### **MARCH 2024**

## PREPARED FOR Tauranga City Council and Bay of Plenty Regional Council

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Health risks of exposure to air pollution in the Mount Maunganui Airshed – a technical review

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## EXECUTIVE SUMMARY

This report provides a review of the Environmental Science and Research (ESR) 2023 report "Air Pollution: Health Risk Assessment (HRA) Mount Maunganui" (henceforth referred to as the ESR report). The purpose of the review is to assist decision-makers with the reliability of findings on the health implications of air quality in and around the Mount Maunganui industrial area.

The Mount Maunganui Airshed (MMA) is unique in a New Zealand context owing to the proximity of residential housing to a very complex pollutant mix.

The ESR report assesses the health impacts on Mount Maunganui residents including quantification for the pollutants particulate matter less than 2.5 microns in diameter ( $PM_{2.5}$ ), particulate matter less than 10 microns in diameter ( $PM_{10}$ ) and nitrogen dioxide ( $NO_2$ ) for a selection of health endpoints. It includes a qualitative assessment for sulphur dioxide ( $SO_2$ ), benzene and hydrogen sulphide ( $H_2S$ ). The approach of quantification of the burden of disease associated with exposure to air contaminants is accepted internationally.

The report uses air quality data from monitoring sites to estimate exposures in surrounding areas. This is the method used in health risk assessments and is appropriate as long-range transport of particulates, including fugitive dust sources, is known to occur over significant distances including across continents. The extrapolation of data from a monitoring site to represent population exposures across a large area is inherent in both risk assessments and the epidemiology underpinning them. The MMA is the most comprehensively monitored in the country and thus extrapolation distances (up to five kilometres) are significantly less than what is typical for risk assessments.

We consider the specific approach to exposure assessment in the ESR report (i.e., the data used) is generally robust although note a slightly lower exposure for PM<sub>10</sub> may be appropriate for Arataki. We do not consider this to be overly significant. We also note that NO<sub>2</sub> impacts may be underestimated as a model was used to derive these that does not take into account shipping emissions.

We consider the concentration response functions (i.e., the relationship between the concentration of pollutants and the response it elicits in the population) used in the HRA to be the best available for New Zealand but note that there are some differences between the Mount Maunganui Airshed and the urban environments used to derive them. The method used for baseline health data appears appropriate.

In our view the Ōtūmoetai comparison should be limited to impacts of PM<sub>10</sub>. Age adjusted baseline health data should be used if differences between areas are to be attributable to air quality impacts.

The quantified impacts for premature mortality from the ESR report range from 19 premature deaths per year to 26 premature deaths per year depending on the model used. Hospital admissions, asthma impacts and restricted activity days are also estimated. They conclude a moderate degree of uncertainty in the analysis, which is considered appropriate for this level of assessment.

In our view the calculations of numbers for premature mortality and hospital admissions identified in the HRA are indicative of the scale of impact for residents of these areas (noting that due to the nature of the analysis any HRA will provide an indication of scale rather than an exact number output).

We note that the HRA was unable to assess the health risk for workers in the MMA. For non-residents that are exposed for prolonged periods there is a health risk to them. This includes over 11,000 workers, and children in childcare centres. Children are particularly susceptible to acute impacts of coarse particulate exposure. Other susceptible groups include the elderly and those with underlying cardiopulmonary disease (a risk factor for smoking).

The ESR report supports the need to manage and minimise emissions of all contaminants in the MMA but with specific attention to  $PM_{10}$ ,  $PM_{2.5}$ ,  $NO_2$  and  $SO_2$ . The main sources of  $PM_{10}$ ,  $PM_{2.5}$  and  $SO_2$  are industrial activities, port activities and shipping. Motor vehicles and shipping are the main sources of  $NO_2$ . Resource

consents and land use planning are tools that can be used to improve air quality from industrial and trade activities and land use planning can assist by minimising exposures.

Overall, our findings are that the risk assessment contributes to the understanding of health impacts of air quality in the Mount Maunganui area and provides an indication of the scale of impact using premature mortality and hospital admissions. We found no issues of substance and concur with the findings that air quality in the area will result in premature mortality and hospital admissions. We consider the calculations of numbers for these health endpoints likely to be indicative of the scale of impact.

## **1 INTRODUCTION**

### 1.1 Scope

This report provides a review of the ESR 2023 report "Air Pollution: Health Risk Assessment Mount Maunganui" (henceforth referred to as the ESR report). The purpose of the review is to assist decision-makers with the reliability of findings on the health implications of air quality in and around the Mount Maunganui industrial area.

The ESR report provides a risk assessment which quantifies the impacts of  $PM_{2.5}$ ,  $PM_{10}$  and  $NO_2$  on health and makes qualitative assessments of  $SO_2$ , benzene and odour impacts.

This review includes an evaluation of the nature of the airshed (sources and contaminant issues) and the health impacts of particulate to assist with assessment of the suitability of the methods in the ESR report.

### 1.2 Background

The Mount Maunganui Airshed (MMA) refers to the area illustrated in Figure 1.1 which has been gazetted under the National Environmental Standards (Ministry for the Environment, 2004). The airshed is "polluted" under that standard owing to breaches of the NESAQ for  $PM_{10}$  (50 µg/m<sup>3</sup>, 24-hour average, one allowable exceedance per year).



