Health and air pollution in New Zealand 2019

(HAPINZ 3.0)

He rangi hauora he iwi ora

Approved methodology

Prepared for

Ministry for the Environment, NZ Transport Agency and Ministry of Transport

17 March 2020

New Zealand Government

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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). Generally 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 a number of 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 this study, health effects were evaluated for 67 urban areas based on the 2001 population and ambient monitoring data. The resulting social costs were presented in NZ$ as at June 2004.

This work was later updated by Kuschel et al (2012) 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 updated study (**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. Approximately half of these effects and costs were associated with anthropogenic (human-generated) sources such as domestic fires, motor vehicles, industry and open burning. The primary deliverables from this work were two reports, an exposure model and a health effects model (which enabled scenarios to be tested around changes in ambient concentrations and population).

Since the release of HAPINZ 2.0, the database of ambient monitoring across New Zealand has expanded considerably to include many more locations, pollutants and sources. In addition, exposure-response functions have come available in the literature to enable quantification of a greater range of health endpoints. In mid-2019, the Ministry for the Environment (**MfE**) issued a request for proposals to update HAPINZ 2.0 to address limitations of the previous model and to provide new, robust analyses that were highly defensible and in line with international best practice. Emission Impossible Ltd put together a team of experienced researchers and was engaged to undertake the latest update - **HAPINZ 3.0** in July 2019.

Air and air quality are both a *taonga*[2] and a part of the *kaitiakitanga*[3] for Māori. In recognition of this importance, the HAPINZ 3.0 study is also named *He rangi hauora he iwi ora* which translates to healthy air means healthy people.

The project is being undertaken in stages. Stage 1 involved the preparation of a literature review and proposed methodology for discussion with the Steering Group. This report covers the approved methodology for proceeding with Stages 2 onwards.

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

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

[3] 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 will be following 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** reported by 16 regional councils, 71 airsheds, 74 territorial local authorities and 139 urban areas
Population covered	100% of 2016 population
Pollutants	**Priority pollutants** • particulate matter (PM_{10} and $PM_{2.5}$) • nitrogen dioxide (NO_2)
Exposure assessment	**PM_{10} and $PM_{2.5}$**: ambient monitoring data averaged for 2015-2018 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_{10} and $PM_{2.5}$** using source apportionment data: marine aerosol, biomass burning, motor vehicles, secondary PM, crustal material **PM_{10} and $PM_{2.5}$** using emissions inventory data: industry, open burning, rail, aviation and shipping (where data allow) **NO_2**: motor vehicle exhaust emissions only
Health endpoints	**Primary health outcomes** • **mortality** and years of life lost (YLL) from long-term **$PM_{2.5}$** for all adults 30+ years, all ethnicities and for Māori/Pasifika • **mortality** and YLL from long-term **NO_2** for all adults 30+ years, all ethnicities • **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 • **respiratory admissions** from long-term **NO_2** for all ages, all ethnicities **Secondary health outcomes (for comparison with HAPINZ 2.0)** • **mortality** from long-term **PM_{10}** for all adults 30+ years, all ethnicities and for Māori/Pasifika • **mortality** from long-term **PM_{10}** for all infants, aged 1 month to 1 year • **restricted activity days** from long-term **$PM_{2.5}$** for all ages, all ethnicities **Childhood asthma outcomes relevant to NZ** • **incidence** due to long-term NO_2 • **exacerbations** due to short-term $PM_{2.5}$
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 hospital admissions • respiratory hospital admission • restricted activity days

Feature	Details
Social costs (cont)	**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/s** enabling sensitivity/scenario testing and designed to be easily updateable A **set of improved exposure-response functions** for use in assessing air pollution health effects on Maori and Pasifika* from a separate cohort study A **final report** suitable for a broad audience, outlining the methodology used and key findings (with all assumptions clearly stated) A draft **messaging guide** to provide evidence based dos and don'ts for anyone wanting to communicate the study findings through various channels

Note: The proposed methodology investigated the inclusion of additional pollutants (black carbon, benzo[a]pyrene, arsenic and lead) in HAPINZ 3.0. Following comments from the international peer reviewers and discussion with the Steering Group, these pollutants were dropped from the current assessment due to concerns about data availability, double-counting and robustness of exposure-response functions.

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 this report and how the report is structured.

1.1 Background

The overall objective of the research is to explicitly identify the effects of air pollution throughout New Zealand, link these effects to the various sources and levels of air pollution, and provide information to assist in the formulation of effective policy that will lead to real and measurable improvements in the health of New Zealanders.

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 Health and Air Pollution in New Zealand (**HAPINZ 1.0**)[4] study undertaken by Fisher et al (2007).

In this study, health effects were evaluated for 67 urban areas based on the 2001 population and ambient monitoring data. The resulting social costs were presented in NZ$ as at June 2004. 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 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 this original study 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

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

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 update in 2012 (HAPINZ 2.0)

In the years following the release of the original HAPINZ study, both data availability and the understanding of air pollution health effects improved significantly. In particular, air quality monitoring was implemented in most urban locations in New Zealand – largely in response to the introduction of a national environmental standard for ambient PM_{10} concentrations in September 2005. HAPINZ 1.0 was formally updated by Kuschel et al (2012) to incorporate population data from the 2006 census and the more comprehensive monitoring being undertaken across New Zealand.

In this update (**HAPINZ 2.0**), health effects were based on PM_{10} only - because the majority of health effects in New Zealand were assumed to be associated with this pollutant and PM_{10} was a good indicator of the sources and effects of other air pollutants. The resulting social costs were presented in NZ$ as at June 2010. The authors 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. Approximately half of these effects and costs were associated with anthropogenic (human-generated) sources such as domestic fires, motor vehicles, industry and open burning.

The primary deliverables from this work were two reports, an exposure model and a health effects model (which enabled scenarios to be tested around changes in ambient concentrations and population).

The key features of this update are summarised in table 2.

Table 2: Key features of the 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 local authorities and 139 urban areas
Population covered	• 4,027,902 covering 100% of 2006 population
Pollutants	• PM_{10} only
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 update (HAPINZ 3.0)

Following the release of HAPINZ 2.0 in 2012, the database of ambient monitoring across New Zealand has expanded considerably to include many more locations, pollutants and sources. In addition, exposure-response functions have become available in the literature to enable quantification of a greater range of health endpoints.

In mid-2019, the Ministry for the Environment (**MfE**) issued a request for proposals to update HAPINZ 2.0 to address limitations of the previous model and to provide new, robust analyses that were highly defensible and in line with international best practice.

Emission Impossible Ltd put together a team of experienced researchers and was engaged to undertake the latest update - **HAPINZ 3.0** in July 2019.

1.3 Project funding and steering committee

This project is being funded by:

- Ministry for the Environment
- NZ Transport Agency
- Ministry of Transport.

The project is being overseen by a steering committee comprising:

- Drew Bingham, Co-chair (Ministry for the Environment
- Shelley Easton, Co-chair (NZ Transport Agency)
- Greg Haldane and Janet Petersen (NZ Transport Agency)
- Iain McGlinchy (Ministry of Transport)
- Sonja Miller (StatsNZ)
- Suz Halligan (Ministry of Health)
- Tim Mallett and Tamsin Mitchell (National Air Quality Working Group).

1.4 Purpose of this report

The project is being undertaken in five stages as shown in table 3.

Table 3: Milestones and key delivery dates for HAPINZ 3.0

Stage	Description	Due date
1	Undertake literature review and propose methodology for steering committee approval	16 Dec 2019
2	Prepare draft versions of all models/reports and present to steering committee	31 Oct 2020
3	Get feedback on draft deliverables from external reviewers and steering committee	27Nov 2020
4	Finalise all models/reports and present to steering committee	30 Jan 2021
5	Develop outreach material and publicly release project findings	15 May 2021

Stage 1 involved the preparation of a literature review and proposed methodology for discussion with the Steering Group. This report covers the approved methodology for proceeding with Stages 2 onwards.

The overarching approach we are following is summarised in a matrix contained in appendix A.

1.5 Report layout

This report is structured as follows:

- Chapter 2 introduces the key steps involved in assessing air pollution health impacts and outlines the best practice general principles we will follow
- Chapter 3 outlines our approach to assessing the exposure of New Zealanders to air pollution in terms of pollutants
- Chapter 4 describes how we will attribute exposure by different sources
- Chapter 5 discusses the range of health endpoints we will address
- Chapter 6 describes our method for estimating social costs associated with air pollution health effects
- Chapter 7 reviews options for more effective communication of key messages arising from the study
- Chapter 8 outlines the calculation methodology, input data and model design
- Chapter 9 discusses how we will address uncertainty
- Chapter 10 summarises the features of our methodology (shown in matrix form in appendix A)

2. Assessing air pollution health impacts

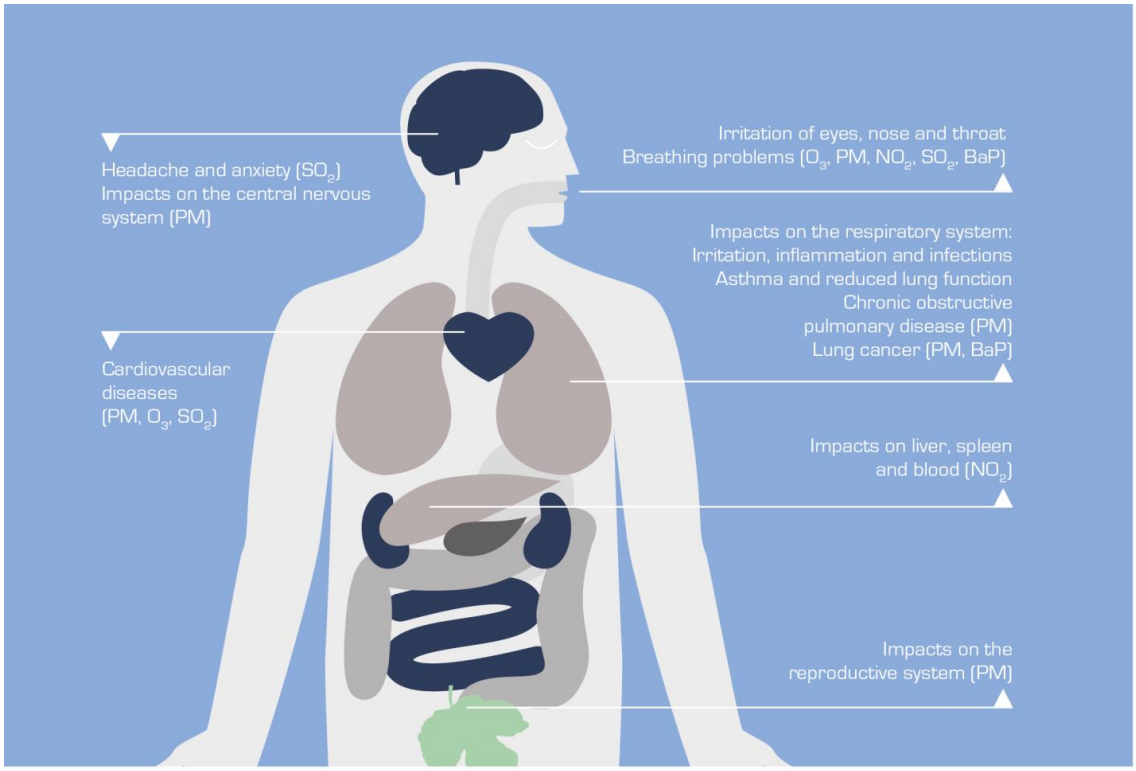
This chapter introduces key air pollutants in New Zealand, identifies the key steps involved in assessing air pollution health impacts and outlines the best practice general principles we will follow.

2.1 Health effects of air pollution

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*" (Ministry for the Environment, 2007). Air and air quality are both a *taonga*[5] and a part of the *kaitiakitanga*[6] for Māori.

Air pollution can cause significant health impacts ranging from respiratory symptoms to premature death. Figure 1 illustrates the potential health effects specifically associated with air pollution.

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.

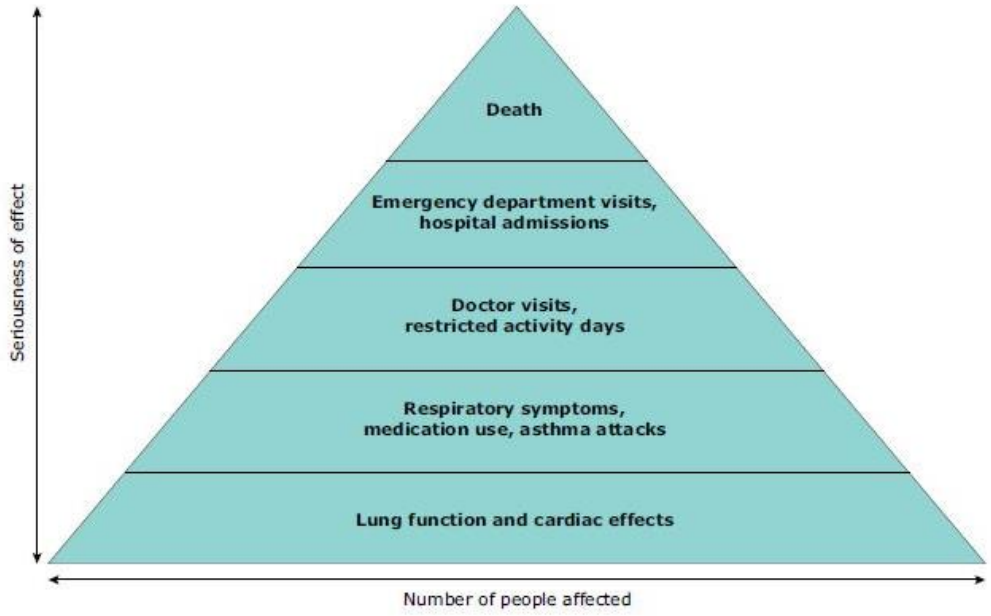
Source: European Environment Agency (2013)

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

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

Some people, for example older adults, children, pregnant women and people with an underlying disease (such as asthma), may be more at risk, and may develop more severe health effects more quickly, when exposed to air pollution (WHO, 2016). More people are affected by less severe health effects than the proportion affected by more severe health effects (figure 2). While there are a large number of acute/short-term health effects, the fewer chronic health impacts incur a much greater social cost.

Figure 2: Pyramid of PM_{10} health effects



Source: European Environment Agency (2013)

2.2 Long-term exposure vs short-term exposure

Air pollution exposure can have two types of effects: short-term (acute) or long-term (chronic) effects. 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).

Short-term effects can include premature death in susceptible individuals, but the major impact of air pollution exposure on life expectancy is through the gradual, cumulative effects on chronic disease. The health burden due to chronic exposure to air pollution is typically 10 times greater than that for acute exposure, based on the relative risk ratios (WHO, 2006). More recently, the Royal College of Physicians in the United Kingdom reported 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)

2.3 Certain people are more vulnerable to effects

Susceptibility to the effects of air pollution depends on factors that are unique for each individual (eg, age, health status, genetic makeup) as well as exposure (eg, time spent outdoors, proximity to major roads).

Based on health reviews, there are groups within the population who are more affected by air pollution than others (Ministry for the Environment, 2011). These susceptible groups are:

- 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.

New Zealand has one of the highest prevalences of asthma in the world, with one in seven children (14%) aged 2–14 years (107,000 children) and one in nine adults (11%) aged over 15 years (389,000 adults) reporting taking current asthma medication (Health Quality & Safety Commission, 2016). The Organisation for Economic Co-operation and Development's (**OECD**) statistics indicate New Zealand has the fourth highest hospital admission rates for asthma of OECD countries.

Māori are 2.9 times and Pacific peoples 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).

2.4 Air pollution is 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 which are correlated with the assessed pollutant. This means that the key air pollutants investigated in an assessment of health impacts may be proxies for the complex mixture (WHO, 2016).

2.5 Key air pollutants in New Zealand

Of the common pollutants present in air pollution, the most significant health impacts (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. In addition, exposure to NO_2, particularly in transport-impacted cities, is increasing.

In terms of additional pollutants, while relatively high concentrations of BC, As, benzo(a)pyrene (**BaP**) and lead (**Pb**) have been measured in New Zealand, ozone (**O_3**) and sulphur dioxide (**SO_2**) levels are of less concern due to historically low or very localised concentrations.

Note: These additional pollutants were investigated in the proposed methodology but were dropped from the current assessment due to concerns about data availability, double-counting and robustness of exposure-response functions.

2.5.1 Particulate matter

Particulate matter (**PM**) comes from anthropogenic (human-caused) sources such as burning coal, oil, wood, petrol and diesel in domestic fires, motor vehicles and industrial processes. Natural sources of particles include sea spray, dust, pollens, volcanic activity and more recently 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.

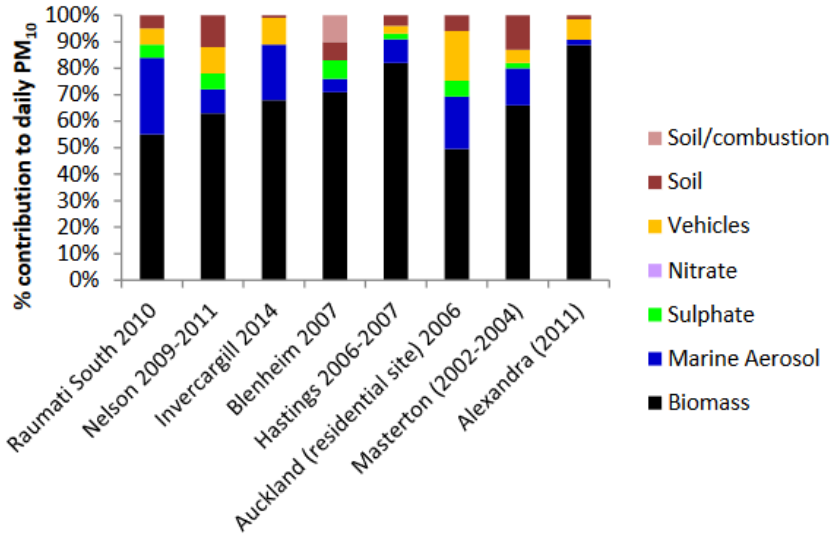
New Zealand is an isolated island nation. This means that, except for rare events such as bushfires and dust storms from Australia, we generate our own pollution (MfE and StatsNZ, 2018). Therefore the composition and sources of pollution in New Zealand are quite different to other parts of the world. 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. Conversely, in New Zealand PM speciation and source apportionment studies have shown that anthropogenic PM is typically dominated by emissions from biomass burning (domestic fires) and motor vehicles (near busy roads and in larger cities), while natural sources of PM are dominated by marine aerosol (Davy and Trompetter, 2018). Figure 3 compares several source apportionment studies and shows that biomass burning (domestic fires) contributed between 50% in Auckland to 89% of daily wintertime PM_{10}. On an annual basis, secondary particulate typically accounts for 10 to 20% of particulate concentration at urban monitoring sites in New Zealand.

In urban atmospheres, UFP (which are measured in terms of number concentration), mainly arise from road traffic but can also from the nucleation (new particle formation) of secondary particles, especially in high insolation urban climates (Brines et al, 2015).

There is a substantial body of evidence that inhaling PM is harmful to human health, especially finer fractions such as PM_{10}, $PM_{2.5}$ and UFP. PM_{10} is a more inclusive, but less specific measure of exposure than $PM_{2.5}$. PM_{10} includes $PM_{2.5}$ plus the coarser $PM_{2.5}$ to PM_{10} fraction. 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). Particles of different

sizes typically have different sources and different chemical and biological composition. However, the mechanisms of particle toxicity are complex and still not fully understood.

Figure 3: Comparison of wintertime source apportionment studies



Source: Davy and Trompetter (2017b)

The health effects[7] of PM are predominantly respiratory and cardiovascular. The impacts range from functional changes (eg, reduced lung function) to symptoms, impaired activities (eg, school absenteeism, days off work), doctors' visits through to hospital admissions, reduced life expectancy and death.

2.5.2 Nitrogen dioxide

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. 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). 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 (Ministry for the Environment & Statistics NZ, 2018).

Evidence of a causal relationship between short-term NO_2 and respiratory outcomes 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 pollutants (such as UFP, $PM_{2.5}$, CO, BC and polycyclic aromatic hydrocarbons) especially in traffic impacted locations (WHO, 2016).

The effects of NO_2 are potentially significant. For example, for 2015 it has been estimated that 79,900 people died prematurely each year in Europe due to long-term exposure to NO_2

[7] Adverse health effects that involve increased illness or disease are generally referred to as 'morbidity' effects, while those involving premature death are classified as 'mortality' effects.

compared to an estimated 422,000 premature deaths due to long-term exposure to $PM_{2.5}$ (EEA, 2018). However, the European Environment Agency (**EEA**) notes that the concentrations of $PM_{2.5}$ and NO_2 are sometimes strongly correlated, which means that the impacts estimated for these cannot be aggregated. Doing so may lead to double counting of up to 30% of the effects of NO_2.

2.5.3 Volatile organic compounds

Volatile organic compounds (**VOCs**) include a wide range of organic gaseous components of anthropogenic and biogenic origins. Some of the anthropogenic VOCs are considered carcinogenic to humans. Of most concern are benzene, formaldehyde and 1-3 butadiene.

Industrial sources, such as paint and resin production, evaporative and exhaust emissions of vehicles and petrol stations, use of paints, pesticides, detergents, and petrochemical plants are major sources of anthropogenic VOCs; while isoprene, monoterpenes and methane from forests and wetlands are the major biogenic VOCs. Both biogenic and anthropogenic VOCs react quickly with ozone (**O_3**) and oxidant radicals in the atmosphere and form oxidised VOCs, such as formaldehyde and acetaldehyde. Thus some VOCs have both primary (emitted directly) and secondary (formed into the atmosphere from later reactions) sources.

2.6 Typical approach for assessing health effects

The effects of air pollution on health are typically assessed in a stepwise process as shown in figure 4.

