3.0 are consistent with values used previously in the EEM/MBCM, with the exception of NO_X which is due to the higher exposure-response function found in the *HAPINZ 3.0 Cohort Study*. Annual average NO_2 values in NZ are much lower than those in the UK and if a supra-linear dose-response is occurring (see section 5.3.2), this would explain why the NZ values are much higher than those reported in the UK (Powell *et al* 2019).

CO_2e

The NZ Treasury publishes guidance on undertaking cost benefit analyses, including recommended annual values for CO_2 equivalent (**CO_2e**) emissions for use in policy assessment.

These values are based on the likely future *abatement* costs[64] rather than the *social* costs associated with the emissions. However, these are the best figures currently available in New Zealand to value CO_2e emissions and ensure consistency and comparability across cost benefit analyses.

We adjusted the NZ Treasury (2021) CBAx central value for CO_2e emissions for June 2019 to arrive at a figure of $88, in all areas – regardless of urban or rural.

Summary

The estimated damage costs developed in HAPINZ 3.0 for harmful emissions are summarised in Table 34, with the NZ Treasury CBAx costs for CO_2e emissions included for completeness. The costs are shown for urban and rural areas as well as New Zealand (on average).

Table 34: Damage costs developed in HAPINZ 3.0 for harmful emissions and the CBAx costs for greenhouse gases (2019 NZ$ values)

Pollutant	Costs in $/tonne Urban	Costs in $/tonne Rural	Costs in $/tonne New Zealand
$PM_{2.5}$	$622,786	$24,473	$382,524
NO_X	$499,526	$11,296	$186,037
SO_2	$36,491	$1,434	$22,413
VOC	$1,433	$56	$880
CO	$4.52	$0.18	$2.78
CO_2e	$88	$88	$88

6.3.6 Uncertainty

Uncertainty in the costs is indicated by the low and high values. These can be used to undertake sensitivity analyses.

In addition, there is uncertainty over the use of VoSL or VoLY for valuing the effects on mortality risk. As noted in earlier discussions in this chapter, the appropriate approach reflects the assumed relationship between the valuation of mortality risk reduction and age. Because this remains a subject of discussion in the literature, consistent with international practice, we have included both measures.

[64] The emissions values provided are based on estimates of future costs of emissions reductions (abatement) required to reach New Zealand's domestic emissions targets, as reflected in the Climate Change Commission's final advice. These values will be updated annually as knowledge improves on New Zealand's costs of abatement.

6.3.7 Summary of our approach

We used two approaches to valuing **mortality impacts** as follows:

- the change in premature mortality multiplied by the current New Zealand-based value of statistical life (VoSL) as was done in HAPINZ 2.0. The current VoSL is based on willingness to pay (WTP) to reduce the risk of road crash deaths.
- the changes to mortality to age-specific death rates in life tables to estimate changes in total life years and then multiply these by a value of life year (VoLY). We estimate a range of VoLYs (based on VoSL and typical discount rates used in policy analyses) then highlight our recommended value with upper and lower bounds.

We valued the following **morbidity impacts**:

- cardiovascular and respiratory hospital admissions using estimates of the costs of hospitalisations per day and the numbers of day per case
- restricted activity days using estimates of lost income
- quality of life impacts from estimates of years lived with disability (YLDs) per hospitalisation and costs of a YLD as a multiple of a VoLY.

We matched *HAPINZ 3.0 Health Effects Model* outputs for health impacts in urban and rural areas, to corresponding emission estimates to develop damage costs for $PM_{2.5}$ and NO_X. The urban/rural cost ratios were then applied to other pollutants with published urban costs. The output is **a suite of damage costs** that can be applied consistently across New Zealand to assess benefits of reducing of exposure to harmful and greenhouse gas emissions resulting from emissions reduction strategies and other policy interventions.

7. Designing the model

This chapter discusses the development of the *HAPINZ 3.0 Health Effects Model*.

It reviews the features in the previous HAPINZ 2.0 models, summarises the requested improvements and outlines the new and improved design of the HAPINZ 3.0 model. Details are provided on the enhanced features, especially for scenario modelling, together with a discussion on the ability to assess uncertainty.

7.1 Approach used in HAPINZ 2.0

HAPINZ 2.0 comprised two models, an exposure model and a health effects model (Kuschel *et al* 2012a). These are described below.

7.1.1 Exposure model

The exposure model provides monitored (for 73% of the population) and modelled (for 27% of the population) annual concentrations of PM_{10} and $PM_{2.5}$ using the methods discussed in section 4.1. Annual concentrations are disaggregated by CAU based on the 2006 Census (**CAU2006**).

The exposure model also provides national emissions estimates disaggregated by CAU and by source (i.e. domestic fires, motor vehicles, industry, open burning and natural).

The exposure model also includes:

- all input data used in the land use regression modelling to estimate pollutant exposure by source
- ambient air quality monitoring data
- exposure indicators (population density, home heating census data, vehicle kilometres travelled)
- corrections for HiVol equivalency
- derivation of default ratios (for $PM_{2.5}$ from available PM_{10} data)
- local emissions inventories
- derivation of default source contributions
- assumptions regarding industrial dispersion
- quality assurance calculations
- and all relevant references.

7.1.2 Health effects model

The health effects model takes the outputs from the exposure model (annual concentrations of PM_{10} and $PM_{2.5}$ by source and census area unit) and applies exposure functions to estimate health effects and social costs.

Estimated health effects and social costs are calculated by source and at the census area unit level (*Base Case Results*), and then summed to give national totals (*Base Case Output Table*). Health effects and social costs may also be aggregated by region, territorial authority, airshed and urban centre for ease of viewing.

The estimates are then tabulated separately by health effect nationally, for each region, for each territorial authority, for each airshed and for each urban area (*Report Tables Base Case*).

The model offered scenario testing whereby the following parameters can be changed (nationally) to estimate consequent changes in health effects and social costs:

- annual PM_{10} and $PM_{2.5}$ concentrations
- population
- all individual exposure-response functions
- all social costs values.

Scenarios may then be compared with the base case in a summary spreadsheet (*Comparison*).

Because the estimates are disaggregated down to CAU, the data may also be used to generate GIS maps which can then be viewed spatially. Instructions are provided in the model on how to do this manually (*Maps*).

7.2 Developments since HAPINZ 2.0

The HAPINZ 2.0 health effects model has been successfully used in numerous projects to date. However, the users asked for improvements in the model design in HAPINZ 3.0 to enhance its utility, in particular:

- Making the model easier to update
- Integrating the health effects and exposure models
- Making it easier to achieve spatial representation of results
- Making the model more robust (less easy to corrupt)
- Providing additional scenario testing options.

7.3 What we did in HAPINZ 3.0

7.3.1 Model format

As a first step, we reviewed a range of options for the format of the *HAPINZ 3.0 Health Effects Model*. Options investigated included an Excel spreadsheet, a GIS toolkit, a database or an automated software tool such as the WHO's AirQ+.[65]

We found that the advantages of retaining a spreadsheet-based model to be:

- Widely accessible as most users have Microsoft Excel.
- Transparent for end users. Most users are familiar with Excel, which means that they can interrogate and understand calculations, and key parameters (e.g. lookup tables).
- Spreadsheets are easier to update and change compared to a GIS tool, which requires specialist GIS input, software, and updates to code/scripts used in the tool.
- The HAPINZ spreadsheet model has significant flexibility compared with software tools such as AirQ+. For example, HAPINZ allows users to evaluate effects at multiple spatial scales (e.g. national, regional, airshed).

The disadvantages of using a spreadsheet-based model included:

- There is a manual process to export results to produce maps when updates or scenarios are developed.
- Spreadsheets can be easily corrupted unless sheets are locked, which then does not allow users to freely interrogate the data and calculations.
- Large datasets and complex calculations are difficult to manage in spreadsheets as they result in large file sizes and slow down the 'model' when trying to either open or run a scenario.

After considering the above, we decided to opt for an improved spreadsheet for the model itself but look to develop a prototype GIS tool, which would be made available online after the results had been publicly released. This tool will make key results available spatially and allow users to undertake sensitivity analysis for key variables.

The *HAPINZ 3.0 Health Effects Model* was built on the approach taken for the HAPINZ 2.0 models but incorporates the following improvements in functionality and ease of use:

- Merging the exposure model and health effects model into one overall model
- Making the spreadsheet easier to update by clearly identifying key inputs (including ambient monitoring concentrations and source apportionment for each location). These data are protected to avoid accidental changes. However, an option to input "user defined" data is provided.
- Simplifying the model to better reflect the accuracy of the source data

[65] AirQ+ is a software tool for health risk assessment of air pollution managed by the WHO's Regional Office for Europe. http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/activities/airq-software-tool-for-health-risk-assessment-of-air-pollution

- Expanding the scenario testing options to include reduction of pollutant concentrations from individual sources. For example, users can test the effectiveness of a policy that is expected to reduce the concentration of pollution attributed to biomass burning (assuming this causes a proportional reduction in effects).

We have minimised the potential for spreadsheet corruption and errors by:

- Using macros, match functions and lookup tables to ensure that formulae are looking at correct data
- Ensuring that input data are clearly identified and there is no duplication of data
- Ensuring that every worksheet is individually peer reviewed and signed off
- Locking cells for data which are not user-defined, and clearly stating where/why they are locked.

7.3.2 Key sources of uncertainty

The key sources of uncertainty in an assessment of air pollution health impacts are described as follows (WHO 2016a).

Air pollutants exist as a complex mixture

There is a considerable body of evidence from epidemiological studies confirming the adverse health effects associated with air pollution. However, the adverse effects attributed to a particular pollutant may actually be attributable to other pollutants in the mixture.

Baseline disease burden

Data on the number of deaths and cases of disease can be uncertain, particularly if data from a number of sources are combined or if projections of future cases are made.

Pollution exposure level

Because there is not full geographical coverage of monitors, some assumptions or modelling are required to estimate exposure. It is not possible to be certain that the assumed concentration coincides with the actual concentration in a given location. Even if population exposure is well estimated, individual exposures can vary substantially, as a result of differences in concentrations in different places and individuals own activity patterns. To accurately assess population exposure, personal monitoring would be required.

The exposure-response function

Exposure-response functions are derived from epidemiological studies, in which assumptions made in the analysis inevitably introduce some uncertainty into the results.

The counterfactual level of air pollution

The counterfactual level of air pollution is the baseline or reference exposure against which the health impacts of air pollution are calculated (e.g. having no air pollution). This is not a source of uncertainty in itself. However, the results of the assessment are sensitive to the counterfactual.

Deliberate simplifications of the model

Practical considerations may require the use of a simplified model, which can lead to increased uncertainty.

7.3.3 Uncertainty in the base case results

Each of the preceding chapters includes an assessment of the likely uncertainty associated with the input data and assumptions used for that component of the assessment process. Together these components generate the HAPINZ 3.0 base case results output by the model.

Exposure assessment (Chapter 3)

The uncertainty in the exposure assessment depends on the pollutant as follows:

- For **PM_{10}**, we used full or partial actual monitoring datasets to assess exposure for 84% of New Zealand's population, with the balance estimated from proxy monitoring sites. It is difficult to quantify the actual uncertainty associated with this approach but we expect the uncertainty in PM_{10} concentrations for unmonitored areas to be in the order of +/-25%.
- For **$PM_{2.5}$**, we used full or partial actual monitoring datasets to assess exposure for 53% of New Zealand's population, with the balance estimated using $PM_{2.5}$ / PM_{10} ratios linked to biomass burning (which were used to assess exposure for 16% of New Zealand's population) or proxy monitoring sites. It is difficult to quantify the actual uncertainty associated with this approach but we expect the uncertainty in $PM_{2.5}$ concentrations for unmonitored areas to be in the order of +/-15% in locations where concentrations are based on biomass burning, and +/-30% in locations based on proxy monitoring sites.
- For **NO_2**, we relied on the estimates generated from the Waka Kotahi VEMT and NVED tools. It is difficult to quantify the actual uncertainty of this approach but, based on validation exercises commissioned by Waka Kotahi (T&T 2020; T&T 2021), we expect the uncertainty in the predicted NO_2 concentrations to be in the order of +/-25%.[66]

Note: As mentioned in section 3.3.8, in HAPINZ 3.0, we developed and used New Zealand specific exposure-response functions for PM_{10}, $PM_{2.5}$ and NO_2. This means that uncertainty in the exposure assessment is captured (to some extent) in the uncertainty of the exposure-

[66] T&T (2021) notes a higher uncertainty for predictions at specific roadside locations. However, we used meshblock average and CAU average values in the *HAPINZ 3.0 Cohort Study* and the *HAPINZ Health Effects Model* which are dominated by background concentrations. These are likely to be more accurate (as they cover a larger spatial area) and also more representative of the average exposure of the population in those areas.

response functions and is therefore captured in the overall uncertainty estimates quoted in the results.

Source attribution (Chapter 4)

The uncertainties in source attribution are estimated in terms of +/-% values.

For example, we estimate the uncertainty in our source apportionment allocations for domestic fires in HAPINZ 3.0 is likely to be less than +/-10%. The uncertainty for other sources is likely to be higher.

Health impacts (Chapter 5)

The uncertainties in the ERFs are reflected in the 95% confidence intervals.

For example, the ERF for premature mortality (and YLLs) in all adults, aged 30 years and over, is 1.105 (1.065 to 1.145) per 10 µg/m^3 annual $PM_{2.5}$.

Social costs (Chapter 6)

The uncertainties in the costs per case are indicated in the low and high value estimates for the central values recommended.

For example, the recommended central VoLY @ 5% is $263,843 (in $2019) with a low value of $62,187 and a high value of $305,502.

7.3.4 Uncertainty in the scenario results

In addition to generating results for the base case (2016), the *HAPINZ 3.0 Health Effects Model* has the capability for sensitivity testing and scenario modelling.

The model's input sheet lists all primary health impacts (due to $PM_{2.5}$ and NO_2) and secondary health impacts (due to PM_{10}). Default ERFs and costs for each health impact are provided, with the confidence intervals or high/low estimates for each also shown (see Figure 23). However, users can define their own ERF and cost values for each health impact to undertake **sensitivity testing**.

Additional functionality is provided to enable users to undertake more detailed **scenario modelling**. PM concentrations by source (e.g. PM from domestic fires) or the overall pollutant concentrations can be adjusted using scalars (see Figure 24). The population (set to a base year of 2016) can also be adjusted.

The likely uncertainty associated with using the model for sensitivity testing (if using the defaults or indicated ranges for the ERF and cost values) is covered earlier in this section regarding the input data and assumptions. However, to give users an indication of the likely uncertainty of using the scalars for scenario modelling we undertook additional analyses as follows.

Figure 23: Defaults and recommended ranges in the model for sensitivity testing

Run Base | Run Scenario | Clear Inputs

Primary Health Outcomes	Relative risk				Cost (as at June 2019)			
All are additive except those indicated by an * (use either premature mortality or YLL not	Default	User defined		Range	Default	User defined		Range
PM2.5								
Mortality								
Premature mortality for all adults (30+ years) due to annual PM2.5 exposure	1.105		per 10 µg/m^3	*1.065 - 1.145*	$4,527,300		$/premature death	*$4,050,742 - $5,242,137*
* Premature mortality for Māori (30+ years) due to annual PM2.5 exposure	1.105		per 10 µg/m^3	*1.065 - 1.145*	$4,527,300		$/premature death	*$4,050,742 - $5,242,137*
* Premature mortality for Pacific adults (30+ years) due to annual PM2.5 exposure	1.105		per 10 µg/m^3	*1.065 - 1.145*	$4,527,300		$/premature death	*$4,050,742 - $5,242,137*
or								
Years of life lost (YLL) for all adults (30+ years) due to annual PM2.5 exposure	1.105		per 10 µg/m^3	*1.065 - 1.145*	$263,843		$/years of life lost	*$62,187 - $305,502*
* Years of life lost (YLL) for Māori (30+ years) due to annual PM2.5 exposure	1.105		per 10 µg/m^3	*1.065 - 1.145*	$263,843		$/years of life lost	*$62,187 - $305,503*
* Years of life lost (YLL) for Pacific (30+ years) due to annual PM2.5 exposure	1.105		per 10 µg/m^3	*1.065 - 1.145*	$263,843		$/years of life lost	*$62,187 - $305,504*
Morbidity								
Cardiovascular hospitalisations for all ages due to annual PM2.5 exposure	1.115		per 10 µg/m^3	*1.084 - 1.146*	$36,666		$/admission	*$10,809 - $473,294*
Respiratory hospitalisations for all ages due to annual PM2.5 exposure	1.070		per 10 µg/m^3	*1.021 - 1.122*	$31,748		$/admission	*$5,891 - $462,770*
Restricted activity days for all ages due to annual PM2.5 exposure	0.9		per 10 µg/m^3	*0.5 - 1.7*	$89		$/RAD	*$49 - $125*
NO2								
Mortality								
Premature mortality for adults (30+ years) due to annual NO2 exposure	1.097		per 10 µg/m^3	*1.074 - 1.120*	$4,527,300		$/premature death	*$4,050,742 - $5,242,137*
or								
Years of life lost (YLL) for all adults (30+ years) due to annual NO2 exposure	1.097		per 10 µg/m^3	*1.074 - 1.120*	$263,843		$/years of life lost	*$62,000 - $226,787*
Morbidity								
Cardiovascular hospitalisations due to long-term NO2 exposure	1.047		per 10 µg/m^3	*1.031 - 1.064*	$36,666		$/admission	*$10,809 - $473,294*
Respiratory hospitalisations for all adults (30+ years) due to annual NO2 exposure	1.130		per 10 µg/m^3	*1.102 - 1.159*	$31,748		$/admission	*$5,891 - $462,770*
*Asthma/wheeze hospitalisations for 0-18 year olds due to annual NO2 exposure	1.182		per 10 µg/m^3	*1.094 - 1.276*	$1,822		$/case	*$911 - $2,733*
Asthma prevalence for 0-18 year olds due to annual NO2 exposure	1.050		per 4 µg/m^3	*1.020 - 1.070*	$128		$/case	*$64 - $192*
Secondary Health Outcomes	**Relative risk**				**Cost (as at June 2019)**			
Not additive - only used for back casting	Default	User defined		Range	Default	User defined		Range
PM10								
Mortality								
Premature mortality for all adults (30+ years) due to annual PM10 exposure	1.111		per 10 µg/m^3	*1.089 - 1.133*	$4,527,300		$/premature death	*$4,050,742 - $5,242,137*
* Premature mortality for Māori adults (30+ years) due to annual PM10 exposure	1.111		per 10 µg/m^3	*1.089 - 1.133*	$4,527,300		$/premature death	*$4,050,742 - $5,242,137*

Figure 24: Adjustments available in the model for scenario modelling

Source (*PM only*)		
Domestic Fires	1.0	
Motor Vehicles	1.0	
Industry	1.0	
Windblown Dust	1.0	
Sea Spray	1.0	n/a
Secondary PM	1.0	n/a

Pollutant		
PM2.5	1.0	
PM10	1.0	
NO2	1.0	

Population		
2016 (base)	1.0	

Using population scalars

Using the population scalar in the model adjusts not only the population but also the baseline mortality and baseline hospitalisation counts in the model, from which we derive the air pollution mortality and morbidity.

We tested a population scalar approach to predict baseline mortality and baseline hospitalisations for 2016 (to compare against the HAPINZ 3.0 data) for:

- a two-year prediction period (from 2014 to 2016)
- a ten-year prediction period (from 2006 to 2016).

We used estimated resident population (**ERP**), averaged over three years, to calculate the population scalar for the different time periods. For example, in the case of the two-year prediction timeframe we used the ratio of 2013-2015 ERP (three-year average) to the 2015-2017 ERP (three-year average) to give us 2014 versus 2016. We assumed stable mortality rates and hospitalisation rates over the relevant timeframes to predict mortality and hospitalisations in each CAU for 2016. **Three-year averages were used to compensate for**

inter-annual variability in temperature which can have a significant impact on annual baseline mortality and morbidity.

See Appendix E.1 for detailed results.

Accuracy of baseline mortality predictions

The scalar calculated from the total ERP gave reasonable results for baseline mortality in 2016 (2015-2017) when predicted **two years into the future** from 2014 (2013-2015). Over this short period, the population increased by 4.2%.

- This approach predicted 29,684 deaths nationally compared with the observed 29,690 deaths (an error of 0.02%).
- At the Territorial Authority (**TA**) level, the differences were greater but for most urban Tas predicted mortality was still within 5% of observed. For example, Auckland City (7,653 predicted vs 7,633 observed, an error of 0.3%). However, less populated Tas recorded greater errors of around 10% on average but up to 41%, in the case of Mackenzie District (which had only 19 observed deaths).
- Results for CAUs were more variable with 59% of CAUs recording less than 20% error in their predicted deaths but 27% of CAUs having over 30% error and 3% of CAUs having over 100% error.

Not surprisingly, results were less accurate for baseline mortality in 2016 (2015-2017) when predicted **ten years into the future** from 2006 (2005-2007). Over this longer period, the population increased by 12.6%.

- This approach predicted 28,885 deaths nationally compared with the observed 29,690 deaths (an error of 2.7%).
- At the Territorial Authority (**TA**) level, the differences were greater but for most urban Tas predicted mortality was still within 10% of observed. For example, Auckland City (7,344 predicted vs 7,633 observed, an error of 3.8%). As before, the less populated Tas recorded greater errors of around 12% on average, with several Tas recording about 20% error.
- Results for CAUs were more variable with 41% of CAUs recording less than 20% error in their predicted deaths but 44% of CAUs having over 30% error and 5% of CAUs having over 100% error.

Accuracy of baseline morbidity predictions

The scalar calculated from the total ERP also gave reasonable results for baseline cardiovascular (CVHA) and respiratory (RHA) hospital admissions in 2016 (2015-2017), when predicted **two years into the future** from 2014 (2013-2015). Over this short period, the population increased by 4.2%.

- This approach predicted 57,181 CVHAs nationally compared with the observed 57,872 CVHAs (an error of 1.2%) and 67,841 RHAs nationally compared with the observed 70,425 RHAs (an error of 3.7%).
- At the Territorial Authority (**TA**) level, the differences were greater but predictions for CVHAs in more urban Tas were still within 10% of observed and for predicted RHAs were within 15% of observed.

- Results for CAUs were more variable. 66% of CAUs recorded less than 20% error in their predicted CVHAs with only 19% of CAUs having over 30% error and only 1% of CAUs having over 100% error. Results for RHAs were similar with 68% of CAUs recording less than 20% error in their predictions with only 19% of CAUs having over 30% error and only 1% of CAUs having over 100% error.

Again, results were less accurate for baseline CVHAs and RHAs in 2016 (2015-2017) when predicted **ten years into the future** from 2006 (2005-2007). Over this longer period, the population increased by 12.6%.

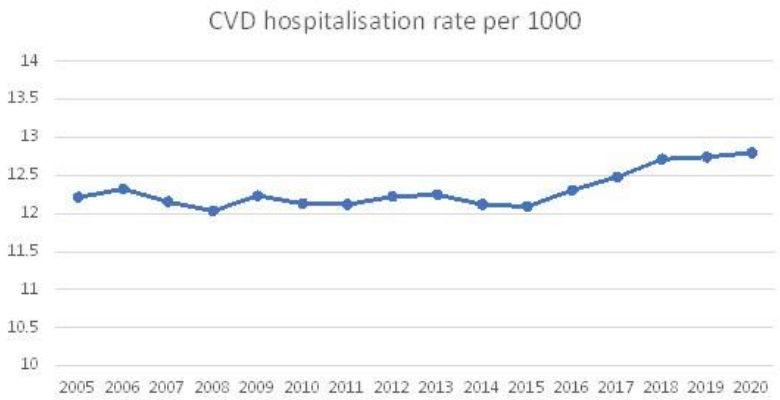
- This approach predicted 57,508 CVHAs nationally compared with the observed 57,872 CVHAs (an error of 0.6%) but only 56,748 RHAs nationally compared with the observed 70,425 RHAs (an error of 19.4%).
- At the Territorial Authority (**TA**) level, the differences were greater with predictions for CVHAs in more urban Tas were generally within 20% of observed and for predicted RHAs were within 40% of observed.
- Results for CAUs were more variable. While 40% of CAUs recorded less than 20% error in their predicted CVHAs, 45% of CAUs had over 30% error and 4% of CAUs had over 100% error. Results for RHAs were similar with 37% of CAUs recording less than 20% error in their predictions with 49% of CAUs having over 30% error and 3% of CAUs having over 100% error.

Caution regarding stable mortality and morbidity rates

The ability to apply a scalar (based on population) for mortality and morbidity, with confidence, assumes stable death rates and stable hospitalisation rates which is not necessarily true over time.

For example, the CVHA rate was relatively stable from 2005 to 2015 but has been steadily increasing since then (see Figure 25), potentially due to an ageing population, and/or changes in underlying risk factors (such as obesity and diabetes) in the population.

Figure 25: Cardiovascular disease hospitalisations per 1000 people, 2005-2020 (unadjusted rate)



Source: National Minimum Dataset

By comparison, the RHA rate gradually increased between 2005 and 2019 but dropped suddenly in 2020 (see Figure 26), most likely due to COVID-19 border closures and lockdowns

severally limiting the impact of seasonal influenza and other diseases (such as the respiratory syncytial virus (RSV) which affects young children).

Consequently, any predictions for mortality and morbidity beyond 2019 should be treated with extreme caution.

Figure 26: Respiratory disease hospitalisations per 1000 people, 2005-2020 (unadjusted rate)

Source: National Minimum Dataset

Conclusion

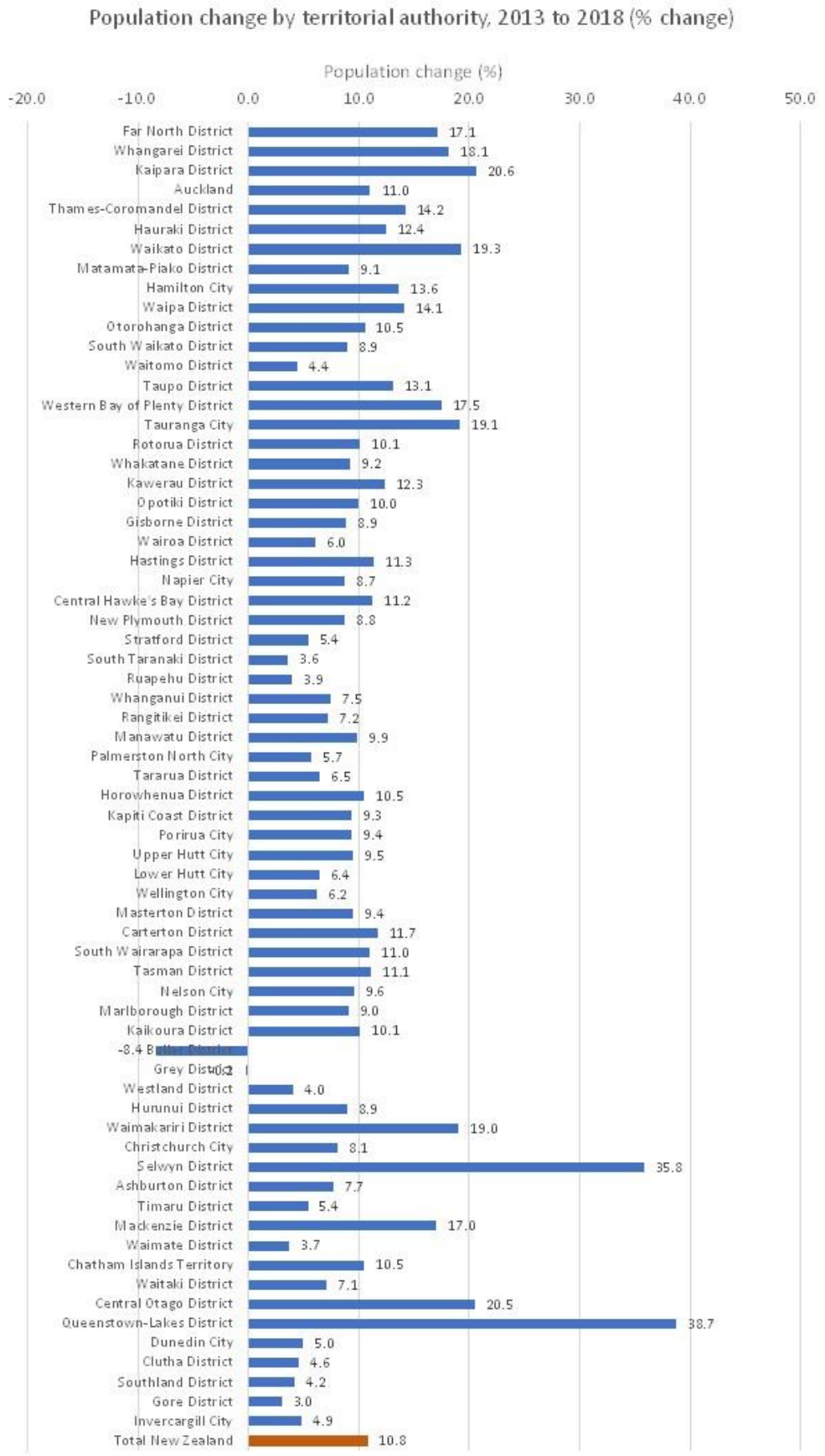
Using estimated resident population as a population scalar is reasonable at the national level for predicting baseline mortality and morbidity (generally accurate to +/-3%) in short-term forecasting (e.g. 2-3 years from the base year). However, longer-term forecasting (e.g. 10 years out) introduces more error (up to +/-5% in most cases, except for RHAs where the error may be as much as +/-20%).