Figure 1.1: MMA Emission Inventory Area (source Bay of Plenty Regional Council, 2022) with risk assessment area overlay (upper left) from the ESR report.

The Port of Tauranga (POT), which extends several kilometers along the western side of the airshed, is a key determinant of industrial activities in the area. Air discharges at the POT include shipping, bulk solid material<sup>1</sup> (BSM) loading and unloading, log handling and storage, transport and cargo handling equipment. The MMA includes BSM handling and storage (range of materials), additional log handling and storage areas, two significant sources of SO<sub>2</sub> (Ballance Agri-Nutrients Limited and Lawter NZ Ltd), combustion sources of PM<sub>2.5</sub> and PM<sub>10</sub> and other products of combustion (Lawter, Waste Management Oil Recovery, Asphalt Plants, Grain Processing), abrasive blasting, cremation and bulk fuel storage facilities.

The 2018 census suggests around 200 residential dwellings are located in the 8 km<sup>2</sup> airshed including at Whareroa Marae, around De Havilland Way, around the Rata Street area to the north, living at the marina and a small number within the area to the east of POT. Additionally, around 11,800 workers including shift workers are exposed to air quality in the Mount Maunganui Central area and a further 1,200 exposed at Sulphur Point (pers com Tauranga City Council, 2024). The ESR report illustrates the airshed includes at least five early childhood education centres including one at Whareroa Marae, one within 300 metres of Rata Street and two within 700m of De Havilland Way.

The industrial area merges into residential and recreational areas to the north and east with the broader Mount Maunganui area comprising around 14 km<sup>2</sup> in total. The residential band to the north and east of the airshed is narrow and spans only a kilometre from the airshed boundary at most points. This area includes Omanu School, Mount Maunganui College, Mount Maunganui Intermediate School, Mount Maunganui Primary School, at least 10 early childhood education centres, and at least five rest homes, retirement villages or aged care facilities. The population of the broader Mount Maunganui area, used in the ESR report risk assessment for 2019 is 16,975.

The ESR report considers the health impacts of five pollutants suspended particulate ( $PM_{10}$ ), fine particulate ( $PM_{2.5}$ ), nitrogen dioxide ( $NO_2$ ), sulphur dioxide ( $SO_2$ ) and benzene as well as impacts of  $H_2S$  (odour) for the Whareroa Marae and in the broader Mount Maunganui area. The quantified risk assessment is limited to  $PM_{2.5}$  and  $NO_2$  (from a joint pollutant model) and  $PM_{10}$  (single pollutant model) which includes impacts of  $PM_{2.5}$  (being a component of  $PM_{10}$ ).

The HAPINZ 3 model on which the HRA is based is typically utilized to estimate health impacts for different cities and regions of New Zealand. Table 1.1 shows summary Regional Data from that study.

PM <sub>2.5</sub>	No. of cases	Cost \$million/yr
Premature mortality - all adults 30+ (annual PM <sub>2.5</sub> )	135	612.6
* Premature mortality - Māori adults 30+ (annual PM <sub>2.5</sub> )	27	124.0
* Premature mortality - Pacific adults 30+ (annual PM <sub>2.5</sub> )	1	5.7
Cardiovascular hospitalisations - all ages (annual PM <sub>2.5</sub> )	285	10.5
Respiratory hospitalisations - all ages (annual PM <sub>2.5</sub> )	239	7.6
Restricted activity days - all ages (annual PM <sub>2.5</sub> )	159,262	14.2
NO <sub>2</sub>		
Premature mortality - all adults 30+ (annual NO <sub>2</sub> )	130	590.4
Cardiovascular hospitalisations - all ages (annual NO <sub>2</sub> )	122	4.5
Respiratory hospitalisations - all ages (annual NO <sub>2</sub> )	412	13.1

#### Table 1.1: HAPINZ 3 health impact assessment output for Bay of Plenty Region for 2016.

<sup>&</sup>lt;sup>1</sup> Bulk solid materials means materials consisting of, or including fragments, that could be discharged as dust or particulate. These materials include but is not limited to: gravel, quarried rock, fertiliser, coal cement, flour, rock aggregate, grains, compost, palm kernel extract, tapioca and wood chip (but do not include logs, salt or other materials not in bulk form such as materials contained in a bag, container or similar).

Asthma/wheeze hospitalisations - 0-18 year olds (annual NO <sub>2</sub> )	51	0.1
Asthma prevalence - 0-18 year olds (annual NO <sub>2</sub> )	659	0.1
Total costs (Prem.mortality, cardio & respiratory hosps, & RADs)	1,252.9	1,252.9
PM10		
PM <sub>10</sub> Premature mortality - all adults 30+ (annual PM <sub>10</sub> )	263	1,192

## 2 THE NATURE OF THE AIRSHED

## 2.1 Air contaminants

Air contaminants in the Mount Maunganui Airshed (MMA) include total suspended particulate (TSP), PM<sub>10</sub>, PM<sub>2.5</sub>, SO<sub>2</sub>, NO<sub>2</sub>, benzene, H<sub>2</sub>S and methyl bromide (Iremonger, (2023)) and these are identified in the ESR report. Health impacts of methyl bromide is not considered in the ESR report and similarly is not evaluated here other than to note that it will contribute to the pollutant mix.