For each area under assessment (eg, a census area unit, **CAU**), the health impacts are generally calculated as follows:

Health Effects (cases) = Exposure * Exposure-Response Function * Population Exposed

Where:

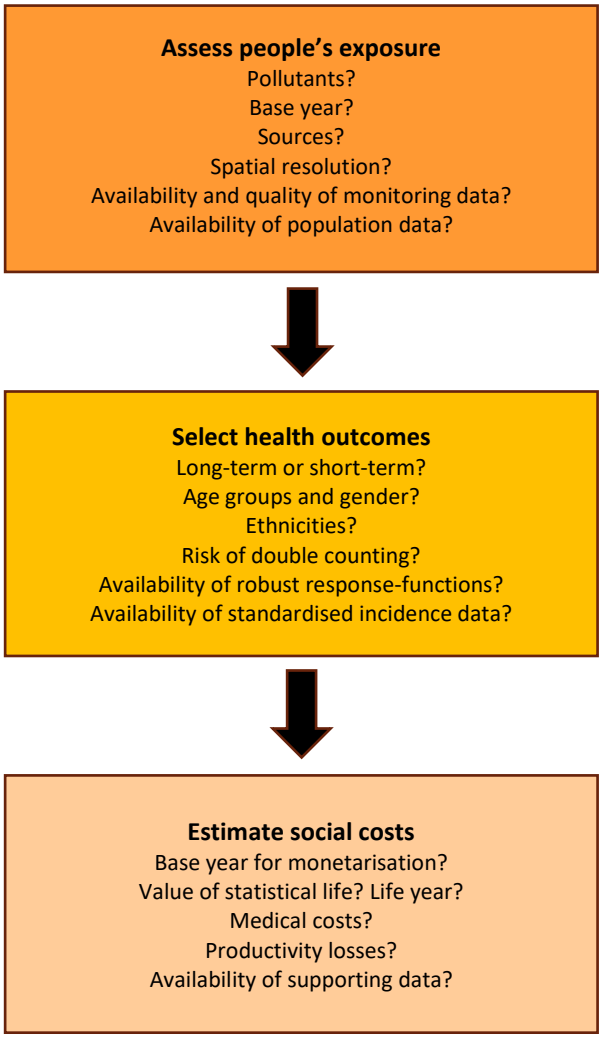
cases are the number of premature deaths, hospital admissions or restricted activity days etc depending on the health outcome being assessed. These are usually assessed relative to a baseline rate that can vary significantly by population and region.

exposure is the concentration of pollutant in the area of interest (eg, annual average PM_{10} concentration in a particular census area unit)

exposure-response function is a formula which shows the change in risk for a particular health outcome (eg, premature death) per unit change in concentration (eg, 10 $\mu g/m^3$ of PM_{10}). These functions are developed from epidemiological studies, which examine the relationship between air pollution exposure and health effects in observed populations.

population exposed is the number of people in the area of interest

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



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

Social Costs = Health Effects (cases) * Cost per case

In simple terms, first we estimate the air pollution concentration and the number of people exposed. Then we apply exposure-response relationships to estimate adverse health effects. These are then combined with published health-cost data 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 benefits of health improvements against the costs of (various) air pollution reduction initiatives
- evaluating the effectiveness of existing policy initiatives (back-casting)
- assessing the likely effects of current population and business as usual trends (forecasting)

- developing targeted strategies for reducing the air pollution exposure of particularly vulnerable groups in the population.

Health impacts 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, 2016).

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

2.7 Best practice guidelines

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

- Health effects assessment 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 such that the end user can see how health outcomes 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.

The following chapters describe the results of our literature review and our approved methodology for the major steps in the air pollution health impact assessment in more detail:

- assessing people's exposure (chapter 3)
- attributing exposure to different sources (chapter 4)
- determining the resultant health effects (chapter 5)
- estimating the overall social costs (chapter 6)
- communicating the findings (chapter 7)
- describing the calculation methodology, input data and model design (chapter 8)
- addressing uncertainty (chapter 9)

3. Evaluating exposure

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 outlines the approach we will be adopting for the HAPINZ 3.0 update.

Understanding exposure is critical to understanding potential health impacts. This is because, the longer people are exposed to air pollutants, and the higher the pollutant concentrations, then the more health effects are likely to occur as a result.[8]

3.1 Approach used in HAPINZ 2.0

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

- **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 (ie, domestic and industry contributions, if applicable, based on housing density and available industrial emissions inventory data).
- **Results were estimated by census area units** 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.

3.2 Developments that have happened since

This section reviews the developments in the availability of monitoring data and exposure models for New Zealand since HAPINZ 2.0.

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

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 (**NES**) for air quality.

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[8] For pollutants where causality has been established

[9] As defined by Statistics New Zealand. See http://archive.stats.govt.nz/browse_for_stats/Maps_and_geography/Geographic-areas.aspx for details

[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).

New Zealand has outstanding 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 4 (Ostro et al, 2018; GBD, 2017). New Zealand has more monitors than Sweden and Australia, countries with similar air quality but more than two and four times our population respectively. Table 5 presents these data on a *per capita* basis for select countries.

Table 4: Number of ground measurements per country used in Global Burden of Disease 2015

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
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)

Table 5: Number of ground measurements from select countries on a per capita basis

Country	Monitors per million persons*
Australia	1.4
Belgium	3.8
Canada	3.4
China	0.1
Finland	4.3
Germany	1.9
Italy	3.9
Japan	0.1
Netherlands	1.4
New Zealand	**8.1**
Norway	2.3
South Africa	0.2
Spain	4.8
Sweden	1.8
Turkey	1.0
United Kingdom	0.8
United States of America	1.1

*Individual country population accessed from Wikipedia on 22 Oct 2019

Ambient PM_{10} air quality data, collected using approved regulatory methods for the purposes of the NES, are available for 57 out of 71 airsheds for the last 13 years (ie, 2006 to 2018). Between 2015 and 2018, there were 51 monitoring sites around the country with three full years of data and 61 monitoring sites with at least one full year of PM_{10} monitoring data.

Monitoring for $PM_{2.5}$ has progressively increased since 2010 across 23 airsheds in nine regions; Northland, Auckland, Waikato, Bay of Plenty, Hawke's Bay, Wellington, Nelson, Marlborough and Canterbury. Between 2015 and 2018, there were 25 monitoring sites around the country with at least one full year of $PM_{2.5}$ monitoring data.

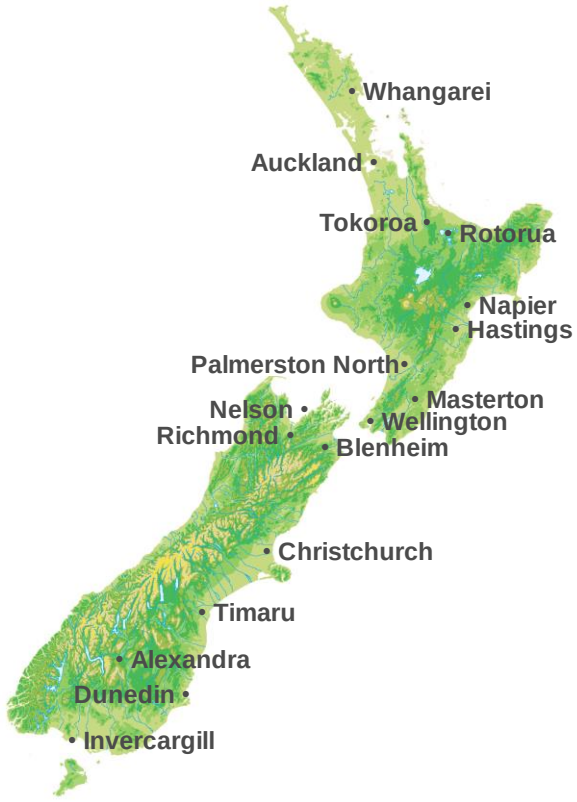
The $PM_{2.5}$ monitoring sites are all co-located with PM_{10} monitoring. Source apportionment analysis has also been undertaken at a number of these sites. This means that there is a considerable amount of data for development of $PM_{2.5}$ to PM_{10} ratios. These allow for the indirect estimation of $PM_{2.5}$ concentrations in airsheds with PM_{10} monitoring.

Ambient air quality data are collected using approved regulatory methods for the purposes of the NES for air quality. We have reviewed this data and it is robust and quality assured for the purpose of assessing annual average exposure.

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. Figure 5 shows the PM speciation sampling locations in New Zealand to date.

Figure 5: Particulate matter speciation sampling locations in New Zealand



Source: Davy and Trompetter (2018)

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

Further details of the PM speciation sampling are provided in appendix B.

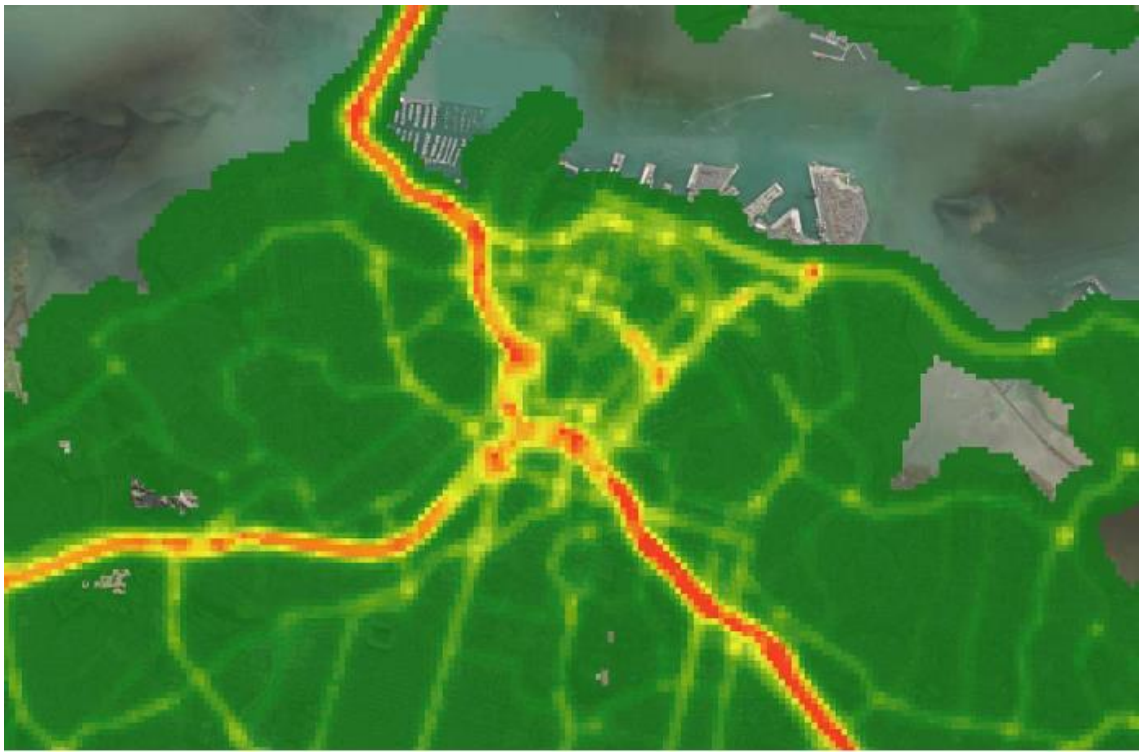
3.2.2 NO_2 exposure

Health effects of NO_2 were not assessed in HAPINZ 2.0 because there was inadequate ambient air quality monitoring data available to assess exposure.

Since the release of HAPINZ 2.0, there has been a considerable increase in the amount of monitoring being undertaken. Regional Councils monitor NO_2 continuously, using regulatory methods, at 15 sites around New Zealand. In addition, the NZ Transport Agency (**NZTA**) has a network of passive monitoring sites at 129 locations (NZTA, 2017). This monitoring dataset has enabled development of high resolution NO_2 exposure assessments.

The NZ Transport Agency is currently developing traffic pollution exposure maps with a 50m resolution. An example output from the NZTA National Vehicle Emissions Database (**NVED**) exposure tool is shown in figure 6.

Figure 6: Example output from the NZTA nitrogen dioxide exposure model



Source: Jacobs [11]

Exposure maps are also being developed by NIWA in their Traffic Impact Model.[12]

Unfortunately, neither the NIWA nor the NZTA model is currently available, so we cannot establish which is more robust and likely to produce more realistic estimates. Both of these models are discussed further in section 3.2.5.

3.2.3 Traffic exposure models

National Vehicle Emissions Dataset (NVED) exposure tool

The NZ Transport Agency is developing a suite of tools to provide for assessment of exposure to traffic pollution. These include:

- The National Vehicle Emissions Dataset (**NVED**) emissions tool which estimates emissions from every road in New Zealand based on the NZTA's Vehicle Emission Prediction Model (**VEPM**) and detailed traffic data.
- The NVED concentration tool, which consists of:
  - NO_2 background concentration maps for urban areas. We understand that these have been developed based on analysis of monitoring results and land use regression techniques.
  - Contour-based GIS datasets of emissions concentrations for annual average daily mean NO_2, PM_{10} and $PM_{2.5}$ within 200m of roadways. These are based on the NVED emissions and simple dispersion equations.

[11] Hastings K (Jacobs). Memo to NZ Transport Agency on the *NVED Exposure Tool*. 12 July 2019

[12] For details see the description of NIWA's Traffic Impact Model: https://niwa.co.nz/node/110129

- The NVED exposure tool which estimates exposure to elevated concentrations based on dwelling locations and population data

The NVED exposure tool is effectively a land use regression model (based on traffic emissions density). We understand that the outputs are currently being validated and results of the concentration tool will be available by December 2019. Results from the exposure tool are likely to be available for NO_2 early in 2020.

For PM_{10} and $PM_{2.5}$, the NVED exposure tool will provide the estimated roadside exposure to exhaust, brake and tyre wear only. The tool will not provide road abrasion, or the estimated contribution of motor vehicles to secondary particulate.

Traffic Impact Model

NIWA has also been developing a Traffic Impact Model which they describe as a 'semi-empirical' model combining measurement data from hundreds of locations collected by several organisations, with physical principles of dispersion to provide coverage of the spatial patterns in road traffic pollution. At present, data for BC and NO_2 are available for approximately 10 New Zealand cities with more still to be added.

NIWA acknowledge that the current version of the model has a number of known limitations, which they are working to address as follows:

- The model predicts (primary-only) pollutant levels due to road traffic sources only. Observed pollutant levels may be slightly higher in the vicinity of other major emission sources, such as industrial boilers, large power plant, airports, seaports and busy rail yards.
- The current model underestimates concentrations in locations impacted by higher than usual proportions of diesel vehicles, such as on roads dominated by buses or trucks.

The model is not currently finalised and does not yet cover towns and cities with very little observational data.

We understand that the NVED exposure tool and the NIWA model have been developing in parallel. The NVED tool uses estimated background NO_2 and NO_x to NO_2 conversion factors that have been developed by NIWA.

Land use regression analysis

In HAPINZ 2.0, regression analysis was used in some locations (including Auckland) to estimate PM_{10} concentration by census area unit based on emissions density. We have updated the regression analysis for 2013 and found that emissions density (by census area unit) does not generally correlate well with measured PM_{10}.

This investigation is summarised in appendix C.

Road dust exposure model

We understand that the NZ Transport Agency is also developing a tool to estimate exposure to dust from unsealed roads but we have no firm date as to when results might be available.

3.2.4 High density monitoring networks

A number of studies have been undertaken, or are underway, to investigate the spatial distribution of pollution in urban areas using low cost sensors (eg, Longley et al, 2019). This is a relatively new area of research with results only available for a few locations.

3.2.5 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, there are other methods available for assessment of exposure as shown in table 6 (Hoek, 2017).

Table 6: 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 more fine spatial scale.

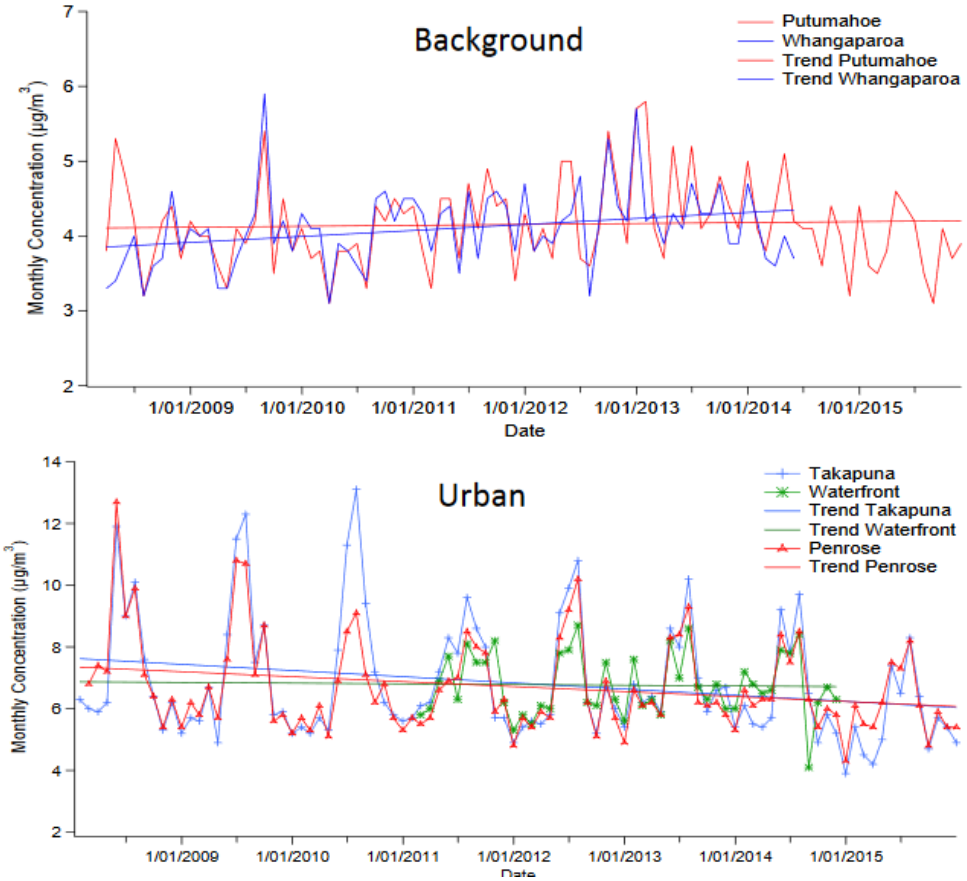
Source: Hoek, 2017

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.

Intra-urban contrasts - PM

Monitoring of $PM_{2.5}$ in Auckland (New Zealand's largest urban area) has shown very similar concentrations of $PM_{2.5}$ across comparable locations. For example, figure 7 shows monthly $PM_{2.5}$ concentrations for two background sites and three urban (traffic influenced) sites.

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



Source: 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. We know 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. We will investigate methods to estimate exposure to the roadside component of PM_{10} and $PM_{2.5}$ such as using the NZ Transport Agency NVED Exposure Tool (provided that this is available and validated within the project time frames) and reviewing source apportionment results in the vicinity of roads.
- We cannot 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. As discussed later in section 4.3.4, we will estimate the overall contribution of industry to the health effects of air pollution in each airshed based on the results of source apportionment and emissions inventories. However, this will be an approximate estimate only. Further work, including development of a comprehensive national inventory of industrial emissions, and dispersion modelling, would be required to more accurately estimate exposure to industrial emissions and the effects of these at a national level.

- There are localised variations in the concentration of air pollution in some locations due to complex topography and meteorology. However, we don't currently have enough information to robustly quantify these variations.

These limitations introduce some uncertainty in the exposure assessment. However, as discussed in section 9, 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.

Ultimately, the most appropriate method and resolution for exposure assessment will reflect the methods and resolution used in the original epidemiological research. For this study, we will develop and use New Zealand specific exposure-response functions for PM_{10} and $PM_{2.5}$. This means that any uncertainty in the exposure assessment will be captured in the uncertainty of the exposure-response functions.

Intra-urban contrasts – NO_2

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

As discussed previously, the exposure assessment should ideally use the same spatial scale and resolution as the original epidemiological research. However, the 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).

As discussed in section 5, there is still considerable uncertainty in exposure-response functions for NO_2. 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). This analysis also found that spatial scale is a significant source of uncertainty for a resolution of less than 1 km. The authors recommend a spatial scale of 100 m for an EU-wide assessment.

3.3 Approved methodology

3.3.1 PM_{10} and $PM_{2.5}$

For the HAPINZ 3.0 update, we will rely primarily on the results of monitoring to estimate exposure to PM_{10} and $PM_{2.5}$. We will investigate options to characterise the intra-urban variation in concentrations, including the NZ Transport Agency NVED exposure tool.

Our overall approach to estimate exposure will be similar to the HAPINZ 2012 methodology. We will develop an exposure model based on actual monitoring data where available. For areas and pollutants where monitoring data are not available, concentrations will be estimated based on comparison with monitored areas (considering key factors such as emissions density, meteorology and topography). There is significantly more information now available than in 2012 when the previous HAPINZ was published. We are therefore confident that this approach will provide a robust assessment of exposure.

Actual monitoring data will be used and averaged for 2015 to 2018 to reduce the influence of year to year variability in meteorological conditions. Although the base assessment year is likely to be 2016, we propose to include 2018 data because there was a significant increase in the availability of $PM_{2.5}$ data in 2018.

In general, the monitored concentration will be applied to the entire airshed. Some judgement will be required, as follows:

- For airsheds with more than one monitor, the most representative site(s) will be selected. The results will either be averaged across all representative monitors, or individual results will be assigned to areas of influence based on an assessment of local topography and emissions density.
- For unmonitored areas, annual concentrations will be based on comparisons with monitored areas that have the 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_{10} to $PM_{2.5}$ will be developed, based on available monitoring and taking into account emissions inventory and source apportionment data (relevant to the non-monitored area).

We will investigate methods to account for elevated concentrations close to roads. This will include consideration of source apportionment studies as well as the NZ Transport Agency high-resolution (50 metre spatial scale) traffic exposure model (provided that this is available and validated within the project time frames).

3.3.2 NO_2

As discussed previously, NO_2 exposure assessment models are being developed by NIWA and NZ Transport Agency. However, neither model is currently available so we cannot establish which model is more robust and likely to produce more realistic estimates. For HAPINZ 3.0, we intend to use the best available data at the time that analysis is undertaken, which on current timing is likely to be the NZ Transport Agency model.

The robustness of the estimate of NO_2 health effects will be limited by the robustness of the exposure-response functions. Provided that reasonable exposure estimates with a resolution of better than 1 km are available by early 2020, we expect to at least provide a preliminary indication of likely health effects from exposure to road transport NO_2.

Note: We will not assess exposure to O_3 or SO_2. Concentrations of O_3 in New Zealand are very low and O_3 is now only monitored at one location (Musick Point in Auckland). Elevated levels of SO_2 tend to be localised and there is insufficient data available to assess impacts at a national level. However, the effects of secondary sulphate particulate will be captured in the assessment of exposure to PM.

3.3.3 Summary of our approach

Particulate matter (PM_{10} and $PM_{2.5}$) will be used as the primary indicator of air pollution exposure due to 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) will be used as an indicator of air pollution exposure due to increased epidemiological evidence of effects, and improved information about exposure to NO_2 from transport in New Zealand.

Actual monitoring data will be used in preference to modelling estimates and **averaged for 2015 to 2018** to reduce the influence of year to year variability in meteorological conditions.

In general, **the monitored concentration will be applied to the entire airshed**. Some judgement will be required, for example:

- For airsheds with more than one monitor, the most representative site(s) will be selected. The results will either be averaged across all representative monitors, or individual results will be assigned to areas of influence based on an assessment of local topography and emissions density.
- For unmonitored areas, annual concentrations will be based on comparisons with monitored areas that have the 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_{10} to $PM_{2.5}$ will be developed, based on available monitoring and taking into account emissions inventory and source apportionment data.

We will investigate methods to account for elevated concentrations close to roads. This will include consideration of source apportionment studies as well as the NZ Transport Agency NVED exposure tool (provided that this is available and validated within the project time frames).

The methodology will not specifically account for elevated concentrations in the vicinity of other localised sources (including for example, unpaved road dust, large industry and ports) because source specific exposure information is not available.

Exposure to **NO_2 from traffic (only)** will be estimated based on the results of the NZ Transport Agency NVED exposure tool (provided that this is available and validated within the project time frames).

4. Attributing source contributions

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 outlines the approach we will be adopting for the HAPINZ 3.0 update.