The error in the predictions increases significantly at finer scales, such as TA and CAU. However, this is mainly because an overall increase in New Zealand's total population results from uneven growth (and even reductions in some areas) across the country as shown in Figure 27.

Consequently, **we recommend using sub-national population scalars if users want to compare results for finer spatial scales**. For example, a population scalar for Tauranga City should be calculated for scenarios involving Tauranga City. This will help to overcome the increased errors seen in finer spatial scales caused by using a national average scalar.

Note: These error estimates in this analysis are based on only one set of time points, and therefore are *indicative* only. As mentioned, the population scalar approach assumes stable mortality and hospitalisation rates; it does not account for any underlying changes in rates (e.g. due to COVID-19), which may lead to increased error.

Figure 27: Population change by individual TA and New Zealand total between 2013 and 2018)



Using pollution scalars

Using the pollution scalars in the model adjusts the population exposure, the source attribution and the air pollution mortality and morbidity impacts associated with the relevant pollutant.

As we had air quality data for years between 2006 and 2019, we took a slightly different approach to that used to assess the population scalars. We tested pollution scalars to predict potential changes in the population exposure for:

- a three-year prediction period (from 2016 to 2019)
- a ten-year prediction period (from 2006 to 2016)

We used passive sampling data collected by Waka Kotahi in the *National Ambient Air Quality Network*[67] to develop an NO_2 scalar for the different time periods. This was based on the trends in the annual NO_2 monitoring results at the 34 sites which have been operating continuously since 2007.

We used ambient air quality data collected by regional councils and others recorded in *Land Air Water Aotearoa* (**LAWA**) [68] to develop a $PM_{2.5}$ scalar for the different time periods. This was based on a population-weighted annual average $PM_{2.5}$ concentrations calculated for sites across New Zealand, some of which have been monitored since 2006 (or even earlier).

See Appendix E.2 for detailed results.

Accuracy of NO_2 predictions

Due to limited ambient monitoring data, the NO_2 exposure dataset in the HAPINZ 3.0 model uses exposure estimates from the Waka Kotahi NVED tool (described in section 3.2.4). Whilst insufficient to provide a robust *assessment* of exposure across New Zealand, monthly passive sampling data from the Waka Kotahi in the *National Ambient Air Quality Network* can be used to assess *trends* over time.

We reviewed available results to identify sites which could be used to assess long-term trends. Thirty-four sites have been monitored continuously (more or less[69]) since 2007. As a first step, we used linear regression to assess the rate of change (slope) at each site from 2016 to 2019 and from 2006 to 2016 then estimated an overall average rate of change. We then compared the trend at each site versus the average trend to assess the error distribution.

The scalar calculated from overall NO_2 trends gave reasonable results for individual site trends for 2019 when predicted **three years into the future** from 2016. From 2016 to 2019, the passive NO_2 concentrations for the 34 sites decreased by 6.5% on average (equivalent to a scalar of 0.9354 for forecasting relative to 2016). 67% of individual sites were within 10% of the overall average trend, 97% within 20% and only 3% (1 site) deviating by over 25%.

---

[67] See https://www.nzta.govt.nz/resources/air-quality-monitoring/

[68] See https://www.lawa.org.nz/

[69] A few sites had insufficient monthly averages to calculate a valid annual average for some years so we interpolated an annual average from the years either side to construct a "complete" dataset for our analysis. This approach was used to estimate 10 missing annual averages out of 442 in total (only 2.3%).

Results were less accurate (but still good) for individual site trends for 2016 when predicted **ten years into the future** from 2006. From 2006 to 2016, the passive NO_2 concentrations for the 34 sites increased by 15.0% on average (equivalent to a scalar 0.8736 for backcasting relative to 2016). 50% of individual sites were still within 10% of the overall average trend, with 74% within 20% and 18% (6 sites) deviating by over 25%.

Accuracy of $PM_{2.5}$ predictions

Sufficient records were available in LAWA for us to develop complete $PM_{2.5}$ 2006 dataset, based on actual monitoring data, with any gaps in the monitoring record filled following the same methodology we used for the 2016 dataset. Using the 2006 population by CAU, we calculated an actual 2006 populated-weighted annual average $PM_{2.5}$ concentration (7.61 $\mu g/m^3$) to compare with the actual value for 2016 (6.53 $\mu g/m^3$). We then applied the ratio of the population-weighted annual average $PM_{2.5}$ concentrations (1.1648 from 7.61/6.53) to backcast the 2016 results by CAU **ten years into the past** to create a predicted 2006 dataset and recalculated the 2006 populated-weighted average, using the 2006 population data.

The $PM_{2.5}$ scalar approach yielded a predicted 2006 populated-weighted annual average of 7.65 $\mu g/m^3$ vs an actual of 7.61 $\mu g/m^3$ (an error of 0.6% overall). At the CAU level, annual predictions of $PM_{2.5}$ were within 10% of the actual $PM_{2.5}$ averages for 48% of individual CAUs (covering 65% of the total 2006 population). 95% of all CAUs were within 20% (covering 96% of the population) with only 2% of CAUs deviating by more than 25%.

Conclusion

Using an NO_2 scalar based on the average overall trends seen at the 34 long-term passive sampling sites is reasonable at the national level for predicting changes in NO_2 exposure in short-term forecasting (e.g. 3 years either side of 2016) and even in longer-term forecasting (e.g. 10 years).

Using a $PM_{2.5}$ scalar based on the population-weighted annual average is reasonable at the national level for predicting changes in $PM_{2.5}$ exposure relative to a known base year, even in long-term forecasting (e.g. 10 years).

The error in both pollution scalars increases significantly at finer scales, such as individual NO_2 sites or CAUs. However, the majority of individual locations still record no more than a 20% error, with close to half being within 10%. As with the population scalars discussed previously, **we recommend using sub-national pollution scalars if users want to compare results for finer spatial scales** to help overcome the increased errors from using national average scalars.

7.3.5 HAPINZ 3.0 model and users' guide

The *HAPINZ 3.0 Health Effects Model* itself is available as a separate deliverable (an Excel workbook) as follows:

Sridhar S et al (2022a). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Health effects model*. Excel model prepared by S Sridhar, J Metcalfe and K Mason for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, March 2022.

A users' guide has been prepared for running the model covering:

- The functions available in the model
- How to change inputs for sensitivity testing or scenario modelling
- How to update the model with air quality or population data.

This is available as a separate document as follows:

Sridhar S et al (2022b). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Health Effects Model – Users' guide*. Guide prepared by S Sridhar, G Kuschel and K Mason for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, March 2022.

8. Communicating findings

This chapter summarises how findings were communicated in the HAPINZ 2.0 study, reviews developments in communicating challenging environmental messages and outlines a more effective approach for messaging in the latest HAPINZ 3.0 update.

8.1 Approach used in HAPINZ 2.0

The deliverables for HAPINZ 2.0 were grouped into two tiers.

For a more general audience:

- A *Summary Report* (volume 1) which presented the main findings of the study and described the workings of the health effects model (Kuschel *et al* 2012b)
- The *Health Effects Model*, based on an Excel spreadsheet, which allowed end-users to output results nationally, regionally, by local areas, or by airshed. End-users were also able to run scenarios to undertake sensitivity testing to test the effects of different assumptions, evaluate the effects of population and emissions trends, or review the effectiveness of different air quality management options.

For a more technical audience:

- A suite of *Technical Reports* (volume 2) which outlined in more detail the methodology followed (Kuschel *et al* 2012c)
- A detailed *Exposure Model* which contained all data, calculations and assumptions used to derive PM_{10} exposure for each CAU by source.

Communicating the results for HAPINZ 2.0 was challenging for several reasons, most notably:

- What do you do when "natural" sources result in nearly half of the impacts?
- What do you do when people think NZ$3.56M is too high a value of statistical life?
- What do you do when the public don't accept that air pollution causes effects?

The HAPINZ 2.0 findings were released through traditional mechanisms (an announcement on the Health Research Council website) but we did prepare in advance a list of questions and answers for media as we were expecting considerable interest from the media. The study findings were widely reported in the print media across New Zealand in late July and early August 2012.

8.2 Developments since HAPINZ 2.0

Despite widespread consensus that air pollution is harmful, most people still do not understand why they should be concerned or how air pollution affects their health. Quantitative estimates of health effects and social costs are important tools in the development of evidence-based policy. However, premature deaths and value of statistical life are poor metrics to use to communicate/engage with the wider community about air quality and health.

This section reviews the literature on improved strategies to more effectively communicate environmental messages that has arisen since HAPINZ 2.0.

8.2.1 What is the goal of good communication about air quality and environmental health?

For experts in environmental health, including in air quality, what matters at the broadest level is that we have a physical environment which promotes good health for all New Zealanders. It is a vision that many New Zealanders share.

To build these healthy environments for all New Zealanders, policy makers need to implement policies and practices based on the best knowledge and evidence. A key part of implementing evidence-led policies and practices for equitable health and wellbeing, is not just public understanding of environmental health and air quality issues, but also their active support for policies and practices that build and support healthy environments.

8.2.2 What is standing in the way of public understanding and support for evidence led policy?

Many barriers exist to public understanding and support for policies that build health environments.

One significant barrier is what the public believe about environmental health, what builds it, who is responsible for it, and how we (as a society) can create good environmental health. Decision-makers are led by public support and demand for new solutions. Public demand reflects dominant cultural understandings about environmental health.

When the prevailing shared cultural stories about environmental health and air quality are too shallow or unproductive, it makes it hard to build support for more effective, but complex, policy solutions.

For example, an interesting study conducted in the United States found that how people thought (or didn't think) about environmental health influenced their thinking about effective ways to build healthy physical environments (Lindland *et al* 2011). Specifically, the most people thought environmental health was limited to being able to identify key threats to people from environmental hazards. People struggled to identify key agencies, institutions, hierarchies, professions, and skill sets in the field of environmental health. This lent itself to patterns of thinking in which responsibility for environmental health was seen to lie at the household level and with individuals. Regarding specific solutions, people consistently focussed on steps individuals should take to increase their awareness and improve their decision making. They could see little or no role for environmental health workers in building healthy physical environments (except for taking some regulatory protective action against significant hazards).

What this research shows is that when experts then try to talk about the role of institutions or organisations in building health physical environments, air quality and improving it, people have limited ways of thinking about it.

However, cultural narratives are not monolithic. Alongside dominant shallow understandings of complex issues, other more nuanced but recessive understandings also exist.

Dominant narratives are ones that:

- show up most often in the public discourse
- are readily available to people (i.e. they are often the first thoughts that someone will have when asked their opinion on an issue)
- are simple and easily accessible by our fast-thinking brain.

Recessive narratives are ones that:

- show up less often in the public discourse
- are harder for people to access (i.e. they are not necessarily the first thought someone might have on the issue)
- often require slower thinking (i.e. more time to reflect on the issue).

In the case of environmental health, the same research in the United States found some recessive understandings that aligned more with expert knowledge. For example:

> Both experts and public informants also recognized that powerful commercial interests are often not aligned with environmental health efforts and represent a substantial challenge to protecting the public's health from negative "man-made" environmental impacts. (Lindland *et al* 2011).

Over time, through strategic, consistent and proven communication across a field of practice, such recessive narratives that support more helpful evidence-based understandings can become more dominant in the public narrative.

If dominant narratives change in this way over time and, for example, environmental health is understood from a structural perspective, the public appetite for new information about threats or challenges to it, and the proposed solutions can also change.

8.2.3 What makes it hard to build public support for evidence led more complex solutions? Why do unproductive understandings prevail across a culture?

The reality is complex. Both our in-built cognitive processes and our information environment can conspire to narrow our thinking about complex issues such as environmental health.

Daniel Kahneman has shown that our fast-thinking brains use many shortcuts to cope with the vast amount of information in the world and protect our existing beliefs (Kahneman 2013). We are designed to unconsciously process information and respond with emotion, and we often use logic to backfill our existing position.

The research is clear that, in our unconscious cognitive processing of information, we grasp the concrete and shy away from the abstract. This is an immense challenge for having a productive public conversation about complex social and environmental issues.

At the same time, we are overloaded by information, including a lot that is poor quality. The digital age has brought new, faster and more targeted ways for us to be exposed to unproductive explanations about complex systems issues.

The combination of these cognitive shortcuts and an overloaded, often misleading, information environment can reinforce dominant cultural narratives that are overly simple or simply wrong.

As experts who communicate on these complex issues, we also play our role. We assume that, if we fill people up with good information, they will understand and act accordingly. This is known as the *information deficit model* (Berentson-Shaw 2018). The evidence is clear that, outside of one-to-one deep dialogues and learning environments, filling up the information deficit is ineffective in deepening how people think.

Another common strategy for improving public understanding of complex issues is to tell compelling personal stories. While stories are important as a default for processing information, only some stories help us achieve our goals. Some stories simply reinforce unhelpful dominant narratives. Others fail to draw attention to the causes of a problem or the role of systems in creating or sustaining the problem. If our stories don't engage people in more productive understandings, we will fail to achieve the systems and structural shifts we need.

The good news is that research that draws on social and cognitive science and narrative traditions can guide us in putting knowledge and reason at the heart of people's thinking on complex issues like environmental health. Research on the importance of intrinsic values to human motivation can show us how to motivate people to support different, more effective approaches.

8.2.4 Developing effective strategies to improve public understanding of complex issues

To reframe communications and put quality knowledge and reason at the heart of people's thinking, we need an evidence-led strategy. There are three key components to this work.

First, we need to understand how people across society currently explain the problems we are concerned with. What chains of reasoning, language, frames, metaphors and values do they engage? Which of these ways of reasoning are the most dominant? By understanding and mapping this cognitive and cultural landscape and how it differs from or aligns to expert understandings of the issue, we can start to find pathways for more productive thinking and identify those pathways that will not take us where we wish to go (Quinn 2005).

Second, we need an evidence-informed communication strategy that helps people navigate from overly simple explanations to more complex and productive understandings, these are sometimes called *simplifying models* or strategies.

Evidence from across the social sciences tells us that advanced communications strategies involve several components including:

- engaging with the psychology of how people process information and misinformation (Lewandowsky *et al* 2012)

- understanding and working with people's values (Dietz 2013; Crompton 2010)
- understanding culturally shared frames and the causes and solutions they engage for people (Kendall-Taylor 2012)
- using language and effective metaphors strategically (Thibodeau *et al* 2017)
- presenting facts in a way that builds new mental models (Kendall-Taylor & Levitt 2017).

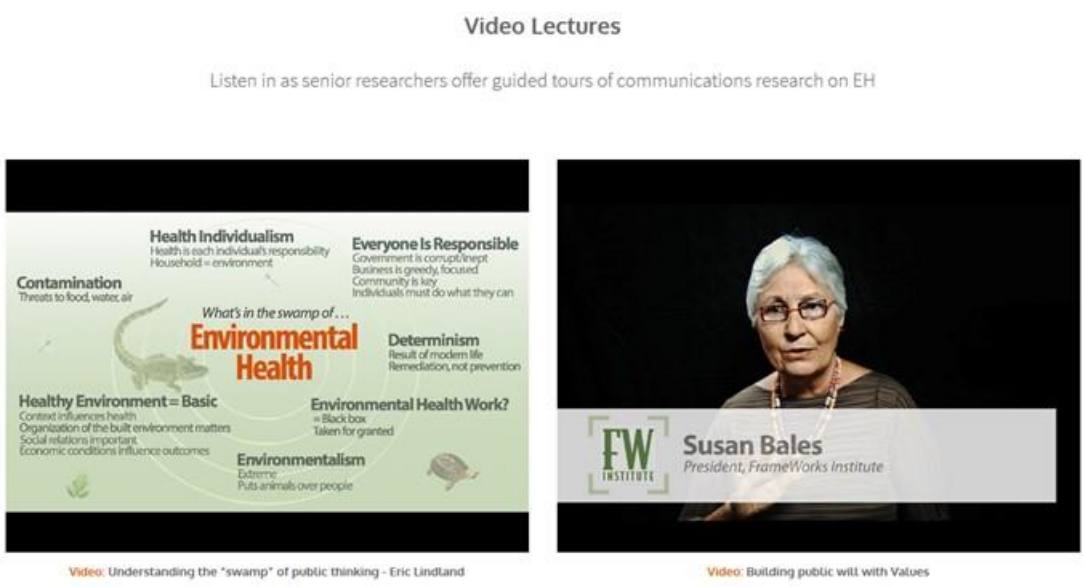
By combining these elements of the science of story into a new communications strategy, we can reframe the conversation and produce more productive ways of thinking.

Finally, we need to equip people across a field of practice with these tested strategies and tools so that everyone moves in the same direction. Advancing our communications on complex issues in our society means a change in our communication approaches across fields of practice.

8.2.5 What strategic communications exist in environmental health and air quality?

A number of tested strategic communications in the area of environmental health already exist. For example, the FrameWorks Institute has identified some values and metaphors proven to shift understanding in the United States public. The American Public Health Association / Centres for Disease Control and Prevention FrameWorks Institute's toolkit (FrameWorks 2014) helps environmental health professionals frame environmental health and related issues as important policy fields and matters of public concern (refer Figure 28).

Figure 28: Good practice example health message regarding air pollution



Source: FrameWorks Institute

Drawing on research from related areas of science, including climate change, can also guide our communications in New Zealand. A summary of this literature was completed by The Workshop in 2019 (The Workshop & Oxfam New Zealand 2019). There are also synergies with

the sustainable transport sector (and area where strategic communications are also being developed in New Zealand).

8.3 What we did in HAPINZ 3.0

Our approach comprised two key steps:

- a review of existing strategic frames and communications strategies
- development of a draft messaging guide.

8.3.1 Review of strategic frames and communication strategies

The core approach to developing strategic frames and communications strategies involved a non-systematic review of existing material. This review did not look at all tested communications techniques in air pollution and environmental health; rather we were guided by a theoretically and empirically driven framework of strategies. Specifically, we searched for research that:

- engaged with the psychology of how people process information (and misinformation)
- worked with people's values
- sought to locate and use culturally shared frames
- tested effective metaphors
- tested the presentation of facts in a way that builds new mental/cognitive models.

The broad aim of the literature review was:

- To map the landscape of current framing and communication strategies that are effective for helping people to think productively about air quality and environmental health.

We were interested in which framing strategies help:

- people understand why they should be concerned with how air pollution affects their health, and
- build public support for policies and practices that support healthy environments.

Search strategy

The review identified the available evidence on effective narrative strategies to talk about air quality and environmental health as it relates to air pollution and emissions. An initial list of keywords related to this specific topic was drawn up. These search term combinations are shown in Table 35 which follows. A scoping search was then conducted using these combinations on the PubMed, Google Scholar and Scopus databases. Potentially relevant articles were screened for mention of key search terms such as messages, narratives, framing, values and futures. Search combinations were discontinued after three consecutive pages with no relevant results. The initial set of references were cross-checked in Google Scholar to see if they contained or were cited by relevant references. The scoping review returned 110

possible articles which were then further evaluated for their direct relevance to the topic. These were narrowed to 40 that were directly relevant.

As well as the articles identified by the scoping review, relevant grey literature from other organisations such as The FrameWorks Institute, and other framing and narrative organisations that work on effective strategies for deepening people's thinking on complex issues, was included where it applied to air quality and environmental health.

Table 35: Messaging literature review initial search term combinations and results

	TOPIC	Air quality	Air pollution	Emissions	Environmental health
AND	Message/ing	3	6	4	4
	Frame/ing (analysis)	3	1	2	7
	Value(s)	1	0	1	5
	Vision(s)	3	3	2	0
	Communication(s)/strategy/ies	2	2	1	2
	Narrative(s)/cultural	1	3	0	4
	Metaphors	0	0	0	1
	Decision/making	5	6	2	3
	Future(s)	2	2	0	0
	COVID-19	2	5	0	0
			& emissions (2); mental health (1)		& social determinants (1); cultural models (1)

Review contents

There are relatively few peer-reviewed studies at the intersection of air quality, environmental health and narrative strategies that examine current framing strategies for helping people to think productively about air quality specifically. Much of the literature focuses on air quality measurement and monitoring, health risk assessments and environmental risk communications which, although related to the topic, was less useful for making conclusions about effective communication techniques to elicit helpful thinking in the public.

In keeping with The Workshop's evidence-led framework of effective narrative strategies, emphasising the importance of framing, the review drew from the available literature on air quality and emissions as well as the related climate change sphere.

We looked for examples of:

- appeals to intrinsic values
- universalist, that is, human rights framing of environmental health and climate change issues
- communications that encompass vision making and are solution-led rather than problem-led
- framing and messaging approaches that were part of successful change processes.

The full literature review is available as a separate report (Bell & Berentson-Shaw 2020).

8.3.2 Development of a draft messaging guide

Review recommendations

The literature review came up with the following five recommendations to inform the development of the messaging guide:

1. **Focus on your audience**
    - Understand who your audience is and tailor your communications specifically for them to be more effective.
    - Use two-way communications developed in collaboration with communities and those with health vulnerabilities that are most affected by air pollution and air quality issues.
    - Make sure you are telling your story – and giving essential information – to those who are open to persuasion.
    - A powerful form of communication is to show communities that something is being done about a problem through actions and policies. This also addresses perception gaps in your audience.
    - Make sure you are taking your audience's cultural beliefs and understandings into account when developing your communications.

2. **Lead with a vision for a better world**
    - First, ask communities what they want for air quality and their environmental health then make sure your communications align with their vision.
    - Focus on positive communications based on transformative visions for the future that motivate people to action. For example, frame your communications about environments that are calm and pleasant where people can move about freely and be in good health.
    - Provide social proof of systems change. For example, temporary reductions in emissions during COVID-19 lockdowns gave people a glimpse of an alternative future of better air quality. Social proof is useful if researchers need the public to take a specific action with policy makers.
    - There is also an aspect of experiential learning where people can be influenced to focus on the long-term future and make more sustainable decisions when they are exposed to more natural and green spaces.
    - Do use a frame of capability that encourages helpful thinking that we can work together to solve the problem as we have done together with other problems before.
    - Do not use frames of fatalism or normalcy bias that tap into unhelpful thinking about the problem of air pollution as a necessary consequence of progress, always going to exist and unable to be solved.

3. **Use intrinsic values to make it matter to your audience**

   - Lead with **intrinsic values**. Emphasise **fairness between places** – this equality value encourages helpful thinking about the collective responsibility and importance of everyone having environmental health conditions for good health and wellbeing. It helps people understand that working to improve environmental health in one place does not take away from others.
   - Use care for the environment as an intrinsic value.
   - Use values that will connect with people's emotions.
   - Avoid extrinsic values. Don't use security values or fear framing that fosters unhelpful thinking about individual's lack of self-efficacy and control over air quality. These values move people to seek simple solutions like denying the problem, not complex policy change solutions. For example, don't emphasise how emissions may impact people's material wellbeing as it reduces willingness to take action.
   - Avoid talking about individual responsibility for managing exposure to air pollution.

4. **Provide better explanations**

   - Use better explanations about the health impacts of air pollution. For example, talk about the long-lasting effects of air pollution on the brains of children and older adults, as well as the respiratory and cardiovascular problems it causes.
   - Present your research findings in a clear explanatory chain of cause, effect and solution, rather than just describing the problem. Start with people's visions/hopes/desires to remind them what they want for their air quality.
   - Avoid communicating about air pollution as invisible and instead talk about it as something concrete and physical.
   - Use helpful frames of health and wellbeing, a common good, and public health to motivate people.
   - Use motivational rather than sacrificial frames. Use specific agentive language to increase people's sense of competence. Name the agents responsible for helping make change so as to not individualise the action. For example, ''I can reduce my emissions if people in government also make changes to cities".
   - Avoid using 'loss' frames so as to be solutions-oriented in your story logic. For example, when discussing the costs associated with reducing emissions, rather than emphasising the loss of future income which is an individualistic frame, talk about a 'foregone gain' – a smaller increase in future income.
   - Use metaphors and tested visual images. For example, exposing people to nature scenes can help people be more future-oriented about decisions to improve air quality.

5. **Use diverse storytellers**

   - The messengers selected to deliver communications will be more effective if the target audience recognises them as part of their own group. They are better able to tap into the cultural understandings and mental models people use.
   - Use a wider variety of trustworthy messengers to speak on environmental health. This helps to make its importance and function clearer to the general public.

- Identify the communication channels commonly used to disseminate information in a community.
- Collaborate with communities on message design and delivery.
- Communicate about others' willingness to make policy changes. This is more effective in gaining support than providing negative information about the problem.

Messaging guide format

The review recommendations were used in the development of a draft easy to read and visually engaging guide on talking about air pollution science and policy. The target audience includes experts, communicators and advocates in the environmental health field with a focus on air quality.

Its purpose is to provide effective communication strategies to:

- help people designing policies and practices that improve air quality to have better conversations with the public
- improve people's understanding of the effects of air pollution on our health and wellbeing
- motivate people to act in support of policies and practices that build healthy environments.

The format was based on previous guides produced by The Workshop in collaboration with others – for example, *How to Talk About Climate Change. A Toolkit for Collection Action* (The Workshop & Oxfam New Zealand 2019) – and was structured around the core strategies of evidence-led communication, including:

- understanding your audience (including your noisy opposition)
- developing your own story
- the importance of avoiding myth busting
- ensuring you have a clear positive vision
- communicating with values
- using effective language
- including metaphors and concrete examples
- how to frame facts effectively
- considering your messages and media.

The draft messaging guide is available as a separate report (Berentson-Shaw & Bell 2020a) as well as a separate checklist for air quality communications (Berentson-Shaw & Bell 2020b).

8.3.3 Summary of our approach

We reviewed literature on existing strategic frames and communication strategies and drafted **an applied messaging guide.**

The draft guide is available as a separate document – *Draft short guide: How to talk about air quality and environmental health* (Berentson-Shaw & Bell 2020a) – and outlines evidence-based dos and don'ts for any individual or organisation wanting to communicate the study findings and air pollution, in general, in a more effective way.

The guide includes a checklist for communications about air quality and environmental health which is also available separately (Berentson-Shaw & Bell 2020b).