The contaminants that have been identified in both reports as having been of concern relative to health guidelines include  $PM_{10}$ ,  $PM_{2.5}$  and  $SO_2$ . Iremonger (2023), proposed monitoring of  $NO_2$  (as a contaminant of emerging significance) and this commenced in August 2023. In the ESR report benzene is noted as a contaminant that could be of concern in the MMA. The airshed also contains industrial activities that can cause odours (e.g., asphalt production, fertilizer manufacture and fibre glassing activities) and has many activities that give rise to nuisance dust issues.

Particulate is one of the main contaminants that causes health and nuisance issues in the MMA. Larger particles (TSP) cause dust nuisance, whilst the smaller size fractions  $PM_{10}$  and  $PM_{2.5}$  penetrate in the lungs resulting in health impacts.

### 2.2 Airshed issues

Key questions relating to the nature of the airshed and health impacts (and assessment of) within the scope of this report are:

- The dominance of the coarse (PM<sub>10</sub>-PM<sub>2.5</sub>) size fraction of PM<sub>10</sub> in the MMA and its health implications relative to the PM<sub>2.5</sub> size fraction.
- If coarse particulate results in less health impacts do its sources still require management (are they contributing to other issues (e.g., dust nuisance) or contaminants with health concerns e.g., PM<sub>2.5</sub> concentrations).
- The extent to which air quality issues might be localized and only cause exposure issues in the vicinity of the sources.
- Are contaminants adequately characterized to enable health impact assessment.

A number of investigations have been carried out as a part of this work to provide better information on the nature of the airshed and some of these key questions to assist with the review of the ESR report. Some detailed analysis of data is included in Appendices A (dilution of fugitive point sources across the airshed) and B ( $PM_{2.5}$  to  $PM_{10}$  ratios). This section evaluates some of the key issues identified above and includes interpretations from Appendices A and B where relevant. It also presents an overview of the airshed sources and concentrations to ensure all have been adequately considered in the health risk assessment. The health implications of coarse particulate are detailed in Chapter 3.

## 2.3 Fine and coarse particulate

The MMA is classified as a polluted airshed because concentrations of  $PM_{10}$  exceed the National Environmental Standard of 50 µg/m<sup>3</sup> (24-hour average) on more than one occasion per year. Additionally annual average concentrations of  $PM_{10}$  and  $PM_{2.5}$  exceed the World Health Organisation (WHO) 2021 guidelines. Particulate is a key contaminant of concern in the MMA and has been the key focus of air quality monitoring programmes (Iremonger, 2023).

Particle size influences health impacts of particulate exposure owing to its impact on the different mechanisms by which particles can impact on health. Historically, epidemiological studies focused on PM<sub>10</sub> as the most prevalent size fraction monitored in the 1990's and 2000's. The methodology for these studies over this period also evolved from a strong dependency on time series studies of acute (short term) exposures to cohort studies and evaluation of chronic (long term) impacts. Initial guidelines were based on daily exposures of PM<sub>10</sub>, whereas current understanding indicates greatest health risks are associated with the PM<sub>2.5</sub> size fraction and chronic exposures (World Health Organisation, 2014). As PM<sub>2.5</sub> is a component of the PM<sub>10</sub> health studies based on PM<sub>10</sub> measurements, where the smaller size fractions are present, will include health impacts of PM<sub>2.5</sub>.

For the purposes of understanding potential health impacts in the MMA we will classify particulate as:

- PM<sub>2.5</sub> (particles less than 2.5 microns in diameter)
- PM<sub>10</sub> (particles less than 10 microns in diameter this includes PM<sub>2.5</sub>)
- Coarse particulate (particles between 2.5 and 10 microns in diameter i.e., PM<sub>10</sub>-PM<sub>2.5</sub>)

Health impacts of the two size fractions may vary as the smaller particles (in the PM<sub>2.5</sub> size fraction) can penetrate deep into the respiratory system and enter the bloodstream whereas larger particles are more likely to settle in the upper airways (Miller & Xu, 2018).

It is also worth considering the nature of the different size fractions as this aids with understanding around predominant sources. Historically, particulate was classified by modes of formation, which were referred to as fine and coarse, and which largely separated particles based on the source of the material. Primary fine-mode particles result from the condensation of molecules, typically from combustion processes, while secondary fine mode particles result through the reaction of gases in the atmosphere.

Coarse-mode particles are formed by mechanical processes such as crushing, grinding and abrasion of surfaces, during which larger pieces of material are broken down to smaller pieces. Fungal spores, pollen, and plant and insect fragments are examples of natural bio-aerosol, which may form part of suspended coarse-mode particles. Coarse particles also include windblown soils and sea-spray. The latter is a marine aerosol derived from the surface of the sea in conjunction with wind. The size distribution of marine aerosol is generally between 0.1 and 20  $\mu$ m, peaking at 6-8  $\mu$ m (USEPA, 2009).