A key policy focus of the HAPINZ studies is the need to consider source contribution so that health effects likely to be attributable to key sources can be estimated.

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 where 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 (eg, other transport including 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 that have happened since

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 concentration of PM_{10} was proportional to the estimated quantity of emissions of PM_{10} 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 or focussing on one source/pollutant. Emission inventories rely on emission factors (eg, the amount of pollution produced per km travelled by petrol cars) and activity data (eg, number of 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 (Metcalfe and 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 are not always consistent and directly comparable (Sridhar and Kuschel, 2018). Some limitations include:

- Most inventories report on the major sources only (motor vehicles, domestic heating and industry) with other sources such as shipping and aviation often excluded.
- Not all pollutants are included in each inventory (as most are PM_{10} inventories only).
- Although the general approach to estimating emissions is the same, the specific methodologies used depend upon available data.
- There are different reporting requirements and periods (eg, annual emissions versus daily winter average).
- The spatial extent and resolution differ between each inventory (eg, region vs airshed).
- Emissions are estimated for different base years and therefore do not reflect the same fuel specifications, emission standards, and regulations in place.

Emissions inventories rely on a significant number of assumptions to estimate emissions from each source, which means that the end results are subject to uncertainty. For example, in Auckland, the overall uncertainty associated with the inventory of PM_{10} emissions from motor vehicles is approximately ± 31% (Sridhar and Metcalfe, 2019) and for home heating the uncertainty is approximately ± 21% (Metcalfe et al, 2018).

Even with a robust emissions inventory, we know that the relative contribution of sources to concentration and subsequent exposure is not necessarily proportional to their emissions. 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).

Another limitation is that emission inventories include only primary emissions. Natural sources of aerosol and secondary PM, formed form organic and inorganic gaseous pollutants in the atmosphere can also contribute to PM concentration.

This means that it is preferable to use source apportionment studies to estimate the relative contributions.

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. Further details of the source apportionment that has been undertaken in New Zealand are provided in appendix B.

The wealth of source apportionment data provides an updated, robust evidential basis for attributing source contribution for assessing exposure. However, the source descriptions and categories available differ significantly from those in HAPINZ 2.0 and the results will not be directly comparable. Table 7 compares key features of the methods used to attribute source contribution in HAPINZ 2.0 with the methods we will use in HAPINZ 3.0 for each key source.

Table 7: Comparison of source apportionment methods with HAPINZ 2.0

Source	Source apportionment method HAPINZ 2.0	Source apportionment method HAPINZ 3.0
Natural	The 'natural' component included sea spray (marine aerosol) and wind blown dusts (crustal matter or soil).	As discussed in appendix B, monitoring has shown that crustal matter is largely anthropogenic, resulting from activities such as construction and industry. We recommend that only marine aerosol (sea spray) should be subtracted from the total PM_{10} concentration to estimate the concentration of PM_{10} from anthropogenic sources.
Marine aerosol (sea spray)	The concentration of marine aerosol was estimated based on the results of source apportionment.	The concentration of marine aerosol will be estimated based on the results of source apportionment. We have substantially more monitoring results available.
Crustal matter	Crustal matter (referred to as 'windblown dusts' or 'soil' in HAPINZ 2.0) was estimated based on the results of source apportionment.	The concentration of crustal matter will be estimated based on the results of source apportionment. We have substantially more monitoring results available which shows that the specific sources of crustal matter vary between locations. We will not attempt to assign crustal matter to specific sources.
Industry	To contribution of industry to anthropogenic PM_{10} was estimated separately for industries with tall stacks and those without, based on emissions inventory data.	Data on the contribution of industrial emissions to exposure is not readily available at a national level. We will use the results of source apportionment, with emission inventory data, to estimate the contribution of industry to PM in each airshed. This will be approximate only.
Domestic fires	The contribution of domestic fires to anthropogenic PM_{10} was estimated based on emission inventory data	The contribution of domestic fires to PM will be estimated based on the results of source apportionment. As discussed in appendix B, the biomass burning signal is dominated by domestic fires in urban areas of New Zealand.

Source	Source apportionment method HAPINZ 2.0	Source apportionment method HAPINZ 3.0
Motor vehicles	The contribution of motor vehicles to anthropogenic PM_{10} was estimated based on emission inventory data including exhaust, brake and tyre wear only.	The contribution of motor vehicles to PM will be estimated based on the results of source apportionment. This includes all PM associated with the source, so it will include re-suspended road dust, brake wear and road abrasion.
Open burning in urban areas	The contribution of open burning to anthropogenic PM_{10} was estimated based on 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.	Open burning of waste is now banned in most polluted urban areas. Source apportionment studies have not identified open burning as a significant source of pollution in urban areas. We will identify airsheds where open burning does occur and make a qualitative assessment of the likely contribution of open burning to PM exposure (based on emissions inventory data).
Open burning in rural areas	As above for open burning in urban 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. We do not propose to estimate the contribution of open burning to the health effects of air pollution in rural areas.
Secondary PM from all sources	Not included because not available from emissions inventories which focus on primary emissions.	Secondary PM will be quantified at locations with monitoring data. Information is available to link secondary PM to specific sources of gaseous precursors so source assessment will be quantitative.

Urban background $PM_{2.5}$ also includes secondary PM derived from atmospheric reactions of precursor gases, including VOCs, SO_2, NO_X and ammonia (**NH_3**). To estimate secondary particulate, receptor modelling (mining data from long $PM_{2.5}$ speciation datasets) or dispersion and chemical modelling (based on emission inventories, meteorological dispersion and chemical reactions to generate secondary PM) are needed. In New Zealand, the results of PM speciation and receptor modelling have been used to derive the contribution of secondary PM to overall PM concentrations. Several speciation monitoring programs (Auckland, Tokoroa, Baring Head, Christchurch and Timaru) have included specific secondary aerosol markers (eg, ammonium, nitrate, sulphate, organic carbon), while hydrogen (a PM marker for ammonium and hydrocarbon aerosol including secondary organics) is routinely analysed in PM speciation samples. From these data, we have a reasonable understanding of the amount of secondary particulate in urban airsheds and this will be included in the overall assessment of the effects of PM (which will be based on monitoring).

For example, the data show that the sources of urban secondary aerosol are split between anthropogenic and natural emissions of precursor gases and that, as annual averages, secondary sulphate is the greatest contributor to urban secondary aerosol (about 10%, or 1 to 2 $\mu g/m^3$) while secondary nitrate contributes about 0.6 $\mu g/m^3$ for both Auckland (population 1.5 million) and Timaru (population 44,000). Speciation and receptor modelling studies in New Zealand have not yet identified a specific secondary organic aerosol (**SOA**) contributor to urban PM, and while it is likely such PM species are present, $PM_{2.5}$ mass closure analysis suggests that the mass contribution of SOA is likely to be somewhat less than 5%.

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)

As already mentioned, there is a widespread consensus that air pollution itself causes adverse health effects. However, assigning different PM toxicities to different PM sources is less clear. Particles from different sources (eg, 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 could estimate the contribution of different sources to the PM concentrations and include source weighting factors (currently set to 1) in the model to future proof the estimates should robust evidence come available at a later date**.** This is discussed in more detail in section 8.3.

To some extent this concern will be addressed in HAPINZ 3.0 by assessing the effects of $PM_{2.5}$ as well as PM_{10}. 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 Approved methodology

4.3.1 PM_{10} and $PM_{2.5}$

We will attribute PM_{10} and $PM_{2.5}$ to various sources based on actual monitoring data (source apportionment studies) where available. Information on the relevant studies that have been undertaken in New Zealand is summarised in appendix B.

The source apportionment studies attribute PM to the following key categories:

- marine aerosol[13] (which is the main 'natural' component of PM)
- biomass burning (domestic fires in urban areas)
- motor vehicles (covering exhaust, brake/tyre wear and re-entrained road dust)
- secondary PM resulting from atmospheric gas to particle conversion, which includes inorganic secondary PM ammonium, calcium and sodium nitrates and sulphates (from combustion of sulphur-containing fuels in shipping, motor vehicles and industry, as well as natural sources such as oceanic and volcanoes), and organic secondary PM (from the oxidation of VOCs emitted from different anthropogenic, biogenic and geogenic sources)
- crustal material[14] (from various sources such as earth works and construction).
- In some areas, localised sources of PM (such as shipping or specific industrial sources) are also identified.

For areas without monitoring data, source attribution will be based on comparisons with monitored areas that have similar characteristics (including urban/rural classification, topography, meteorology and emissions inventory results).

The estimated contribution of industrial emissions to PM based on source apportionment is very location specific (depending on the industries in the vicinity of the site). We will use the results of source apportionment and emission inventories to estimate the likely contribution of industry in each airshed. This will be an approximation only.

The results of monitoring suggest that open burning is not a significant source of exposure in urban areas. We will identify airsheds where open burning occurs and make a qualitative assessment of the likely contribution of open burning to PM exposure (based on emissions inventory data).

Note: PM emission estimates for other sources (eg, aviation, shipping, rail, as well as off-road construction, farming and agriculture vehicles) are still not reported consistently in emission inventories across New Zealand so these other sources will not be included unless there is supporting information or a specific signal identified in the source apportionment data. Neither can the secondary and natural PM can be inferred from emission inventories.

4.3.2 NO_2

We will assess the effects of exposure to NO_2 from motor vehicles only. This is expected to account for the vast majority of exposure in urban areas.

The effects of other sources will not be quantified but we will review the data and make a qualitative (only) assessment of their likely contribution to exposure.

4.3.3 Relative contributions of different sources to PM health effects

We can estimate the contribution of different sources to PM *concentrations*. However, this does not necessarily translate to the contribution of different sources to PM *effects* if the toxicity varies by source.

[13] Referred to as 'sea spray' in HAPINZ 2.0

[14] Referred to as 'windblown dust' in HAPINZ 2.0

To better address the contribution of different sources to PM health effects in HAPINZ 3.0, we will:

- recognise that the likely contribution of different sources to PM health effects is uncertain and that this needs to be carefully communicated
- consider reporting the effects of anthropogenic (only) air pollution. As an example, Kings College (2015) estimates effects based on the anthropogenic PM concentration, which excludes sea salt.
- estimate exposure and effects of BC, which is from anthropogenic (only) sources
- consider including source weighting factors applied to the exposure-response functions (currently set to 1) in the health effects model to future proof the estimates should robust evidence come available at a later date.

4.3.4 Summary of our approach

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

- marine aerosol (which is the main 'natural' component of PM)
- biomass burning (domestic fires in urban areas)
- motor vehicles (covering exhaust, brake/tyre wear and re-entrained road dust)
- secondary PM (including ammonium sulphates and nitrates and secondary organic carbon)
- crustal material (from various sources such as earth works and construction).

We will use the source apportionment and emission inventories to estimate the likely contribution of industry to PM exposure in each airshed. This will be an approximation only.

We will identify airsheds where open burning occurs and make a qualitative assessment of the likely contribution to PM exposure (based on emissions inventory data). We will investigate including rail, aviation and shipping (where supporting information allow).

NO_2 exposure and effects will be assessed for motor vehicles (only) with qualitative discussion of the likely impact of other sources included where relevant.

5. Selecting health outcomes

This chapter summarises the health outcomes selected for the HAPINZ 2.0 study, reviews developments that have occurred since in epidemiology since and their relevance to New Zealand and outlines the health outcomes to be assessed in the latest HAPINZ 3.0 update. The method for calculating health outcomes is also summarised.

5.1 Approach used in HAPINZ 2.0

HAPINZ 2.0 assessed the following primary health outcomes:

- **Premature mortality** from long-term exposure (PM_{10} annual mean)
  - Adults, aged 30 years and over: 1.07 (1.03 to 1.10) per 10 µg/m^3
  - Infants, aged 1 month to 1 year: 1.05 (1.02 to 1.08) per 10 µg/m^3
- **Hospital admissions** from short-term exposure (PM_{10} daily mean)
  - cardiac hospital admissions, all ages: 1.006 (1.003 to 1.009) per 10 µg/m^3
  - respiratory hospital admissions, all ages: 1.1 (1.06 to 1.017) per 10 µg/m^3
- **Restricted activity days** from long-term exposure ($PM_{2.5}$ annual mean[15])
  - restricted activity days, all ages: 0.9 (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: 1.20 (1.07 to 1.33) per 10 µg/m^3 (PM_{10} annual mean)
- Respiratory hospital admissions for children aged 1 to 4 years: 1.01 (1.006 to 1.017) per 10 µg/m^3 (PM_{10} daily mean)
- Respiratory hospital admissions for children aged 5 to 14 years: 1.03 (1.0 to 1.05`) per 10 µg/m^3 (PM_{10} daily mean)

Sensitivity analyses were conducted on the 95% confidence intervals or upper and lower bounds of the 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 $PM_{2.5}$ as opposed to PM_{10} follows:

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

5.2 Developments that have happened since

This section reviews the developments in the literature on health impacts of long-term air pollution exposure since HAPINZ 2.0. We consider robustness of exposure-response functions by pollutant, the availability of exposure and health outcome data, and the likely public health

[15] Assuming that 60% of annual PM_{10} in urban areas and 40% of annual PM_{10} in rural is $PM_{2.5}$

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-term exposure and long-term exposure to PM are associated with a wide range of health endpoints, including premature mortality and a range of cardiovascular and respiratory diseases. There is increasing evidence of effects of PM exposure on adverse birth outcomes (Laurent, Hu et al, 2016; Huang, Woodruff et al, 2018; Seeni, Williams et al, 2019), childhood respiratory diseases (Guarnieri and Balmes, 2014; Bowatte, Lodge et al, 2015; Burte, Nadif et al, 2016; Hehua, Qing et al, 2017; Khreis, Kelly et al, 2017; Munoz, Barreiro et al, 2019; Williams, Phaneuf et al, 2019), diabetes and dementia (WHO, 2013; Peters et al; 2019). The immune system is also involved in the toxicity of inhaled pollutants, especially in the case of respiratory diseases (Falcon-Rodriguez, Osornio-Vargas et al, 2016).

Considerable scientific effort has been directed to identifying specific constituents of PM that are primarily responsible for health impacts. In general, there are stronger and more consistent associations between health outcomes and $PM_{2.5}$ than for PM_{10}. A recent study reported that the sulphur content of PM was most strongly associated with mortality (Beelen, Hoek 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-Nielsen, Beelen et al, 2016)

Ostro (2015) reported that $PM_{2.5}$ from high sulphur fuel combustion was associated with mortality in teachers (Ostro, Hu et al, 2015). However, there were no significant associations with $PM_{2.5}$ from petrol or diesel for land-based transport, wood smoke or meat cooking. Thurston, Burnett et al (2016) investigated associations between $PM_{2.5}$ from different sources and ischaemic heart disease mortality (Thurston et al, 2016). 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 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, Luben et al, 2019)

Health outcomes from exposure to BC could be estimated based on exposure-response functions from WHO (2012) or from a more recent cohort study undertaken in Denmark (Hvidtfeldt et al, 2019). The latter includes an exposure-response function for all-cause mortality of 1.09 (1.04 to 1.15) per 1 $\mu g/m^3$ BC. However, there is a major study currently underway - Effects of Low-Level Air Pollution: A Study in Europe (**ELAPSE**) - which is focussing particularly on the effects of BC, with results due for release in 2019/2020.[16]

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.

5.2.2 NO_2 health endpoints

A review reported statistically significant associations between NO_2 and mortality, but with substantial heterogeneity of the effect size (Hoek, Krishnan et al, 2013) while a meta-analysis reported more consistent associations between long term exposure to NO_2 and mortality (Faustini, Rapp et al, 2014). In the meta-analysis, the associations were broadly similar in strength to those of $PM_{2.5}$, and in four studies, 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, COMEAP (UK) 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 long term exposure on mortality, several members of the committee recommended using an adjusted coefficient of 1.006 to 1.013 per 10 $\mu g/m^3$ of NO_2, although other members of the committee considered that the evidence was not strong enough to justify this. However, 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 (1.023 (95% CI: 1.008, 1.037 per 10 $\mu g/m^3$ 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, Lodge et al, 2015; Burte, Nadif et al, 2016; Khreis, Kelly et al,

[16] https://www.healtheffects.org/research/ongoing-research/mortality-and-morbidity-effects-long-term-exposure-low-level-pm25-black

2017; Khreis and Nieuwenhuijsen, 2017). 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 (1.02, 1.07) per 4 $\mu g/m^3$ NO_2 (Khreis, Kelly et al, 2017). Another meta-analysis reported associations between air pollution exposure and asthma exacerbations (Orellano, Quaranta 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 exposure (Achakulwisut, Brauer et al, 2019). Exposure estimates at 100 m resolution were used for this study, and are available globally (including for NZ).

The 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)

To calculate total mortality from long term exposure to $PM_{2.5}$ and NO_2, we recommend adding together the effects of $PM_{2.5}$ and NO_2, but reducing the effects of NO_2 by 30% to account for the possible overlap in effects (as recommended by Kings College, 2015).

5.2.3 Sensitive populations

People with poor diet, chronic disease, children, the elderly, pregnant women and certain ethnic groups may be particularly susceptible to the health impacts of air pollution (Wang, Kloog et al, 2016; Tibuakuu, Michos 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, Krishnan et al, 2013)

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, Blakely et al, 2012).

5.3 Approved methodology

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 endpoints we will assess are predominantly long-term (which rely typically on annual average concentration datasets).

Where short-term effects have been included, we will estimate effects also based on the annual mean to enable more representative coverage across New Zealand. While this is not ideal, we note that the major impact of air pollution exposure on life expectancy (the dominant social cost) is through the gradual, cumulative effects on chronic disease as discussed previously in section 2.2.

5.3.1 Cohort study

Following on from the earlier work (Hales, Blakely et al, 2012), we will undertake further study to investigate the susceptibility of Māori and potentially extend the assessment to Pasifika peoples. Since the previous study was published, data availability, methods of exposure estimation and of data analysis have advanced considerably. Since there are stronger and more consistent associations between most health outcomes and $PM_{2.5}$ than PM_{10}, we recommend using $PM_{2.5}$ as the principal exposure metric for mortality. However, we will also assess PM_{10}.to compare with the exposure-response functions used in HAPINZ 2.0.

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

Base cohort study

A study of mortality from non-external causes in relation to estimated long-term average air pollution exposure at small area level has the potential to refine our understanding at relatively low cost. This study requires estimates of the spatial pattern of air pollution exposures for census area units or other administrative boundaries to be imported into the IDI.

The simplest cohort study, which is feasible within the available budget, involves statistical models of mortality in adults (2013-2017), for all non-external causes, and by sub-group of major cause with adjustment for confounding by ambient temperature, age, sex, ethnicity, equivalized household income and smoking status, analysed in relation to the estimated exposure to $PM_{2.5}$ and PM_{10} at the place of usual residence at baseline (2013).

We will use Cox proportional hazards regression models in order to estimate years of life lost (**YLL**). Effect modification will be assessed in subgroup analyses and by including interaction terms in the model.

Additional analyses

The following additional analyses will be undertaken to increase the study power and extend the assessment to different health outcomes of particular relevance (such as childhood asthma)

Extending the period of exposure in relation to health impacts

One refinement concerns the relevant period of exposure in relation to health impacts. As discussed earlier, air pollution has both short-term and long-term health impacts. The long-term effects would ideally be assessed with reference to lifetime (including *in utero*) exposures.

This is not practical for the present study, but extending the study to include 2013 census data, may make it possible to link cases to historical addresses and so derive more inclusive estimates of past exposure. Consequently, we will analyse data for the years 2006-2017.

Undertaking a finer scale assessment of NO_2 effects

For models of NO_2 effects, it is necessary to assess exposure at sub-kilometre level, due to the strong exposure gradients near to roads. For the same reason, for NO_2 exposure, the use of address of usual residence is a more serious limitation. We will investigate the feasibility of performing the analysis at meshblock scale, with geocoding of cases refined by inclusion of work address as well as address of usual residence.

Extending the mortality cohort study to cover morbidity

Long-term air pollution exposure leads to chronic diseases as well as mortality. Life shortening is the most important health impact of air pollution, but effects on morbidity can also be estimated.

As for mortality, the most directly relevant evidence for effects on morbidity is derived from studies in the population of interest, rather than by extrapolation of studies in external populations. Given the availability of detailed data on hospital admissions, the design of the mortality cohort study will be extended to analysis of hospital data.

Developing proxy indicators for childhood asthma

We will investigate developing suitable proxy indicators for **childhood asthma incidence or prevalence** so external exposure-response estimates can be applied to these (Khreis, Kelly et al, 2017; Orellano, Quaranta et al, 2017).

5.3.2 Exposure-response functions for health outcomes

The exposure-response functions we will adopt fall into two categories:

- Those which WHO has confirmed there is sufficient data to enable quantification of effects and the effects are largely additive.
- Those which enable back-casting of health effects with the previous HAPINZ.

In addition, we will establish appropriate proxy indicators to enable assessment of childhood asthma.

Primary exposure-response functions

Of most importance, we will assess the following pollutant-outcome pairs for which WHO has confirmed that there is enough data to enable quantification of effects, and for which the effects are approximately additive (Héroux et al, 2015). These include:

- **Premature mortality and YLL** from long-term exposure ($PM_{2.5}$ annual mean, no threshold)
  - Adults, aged 30 years and over: 1.062 (1.040 to 1.083) per 10 µg/m$^{3.}$
- **Premature mortality and YLL** from long-term exposure (NO_2 annual mean over 0 µg/m^3 and NO_2 annual mean over 20 µg/m^3) [17]
  - Adults, aged 30 years and over: 1.055 (1.031 to 1.080) per 10 µg/m^3
- **Hospital admissions** from short-term exposure (based on $PM_{2.5}$ annual mean)
  - CVD (including stroke), all ages: 1.0091 (1.0017 to 1.0166) per 10 µg/m^3
  - Respiratory diseases, all ages: 1.0190 (0.9982 to 1.0402) per 10 µg/m^3
- **Hospital admissions** from short-term exposure (based on NO_2 annual mean)
  - Respiratory diseases, all ages: 1.0180 (1.0115 to 1.0245) per 10 µg/m^3

Note: Ethnicity-based outcomes resulting from long-term exposure to $PM_{2.5}$ will be investigated in the base cohort study which may yield updated exposure-response functions for $PM_{2.5}$. The NO_2 exposure-response estimates are less certain than those for $PM_{2.5}$.

Secondary exposure-response functions (for back-casting)

We will also assess the following pollutant-outcome pairs used in the HAPINZ 2.0 report, to allow comparison with the previous results. These include:

- **Premature mortality** from long-term exposure (PM_{10} annual mean)
  - All Adults, aged 30 years and over: 1.07 (1.03 to 1.10) per 10 µg/m^3
  - Māori adults, aged 30 years and over: 1.20 (1.07 to 1.33) per 10 µg/m^3
  - Infants, aged 1 month to 1 year: 1.04[18] (1.02 to 1.07) per 10 µg/m^3
- **Restricted activity days** from long-term exposure ($PM_{2.5}$ annual mean)
  - restricted activity days, all ages: 0.9 (0.5-1.7) per 10 µg/m^3

Note: Ethnicity-based outcomes resulting from long-term exposure to PM_{10} will be investigated in the base cohort study which may yield updated exposure-response functions for PM_{10}. We will not be back-casting hospitalisations as the recommended primary exposure-response functions based on $PM_{2.5}$ and NO_2 are more robust.