9. Summary of methodology

The key features of the approach we adopted are summarised in the following table:

Key features of the HAPINZ 3.0 update

Feature	Details
Base year	2016 for population
Spatial resolution	**Calculations** undertaken using 2013 census area unit boundaries **Results** aggregated by 16 regional councils, 20 district health boards, 67 territorial authorities and 89 airsheds
Population covered	100% of 2016 population
Pollutants	**Priority pollutants** • particulate matter ($PM_{2.5}$ and PM_{10}) • nitrogen dioxide (NO_2)
Exposure assessment	**$PM_{2.5}$ and PM_{10}**: ambient monitoring data typically averaged for 2015-2017 covering the majority of urban areas in New Zealand, with proxy monitoring used in unmonitored areas **NO_2**: modelling estimates from the NZ Transport Agency NVED exposure tool
Source attributions	**$PM_{2.5}$ and PM_{10}**: using source apportionment data and assigned to domestic fires, motor vehicles, industry, windblown dust, sea spray, and secondary PM **NO_2**: no source apportionment data available but assigned to motor vehicles (estimated to contribute approximately 90% of NO_2 exposure in urban areas)
Health endpoints	**Primary health impacts** • **mortality** and years of life lost (YLL) from long-term **$PM_{2.5}$** for all adults 30+ years, all ethnicities and Māori/Pacific peoples • **cardiac admissions** from long-term **$PM_{2.5}$** for all ages, all ethnicities • **respiratory admissions** from long-term **$PM_{2.5}$** for all ages, all ethnicities • **restricted activity days** from long-term **$PM_{2.5}$** for all ages, all ethnicities • **mortality** and YLL from long-term **NO_2** for all adults 30+ years, all ethnicities • **cardiac admissions** from long-term **NO_2** for all ages, all ethnicities • **respiratory admissions** from long-term **NO_2** for all ages, all ethnicities **Secondary health impacts (for comparison with HAPINZ 2.0)** • **mortality** from long-term **PM_{10}** for all adults 30+ years, all ethnicities and for Māori • **restricted activity days** from long-term **$PM_{2.5}$** for all ages, all ethnicities (also in primary health impacts) **Childhood asthma impacts relevant to NZ** • **asthma/wheeze hospitalisations** due to long-term **NO_2** for all 0-18 years • **asthma prevalence** due to long-term **NO_2** for all 0-18 years

Feature	Details
Social costs	**Valuation of mortality costs** • change in mortality multiplied by current NZ Value of a Statistical Life (**VoSL**) • change in total life years multiplied by a NZ Value of a Life Year (**VoLY**) **Valuation of morbidity costs** • cardiovascular and respiratory hospital admissions • restricted activity days • childhood asthma costs from GP visits, medication and hospitalisation **Development of a suite of NZ-specific damage costs** for consistent assessment of benefits to society in reducing harmful emissions and greenhouse gases
Key outputs	Combined **exposure/health effects model** enabling sensitivity/scenario testing and designed to be easily updateable together with a Users' Guide A **set of New Zealand-specific exposure-response functions** for assessing effects of air pollution on mortality and morbidity amongst New Zealanders A **detailed report**, suitable for a technical audience, outlining the methodology adopted and clearly stating all assumptions (Volume 2) A **summary report**, suitable for a more general audience, presenting the key findings and discussing their implications (Volume 1) A draft **messaging guide** to provide evidence-based dos and don'ts for anyone wanting to communicate the study findings through various channels together with a checklist

Note: For further details on the development of the methodology outlined in this report, please see:

Bell S & Berentson-Shaw J (2020). *Literature review: Framing air quality and environmental health*. Supplementary report for HAPINZ 3.0 prepared by The Workshop. Wellington, NZ, October 2020.

Davy PK & Trompetter WJ (2020). *An empirical model for attributing sources of particulate matter*. Supplementary report for HAPINZ 3.0 prepared by GNS Science, GNS Science consultancy report 2020/33, Institute of Geological and Nuclear Sciences, NZ, November 2020.

Kuschel G *et al* (2020). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Approved methodology*. Prepared by G Kuschel, J Metcalfe, P Davy, K Hastings, K Mason, T Denne, J Berentson-Shaw, S Hales, J Atkinson and A Woodward for Ministry for the Environment, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, 17 March 2020.

Sridhar S & Metcalfe J (2021). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Sources*. Excel model prepared by S Sridhar and J Metcalfe for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, February 2021.

Glossary

Term	Definition
acute	short-term duration but severe
airshed	a geographic area established to manage air pollution within the area as defined by the NESAQ
anthropogenic	generated by human activities, such as the combustion of fuels or processing of raw materials
As	arsenic
BaP	benzo(a)pyrene
BC	black carbon, both a harmful pollutant and a greenhouse gas
biomass burning	the burning of wood in domestic fires used for heating homes in winter – considered an anthropogenic source in HAPINZ 3.0
BMI	body mass index, a value derived from the mass and height of a person to indicate whether they are under or over weight
cardiac	of, pertaining to, or affecting the heart
cardiovascular	of, pertaining to, or affecting the heart and blood vessels
CAU	census area unit, a non-administrative geographic area normally with a population of 3,000–5,000 people in an urban area
CAU2006	census area unit based on the 2006 Census boundaries
CAU2013	census area unit based on the 2013 Census boundaries
CBA	cost-benefit analysis
CVD	cardiovascular disease
CVHA	cardiovascular hospital admission
chronic	long-term duration or constantly recurring
CO	carbon monoxide, a harmful pollutant
CO_2	carbon dioxide, a greenhouse gas
CO_2e	carbon dioxide equivalent, a way to express the impact of each different greenhouse gas in terms of the amount of CO_2 that would create the same amount of warming
coarse particulate	particles in the $PM_{2.5}$ to PM_{10} fraction
COMEAP	Committee on the Medical Effects of Air Pollutants
crustal material	particulate matter from construction, windblown dust etc. – considered an anthropogenic source in HAPINZ 3.0
CVD	cardiovascular disease
DALY	disability-adjusted life year is a measure of years in perfect health lost whereas QALYs are a measure of years lived in perfect health gained
DEFRA	Department of Environment, Food and Rural Affairs, UK
DMRB	Design Manual for Roads and Bridges

domestic fire	a solid-fuel heating appliance which is intended primarily to heat a residential dwelling
DTCC	Domestic Transport Costs and Charges
DW	disability weight
EC	elemental carbon
EEA	European Environment Agency
ELAPSE	Effects of Low-Level Air Pollution: A Study in Europe which is focussing particularly on the effects of BC
ERF	exposure-response function or relative risk function, the increase in risk for every increment in pollution
ERP	estimated resident population
ETS	Emissions Trading Scheme
extrinsic/individual values	values centered on external approval or individual rewards and losses, e.g. social power, money, or concern about image
fine particulate	particles in the $PM_{2.5}$ fraction
frames	frames are 'pre-packaged' mental models that help us to make sense of ideas and help to direct people where to look and interpret what they see
GBD	Global Burden of Disease study
GDP	gross domestic product
GIS	Geographic Information System, a computer system that analyzes and displays geographically referenced information.
HAPINZ	Health and Air Pollution in New Zealand
HAPINZ 1.0	the original HAPINZ study for 2001, undertaken by Fisher *et al* (2007)
HAPINZ 2.0	the first HAPINZ update for 2006, undertaken by Kuschel *et al* (2012a)
HAPINZ 3.0	the current HAPINZ update for 2016, undertaken by Kuschel *et al* (2022)
harmful pollutant	an air pollutant which causes adverse health effects
home heating emissions	emissions from solid-fuel heating appliances (also known as domestic fires) used to heat homes in winter. These are typically wood-fuelled (biomass burning).
IARC	International Agency for Research on Cancer
ICD-10 AM	International Classification of Diseases 10th revision – Australian Modification
IDI	The Stats NZ Integrated Data Infrastructure which has detailed data on national mortality rates and hospital admissions for up to the past 20 years
incidence	the proportion or rate of persons who *develop* a condition during a particular time period
intrinsic/collective values	values centered on internal or collective rewards and losses e.g. care for others or connection with nature
kaitiakitanga	in Māori culture, a kaitiaki is a guardian, and the process and practices of protecting and looking after the environment are referred to as kaitiakitanga
LAWA	Land Air Water Aotearoa, a website which presents environmental data collected across New Zealand
LEZ	low emission zone

marine aerosol	also known as sea spray– considered a natural source in HAPINZ 3.0
MAC	marginal abatement cost, the cost of reducing one more unit of pollution
MBCM	Monetised Benefits and Costs Manual, produced by Waka Kotahi
meshblock	the smallest geographic unit for which statistical data is collected and processed by Stats NZ. A meshblock is defined by a geographic area, which can vary in size from part of a city block to a large area of rural land. Meshblocks are added together to build up larger geographic areas such as census area units (CAU) and urban rural areas.
MfE	Ministry for the Environment
MoH	Ministry of Health
MoT	Te Manatū Waka Ministry of Transport
morbidity	ill health or suffering
mortality	death
motor vehicles	vehicles registered to travel on public roads, including cars, light commercial vehicles, trucks, buses and motorcycles – considered an anthropogenic source in HAPINZ 3.0
natural	generated by natural activities, such as sea spray, sand storms, vegetation, animals or volcanoes
NESAQ	National Environmental Standards for Air Quality
NH_3	ammonia
NO	nitric oxide
NO_2	nitrogen dioxide, a harmful pollutant
NO_X	oxides of nitrogen
NPV	net present value
NMDS	National Minimum Dataset, which holds data on date of birth, date of hospital admission and discharge, primary diagnosis (ICD-10AM code), ethnic groups, domicile code (CAU), and other useful information
NVED	National Vehicle Emissions Dataset
NZTA	Waka Kotahi NZ Transport Agency
NZU	New Zealand Unit (a tradable unit under the ETS)
O_3	Ozone, a harmful pollutant
OECD	Organisation for Economic Co-operation and Development
open burning	burning of biomass and waste in the outdoors – considered an anthropogenic source in HAPINZ 3.0
Pacific peoples	indigenous peoples from the Island nations in the South Pacific, and in its narrowest sense Pacific peoples in New Zealand
PAF	population attributable fraction, the estimated percentage of total health cases that are attributable to the air pollution exposure
Pb	lead
PM	particulate matter

$PM_{2.5}$	particulate matter less than 2.5 µm in diameter, sometimes referred to as fine particulate – also known as respirable particulate because it deposits deeper in the gas-exchange region including the respiratory bronchioles and alveoli
$PM_{2.5}$ / PM_{10} ratio	the fraction of $PM_{2.5}$ (by concentration or weight) in PM_{10}
PM_{10}	particulate matter less than 10 µm in diameter, includes fine particulate (less than 2.5 µm) and coarse particulate (2.5 to-10 µm) – also known as thoracic particulate because it deposits within the lung airways and the gas-exchange region, including the trachea, bronchi, and bronchioles
PPI	producer price index
prevalence	the proportion of a population who *have* a specific characteristic in a given time period
PV	present value
QALY	quality-adjusted life year is a generic measure of disease burden, including both the quality and the quantity of life lived. One QALY equates to one year in perfect health.
RAD	restricted activity day, a day on which people cannot do the things they might otherwise have done if air pollution was not present
RCP	Royal College of Physicians, UK
respiratory	of, pertaining to, or affecting the lungs and airways
REVIHAAP	Review of Evidence on Health Aspects of Air Pollution
RHA	respiratory hospital admission
SA1 and SA2	Statistical Area 1 and Statistical Area 2
SCC	social cost of carbon
secondary PM	particles created when gases react in the atmosphere
secondary sulphate	sulphate particles created when sulphur-containing gases react in the atmosphere, a subset of secondary PM - – considered a natural source in HAPINZ 3.0
SO_2	sulphur dioxide, a harmful pollutant
SOC	social opportunity cost of capital
solid fuel	coal and wood (including wood pellets)
Stats NZ	The public service department charged with the collection of statistics related to the economy, population and society of NZ
taonga	in Māori culture, a taonga is a treasured thing, whether tangible or intangible
TA	Territorial Authority, such as city or district council
UFP	ultrafine particles
µg	microgram, one millionth of a gram
$µg/m^3$	microgram per cubic metre, a unit of concentration
µm	micrometre, one millionth of a metre
UK	United Kingdom
US	United States of America

US EPA	United States Environmental Protection Agency
VEMT	Vehicle Emissions Mapping Tool
VEPM	Vehicle Emission Prediction Model
VFRR	value of fatality risk reduction
VoLY	value of a life year
VoSL	value of statistical life
VPF	value of preventing a fatality
VOCs	volatile organic compounds
VRMR	value of reduced mortality risk
VRR	value of reduced risk
Waka Kotahi	Waka Kotahi NZ Transport Agency
WHO	World Health Organization
wood burner	a domestic heating appliance that burns wood which is not an open fire, a multifuel heater, a pellet heater, a cooking stove or a coal burning heater
WTP	willingness to pay
WTS	willingness to swap
YLD	year lived with disability
YLL	year of life lost

References

Achakulwisut P *et al* (2019). Global, national, and urban burdens of paediatric asthma incidence attributable to ambient NO_2 pollution: estimates from global datasets. *The Lancet Planetary Health* **3**(4): e166-e178.

Abelson P (2008). Establishing a Monetary Value for Lives Saved: Issues and Controversies. *Working papers in cost-benefit analysis*. WP 2008-02. Office of Best Practice Regulation, Department of Finance and Deregulation.

Ackerman F & L Heinzerling (2002). Pricing the priceless: cost-benefit analysis of environmental protection. *University of Pennsylvania Law Review* **150**: 1553-1584.

Adler MD (2016). Benefit–Cost Analysis and Distributional Weights: An Overview. *Review of Environmental Economics and Policy* **10**(2): 264–285.

Adler MD *et al* (2019). Fair Innings? The Utilitarian and Prioritarian Value of Risk Reduction over a Whole Lifetime. *Duke Law School Public Law & Legal Theory Series* No. 2019-79.

AEA Technology Environment (2005). *Methodology for the Cost-Benefit Analysis for CAFE: Volume 2: Health Impact Assessment. Service Contract for Carrying out Cost-Benefit Analysis of Air Quality Related Issues, in particular in the Clean Air for Europe (CAFE) Programme*. Prepared for the European Commission, DG Environment. Oxon, UK.

Akehurst G *et al* (2019). *Air Quality Cost-Benefit Analysis – Update Review of the National Environmental Standards for Air Quality– Policy Options*. Prepared for the Ministry for the Environment by Market Economics.

ALA (1995). *Dollars and cents: The economic and health benefits of potential particulate matter reduction in the United States.* American Lung Association, 1740 Broadway, New York.

Alberini A *et al* (2004). Does the value of a statistical life vary with age and health status? Evidence from the US and Canada. *Journal of Environmental Economics and Management* **48**: 769–792.

Alberini A and Ščasný M (2013). Exploring heterogeneity in the value of a statistical life: Cause of death v. risk perceptions. *Ecological Economics* **94**: 143–155.

Aldy JE & Smyth SJ (2014). Heterogeneity in the value of life. *National Bureau of Economic Research Working paper* No 20206.

Aldy JE & Viscusi WK (2007). Age Differences in the Value of Statistical Life: Revealed Preference Evidence. *Review of Environmental Economics and Policy* **1**(2): 241–260.

Aldy JE & Viscusi WK (2008). Adjusting the Value of a Statistical Life for Age and Cohort Effects. *Review of Economics and Statistics* **90**(3):573-581

Amann *et al* (2017). *Costs, benefits and economic impacts of the EU Clean Air strategy and their implications on innovation and competitiveness*. Prepared for the European Commission, DG Environment.

Amato *et al* (2016). AIRUSE-LIFE+: a harmonized PM speciation and source apportionment in five southern European cities. *Atmospheric Chemistry and Physics* **16**: 3289–3309.

Anand S & Reddy SG (2019). *The Construction of the DALY: Implications and anomalies: Department of Economics Discussion Paper Series, Number 877.* Oxford: Department of Economics, University of Oxford.

Ancelet T *et al* (2010). A comparison of particulate and particle-phase PAH emissions from a modern wood burner with those of an old wood burner. *Air Quality and Climate Change* **44:** 21-24.

Ancelet T *et al* (2011). Carbonaceous aerosols in an urban tunnel. *Atmospheric Environment* **45**: 4463-4469.

Ancelet T *et al* (2011b). Characterisation of particulate matter emissions from a modern wood burner under varying burner conditions. *Air Quality and Climate Change* **45:** 21-27.

Andersson H *et al* (2019). Private and public willingness to pay for safety: A validity test. *Accident Analysis & Prevention* **123**: 170–175.

APHEIS (2004). *Health Impact Assessment of Air Pollution and Communication Strategy*, Third Year Report 2002 -2003. https://www.santepubliquefrance.fr/determinants-de-sante/pollution-et-sante/air/documents/rapport-synthese/apheis.-health-impact-assessment-of-air-pollution-and-communication-strategy.-third-year-report

Arrow KJ (1950). A Difficulty in the Concept of Social Welfare. *Journal of Political Economy* **58**(4): 328–46.

Asthma Foundation (2016). *Key Stats, Asthma & Respiratory Foundation NZ*. NZ. https://www.asthmafoundation.org.nz/research-stats/key-statistics

Åström S *et al* (2018). The costs and benefits of a nitrogen emission control area in the Baltic and North Seas. *Transportation Research Part D* **59**: 223–236.

Australian Institute of Health & Welfare (2020). *Australia's health snapshots 2020*. Canberra: AIHW.

Australian Safety & Compensation Council (2008). *The Health of Nations: the Value of a Statistical Life*. Commonwealth of Australia.

Austroads (2012). *Guide to Project Evaluation Part 4: Project Evaluation Data*. Austroads Publication No. AGPE04-12, Australia.

Barnes J *et al* (2019). Emissions vs Exposure: Increasing injustice from road traffic-related air pollution in the United Kingdom. *Transportation Research Part D: Transport and Environment* **73**: 56-66.

Barnett *et al* (2005). Air Pollution and Child Respiratory Health: A Case-Crossover Study in Australia and New Zealand. *American Journal of Respiratory and Critical Care Medicine* **171**(11): 1272–1278.

Beelen R *et al* (2015). Natural-cause mortality and long-term exposure to particle components: an analysis of 19 European cohorts within the multi-center ESCAPE project. *Environmental Health Perspectives* **123**(6): 525-33.

Bell S & Berentson-Shaw J (2020). *Literature review: Framing air quality and environmental health.* Supplementary report for HAPINZ 3.0 prepared by The Workshop. Wellington, NZ, October 2020

Berentson-Shaw J (2018). *A Matter of Fact*. BWB Text, Wellington, NZ.

Berentson-Shaw J & Bell S (2020a). *Draft short guide: How to talk about air quality and environmental health*. Report for HAPINZ 3.0 prepared by The Workshop. Wellington, NZ, November 2020.

Berentson-Shaw J & Bell S (2020b). *Checklist : How to talk about air quality and environmental health*. Report for HAPINZ 3.0 prepared by The Workshop, Wellington, NZ, November 2020.

Bertram MY *et al* (2016). *Cost–effectiveness thresholds: pros and cons*. Bulletin of the World Health Organization **94**: 925–930.

Bino M *et al* (2017). *Improved Methodologies for NO_2 Exposure Assessment in the EU*. Report no.: 2017/RMA/R/1250 prepared by VITO and Kings College London for the European Commission, DG-Environment. https://ec.europa.eu/environment/air/pdf/NO2%20exposure%20final%20report.pdf

Birchby D *et al* (2019). *Air quality damage cost update 2019*. Report prepared for the Department of Environment, Food and Rural Affairs by Ricardo Energy & Environment. Ref: AQ0650. Didcot, UK.

Bluett J *et al* (2016). *Impacts of exposure to dust on unsealed roads*. NZTA Research report 590 prepared by Golder Associates Ltd, NZ. August 2016. https://www.nzta.govt.nz/resources/research/reports/590/

Bognar G (2008). Age-weighting. *Economics and Philosophy* **24**: 167-189.

Bognar G (2015). Fair Innings. *Bioethics* **29**(4): 251-261.

Bowling A (2011). Do older and younger people differ in their reported well-being? A national survey of adults in Britain. *Family Practice 2011* **28**: 145–155.

Bowatte G *et al* (2015). The influence of childhood traffic-related air pollution exposure on asthma, allergy and sensitization: a systematic review and a meta-analysis of birth cohort studies. *Allergy* **70**(3): 245-56.

Brines M *et al* (2015). Traffic and nucleation events as main sources of ultrafine particles in high-insolation developed world cities. *Atmospheric Chemistry and Physics* **15**: 5929–5945.

Brook R *et al* (2010). Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement from the American Heart Association. *Journal of the American Heart Association. Circulation 2010* **121**: 2331-2378.

Brunekreef B *et al* (2007). The Brave New World of Lives Sacrificed and Saved, Deaths Attributed and Avoided. *Epidemiology* **18**(6): 785–788.

Brunekreef B *et al* (2021). *Mortality and Morbidity Effects of Long-Term Exposure to Low-Level $PM_{2.5}$, BC, NO_2, and O_3: An Analysis of European Cohorts in the ELAPSE Project*. HEI Research Report 208, Health Effects Institute, Boston, MA, United States, September. https://www.healtheffects.org/publication/mortality-and-morbidity-effects-long-term-exposure-low-level-pm25-bc-no2-and-o3-analysis

Burte E *et al* (2016). Susceptibility factors relevant for the association between long-term air pollution exposure and incident asthma. *Current Environmental Health Reports* **3**(1): 23-39.

Cameron TA (2010). Euthanizing the value of a statistical life. *Review of Environmental Economics and Policy* **4**(2): 161-178.

Cameron TA *et al* (2010). Demand for health risk reductions: A cross-national comparison between the US and Canada. *Journal of Risk and Uncertainty* **41**:245–273.

Cameron T & Ostro B (2004). *Advisory Council on Clean Air Compliance Analysis Response to Agency Request on Cessation Lag*. Letter to MO Leavitt, US EPA. EPA-COUNCIL-LTR-05-001. 6 December 2004.

Carswell AC et al (2015). *Costs of childhood hospitalisation for asthma*. Department of Public Health, University of Otago, Wellington, NZ.

Chen J. & Hoek G (2020). Long-term exposure to PM and all-cause and cause-specific mortality: A systematic review and meta-analysis. *Environment International* **143**: 105974.

Chilton S *et al* (2004). *Valuation of health benefits associated with reductions in air pollution.* Final Report. Prepared for the Department of Environment, Food & Rural Affairs, UK.

Chilton S *et al* (2020). *A scoping study on the valuation of risks to life and health: the monetary value of a life year (VOLY)* Final report. UK Health & Safety Executive.

Clough P *et al* (2018). A New Approach to Environmental Valuation for New Zealand. *Policy Quarterly* **14**(2): 50-57.

COMEAP (2010). *The mortality effects of long-term exposure to particulate air pollution in the United Kingdom*. A report by the Health Protection Agency for the Committee on the Medical Effects of Air Pollutants, UK.

COMEAP (2015). *Statement on the evidence for the effects of nitrogen dioxide on health*. Prepared by the Committee on the Medical Effects of Air Pollutants, UK, 12 March 2015. https://www.gov.uk/government/publications/nitrogen-dioxide-health-effects-of-exposure

COMEAP (2018). *Associations of long term average concentrations of nitrogen dioxide with mortality.* Prepared by the Committee on the Medical Effects of Air Pollutants, UK, August 2018. https://www.gov.uk/government/publications/nitrogen-dioxide-effects-on-mortality/associations-of-long-term-average-concentrations-of-nitrogen-dioxide-with-mortality-2018-comeap-summary

Correia AW *et al* (2013). Effect of Air Pollution Control on Life Expectancy in the United States. *Epidemiology* **24**(1): 23–31.

Crompton T (2010). *Common cause: The case for working with our cultural values*. The Common Cause Foundation, UK.

Cropper ML *et al* (1994). Preferences for Life Saving Programs: How the Public Discounts Time and Age. *Journal of Risk and Uncertainty* **8**:243-265.

DANIDA (2000). *Who Suffers? Identifying the Vulnerable Groups*. Paper presented at the DANIDA Workshop Papers: Improving the Urban Environment and Reducing Poverty, Copenhagen, Denmark, 5 December 2000. http://web.mit.edu/urbanupgrading/urbanenvironment/issues/vulnerable-groups.html

Davy PK *et al* (2009). *Elemental analysis of wood burner emissions*. GNS Science Client Report 2009/258, Institute of Geological and Nuclear Sciences, NZ.

Davy PK *et al* (2011). *Concentration, composition and sources of particulate matter in the Johnstone Hills Tunnel, Auckland*. GNS Science Client Report 2010/296, Institute of Geological and Nuclear Sciences, NZ.

Davy PK *et al* (2017). *Source apprtionment and trend analysis of air particulate matter in the Auckland region*. GNS Science Client Report 2014/194, Institute of Geological and Nuclear Sciences, NZ.

Davy PK & Trompetter WJ (2017). *Apportionment of PM_{10} sources in the Tokoroa airshed, Waikato region*. GNS Science consultancy report. Institute of Geological and Nuclear Sciences, NZ.

Davy PK & Trompetter WJ (2018). *Heavy metals, black carbon and natural sources of particulate matter in New Zealand.* GNS Science consultancy report 2017/238, Institute of Geological and Nuclear Sciences, NZ.

Davy PK & Trompetter WJ (2020). *An empirical model for attributing sources of particulate matter.* Supplementary report prepared for HAPINZ 3.0 by GNS Science, GNS Science consultancy report 2020/33, Institute of Geological and Nuclear Sciences, NZ, November 2020.

DEFRA *et al* (2007). *An Economic Analysis to inform the Air Quality Strategy*. Updated Third Report of the Interdepartmental Group on Costs and Benefits. Department of Environment, Food & Rural Affairs in partnership with the Scottish Executive, Welsh Assembly Government and Department of Environment Northern Ireland.

Degobbi C, Saldiva PHN & Rogers C (2010). Endotoxin as modifier of particulate matter toxicity: a review of the literature. *Aerobiologia* 27:97-105

Denne T & Atkins S (2015). *Policies to reduce harmful emissions from vehicles: Costs and benefits*. Final Report prepared for Ministry of Transport by Covec Ltd. https://www.transport.govt.nz//assets/Uploads/Report/Policies-to-Reduce-Harmful-Emissions-from-Vehicles-Costs-and-Benefits-May-2015.pdf

Department for Business, Energy & Industrial Strategy (2018). *Updated short-term traded carbon values*. Used for public policy appraisal, UK.

Department for Business, Energy & Industrial Strategy (2019). *Valuation of Energy Use and Greenhouse Gas*. Supplementary guidance to the HM Treasury Green Book on Appraisal and Evaluation in Central Government, UK.

Desaiguesa B *et al* (2011). Economic valuation of air pollution mortality: A 9-country contingent valuation survey of value of a life year (VoLY). *Ecological Indicators* **11**: 902–910.

Dietz T (2013). Bringing values and deliberation to science communication. *Proceedings of the National Academy of Sciences* **110**. Supplement 3: 14081-14087.

Dockins C *et al* (2018). *What's in a Name? A Systematic Search for Alternatives to "VSL".* Resources for the Future Working Paper 18-14. Washington, DC.

Dolan P *et al* (2005). QALY maximisation and people's preferences: a methodological review of the literature. *Health Economics* **14**: 197–208.

Dolan P *et al* (2008). Valuing lives and life years: anomalies, implications, and an alternative. *Health Economics, Policy and Law* **3**: 277–300

Dolan P & Tsuchiya A (2012). It is the lifetime that matters: public preferences over maximising health and reducing inequalities in health. *Journal of Medical Ethics* **38**: 571-573.

Donovan G *et al* (2018). Vegetation diversity protects against childhood asthma: results from a large New Zealand birth cohort. *Nature Plants* 2018. **4**: 358-364.

EEA (2014). *Health Impacts of Air Pollution*. European Environment Agency, EEA infographic INF-12-en. https://www.eea.europa.eu/signals/signals-2013/infographics/health-impacts-of-air-pollution/view

EEA (2018). *Air Quality in Europe – 2018 report*. European Environment Agency, EEA report number 12/2018. https://www.eea.europa.eu/publications/air-quality-in-europe-2018

EFCA (2019). *Ambient ultrafine particles: evidence for policy makers*. White paper prepared by the 'Thinking outside the box' team, European Federation of Clean Air and Environmental Protection Associations. Version 1, 25 October 2019. https://efca.net/files/WHITE%20PAPER-UFP%20evidence%20for%20policy%20makers%20(25%20OCT).pdf

EHINZ (2021). *Ethnic profile*. Webpage prepared by Environmental Health Intelligence New Zealand, Wellington, NZ. Accessed August 2021. https://www.ehinz.ac.nz/indicators/population-vulnerability/ethnic-profile/

European Commission (2001). *Recommended Interim Values for the Value of Preventing a Fatality in DG Environment Cost Benefit Analysis*. https://ec.europa.eu/environment/enveco/others/pdf/recommended_interim_values.pdf

Falcon-Rodriguez CI *et al* (2016). Aeroparticles, composition, and lung diseases. *Frontiers in Immunology*, 7(3).

Faustini A *et al* (2014). Nitrogen dioxide and mortality: review and meta-analysis of long-term studies. *European Respiratory Journal* **44**(3): 744-753.

Fisher *et al* (2007). *Health and Air Pollution in New Zealand: Main Report*. Research report prepared by G Fisher, T Kjellstrom, S Kingham, S Hales, R Shrestha and others for Health Research Council of New Zealand, Ministry for the Environment, and Ministry of Transport, Auckland, NZ.