The majority of fine-mode particles typically fall within the  $PM_{2.5}$  size fraction (particles less than 2.5 microns in diameter) and similarly coarse-mode particles within the  $PM_{10}$ - $PM_{2.5}$  size fraction. However, the  $PM_{2.5}$  size fraction will include coarse-mode particles and the  $PM_{10}$ - $PM_{2.5}$  size fraction can include fine-mode particles. Classification is now based on size with measurements made for  $PM_{10}$  and  $PM_{2.5}$  size fractions and with the coarse particulate ( $PM_{10}$ - $PM_{2.5}$ ) comprising the difference between these two measurements.

### 2.4 Transport of pollutants – impacts on exposure

The transport and dispersion of pollutants in the air is a key variable that impacts exposures. The impact of emissions from a single source will depend on characteristics of the source as well as meteorological conditions. Dispersion of contaminants from a stack at temperature will be different to dispersion from a ground level source at ambient temperatures. The impacts of meteorological variables such as atmospheric stability and wind speed will also significantly impact the dilution of an emission source. High winds are often associated with greater dispersion but for some sources, such as particulate from the storage and handling of logs and from the handling of bulk solid materials, high wind speeds can also increase the emission rate. There is ample evidence of long-range transport of fugitive dust sources over significant distances including countries and even continents (e.g., Tanaka & Chiba, 2006; UNEP, WMO & UNCCD, 2016; Middleton, 2017).

The MMA has a mixture of industrial storage, handling, process and combustion sources, shipping combustion sources, port activities, fugitive dust sources, transport and equipment operation combustion emissions. In addition, marine aerosol will contribute to concentrations of PM<sub>10</sub> and PM<sub>2.5</sub> under certain wind and sea state conditions. These sources will have different dispersion characteristics under different meteorological

conditions and will impact different locations under different wind conditions. Collectively the impact will also vary depending on the meteorological conditions.

The extrapolation of data from a monitoring site to represent population exposures across a large area is inherent in both risk assessments and the epidemiology underpinning them. In New Zealand monitoring data are typically extrapolated between 10 and 30 kilometers and in the MMA extrapolations are much smaller (five kilometers maximum).

Industrial airsheds do typically include more fugitive PM<sub>10</sub> sources, however, and to address potential dispersion issues with these sources an additional evaluation was carried out (Appendix B). Appendix B suggests that sources of PM<sub>10</sub> located close to and upwind of the Rail Yard South (RYS) monitoring site under a south-west (SW) wind likely also impact on the Rata Street monitoring site, which is located 1.2 kilometers away. Under these conditions and for higher wind speeds the Rata Street concentrations are most commonly around 60-70% of those at RYS. Appendix B also illustrates that when the wind is blowing from the south-east (from the industrial area to the monitoring sites) and there is a correlation between the two sites that the concentrations at Rata Street are not much less than at RYS when the windspeed is 2-4 ms<sup>-1</sup>. Whilst this analysis does not quantify the impact of transport across the airshed it demonstrates that sources of PM<sub>10</sub> in the airshed do not just result in localised impacts and that PM<sub>10</sub> transportation is occurring.

Whilst meteorological conditions giving rise to peak concentrations (short term averages) may result in localised effects, the literature and data analysis suggests that these emissions will also be transported, and this will have an impact on chronic exposures in residential areas surrounding the MMA. It is also noted that spatial variability in PM<sub>2.5</sub> concentrations tends to be lower than that observed for other size fractions (USEPA, 2019).

## 2.5 Sources of $PM_{10}$ , $PM_{2.5}$ , $NO_2$ , $SO_2$ and benzene

A health risk assessment carried out by ESR quantifies the health risks associated with  $PM_{2.5}$ ,  $PM_{10}$  and NOx. This section considers these contaminants in the context of the MMA and SO<sub>2</sub>, as a contaminant that has exceeded guideline values historically in the MMA. A qualitative analysis of health impacts of SO<sub>2</sub> is included in the ESR report. Whilst these contaminants are the main focus, the pollutant mix that residents, workers and other users of the area are exposed to will be more complex than most areas of New Zealand and consequently more complex than the areas used as the basis for the concentration response functions<sup>2</sup> (CRFs) underpinning risk assessment in New Zealand.

#### 2.5.1 Emission inventory assessment - PM<sub>10</sub>, PM<sub>2.5</sub> in the MMA

An emission inventory carried out in 2022 shows the estimated relative contribution of sources to PM<sub>10</sub> and PM<sub>2.5</sub> emission in the MMA (Figure 2.1) (Wilton, 2023). The inventory emission estimates represent total discharges into the airshed but do not take into account dispersion mechanisms that impact on ground level concentrations. For example, emissions from shipping are discharged at height with velocities and temperatures which increase the buoyancy of the plumes resulting in increased dispersion prior to reaching ground level. Thus, whilst providing an indication of sources into an airshed an emission inventory may not represent the impacts of different sources on concentrations measured at a monitoring site or exposures at different locations.