Childhood asthma

We will assess childhood asthma by developing suitable proxy indicators. Relevant exposure-response functions include:

[17] Note Héroux, Anderson et al, 2015 conclude that calculation of impacts for levels over 20 µg/m^3 only, ignoring potential effects at lower concentrations, may underestimate effects so we propose to assess impacts for levels over 0 µg/m^3 as well as 20 µg/m^3.

[18] Note this is the updated exposure-response-function from Héroux, Anderson et al, 2015 and differs slightly to that used in HAPINZ 2.0 (refer section 5.1)

- **Incidence** of childhood asthma due to long-term exposure (based on NO_2 annual mean): 1.05 (1.02 to 1.07) per 4 µg/m^3 (Khreis, Kelly et al, 2017)
- **Exacerbations** of childhood asthma due to short-term exposure (based on $PM_{2.5}$ annual mean: 1.028 (1.009 to 1.047) per 10 µg/m^3 (Orellano et al, 2017).

5.3.3 Summary of our approach

The primary health outcomes of the assessment include:

- **Premature mortality and YLL** from long-term exposure ($PM_{2.5}$ annual mean, no threshold)
  - Adults, aged 30 years and over: non external causes (Héroux et al, 2015) 1.062 (1.040–1.083) per 10 µg/m^3
  - Adults, aged 30 years and over: YLL *age specific exposure-response from the New Zealand cohort study*
- **Premature mortality and YLL** from long-term exposure (NO_2 annual mean over 0 µg/m^3 and over 20 µg/m^3)
  - Adults, aged 30 years and over: non external causes (Héroux et al, 2015) 1.055 (1.031–1.08) per 10 µg/m^3
- **Hospital admissions** from long-term exposure ($PM_{2.5}$ annual mean)
  - CVD (including stroke) hospital admissions, all ages (Héroux et al, 2015) 1.0091 (1.0017–1.0166) per 10 µg/m^3
  - Respiratory disease hospital admissions, all ages (Héroux et al, 2015) 1.0190 (0.9982–1.0402) per 10 µg/m^3

  Or:

  Cardiac hospital admissions and Respiratory hospital admissions, *age specific exposure-response from the New Zealand study*
- **Hospital admissions** from long-term exposure (NO_2 annual mean)
  - Respiratory disease hospital admissions, all ages (Héroux et al, 2015) 1.018 (1.0115-1.0245) per 10 µg/m^3

  Or:

  Respiratory disease hospital admissions, *age specific exposure-response from the New Zealand study, children and adults separately*

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

- **Premature mortality** from long-term exposure (PM_{10} annual mean)
  - Adults, aged 30 years and over: (Hales et al, 2012) 1.07 (1.03 to 1.10) per 10 µg/m^3
  - Māori adults, aged 30 years and over: (Hales et al, 2012) 1.20 (1.07 to 1.33) per 10 µg/m^3
  - Infants, aged 1 month to 1 year: (Héroux et al, 2015) 1.04 (1.02 to 1.07) per 10 µg/m3
- **Restricted activity days** from long-term exposure ($PM_{2.5}$ annual mean)
  - Restricted activity days, all ages: (ALA, 1995) 0.9 (0.5-1.7) per 10 µg/m^3

Or:

Premature mortality *exposure-response functions from the New Zealand study ($PM_{2.5}$ annual mean).*

Childhood asthma outcomes of relevance to New Zealand include:

- **Incidence** due to long-term exposure (NO_2 annual mean): (Khreis, Kelly et al, 2017) 1.05 (1.02 to 1.07) per 4 µg/m^3
- **Exacerbations** due to short-term exposure ($PM_{2.5}$ annual mean: (Orellano et al, 2017) 1.028 (1.009 to 1.047) per 10 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 starts by setting out the nature of the social costs of air pollution and the implications for measuring average and marginal costs.

6.1 Approach used in HAPINZ 2.0

HAPINZ 2.0 estimated the social costs as follows:

- All costs were estimated in New Zealand dollars as at June 2010.
- A **transport risk (road safety) based Value of Statistical Life (VOSL) of NZ$3.56 million** was used for all cases of premature mortality due to air pollution.
- Average costs of **NZ$6,350 (cardiovascular)** and **NZ$4,535 (respiratory)** were used for all **hospital admissions**. These included medical costs and loss of output during hospitalisation but did not include loss of life quality due to prolonged pain and suffering.
- Restricted activity days were valued at **NZ$62**, based on the average loss of output per day (irrespective of a working or non-working day).

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 that have happened since

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 economic 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.[19] 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, the Ministry of Transport (**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

[19] See for example ExternE studies, eg, Rabl A & Spadaro J et al (2005) and Hohmeyer (1998)

this hierarchy of effects but have concentrated on health effects. Health effects are the focus of this economic analysis also.

6.2.1 Policy studies in New Zealand

Although HAPINZ 2.0 was an analysis of the total costs of air pollution, the values have been used in policy studies which are based on marginal cost analysis, ie, a measure of the change in total costs resulting from a small change in concentrations (or emissions) of pollutants. As we discuss further below, marginal costs may be significantly different from average costs because the most important effects are cumulative and the benefits depend on repair to damaged health.

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.

Ministry for the Environment (2004)

The Ministry for the Environment analysed the costs and benefits of proposed national environmental standards for air pollution (MfE, 2004). The study included modelling of the expected impacts of the air quality standards on concentrations of PM_{10} in 24 sites across New Zealand for the years 2001 to 2021. MfE estimated the number of premature deaths falling by 54 per annum by 2020 as a result of the standards.

It uses these results with a value of statistical life of $2.5 million, adjusted downwards to reflect age (see discussion in the next section). Total benefits were estimated to include 625 premature deaths prevented by 2020, total benefits with a present value (to 2004 at a 10% discount rate) of $429 million, of which $420 million were from lives saved. The costs of the policy measures were estimated to be $111 million, yielding a net present value (**NPV**) of $318 million ($554 million at a 5% discount rate). The analysis was also expressed as a cost per life saved of $177,000 ($232,000 at 5% rate), suggesting that much smaller benefit levels would justify the costs of the standards.

NZ Institute of Economic Research (2009)

The NZ Institute of Economic Research (**NZIER**) conducted a review and update of the 2004 Cost Benefit Analysis (**CBA**) (NZIER, 2009). The authors suggested several shortcomings of the original analysis, including (on the benefit side):

- the reduction in the VoSL because of the expected age of those affected – NZIER suggests that there is no empirical basis for assuming either that elderly people are most affected or that the VoSL differs with age (we discuss these issues later)
- the absence of any assessment of the costs of loss of life quality for those who suffer from chronic ill-health (see later)
- that no explicit allowance was made for medical costs saved by reducing bad air days.

The approach retains the structure of the 2004 analysis, but it increases the costs of some impacts, including an increased VoSL of $3.35 million (table 8). NZIER also reduced the discount rate from 10% to 8%, consistent with updated NZ Treasury guidance on discount rates for public policy (NZ Treasury, 2008).

Table 8: Costs of health impacts assumed in NZIER study

Health effect	Cost per event
Premature mortality (all)	$3.35 million
Hospitalisation (medical costs per event)	$7,700
Hospitalisation (loss of income per day)	$713
Restricted activity day	**$46.50**

Assumes $60.43/day, 6.8 days in hospital and 5 days recuperation
Source: NZIER (2009).

NZIER's updated estimate of benefits of standards being met by 2013 was $1,289 million (up from $429 million in the 2004 study).

Market economics (2013)

McIlrath (2013) assessed the costs and benefits of reducing emissions associated with domestic fires in Auckland. The study used the cost per case assumptions from the HAPINZ 2.0 study section 6.1. However, it added a cost per chronic obstructive pulmonary disease (**COPD**) using values included in HAPINZ 1.0, despite the updated HAPINZ 2.0 not including COPD effects because of "limited scientific consensus on the relationships with air pollution" (Kuschel et al, 2012).

Covec/Tonkin + Taylor (2015)

A 2015 study for Ministry of Transport addressed the costs and benefits of introducing Low Emission Zones (**LEZs**), regional emissions testing and road pricing in Auckland (Denne and Atkins 2015). It discussed developments in international CBAs including those that took account of cessation lag[20] and the use of values of life years lost (**VoLYs**) rather than VoSL. Using simple life tables for analysis, estimates were made of the life years gained from air quality policy options. The results showed a significant difference between the net benefits using a VoLY and a VoSL-based analysis.

6.2.2 Valuing mortality impacts

In this section we address several issues that are raised by the studies to date in New Zealand. Specifically, these are:

- Marginal effects – the implications for analysis of health effects being dominated by chronic mortality, with full benefits only emerging after some time;
- Whether the mortality impacts should be characterised as premature deaths and whether this affects the analysis;
- VoSL vs VoLY.

[20] This was in recognition 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

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).

These mechanisms are important in defining the marginal effects.

The delay issues have been recognised in international studies for some time. In the United States of America (**US**), the United Kingdom (**UK**) and elsewhere in Europe, studies of the costs and benefits of air pollution use lagged benefits. This reduces the present value of benefits because of the impacts of discounting. The approaches used are still developing and there is increased focus on studies that are testing the extent of lag, including some US studies that suggest a significant proportion of the benefit is gained soon after a reduction in emissions (Lepeule et al, 2012).

Against this, the UK Committee on the Medical Effects of Air Pollutants (**COMEAP**)[21] suggests that:

> the US cohort studies do not, and cannot, lead to any clear conclusion on the likely latency between a change in average pollution levels and the appearance of effects. (COMEAP, 2009)

while also noting that

> …current thinking suggests that the exposure in the weeks, months and short number of years prior to death is the most biologically relevant time period of exposure for deaths from cardiovascular (or cardiorespiratory) causes, whereas the effect of exposure on lung cancer is likely to have a longer latency.

United States of America

In the USA, prior to 2004, the Environmental Protection Agency (**US EPA**) and the Health Effects Subcommittee (**HES**) used a weighted 5-year time course of benefits in which 25% of the PM-related mortality benefits were assumed to occur in the first and second year, and 16.7% were assumed to occur in each of the remaining 3 years (US EPA, 2004a). Subsequently, following a suggestion from the EPA (US EPA, 2004b), the Science Advisory Board (**SAB**) 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 and 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 (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).

[21] COMEAP has been established as an expert committee to advise the UK government on all matters concerning the health effects of air pollutants

European Union

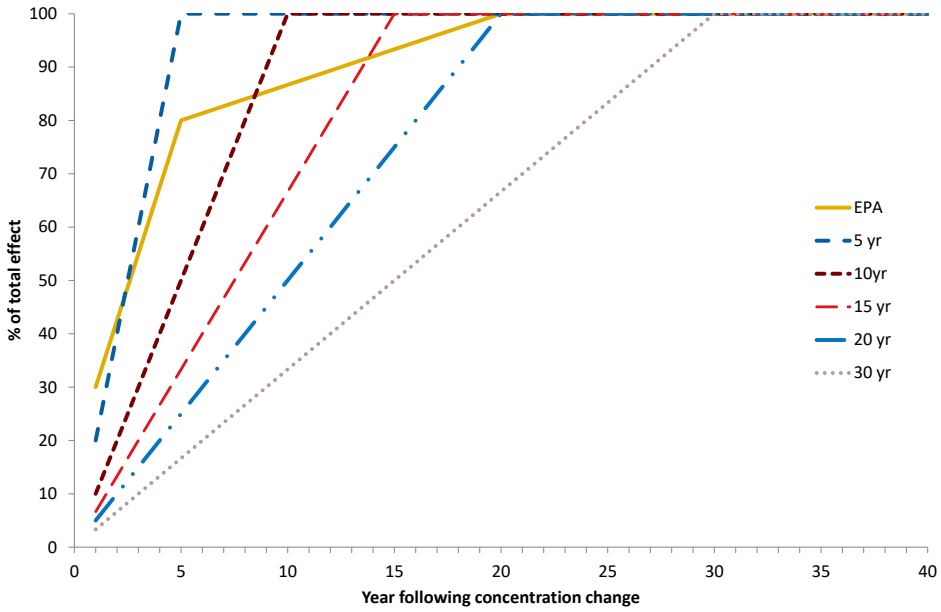
Work for the European Commission has examined the effects associated with a 1-year pulse change (ie, a sudden reduction in pollution for one year) as a way to understand the marginal effects (AEA Technology Environment, 2005). Here, in contrast to a simple immediate 6% increase in mortality for a 10 μg/m^3 increase in $PM_{2.5}$ concentrations otherwise used, 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 (NECD) 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 5, 10, 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 8. Table 9 shows the implications of these different lag structures on damage estimates in relative terms, using different discount rates. At a 6% discount rate, usually used for public policy analysis in New Zealand, the US EPA approach reduces the impact to 84% of what it would be with no lag and a 30-year lag reduces the impact to 49%.

Figure 8: Selection of lag structures examined by Walton (2010)



Source: Walton (2010)

Table 9: Implications of lag structures for impact estimates (Index: no lag = 100)

Discount Rate	No lag	EPA	5 yr	10yr	15 yr	20 yr	30 yr
0.0%	100	100	100	100	100	100	100
2.0%	100	93	96	92	87	83	76
4.0%	100	88	93	84	77	71	60
6.0%	**100**	**84**	**89**	**78**	**69**	**61**	**49**
8.0%	100	80	86	72	62	53	41

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. But a zero lag with an instantaneous response to reductions in emissions over-estimates the measured impact.

The US EPA lag formula appears to be the most widely used approach, including most recently by Birchby et al (2019) in advice to Department for Environment, Food & Rural Affairs (DEFRA) in the UK. Within the identified range of possible lag structures, it is conservative, ie, 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 over 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 (2010) addressed this issue to some extent by limiting the analysis (in sensitivity analysis) to those who were in the same location five year's previously. However, in general, data limitations will preclude this analysis.

Premature death or life years lost

Impact studies in New Zealand have characterised the mortality impacts as increases in premature deaths, building on dose-response functions that express the impacts in that way. Using premature mortality makes estimating the costs simple; the estimated number of premature deaths is multiplied by the value of a statistical life (VoSL). However, other researchers and jurisdictions have measured the effects in terms of the life years lost and in this section we explore these issues.

The nature of the effect

The difficulty with the concept of premature mortality is that, effectively, everyone dies prematurely, of something. Air pollution may be a causal effect, but it is adding to other factors that would lead to premature deaths, regardless. Thus, the number of deaths quantified as being attributable to air pollution is less than the total number of deaths for which air pollution is a contributory factor and there are likely to be no deaths for which air pollution is the sole cause of death; other factors will have contributed to their health.

When life is shortened by the effects of air pollution, death rates increase amongst younger age groups and, because people die once only, death rates necessarily decrease for older age groups. Deaths from air pollution cannot be observed (Rabl, 2003); only changes in age-specific all-cause death rates can be. Epidemiological studies used to observe changes in death rates have separated out the age groups. For example, Hales et al (2010) developed dose-response functions by analysing data for those aged under 75.

Some studies have provided estimates of the impacts on life expectancy (or life years). Pope et al (2009) find a 0.61 year increase in life expectancy from a 10 $\mu g/m^3$ reduction in $PM_{2.5}$; Künzli et al (2000) note that life expectancy is shortened by about 6 months per 10 $\mu g/m^3$; COMEAP estimates that for England and Wales, a population-weighted average concentration of $PM_{2.5}$ of 9.46 $\mu g/m^3$ throughout their life results in approximately 6.5 months lower life expectancy (COMEAP, 2010).

These studies provide a basis for expressing the effect in terms of life years lost. The change in the number of premature deaths may be useful as a population aggregate, but it is important not to consider these as identifiable individuals.

Premature mortality as a summary of the effects has particular difficulties when used in policy analysis. If pollution is reduced, premature deaths are not eliminated and any changes in age-specific death rates are likely to be caused by life extension for all (or many) people rather than the elimination of premature death for some (and not for others). In other words, the impacts are not on the number of premature deaths but on the prematurity of the deaths. If it is assumed that the cost of air pollution (and the benefit of air quality policy) is associated with the fact of prematurity, rather than the extent of prematurity, then policy may be considered to have no measurable benefit: people still die prematurely.

The observed (and/or predicted) change in the number of premature deaths is simply the statistical outcome of changes in life expectancy. It is useful for this to be clearly expressed and valued.

Addressing the question of presentation (as premature mortality or life years lost), COMEAP notes that:

> there is, to some extent, 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,[22] which means it cannot be compared simply with the effects of road traffic accidents, suicide, or HIV/AIDS, which by comparison 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 life-years gained).

International practice

In the 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, eg, in the economic analysis to inform the Air Quality Strategy (DEFRA et al, 2007). Birchby et al (2019) in their report on damage costs to DEFRA use a VoLY-based approach.

In the EU, a 2005 CBA of the Clean Air for Europe (CAFE) programme recommends that:

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

[22] 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

However, in response to peer review recommendations, they also include estimates of the number of deaths per year attributable to long-term exposure to ambient $PM_{2.5}$ despite their acknowledging that it will over-estimate the impact. While they argue that it has computational problems but is easy to understand.

Despite the problems with premature deaths as a characterisation, we note that in the US the practice[23] has been more to use premature deaths and VoSL rather than life years lost and VoLY. However, the government's 2003 guidance on regulatory impact analysis suggests that it is :

> appropriate to consider providing estimates of both VSL and VSLY, while recognizing the developing state of knowledge in this area" (US Office of Management and Budget, 2003).

Consistent with this, the 2011 CBA of the Clean Air Act Amendments (**CAAA**) included results in terms of avoided premature mortality, life-years lost and changes in life expectancy (US EPA, 2011).

The emerging international consensus is moving towards the use of life years lost as the key metric for analysis. We agree with this direction. Our preference is to use life years and value of life years as the primary means of quantifying the monetary impacts of air quality impacts.

This emphasis is particularly for policy (marginal) analyses, but can apply also to the analysis of total impacts.

Determining Life Years Lost

Estimating impacts using life years lost employs a similar methodology to estimating impacts on premature mortality, but incorporating lifetables.

For each death, the remaining life expectancy is calculated, based on 5-year age categories from the life tables. These remaining life expectancies take into account the changing the risk of death for each age category. The life years lost are simply the number of remaining life years for someone in that age group, at the time of death.

The years of life lost attributable to exposure to air pollution are estimated in a similar way to estimating the attributable number of deaths due to air pollution, by applying the population attributable fraction (based on the exposure-response function, and air pollutant exposure level).

The years of life lost attributable to air pollution are then combined with VoLY to estimate the total social cost of air pollution premature mortality.

Estimating VoLY

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. This is the approach adopted in the cost benefit analysis of the EU CAFE programme (AEA Technology Environment, 2005). The formula used is:

[23] For example, in Industrial Economics (2006)

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

Where: VoSL = value of statistical life, currently $4.34 million (MoT, 2019)
r = the discount rate
n = years over which the annuity is calculated (ie, the life expectancy of the average crash victim)

This formula is represented by the PMT function in Excel, ie, VoLY = PMT(*r, n, -VoSL*) .

In 2018 the average age of death for car drivers is 45,[24] suggesting a weighted average life expectancy of approximately 38 years.[25] This would suggest a VoSL-derived VoLY (using a 6% discount rate) of approximately $292,000. The appropriate discount rate is that which would apply to the individuals: a social rate of time preference, rather than the Treasury's recommended discount rate for public policy analysis which reflects an opportunity cost of investment. Previous analyses of the social rate of time preference for New Zealand suggest that it would be in the 3-5% range.[26] Table 10 shows the estimated value of a VoLY (based on VoSL) at different discount rates when discounted over 38 years.

Table 10: Estimation of VoLY from VoSL

Indicator	Value
VoSL	$4.34 million
VoLY (@ 3%)	$192,954
VoLY (@ 6%)	$292,334
VoLY (@ 8%)	$366,899

Telfar-Barnard and Zhang (2019) used this same approach in estimating the costs of respiratory disease in New Zealand; a 3% discount rate and the 2015 VoSL was used 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.

Values from surveys

Other approaches to defining VoLY have used survey-based approaches. Swedish researchers Johannesson and Johansson administered a telephone survey in 1995 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 and Johansson, 1997).

The resulting VoLY values are between US$700 and US$1,300 in 1995 dollars.

[24] Ministry of Transport crash statistics

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

[26] See for example: Ministry of Economic Development (2006); Parker (2009)

Half of the sample had a willingness-to-pay (**WTP**) of zero; the average of positive WTP was about US$2,700. These values are estimates of the present value of some future benefit.

Dolan et al (2008) review other studies with similarly low values (eg, £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 cites Chilton et al (2004) who derived VoLYs of £6,040 to £27,630 in 2002 prices. More recently surveys in a number of European countries were undertaken to suggest 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 (2004) study, Birchby et al (2019) use a VoLY of £42,800 within a range of £32,000 – £53,300 (£2017 prices).

QALY-based values

Dolan et al (2008) suggest that VoLYs can be estimated from using a ratio to a quality adjusted life year (**QALY**), essentially a life year in perfect health. They suggest a ratio of 1 QALY to 1.2 VoLYs.[27]

In New Zealand health studies, it is common to use a threshold value for how much to spend to achieve a QALY, eg, using GDP per capita as the maximum amount to pay to achieve a QALY.[28] GDP per capita in New Zealand is currently approximately $61,000; this would suggest a VoLY of approximately $51,000.

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 these morbidity impacts are estimated to have damage costs in the order of 8-10% of the mortality impacts,[29] although HAPINZ 2.0 estimated costs totalling only 2.2% of total social costs (see table 8 previously).

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) summing to $6,350 and $4,535 per hospitalisation (June 2010 prices) for cardiovascular and respiratory hospital admissions (**CHA** and **RHA**) respectively. These were based on NZIER (2009) which used hospitalisation costs from traffic accidents (from the MoT social costs of road crashes and injuries report),[30] adjusted for the days in hospital for RHAs and CHAs. These estimates were updated in HAPINZ 2.0 to take account of more recent information on the length of hospital stays: the values used were 5 days for CHAs and 3.3 days for RHAs. The hospitalisation costs were estimated as $6,350 and $4,535 for CHAs and RHAs respectively (table 11).

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

[28] See, for example: Webber-Foster et al (2014)

[29] WHO Regional Office for Europe and OECD (2015)

[30] The latest version is Ministry of Transport (2019)

Table 11: Hospitalisation costs in HAPINZ 2.0

	Cardiovascular	Respiratory
$/day	$674.60	
Follow-up ($/day)	$333.33	
Fixed (emergency) cost	$1,000	
Lost income ($/day)	$62	
Days	5	3.3
Total ($2010)	**$6,350**	**$4,535**

Source: Kuschel et al (2012); Statistics NZ Infoshare PPI020AA (Health Care and Social Assistance)

Kuschel et al (2012) note that the loss of output after hospitalisation has not been included. In the sensitivity analysis, a loss of life quality after hospitalisation is added based on 10% of the VoSL (ie, $356,000).