Forastiere F & Peters A (2021). The NO_2 and Mortality Dilemma Solved? Almost There! *Environmental Health Perspectives* **129**(12): Invited Perspective, 28 December. https://ehp.niehs.nih.gov/doi/10.1289/EHP10286

FrameWorks (2014). *Environmental health toolkit: A brief intro to strategic frame analysis*. FrameWorks Institute, Washington DC. https://www.frameworksinstitute.org/publication/environmental-health-toolkit-a-brief-intro-to-strategic-frame-analysis/

GBD (2017). Global, regional, and national age-sex specific mortality for 264 causes of death, 1980–2016: a systematic analysis for the Global Burden of Disease Study 2016. Prepared by the Global Burden of Disease 2016 Collaborators. *The Lancet* **390** (10100): 1151 – 1210

Glover D & Henderson J (2010). *Quantifying health impacts of government policies. A how-to guide to quantifying the health impacts of government policies*. Department of Health, UK.

González RM *et al* (2018). Estimating the value of risk reductions for car drivers when pedestrians are involved: a case study in Spain. *Transportation* **45**(2): 499–521.

Guarnieri M & Balmes JR (2014). Outdoor air pollution and asthma. *The Lancet* **383**(9928): 1581-1592.

Gyrd-Hansen D (2005). Willingness to Pay for a QALY: Theoretical and Methodological Issues. *Pharmacoeconomics* **23**(5): 423-432.

Hales S *et al* (2012). Air pollution and mortality in New Zealand: cohort study. *Journal of Epidemiology and Community Health* **66**:468-473, May. https://doi.org/10.1136/jech.2010.112490

Hales S *et al* (2021). Long term exposure to air pollution, mortality and morbidity in New Zealand: Cohort study. *Science of Total Environment* **801** (2021) 149660, August. https://doi.org/10.1016/j.scitotenv.2021.149660

Hammitt JK (2013). Admissible utility functions for health, longevity, and wealth: integrating monetary and life year measures. *Journal of Risk and Uncertainty* **47**(3): 311–325.

Harris J (1985). *The Value of Life. An introduction to medical ethics*. Routledge.

Health Canada (2016). *Human Health Risk Assessment for Ambient Nitrogen Dioxide*. Water and Air Quality Bureau, Health Canada.

Hehua Z *et al* (2017). The impact of prenatal exposure to air pollution on childhood wheezing and asthma: A systematic review. *Environmental Research* **159**: 519-530.

Hensher DA *et al* (2009). Estimating the willingness to pay and value of risk reduction for car occupants in the road environment. *Transportation Research Part A* **43**(7): 692–707.

Hensher DA *et al* (2011). Estimating the Value of Risk Reduction for Pedestrians in the Road Environment: An Exploratory Analysis. *Journal of Choice Modelling* **4**(2): 70-94.

Héroux ME *et al* (2015). Quantifying the health impacts of ambient air pollutants: recommendations of a WHO/Europe project. *International Journal of Public Health* **60**(5): 619-627.

HM Treasury (2018). *The Green Book. Central Government Guidance on Appraisal and Evaluation.* UK.

Hoek G (2017). Methods for assessing long-term exposures to outdoor air pollutants. *Current Environmental Health Reports* **4**: 450–462, October. https://doi.org/10.1007/s40572-017-0169-5

Hoek G *et al* (2013). Long-term air pollution exposure and cardio- respiratory mortality: a review. *Environmental Health* **12**(1): 43.

Hohmeyer O (1998). *The social costs of energy consumption*. Springer Verlag. Berlin.

Holland M (2019). Cost-Benefit Analysis and Air Quality Related Health Impacts: A European Perspective. *Encyclopedia of Environmental Health*, 2nd edition, Volume 1.

Holland M *et al* (2005). *Methodology for the Cost-Benefit Analysis for CAFE: Volume 1: Overview of Methodology*. AEAT/ED51014/Methodology Paper Issue 4.

Holland MR *et al* (2013). *The ALPHA Benefit Assessment Model.* EC4MACS Modelling Methodology. European Consortium for Modelling of Air Pollution and Climate Strategies - EC4MACS.

Holt S & Beasley R (2002). *The burden of asthma in New Zealand*. Asthma and Respiratory Foundation of New Zealand, Wellington, NZ.

Horowitz JK & Carson RT (1990). Discounting Statistical Lives. *Journal of Risk and Uncertainty* **3**: 403-413.

Howard RA (1984). On Fates Comparable to Death. *Management Science* **30**(4):407-422.

HQSC (2016). *Atlas of Healthcare Variation: Asthma*. Health Quality & Safety Commission, NZ. http://www.hqsc.govt.nz/our-programmes/health-quality-evaluation/projects/atlas-of-healthcare-variation/asthma/

Huang H *et al* (2018). Investigation of association between environmental and socioeconomic factors and preterm birth in California. *Environment International* **121**: 1066-1078.

Huangfu P & Atkinson R (2020). Long-term exposure to NO_2 and O_3 and all-cause and respiratory mortality: A systematic review and meta-analysis. *Environment International* **144**: 105998.

IARC (2012). *Diesel engine exhaust carcinogenic*. Press release No 213, International Agency for Research on Cancer, WHO, Lyon, France, 12 June 2012. https://www.iarc.who.int/pressrelease/#year:2012

IARC (2013). *Outdoor air pollution a leading environmental cause of cancer deaths*. Press release No 221, International Agency for Research on Cancer, WHO, Lyon, France, 17 October 2013. https://www.iarc.who.int/pressrelease/#year:2013

Industrial Economics (2006). *Expanded expert judgment assessment of the concentration-response relationship between $PM_{2.5}$ exposure and mortality*. Prepared for Office of Air Quality Planning and Standards, US Environmental Protection Agency.

Iragüen P & de D Ortuzar J (2004). Willingness-to-pay for reducing fatal accident risk in urban areas: an internet-based Web page stated preference survey. *Accident Analysis and Prevention* **36**: 513-524.

Jacobs (2016). *Local Authority Vehicle Emission GIS Mapping, Prototype Development for Four Local Authorities.* Prepared for Waka Kotahi NZ Transport Agency, 26 September. https://www.nzta.govt.nz/roads-and-rail/highways-information-portal/technical-disciplines/air-quality-climate/planning-and-assessment/vehicle-emissions-mapping-tool/

Jacobs (2018). *National Vehicle Emission GIS Mapping - National Vehicle Emission Dataset 2016*. Prepared for Waka Kotahi NZ Transport Agency, 7 September. https://www.nzta.govt.nz/roads-and-rail/highways-information-portal/technical-disciplines/air-quality-climate/planning-and-assessment/vehicle-emissions-mapping-tool/

Jakob A *et al* (2006). Transport Cost analysis: a case study of the total costs of private and public transport in Auckland. *Environmental Science and Policy* **9**(1): 55-66.

Johannesson M & Johansson PO (1997). Quality of life and the WTP for an increased life expectancy at an advanced age. *Journal of Public Economics* **65**: 219-228.

Johansson PO (2002). On the definition and age-dependency of the value of a statistical life. *Journal of Risk and Uncertainty* **25**(3): 251–63.

Jones-Lee MW *et al* (1985). The Value of Safety: Results of a National Sample Survey. *The Economic Journal* **95**(377): 49-72.

Jones-Lee M *et al* (2015). Valuing Gains in Life Expectancy: Clarifying Some Ambiguities. *Journal of Risk and Uncertainty* **51**: 1–21.

Kahneman D (2013). *Thinking Fast and Slow*. New York, NY. Farrar, Straus and Giroux.

Kendall-Taylor N (2012). Conflicting models of mind: Mapping the gaps between expert and public understandings of child mental health. *Science Communication* **34**: 695–726.

Kendall-Taylor N & Levitt P (2017). Beyond hat in hand: Science advocacy is foundational for policy decisions. *Neuron* **94**(4): 708-712.

Khreis H & Nieuwenhuijsen MJ (2017). Traffic-related air pollution and childhood asthma: Recent advances and remaining gaps in the exposure assessment methods. *International Journal of Environmental Research and Public Health* **14**(3).

Khreis H *et al* (2017). Exposure to traffic-related air pollution and risk of development of childhood asthma: A systematic review and meta-analysis. *Environment International* **100**: 1-31.

King's College (2015). *Understanding the Health Impacts of Air Pollution in London*. Report prepared for Transport for London, July 2015. https://www.london.gov.uk/WHAT-WE-DO/environment/environment-publications/understanding-health-impacts-air-pollution-london

Kirrane EF *et al* (2019). A systematic review of cardiovascular responses associated with ambient black carbon and fine particulate matter. *Environment International* **127**: 305-316.

Klompmaker JO *et al* (2021). Comparison of associations between mortality and air pollution exposure estimated with a hybrid, a land-use regression and a dispersion model. *Environment International* **146**: 106306.

Kloog I *et al* (2012). Acute and Chronic Effects of Particles on Hospital Admissions in New-England. *PLOS One* **7**(4): e34664.

Kolasa-Więcek A & Suszanowicz D (2019). Air pollution in European countries and life expectancy—modelling with the use of neural network. *Air Quality, Atmosphere & Health* **12**: 1335–1345.

Krupnick A (2007). Mortality-risk Valuation and Age: Stated Preference Evidence. *Review of Environmental Economics and Policy* **1**(2): 261–282. https://doi.org/10.1093/reep/rem016

Künzli N *et al* (2000). Public-health impact of outdoor and traffic-related air pollution: a European assessment. *The Lancet* **356**: 795-801.

Kuschel G *et al* (2012a). *Updated Health and Air Pollution in New Zealand Study*. Various reports and models prepared by G Kuschel, J Metcalfe, E Wilton, J Guria, S Hales, K Rolfe and A Woodward for Health Research Council of New Zealand, Ministry for the Environment, Ministry of Transport and New Zealand Transport Agency, March 2012.

Kuschel G *et al* (2012b). *Updated Health and Air Pollution in New Zealand Study Volume 1: Summary Report*. Prepared by G Kuschel, J Metcalfe, E Wilton, J Guria, S Hales, K Rolfe and A Woodward for Health Research Council of New Zealand, Ministry for the Environment, Ministry of Transport and New Zealand Transport Agency, March 2012.

Kuschel G *et al* (2012c). *Updated Health and Air Pollution in New Zealand Study Volume 2: Technical Reports*. Prepared by G Kuschel, J Metcalfe, E Wilton, J Guria, S Hales, K Rolfe and A Woodward for Health Research Council of New Zealand, Ministry for the Environment, Ministry of Transport and New Zealand Transport Agency, March 2012.

Kuschel G *et al* (2017). Evaluating Bus Emissions: What colour, how big and how much is that elephant in the window? Paper by G Kuschel, J Metcalfe and A Cooper, *Australasian Transport Research Forum 2017 Proceedings*, 27 – 29 November 2017, Auckland, NZ.

Kuschel G *et al* (2020). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Approved methodology*. Prepared by G Kuschel, J Metcalfe, P Davy, K Hastings, K Mason, T Denne, J Berentson-Shaw, S Hales, J Atkinson and A Woodward for Ministry for the Environment, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, 17 March 2020.

Kuschel G *et al* (2021). *Domestic Transport Costs and Charges: Working Paper D4 Air quality and greenhouse gas emissions*. Report prepared by G Kuschel, S Sridhar and J Metcalfe for Te Manatū Waka Ministry of Transport, *publication pending*.

Kuschel G *et al* (2022). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Volume 1 – Findings and implications*. Report prepared by G Kuschel, J Metcalfe, S Sridhar, P Davy, K Hastings, K Mason, T Denne, J Berentson-Shaw, S Bell, S Hales, J Atkinson and A Woodward for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, March 2022.

Lacasaña M *et al* (2005). Exposure to ambient air pollution and prenatal and early childhood health effects. *European Journal of Epidemiology* **20**: 183-189.

Laughland D *et al* (2007). *Exploring the Role of Cost-Benefit Analysis in Government Regulations*. Research Report, AARP Public Policy Institute.

Laurent O *et al* (2016). A statewide nested case control study of preterm birth and air pollution by source and composition. *Environmental Health Perspectives* **124**(9): 1479-1486.

Lepeule J *et al* (2012). Chronic exposure to fine particles and mortality: an extended follow-up of the Harvard Six Cities Study from 1974 to 2009. *Environmental Health Perspectives* **120**(7): 965-97.

Lewandowsky S *et al* (2012). Misinformation and its correction: Continued influence and successful debiasing. *Psychological Science in the Public Interest* **13**(3): 106-131.

Lewis PA & Charny M (1989). Which of two individuals do you treat when only their ages are different and you can't treat both? *Journal of Medical Ethics* **15**: 28-32.

Lindland EH & Kendall-Taylor N (2011). *People, Polar Bears, and the Potato Salad: Mapping the Gaps Between Expert and Public Understandings of Environmental Health*. A FrameWorks Research Report, FrameWorks Institute, Washington, DC. https://www.frameworksinstitute.org/publication/people-polar-bears-and-the-potato-salad-mapping-the-gaps-between-expert-and-public-understandings-of-environmental-health/

Liu S *et al* (2020). Long-term exposure to low-level air pollution and incidence of asthma: the ELAPSE project. *European Respiratory Journal*: 2003099.

Longley I & Sommervell E (2019). *Review of NO_2/NO_X empirical conversion equations*. Report prepared by NIWA.

Longley I (2020). *NIWA Traffic Impact Model v3.1*. Memo prepared by NIWA for PDP, 29 June.

Marglin SA (1963). The Social Rate of Discount and Optimal Rate of Saving. *Quarterly Journal of Economics* **77**(1): 95-111.

Mason H *et al* (2009). Modelling the monetary value of a QALY: a new approach based on UK data. *Health Economics* **18**(8): 933-950.

Metcalfe J & Sridhar S (2018). *National air emissions inventory 2015*. Report prepared by Emission Impossible Ltd for Ministry for the Environment, NZ, October 2018. https://environment.govt.nz/publications/national-air-emissions-inventory/

MfE (2004). *Proposed National Environmental Standards for Air Quality: Resource Management Act S32: Analysis of the Costs and Benefits.* Ministry for the Environment, NZ. https://environment.govt.nz/publications/proposed-national-environmental-standards-for-air-quality-resource-management-act-section-32-analysis-of-the-costs-and-benefits/

MfE (2007). *Environment New Zealand 2007.* Report ME847, Ministry for the Environment, NZ. https://environment.govt.nz/publications/environment-new-zealand-2007/

MfE (2011a). *Clean healthy air for all New Zealanders: The National Air Quality Compliance Strategy to meet the PM_{10} standard*. Prepared by Emission Impossible Ltd for the Ministry for the Environment, NZ, August 2011. https://environment.govt.nz/assets/Publications/Files/air-quality-compliance-strategy.pdf

MfE (2011b). *Resource Management (National Environmental Standards for Air Quality) Regulations 2004 plus 2011 Amendment Regulations*. NZ Government, NZ, June 2011. https://www.legislation.govt.nz/regulation/public/2004/0309/latest/DLM286835.html

MfE & Stats NZ (2018). *New Zealand's Environmental Reporting Series: Our air 2018*. Ministry for the Environment and Stats NZ, Wellington, NZ, October 2018. https://www.stats.govt.nz/information-releases/new-zealands-environmental-reporting-series-our-air-2018

Miller B *et al* (2011). *Health impact assessment for the National Emissions Ceiling Directive (NECD) – Methodological Issues*. IOM Research Report TM/11/03.

Miller T & Guria J (1991). *The Value of Statistical Life in New Zealand*. Market Research on Road Safety. Land Transport Division, Ministry of Transport, Wellington.

Morgan G *et al* (2017). Rethinking the Social Cost of Carbon Dioxide. *Issues in Science and Technology* **33**(4): 1-12.

MoH (2020a). *Annual Data Explorer 2019/20: New Zealand Health Survey.* Data file, Ministry of Health, Wellington, NZ. https://minhealthnz.shinyapps.io/nz-health-survey-2019-20-annual-data-explorer/

MoH (2020b). *Longer, Healthier Lives: New Zealand's Health 1990–2017. A report on the health loss estimates of the 2017 Global Burden of Disease Study.* Ministry of Health, Wellington, NZ.

MoH (2021a). *Mortality Collection.* Data file, Ministry of Health, Wellington, NZ. https://www.health.govt.nz/nz-health-statistics/national-collections-and-surveys/collections/mortality-collection

MoH (2021b). *National Minimum Dataset (Hospital Events).* Data file, Ministry of Health, Wellington, NZ. https://www.health.govt.nz/nz-health-statistics/national-collections-and-surveys/collections/national-minimum-dataset-hospital-events

MoT (1996). *Land transport pricing study: Environmental externalities*. Discussion Paper. Ministry of Transport, Wellington, NZ.

MoT (2010). *The Social Cost of Road Crashes and Injuries June 2010 update.* Ministry of Transport, Wellington, NZ.

MoT (2020). *Social cost of road crashes and injuries - June 2019 update*. Te Manatū Waka Ministry of Transport, Wellington, NZ.

Muñoz X *et al* (2019). Diesel exhaust particles: Their role in increasing the incidence of asthma. Reviewing the evidence of a causal link. *Science of the Total Environment* **652**: 1129-1138.

National Health Committee (2013). *Strategic overview. Cardiovascular Disease in New Zealand*.

Nielsen JS *et al* (2010). How would you like your gain in life expectancy to be provided? An experimental approach. *Journal of Risk and Uncertainty* **41**(3):195-218.

Nord E *et al* (1996). The Significance of Age and Duration of Effect in Social Evaluation of Health Care. *Health Care Analysis* **4**(2): 103–111.

Nordhaus WD (2016). Revisiting the social cost of carbon. *Proceedings of the National Academy of Science* **114**(7): 1518–1523.

NZIER (2009). *The value of air quality standards. Review and update of cost benefit analysis of National Environmental Standards on air quality*. Report prepared by the NZ Institute of Economic Research for the Ministry for the Environment.

NZTA (2020). *Ambient air quality (nitrogen dioxide) monitoring network - Annual report 2007-19*. Prepared by Tonkin & Taylor for Waka Kotahi NZ Transport Agency, Wellington, NZ, August 2020. https://www.nzta.govt.nz/resources/air-quality-monitoring/

NZTA (2021). *Monetised benefits and costs manual. Version 1.4*. Waka Kotahi NZ Transport Agency, February 2021. https://www.nzta.govt.nz/resources/monetised-benefits-and-costs-manual/

NZ Treasury (2015). *Guide to Social Cost Benefit Analysis*. NZ Treasury, Wellington, NZ.

NZ Treasury (2020). *CBAx Tool User Guidance. Guide for departments and agencies using Treasury's CBAx tool for cost benefit analysis*. NZ Treasury, Wellington, NZ, 11 December 2020.

NZ Treasury (2021). *CBAx Tool User Guidance. Guide for departments and agencies using Treasury's CBAx tool for cost benefit analysis*. NZ Treasury, Wellington, NZ, September 2021. https://www.treasury.govt.nz/publications/guide/cbax-tool-user-guidance

O'Brien J (2018). Age, Autos, and the Value of a Statistical Life. *Journal of Risk and Uncertainty* **57**: 51–79.

O'Dea D & Wren J (2012). *New Zealand Estimates of the Total Social and Economic Cost of Injuries. For All Injuries, and the Six Injury priority areas. For Each of Years 2007 to 2010. In June 2010 dollars*. Report to New Zealand Injury Prevention Strategy, Wellington, NZ.

OECD (2010). *Valuing lives saved from environmental, transport and health policies: a meta-analysis of stated preference studies.* OECD report ENV/EPOC/WPNEP(2008)10/FINAL, Organisation for Economic Co-operation and Development.

OECD (2012). *Mortality Risk Valuation in Environment, Health and Transport Policies*. Organisation for Economic Co-operation and Development, OECD Publishing, Paris.

OECD (2016). *The Economic Consequences of Outdoor Air Pollution*. Organisation for Economic Co-operation and Development, OECD Publishing, Paris.

OECD (2019). *Health at a Glance 2019*. Organisation for Economic Co-operation and Development, November 2019. https://www.oecd-ilibrary.org/social-issues-migration-health/asthma-and-copd-hospital-admission-in-adults-2017-or-nearest-year_e1a68f0a-en

Orellano P *et al* (2017). Effect of outdoor air pollution on asthma exacerbations in children and adults: Systematic review and multilevel meta-analysis. *PLOS One* **12**(3): e0174050.

Ostro B (1987). Air pollution and morbidity revisited: A specification test. *J Environ Econ Manage* **14**, 87-98.

Ostro B *et al* (2015). Associations of mortality with long-term exposures to fine and ultrafine particles, species and sources: Results from the California Teachers Study Cohort. *Environmental Health Perspectives* **123**(6): 549-556.

Ostro B *et al* (2018). Assessing the recent estimates of the global burden of disease for ambient air pollution: Methodological changes and implications for low- and middle-income countries. *Environmental Research* **116**: 713 – 725. https://sci-hub.tw/10.1016/j.envres.2018.03.001

Papadogeorgou G *et al* (2019). Low Levels of Air Pollution and Health: Effect Estimates, Methodological Challenges, and Future Directions. *Current environmental health reports* **6**(3): 105-115.

Pearce DW (2000). *Valuing Risks to Life and Health. Towards Consistent Transfer Estimates in the European Union and Accession States*. Paper prepared for the European Commission (DGXI) Workshop on Valuing Mortality and Valuing Morbidity, November 13, 2000, Brussels. Revised, December 2000.

Peters R *et al* (2019). Air pollution and dementia: A systematic review. *Journal of Alzheimer's Disease* **70**(s1): S145-S163.

Pezzey JCV (2019). Why the social cost of carbon will always be disputed. *WIREs Climate Change* **10**: e558.

PHARMAC (2015). *Prescription for Pharmacoeconomic Analysis. Methods for cost-utility analysis*. Version 2-2. Pharmaceutical Management Agency, NZ.

PHARMAC (2017). *Annual Report For the year ended 30 June 2017*. Pharmaceutical Management Agency, NZ.

PHARMAC (2018). *Cost Resource Manual Version 3*. Pharmaceutical Management Agency, NZ.

PHARMAC (2019). *Annual Report For the year ended 30 June 2019*. Pharmaceutical Management Agency, NZ.

PHARMAC (2021). *Annual Report For the year ended 30 June 2021*. Pharmaceutical Management Agency, NZ.

Pindyck RS (2013). Climate Change Policy: What Do the Models Tell Us? *Journal of Economic Literature* **51**(3): 860—872.

Pindyck RS (2015). *The Use and Misuse of Models for Climate Policy*. National Bureau of Economic Research Working Paper 21097.

Powell E *et al* (2019). *Air quality damage cost guidance*. Prepared for Department of Environment, Food, Rural Affairs, UK.

Pope CA III *et al* (2009). Fine-particulate air pollution and life expectancy in the United States. *The New England Journal of Medicine* **360**(4): 376-386.

Prüss-Üstün A *et al* (2003). *Introduction and methods: assessing the environmental burden of disease at national and local levels.* Geneva: World Health Organization. (WHO Environmental Burden of Disease Series, No. 1). https://apps.who.int/iris/handle/10665/42750

Quinn N (2005). How to reconstruct schemas people share, from what they say. In: Quinn N (eds) *Finding culture in talk*. Palgrave Macmillan, New York, pp35-81.

Raaschou-Nielsen O *et al* (2016). Particulate matter air pollution components and risk for lung cancer. *Environment International* **87**: 66-73.

Rabl A (2003). Interpretation of Air Pollution Mortality: Number of Deaths or Years of Life Lost? *J Air & Waste Manage. Assoc.* **53**: 41–50. https://doi.org/10.1080/10473289.2003.10466118

Rabl A *et al* (2005). *Externalities of energy: Extension of accounting framework and policy applications*. Final Technical Report ENG1-CT2002-00609.

RCP (2016). *Every breath we take: the lifelong impact of air pollution*. Working Party Report, Royal College of Physicians (RCP) UK. https://www.rcplondon.ac.uk/projects/outputs/every-breath-we-take-lifelong-impact-air-pollution

Ricardo-AEA (2014). *Update of the Handbook on External Costs of Transport.* Report for the European Commission, DG MOVE Ricardo-AEA/R/ ED57769.

Rivlin MM (2000). Why the fair innings argument is not persuasive. *BMC Medical Ethics* **1**: 1. https://bmcmedethics.biomedcentral.com/articles/10.1186/1472-6939-1-1

Rizzi LI & de D Ortúzar J (2003). Stated preference in the valuation of interurban road safety. *Accident Analysis and Prevention* **35**: 9–22.

Rizzi LI & de D Ortúzar J (2006a). Estimating the Willingness-to-Pay for Road Safety Improvements. *Transport Reviews* **26**(4): 471–485.

Rizzi LI & de D Ortúzar J (2006b). Road safety valuation under a stated choice framework. *Journal of Transport Economics and Policy* **40**(1): 69–94.

Robinson LA *et al* (2018). *Valuing Mortality Risk Reductions in Global Benefit-Cost Analysis. Guidelines for Benefit-Cost Analysis Project Working Paper No. 7*. Prepared for the Benefit-Cost Analysis Reference Case Guidance Project Funded by the Bill and Melinda Gates Foundation.

Röösli M *et al* (2005). Years of life lost attributable to air pollution in Switzerland: dynamic exposure-response model. *International Journal of Epidemiology* **34**(5): 1029-35.

Sagoff M (1988). *The Economy of the Earth. Philosophy, Law and the Environment.* Cambridge University Press.

Salomon JA *et al* (2015). Disability weights for the Global Burden of Disease 2013 study. *Lancet Global Health* **3**(11): e712–723.

Scovronick N et al (2019). Human health and the social cost of carbon: A primer and call to action. *Epidemiology*, 30(5): 642-647.

Seeni I *et al* (2019). Acute air pollution exposure and NICU admission: a case-crossover analysis. *Annals of Epidemiology* **37**: 64-70.e2.

Seethaler RK *et al* (2003). Economic Costs of Air Pollution Related Health Impacts: An Impact Assessment Project of Austria, France and Switzerland. *Clean Air and Environmental Quality* **37**(1): 35-43.

Shepard DS & Zeckhauser RJ (1984). Survival versus consumption. *Management Science* **30**(4): 423–39.

Simpson J *et al* (2017). *The Health Status of Pacific Children and Young People in New Zealand 2015.* Dunedin: New Zealand Children and Youth Epidemiology Service, University of Otago.

Sridhar S *et al* (2022a). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Health Effects Model*. Excel model prepared by S Sridhar, J Metcalfe and K Mason for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, March 2022.

Sridhar S *et al* (2022b). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Health Effects Model – Users' guide.* Guide prepared by S Sridhar, G Kuschel and K Mason for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, March 2022.

Sridhar S & Kuschel G (2018). *Transport-Related Air emissions INventory Stocktake (TRAINS)*. Report prepared by Emission Impossible Ltd for NZ Transport Agency, NZ.

Sridhar S & Metcalfe J (2019). *Auckland Air Emissions Inventory 2016 – Transport (Revised)*. Auckland Council Technical Report 2018/016-2, March 2019. https://knowledgeauckland.org.nz/media/1093/tr2018-016-2-auckland-air-emissions-inventory-2016-transport-revised.pdf

Sridhar S & Metcalfe J (2021). *Health and air pollution in New Zealand 2016 (HAPINZ 3.0): Sources*. Excel model prepared by S Sridhar and J Metcalfe for Ministry for the Environment, Ministry of Health, Te Manatū Waka Ministry of Transport and Waka Kotahi NZ Transport Agency, February 2021.

Stats NZ (2017). *ANZLIC metadata for urban area, 2017.* Stats NZ, Wellington, NZ. https://datafinder.stats.govt.nz/document/689-anzlic-metadata-2017-urban-area/

Stats NZ (2018). *New Zealand abridged period life table: 2015–17 (final).* Stats NZ, Wellington, NZ. https://www.stats.govt.nz/information-releases/new-zealand-abridged-period-life-table-201517-final

Steenhof M *et al* (2011). In vitro toxicity of particulate matter (PM) collected at different sites in the Netherlands is associated with PM composition, size fraction and oxidative potential – the RAPTES project. *Particle and Fibre Toxicology* **8**: 26. https://doi.org/10.1186/1743-8977-8-26

Stieb D *et al* (2021). Systematic review and meta-analysis of cohort studies of long term outdoor nitrogen dioxide exposure and mortality. *PLOS One* **16**(2): e0246451.

Strak M *et al* (2021). Long term exposure to low level air pollution and mortality in eight European cohorts within the ELAPSE project: pooled analysis. *BMJ 2021* **374**: n1904. https://doi.org/10.1136/bmj.n1904

Sunstein CR (2013). *The Value of a Statistical Live: Some Clarifications and Puzzles*. Mossavar-Rahmani Center for Business and Government. Harvard Kennedy School. RPP-2013-18.