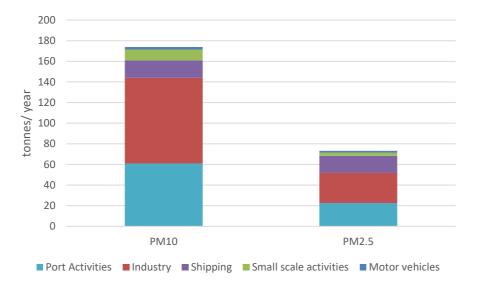
The main sources of  $PM_{10}$  and  $PM_{2.5}$  as indicated by the emission inventory are industrial activities including those from the Port of Tauranga (identified separately in Figure 2.1).

The extent to which sources of PM<sub>10</sub>, which are known to exceed national environmental standards and guidelines also contribute to PM<sub>2.5</sub> may also be relevant from an air quality management viewpoint. As indicated previously, sources of coarse mode particulate (formed through mechanical or abrasive activities) can result in

<sup>&</sup>lt;sup>2</sup> Concentration response function (also referred to as exposure-response function) is the increase in health impact for an incremental increase in contaminant concentrations. This can be expressed as a relative risk (RR) (e.g., 1.11 for a 10  $\mu$ g/m<sup>3</sup> increase in PM<sub>10</sub> in the ESR report) or a percentage increase in baseline health data (e.g., 11.1% per 10  $\mu$ g/m<sup>3</sup> exposure).

discharges in the PM<sub>2.5</sub> size fraction. Emissions data from United States Environmental Protection Agency (USEPA) indicate that fugitive PM<sub>10</sub> sources including BSM handling, log handling and log storage (included in both Port Activities and Industrial activities) result in PM<sub>2.5</sub> discharges with emission factor data suggesting the PM<sub>2.5</sub> fraction ranging from 13% to 50% of the PM<sub>10</sub>. These factors have a high degree of uncertainty and will vary depending on the material being handled and the processes used. Port activities included in the estimates include non-fugitive dust sources such as equipment operation and vehicle movements at Port. Appendix B of this report details analysis of PM<sub>2.5</sub> and PM<sub>10</sub> monitoring data at the Totara Street monitoring site with a view to better understanding if contributions from these more fugitive diffuse sources are occuring.

The analysis is complex and some confounding by combinations of sources is expected. However, results highlight sources of  $PM_{10}$  in the airshed from a range of wind directions with low  $PM_{2.5}$  to  $PM_{10}$  ratios as would be expected from such sources.



# Figure 2.1: Estimated contribution of sources to $PM_{10}$ and $PM_{2.5}$ emissions in the MMA (2022) (adapted from (Wilton, 2023)

#### 2.5.2 Monitoring data – PM<sub>2.5</sub> and PM<sub>10</sub> in the MMA

Monitoring of  $PM_{10}$  in the MMA is comprehensive with seven reference method samplers located across the airshed. Historically,  $PM_{10}$  and TSP have been correlated in the MMA with around 50-60% of the TSP being in the  $PM_{10}$  size fraction (Iremonger, 2023). Thus, sources of dust nuisance are also sources of  $PM_{10}$ . An analysis of  $PM_{2.5}$  and  $PM_{10}$  data in Appendix B suggests that these sources also likely contribute to  $PM_{2.5}$ .

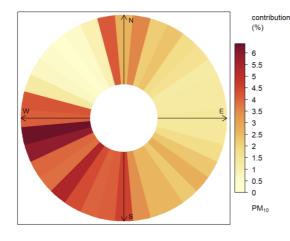
Exceedances of the NES for  $PM_{10}$  of 50  $\mu$ g/m<sup>3</sup> have occurred regularly since monitoring commenced. These have occurred across most monitoring sites with RYS regularly recording exceedances prior to the establishment of a wind fence in February 2020 (Iremonger, 2023).

Annual average concentrations at each site since 2019 are shown in Table 2.1. As the risk assessment uses 2019 as a base year, any downward trend in concentrations since then may indicate a lower health risk.

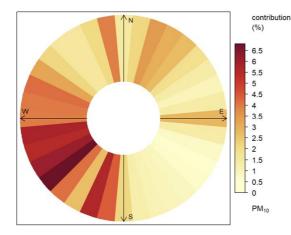
Year	Rata Street	Rail Yard South	Totara Street	Whareroa Marae	De Havilland Way	Sulphur Point	Bridge Marina
2019	20	31	25	17	20	14	16
2020	18	23	21	14	18	13	14
2021	19	24	21	11	19	13	15
2022	21	23	22	10	18	14	15

Table 2.1: Temporal variation in annual average PM<sub>10</sub> concentrations in the MMA (from Iremonger, 2023).

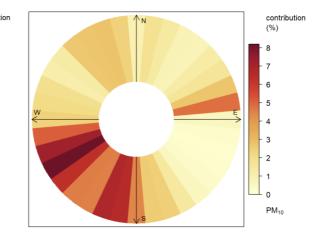
The relative contributions to annual average PM<sub>10</sub> weighted for wind direction prevalence for Whareroa Marae, Rata Street, Totara Street and RYS for 2019 are shown in Figure 2.2.