Telfar-Bernard and Zhang (2019) estimate total costs from RHAs of $333 million in 2015 values, averaging approximately $4,215 per RHA.[31] Using the HAPINZ 2.0 numbers for hospital costs only (ie, ignoring the follow-up and lost-income costs), and inflating to mid-2015, would produce costs of approximately $3,400/RHA. The National Health Committee, an independent statutory body advising the New Zealand Minister of Health, estimated costs for different types of CHA (table 12).

Table 12: Cardiovascular disease impacts and costs (2012 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
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**

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

[31] This is based on a respiratory hospitalisation rate of 1721.8 per 100,000 (Table A100 on p145) and a NZ population of 4,595,703 (p28) – approach recommended by Lucy Telfar-Barnard (personal communication)

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

Source: National Health Committee (2013)

The weighted average cost (for the 2011/12 financial year) was $9,122/CHA and an average of 3.9 days ($2,334/day). Using the same Producers Price Index (**PPI**) as used in table 8, this would inflate to $10,020 in 2019 Q2 values ($2,564/day).[32]

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.

Childhood asthma

The social costs of childhood asthma were not evaluated in HAPINZ 2.0.

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, or $2,026 per hospitalisation for an average of 1.45 days.

6.2.4 Damage costs and valuing CO_2 emissions

Damage costs are a way to value changes in emissions to air to compare the benefits to society of a change in policy/operation versus the cost of implementing the change. They can also be used to compare options to identify which will produce the best overall outcome. Many government agencies internationally publish relevant values to be used in the assessment of costs and benefits of policy options in their jurisdictions (eg, DEFRA, 2019).

Damage costs can be used to capture benefits of emission reductions of both harmful pollutants (eg, PM_{10}) and greenhouse gases (GHGs eg, CO_2). As an example, The Guide to Project Evaluation Part 4 published by Austroads (2012) includes unit values of emissions in AUD$ per tonne (as at 2010), shown in table 13.

In New Zealand, the application of damage costs has largely been in transport projects, such as assessing environmental outcomes of roading infrastructure projects (NZTA, 2018) 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 translate more or less into exposure) and reviewing overseas values as a crosscheck. Unit costs of CO_2 emissions are included in the EEM based on values in Austroads (2012), which in turn are based on European estimates of global damage costs.

[32] Index values (PPI020AA) of 1027 (2012 Q1) and 1128 (2019 Q2)

Table 13: Unit values of emissions in $/tonne published by Austroads (2012)

Pollutant	Costs in AUD/tonne	Value Base Date
CO_2	$52.40	2010
PM_{10}	$332,506	2010
NO_X	$2,089	2010
CO	$3.30	2010
HC	$1,047	2010

While determining damage costs to society resulting from harmful emissions is relatively straightforward, establishing comparable costs for GHG emissions (CO_2) is more difficult because the effects are global rather than purely local.

When costs and benefits are identified for use in a CBA, or other analysis, a perspective is taken. Normally, for policy purposes, costs and benefits are identified as those that accrue to New Zealand and/or New Zealanders. For example, if evaluating a policy change that results in increased exports of New Zealand goods, the benefits to New Zealand are the export value of those goods, less the opportunity costs of their production; we do not count as a benefit any surplus obtained by the consumers in some other country who may have been willing to pay more for the goods they import. Likewise, we do not usually take account of the environmental costs of production in some other country when the costs of imported goods are included in a national policy or investment analysis. Usually it is assumed that the exporting country should decide itself on the right level of environmental or other protection that might affect the production costs.

CO_2 emissions pose a different problem in that, because the molecules are long-lived and mix thoroughly in the atmosphere, the environmental impacts of emissions from New Zealand are borne largely by other countries. Because of this characteristic of CO_2 emissions, few if any countries have an incentive to act alone and commitments to reduce emissions are being coordinated internationally. New Zealand has agreed to limit its emissions within national emission budgets.

This then poses the dilemma. **Should the costs of CO_2 emissions be counted in the same way as other effects of policies (ie, based only on the costs to New Zealand) or should they include the wider damage costs falling on people in other countries?**

New Zealand costs only

If taking a New Zealand-centric approach, as is usual in CBA, the damage costs of CO_2 emissions originating from New Zealand falling on New Zealand are very small; they are effectively zero. However, what is more significant is that any additional emissions from New Zealand impose a cost because there is a requirement to take additional action to come back into compliance with the national cap on emissions (presuming business as usual emissions are greater than the cap). CO_2 emissions from fossil fuel combustion are priced in the market via the emissions trading scheme (**ETS**) and are incorporated into fuel prices.[33] The costs of New Zealand Units (**NZUs**) under the emissions trading scheme (ETS) provides a readily available number to assign to this cost.

[33] For emissions accounting, carbon in fuels is assumed to completely oxidise to CO_2.

This approach is consistent with that used elsewhere. For example, in the UK, originally a damage cost-based approach was used for public policy purposes. This was defined as a Shadow Price of Carbon, based on estimates of the lifetime damage costs associated with GHG emissions drawn from the Stern Review (known as the Social Cost of Carbon) (UK Department of Energy & Climate Change, 2009). But following the introduction of limits on emissions, the approach shifted to one based on estimates of the abatement costs that will need to be incurred to meet specific emissions reduction targets. Now, "short-term traded carbon values" are used to for valuing the impact of government policies on emissions from sectors covered by the EU ETS (UK Department of Business, Energy & Industrial Strategy, 2018). Short-term traded values are estimated from the average daily settlement prices of end year EU Allowance (**EUA**) futures contracts of 2018 and 2019 vintages, averaged over a period of three months.

Using the same approach in New Zealand, the current NZU price would be used. However, although this is an available price, it might not be the right 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 that 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 a fixed price option is currently available, allowing obligated entities to pay $25/tonne rather than submit NZUs.[34]

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. MfE has developed some marginal abatement cost (**MAC**) curves for emission reductions and is funding the development of more sophisticated economy-wide models. The results of this process should yield values for estimating the marginal costs of New Zealand complying with its obligations.

Global damage costs

The full damage costs of GHG emissions (ie the social costs of carbon - **SCC**) include the effects on people in other countries as well as New Zealand. These costs have been estimated and used in CBAs in several countries, including the USA (Scovronick et al, 2019).[35] Global damage values, based on Austroads (2012) as discussed above, are included in the EEM. Global damage costs of emissions from New Zealand have been estimated to produce a shadow price of CO_2-e emissions and included in New Zealand's Fourth Biennial Report under the United Nations Framework Convention on Climate Change (**UNFCCC**).

However, there are two problems with this approach.

The first is that, because New Zealand has set an absolute cap on its emissions, an additional emission from any one source (such as a motor vehicle) does not add to total emissions, it simply shifts the location (or possibly the timing) of emissions. This process is facilitated in New Zealand via the ETS. Likewise, a reduction in emissions from one source does not change the total emissions from the country, only who emits. It is the cap which determines total emissions, not levels of activity in any sector.

[34] Current prices (December 2019) are just below $25/t (see https://www.commtrade.co.nz/)

[35] We note that estimates of the SCC used in US Federal CBAs now includes only damage costs in the USA rather than those in other countries also (Rennert and Kingdon, 2019)

The second is that, even if there was no binding cap on emissions, effects should be estimated using a consistent perspective for analysis. Using global damage costs for GHG emissions is taking a different perspective to a CBA from that which is normally taken. It is taking account of the effects on people outside of New Zealand. There is nothing wrong with taking a global perspective for analysis, but it should be used consistently. Otherwise, by only using a global perspective for GHGs, we are distorting consumption from GHG-producing activities to potentially more damaging alternatives.

For a global perspective to be used consistently in analysis would require the external costs of production of imports to be added to current prices to produce shadow prices for all goods and services. For example, in analysing the benefits of electric vehicles, the full costs of vehicle manufacture, including production externalities in Japan or elsewhere, should be estimated rather than simply the import price. Likewise, the benefits (or costs) of New Zealand exports beyond their market price should be included in analysis.

And note, if the SCC is used as a cost of CO_2, it is additional to the New Zealand compliance costs, discussed above. The compliance costs (the marginal costs of emission reduction) are an estimate of the costs to New Zealand. The SCC is an estimate of the costs falling largely on other countries.

The SCC might be a useful metric in the absence of abatement cost estimates for New Zealand. This would be on the assumption that New Zealand's emission limits were set on the basis of it optimising its efforts, ie the marginal costs of emission reductions are equal to the marginal damage costs of those emissions. However, no such analysis has been undertaken. Other approaches to estimating carbon costs are discussed by Denne and Bond-Smith (2010).

6.3 Approved methodology

6.3.1 Mortality impacts

We will use two approaches for valuing mortality impacts as follows:

- use the change in premature mortality[36] multiplied by the current New Zealand-based VoSL as was done in HAPINZ 2.0. The current VoSL is based on road crash deaths
- apply changes to mortality to age-specific death rates in life tables to estimate changes in total life years and then multiply these by VoLY.

We will 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.

Population data will be taken from the census. Life expectancy at each age group will be taken from the Stats NZ's sub-national Life Tables.

[36] If cancer cases are included in HAPINZ 3.0, it is likely that the cost per case will be assumed to be equivalent to that for a premature death.

6.3.2 Morbidity impacts

We will value the following morbidity impacts:

- cardiovascular hospital admissions
- respiratory hospital admissions
- restricted activity days.

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.

Cost of hospitalisations

We will take the daily costs of hospitalisations from Telfar-Bernard and Zhang (2019) for RHAs and from National Health Committee (2013) for CHAs. We will then use hospitalisation data to calculate the actual number of hospital days (rather than using published 'average length of stay' figures).

Costs of lost earnings

We will recalculate the cost of lost earnings. Average weekly income will be multiplied by the working population to estimate total weekly earnings. This will be divided first by the total population and then by seven to produce average daily lost earnings. This translates currently to NZ$62 in 2019 values (table 14).

Table 14: Lost earnings ($/day) (2019 values)

	Stats NZ Infoshare Dataset	Amount (Q2 2019)
Average weekly income (seasonally adjusted)	QEX045AA	$1,088
Total in employment	QEX039AA	1,971,789
Total income ($m/week)		$2,145
Total population	DPE059AA	4912000
Average income per day		**$62**

Costs of rehabilitation and reduced quality of life

Costs for rehabilitation and the associated reduction in quality of life differ with specific diseases. For example, National Health Committee (2013) estimates rehabilitation of 23-26 days for 30% of stroke patients at $750 per day.

In the absence of other data, we will use the same percentage of hospitalisation costs (50%) in this analysis: currently $2,348 and $5,209 respectively for RHAs and CHAs.

Restricted activity days

The social costs of restricted activity days will be the same one calculated for the cost of lost earnings needed to assess hospitalisations (see table 14).

6.3.3 Damage costs

We will look to develop emission marginal cost estimates expressed as a $/tonne, suitable for inputs to policy and cost benefit analysis.

For harmful emissions, damage costs will be developed by using the updated HAPINZ 3.0 model to output health impacts for areas with robust emissions inventories - thereby enabling us to develop a suite of damage costs for areas with different population densities.

Being able to assess co-benefits is very important. This study offers the opportunity to consider costs more holistically by developing a suite of damage costs that can be applied consistently across New Zealand to assess all benefits (in terms reducing of exposure to harmful emissions **and** GHGs) resulting from emissions reduction strategies and other policy interventions.

For CO_2 we will examine the development of MAC curves for New Zealand as the primary source for estimates of costs. In the absence of such cost estimates, we will examine whether estimates of the SCC provide useful guidance for costs.

6.3.4 Summary of our approach

We will use two approaches to valuing **mortality impacts** as follows:

- use the change in premature mortality multiplied by the current New Zealand-based VoSL as was done in HAPINZ 2.0. The current VoSL is based on road crash deaths.
- apply changes to mortality to age-specific death rates in life tables to estimate changes in total life years and then multiply these by VoLY

We will 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.

Population data will be taken from the census. Life expectancy at each age group will be taken from the Stats NZ's sub-national Life Tables.

We will value the following **morbidity impacts**:

- cardiovascular hospital admissions
- respiratory hospital admissions
- restricted activity days.

We will look to develop emission marginal cost estimates expressed as a $/tonne, suitable for inputs to policy and cost benefit analysis. We intend to develop **a suite of damage costs** that can be applied consistently across New Zealand to assess all benefits (in terms reducing of exposure to harmful emissions and GHGs) resulting from emissions reduction strategies and other policy interventions.

7. 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.

7.1 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
- The *Health Effects Model*, based on an Excel spreadsheet, which allowed end-users to output results nationally, regionally, by Territorial Authority (**TA**), by Statistics NZ urban 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
- 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 as a value of statistical life (**VoSL**) is too high?
- 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.

7.2 Developments that have happened since

Despite widespread consensus that air pollution is harmful, most people 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 aren't good metrics to use to communicate/engage with the wider community about air quality and health. The steering

committee wants the results of this update to be more accessible to a broad audience and to ensure that the results of the update feed into policy development.

This section reviews the literature on improved strategies to more effectively communicate environmental messages that has arisen since HAPINZ 2.0.

7.2.1 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.

In order 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.

7.2.2 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. With regard to 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 (with the exception of 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 (ie, 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 (ie, they are not necessarily the first thought someone might have on the issue)
- often require slower thinking (ie, 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.

7.2.3 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 the default way we process information, only some stories will 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.

7.2.4 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? And 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 a number of 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, 2017)
- presenting facts in a way that builds new mental models (Kendall-Taylor et al, 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.

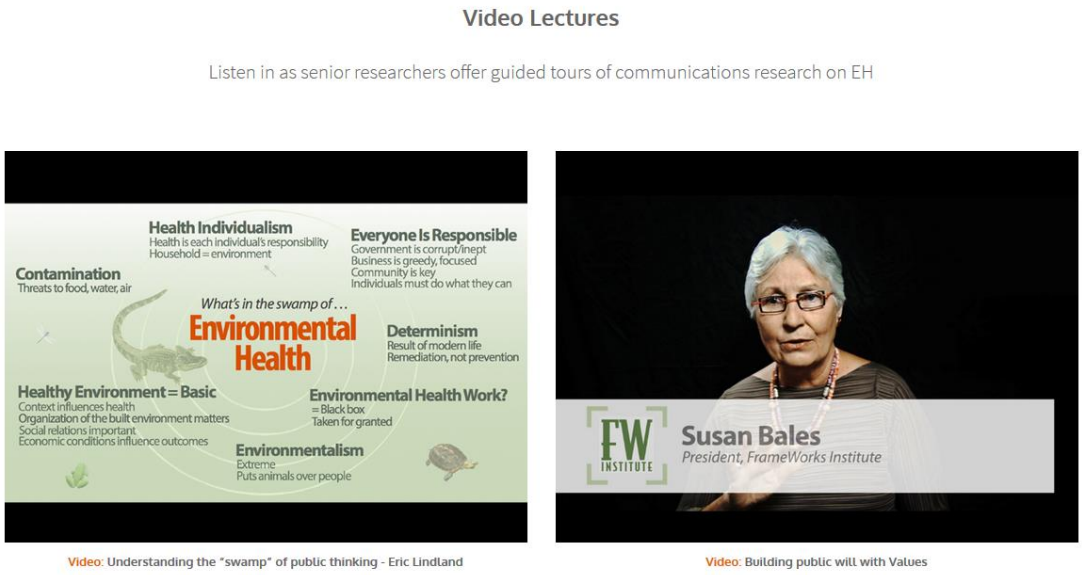
7.2.5 What strategic communications exist in environmental health and air quality?

A number of tested strategic communications in the area of environmental health already exist. For example, FrameWorks Institute research 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[37] helps environmental health professionals frame environmental health and related issues as important policy fields and matters of public concern (refer figure 9). We don't expect there to be a large body of research that has tested strategic communications specifically on air pollution.

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).

Until we complete a comprehensive review of strategies that work, we cannot highlight other existing best practice examples in the area of air quality and pollution.

Figure 9: Good practice example health message regarding air pollution (FrameWorks Institute)



Video: Understanding the "swamp" of public thinking - Eric Lindland

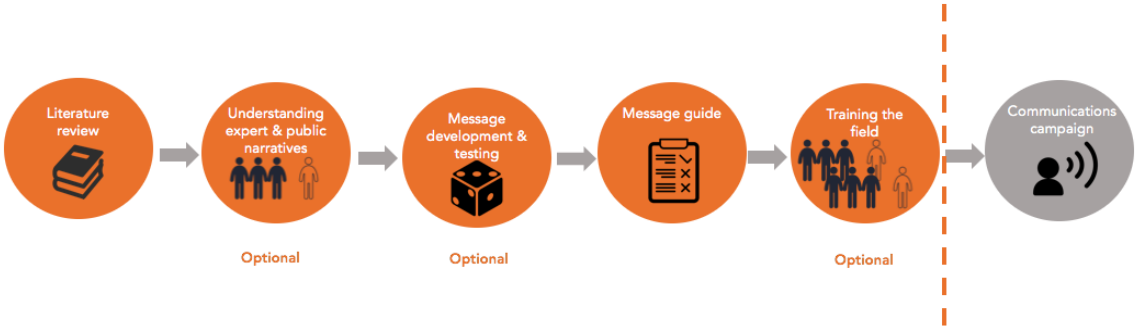
Video: Building public will with Values

[37] http://frameworksinstitute.org/toolkits/environmentalhealth/

7.2.6 Communication strategy and messaging

In the next section, we outline the different methodological approaches available for this project, depending on the steering group's priorities. Regardless of the methods chosen the core components will comprise a review of the existing strategic framing literature, and the development of guidance on how to effectively and strategically frame the study's key findings. This framing work does not include the development of either a communications campaign or the products that comprise such a campaign, as described in figure 10. Rather it provides evidence based do and don'ts for any individual or organisation wanting to communicate the study's findings through various channels.

Figure 10: Methodology for developing strategic communications on air quality



7.3 Approved methodology

Our approach comprises two key steps:

- a review of existing strategic frames and communications strategies
- development of a draft messaging guide.

7.3.1 Review of existing strategic frames and communication strategies

The core approach to developing strategic frames and communications strategies involves a non-systematic review of existing material. This review doesn't look at all tested communications techniques in the area of air pollution and environmental health; rather we are guided by a theoretically and empirically driven framework of strategies. Specifically we search for research that:

- engages with the psychology of how people process information (and misinformation)
- works with people's values
- seeks to locate and use culturally shared frames
- tests effective metaphors
- tests the presentation of facts in a way that builds new mental/cognitive models.

7.3.2 Development of a draft messaging guide

From the review we will develop a draft messaging guide. Taking existing communication strategies employed within New Zealand and internationally, we will outline the development of an easy to read and visually engaging guide on talking about air pollution science and policy.

This draft guide will be 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 and
- considering your messages and media.

For an example of a finalised guide see: The Workshop & Oxfam New Zealand (2019) How to Talk About Climate Change. A Toolkit for Collection Action. The Workshop. Wellington.

7.3.3 Summary of our approach

We will:

- review existing strategic frames and communication strategies and
- draft **an applied messaging guide.**

The draft guide will outline evidence based dos and don'ts for any individual or organisation wanting to communicate the study findings through various channels.

8. Other considerations

This chapter reviews other methodological considerations including:

- Calculation method
- Population and health incidence data
- Base year
- Spatial unit
- Exposure model functionality

This chapter summarises the methodologies used in the HAPINZ 2.0 study, reviews developments that have occurred since and outlines our methodology for the HAPINZ 3.0 update.

8.1 Calculation method

8.1.1 Approach used in HAPINZ 2.0

The calculation methods used in HAPINZ 2.0 were broadly consistent with the methods we are using for this update so will not be repeated here. Any differences (eg, in terminology) are highlighted in the following sections.

8.1.2 Developments since

We have updated the terminology in this report to align with guidance and methods produced by WHO. However, the calculation methods are consistent with the previous HAPINZ.

8.1.3 Recommendations

Our approach to estimating the health burden attributable to air pollution follows the method used in the Global Burden of Disease study (WHO, 2018) and summarised in figure 11. It is also consistent with the approach used in previous HAPINZ studies.

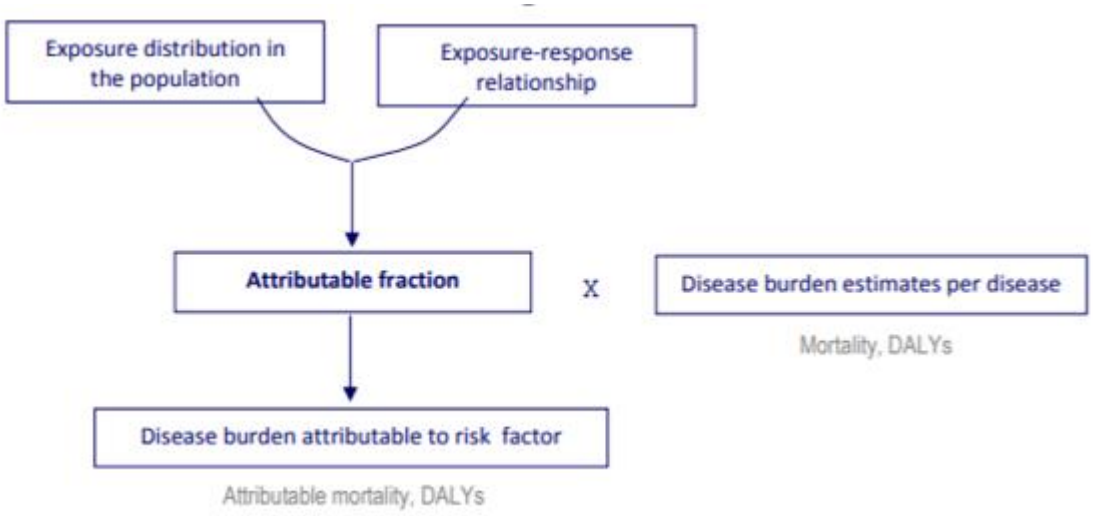
This method uses the following terminology:

Population attributable fraction (**PAF**) (also known as the attributable fraction): proportion of the health burden attributable to a specified risk factor

Attributable burden: disease burden attributable to a specified risk factor.

Note: In the previous HAPINZ report, the attributable burden was referred to as $Cases_{AP}$ (number of extra cases that arose due to exposure to air pollution).

Figure 11: Method for burden of disease estimation. DALYs: disease adjusted life years



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

For the estimated health burden of PM, we will use the following formula for the PAF, which will be calculated for each area unit:

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

In this formula,

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

E (exposure) is the concentration of pollutant in the area of interest (eg, annual average PM_{10} concentration in a particular census area unit)

PAF is the population attributable fraction, which can be interpreted as the estimated percentage of total health cases that are attributable to the exposure (ie, 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:

$$\mathbf{Health\ effects}_{(\mathbf{Cases})} = \mathbf{PAF} \times \mathbf{Cases}_{(\mathbf{Total})}$$

Where:

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

PAF = population attributable fraction calculated above

Cases$_{(Total)}$ is the total number of health cases in a specific area unit

The health cases can include any type of health burden, such as:

- deaths
- hospitalisations

- years of life lost (based on deaths and life tables)
- length of stay in hospital.