Svensson M & Johansson MV (2010). Willingness to pay for private and public road safety in stated preference studies: Why the difference? *Accident Analysis and Prevention* 42: 1205-1212.

T&T (2020). *Validation of the DMRB model output for $PM_{2.5}$.* Report prepared by Tonkin & Taylor for Waka Kotahi, June.

T&T (2021). *Vehicle emissions mapping tool – Summary report*. Draft version 2. Report prepared by Tonkin & Taylor for Waka Kotahi, 16 April.

Talbot N *et al* (2017). *Auckland ambient air quality trends for $PM_{2.5}$ and PM_{10} – 2006 – 2015*. Auckland Council Technical Report 2017/029. Auckland, NZ. http://knowledgeauckland.org.nz

Telfar-Barnard L *et al* (2011). *The impact of retrofitted insulation and new heaters on health services utilisation and costs, pharmaceutical costs and mortality Evaluation of Warm Up New Zealand: Heat Smart.* Report to Ministry of Economic Development, NZ.

Telfar-Barnard L & Zhang J (2019). *The impact of respiratory disease in New Zealand: 2018 update*. Asthma + Respiratory Foundation New Zealand.

The Highways Agency (2007). *Design Manual for Roads and Bridges, Volume 11, Section 3 Environmental Assessment Techniques, Part 1 HA207/07 Air quality*. The Highways Agency, UK, May.

The Workshop & Oxfam New Zealand (2019). *How to Talk About Climate Change. A Toolkit for Collection Action*. The Workshop. Wellington, NZ. https://www.theworkshop.org.nz/publications/how-to-talk-about-climate-change-a-short-guide-2019

Thibodeau PH *et al* (2017). How linguistic metaphor scaffolds reasoning. *Trends in Cognitive Sciences* 21(11): 852-863.

Thurston GD *et al* (2016). Ischemic heart disease mortality and long-term exposure to source-related components of U.S. fine particle air pollution. *Env Health Perspectives*, 124(6): 785-94.

Tibuakuu M *et al* (2018). Air pollution and cardiovascular disease: a focus on vulnerable populations worldwide. *Current Epidemiology Reports*, 5(4): 370-378.

Torfs R *et al* (2007). Deliverable 3.7 - RS1b/WP3 *"A set of concentration-response functions"*. New Energy Externalities Developments for Sustainability (NEEDS), EU Sixth Framework Programme.

US EPA (2002). *Technical Addendum: Methodologies for the Benefit Analysis of the Clear Skies Initiative*. US Environmental Protection Agency: Research Triangle Park, NC.

US EPA (2004a). *Advisory on plans for health effects analysis in the analytical plan for EPA's second prospective analysis—Benefits and costs of the Clean Air Act, 1990-2020*. Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis, US Environmental Protection Agency: Research Triangle Park, NC.

US EPA (2004b). Letter to Dr Trudy Cameron and Dr Bart Ostro, Science Advisory Board, 11 August 2004. US Environmental Protection Agency: Research Triangle Park, NC.

US EPA (2010). *Mortality Risk Valuation Estimates.* Appendix B in: Guidelines for Preparing Economic Analyses. EPA 240-R-10-001, US Environmental Protection Agency: Research Triangle Park, NC.

US EPA (2011). *The benefits and costs of the Clean Air Act from 1990 to 2020*. Final Report Rev A, US Environmental Protection Agency: Research Triangle Park, NC.

US EPA (2016). *Integrated science assessment for oxides of nitrogen – Health criteria.* US Environmental Protection Agency: Research Triangle Park, NC.

US EPA (2018). *Integrated science assessment for particulate matter (review draft)*. US Environmental Protection Agency: Research Triangle Park, NC.

US Office of Management and Budget (2003). *Circular A-4 Regulatory Analysis*.

Vincent J (2012). The Anti-Aging Movement. Contemporary Cultures and the Social Construction of Old Age. In: Schermer M and Pinxten W (Eds) *Ethics, Health Policy and (Anti-) Aging: Mixed Blessings*. Springer.

Viscusi WK (2005). *The value of life*. John M Olin Center for Law, Economics and Business discussion paper no.517.

Viscusi WK (2009). The devaluation of life. *Regulation & Governance*, 3: 103–127.

Walton HA (2010). *Supporting paper to COMEAP 2010 report: The mortality effects of long-term exposure to particulate air pollution in the United Kingdom*. Working paper: Development of proposals for cessation lag(s) for use in total impact calculations.

Wang Y *et al* (2016). Estimating causal effects of long-term $PM_{2.5}$ exposure on mortality in New Jersey. *Environmental Health Perspectives*, 124(8): 1182-1188.

Webber-Foster R *et al* (2014). Cost-effectiveness analysis of docetaxel versus paclitaxel in adjuvant treatment of regional breast cancer in New Zealand. *Pharmacoeconomics*, 32: 707–724.

WHO (2006). *Air quality guidelines – global update 2005*. World Health Organization. https://www.who.int/phe/health_topics/outdoorair/outdoorair_aqg/en/

WHO (2012). *Health effects of black carbon*. World Health Organization. https://www.euro.who.int/en/publications/abstracts/health-effects-of-black-carbon-2012

WHO (2013). *Review of evidence on health aspects of air pollution –REVIHAAP Project*. World Health Organization. https://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/2013/review-of-evidence-on-health-aspects-of-air-pollution-revihaap-project-final-technical-report

WHO (2014). *WHO Handbook for Guideline Development*. 2nd Edition. World Health Organization. https://apps.who.int/iris/handle/10665/145714

WHO (2015). *Economic cost of the health impact of air pollution in Europe: Clean air, health and wealth*. World Health Organization Regional Office for Europe and OECD.

WHO (2016a). *Health risk assessment of air pollution – general principles*. World Health Organization. https://www.euro.who.int/en/publications/abstracts/health-risk-assessment-of-air-pollution.-general-principles-2016

WHO (2016b). *Ambient air pollution: A global assessment of exposure and burden of disease*. World Health Organization. https://apps.who.int/iris/bitstream/handle/10665/250141/9789241511353-eng.pdf

WHO (2018). *Burden of disease from ambient air pollution for 2016 – Description of method*. Version 5 May 2018, World Health Organization. https://www.who.int/airpollution/data/AAP_BoD_methods_Apr2018_final.pdf

WHO (2020). *Risk of Bias Assessment Instrument for Systematic Reviews Informing Who Global Air Quality Guidelines.* World Health Organization, WHO Regional Office for Europe, Copenhagen, Denmark

WHO (2021). *WHO global air quality guidelines. Particulate matter ($PM_{2.5}$ and PM_{10}), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide.* World Health Organization. https://apps.who.int/iris/handle/10665/345329

Wilton E *et al* (2008). *New Zealand sulphur dioxide industrial emissions inventory 2007*. Prepared by Environet Ltd for Ministry for the Environment.

Wilton E *et al* (2015). *Home heating emissions inventory and other sources evaluation*. Also known as *2013 National Air Emissions Inventory.* Prepared by Environet Ltd and Golders Associates for Ministry for the Environment and Stats NZ. Spreadsheet available at https://data.mfe.govt.nz/data/category/environmental-reporting/air/home-heating-emissions/

Williams A (1997). Intergenerational Equity: An Exploration of the "Fair Innings" Argument. *Health Economics* 6(2): 117–132.

Williams AM *et al* (2019). Short-term impact of $PM_{2.5}$ on contemporaneous asthma medication use: Behavior and the value of pollution reductions. *Proceedings of the National Academy of Sciences*, 116(12): 5246-5253.

Xie S *et al* (2019). *Auckland Air Emissions Inventory 2016*. Auckland Council Technical Report 2019/024, December 2019. https://knowledgeauckland.org.nz/media/1558/tr2019-024-auckland-air-emissions-inventory-2016.pdf

Yazdi M *et al* (2021). Long-Term Association of Air Pollution and Hospital Admissions Among Medicare Participants Using a Doubly Robust Additive Model. *Circulation* **143**: 1584-1596. https://doi.org/10.1161/CIRCULATIONAHA.120.050252

Yitshak-Sade M *et al* (2018). The association between short and long-term exposure to $PM_{2.5}$ and temperature and hospital admissions in New England and the synergistic effect of the short-term exposures. *Science of The Total Environment* **639**: 868-875.

Appendix A: PM_{10} concentration vs emissions density

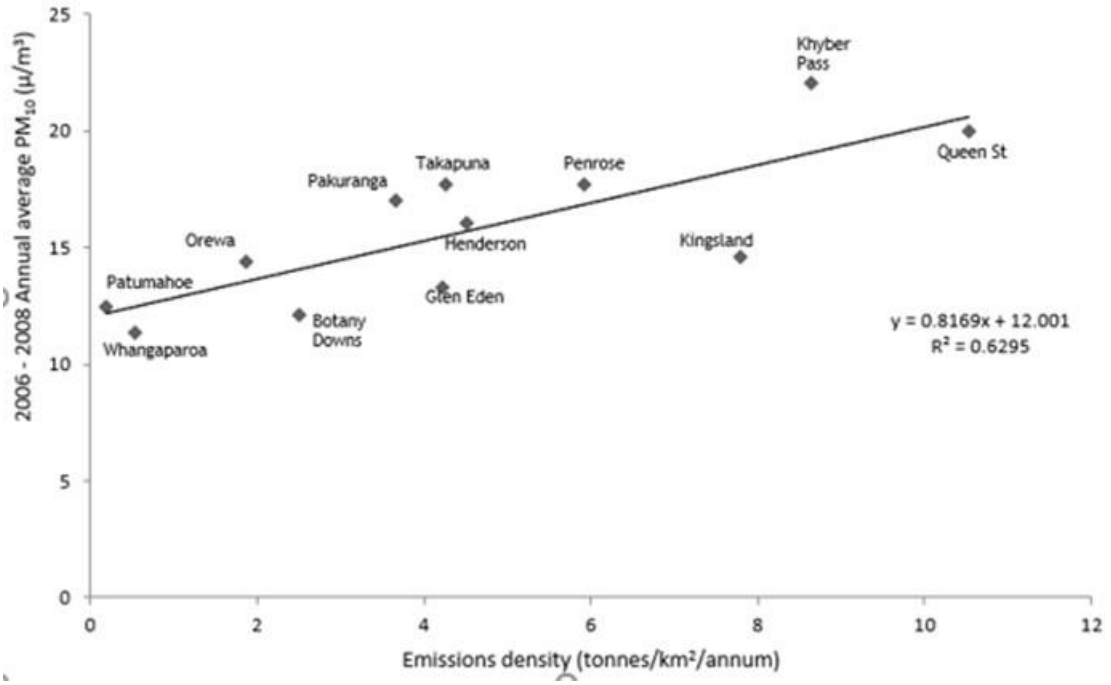
This appendix summarises the analyses undertaken to determine whether relationships between annual average PM_{10} concentrations and emission densities used in HAPINZ 2.0 could be used in HAPINZ 3.0 to provide more spatially representative concentrations across Auckland other locations.

A.1 Correlations for HAPINZ 2.0 (based on 2006-2008)

Previous regression analyses indicated a correlation between estimated emissions density and annual average PM_{10} in Auckland, Rotorua and Invercargill. Emissions density was based on estimated PM_{10} emissions from home heating, open burning and motor vehicles in the census area unit where the monitor was located.

Figure 29 shows the correlation between annual average PM_{10} (for the years 2006–2008) and estimated emissions density in Auckland from HAPINZ 2.0.

Figure 29: Correlation between emissions density and annual average PM_{10} in Auckland in HAPINZ 2.0



Source: HAPINZ 2.0 Exposure Model, App1, Kuschel *et al* (2012a)

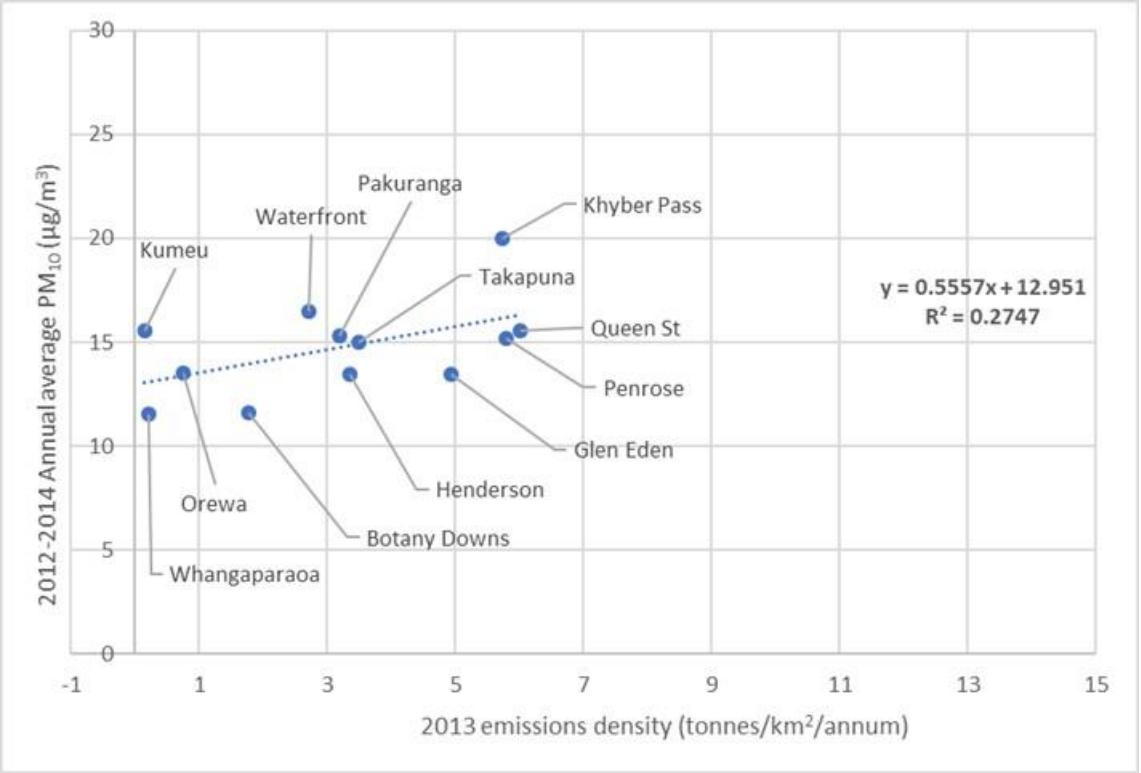
Based on the robustness of these correlations for 2006 to 2008, emissions density was used in HAPINZ 2.0 to estimate concentrations in areas to better represent spatial variability.

A.2 Findings for HAPINZ 3.0 (based on 2012-2014)

For HAPINZ 3.0, we repeated this analysis based on estimated 2013 emissions density and monitoring data to see whether emissions density might still be useful.

Figure 30 presents the analysis for Auckland using 2012–2014 data and shows no clear correlation with the recent data. The concentration of PM_{10} is reasonably consistent across the Auckland airshed and is no longer significantly influenced by localised emissions density.

Figure 30: Updated correlation between emissions density and annual average PM_{10} in Auckland



Since 2007, concentrations have declined appreciably at peak locations (such as Queen Street) due to vehicle fuel and domestic fire emissions improvements and we hypothesise that the influence of local factors such as meteorology, topography and proximity of the monitoring sites to roads are now more significant.

As the strong correlations found in HAPINZ 2.0 were no longer evident, we did not use emissions density in HAPINZ 3.0.

Appendix B: PM source attribution results

This appendix presents the final adjusted source attribution profiles for PM_{10} (Table 36) and $PM_{2.5}$ (Table 37) by region and location used in the *HAPINZ 3.0 Health Effects Model*.

Table 36: Final adjusted source attribution profiles for PM_{10} in all areas

Region	Location	Total PM_{10} µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
All Regions	Unmonitored industrial areas	18.2	2.0	2.9	1.5	5.3	1.0	5.6
	Unmonitored rural areas	9.2	0.9	0.0	1.5	5.3	0.0	1.5
Northland	Urban areas outside airsheds	13.7	2.2	1.6	1.5	6.6	0.0	1.8
	Kaitaia	16.0	3.8	1.7	1.5	6.6	0.0	2.5
	Keri Keri	13.7	2.2	1.6	1.5	6.6	0.0	1.7
	Whangarei	13.7	2.2	1.8	1.5	6.6	0.0	1.6
	Marsden Point	12.3	0.0	1.6	1.5	6.6	1.0	1.7
	Dargaville	13.7	2.2	1.6	1.5	6.6	0.0	1.7
Auckland	Urban areas outside airsheds	11.6	1.7	1.6	1.4	6.6	0.0	0.3
	Wellsford	11.6	1.7	1.7	1.4	6.5	0.0	0.3
	Warkworth	13.2	1.9	1.8	1.5	6.6	0.0	1.5
	Snells Beach	11.6	1.7	1.6	1.4	6.5	0.0	0.3
	Helensville	13.3	2.0	1.6	1.5	6.6	0.0	1.6

Region	Location	Total PM_{10} µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
	Kumeu	13.2	1.9	1.8	1.5	6.6	0.0	1.5
	Riverhead	11.6	1.7	1.6	1.4	6.5	0.0	0.3
	Auckland CBD (Auckland Central East & West)	16.8	1.4	5.1	1.5	6.6	0.0	2.3
	Harbourside	17.7	2.1	3.7	1.5	6.6	0.0	3.7
	Newmarket, Epsom North, Grafton East & West, Newton, Eden Tce, Arch Hill	20.0	1.7	5.0	1.5	6.6	0.0	5.1
	Auckland Urban	13.2	2.1	2.3	1.5	6.6	0.0	0.8
	Waiheke Island	12.2	1.7	1.6	1.5	6.6	0.0	0.9
	Beachlands & Maraetai	12.0	1.8	1.6	1.5	6.6	0.0	0.5
	Pukekohe	13.2	1.9	1.7	1.5	6.6	0.0	1.5
	Waiuku	13.2	2.3	1.7	1.5	6.6	0.0	1.1
	Auckland Rural	11.2	1.0	0.0	1.5	6.6	0.0	2.2
Waikato	Urban areas outside airsheds	9.9	2.6	1.6	1.4	3.9	0.0	0.4
	Whitianga	8.5	2.8	1.3	1.1	3.1	0.0	0.3
	Thames	8.5	2.7	1.3	1.1	3.0	0.0	0.3
	Whangamata	8.5	2.7	1.3	1.1	3.0	0.0	0.3
	Tuakau	11.6	2.6	1.6	1.5	3.9	0.0	2.0
	Paeroa	11.6	2.6	1.6	1.5	3.9	0.0	2.0
	Waihi	12.3	2.6	1.6	1.5	3.9	0.7	2.0
	Huntly	9.9	2.6	1.6	1.4	3.8	0.0	0.4
	Te Aroha	11.6	2.6	1.6	1.5	3.9	0.0	2.0

Region	Location	Total PM_{10} µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
	Ngaruawahia	11.6	2.6	1.7	1.5	3.9	0.0	1.9
	Morrinsville	11.6	2.6	1.7	1.5	3.9	0.0	1.9
	Hamilton City	11.9	2.4	2.0	1.5	3.9	0.0	2.2
	Matamata	11.6	2.6	1.7	1.5	3.9	0.0	1.9
	Cambridge	11.0	2.8	1.7	1.5	3.9	0.0	1.1
	Te Awamutu & Kihikihi	11.8	2.8	1.7	1.5	3.9	0.0	1.9
	Putaruru	12.5	4.8	1.7	1.5	3.9	0.0	0.7
	Otorohanga	11.6	2.6	1.8	1.5	3.9	0.0	1.9
	Tokoroa	15.4	9.0	1.4	1.2	3.3	0.0	0.4
	Te Kuiti	14.9	5.5	1.7	1.5	3.9	0.0	2.4
	Taupo	12.7	5.5	1.7	1.4	3.8	0.0	0.3
	Turangi	10.0	3.8	1.4	1.2	3.3	0.0	0.3
Bay of Plenty	Urban areas outside airsheds	10	2.0	1.8	1.4	4.3	0.0	0.3
	Rotorua	16.8	6.9	1.8	1.5	3.9	0.0	2.6
	Mt Maunganui	20.0	2.6	2.9	1.5	4.5	2.5	6.1
Gisborne	Urban areas outside airsheds	13.5	4.7	1.8	1.5	4.5	0.0	1.1
Manawatū-Whanganui	Urban areas outside airsheds	11.6	3.7	1.6	1.5	3.9	0.0	1.0
	Taumarunui	11.6	4.6	1.5	1.4	3.7	0.0	0.3
	Taihape	13.2	3.8	1.6	1.5	3.9	0.0	2.5

Region	Location	Total PM_{10} µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
Taranaki	Urban areas outside airsheds	11.9	2.4	2.0	1.5	4.5	0.0	1.6
Hawke's Bay	Urban areas outside airsheds	13.2	3.6	1.6	1.5	4.5	0.0	2.1
	Whirinaki	13.2	3.6	1.6	1.5	4.5	0.0	2.1
	Napier	13.2	3.4	1.9	1.5	4.5	0.0	1.9
	Hastings	14.5	5.5	1.8	1.5	4.5	0.0	1.3
	Awatoto	18.6	1.7	1.9	1.5	9.1	1.1	3.4
Wellington	Urban areas outside airsheds	10.4	2.6	1.7	1.5	4.3	0.0	0.4
	Kapiti Coast	10.4	2.6	1.8	1.5	4.3	0.0	0.3
	Masterton	14.2	7.1	1.5	1.3	3.9	0.0	0.3
	Porirua	12.0	3.5	1.9	1.5	4.3	0.0	0.9
	Upper Hutt	10.4	2.9	1.7	1.4	4.1	0.0	0.3
	Lower Hutt	11.3	1.9	1.9	1.5	4.3	0.0	1.7
	Wainuiomata	10.8	3.0	1.7	1.5	4.3	0.0	0.4
	Wellington City	11.3	1.6	2.3	1.5	4.3	0.0	1.7
	Karori	10.4	2.6	1.7	1.5	4.3	0.0	0.4
Nelson	Urban areas outside airsheds	11.0	3.3	1.8	1.5	3.6	0.0	0.8
	Nelson A - Nelson South	17.0	7.7	1.8	1.5	3.6	0.0	2.4
	Nelson B1 - Tahunanui	18.3	4.6	1.8	1.5	3.6	2.5	4.4
	Nelson B2 - Stoke	11.0	3.3	1.8	1.5	3.6	0.0	0.8
	Nelson C - Nelson City	12.0	4.0	1.8	1.5	3.6	0.0	1.1

Region	Location	Total PM_{10} µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
Tasman	Urban areas outside airsheds	11.0	3.3	1.8	1.5	3.6	0.0	0.8
	Richmond	17.7	8.2	1.7	1.5	3.6	0.0	2.7
Marlborough	Urban areas outside airsheds	9.7	2.0	1.8	1.5	2.0	0.0	2.4
	Blenheim	18.3	9.8	1.8	1.5	2.0	0.0	3.3
West Coast	Urban areas outside airsheds	16.4	7.5	1.4	1.2	5.9	0.0	0.4
	Reefton	16.4	7.5	1.4	1.2	5.9	0.0	0.4
Canterbury	Urban areas outside airsheds	17.3	5.8	1.6	1.5	6.9	0.0	1.6
	Rangiora	17.7	5.8	1.7	1.5	6.9	0.0	1.9
	Kaiapoi	19.0	7.2	1.8	1.5	6.9	0.0	1.7
	Christchurch	18.3	5.5	2.1	1.5	6.9	0.0	2.5
	Ashburton	18.3	5.9	1.8	1.5	6.9	0.0	2.3
	Geraldine	18.0	7.0	1.6	1.5	6.9	0.0	1.1
	Timaru	24.0	9.6	1.8	1.5	6.9	0.0	4.3
	Waimate	17.3	6.0	1.6	1.5	6.9	0.0	1.4
	Canterbury Rural	9.2	0.9	0.0	1.5	5.3	0.0	1.5
Otago Region	Urban areas outside airsheds	15.7	2.8	1.9	1.5	4.9	0.0	4.6
	Dunedin	15.7	2.8	1.9	1.5	4.9	0.0	4.6
	Otago 1	19.1	12.6	1.3	1.1	3.8	0.0	0.3
	Otago 2	21.7	9.4	1.7	1.5	4.9	0.0	4.2
	Otago 3	18.2	11.9	1.2	1.1	3.7	0.0	0.3

Region	Location	Total PM_{10} µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
	Otago 4	17.0	10.9	1.2	1.1	3.6	0.0	0.3
Southland	Urban areas outside airsheds	10.3	2.2	1.6	1.4	4.7	0.0	0.4
	Gore	19.3	6.5	1.7	1.5	4.9	0.0	4.8
	Invercargill	20.3	7.9	1.8	1.5	4.9	0.0	4.3

Table 37: Final adjusted source attribution profiles for $PM_{2.5}$ in all areas

Region		Total $PM_{2.5}$ µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
All Regions	Unmonitored industrial areas	6.5	2.0	1.5	1.1	1.3	0.3	0.4
	Unmonitored rural areas	4.4	0.9	0.0	1.4	1.6	0.0	0.5
Northland	Urban areas outside airsheds	6.0	2.2	0.7	1.2	1.5	0.0	0.4
	Kaitaia	7.4	3.8	0.7	1.2	1.4	0.0	0.4
	Keri Keri	6.0	2.2	0.7	1.2	1.5	0.0	0.4
	Whangarei	6.0	2.2	0.8	1.2	1.4	0.0	0.4
	Marsden Point	4.1	0.0	0.6	1.2	1.5	0.4	0.4
	Dargaville	6.0	2.2	0.7	1.2	1.5	0.0	0.4
Auckland	Urban areas outside airsheds	4.4	1.7	0.4	0.9	1.1	0.0	0.3
	Wellsford	4.4	1.7	0.5	0.9	1.1	0.0	0.3
	Warkworth	5.0	1.9	0.7	1.0	1.2	0.0	0.3

Region		Total $PM_{2.5}$ µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
	Snells Beach	4.4	1.7	0.5	0.9	1.1	0.0	0.3
	Helensville	5.1	2.0	0.5	1.0	1.2	0.0	0.3
	Kumeu	5.0	1.9	0.6	1.0	1.2	0.0	0.3
	Riverhead	4.4	1.7	0.5	0.9	1.1	0.0	0.3
	Auckland CBD (Auckland Central East & West)	6.7	1.4	3.0	0.9	1.1	0.0	0.3
	Harbourside	7.4	2.1	2.4	1.1	1.4	0.0	0.4
	Newmarket, Epsom North, Grafton East & West, Newton, Eden Tce, Arch Hill	7.0	1.7	2.9	0.9	1.1	0.0	0.3
	Auckland Urban	5.8	2.1	1.1	1.1	1.3	0.0	0.4
	Waiheke Island	4.4	1.7	0.4	0.9	1.1	0.0	0.3
	Beachlands & Maraetai	4.7	1.8	0.5	1.0	1.1	0.0	0.3
	Pukekohe	5.0	1.9	0.6	1.0	1.2	0.0	0.3
	Waiuku	6.1	2.3	0.7	1.2	1.5	0.0	0.4
	Auckland Rural	4.6	1.0	0.0	1.4	1.7	0.0	0.5
Waikato	Urban areas outside airsheds	5.8	2.6	0.5	1.1	1.3	0.0	0.4
	Whitianga	5.3	2.8	0.4	0.8	1.0	0.0	0.3
	Thames	5.3	2.7	0.5	0.8	1.0	0.0	0.3
	Whangamata	5.3	2.7	0.5	0.8	1.0	0.0	0.3
	Tuakau	5.9	2.6	0.6	1.1	1.3	0.0	0.4
	Paeroa	5.9	2.6	0.6	1.1	1.3	0.0	0.4
	Waihi	5.9	2.6	0.6	1.1	1.3	0.0	0.4