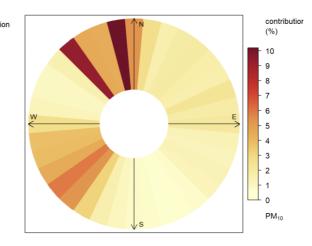
Rata Street - 2019 polar frequency plot



Totara Street - 2019 polar frequency plot



Rail Yard South - 2019 polar frequency plot



Whareroa Marae - 2019 polar frequency plot

#### Figure 2.2: Polar frequency plots for Rata Street, RYS, Totara Street and Whareroa Marae.

Marine aerosol was found to contribute to over half of the  $PM_{10}$  exceedances at the Rata Street monitoring site and these exceedances have been classified as exceptional events that do not constitute breaches (Iremonger, 2023). Figure 2.2 shows that the greatest contributions to annual 2019  $PM_{10}$  concentrations at Rata Street come from the southwest (SW) quadrant. The southwest to westerly direction likely represent emissions from the Port of Tauranga. Contributions from the south to SW sectors could include contributions from yard directly south of the Rata Street monitoring site but likely also includes contributions from the Port of Tauranga as indicated in Appendix A. Contributions from the north and west could include marine aerosol contributions.

Monitoring of PM<sub>2.5</sub> has been carried out at Totara Street since 2019. The 2019 annual average PM<sub>2.5</sub> concentrations was 8  $\mu$ g/m<sup>3</sup>, with subsequent averages for 2020, 2021 and 2022 being around 6  $\mu$ g/m<sup>3</sup>. Whilst daily PM<sub>2.5</sub> concentrations typically comply with the WHO 2021 guidelines, annual average PM<sub>2.5</sub> concentrations exceed the WHO 2021 annual guideline of 5  $\mu$ g/m<sup>3</sup>.

#### 2.5.3 Emission inventory assessment - NO<sub>2</sub> and SO<sub>2</sub> in the MMA

The 2022 air emission inventory for the MMA includes both NOx and SOx emissions but does not include benzene. Figure 2.2 shows shipping to the main source of NO<sub>x</sub> in the airshed. It is noted that dispersion from shipping is likely greater than from other sources in Figure 2.2 and thus the contributions at ground level may vary. A small proportion of the NOx will be emitted as NO<sub>2</sub> and conversion of NO to NO<sub>2</sub> will occur in the atmosphere. Thus, the inventory NOx estimates may not represent NO<sub>2</sub> contributions at ground level. However, Figure 2.2 illustrates that shipping NO<sub>2</sub> emissions are a source that should be considered when evaluating NO<sub>2</sub> exposures if monitoring data are not being used to estimate exposure (as in the HAPINZ 3 model).

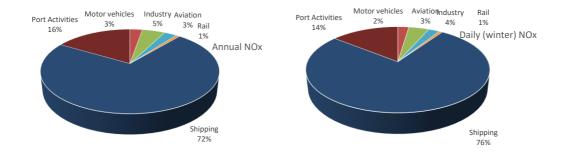
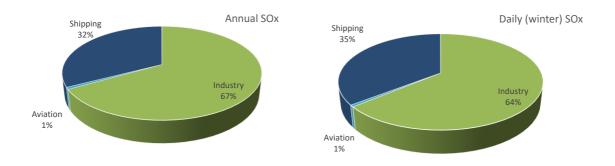


Figure 2.3: Estimated contribution of sources to NO<sub>2</sub> emissions in the MMA (2022) (from Wilton, 2023)



#### Figure 2.4: Estimated contribution of sources to SO<sub>2</sub> emissions in the MMA (2022) (from Wilton, 2023)

Industry is the main source of SO<sub>2</sub> in the airshed with two key industrial sources (Ballance Agri-Nutrients Limited and Lawter NZ Ltd) each contributing around half of the industrial SO<sub>2</sub> emissions.

#### 2.5.1 Evaluation of monitoring data - NO<sub>2</sub> and SO<sub>2</sub> in the MMA

The BOPRC 2023 Air Quality Monitoring Report (Iremonger, 2023) provides an evaluation of SO<sub>2</sub> concentrations which have been measured at six sites (Totara Street, RYS, Sulphur Point, Bridge Marina and Whareroa Marae) in the MMA.

The highest hourly average SO<sub>2</sub> concentrations measured in the MMA was 751  $\mu$ g/m<sup>3</sup> and was recorded at Whareroa Marae in 2016. This exceeds the National Environmental Standard (NES) upper limit SO<sub>2</sub> concentrations which allows for no exceedances. In 2019 the same hourly NES was exceeded at Rata Street. Both of these episodes constitute a breach of the NES for SO<sub>2</sub> in the MMA. Historically, the hourly average SO<sub>2</sub> concentrations have also exceeded the short term (hourly) average NES limit value of 350  $\mu$ g/m<sup>3</sup> (nine allowable exceedances per year) at Totara. This did not comprise a breach of the standard. Sources of these peak SO<sub>2</sub> concentrations likely include industry and shipping.