The previous formula gives the same results as the formula used in HAPINZ 2.0 (where Health effects$_{(Cases)}$ is the same as Cases$_{AP}$):

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

Approach for this study – based on HAPINZ 2.0

For HAPINZ 3.0, we will calculate the attributable burden at the small area level (eg, census area units) then sum across the whole country. In this approach, we assume that in a census area unit (**CAU**) everybody is exposed to the same level of air pollution. We then calculate the PAF and attributable burden for each CAU, and then aggregate up across the total country.

Calculating the attributable burden for NO_2 (and possibly roadside PM_{10} and $PM_{2.5}$)

Exposure to traffic pollutants, in particular NO_2, is higher near busy roads. Therefore, some people in a CAU will have exposure to higher levels of NO_2 than other people in a CAU, depending on where they live.

To account for this, a population weighted average exposure could be calculated in GIS for each CAU. This would be based on population exposure estimated at a 50 metre resolution in the NZ Transport Agency NVED exposure tool. This population weighted exposure could then be used to calculate the PAF and attributable burden using the same calculation procedure described above.

The NZ Transport Agency NVED exposure tool also estimates roadside exposure of PM_{10} and $PM_{2.5}$. Further work is required to determine whether these estimates will be included in HAPINZ 3.0.

8.2 Base analysis year and spatial unit

To ensure consistency, we need to align the year(s) and spatial unit(s) for population data, exposure data and health data.

8.2.1 Approach used in HAPINZ 2.0

For HAPINZ 2.0, the analysis was based on 2006 census data and 2005-2007 health incidence data. The analysis was based on 2006 CAUs. These small areas were aggregated to give population impacts of air quality at the required geographic areas (including TA, airshed, and regional council).

8.2.2 Developments since

In terms of population data, two censuses have been undertaken since HAPINZ 2.0 as follows:

- 2013 Census, which employed CAUs that were slightly different to those employed in 2006 and

- 2018 Census, which employed new statistical area units (**SA1**s and **SA2**s) as a base unit. These differ to the CAUs employed in both Census 2013 and 2006.

In addition, there have also been some amendments to airsheds (eg, Bay of Plenty regional council has gazetted a new airshed for the Mount Maunganui Port/Industrial area).

National health incidence data utilises domicile codes which relate one-to-one to CAUs. However, these domicile codes lag behind the census. For example, 2006 domicile codes were assigned to health records up to mid-2015. After this time, the domicile codes relate one-to-one to 2013 CAUs.

Table 15 summarises the cross-over dates applicable to each base unit.

Table 15: National database units

Database	Base unit	Domicile code
2006 Census	2006 CAU	Assigned to 2001 CAU up to 30 Jun 2008
2013 Census	2013 CAU	Assigned to 2006 CAU up to 30 Jun 2015
2018 Census	2018 SAU	Assigned to 2013 CAU up to now

There is not yet any way to translate health domicile codes (of the national health datasets) to the new SA1s or SA2s.

8.2.3 Recommendations

We will be utilising the following key datasets:

- Population data from the New Zealand Census, which is published by Statistics NZ.
- Mortality data from the New Zealand Mortality Collection, which is available from the Ministry of Health. The Mortality Collection has date of birth, date of death, underlying cause of death (ICD-10AM code), ethnic groups, domicile code (census area unit).
- Hospitalisations from the confidentialised unit record data from the National Minimum Dataset (**NMDS**), which is available from the Ministry of Health. The NMDS Collection has date of birth, date of hospital admission and discharge, primary diagnosis (ICD-10AM code), ethnic groups, domicile code (census area unit), and other useful information.

Data will be analysed at a small spatial unit (census area unit) to calculate health effects. For some health outcomes data will be analysed for specific population sub-groups (eg, age and ethnicity).

To ensure consistency, we need to align the year(s) of data for population data, exposure data and health data. As for HAPINZ 2.0, we recommend averaging health incidence data across three years. This means that, **provided provisional mortality data for 2017 is of sufficient quality and fit for purpose, a base analysis year of 2016 is feasible**.

Population data is already available from 2013 and 2018 Census data. Population data for the base year (2015 or 2016) will be based either on linear interpolation between Census points, or other data sources (such as the population spine in the Integrated Data Infrastructure).

At this stage, there is no way to translate health domicile codes (used for national health incidence data) to the new 2018 SA1s or SA2s. **This means we recommend using 2013 CAUs as our base spatial unit.**

8.3 Model design and functionality

8.3.1 Approach used in HAPINZ 2.0

HAPINZ 2.0 comprised two models, an exposure model and a health effects model. These are described below.

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.

The exposure model also provides national emissions estimates disaggregated by CAU and by source (ie, 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.

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 local 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 offer scenario testing whereby the following parameters can be changed (nationally) to estimate consequent changes in health effects and social:

- annual PM_{10} and $PM_{2.5}$ concentrations
- population
- all individual exposure-response functions
- all social costs.

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).

8.3.2 Developments since

The HAPINZ 2.0 spreadsheet model has been successfully used for a range of projects. The steering group has asked for improvements in the model design, in particular:

- Make the model easier to update
- Integrate the health effects and exposure models
- Make it easier to achieve spatial representation of results
- Make the model more robust (less easy to corrupt)
- Provide additional scenario testing options

We have considered whether a spreadsheet is the best option for HAPINZ 3.0. Other options could include a GIS toolkit,a database or an automated software tool such as the WHO's AirQ+.[38]

Advantages of using a spreadsheet-based model include:

- Widely accessible as most users have Microsoft Excel.
- Transparent for end users. Most users are reasonably familiar with Excel, which means that they can interrogate and understand calculations, and key parameters (eg, lookup tables)
- Spreadsheets are easier to update and change compared to a GIS tool, which requires specialist GIS input, software, and updates to any 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 (eg, national, regional, airshed).

Disadvantages of using a spreadsheet-based model include:

- There is a manual process to export results to produce maps when updates or scenarios are developed.

[38] AirQ+ is a software tool for health risk assessment of air pollution managed by the WHO's Regional Office for Europe. Available at: http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/activities/airq-software-tool-for-health-risk-assessment-of-air-pollution

- Spreadsheets can be easily corruptible and subject to errors 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.

8.3.3 Recommendations

For HAPINZ 3.0 we will develop a spreadsheet model. We also propose to develop a prototype GIS tool, which will be available online. This tool will make key results available spatially and allow users to undertake sensitivity analysis for key variables.

The modelling approach will be similar to HAPINZ 2.0. However, based on our experience using the model, as well as feedback from the steering committee, we will improve the functionality and ease of use of HAPINZ 3.0. Improvements will include:

- Merging the exposure model and health effects model
- Making the spreadsheet easier to update by clearly identifying key inputs (including ambient monitoring concentrations and source apportionment for each location). These data will be protected to avoid accidental changes. However, an option to input "user defined" data could be provided. Alternatively, instructions to unlock and overwrite data will be provided.
- Simplifying the model. Some of the complexity in the HAPINZ 2.0 model was not justified given the limitations in the accuracy of the source data.
- Considering expanding the scenario testing options to include reduction of pollutant concentrations from key sources. For example, this would allow users to test the effectiveness of a policy that is expected to achieve a reduction in the concentration of pollution attributed to domestic fires (assuming that this causes a proportional reduction in effects).
- Developing a prototype GIS tool which will be available online. This tool will make key results available spatially and allow users to undertake sensitivity analysis for key variables.

We will minimise 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.

9. Addressing uncertainty

This chapter reviews key sources of uncertainty in an assessment of health impacts from air pollution and describes how we will assess uncertainty in HAPINZ 3.0.

9.1 Key sources of uncertainty

The key sources of uncertainty in an assessment of air pollution health impacts are described as follows (WHO, 2016).

9.1.1 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.

9.1.2 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.

9.1.3 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.

9.1.4 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.

9.1.5 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. This is not a source of uncertainty in itself, however the results of the assessment are sensitive to the counterfactual.

9.1.6 Deliberate simplifications of the model

Practical considerations may require the use of a simplified model, which can lead to increased uncertainty.

9.2 Recommended methodology

In HAPINZ 3.0 each of the key sources of uncertainty will be acknowledged and described as fully as possible.

Uncertainty will be quantified where possible. This will help to give a sense of the precision of the estimates and help to prioritise future research and improvements in the methodology.

10. Summary of approved approach

The key features of the approach we will be following 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** reported by 16 regional councils, 71 airsheds, 74 territorial local authorities and 139 urban areas
Population covered	100% of 2016 population
Pollutants	**Priority pollutants** • particulate matter (PM_{10} and $PM_{2.5}$) • nitrogen dioxide (NO_2)
Exposure assessment	**PM_{10} and $PM_{2.5}$**: ambient monitoring data averaged for 2015-2018 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_{10} and $PM_{2.5}$** using source apportionment data: marine aerosol, biomass burning, motor vehicles, secondary PM, crustal material **PM_{10} and $PM_{2.5}$** using emissions inventory data: industry, open burning, rail, aviation and shipping (where data allow) **NO_2**: motor vehicle exhaust emissions only
Health endpoints	**Primary health outcomes** • **mortality** and years of life lost (YLL) from long-term **$PM_{2.5}$** for all adults 30+ years, all ethnicities and for Māori/Pasifika • **mortality** and YLL from long-term **NO_2** for all adults 30+ years, all ethnicities • **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 • **respiratory admissions** from long-term **NO_2** for all ages, all ethnicities **Secondary health outcomes (for comparison with HAPINZ 2.0)** • **mortality** from long-term **PM_{10}** for all adults 30+ years, all ethnicities and for Māori/Pasifika • **mortality** from long-term **PM_{10}** for all infants, aged 1 month to 1 year • **restricted activity days** from long-term **$PM_{2.5}$** for all ages, all ethnicities **Childhood asthma outcomes relevant to NZ** • **incidence** due to long-term NO_2 • **exacerbations** due to short-term $PM_{2.5}$

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 hospital admissions • respiratory hospital admission • restricted activity days **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/s** enabling sensitivity/scenario testing and designed to be easily updateable A **set of improved exposure-response functions** for use in assessing air pollution health effects on Maori and Pasifika from a separate cohort study A **final report** suitable for a broad audience, outlining the methodology used and key findings (with all assumptions clearly stated) A draft **messaging guide** to provide evidence based dos and don'ts for anyone wanting to communicate the study findings through various channels

Note: The proposed methodology investigated the inclusion of additional pollutants (black carbon, benzo[a]pyrene, arsenic and lead) in HAPINZ 3.0. Following comments from the international peer reviewers and discussion with the Steering Group, these pollutants were dropped from the current assessment due to concerns about data availability, double-counting and robustness of exposure-response functions.

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 AQNES
Al	aluminium
anthropogenic	generated by human activities, such as the combustion of fuels or processing of raw materials
AQNES	National Environmental Standards for Air Quality
As	arsenic
BaP	benzo(a)pyrene
BC	black carbon, both a harmful pollutant and a greenhouse gas
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
CBA	cost-benefit analysis
CCA	copper chrome arsenate (a treatment used to preserve timber)
CHA	cardiovascular hospital admission
chronic	long-term duration or constantly recurring
CO	carbon monoxide, a harmful pollutant
CO_2	carbon dioxide, a greenhouse gas
coarse particulate	particles in the $PM_{2.5}$ to PM_{10} fraction
COMEAP	Committee on the Medical Effects of Air Pollutants
COPD	chronic obstructive pulmonary disease, which includes a range of conditions such as bronchitis, chronic bronchitis, emphysema, bronchiectasis, extrinsic allegoric alveolitis, and chronic airways obstruction
Cr	chromium
Cu	copper
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
DMS	dimethyl sulphide
domestic fire	a solid-fuel heating appliance which is intended primarily to heat a residential dwelling
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
ETS	New Zealand Emissions Trading Scheme

EUA	European Union Allowance (the tradable unit under the European Union Emissions Trading Scheme)
Fe	iron
fine particulate	particles in the $PM_{2.5}$ fraction
greenhouse gas	an air pollutant which contributes to atmospheric warming
HAPINZ	Health and Air Pollution in New Zealand
harmful pollutant	an air pollutant which causes adverse health effects
H_2S	hydrogen sulphide
IBA	ion beam analysis
IDI	The Statistics New Zealand Integrated Data Infrastructure which has detailed data on national mortality rates and hospital admissions for up to the past 20 years.
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
kg	kilogram
LEZ	low emission zone
m	metre
MAC	marginal abatement cost
MfE	Ministry for the Environment
MoH	Ministry of Health
MoT	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
natural	generated by natural activities, such as wind-blown dust, sea spray, vegetation, animals or volcanoes
ng/m^3	nanogram per cubic metre, a unit of concentration
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
NZTA	New Zealand Transport Agency
NZU	New Zealand Unit (a tradable unit under the ETS)
OC	organic carbon
OEHHA	California Office of Environmental Health Hazard Assessment
open burning	burning of biomass and waste in the outdoors
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_{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
PMF	positive matrix factorisation
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.
RADs	restricted activity days are days on which people cannot do the things they might otherwise have done if air pollution was not present.
respiratory	of, pertaining to, or affecting the lungs and airways
RHA	respiratory hospital admission
S	sulphur
SCC	social cost of carbon
Se	selenium
Si	silicon
SO_2	sulphur dioxide, a harmful pollutant
SOA	secondary organic aerosol
solid fuel	coal and wood (including wood pellets)
taonga	in Māori culture, a taonga is a treasured thing, whether tangible or intangible
TLA	Territorial Local Authority, such as city or district council
UFP	ultrafine particles
µg	microgram, one millionth of a gram
$\mu g/m^3$	microgram per cubic metre, a unit of concentration
UNFCCC	United Nations Framework Convention on Climate Change
µm	micrometre, one millionth of a metre
UK	United Kingdom
USA	United States of America
US EPA	United States Environmental Protection Agency
V	vanadium
VKT	vehicle kilometres travelled
VoLY	value of a life-year
VoSL	value of statistical life
VOCs	volatile organic compounds
WHO	World Health Organization
wood burner	a domestic heating appliance that burns wood but which is not an open fire or a multifuel heater, a pellet heater or a coal burning heater or a cooking stove

WTP	willingness to pay
XRF	X-ray fluorescence
YLL	years of life lost

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Appendix A: Features of the approved methodology

The outcomes we will be assessing in HAPINZ 3.0 are summarised in the following two matrices

Health Outcomes	Valid RR or RF?	Valid incidence data?	Valid AQ data?	Valid Source Info?				Valid Social Costs?
				Domestic Fires	Motor Vehicles	Industry, Open Burning, Rail, Aviation, Shipping	Natural sea spray/soil	
$PM_{2.5}$ - annual exposure								
Premature mortality and YLL (all adults 30+, all ethnicities)	Yes	Yes	Yes	Yes	Yes	Approx only	Yes	Yes
Hospital admissions: CVD (all ages, all ethnicities)	Yes	Yes						Yes
Hospital admissions: Respiratory (all ages, all ethnicities)	Yes	Yes						Yes
Restricted activity days (all ages, all ethnicities)	HAPINZ 2.0	n/a						Yes
Exacerbation of childhood asthma (all ethnicities)	Yes	Proxy						Based on proxy
NO_2 - annual exposure								
Premature mortality and YLL (all adults 30+, all ethnicities)	Yes	Yes	Pending NZTA exposure model	Data for motor vehicles **only** pending NZTA exposure model			n/a	Yes
Hospital admissions: Respiratory (all ages, all ethnicities)	Yes	Yes						Yes
Incidence of childhood asthma (all ethnicities)	Yes	Proxy						Based on proxy

Health Outcomes	Valid RR or RF?	Valid incidence data?	Valid AQ data?	Valid Source Info?				Valid Social Costs?
				Domestic Fires	Motor Vehicles	Industry, Open Burning, Rail, Aviation, Shipping	Natural sea spray/soil	
PM_{10} - annual exposure								
Premature mortality (all adults 30+, all ethnicities)	HAPINZ 2.0	Yes	Yes	Yes	Yes	Approx only	Yes	Yes
Premature mortality (all adults, Māori only)	HAPINZ 2.0	Yes						Yes
Premature mortality (infants, all ethnicities)	HAPINZ 2.0	Yes						Yes

Appendix B: Source apportionment

This appendix summarises the PM speciation studies that have been undertaken in New Zealand to date.

B.1 PM speciation sampling sites in New Zealand

The concentration of specific contaminants in PM (including BC, As and Pb) is available from the results of particulate speciation studies.

Particulate matter samples have been collected and analysed at approximately 40 sites across New Zealand, with some urban areas including multiple sites. For example, Auckland PM data includes sites at Takapuna, Henderson, Kingsland, Newmarket, Auckland CBD (Queen Street), Penrose and Patumahoe (40 km southwest of the CBD). All sites where PM has been collected have included analyses for BC and multi-elemental speciation (Na to U) with the accompanying receptor modelling (source apportionment) and reporting. Figure B1 and table B1 presents the PM speciation sampling locations in New Zealand to date.

Figure B1: Particulate matter speciation sampling locations in New Zealand

In addition to the urban speciation monitoring locations, several studies have targeted source specific PM. These include motor vehicle tunnels (Ancelet et al, 2011b; Davy et al, 2011a) and wood burner emissions (Davy et al, 2009b; Ancelet et al, 2010; Ancelet et al, 2011a) in order to better understand emission source characteristics and composition. The majority of PM sampling and analysis campaigns have been targeted studies collecting 24-hour time integrated samples that ran for 1-2 years in order to better understand the local drivers of air pollution for air quality management purposes. The exception to this is the Auckland multi-site air PM speciation database that has been running since mid-2004 and, using archived filters, the BC measurements have been extended back to 1998 at some sites providing a 20-year BC dataset. For several locations, high-resolution sampling (hourly) and analysis was undertaken as part of a research programme[39] in order to better understand the source contributing to the

[39] MBIE Contract C05X0903: Understanding air particulate matter pollution. *Sources, patterns and transport of air particulate matter in polluted New Zealand urban environments*

observed diurnal variation in PM concentrations in New Zealand urban centres (Trompetter et al, 2010; Ancelet et al, 2012; Ancelet, Davy et al, 2014b; 2014a).

Table B1: 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

B.2 Analysis of PM composition

Two multi-elemental analysis techniques have been used routinely to provide the PM composition analysis, these are accelerator-based ion beam analysis (**IBA**) and X-ray fluorescence analysis (**XRF**), while light reflectance has been used to determine BC concentrations in all samples. These are well established and internationally accepted methods for determining PM elemental composition (Horvath, 1993; Landsberger and Creatchman, 1999; Maenhaut and Malmqvist, 2001; Bond and Bergstrom, 2006). Full descriptions of these techniques are provided in Davy and Trompetter (2018). IBA and XRF are both non-destructive analytical techniques and provide complimentary elemental results where XRF is more sensitive (lower limits of analytical detection) for heavier elements, particularly heavy metals, and IBA is more sensitive for lighter elements (Na to K) with the ability to determine hydrogen concentrations, a useful marker for hydrocarbon and secondary aerosol species.

GNS Science has used the accelerator-based IBA techniques to measure elemental concentrations in New Zealand PM samples since 1996. In 2013, the analysis capability was extended by acquiring the XRF analytical facility (Epsilon 5, Panalytical Pty, Netherlands).

At several locations (such as Auckland, Tokoroa, Wellington, Christchurch and Timaru) speciation monitoring programs have included a wider range of analytes such as ions (eg NH_4^+, NO_3^- and SO_4^{2-}), organic compounds (eg PAHs, levoglucosan and retene) and the organic carbon/elemental carbon (**OC/EC**) split by thermal optical reflectance techniques (Scott, 2006; Ancelet et al, 2011; Salako et al, 2012; Ancelet et al, 2013; Scott, 2014; Davy et al, 2016; Davy and Trompetter, 2017). The extra analytes provided insights for secondary aerosol species and carbonaceous aerosol composition and sources.

B.3 Receptor modelling of PM composition

The multivariate analysis of air PM sample composition (also known as receptor modelling or source apportionment) provides groupings (or factors) of elements that vary together over time. This technique effectively 'fingerprints' the sources that are contributing to airborne PM concentrations and the mass of each element (including BC, As, Pb) attributed to that source. Most commonly used receptor models are based on conservation of mass from the point of emission to the point of sampling and measurement (Hopke, 1999). Their mathematical formulations express ambient chemical concentrations as the sum of products of species abundances in source emissions and source contributions. In other words, the chemical composition of filter-based samples of PM collected at a monitoring station is resolved mathematically to be the sum of a number of different factors or sources of those particles.

GNS Science has used the receptor modelling approach to identify sources of PM in New Zealand airsheds by applying a technique known as Positive Matrix Factorisation (**PMF**) analysis to PM composition data (Paatero and Tapper, 1994; Hopke et al, 1999). A direct result of using this technique is that the sources of BC and heavy metals (or any other variable) were also derived and the mass contribution of each emission source to atmospheric BC and heavy metal concentrations was determined.

While the source apportionment data are spread across time and space, each study has offered up the relative contribution of sources to total PM concentrations for which, as annual averages, the relative proportions are unlikely to change radically over the years. The Auckland source apportionment dataset shows that any such changes are observable in total PM concentrations and can be accounted for through appropriate data analysis techniques.

B.4 Sources of PM_{10} and $PM_{2.5}$

PM_{10} by definition also includes $PM_{2.5}$ and the contribution of $PM_{2.5}$ sources. There are five key source types contributing to PM_{10} that have been identified across all urban source apportionment studies in New Zealand to date. These are defined by the emission source, composition and particle size.

The five source types are motor vehicles, biomass combustion (dominated by domestic solid fuel fires), secondary aerosol (primarily secondary sulphate), marine aerosol (sea salt) and crustal matter (soil). The combustion sources (motor vehicle tailpipe emissions, domestic fires) and secondary aerosol are the main $PM_{2.5}$ sources. Marine aerosol, crustal matter and the resuspended road dust generated by the turbulent passage of motor vehicles across a road surface (includes tyre wear, brake wear, road surface wear and whatever else has been deposited on the road surface) are mainly coarse particle ($PM_{10-2.5}$) sources but with some more minor component that extends into the $PM_{2.5}$ size range. The relative contributions of each of these source types to PM_{10} and $PM_{2.5}$ have been well-defined by source apportionment studies both in New Zealand and overseas.

B.4.1 Derivation of natural sources of particulate matter

New Zealand's isolated Southern Hemisphere location results in a significant contribution from oceanic generated aerosol (mainly sea salt) to terrestrial PM concentrations. There is also a component derived from local volcanic emissions, mainly to secondary sulphate concentrations. Occasionally dust from Australian desert storms may be lifted sufficiently high in the atmosphere to cross the Tasman Sea. Collectively these sources are considered the natural PM component.

One of the key results from receptor modelling analyses is the derivation of mass contributions to ambient aerosol concentrations from natural sources and sources for which little useful information is available from other methods of source apportionment such as emissions inventories. The information is vital for air quality management as the proportion of particle mass from natural and other (uncontrollable) sources needs to be factored into any air pollution reduction strategy. A straightforward definition of natural sources of PM is that the source can only be considered 'natural' if it involves no direct or indirect human activity[40]. For example, PM pollution from a wildfire can only be considered natural if it was ignited by lightening or similar. If the fire was due to accidental or deliberate human activity as the ignition source, then it is considered an anthropogenic source.