Region		Total $PM_{2.5}$ µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
	Huntly	5.9	2.6	0.5	1.1	1.3	0.0	0.4
	Te Aroha	5.9	2.6	0.5	1.1	1.3	0.0	0.4
	Ngaruawahia	5.9	2.6	0.6	1.0	1.2	0.0	0.4
	Morrinsville	5.9	2.6	0.6	1.0	1.3	0.0	0.4
	Hamilton City	5.8	2.4	0.8	1.0	1.2	0.0	0.3
	Matamata	5.9	2.6	0.6	1.0	1.3	0.0	0.4
	Cambridge	5.9	2.8	0.6	1.0	1.2	0.0	0.3
	Te Awamutu & Kihikihi	6.0	2.8	0.6	1.0	1.2	0.0	0.3
	Putaruru	8.0	4.8	0.6	1.0	1.2	0.0	0.3
	Otorohanga	5.9	2.6	0.6	1.0	1.2	0.0	0.3
	Tokoroa	12.5	9.0	0.6	1.1	1.3	0.0	0.4
	Te Kuiti	8.9	5.5	0.6	1.1	1.3	0.0	0.4
	Taupo	8.6	5.5	0.6	1.0	1.2	0.0	0.3
	Turangi	6.6	3.8	0.5	0.9	1.1	0.0	0.3
Bay of Plenty	Urban areas outside airsheds	5.0	2.0	0.7	0.9	1.1	0.0	0.3
	Rotorua	10.2	6.9	0.7	1.0	1.2	0.0	0.3
	Mt Maunganui	7.1	2.6	1.0	1.2	1.4	0.6	0.4
Gisborne	Urban areas outside airsheds	8.0	4.7	0.7	1.1	1.3	0.0	0.4
Manawatū-Whanganui	Urban areas outside airsheds	6.1	3.7	0.4	0.8	0.9	0.0	0.3
	Taumarunui	7.6	4.6	0.5	1.0	1.2	0.0	0.3

Region		Total $PM_{2.5}$ µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
	Taihape	6.9	3.8	0.5	1.0	1.2	0.0	0.3
Taranaki	Urban areas outside airsheds	5.8	2.4	0.8	1.0	1.2	0.0	0.3
Hawke's Bay	Urban areas outside airsheds	7.0	3.6	0.5	1.1	1.4	0.0	0.4
	Whirinaki	7.0	3.6	0.5	1.1	1.4	0.0	0.4
	Napier	7.0	3.4	0.8	1.1	1.3	0.0	0.4
	Hastings	8.7	5.5	0.6	1.0	1.2	0.0	0.3
	Awatoto	6.2	1.7	0.9	1.2	1.5	0.4	0.4
Wellington	Urban areas outside airsheds	5.2	2.6	0.5	0.8	1.0	0.0	0.3
	Kapiti Coast	5.2	2.6	0.5	0.8	1.0	0.0	0.3
	Masterton	10.2	7.1	0.6	1.0	1.2	0.0	0.3
	Porirua	6.6	3.5	0.7	1.0	1.2	0.0	0.3
	Upper Hutt	5.9	2.9	0.6	0.9	1.1	0.0	0.3
	Lower Hutt	5.2	1.9	0.8	1.0	1.2	0.0	0.3
	Wainuiomata	6.1	3.0	0.6	1.0	1.2	0.0	0.3
	Wellington City	5.4	1.6	1.1	1.1	1.3	0.0	0.4
	Karori	5.2	2.6	0.5	0.8	1.0	0.0	0.3
Nelson	Urban areas outside airsheds	6.4	3.3	0.7	1.0	1.2	0.0	0.3
	Nelson A - Nelson South	11.0	7.7	0.7	1.0	1.2	0.0	0.3
	Nelson B1 - Tahunanui	9.5	4.6	0.7	1.0	1.2	1.6	0.4
	Nelson B2 - Stoke	6.4	3.3	0.7	1.0	1.2	0.0	0.3

Region		Total $PM_{2.5}$ µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
	Nelson C - Nelson City	6.9	4.0	0.6	0.9	1.1	0.0	0.3
Tasman	Urban areas outside airsheds	6.4	3.3	0.7	1.0	1.2	0.0	0.3
	Richmond	11.6	8.2	0.7	1.1	1.3	0.0	0.4
Marlborough	Urban areas outside airsheds	5.6	2.0	0.6	0.9	1.1	0.0	0.3
	Blenheim	13.5	9.8	0.8	1.2	1.4	0.0	0.4
West Coast	Urban areas outside airsheds	10.9	7.5	0.6	1.1	1.4	0.0	0.4
	Reefton	10.9	7.5	0.6	1.1	1.4	0.0	0.4
Canterbury	Urban areas outside airsheds	9.3	5.8	0.6	1.1	1.4	0.0	0.4
	Rangiora	9.3	5.8	0.7	1.1	1.3	0.0	0.4
	Kaiapoi	11.0	7.2	0.8	1.2	1.4	0.0	0.4
	Christchurch	10.1	5.5	1.2	1.4	1.6	0.0	0.5
	Ashburton	9.8	5.9	0.8	1.2	1.4	0.0	0.4
	Geraldine	10.8	7.0	0.7	1.2	1.5	0.0	0.4
	Timaru	13.9	9.6	0.9	1.4	1.6	0.0	0.5
	Waimate	9.7	6.0	0.6	1.2	1.4	0.0	0.4
	Canterbury Rural	4.4	0.9	0.0	1.4	1.6	0.0	0.5
Otago Region	Urban areas outside airsheds	7.0	2.8	1.0	1.3	1.5	0.0	0.4
	Dunedin	7.0	2.8	1.0	1.3	1.5	0.0	0.4
	Otago 1	15.4	12.6	0.5	0.9	1.1	0.0	0.3

Region		Total $PM_{2.5}$ µg/m³	Domestic fires µg/m³	Motor vehicles µg/m³	Secondary PM µg/m³	Sea spray µg/m³	Industry µg/m³	Windblown dust µg/m³
	Otago 2	13.5	9.4	0.8	1.3	1.6	0.0	0.4
	Otago 3	14.7	11.9	0.5	0.9	1.1	0.0	0.3
	Otago 4	13.7	10.9	0.5	0.9	1.1	0.0	0.3
Southland	Urban areas outside airsheds	5.6	2.2	0.6	1.1	1.3	0.0	0.4
	Gore	10.5	6.5	0.8	1.3	1.5	0.0	0.4
	Invercargill	11.8	7.9	0.8	1.2	1.5	0.0	0.4

Appendix C: ERF sensitivity analyses

This appendix presents the detailed results of the sensitivity analyses conducted to confirm the robustness of the *HAPINZ 3.0 Cohort Study* exposure-response functions, especially those relating to the effect of NO_2.

C.1 Risk of bias

The following outlines our assessment of the risk of bias in the *HAPINZ 3.0 Cohort Study* using the WHO (2020) guidance.

1. **Confounding:** There is a *low to moderate* risk of bias in this domain, due to our inability to control for BMI or noise.

An unmeasured factor which is associated with both NO_2 exposure and mortality could bias the results. This type of bias can never be completely excluded. For example, **NO_2 might be acting as a proxy for exposure to traffic, and the true causal factor might be another traffic related exposure such as noise**. Studies of air pollution and health which control for noise have reported mixed results (see Steib *et al* 2021, p13-14). If noise is a confounder in the relation of NO_2 to mortality, then this cannot explain why the NZ result is different to other places, where noise has not usually been included in the models. Nonetheless, future air pollution studies in NZ should ideally control for noise.

WHO **recommends controlling for body mass index (BMI**) which we were unable to do. However, we found no significant difference in effect of NO_2 by ethnicity. Including ethnicity in the models would be expected to control for BMI indirectly, since BMI is associated with ethnicity in NZ. Therefore, lack of control for BMI is unlikely to have affected the results of HAPINZ 3.0.

We were able to control for effects of age, sex, ethnicity, education, income and smoking at individual level, based on census data linkage. These data are likely to be of high quality. We controlled for ambient temperature and co-pollutants at census area level. We carried out a stepwise analysis which allowed assessment of potential confounding at each step. The effects of NO_2 were not substantially attenuated (reduced) by control for confounding, which supports a causal inference.

2. **Selection bias:** *low* risk of bias

This is unlikely to be a problem in this population-based study with high levels of participation.

3. **Exposure assessment:** *low* risk of bias

Exposure levels were assessed with appropriate methods. For the two pollutant models, NO_2 exposure was estimated at meshblock level, based on a map of concentrations at 50m resolution, while PM exposure was estimated at census area level. It is possible that this may lead to an under-estimate of effects of PM, if exposure misclassification was greater for this pollutant class. However, the results were not altered substantially when analyses were repeated using average NO_2 exposure at census area level. The estimates of coarse PM

concentration ($PM_{2.5-10}$) may be subject to greater bias because this involves subtraction of one modelled estimate from another, for many CAUs where measurements of both $PM_{2.5}$ and PM_{10} were not available.

Results were substantially unchanged when decadal average pollutant exposures were used. Both actual and modelled exposure contrasts may be relatively well defined in NZ, compared to other settings, due to lack of long-distance transport of air pollution.

4. **Outcome measurement:** *low* risk of bias

Mortality and hospital discharge data used in the analysis are expected to be of high quality by international standards.

5. **Missing data:** *low* risk of bias

Models had less than 5% missing outcome and confounder data (approximately 100,000 observations out of ~2.3 million). Pollution exposure was almost complete.

6. **Selective reporting:** *low* risk of bias

Main aims and analyses were defined in advance and are reported in full.

C.2 Mortality model selection

The two statistical models most often used for assessing *mortality* events are the Cox proportional hazards model and the Poisson regression model.

The **Cox proportional hazards regression model** gets its name, in part, from the assumption that the hazard function or death rate for one group of patients will be proportional to the hazard function or death rate from another group. This is equivalent to assuming the relative risk of death between the two groups will be constant over time.

One of the chief advantages of the Cox model is that there are no assumptions regarding what the *shape* of the underlying hazard or death rate looks like. It is for this reason that estimates of relative risk are more robust under the Cox model than what might otherwise be obtained using a fully parametric model. Unfortunately, this is also a disadvantage in that it does not allow one to formally test hypotheses about the shape of the hazard function although one can still estimate and describe its shape.

An alternative is the **Poisson regression model**. The Poisson model is similar to the Cox model in that both account for *censored*[70] data and assume the death rates between any two groups of patients will be proportional to one another. Like the Cox model, the Poisson model also accommodates non-proportional death rates through the use of time-dependent covariates. However, unlike the Cox model, the Poisson model is semi-parametric in that it assumes death

[70] Censored observations are subjects who either die of causes other than the disease of interest or are lost to follow-up. Ignoring these censored observations results in potentially valuable information on survival being thrown away so it is important to account for these data.

rates are constant within specified intervals of time. In fact, this is the key difference between the two models.

In the Cox model, the reference population's death rate over a specified interval of time is left unspecified, while in the Poisson model it is assumed constant. Both models assume the death rate for a comparative group of patients will be proportional to the death rate for the reference group within each specified interval of time.

For short intervals of follow-up (e.g., every 3 months or every 6 months), it is reasonable to assume that the death rates will be approximately constant. Consequently, by choosing appropriate intervals of follow-up, an interval Poisson model and an interval Cox model will give very nearly the same results with respect to relative risks.

We opted for Poisson regression in the *HAPINZ 3.0 Cohort Study* because we wanted to use the model to also assess *morbidity* impacts and Poisson enables us to consider multiple events (i.e. multiple hospitalisations) for the same affected person.

We re-ran the analyses using the Cox model and found the results for non-external causes of mortality were virtually identical to those from Poisson regression (Table 38).

Table 38: Comparison of the relative risks for all adult mortality derived from the Cox proportional hazards versus Poisson regression models

Model	Pollutant	Relative Risk (per 10 µg/m³)	Lower CI	Upper CI
Two pollutant model: 2016 estimates, Cox model	NO_2	1.097	1.074	1.120
	$PM_{2.5}$	1.105	1.066	1.146
Two pollutant model: 2016 estimates, Poisson model	NO_2	1.097	1.074	1.120
	$PM_{2.5}$	1.105	1.065	1.145

C.3 Assessment year

Results of the main models for non-external causes of mortality were not substantially altered whether estimated concentrations of $PM_{2.5}$ and NO_2 were based on 2016 estimates or decadal average (2006 to 2016) estimates (Table 39).

Table 39: Comparison of the relative risks for all adult mortality derived from 2016 pollutant concentration estimates or decadal average (2006 to 2016)

Model	Pollutant	Relative Risk (per 10µg/m³)	Lower CI	Upper CI
Two pollutant model: 2016 estimates	NO_2	1.097	1.074	1.120
	$PM_{2.5}$	1.105	1.065	1.145
Two pollutant model: decadal average (2006 to 2016)	NO_2	1.096	1.071	1.121
	$PM_{2.5}$	1.121	1.083	1.160

C.4 Three pollutant modelling

In models including $PM_{2.5}$, $PM_{2.5-10}$ and NO_2 concentrations, associations between $PM_{2.5}$, NO_2 and mortality were strengthened, while $PM_{10-2.5}$ had an apparent negative effect (shown by the RRs<1.0 in Table 40).

Table 40: Poisson regression model of all adult mortality including $PM_{2.5}$ $PM_{10-2.5}$ and NO_2

Pollutant	Relative Risk (per 10 µg/m³)	Lower CI	Upper CI
NO_2	1.123	1.098	1.149
$PM_{2.5}$	1.125	1.085	1.167
$PM_{10-2.5}$	0.879	0.837	0.924

Similar results were obtained when this model was run for Auckland urban area alone; but the association with $PM_{10-2.5}$ was weaker and no longer significant following adjustment for potential confounders (not shown).

In analyses restricted to specific ethnic groups, associations with $PM_{2.5}$ were significantly stronger among Māori and Pacific peoples compared to other ethnicities (Table 41). Again, $PM_{10-2.5}$ had an apparent negative effect on mortality. Since this is implausible, it is not clear how these models should be interpreted.

Table 41: Poisson regression models of mortality including $PM_{2.5}$ $PM_{2.5-10}$ and NO_2, by ethnicity

Ethnicity	Pollutant	Relative Risk (per 10µg/m³)	Lower CI	Upper CI
Māori	NO_2	1.011	0.934	1.095
	$PM_{2.5}$	1.420	1.234	1.634
	$PM_{10-2.5}$	0.836	0.707	0.989
Pacific peoples	NO_2	1.239	1.071	1.434
	$PM_{2.5}$	1.647	1.149	2.361
	$PM_{10-2.5}$	0.622	0.400	0.967
Asian	NO_2	1.164	1.004	1.350
	$PM_{2.5}$	0.730	0.473	1.125
	$PM_{10-2.5}$	0.784	0.504	1.219
All other ethnicities	NO_2	1.124	1.097	1.153
	$PM_{2.5}$	1.097	1.056	1.140
	$PM_{10-2.5}$	0.890	0.845	0.939

C.5 Shape of ERF at low concentrations

When pollutants were classified in quintiles, mortality from non-external causes was positively associated with $PM_{2.5}$ in the upper two quintiles and with NO_2 in the upper four quintiles. The shape of the association between mortality and NO_2 (but not $PM_{2.5}$) appeared supra-linear.

Consistent with a supra-linear association between mortality and NO_2, in models restricted to pollutant concentrations below or equal to the median the association between mortality and NO_2 was substantially strengthened (Table 42).

Table 42: Comparison of the final model with one restricted to CAUs with pollutant concentrations below or equal to the median

Model	Pollutant	Relative Risk (per 10µg/m³)	Lower CI	Upper CI
Two pollutant model: based on 2016 for all CAUs regardless of concentration	NO_2	1.097	1.074	1.120
	$PM_{2.5}$	1.105	1.065	1.145
Two pollutant model: based on 2016 for CAUs below national median concentrations for both pollutants	NO_2	1.973	1.453	2.678
	$PM_{2.5}$	0.759	0.449	1.286

Appendix D: VoSL and age

This appendix reviews the literature relevant to the age-VoSL relationship in more detail.

D.1 Theoretical models

VoSL declining with age has a simple logic: there is greater value in reducing the risk of death for a young person because there are more life years to protect (Adler *et al* 2019; Bognar 2015; Viscusi 2009). When an individual reveals their WTP to reduce their own death (and we discuss below whether this is the best way to derive or consider VoSL), the assumption is that the WTP reflects the product of the change in the risk of death and the (discounted) aggregate of the future wellbeing they anticipate. Under simplifying assumptions reflecting an economic ideal[71] this would mean WTP for mortality risk reduction would peak at birth and decline throughout life (Aldy & Viscusi 2007, 2008).

A greater value for a longer life assumes people have positive wellbeing throughout their lives[72] so the more life years, the more (cumulative) wellbeing.[73] The present value (PV) of future wellbeing would fall with age, as would the WTP for mortality risk reduction and VoSL. The only way to approximate a constant PV of future wellbeing with age (and thus a constant VoSL) is with a very high discount rate (so that effectively only immediate wellbeing is considered).

D.1.1 Does wellbeing vary with age?

WTP is often used as a measure of wellbeing received, but it may not be so simple. People's WTP may change over their lifetime because of factors that include income and life circumstances. Viscusi (2009) suggests the theoretical outcome of WTP peaking while young and then declining may not be seen in practice because of:

- imperfect information, e.g. about how much income a person might earn in the future or their expected lifespan
- limited access to capital, such as their ability to borrow against future income
- life circumstances, e.g. whether a person has a partner or children and therefore who else is affected by a death

[71] The theoretical world would include rational individuals, perfect information (e.g. about future income and the length of life) and unconstrained access to capital (such as ability to borrow against future income or to purchase annuities to fund consumption in old age). In the theoretical market, people borrow and save to ensure they consume the same amount during every year of life (Aldy & Viscusi 2007).

[72] Shepherd and Zeckhauser (1984) suggest "living is a generally enjoyable activity for which consumers should be willing to sacrifice other pleasures, such as consumption" and that a longer life provides more opportunities for wellbeing enhancement. This is consistent with findings of life satisfaction surveys which show positive wellbeing for all age groups, in New Zealand (https://www.stats.govt.nz/information-releases/wellbeing-statistics-2018) and elsewhere (e.g. Bowling 2011).

[73] A person may not think in these terms, but implicitly a calculation of this nature is assumed to occur.

- income.

Shepard and Zeckhauser (1984) estimate the change in WTP with age using theoretical models with and without access to capital markets; they use future income and wealth (and therefore consumption) as the basis for a discounted future value of life. Without the possibility of transferring money from one time period to another,[74] WTP (and VoSL) follows an inverted 'U' (or hump) shape peaking in mid-life; the shape follows the pattern of income and consumption over the life of an individual. Aldy and Smyth (2014) similarly developed a theoretical model which produced an inverted 'U' shape. In their analysis, the shape is driven particularly by people's inability to insure against income shocks, rather than constraints on borrowing.

D.2 Empirical results

Unlike the theoretical models, empirical studies have produced more mixed results (Johansson 2002), including positive and negative correlations with age, no relationship, in addition to the inverted 'U' shape.

The original 1991 New Zealand WTP study to establish a VoSL found a statistically significant lower WTP for those aged 60 and over (Miller & Guria 1991). Jones-Lee *et al* (1985) identified an inverted 'U' relationship in a 1982 stated preference (SP) survey in the UK. Pearce (2000) reviewed the available studies by Jones-Lee and others and concluded that a reasonable working hypothesis would be that older age groups (over 70 years old) at risk from air pollution have WTPs of a third to three quarters of the 'mean' WTP. The European Commission (2021) came to similar conclusions, suggesting that there are

> strong theoretical and empirical grounds for believing that the value for preventing a fatality declines with age.

They suggested a VoSL adjustment of 0.7 for air pollution because of its greater impact on older people. At the same time, the US EPA used the UK (Jones-Lee *et* al 1985) results as the basis for a 37% "senior death discount" to VoSL for adults over 65 (US EPA 2002), before this approach was rejected after protest objections particularly from elderly citizen groups (see discussion in Laughland *et al* 2007 and Viscusi 2009). Krupnick (2007) suggests

> the senior discount effect, if it exists, is not robust.

However, he also notes that

> qualitative meta-analysis suggests that larger samples are associated with finding a senior discount

and that alternative specifications for age might have found age effects.

[74] They termed this the Robinson Crusoe case, i.e. an individual is limited to their own resources (wealth and earnings) and cannot borrow, e.g. against future expected income.

More recently, Robinson *et al* (2018) suggest that

> values for working age adults may follow an inverse 'U' pattern that peaks in middle-age, and values at older ages may remain constant, increase, or decrease. However, the results across studies are inconsistent and raise questions about the robustness of these findings.

They note that several studies suggest that WTP for reduced current morbidity or mortality risks to children may be noticeably greater (perhaps by a factor of two) than adult WTP to reduce their own risks, although the magnitude of the difference varies across studies.

Other SP studies have found an inverted-U relationship, e.g. Cameron *et al* (2010), but rates of increase and decrease and the age at which VSL peaks vary significantly across studies, and some studies have found no statistically significant relationships between VoSL and age, while others find that the VoSL increases or decreases among older individuals in varying patterns and amounts (Robinson *et al* 2018).

O'Brien (2018) summarises the results of several revealed preference (RP)[75] and SP studies. He notes that RP studies "generally support a flat or inverted-U shaped age-VSL relationship" and that SP studies

> typically show flat, slightly decreasing, or weakly inverted-U shaped age-VSL estimates.

His own RP study used vehicle purchase decisions to estimate a significant inverted-U shape to the age-VoSL function.

The OECD (2012) reviewed the SP literature and concluded that the results were too ambiguous to adjust the VoSL for adult age, but that if a regulation is targeted on reducing children´s risk, VoSL should be a factor of 1.5 – 2.0 higher than adult VSL.[76]

D.3 Arguments for VoSL declining with age

Arguments against WTP and VoSL declining with age, based on the empirical results and adjusted theoretical models, ignore two issues:

1. The results reflect a private rather than public estimate of WTP, and the latter may be more appropriate for use in public policy.
2. The presence of market failures including information gaps, which affect the WTP. This includes the effects of income when our interest is in wellbeing, independent of income.

Below we discuss these issues, turning first to the issue of private versus public perspectives for analysis.

---

[75] These studies include hedonic wage (HW) studies which identify values revealed by wage-risk trade-offs (estimates of worker compensation required for them to work in higher risk jobs), quantile wage (QW) studies which are a type of HW study and hedonic vehicle price (HVP) studies which examine the vehicle price-risk trade-off.

[76] The economic analysis in OECD (2016) also used VoSL only.

D.3.1 Public or private values

A private perspective measures a person's WTP to reduce the risk of their own death and (in an imperfect market) this is expected to increase with income, wealth and maturity. In contrast, a public perspective is the WTP for reduced risk of death in the community at large.

Most stated preference (SP) studies to derive VoSL have used an individual (or private) perspective, but it is not clear that this is appropriate (Abelson 2008; Andersson *et al* 2019; Sagoff 1988). People often appear to have different preferences when acting as a citizen rather than as a consumer (Arrow 1950; Marglin 1963; Ackerman & Heinzerling 2002), and as Svensson & Johansson (2010) suggest, in cases when the intended use is for public policy purposes, the WTP eliciting scenario should be public. In analysing the VoSL-age relationship, the interest is in the WTP for a reduction in the risk of death for an average person (of different ages).

When considering the average, it might be expected that the potential (financial and other) future positive contribution of people of different ages to the community would be relevant, in addition to any altruistic considerations for other people's potential lifetime wellbeing, offset by the potential costs to the community of people of different ages, e.g. for the education of children and the care of the elderly.

Surveys have been used to analyse public preferences for saving the lives of people of different ages (Bognar 2008), including:

- an Australian study in which respondents considered extending the lives of four 20-year-olds as equivalent to extending the lives of ten 60-year olds by the same amount; (Nord *et al* 1991)
- a US study that found saving one 20-year-old equivalent to saving seven 60-year-olds (Cropper *et al* 1994)
- a Swedish study in which median respondent is indifferent between saving the life of one 30-year-old, five 50-year olds, and thirty-four 70-year olds (Johannesson & Johansson 1997)
- a UK study in which one year in full health for a 50-year-old is valued the same as 10 years for a 70-year-old (Dolan & Tsuchiya 2012).

Bognar (2008) noted that, in the US and Swedish studies, the age of the respondents had no effect on their preferences, i.e. people seem to prefer saving the younger person regardless of their own age.

The studies do not all suggest a simple declining value (or preference) with age. Cropper *et al* (1994) found reducing fatalities for 20-year-olds and 40-year-olds was valued similarly, and that a saved life has its highest value around age 30.[77] They also found most respondents gave greater weight to life years saved per person than the total life years saved, so they would prefer programmes in which a smaller number of young people were saved than a large number of old people, and by implication favour life years saved over lives. Lewis and Charny (1989) find people have a very strong preference for who should be treated for leukaemia

[77] Saving eight 60-year-olds was judged equivalent to saving one 20-year-old; saving eleven 60-year-olds was judged equivalent to saving one 30-year-old; and saving seven 60-year-olds was judged equivalent to saving one 40-year-old.

given limited resources;[78] respondents favoured saving the lives of five-year olds over 70-year-olds, had a strong preference for 35-year-olds over 60-year-olds and a slight preference for eight-year olds over two-year-olds.[79]

Rather than assuming people operate with a utilitarian basis for preferring to reduce fatality risks for younger people, preferences may reflect notions of fairness, such as the "fair innings" concept from public health literature. This is that

> there is some span of years that we consider a reasonable life, a fair innings … everyone [should] be given an equal chance to have a fair innings, to reach the appropriate threshold but, having reached it, they have received their entitlement. The rest of their life is the sort of bonus which may be cancelled when this is necessary to help others reach the threshold.[80]

The issue is not that the "fair innings" concept is a useful decision criterion[81] but that it might explain (in part at least) public preferences for mortality risk reduction for the young (Adler *et al* 2019). However, there are other potential principles that might also determine public preferences, including the injustice of death at any age (Harris 1985 p91).[82]

These arguments all take a public approach, i.e. they are the considerations that are included when we ask the question for the community as a whole, rather than an individual's own life. The results are not unambiguous but tend to suggest that reducing the risks of death is valued more highly for a young person than for an older person.

The argument of falling value with age is simpler to use as the basis for adjusting the VoSL of young people upwards than it is to justify a reduction in the VoSL (a senior discount) that otherwise would be based on the WTP of the older people themselves. This is to argue that that a person's life is worth less to society than it is to them.[83] Simpler still is to use a VoLY.

D.3.2 Market failure

In addition to the community versus private basis for analysis as a reason for suggesting VoSL may differ with age, another argument is from the perspective of market failure, which may explain the differences between the theoretical and empirical results. If the WTP results reflect significant market failures, they do not provide useful guidance for public policy purposes (Sunstein 2013).

---

[78] See interpretation by Dolan *et al* (2005).

[79] Lewis and Charny (1989) note that, when they questioned respondents on why they favoured medical interventions to save an eight-year-old over a two-year-old, this included statements such as "*to protect the investments that have already been made*". Whether knowingly or not, this is effectively saying a young child has both a long potential life ahead with the potential to contribute to a society, but an initial net annual cost for the community when raising that child.

[80] See also Williams (1997)

[81] See Rivlin (2000) for ethical and other objections

[82] This injustice argument is used by Bognar (2008) in suggesting the *Anti-Ageist Principle* that each person should have an equal claim for a lifesaving resource (and thus to fatality risk reduction), even if some person's prospect with respect to the quality or length of their life is worse than the prospects of others.

[83] As suggested by the anti-aging movement, for example (see e.g. Vincent 2012)

Market failures are defined from a particular perspective, which is that resources are used optimally when they maximise the wellbeing of the whole community. Markets are said to fail when they do not maximise wellbeing, given available resources. When estimating non-market values, analysts will often use stated preferences in hypothetical markets, and these can be subject to market failures in the same way as actual markets. For example, a young survey respondent expressing how much they value a reduction in the risk of their own death will not know how much they will enjoy life in the future and what their income level will be; they will also be constrained by their current income in expressing their WTP. Some of the studies discussed above have developed theoretical models to take account of information and income limitations (e.g. Shepard & Zeckhauser 1984; Aldy & Smith 2014), but this is adjusting a theoretical model to mimic a market in which market failures exist. For public policy purposes we are better served by a theoretical model without market failure, i.e. the theoretical ideal with full information and efficient capital markets.

D.3.3 Income effects

Theoretical models and empirical studies have found that WTP increases with income. However, when we are defining values based on WTP in public policy decisions, the monetary values are used as a proxy for wellbeing. An individual's WTP is assumed to reflect what they would give up to obtain more of something else; in this case a lower risk of death. At any point in time this can provide an accurate picture of relative values and thus the relative contribution to wellbeing, e.g. lowering the risk of a fatal accident versus a faster trip.[84] But it can fail when comparing relative wellbeing across the population or for an individual over time.