The impact of MARPOL regulations (Annex V1 limiting the sulphur content of fuels or technological equivalents) for shipping from 1 January 2020 combined with a reduction in cruise shipping visiting Port has resulted in significant improvements in annual average SO<sub>2</sub> concentrations in the MMA (Iremonger, 2023). Annual average SO<sub>2</sub> concentrations have decreased at Rata Street by around 15  $\mu$ g/m<sup>3</sup>, at RYS by around 19  $\mu$ g/m<sup>3</sup>, at Totara by around 9-11  $\mu$ g/m<sup>3</sup>. Reductions in SO<sub>2</sub> are not as significant at Whareroa Marae, Sulphur Point and Bridge Marina (at around 5  $\mu$ g/m<sup>3</sup> or less).

Exceedances of the WHO 2021 daily guideline for SO<sub>2</sub> of 40  $\mu$ g/m<sup>3</sup> in the MMA summarised for 2019 to 2022 in Appendix A of the ESR report show that this daily guideline level is exceeded in the MMA after the 1 January 2020 at Bridge Marina and Whareroa Marae. Similarly, one breach of the WHO 2021 10 -minute guideline value has occurred at Bridge Marina since 2019. It is noted that the value recorded was 1247  $\mu$ g/m<sup>3</sup> and compares with a guideline of 500  $\mu$ g/m<sup>3</sup> (WHO,2021). Based on wind direction analysis, the ESR report indicates industry is the likely source of the elevated daily and 10-minute SO<sub>2</sub> concentrations in these locations.

Monitoring of NO<sub>2</sub> commenced at the Whareroa Marae site in August 2023. Waka Kotahi have carried out NO<sub>2</sub> monitoring at roadside sites in the MMA, as referred to in the ESR report, as motor vehicles are typically a major source of NO<sub>2</sub>. These show annual average concentrations often around 20  $\mu$ g/m<sup>3</sup> but are likely impacted on by predominantly by motor vehicles. In the MMA shipping is likely a significant contributor to NO<sub>2</sub> emissions.

Comparison of the Tauranga Air Emission Inventory and a more recent post MARPOL inventory for the MMA shows emission rates are similar for NO<sub>x</sub> and SO<sub>x</sub> pre MARPOL (from shipping). However, the proportion of NO<sub>2</sub> in the NO<sub>x</sub> will differ to the SO<sub>2</sub> in the SO<sub>x</sub> and will also be influenced by other variables (for example NO<sub>2</sub> is emitted directly and through atmospheric reactions of NO with O<sub>3</sub>. Krause et al., (2021) evaluated NO<sub>x</sub>, NO<sub>2</sub> and SO<sub>2</sub> from over 7204 ship passages in 2018 and found NO<sub>2</sub> emission rates (estimated from downwind concentrations) to be slightly higher than the SO<sub>2</sub> rate but the applicability to the MMA is uncertain. Whilst there are uncertainties in the evaluation it does highlight the potential for underestimation of NO<sub>2</sub> in the MMA.

For example, if NO<sub>2</sub> and SO<sub>2</sub> from shipping were similar, annual average concentrations of a minimum<sup>3</sup> of 10  $\mu$ g/m<sup>3</sup> (Totara St), 15  $\mu$ g/m<sup>3</sup> (RYS) and 19  $\mu$ g/m<sup>3</sup> (Rata Street) from shipping could be expected based on the reduction in SO<sub>2</sub> concentrations attributed to MARPOL Annex VI. These compare with WHO (2021) guideline values for NO<sub>2</sub> of 10  $\mu$ g/m<sup>3</sup>.

 $<sup>^3</sup>$  The estimate is based on the improvement in  $SO_2$  concentrations since MARPOL and will not account for non-improved component

## **3 PARTICULATE AND HEALTH IMPACTS**

This section considers the health impacts with a focus on potential implications of the differences in the character of the Mount Maunganui airshed. The main character issue evaluated is the greater proportion of  $PM_{10}$  that is coarse in the Mount Maunganui airshed. The issue arises because the concentration response function (CRF) for  $PM_{10}$  used in the ESR report to estimate health impacts in Mount Maunganui is based on epidemiological studies carried out throughout New Zealand, mostly in urban areas that are different in character to the MMA because they have a higher proportion of  $PM_{2.5}$  and a lower proportion of coarse particulate matter.

The average ratio of  $PM_{2.5}$  to  $PM_{10}$  from the HAPINZ 3 study<sup>4</sup> and cited in the ESR report is 0.53 and compares with a ratio of 0.32 at the Totara Street monitoring site in the MMA. If the CRF for premature mortality and other health endpoints derived in New Zealand is dominated by the  $PM_{2.5}$  size fraction, use of the CRFs to estimate impacts in the MMA might overestimate impacts if the coarse size fraction does not contribute to premature mortality and other health endpoints to the same extent.

This review seeks to determine:

- The health impacts that can be expected as a result of exposure to particulate in Mount Maunganui.
- Whether the coarse particulate size fraction causes adverse health impacts and if so which ones.
- If the size of the health impacts caused by coarse particulate are likely the same as PM<sub>2.5</sub>.

The process used in this assessment was reliance on significant bodies of work by credible agencies e.g., World Health Organisation (WHO) including those with specific evaluations e.g., Health Canada have a detailed causality assessment. This was supplemented by a summary of outcomes of more recent studies of significance that might further understanding.