The New Zealand datasets show that oceanic or *marine aerosol* (sea salt) is the primary source of natural aerosol present in New Zealand urban atmospheres. *Secondary sulphate* aerosol formed from gas-to-particle atmospheric reactions has both natural (oceanic phytoplankton, volcanic emissions) and anthropogenic (combustion of sulphur containing fuels, industrial emissions) gaseous precursor sources. The third component of urban PM that may be of natural origin is crustal matter, generally referred to as *soil* in source apportionment studies. However, time-variation analyses across multiple datasets show that urban soil PM concentrations are lower on weekends than weekdays indicating that the generation of airborne crustal matter in urban locations is largely the result of human activities (construction/demolition, earthworks, roadworks, passage of vehicles on roads and unpaved

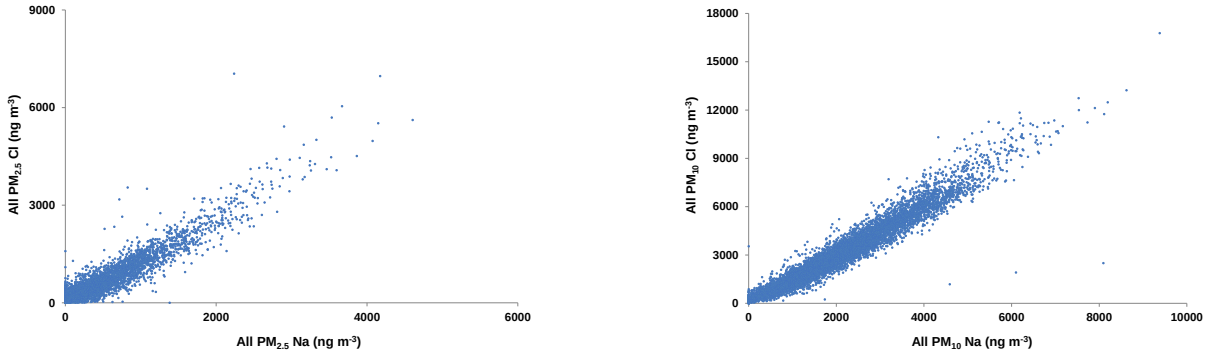
[40] *Particulate matter from natural sources and related reporting under the EU Air Quality Directive in 2008 and 2009*. Technical report No. 10/2012. European Union 2012, https://www.eea.europa.eu/publications/particulate-matter-from-natural-sources

areas) and therefore does not meet the 'natural source' criteria discussed above. The following sections provide further detail on each of these sources.

Marine aerosol

Sodium and chlorine are the primary constituents of marine aerosol or sea salt, and were also significant elemental contributors to both $PM_{2.5}$ and PM_{10} mass at New Zealand monitoring sites along with the more minor components (K, Ca, Mg, S) of sea salt. The elements were highly correlated (as shown for the Auckland dataset in figure B2) and present in the same ratio at peak concentrations as found in sea salt ([Na] = 0.56[Cl]) (Lide, 1992). The analytical results demonstrate the relative influence of this natural aerosol source on urban PM concentrations in New Zealand, even for inland locations, due to the isolated oceanic location of the New Zealand landmass. Other sources of Na and Cl include biomass burning, motor vehicle emissions, crustal matter, fireworks and industrial emissions.

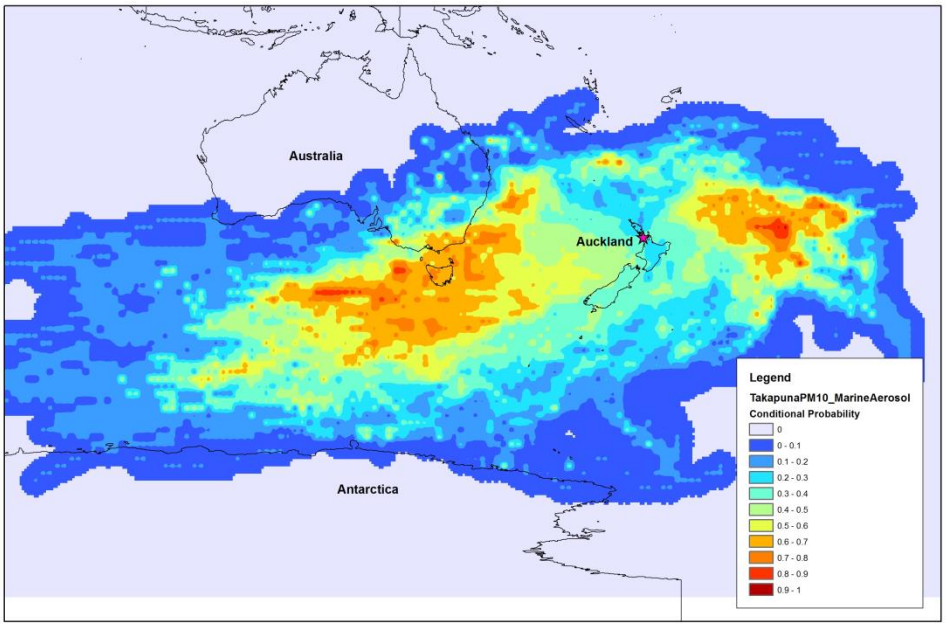
Figure B2: Scatterplots for sodium and chlorine in $PM_{2.5}$ (left) and PM_{10} (right) for all Auckland PM samples



Research has shown that the concentration of marine aerosol shows a strong dependence on wind speed across the ocean surface and ranges from about 2 µg/m^3 to as much as 50 µg/m^3 or more at wind speeds in excess of 15 m/s (Fitzgerald, 1991) and the Auckland data corroborates those potential concentration ranges. Therefore, marine aerosol concentrations in New Zealand urban areas are largely influenced by meteorological and long-range transport mechanisms as shown previously (Davy et al, 2011d).

The marine aerosol component of urban air PM is considered to be part of the 'natural' background and therefore is that proportion that cannot be managed. It has been shown previously that the primary marine aerosol generation and source regions were in the Southern Ocean below Australia and to the northeast of Auckland out in the Pacific Ocean as shown in the PSCF plot presented in figure B3 (Davy et al, 2011c; Davy et al, 2011d). Similar plots generated for other New Zealand locations show analogous source regions depending of predominant local air mass trajectories.

Figure B3: **PSCF plot (using 96-hour back trajectories) for the Takapuna PM_{10} marine aerosol source contribution data (2006 – 2013) showing that the most likely source regions are in the Southern Ocean below Australia and Pacific Ocean to the northeast of Auckland**



Secondary aerosol

Urban background $PM_{2.5}$ also includes secondary PM derived from atmospheric reactions of precursor gases, including VOCs, SO_2, NO_X and ammonia (**NH_3**). To estimate secondary particulate, receptor modelling (mining data from long $PM_{2.5}$ speciation datasets) or dispersion and chemical modelling (based on emission inventories, meteorological dispersion and chemical reactions to generate secondary PM) are needed. In New Zealand the results of PM speciation and receptor modelling have been used to derive the contribution of secondary PM to overall PM concentrations.

Several speciation monitoring programs (Auckland, Tokoroa, Baring Head, Christchurch and Timaru) have included specific secondary aerosol markers (eg ammonium, nitrate, sulphate, organic carbon), while hydrogen (a PM marker for ammonium and hydrocarbon aerosol including secondary organics) is routinely analysed in PM speciation samples. The data show that the sources of urban secondary aerosol are split between anthropogenic and natural emissions of precursor gases and that, as annual averages, secondary sulphate is the greatest contributor to urban secondary aerosol (about 10% or 1 to 2 µg/m^3) while secondary nitrate contributes about 0.6 µg/m^3 for both Auckland (population 1.5 million) and Timaru (population 44,000).

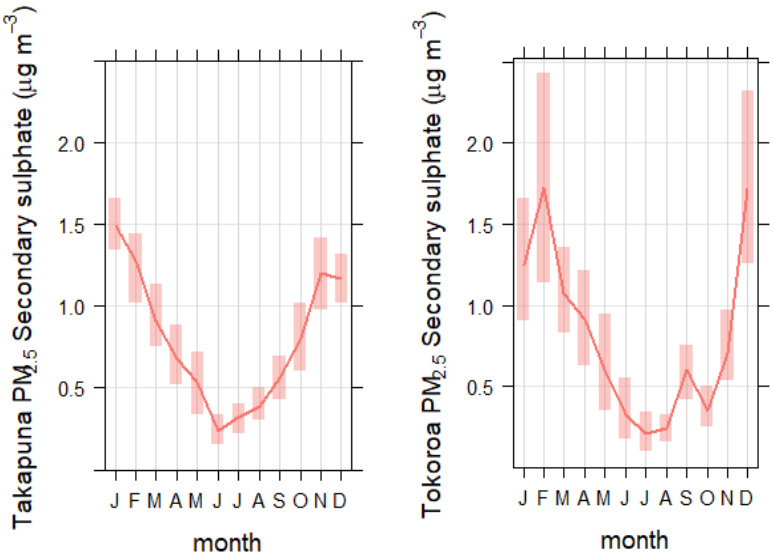
Speciation and receptor modelling studies in New Zealand have not yet identified a specific secondary organic aerosol (**SOA**) contributor to urban PM, and while it is likely such PM species are present, $PM_{2.5}$ mass closure analysis suggests that the mass contribution of SOA is likely to be somewhat less than 5%.

Secondary sulphate aerosol

The presence of sulphur in airborne PM is generated from a variety of sources including sulphur incorporated in mineral structures of crustal matter, cell structure of trees (released during biomass combustion), volcanic emissions, marine aerosol, and the combustion of

sulphur containing fuels including automotive fuels (petrol, diesel, fuel oils used by ships) and other fossil fuels such as coal. Sulphur-containing PM is also derived from precursor gases such as SO_2, hydrogen sulphide (**H_2S**) or dimethyl sulphide (**DMS**) from the gas-to-particle reaction process in the atmosphere. These reactions can take hours to days depending on the reaction pathway followed, the availability of catalytic metals (eg, Fe, Mn), relative humidity and the strength of solar radiation (Seinfeld and Pandis, 2006). Therefore, concentrations of S containing PM from secondary sulphate sources are likely to be highest some distance downwind of a precursor gas emission source (Polissar et al, 2001). Seasonal patterns show that secondary sulphate concentrations generally have a summer maximum and a winter minimum (figure B4), reflecting the relative influence of solar forcing on atmospheric reaction pathways.

Figure B4: Seasonal variation in secondary sulphate concentrations at (left) Takapuna, Auckland (2006-2013) and (right) Tokoroa, Waikato (2016)



The New Zealand source apportionment data indicates that there are both natural (oceanic, volcanic) and anthropogenic (shipping, motor vehicle and industrial emissions) sources of secondary sulphate aerosol. The relative contribution secondary sulphate particles to PM concentrations at a given air quality monitoring site is dependent on:

- local source precursor gas emission activity (both anthropogenic and natural)
- the proximity of a PM sampling site to such activities
- atmospheric chemical reaction kinetics (ie, the drivers for the gas-to-particle reaction pathway)
- long-range transport of natural source (volcanic and oceanic) secondary sulphate.

Receptor modelling studies of PM composition from around New Zealand show that PM monitoring sites near ports are likely to be influenced by secondary sulphate associated with emissions of precursor gases from ships engines. Also, some monitoring locations were influenced by direct emissions of combustion-derived PM from ships engines.

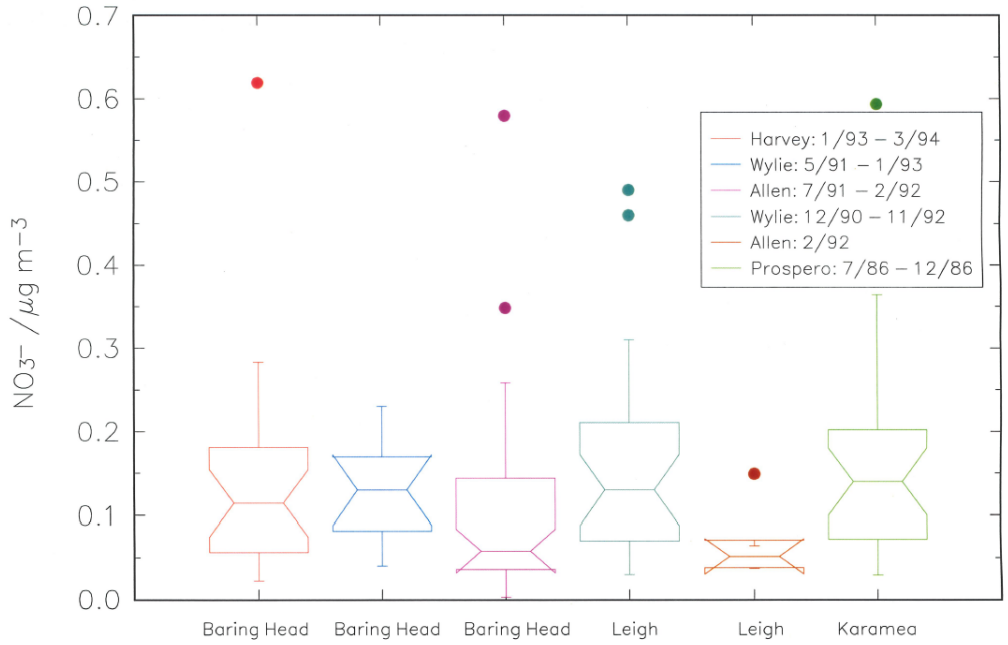
Secondary nitrate aerosol

Atmospheric concentrations of inorganic nitrogen-containing aerosol in the land-based lower boundary layer have primarily been measured at urban locations in New Zealand due to the

focus on PM compliance monitoring for health-effect based guidelines and standards. However, several studies of background oceanic aerosol in the Southern Ocean and coastal locations around New Zealand (Allen et al, 1996; Kristament et al, 1993; Ooki et al, 2007; Wylie and de Mora, 1996) provide baseline concentrations inorganic nitrogen-containing aerosol that are likely to impact across the New Zealand landmass as part of the west->east flow in Southern Hemisphere circulation patterns. Most of nitrogenous aerosol were measured as ionic nitrate (NO_3^-) and ammonium (NH_4^+) species, with much of the nitrate present as the ammonium salt (NH_4NO_3) (Allen et al, 1996).

Figure B5 provides a summary of nitrate concentrations measured at New Zealand coastal locations showing that average concentrations range between 50 and 150 ng/m^3.

Figure B5: Nitrate aerosol concentrations measured at New Zealand coastal locations



Source M J Harvey, NIWA

As a result of sampling campaigns during ship based voyages across the South Pacific, Jung and co-workers found that inorganic nitrogen in aerosols was composed of approximately 32% NO_3^- and 68% NH_4^+ since ammonium ions are also an important component of inorganic sulphate aerosol species (as $(NH_4)_2SO_4$) (Jung et al, 2011). Average ammonium ion concentrations in background oceanic air at Baring Head and the Southern Pacific were found to range between 50 and 80 ng/m^3 with maximum concentrations during summer (Allen et al, 1996; Jung et al, 2011; Ooki et al, 2007).

Monitoring of nitrogen aerosol at urban locations

There have been relatively few urban monitoring studies in New Zealand that have included nitrogen aerosol, with most being short-duration campaigns. This is partly because of challenges in effective sampling of nitrate aerosol due to its thermodynamic stability and physiochemical properties, but primarily due to an urban focus on anthropogenic combustion sources (motor vehicles, fossil fuels, biomass) as those most likely to be responsible for urban air pollution events. The studies of nitrate aerosol concentrations that have been conducted at various locations do allow for some generalisations about ambient concentrations, seasonality and origins of nitrate and ammonium species in urban areas.

In a series of short-term monitoring campaigns at Auckland and Christchurch between 2000 and 2004, Wang and co-workers analysed collected PM for nitrate and ammonium species amongst other parameters. Table B2 presents average nitrate and ammonia concentrations in the two cities. They found that, in general, nitrate was highest during winter at Christchurch and ascribed this to emissions from solid-fuel (wood) fires for home heating (Wang et al, 2005; Wang and Shooter, 2001).

Table B2: Average (maximum) concentrations of nitrate and ammonium species at Auckland and Christchurch (ng/m^3)

Species	Auckland	Christchurch
Nitrate (ng/m^3)	233 (1170)	734 (1670)
Ammonium (ng/m^3)	84 (290)	270 (960)

Source Wang et al (2005)

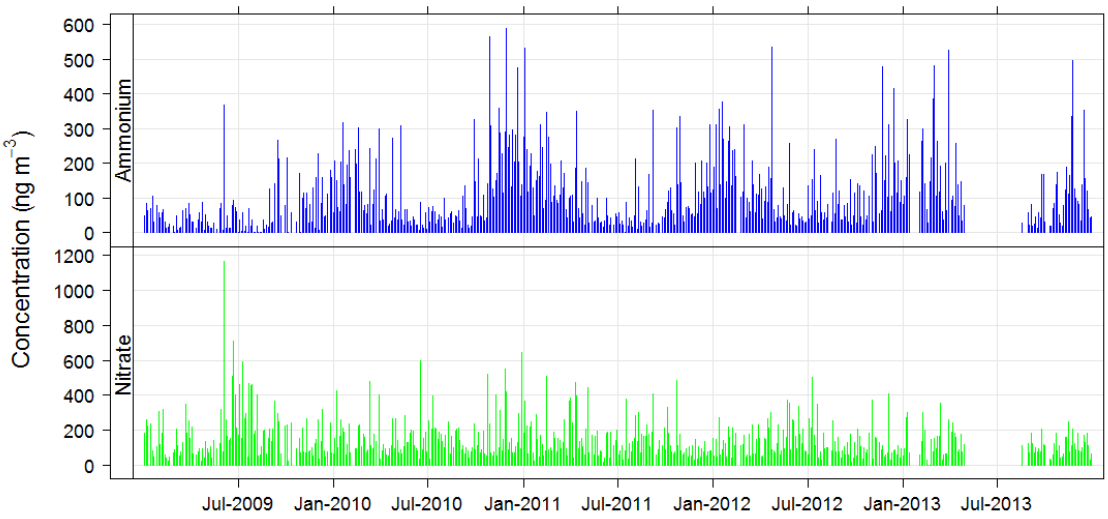
Without access to the detailed data, it is difficult to extract information from these studies that may inform the current discussion.

Analysis of urban sources of nitrate and ammonium

Takapuna, Auckland

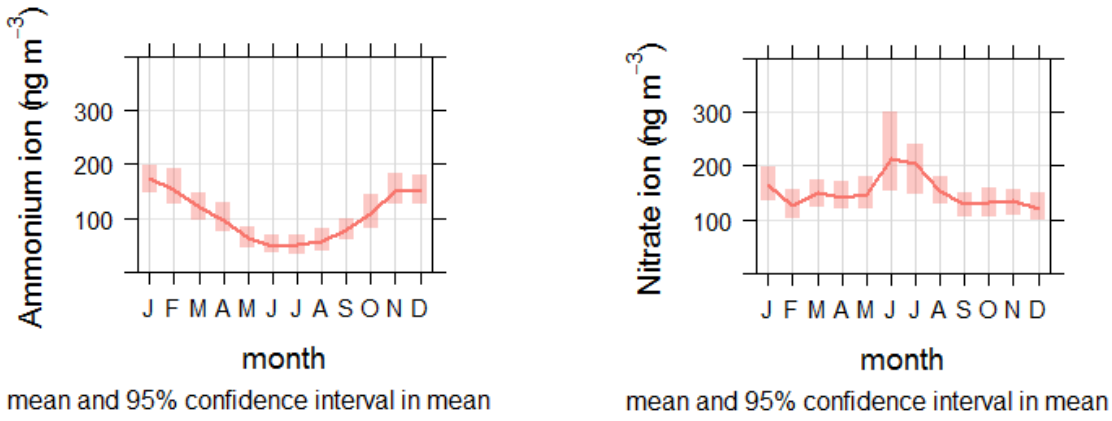
Nitrate concentrations were determined as part of a $PM_{2.5}$ dataset from an Auckland Council monitoring site at Takapuna on the North Shore (Davy et al, 2014). Sample collection for analysis, including nitrate and ammonium species, was from January 2009 to December 2013 (Selleck and Keywood, 2012). Figure B6 presents the time series data for nitrate and ammonium at the Takapuna site with average concentrations 150 and 106 ng/m^3 respectively.

Figure B6: Nitrate and ammonium concentrations at the Takapuna site (2009-2013)



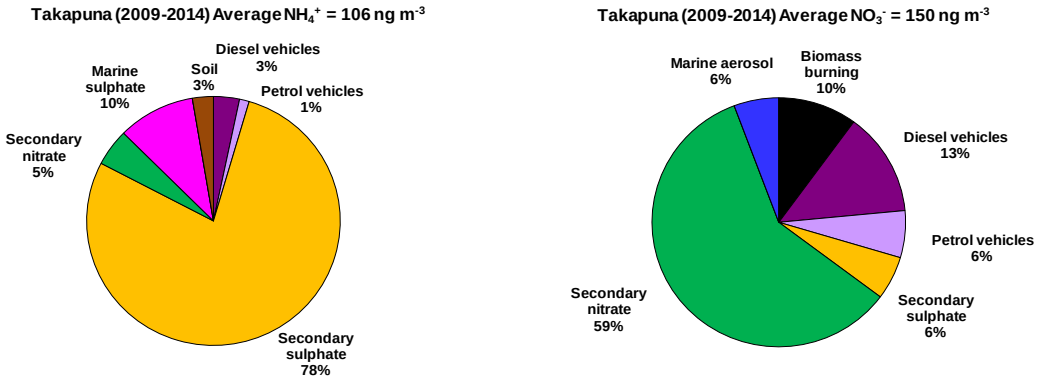
When the seasonality of concentrations was examined (figure B7), it showed that ammonium ion concentrations have a summer peak and winter minimum, while conversely nitrate had a small winter maximum which suggests differing sources for the two species.

Figure B7: Monthly average concentrations for ammonium and nitrate at the Takapuna site



Analysis of the Takapuna data by receptor modelling techniques was used to apportion the sources of $PM_{2.5}$ (Davy et al, 2014). Figure B8 presents the relative contributions of sources to ambient concentrations of ammonium and nitrate. It shows that most of the ammonium was associated with secondary sulphate aerosol formed from gas-to-particle atmospheric chemical reactions. The gaseous precursors were likely to have been generated from the combustion of sulphur containing fuels, which for Auckland, has been shown to be dominated by shipping emissions. However, at times volcanic emissions of sulphur gases can also contribute to the secondary sulphate (and therefore the associated ammonium ion) burden.