Because WTP reflects ability to pay, the preferences of the rich might be given greater weight than those of the poor. Very often this does not matter, e.g. if assessments are being made for a population as a whole and population average values are used. However, if the effects are on a particular sector or location with income levels different from the average (and assuming we care equally about the wellbeing of all people), adjustments can be made to account for income (Adler 2016; NZ Treasury 2015). This same kind of correction is valid for comparing the preferences and WTP of a person over their lifetime when they have different levels of income. Even this may fail to take account of all the preferences which may not be expressed via an increase in available money. For example, the value to children of playing, or to the elderly of talking with friends (Australian Safety and Compensation Council 2008). However, the simple conclusion is that it makes no sense to use ability to pay as the basis for adjusting WTP with age when our interest is in relative wellbeing over time.

As cited in HAPINZ 2.0, Mason *et al* (2009) comment on the finding of an inverted U-shape in empirical studies when income is controlled for (e.g. by Jones-Lee *et al* 1985) and note that, at least over early years of adult life, value increases as life expectancy falls. They suggest this is because of increasing personal maturity resulting in a

> fundamental change in preferences and attitudes rather than a change in an individual's future hazard rates.

[84] This is the approach taken in many studies used to define VoSL. See for example Rizzi & Ortúzar 2003; 2006a; 2006b; Hensher *et al* 2009; Hensher *et al* 2011; Iragüen & Ortúzar 2004.

Given this, they suggest valuation should be based only on the time interval over which the VPF is a decreasing function of age. Kuschel *et al* (2012c) disagree, suggesting that the value per QALY changes with age, as suggested by Aldy and Viscusi (2008). However, as noted above, we are not trying to mimic the valuation of an individual with limited information, but to optimise decision making given full information, as is available to a decision maker making decisions on behalf of the population as a whole and with the knowledge of population averages.

When taking a societal perspective, with full information about patterns of preference over time, it is reasonable to assume the value of reduced risk of death when young is at least as high as the discounted value of reduced risk of death when older. This is consistent with using changes in life years and VoLY for analysis.

Appendix E: Scenario modelling uncertainty

This appendix provides more detailed results of the assessment undertaken to test the accuracy of predictions made using population and pollutant scalars to run scenarios in the *HAPINZ 3.0 Health Effects Model*.

E.1 Population scalars

The accuracy of using estimated resident population as the population scalar was tested for two different prediction timeframes to 2016 (the HAPINZ 3.0 base year):

- two years from 2014 to 2016 using annual data averaged over three-years
- ten years from 2006 to 2016 using annual data averaged over three-years.

E.1.1 Baseline mortality

Two-year predictions

We used the annual estimated resident populations for 2013-2015 relative to 2015-2017 to calculate the annual average scalars shown in Table 43. The resulting national mortality predictions are shown in Table 44, by TA in Table 45, with the CAU error distribution presented in Figure 31.

Table 43: Scalars used to predict 2015-2017 annual average mortality based on 2013-2015

Population	National data (Stat NZ estimates) of estimated resident population		% change	Scalar
	2013-2015	2015-2017		
Total population	4,522,667	4,712,367	4.2%	1.04194
30+ population	2,677,217	2,792,357	4.3%	1.04301
55+ population	1,173,447	1,246,597	6.2%	1.06234
60+ population	893,560	948,807	6.2%	1.06183

Table 44: Observed vs predicted 2015-2017 annual average mortality based on 2013-2015

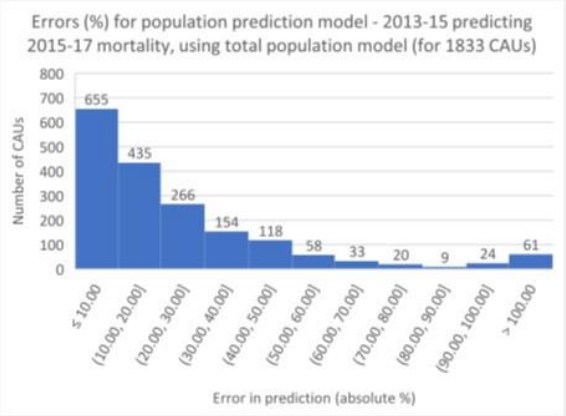
Population	Annual average deaths (2015-2017)		Difference (Observed-Predicted)	Error (%)
	Observed	Predicted		
Total population	29,690	29,684	6	0.0%
30+ population	29,690	29,676	14	0.0%
55+ population	29,690	30,211	-521	-1.8%
60+ population	29,690	30,193	-503	-1.7%

Table 45: Observed vs predicted 2015-2017 annual average mortality by TA based on 2013-2015

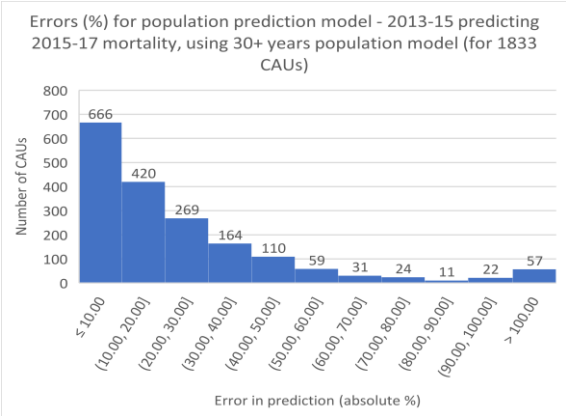
Base year 2013-15, scalar		Predicted deaths 2015-17 - scalar model				Differences (observed-predicted)				Errors (%)			
Row Labels	Observed deaths 2015-17	Total pop model	30+ model	55+ years model	60+ years model	Total pop model	30+ model	55+ model	60+ model	Total model	30+ model	55+ model	60+ model
New Zealand	**29,690**	**29,684**	**29,676**	**30,211**	**30,193**	6	14	-521	-503	0.0	0.0	-1.8	-1.7
Far North District	530	495	495	504	504	35	35	26	26	6.5	6.6	4.9	4.9
Whangarei District	699	683	683	695	695	15	16	3	4	2.2	2.2	0.5	0.5
Kaipara District	176	161	161	164	164	16	16	13	13	8.8	8.9	7.2	7.3
Auckland	7,633	7,653	7,651	7,789	7,785	-20	-17	-156	-151	-0.3	-0.2	-2.0	-2.0
Thames-Coromandel District	306	296	296	301	301	10	10	5	5	3.4	3.4	1.6	1.7
Hauraki District	185	174	174	177	177	11	11	8	8	5.8	5.9	4.2	4.2
Waikato District	375	373	372	379	379	2	2	-4	-4	0.6	0.6	-1.2	-1.1
Matamata-Piako District	266	267	267	272	272	-2	-1	-6	-6	-0.6	-0.5	-2.4	-2.3
Hamilton City	847	818	818	833	832	28	29	14	14	3.4	3.4	1.6	1.7
Waipa District	372	357	357	363	363	15	15	9	9	4.0	4.1	2.3	2.4
Otorohanga District	51	50	50	51	51	1	1	0	0	1.8	1.8	0.0	0.1
South Waikato District	186	186	186	189	189	0	0	-4	-4	-0.3	-0.2	-2.0	-2.0
Waitomo District	69	75	75	76	76	-6	-6	-8	-8	-9.4	-9.4	-11.4	-11.3
Taupo District	268	282	282	287	287	-14	-14	-19	-19	-5.2	-5.1	-7.0	-7.0
Western Bay of Plenty District	353	352	352	359	359	1	1	-5	-5	0.3	0.3	-1.5	-1.5
Tauranga City	1,067	1,023	1,023	1,041	1,040	44	44	26	27	4.1	4.1	2.4	2.5
Rotorua District	508	505	505	514	514	3	3	-6	-6	0.6	0.6	-1.1	-1.1
Whakatane District	278	271	271	275	275	8	8	3	3	2.7	2.8	1.0	1.1
Kawerau District	63	61	61	62	62	2	2	1	1	3.4	3.4	1.6	1.7
Opotiki District	82	84	84	85	85	-2	-2	-3	-3	-2.3	-2.2	-4.1	-4.0
Gisborne District	354	362	362	369	368	-8	-8	-15	-14	-2.3	-2.3	-4.1	-4.1
Wairoa District	79	73	73	75	75	6	6	5	5	7.5	7.5	5.8	5.9
Hastings District	612	595	595	606	606	17	17	6	7	2.8	2.8	1.1	1.1
Napier City	555	555	554	564	564	0	1	-9	-9	0.1	0.1	-1.7	-1.6
Central Hawke's Bay District	101	94	94	95	95	8	8	6	6	7.6	7.7	6.0	6.1
New Plymouth District	654	648	648	660	659	6	6	-6	-5	0.9	0.9	-0.9	-0.8
Stratford District	79	74	74	75	75	5	5	4	4	6.6	6.6	4.9	5.0
South Taranaki District	207	202	202	206	206	5	5	1	1	2.3	2.4	0.6	0.7
Ruapehu District	88	90	90	91	91	-1	-1	-3	-3	-1.6	-1.6	-3.4	-3.4
Wanganui District	474	466	466	474	474	8	8	0	0	1.6	1.7	-0.1	0.0
Rangitikei District	104	109	109	110	110	-5	-5	-7	-7	-5.0	-5.0	-6.6	-6.5
Manawatu District	227	198	198	202	202	28	28	25	25	12.5	12.5	11.0	11.0
Palmerston North City	537	556	555	566	565	-18	-18	-28	-28	-3.4	-3.4	-5.2	-5.2
Tararua District	152	147	147	150	150	4	5	2	2	3.0	3.0	1.2	1.3
Horowhenua District	371	367	367	374	374	4	4	-3	-3	1.0	1.0	-0.8	-0.7
Kapiti Coast District	586	566	566	576	576	20	20	10	10	3.3	3.4	1.6	1.7
Porirua City	286	285	285	290	289	1	1	-4	-3	0.5	0.5	-1.3	-1.2
Upper Hutt City	317	317	317	323	322	0	0	-5	-5	0.1	0.1	-1.7	-1.6
Lower Hutt City	621	630	630	641	641	-9	-9	-20	-20	-1.5	-1.4	-3.3	-3.2
Wellington City	830	838	838	853	853	-8	-8	-23	-23	-1.0	-1.0	-2.8	-2.8
Masterton District	245	255	255	259	259	-10	-10	-14	-14	-3.9	-3.9	-5.8	-5.7
Carterton District	68	67	67	68	68	1	1	0	0	1.3	1.3	-0.5	-0.4
South Wairarapa District	75	79	79	80	80	-4	-4	-5	-5	-5.3	-5.3	-7.2	-7.1
Tasman District	349	350	350	356	356	-1	-1	-7	-7	-0.2	-0.2	-2.0	-1.9
Nelson City	437	416	416	423	423	21	21	14	14	4.8	4.8	3.1	3.1
Marlborough District	388	389	389	396	396	-2	-2	-9	-8	-0.4	-0.4	-2.2	-2.2
Kaikoura District	29	25	25	25	25	4	4	3	3	12.6	12.6	11.1	11.1
Buller District	93	101	101	102	102	-8	-8	-9	-9	-8.1	-8.1	-10.0	-10.0
Grey District	111	112	112	114	114	-1	-1	-3	-3	-0.9	-0.9	-2.7	-2.7
Westland District	64	59	59	60	60	5	5	4	4	8.1	8.1	6.4	6.5
Hurunui District	73	79	79	80	80	-6	-6	-8	-8	-8.7	-8.7	-10.6	-10.6
Waimakariri District	351	348	347	354	352	4	4	-2	0	1.1	1.1	-0.7	-0.1
Christchurch City	2,627	2,717	2,716	2,766	2,764	-91	-90	-139	-137	-3.4	-3.4	-5.3	-5.2
Selwyn District	159	144	144	147	147	15	15	12	12	9.2	9.2	7.6	7.6
Ashburton District	243	242	242	247	247	0	0	-4	-4	0.1	0.1	-1.7	-1.7
Timaru District	434	458	458	466	466	-24	-24	-32	-32	-5.5	-5.5	-7.4	-7.3
Mackenzie District	19	27	27	27	27	-8	-8	-8	-8	-41.0	-41.0	-43.5	-43.4
Waimate District	71	75	75	76	76	-4	-4	-6	-6	-6.3	-6.3	-8.2	-8.2
Waitaki District	206	239	239	243	243	-33	-33	-37	-37	-15.9	-15.8	-17.9	-17.9
Central Otago District	136	142	142	145	145	-6	-6	-8	-8	-4.4	-4.3	-6.2	-6.2
Queenstown-Lakes District	94	100	99	101	101	-6	-5	-7	-7	-5.9	-5.8	-7.7	-7.7
Dunedin City	943	992	992	1,010	1,009	-49	-49	-67	-66	-5.2	-5.2	-7.1	-7.0
Clutha District	121	123	123	125	125	-2	-2	-4	-4	-1.5	-1.5	-3.3	-3.3
Southland District	149	152	152	155	155	-3	-3	-6	-5	-2.0	-2.0	-3.9	-3.6
Gore District	130	128	128	130	130	2	2	0	0	1.8	1.8	0.0	0.1
Invercargill City	523	522	522	532	531	1	1	-9	-8	0.1	0.2	-1.6	-1.6
Chatham Islands Territory	5	5	5	5	5	0	0	0	0	2.6	2.6	0.9	0.9

Figure 31: Errors (%) in predicting 2015-2017 annual average mortality by CAU based on 2013-2015 for (a) a total population model and (b) a 30+ years population model

(a) total population model



(b) 30+ years model



Ten-year predictions

For this analysis, we used the annual estimated resident populations for 2005-2007 relative to 2015-2017 to calculate the annual average scalars shown in Table 46. The resulting national mortality predictions are shown in Table 47, by TA in Table 48, with the CAU error distribution presented in Figure 32.

Table 46: Scalars used to predict 2015-2017 annual average mortality based on 2005-2007

Population	National data (Stat NZ estimates) of estimated resident population		% change	Scalar
	2005-2007	2015-2017		
Total population	4,184,600	4,712,367	12.6%	1.12612
30+ population	2,431,180	2,792,357	14.9%	1.14856
55+ population	941,290	1,246,597	32.4%	1.32435
60+ population	698,280	948,807	35.9%	1.35878

Table 47: Observed vs predicted 2015-2017 annual average mortality based on 2005-2007

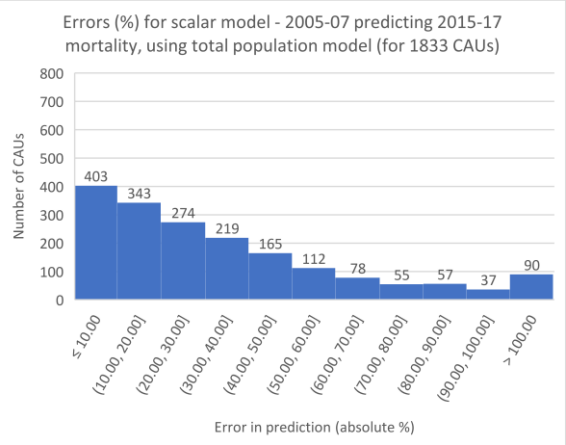
Population	Annual average deaths (2015-2017)		Difference (Observed-Predicted)	Error (%)
	Observed	Predicted		
Total population	29,690	28,885	805	2.7%
30+ population	29,690	29,461	229	0.8%
55+ population	29,690	33,948	-4,258	-14.3%
60+ population	29,690	34,816	-5,126	-17.3%

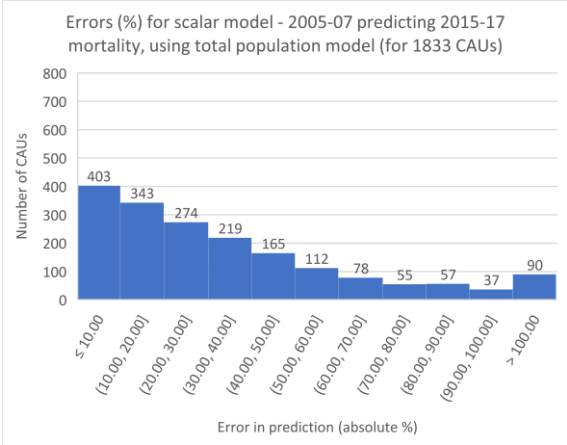
Table 48: Observed vs predicted 2015-2017 annual average mortality by TA based on 2005-2007

Base year 2005-07, scalar		Predicted deaths 2015-17 - scalar model				Differences (observed-predicted)				Errors (%)			
Territorial authority	Observed deaths 2015-17	Total pop model	30+ model	55+ years model	60+ years model	Total pop model	30+ model	55+ model	60+ model	Total model	30+ model	55+ model	60+ model
Total New Zealand	**29,690**	**28,885**	**29,461**	**33,948**	**34,816**	**805**	**229**	**-4258**	**-5126**	**2.7**	**0.8**	**-14.3**	**-17.3**
Far North District	530	450	459	530	544	79	70	0	-14	15.0	13.3	0.0	-2.6
Whangarei District	699	614	627	722	741	84	72	-24	-42	12.0	10.3	-3.4	-6.1
Kaipara District	176	145	147	170	174	32	29	6	2	18.0	16.4	3.6	1.1
Auckland	7,633	7,344	7,490	8,632	8,849	289	143	-998	-1215	3.8	1.9	-13.1	-15.9
Thames-Coromandel District	306	290	296	341	350	16	10	-35	-44	5.2	3.3	-11.5	-14.4
Hauraki District	185	160	163	188	193	25	22	-3	-8	13.5	11.8	-1.7	-4.3
Waikato District	375	325	332	383	393	49	43	-8	-18	13.1	11.4	-2.1	-4.8
Matamata-Piako District	266	256	261	301	309	10	5	-35	-43	3.6	1.7	-13.3	-16.3
Hamilton City	847	819	835	963	988	28	11	-117	-142	3.3	1.3	-13.8	-16.7
Waipa District	372	339	346	399	409	33	26	-27	-37	8.8	7.0	-7.3	-10.0
Otorohanga District	51	50	51	58	60	1	0	-7	-9	2.8	0.9	-14.3	-17.2
South Waikato District	186	169	172	199	204	17	13	-13	-18	9.0	7.2	-7.0	-9.8
Waitomo District	69	74	75	87	89	-5	-6	-18	-20	-7.1	-9.3	-26.0	-29.3
Taupo District	268	234	239	274	280	34	29	-6	-12	12.7	11.0	-2.3	-4.6
Western Bay of Plenty District	353	316	322	371	381	38	31	-18	-28	10.7	8.9	-5.1	-7.8
Tauranga City	1,067	980	999	1,151	1,181	87	68	-84	-114	8.2	6.3	-7.9	-10.7
Rotorua District	508	498	508	586	601	10	0	-77	-93	1.9	0.0	-15.2	-18.2
Whakatane District	278	267	272	313	321	12	7	-35	-43	4.2	2.3	-12.6	-15.4
Kawerau District	63	47	47	55	56	16	16	8	7	26.1	24.6	13.1	10.9
Opotiki District	82	87	88	102	105	-5	-6	-20	-23	-5.7	-7.9	-24.4	-27.6
Gisborne District	354	408	417	480	493	-54	-63	-126	-139	-15.4	-17.7	-35.7	-39.2
Wairoa District	79	68	70	80	82	11	10	-1	-3	13.9	12.2	-1.3	-3.9
Hastings District	612	596	608	701	720	16	4	-89	-107	2.6	0.6	-14.6	-17.5
Napier City	555	548	559	644	661	7	-4	-89	-106	1.3	-0.6	-16.0	-19.1
Central Hawke's Bay District	101	100	102	117	120	1	-1	-16	-19	1.5	-0.5	-15.9	-18.9
New Plymouth District	654	639	652	752	771	15	2	-98	-117	2.3	0.3	-15.0	-17.9
Stratford District	79	86	87	101	103	-6	-8	-21	-24	-7.9	-10.0	-26.9	-30.2
South Taranaki District	207	216	221	254	261	-9	-14	-47	-54	-4.5	-6.5	-22.8	-26.0
Ruapehu District	88	102	105	121	123	-14	-16	-32	-34	-16.0	-18.3	-36.4	-39.0
Wanganui District	474	468	477	550	564	6	-3	-76	-91	1.3	-0.7	-16.1	-19.1
Rangitikei District	104	113	116	133	137	-10	-12	-30	-33	-9.4	-11.5	-28.6	-31.9
Manawatu District	227	197	201	232	238	30	26	-5	-11	13.1	11.3	-2.2	-4.9
Palmerston North City	537	540	551	635	652	-3	-14	-98	-114	-0.5	-2.5	-18.2	-21.3
Tararua District	152	151	154	177	182	1	-2	-25	-30	0.8	-1.2	-16.7	-19.8
Horowhenua District	371	354	361	416	427	17	10	-45	-56	4.6	2.7	-12.2	-15.1
Kapiti Coast District	586	522	532	614	630	64	54	-28	-44	11.0	9.2	-4.7	-7.4
Porirua City	286	252	257	296	303	34	29	-10	-17	11.9	10.2	-3.4	-5.9
Upper Hutt City	317	278	283	327	333	40	34	-9	-16	12.5	10.7	-2.9	-5.0
Lower Hutt City	621	651	663	764	784	-30	-42	-143	-163	-4.8	-6.8	-23.0	-26.2
Wellington City	830	888	905	1,039	1,066	-58	-75	-209	-236	-7.0	-9.1	-25.2	-28.4
Masterton District	245	233	238	274	281	12	7	-29	-36	4.9	3.0	-11.9	-14.8
Carterton District	68	62	63	72	74	6	5	-5	-7	9.0	7.2	-7.0	-9.8
South Wairarapa District	75	64	65	75	77	11	10	0	-2	14.9	13.2	-0.1	-2.7
Tasman District	349	337	344	396	406	12	6	-47	-57	3.5	1.6	-13.4	-16.3
Nelson City	437	388	395	456	468	49	41	-19	-31	11.2	9.4	-4.4	-7.1
Marlborough District	388	390	397	458	470	-2	-10	-71	-82	-0.5	-2.5	-18.2	-21.3
Kaikoura District	29	27	28	32	33	1	1	-4	-4	4.4	2.5	-12.4	-15.3
Buller District	93	82	84	97	99	11	9	-4	-6	11.6	9.8	-4.0	-6.7
Grey District	111	102	105	121	124	8	6	-10	-13	7.4	5.6	-8.9	-11.7
Westland District	64	71	72	83	85	-7	-8	-19	-20	-10.3	-12.5	-29.7	-31.7
Hurunui District	73	73	74	86	88	0	-2	-13	-15	-0.2	-2.2	-17.9	-20.9
Waimakariri District	351	276	282	319	327	75	70	33	24	21.4	19.8	9.3	6.9
Christchurch City	2,627	2,885	2,942	3,393	3,481	-258	-316	-766	-854	-9.8	-12.0	-29.2	-32.5
Selwyn District	159	127	129	149	153	32	30	10	6	20.2	18.6	6.2	3.7
Ashburton District	243	238	243	280	287	5	0	-37	-44	1.9	0.0	-15.3	-18.3
Timaru District	434	452	461	532	546	-18	-27	-98	-112	-4.2	-6.3	-22.6	-25.8
Mackenzie District	19	18	19	22	22	1	0	-3	-3	3.2	1.3	-13.8	-16.8
Waimate District	71	71	72	83	86	0	-2	-13	-15	-0.4	-2.4	-18.1	-21.1
Waitaki District	206	214	218	251	258	-8	-12	-45	-52	-3.7	-5.7	-21.9	-25.1
Central Otago District	136	137	139	161	165	0	-3	-24	-29	-0.2	-2.2	-17.9	-20.9
Queenstown-Lakes District	94	74	75	87	89	20	19	7	5	21.3	19.8	7.5	5.1
Dunedin City	943	996	1,016	1,170	1,201	-53	-72	-227	-257	-5.6	-7.7	-24.1	-27.3
Clutha District	121	136	138	159	164	-14	-17	-38	-42	-11.7	-13.9	-31.3	-34.8
Southland District	149	144	147	169	173	6	3	-20	-24	3.7	1.8	-13.2	-15.9
Gore District	130	119	122	140	144	11	8	-10	-14	8.2	6.3	-8.0	-10.8
Invercargill City	523	528	539	621	637	-5	-16	-98	-114	-1.0	-3.0	-18.8	-21.8
Chatham Islands Territory	5	3	3	3	3	2	2	2	2	47.4	46.4	38.2	36.6

Figure 32: Errors (%) in predicting 2015-2017 annual average mortality by CAU based on 2005-2007 for (a) a total population model and (b) a 30+ years population model

(a) total population model (b) 30+ years model





E.1.2 Baseline hospitalisations

Two-year predictions

We applied the same scalars shown in Table 43 to cardiovascular (CVHA) and respiratory (RHA) hospital admissions. The resulting national morbidity predictions are shown in Table 49, by TA in Table 50, with the CAU error distribution presented in Figure 33.

Table 49: Observed vs predicted 2015-2017 annual average hospitalisations based on 2013-2015

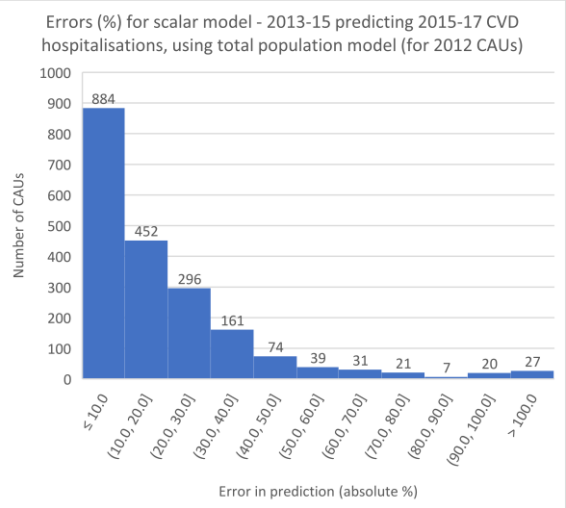
Population	CVHAs in 2015-2017		Error (%)	RHAs in 2015-2017		Error (%)
	Observed	Predicted		Observed	Predicted	
Total population	57,872	57,181	1.2%	70,425	67,841	3.7%

Table 50: Observed vs predicted 2015-2017 annual average hospital admissions by TA based on 2013-2015

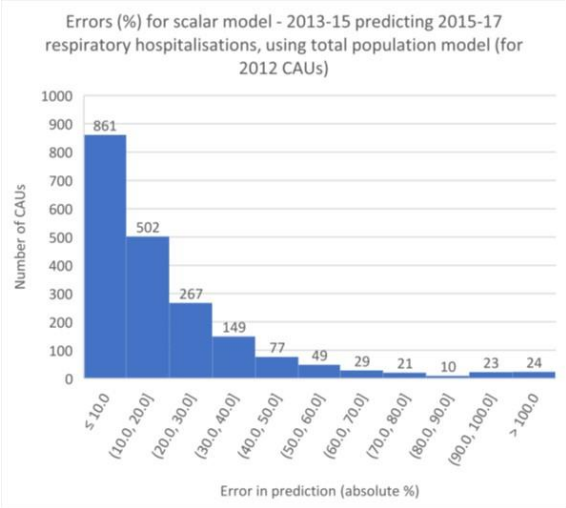
Scalar - 2013-15 to 2015-17	CVD hospitalisations				Respiratory hospitalisations			
Row Labels	Observed CVD hosps 2015-17 (2013-15 model)	Predicted CVD hosps 2015-17 (2013-15 model)	Difference (observed-predicted)	Errors (%)	Observed resp hosps 2015-17 (2013-15 model)	Predicted resp hosps 2015-17 (2013-15 model)	Difference (observed-predicted)	Errors (%)
Total New Zealand	**57,872**	**57,181**	**690**	**1.2**	**70,425**	**67,841**	**2,584**	**3.7**
Far North District	842	859	-17	-2.0	1,022	1,025	-2	-0.2
Whangarei District	1,527	1,480	47	3.1	1,893	1,780	113	6.0
Kaipara District	307	308	-1	-0.5	288	268	20	6.9
Auckland	16,717	16,495	222	1.3	22,712	22,739	-27	-0.1
Thames-Coromandel District	563	533	30	5.4	453	396	57	12.6
Hauraki District	362	332	29	8.1	397	362	35	8.7
Waikato District	828	709	119	14.4	1,149	1,034	114	10.0
Matamata-Piako District	442	408	34	7.6	497	439	58	11.7
Hamilton City	1,787	1,725	62	3.5	3,121	2,828	293	9.4
Waipa District	697	624	73	10.5	728	636	92	12.6
Otorohanga District	103	101	2	1.6	153	113	40	26.2
South Waikato District	406	368	38	9.4	490	398	91	18.6
Waitomo District	130	131	-1	-0.7	172	176	-4	-2.4
Taupo District	466	457	9	2.0	505	462	43	8.5
Western Bay of Plenty District	669	650	18	2.8	727	708	18	2.5
Tauranga City	1,932	1,871	60	3.1	2,329	2,229	100	4.3
Rotorua District	1,069	994	75	7.0	1,666	1,500	167	10.0
Whakatane District	586	607	-22	-3.7	742	738	4	0.6
Kawerau District	130	129	1	0.6	178	162	16	8.9
Opotiki District	156	154	3	1.8	198	176	22	11.1
Gisborne District	558	590	-32	-5.8	885	845	40	4.5
Wairoa District	138	132	6	4.4	134	163	-30	-22.1
Hastings District	1,190	1,176	14	1.2	1,409	1,274	135	9.6
Napier City	1,063	1,106	-42	-4.0	898	856	42	4.6
Central Hawke's Bay District	223	202	21	9.3	235	194	40	17.1
New Plymouth District	1,188	1,160	28	2.4	1,318	1,138	180	13.7
Stratford District	146	123	23	15.8	161	140	21	12.9
South Taranaki District	411	390	21	5.2	569	520	49	8.6
Ruapehu District	196	174	23	11.5	261	291	-31	-11.8
Whanganui District	836	875	-39	-4.7	1,048	984	64	6.1
Rangitikei District	210	236	-26	-12.5	267	235	32	11.8
Manawatu District	430	389	41	9.5	488	448	40	8.1
Palmerston North City	1,022	1,070	-48	-4.7	1,295	1,265	30	2.3
Tararua District	288	264	24	8.5	320	328	-8	-2.4
Horowhenua District	651	672	-22	-3.3	619	611	8	1.3
Kapiti Coast District	1,399	1,247	152	10.9	741	679	62	8.3
Porirua City	607	615	-8	-1.3	987	967	20	2.0
Upper Hutt City	499	499	0	-0.1	609	589	20	3.3
Lower Hutt City	1,195	1,268	-73	-6.1	1,849	1,858	-9	-0.5
Wellington City	1,594	1,651	-57	-3.6	2,163	2,158	4	0.2
Masterton District	409	408	0	0.1	472	453	19	4.1
Carterton District	143	119	25	17.1	119	106	13	10.7
South Wairarapa District	130	132	-2	-1.5	127	118	10	7.5
Tasman District	521	547	-26	-4.9	458	462	-4	-0.8
Nelson City	622	611	11	1.8	577	534	43	7.5
Marlborough District	698	741	-44	-6.2	601	572	30	4.9
Kaikoura District	56	47	9	16.3	43	29	14	32.7
Buller District	168	150	18	10.9	149	138	11	7.2
Grey District	201	204	-3	-1.4	200	171	29	14.4
Westland District	101	108	-7	-7.3	104	90	14	13.5
Hurunui District	130	132	-2	-1.8	108	114	-5	-4.8
Waimakariri District	743	717	27	3.6	688	611	77	11.2
Christchurch City	4,390	4,438	-48	-1.1	4,833	4,815	18	0.4
Selwyn District	463	386	77	16.7	532	404	127	24.0
Ashburton District	395	367	28	7.1	395	366	29	7.3
Timaru District	736	804	-69	-9.3	584	520	64	10.9
Mackenzie District	39	44	-4	-11.3	32	27	5	15.1
Waimate District	126	127	-1	-0.9	92	83	9	9.7
Waitaki District	366	352	15	4.0	271	250	21	7.7
Central Otago District	273	272	1	0.4	190	179	11	5.7
Queenstown-Lakes District	210	205	5	2.4	210	218	-7	-3.5
Dunedin City	1,741	1,878	-137	-7.9	2,111	2,045	66	3.1
Clutha District	219	221	-2	-0.7	223	203	21	9.2
Southland District	325	309	15	4.7	352	342	10	2.8
Gore District	201	200	1	0.6	187	208	-21	-11.2
Invercargill City	898	881	17	1.9	1,089	1,061	28	2.5
Chatham Islands Territory	9	10	-1	-7.9	3	7	-4	-143.1

Figure 33: Errors (%) in predicting 2015-2017 annual average hospitalisations by CAU based on 2013-2015 for (a) a total population model and (b) a 30+ years population model

(a) cardiovascular hospitalisations



(b) respiratory hospitalisations



Ten-year predictions

We applied the same scalars shown in Table 47 to cardiovascular (CVHA) and respiratory (RHA) hospital admissions. The resulting national morbidity predictions are shown in Table 51, by TA in Table 52, with the CAU error distribution presented in Figure 34.

Table 51: Observed vs predicted 2015-2017 annual average hospitalisations based on 2005-2007

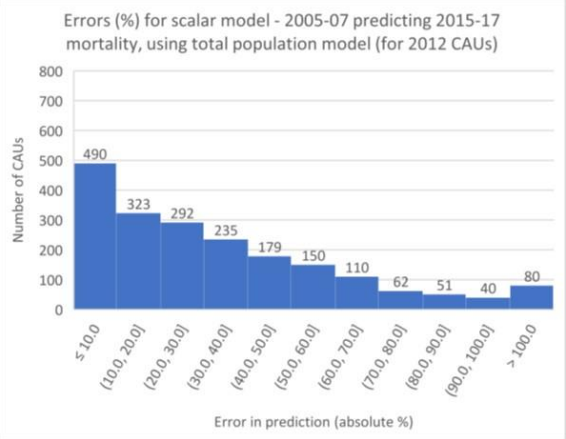
Population	CVHAs in 2015-2017		Error (%)	RHAs in 2015-2017		Error (%)
	Observed	Predicted		Observed	Predicted	
Total population	57,872	57,508	0.6%	70,425	56,748	19.4%

Table 52: Observed vs predicted 2015-2017 annual average hospital admissions by TA based on 2005-2007

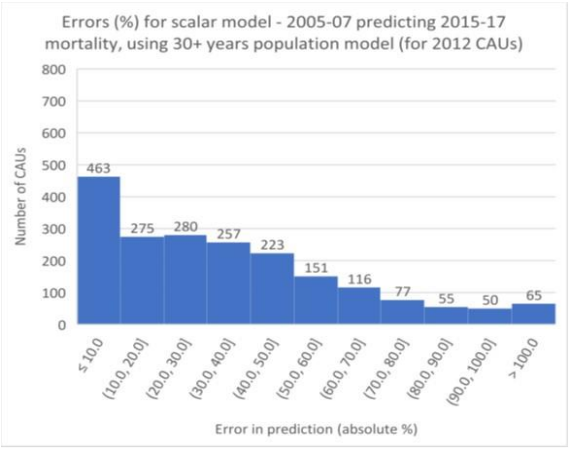
Scalar - 2005-07 to 2015-17	CVD hospitalisations (all ages)				Respiratory hospitalisations (all ages)			
Row Labels	Observed CVD hosps 2015-17 (2005-07 model)	Predicted CVD hosps 2015-17 (2005-07 model)	Difference (observed-predicted)	Errors (%)	Observed resp hosps 2015-17 (2005-07 model)	Predicted Resp hosps 2015-17 (2005-07 model)	Difference (observed-predicted)	Errors (%)
Total New Zealand	**57,872**	**57,508**	**363**	**0.6**	**70,425**	**56,748**	**13677**	**19.4**
Far North District	842	963	-122	-14.4	1,022	946	77	7.5
Whangarei District	1,527	1,490	37	2.4	1,893	1,372	521	27.5
Kaipara District	307	274	32	10.5	288	253	35	12.0
Auckland	16,717	17,208	-491	-2.9	22,712	19,727	2985	13.1
Thames-Coromandel District	563	541	22	3.9	453	272	181	39.9
Hauraki District	362	291	71	19.6	397	231	166	41.8
Waikato District	828	639	189	22.8	1,149	661	487	42.4
Matamata-Piako District	442	441	1	0.2	497	308	190	38.1
Hamilton City	1,787	1,724	63	3.5	3,121	1,772	1349	43.2
Waipa District	697	666	31	4.4	728	462	266	36.5
Otorohanga District	103	90	13	12.3	153	98	55	36.1
South Waikato District	406	386	20	5.0	490	295	195	39.7
Waitomo District	130	149	-19	-14.9	172	155	17	9.6
Taupo District	466	553	-86	-18.5	505	407	98	19.4
Western Bay of Plenty District	669	578	90	13.5	727	509	218	30.0
Tauranga City	1,932	1,848	84	4.4	2,329	1,696	633	27.2
Rotorua District	1,069	1,250	-181	-16.9	1,666	1,401	265	15.9
Whakatane District	586	657	-71	-12.2	742	729	13	1.8
Kawerau District	130	149	-19	-14.9	178	178	0	0.3
Opotiki District	156	175	-18	-11.7	198	177	21	10.7
Gisborne District	558	675	-118	-21.1	885	984	-99	-11.1
Wairoa District	138	138	0	0.2	134	136	-2	-1.7
Hastings District	1,190	1,139	51	4.3	1,409	991	419	29.7
Napier City	1,063	1,116	-52	-4.9	898	666	232	25.9
Central Hawke's Bay District	223	196	27	12.3	235	146	88	37.6
New Plymouth District	1,188	1,060	128	10.7	1,318	917	401	30.4
Stratford District	146	129	17	11.6	161	113	48	29.9
South Taranaki District	411	408	3	0.7	569	383	186	32.6
Ruapehu District	196	203	-7	-3.6	261	266	-5	-2.0
Whanganui District	836	864	-28	-3.4	1,048	914	134	12.8
Rangitikei District	210	247	-37	-17.8	267	227	40	14.9
Manawatu District	430	334	96	22.2	488	266	222	45.4
Palmerston North City	1,022	1,129	-108	-10.6	1,295	1,029	266	20.6
Tararua District	288	306	-18	-6.4	320	274	47	14.6
Horowhenua District	651	704	-53	-8.2	619	518	101	16.3
Kapiti Coast District	1,399	672	727	52.0	741	536	205	27.6
Porirua City	607	462	145	23.9	987	758	228	23.1
Upper Hutt City	499	581	-83	-16.6	609	586	23	3.7
Lower Hutt City	1,195	1,428	-233	-19.5	1,849	1,725	123	6.7
Wellington City	1,594	1,302	292	18.3	2,163	1,349	813	37.6
Masterton District	409	494	-85	-20.9	472	445	27	5.8
Carterton District	143	151	-8	-5.5	119	82	37	31.0
South Wairarapa District	130	135	-5	-3.7	127	121	6	5.1
Tasman District	521	516	5	1.0	458	360	98	21.3
Nelson City	622	651	-29	-4.7	577	467	110	19.1
Marlborough District	698	791	-93	-13.4	601	490	111	18.5
Kaikoura District	56	46	10	17.6	43	47	-4	-9.1
Buller District	168	164	4	2.3	149	118	31	20.6
Grey District	201	181	20	10.0	200	184	16	8.1
Westland District	101	80	21	21.2	104	88	16	15.2
Hurunui District	130	111	19	14.2	108	107	1	1.2
Waimakariri District	743	533	211	28.3	688	462	226	32.9
Christchurch City	4,390	4,666	-276	-6.3	4,833	4,627	206	4.3
Selwyn District	463	273	190	41.1	532	282	250	47.0
Ashburton District	395	491	-96	-24.2	395	377	17	4.4
Timaru District	736	787	-51	-7.0	584	527	57	9.7
Mackenzie District	39	36	4	9.3	32	27	5	15.3
Waimate District	126	122	4	2.9	92	71	21	22.8
Waitaki District	366	382	-15	-4.2	271	209	62	22.9
Central Otago District	273	243	29	10.8	190	205	-15	-8.1
Queenstown-Lakes District	210	179	31	14.8	210	144	66	31.5
Dunedin City	1,741	1,746	-5	-0.3	2,111	1,540	571	27.0
Clutha District	219	238	-19	-8.5	223	172	51	23.0
Southland District	325	289	36	11.0	352	245	108	30.5
Gore District	201	170	32	15.7	187	121	66	35.3
Invercargill City	898	859	39	4.3	1,089	792	297	27.2
Chatham Islands Territory	9	9	0	-0.5	3	4	-1	-37.6

Figure 34: Errors (%) in predicting 2015-2017 annual average hospitalisations by CAU based on 2005-2007 for (a) a total population model and (b) a 30+ years population model

(a) cardiovascular hospitalisations



(b) respiratory hospitalisations



E.2 Pollution scalars

E.2.1 NO_2 predictions

Three-year predictions

We used the average of the rate of change (slope) at each site from 2016 to 2019 to estimate the overall average change in NO_2 concentrations (a scalar of 0.9354 for forecasting 2019 relative to 2016).

Results for each of the 34 sites are shown in Table 53 and the site error distribution is shown in Figure 35.

Ten-year predictions

We used the average of the rate of change (slope) at each site from 2006 to 2016 to estimate an overall average change in NO_2 concentrations (a scalar of 0.8736 for backcasting relative to 2016).

Results for each of the 34 sites are shown in Table 54 and the site error distribution is shown in Figure 36.

Table 53: Difference in the individual site trends vs the average trend for 2016 to 2019

		0	1	2	3	Slope	Intercept, y0	2019	%change	%error	Error
Monitoring Zone	**Site Id**	**2016**	**2017**	**2018**	**2019**	2016-2019	2016	3	**2016-2019**	vs ave	distribution
Auckland - Northern	AUC004	15.0	15.1	13.9	12.8	-0.769	15.339	13.0	0.850	-9.2%	0-5%
	AUC007	24.3	25.1	23.9	20.3	-1.332	25.393	21.4	0.843	-9.9%	12
Auckland - Western	AUC020	14.8	16.2	16.0	13.4	-0.436	15.746	14.4	0.917	-2.0%	35%
Auckland - Central	AUC008	23.4	24.4	23.8	20.9	-0.813	24.372	21.9	0.900	-3.8%	5-10%
	AUC009	47.9	48.2	43.6	40.4	-2.705	49.072	41.0	0.835	-10.8%	11
	AUC011	33.8	33.6	31.3	29.9	-1.394	34.278	30.1	0.878	-6.1%	32%
	AUC013	29.0	29.3	28.2	27.3	-0.626	29.379	27.5	0.936	0.1%	10-15%
	AUC014	28.5	30.0	27.5	26.7	-0.800	29.387	27.0	0.918	-1.8%	8
	AUC015	27.1	29.3	28.2	28.1	0.181	27.918	28.5	1.019	9.0%	24%
	AUC022	25.5	25.1	25.1	21.7	-1.138	26.058	22.6	0.869	-7.1%	15-20%
Auckland - Southern	AUC018	27.4	29.4	29.7	27.9	0.195	28.288	28.9	1.021	9.1%	2
	AUC019	23.8	23.7	23.9	21.6	-0.663	24.243	22.3	0.918	-1.9%	6%
	AUC026	21.8	23.2	22.2	18.8	-0.998	23.002	20.0	0.870	-7.0%	20-25%
Hamilton	HAM001	29.3	30.2	31.9	30.7	0.586	29.664	31.4	1.059	13.2%	0
	HAM003	40.5	42.9	42.5	41.4	0.213	41.505	42.1	1.015	8.6%	0%
Cambridge	HAM004	21.8	27.9	27.8	27.1	1.576	23.788	28.5	1.199	28.2%	>25%
Taupo	HAM005	16.3	18.1	17.2	15.8	-0.264	17.253	16.5	0.954	2.0%	1
Tauranga	HAM007	30.9	34.1	30.2	28.6	-1.107	32.613	29.3	0.898	-4.0%	3%
	HAM008	32.1	34.3	34.1	34.1	0.576	32.797	34.5	1.053	12.5%	
	HAM010	30.8	31.8	29.5	31.0	-0.161	31.037	30.6	0.984	5.2%	
Gisborne	NAP001	16.7	17.2	15.3	13.5	-1.144	17.396	14.0	0.803	-14.2%	
	NAP003	25.6	27.9	26.7	24.3	-0.513	26.878	25.3	0.943	0.8%	
Palmerston North	WAN004	21.9	23.5	22.1	22.1	-0.054	22.489	22.3	0.993	6.1%	
Porirua	WEL005	18.8	20.7	19.5	16.8	-0.715	19.982	17.8	0.893	-4.6%	
Wellington	WEL007	16.7	18.3	16.4	14.1	-0.951	17.808	15.0	0.840	-10.2%	
	WEL008	38.2	40.3	37.8	31.1	-2.393	40.428	33.3	0.822	-12.1%	
Nelson	WEL009	24.3	25.3	23.8	21.2	-1.062	25.253	22.1	0.874	-6.6%	
	WEL011	15.6	16.0	15.3	15.6	-0.058	15.723	15.5	0.989	5.7%	
Blenheim	WEL012	13.2	12.8	12.4	12.5	-0.248	13.121	12.4	0.943	0.8%	
Christchurch	CHR002	29.4	30.0	28.1	22.1	-2.394	30.971	23.8	0.768	-17.9%	
	CHR003	26.2	28.6	27.6	24.2	-0.668	27.648	25.6	0.927	-0.8%	
	CHR004	14.5	14.4	14.1	12.9	-0.507	14.710	13.2	0.897	-4.1%	34
Greymouth	CHR001	11.1	11.0	11.9	11.2	0.128	11.115	11.5	1.035	10.6%	sites total
Dunedin	DUN001	25.5	25.7	26.0	22.9	-0.742	26.145	23.9	0.915	-2.2%	
	DUN002	15.9	16.2	16.5	16.8	0.301	15.877	16.8	1.057	13.0%	
Queenstown	DUN004	23.4	26.8	26.4	25.9	0.698	24.591	26.7	1.085	16.0%	
									0.9354		
						pop wt'd close			0.768		
									1.199		
								change 2016-2019	**-6.5%**		
								2019 factor vs 2016	0.9354	forward in time	

Figure 35: Error distribution in the 2016-2019 trends for the individual NO_2 sites

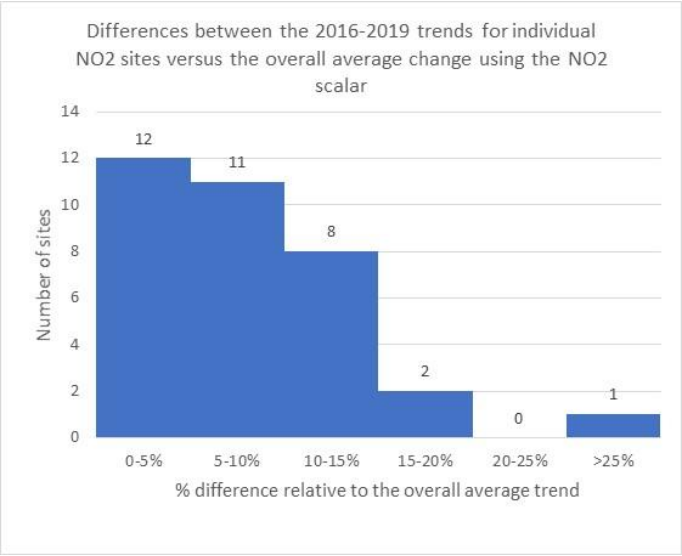
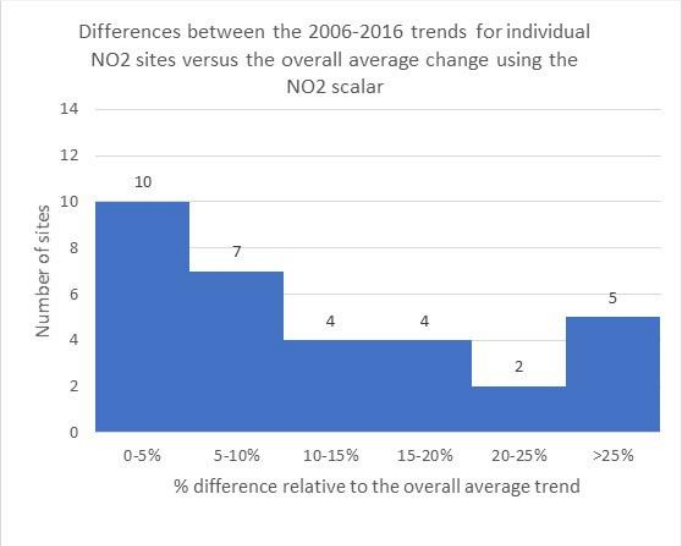


Table 54: Difference in the individual site trends vs the average trend for 2006 to 2016

		0	1	2	3	4	5	6	7	8	9	Slope	Intercept, y0	2006	2016	%change	%error	Error
Monitoring Zone	**Site Id**	**2007**	**2008**	**2009**	**2010**	**2011**	**2012**	**2013**	**2014**	**2015**	**2016**	2007-2016	2007	-1	9	**2006-2016**	vs ave	distribution
Auckland - Northern	AUC004	12.3	15.4	10.9	10.8	10.7	10.0	10.4	10.8	14.2	15.0	0.082	11.672	11.6	12.4	1.071	-6.9%	0-5%
	AUC007	23.3	25.0	25.1	28.5	28.8	28.2	27.7	22.5	26.0	24.3	-0.002	25.950	26.0	25.9	0.999	-13.1%	10
Auckland - Western	AUC020	14.3	14.7	15.9	16.6	16.2	16.0	17.5	14.5	16.5	14.8	0.079	15.340	15.3	16.1	1.052	-8.5%	29%
Auckland - Central	AUC008	25.3	26.6	23.9	27.6	28.3	28.5	28.3	23.2	26.1	23.4	-0.134	26.729	26.9	25.5	0.950	-17.4%	5-10%
	AUC009	41.9	35.8	44.0	40.8	43.9	41.3	46.5	41.0	44.4	47.9	0.693	39.626	38.9	45.9	1.178	2.5%	7
	AUC011	31.0	31.3	36.9	36.7	37.5	36.3	38.0	35.0	37.5	33.8	0.374	33.729	33.4	37.1	1.112	-3.3%	21%
	AUC013	27.6	23.5	32.8	30.1	31.7	29.6	32.6	30.8	29.7	29.0	0.309	28.350	28.0	31.1	1.110	-3.4%	10-15%
	AUC014	26.4	23.1	29.4	30.1	30.8	28.7	30.5	30.0	29.8	28.5	0.411	26.876	26.5	30.6	1.155	0.5%	4
	AUC015	27.2	25.2	28.9	29.3	30.3	28.0	32.1	29.1	29.5	27.1	0.221	27.665	27.4	29.7	1.081	-6.0%	12%
	AUC022	30.0	29.4	31.6	32.3	34.1	31.4	31.7	26.6	27.9	25.5	-0.488	32.245	32.7	27.9	0.851	-26.0%	15-20%
Auckland - Southern	AUC018	26.5	26.0	30.7	30.2	33.1	29.9	29.3	28.2	30.6	27.4	0.127	28.616	28.5	29.8	1.044	-9.2%	4
	AUC019	20.1	21.2	19.6	23.0	24.1	23.4	23.8	21.4	23.6	23.8	0.366	20.761	20.4	24.1	1.179	2.6%	12%
	AUC026	19.3	20.0	17.0	22.0	25.0	25.0	23.7	19.9	22.4	21.8	0.354	20.030	19.7	23.2	1.180	2.6%	20-25%
Hamilton	HAM001	21.4	22.6	26.3	28.4	30.1	29.4	30.2	30.9	30.5	29.3	0.928	23.729	22.8	32.1	1.407	22.4%	3
	HAM003	36.1	38.3	40.0	39.0	40.4	37.5	38.4	38.7	41.4	40.5	0.303	37.671	37.4	40.4	1.081	-6.0%	9%
Cambridge	HAM004	24.1	23.2	27.0	27.9	30.6	27.3	29.0	30.1	31.5	21.8	0.323	25.785	25.5	28.7	1.127	-2.0%	>25%
Taupo	HAM005	17.2	18.6	20.2	20.3	17.3	15.8	17.1	15.6	16.0	16.3	-0.361	19.063	19.4	15.8	0.814	-29.2%	6
Tauranga	HAM007	24.7	23.1	27.0	28.5	32.1	29.1	29.2	29.0	31.0	30.9	0.732	25.157	24.4	31.7	1.300	13.0%	18%
	HAM008	23.5	22.6	24.9	25.6	29.1	29.8	28.8	31.1	29.0	32.1	0.988	23.206	22.2	32.1	1.444	25.6%	
	HAM010	33.3	29.8	38.3	25.9	29.6	26.7	27.4	26.2	29.9	30.8	-0.487	31.982	32.5	27.6	0.850	-26.1%	
Gisborne	NAP001	11.7	10.6	12.4	13.8	16.1	14.7	16.2	15.5	15.5	16.7	0.609	11.567	11.0	17.1	1.556	35.3%	
	NAP003	19.4	19.8	22.4	24.1	27.8	24.6	25.0	24.2	23.5	25.6	0.547	21.170	20.6	26.1	1.265	10.0%	
Palmerston North	WAN004	19.3	21.1	15.1	25.1	25.4	21.5	22.3	21.2	23.1	21.9	0.342	20.042	19.7	23.1	1.174	2.1%	
Porirua	WEL005	20.1	16.8	20.2	20.2	21.8	18.7	20.9	18.8	18.5	18.8	-0.049	19.689	19.7	19.2	0.975	-15.2%	
Wellington	WEL007	18.5	17.3	19.2	19.9	21.7	17.4	19.0	15.6	17.4	16.7	-0.243	19.362	19.6	17.2	0.876	-23.8%	
	WEL008	39.4	31.3	33.3	35.5	34.8	32.7	36.7	36.4	38.3	38.2	0.340	34.114	33.8	37.2	1.101	-4.3%	
Nelson	WEL009	18.3	20.2	20.8	22.8	24.0	22.7	21.8	21.0	23.4	24.3	0.441	19.943	19.5	23.9	1.226	6.7%	
	WEL011	11.8	13.0	14.3	15.1	17.2	15.9	18.4	15.2	16.2	15.6	0.424	13.349	12.9	17.2	1.328	15.5%	
Blenheim	WEL012	12.3	13.4	14.5	14.1	16.7	14.5	14.9	13.7	13.7	13.2	0.038	13.944	13.9	14.3	1.027	-10.6%	
Christchurch	CHR002	21.0	22.2	23.0	26.8	29.0	30.7	28.9	28.2	31.1	29.4	1.041	22.359	21.3	31.7	1.488	29.4%	
	CHR003	22.3	21.7	24.8	26.9	30.0	29.8	24.4	27.8	26.5	26.2	0.462	23.958	23.5	28.1	1.197	4.1%	
	CHR004	12.3	12.2	11.3	15.0	16.3	14.4	18.1	14.5	14.1	14.5	0.342	12.730	12.4	15.8	1.276	11.0%	34
Greymouth	CHR001	10.8	11.7	14.4	12.9	16.0	13.0	14.3	11.7	12.4	11.1	-0.032	12.971	13.0	12.7	0.976	-15.1%	sites total
Dunedin	DUN001	20.3	22.7	25.6	27.0	26.4	27.9	27.1	27.8	25.4	25.5	0.470	23.468	23.0	27.7	1.204	4.8%	
	DUN002	12.5	12.4	12.7	15.3	19.2	15.0	15.6	14.8	14.5	15.9	0.315	13.366	13.1	16.2	1.241	8.0%	
Queenstown	DUN004	15.0	17.9	19.8	22.5	18.7	21.7	18.9	21.8	23.1	23.4	0.700	17.134	16.4	23.4	1.426	24.0%	
																1.1498		
																0.814		
																1.556		
															change 2006-2016	**15.0%**		
															2006 factor vs 2016	0.8697	back in time	

Figure 36: Error distribution in the 2006-2016 trends for the individual NO_2 sites



E.2.2 $PM_{2.5}$ predictions

Ten-year predictions

We calculated the population-weighted annual average $PM_{2.5}$ concentrations for the actual 2006 and 2016 datasets then used the ratio (1.1648 from 7.61/6.53) to backcast the 2016 results to create a predicted 2006 dataset for comparison.

The errors in the difference between the predicted and actual $PM_{2.5}$ for the 2006 CAUs are shown in Table 55 and Figure 37.

Table 55: Error in the predicted versus actual 2006 population-weighted annual average $PM_{2.5}$ concentrations by CAU and the affected population

Difference between predicted and actual $PM_{2.5}$	Number of CAUs	% CAUs	2006 Population	% Population
0-5%	481	23.9%	1,487,160	35.5%
5-10%	479	23.8%	1,245,260	29.8%
10-15%	196	9.7%	455,190	10.9%
15-20%	760	37.8%	819,290	19.6%
20-25%	64	3.2%	106,040	2.5%
>25%	32	1.6%	71,710	1.7%
Total	**2,012**	**100%**	**4,184,650**	**100%**

Figure 37: Error distribution in the 2006 $PM_{2.5}$ annual average concentrations by CAU