Figure B8: Sources of ammonium (left) and nitrate (right) at the Takapuna site

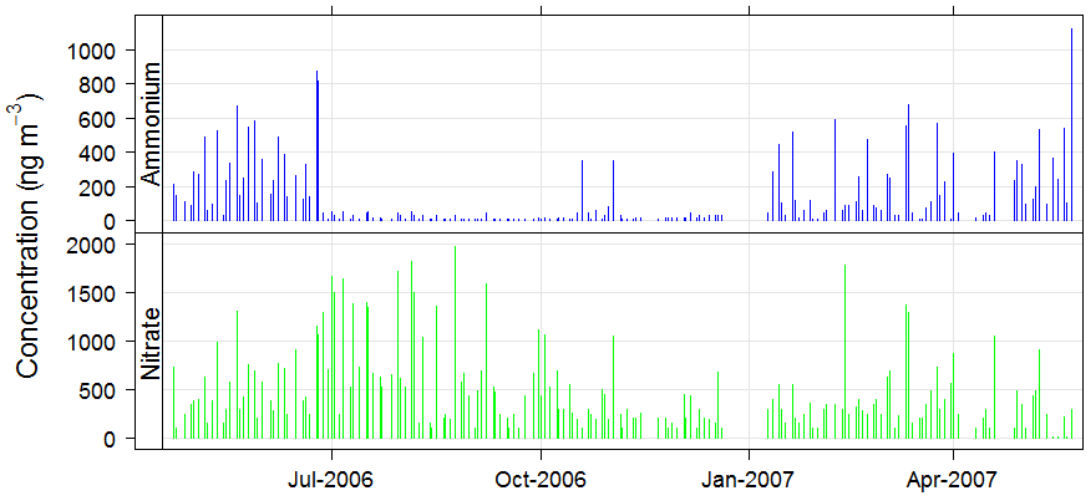


Nitrate concentrations at Takapuna were found to be dominated by a secondary aerosol source (analogous to the secondary sulphate production mechanism) that may be due to anthropogenic precursor combustion gases (dominated by motor vehicle NO_X emissions). However, it could also be representative of the oceanic background given the nitrate concentration associated with this source is around 90 ng/m^3 similar to those background sites presented earlier.

Timaru, Canterbury

A study of $PM_{2.5}$ sources carried out in Timaru from May 2006 to May 2007 also analysed for nitrate and ammonium aerosol species amongst a range of other variables (Scott, 2014). Figure B9 presents the nitrate and ammonium concentration data from the Timaru study showing that peak nitrate concentrations occurred during winter months June to August) at Timaru.

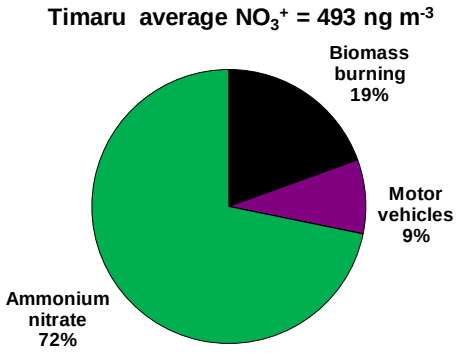
Figure B9: Nitrate and ammonium concentrations at the Timaru site (2006-2007)



Source: Environment Canterbury

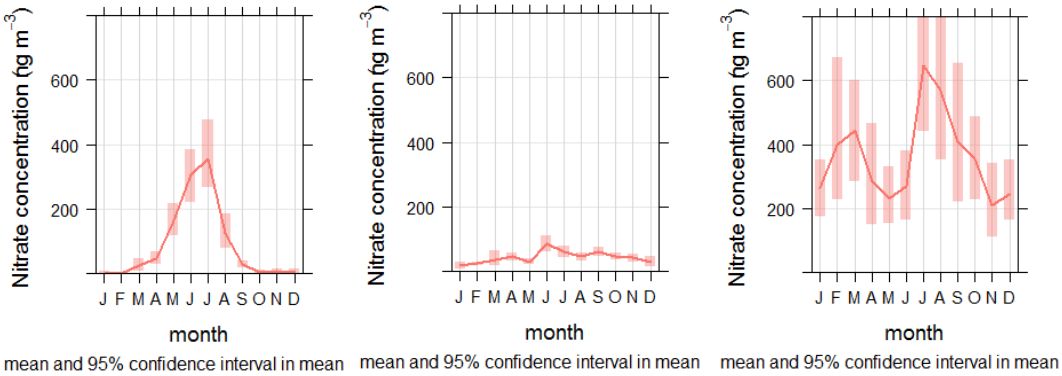
Analysis of the Timaru data by receptor modelling techniques indicates three primary sources of nitrate, biomass burning, motor vehicles and secondary aerosol ammonium nitrate which dominated nitrate concentrations as presented in figure B10.

Figure B10: Sources of nitrate aerosol at Timaru



Average nitrate concentrations at Timaru (493 ng/m^3) were significantly higher than Auckland (150 ng/m^3) but since the monitoring periods were not coincident there can be no direct comparison between the results. An examination of the seasonality of the Timaru nitrate sources showed that the biomass burning source was mainly present during the winter months due to the association with solid fuel fires for home heating, the motor vehicle contribution was at low levels across all the months, while the ammonium nitrate source had higher but variable concentrations all year as shown in figure B11.

Figure B11: Monthly average nitrate concentrations associated with biomass burning (left), motor vehicles (middle) and ammonium nitrate sources at Timaru

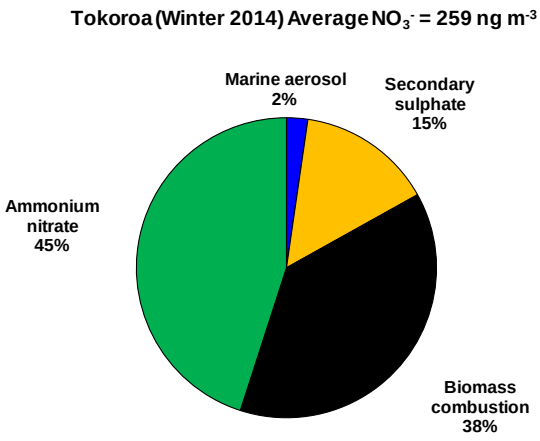


The Timaru data suggests that the ammonium nitrate source may be a combination of the background atmospheric (oceanic) nitrate with more local land-based emissions that could potentially be from rural sources.

Tokoroa, Waikato

A PM monitoring programme was conducted from May to October 2014 at Tokoroa to assess the sources contributing to high winter concentrations of PM pollution (Ancelet and Davy, 2015). Nitrate and ammonium species were measured as part of the programme. Receptor modelling of the data found that the nitrate was associated with biomass combustion, secondary sulphate, ammonium nitrate and a minor component with marine aerosol (sea salt). The sources contributing to nitrate concentrations are presented in figure B12. Most of the measured ammonium (92%), was associated with the ammonium nitrate source. Biomass combustion, a major contributor to ambient nitrate concentrations during winter in Tokoroa, represents emissions from solid fuel (wood) fires for home heating and is the primary particulate matter source in the town.

Figure B12: Sources of nitrate aerosol at Tokoroa



Due to the brevity of the Tokoroa study the exact source of the ammonium nitrate was uncertain. The study indicated that the ammonium nitrate source contributions peaked under low wind speeds from the north and that it was possible that agricultural emissions, associated with pasture and livestock, were responsible for elevated ammonium nitrate contributions.

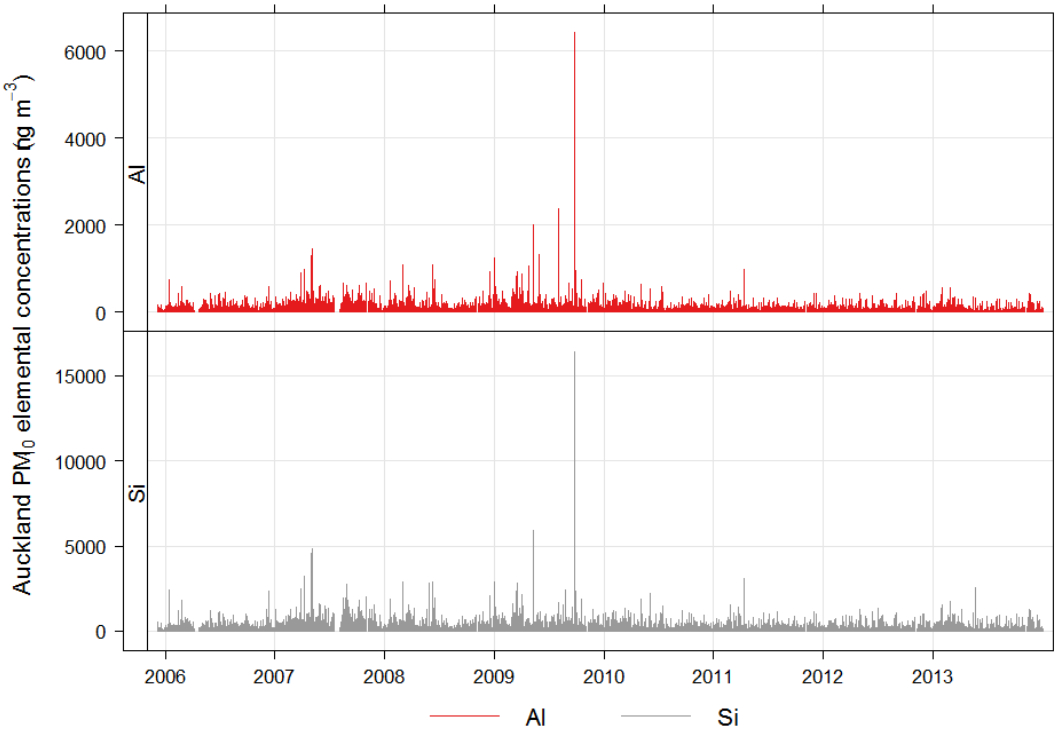
However, contributions from oceanic background nitrate cannot be ruled out given that the average concentration of the ammonium nitrate source was 117 ng/m^3 and was of the same order as those nitrate concentrations presented earlier for oceanic and background sites.

Crustal matter

Crustal matter is primarily composed of aluminosilicate minerals and the source profiles extracted from receptor modelling reflect this, with Al and Si being the primary constituents and Mg, K, Ca, Ti and Fe commonly present. The mass ratio of Si/Al is consistently about 3:1 for both PM_{10} and $PM_{2.5}$ size fractions across all New Zealand monitoring sites and is similar to the Si/Al ratio in aluminosilicate minerals. Al and Si concentrations were primarily associated with crustal matter (synonymous with soil as a source reference) which is predominantly a coarse particle source generated by mechanical abrasion of surface material. In urban locations, the passage of motor vehicles over roads can be the primary source of crustal matter suspension and resuspension (Thorpe and Harrison, 2008).

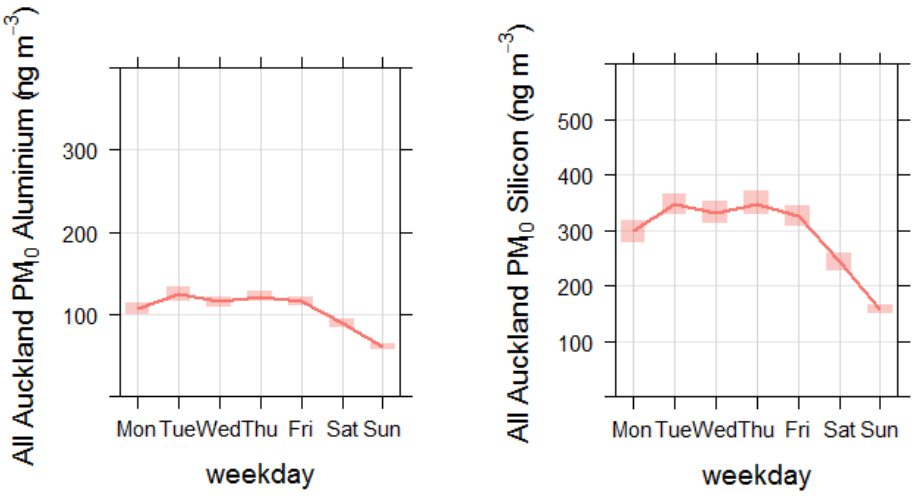
A specific dust event that resulted in PM_{10} exceedances across the Auckland region was identified as originating from a dust storm (a natural event) in the Australian desert during September 2009 (Davy et al, 2011d), the influence of which can be seen in the time-series plots for Al and Si in all Auckland PM_{10} samples presented in figure B13.

Figure B13: Time-series plots for aluminium and silicon in all Auckland PM_{10} samples



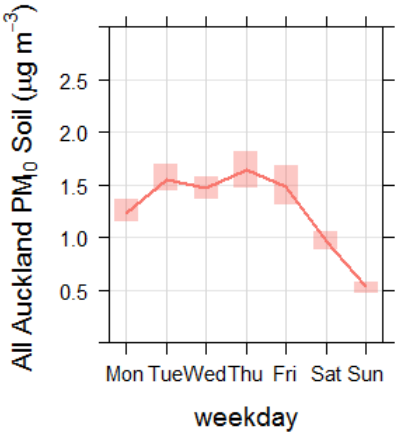
The temporal variation for both Al and Si concentrations indicate that airborne concentrations are primarily from anthropogenic activities because of the day-of-the-week concentration dependence with weekend concentrations significantly lower than weekdays as presented for Auckland data in figure B14.

Figure B14: Temporal variations in aluminium (left) and silicon (right) in all Auckland PM_{10} samples (the shaded bars are the 95 percentile confidence limits in the mean)



Crustal matter source contributions at the monitoring sites were likely to be a combination of windblown soil, road dust and dust generated by earthworks, construction and road works. Concentrations were found to vary from site to site depending on meteorological conditions and local dust generating activities. It can be seen from figure B15 that the temporal variation for PM_{10} crustal matter contributions (Auckland data) that concentrations during the weekend were significantly lower than during weekdays which indicates that crustal matter source emissions were primarily driven by human activity because any randomly generated emissions such as wind -blown dust, would not show a bias for day of the week due to the random nature of meteorological events.

Figure B15: Temporal variations in crustal matter contributions for all Auckland PM_{10} samples showing lower weekend concentrations (the shaded bars are the 95 percentile confidence limits in the mean)



B.4.2 Shipping emissions

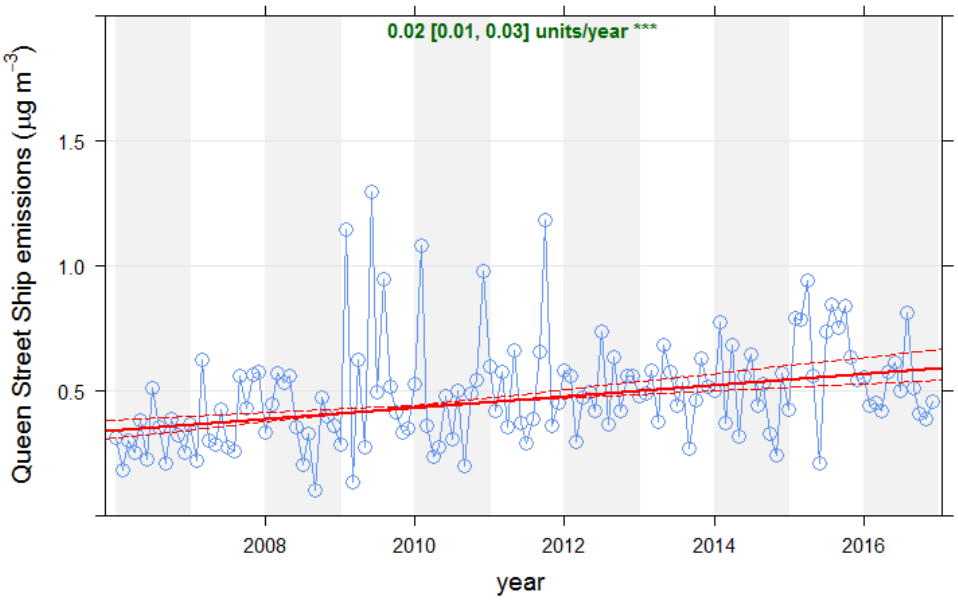
Receptor modelling studies of PM composition from around New Zealand show that PM monitoring sites near ports are likely to be influenced by secondary sulphate associated with emissions of precursor gases from ships engines. Also, some monitoring locations were influenced by direct emissions of combustion-derived PM from ships engines.

Concentrations of vanadium (**V**) and nickel (**Ni**) were found to be highest in PM samples from monitoring sites near port facilities and were found to be associated with combustion products from ships engines (Davy et al, 2008; Davy et al, 2011d; Davy et al, 2011b; Ancelet et al, 2014d; Davy et al, 2017). The major factor is the use of residual or bunker oil as fuel for ships which is generally of poor quality, high in sulphur, PAHs and heavy metals that can result in high sulphate containing PM emissions contaminated with alkali earth and transition metals (V, Ni, Ca, Fe) (Fridell et al, 2008; Moldanová et al, 2009).

Emissions of combustion products from ships engines can impact on local air quality in port areas, regional air quality and global climate (Huebert, 1999; Endresen et al, 2003; Ault et al, 2009; Eyring et al, 2010; Hellebust et al, 2010; Matthias et al, 2010). Species emitted to atmosphere from ships engines include usual combustion products (CO_x, NO_x), gaseous sulphur oxides (SO_X) that relate to fuel composition, VOCs from incomplete fuel combustion and PM which includes trace heavy metals (eg, V and Ni) (Healy et al, 2009; Agrawal et al, 2008b; Agrawal et al, 2008a; Fridell et al, 2008).

The analysis of PM data from the Auckland Council Queen Street monitoring site showed that there was an impact on the Auckland CBD due to combustion emissions from ships engines and that there appeared to be an upward trend in concentrations as presented in figure B16 most likely due to increasing shipping activity at the Port of Auckland (Davy et al, 2017).

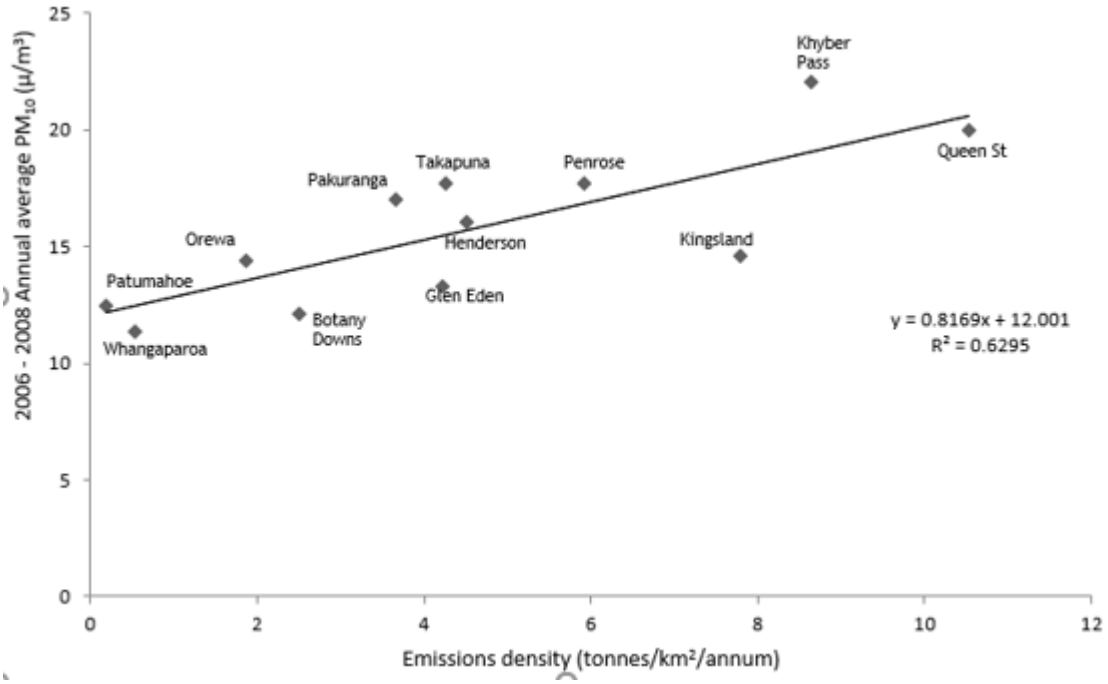
Figure B16: Trend in PM_{10} shipping emission source concentrations at Queen Street (statistically significant at the 99.9 % confidence interval)



Appendix C: Spatial analysis

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. For example, figure C1 shows the correlation between annual average PM_{10} (for the years 2006 – 2008) and estimated emissions density in Auckland from HAPINZ 2.0.

Figure C1: Example 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 (2012)

We have updated this analysis based on estimated 2013 emissions density and monitoring data. Figure C2 shows the same data for Auckland in 2012 – 2014, it is clear that there is no correlation for Auckland.

Figure C2: 2013 correlation between emissions density and annual average PM_{10} in Auckland

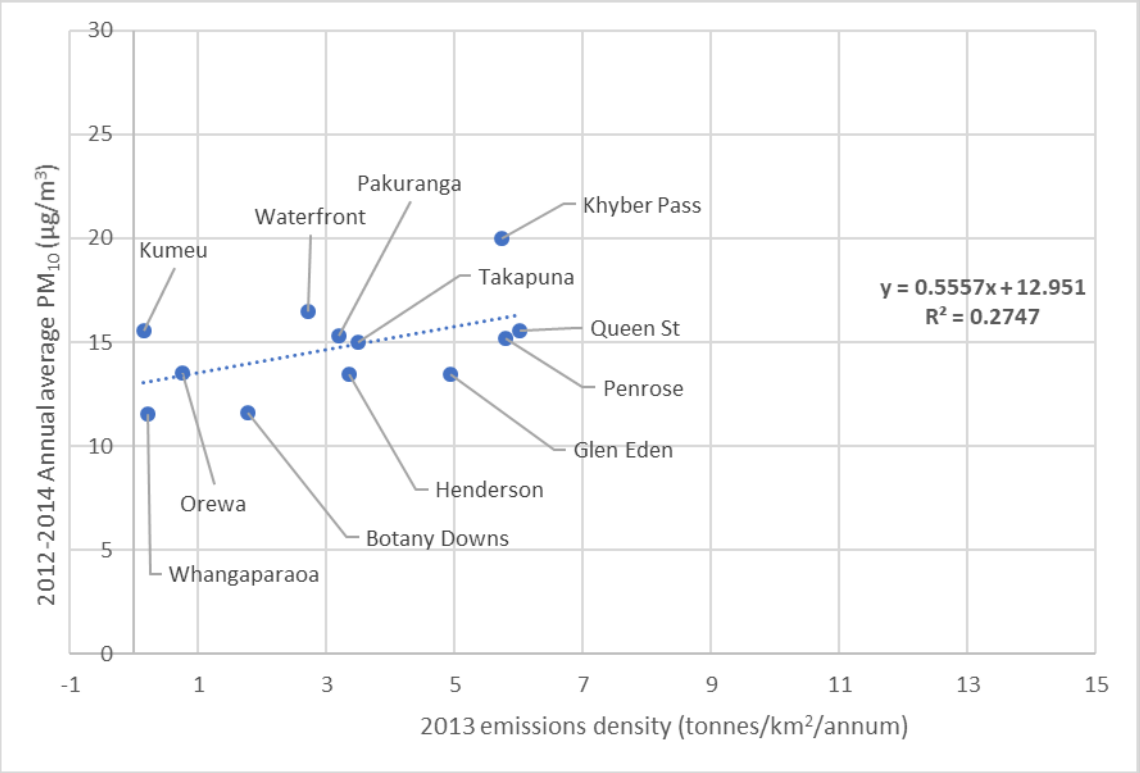


Figure C2 shows that the concentration of PM_{10} is reasonably consistent across the Auckland airshed, and is not significantly influenced by localised emissions density. We hypothesise that the influence of local factors such as meteorology, topography and proximity of the monitoring sites to roads are more significant.