### 3.1 Health impacts of fine particulate (PM<sub>2.5</sub>)

Health impacts of particulate exposure in Mount Maunganui include exposure to concentrations in the  $PM_{2.5}$  size fraction. Both long-term and short-term exposures to  $PM_{2.5}$  are considered causal for numerous health endpoints (Table 3.1) including all-cause<sup>5</sup> mortality and cause specific mortality.

Health endpoint	Exposure duration	Health Canada -causality determination	USEPA (2019) - causality determination
All cause mortality	Long term	Causal	Causal
All cause mortality	Short term	Causal	Causal
Cardiovascular mortality	Long term	Causal	
Cardiovascular mortality	Short term	Causal	
Cardiovascular morbidity	Long term	Likely to be causal	Causal
Cardiovascular morbidity	Short term	Causal	Causal
Respiratory mortality	Short term	Causal	
Respiratory mortality	Long term	Suggestive of but not	
		sufficient to infer a causal	
		relationship	
Respiratory morbidity	Short term	Causal	Likely to be causal
Respiratory morbidity	Long term	Likely causal	Likely to be causal

#### Table 3.1: Summary of Health Canada (2016a) and USEPA (2019) causality assessment for PM2.5

<sup>5</sup> All cause mortality counts the total number of deaths due to any cause within a specified year, whereas cause-specific mortality statistics count the number of deaths due to a particular cause in a specified year.

<sup>&</sup>lt;sup>4</sup> This is not a population or exposure adjusted ratio

Cancer morbidity and mortality	Long term	Likely causal	Likely to be causal
Metabolic effects	Short term		Suggestive of, but not sufficient to infer
Metabolic effects	Long term		Suggestive of, but not sufficient to infer
Reproductive effects	(duration of pregnancy)	Inadequate to infer a causal relationship	Male and female reproduction and fertility - Suggestive of, but not sufficient to infer
Developmental effects	Range of exposures	Suggestive of, but not sufficient to infer, a causal relationship	Birth outcomes - Inadequate to infer a causal relationship
Neurological effects	Short and long term	Suggestive of, but not sufficient to infer, a causal relationship	Short term - Suggestive of, but not sufficient to infer Long term - Likely to be causal

Morbidity impacts of PM<sub>2.5</sub> exposure include reduced lung function in both adults and in children. In children, increased respiratory allergy symptoms, bronchitis symptoms and, to some extent, wheezing symptoms are associated with exposure to PM<sub>2.5</sub>. An increased risk of asthma diagnosis, as well as asthma exacerbation-related hospital visits (including hospital admissions and emergency room visits), were observed with long-term exposure to PM<sub>2.5</sub> in both adults and children but especially in children (Health Canada (2016a)).

Cardiovascular related morbidity impacts associated with long term PM<sub>2.5</sub> exposure include increased risk of hospital admissions or development of a number of cardiovascular conditions including ischemic heart disease, congestive heart failure, cerebrovascular disease, myocardial infarction, hypertension, and peripheral vascular disease (Health Canada, 2016a).

In terms of reproductive and developmental effects, PM<sub>2.5</sub> exposure during the full length of pregnancy has been associated with increased risk of adverse birth outcomes in infants (reduced birth weight, increased risk of low birth weight, small for gestational age, preterm birth) and in one study exposure in has been associated with post neonatal infant mortality (Health Canada 2016a).

Associations between long-term exposure to PM<sub>2.5</sub> and neurological effects include associations with neurodegenerative diseases (i.e., Parkinson's disease, Alzheimer's disease and dementia), autism, cognitive functions (e.g., cognitive impairment, visual-motor skills, verbal learning), and morphological changes in the brain (i.e., smaller white and grey matter and total cerebral brain volume). One study also reports an association between short term PM<sub>2.5</sub> exposure and Alzheimer's disease (Health Canada, 2016a).

## 3.2 Health impacts of coarse (PM<sub>10</sub>-PM<sub>2.5</sub>) particulate

There is significant health evidence and research relating to the  $PM_{10}$  size fraction which includes the  $PM_{2.5}$  component, identified above as being causal in many health endpoints.

The WHO guidelines, whilst including guidelines for  $PM_{10}$  do not separately address health impacts of the coarse size fraction ( $PM_{10}$ - $PM_{2.5}$ ). They do consider health impacts of sand and desert storm dust (SDS) which is coarse-mode particulate that will include a substantive  $PM_{10}$ - $PM_{2.5}$  component.

#### 3.2.1 Short term exposures (acute effects) and mortality

In 2016, the relationship between exposure to sources of coarse-mode particulate and premature mortality impacts was examined in a review of 26 studies using time-series epidemiology and case- crossover design<sup>6</sup> by Health Canada (2016). The health endpoints included:

- Daily mortality (18 studies, positive associations in all, statistical significance in seven,
- Respiratory mortality (examined in most studies)
- Cardiovascular mortality (examined in most studies)
- Cerebrovascular and cancer mortality (examined in a small number)
- More specific outcomes (myocardial infarction (MI), stroke and intracerebral haemorrhage)

Health Canada (2016) conclude that there are "fairly consistent positive associations between short-term exposure to ambient coarse PM and non-accidental, respiratory and cardiovascular mortality in the available epidemiological studies". They also note that the associations observed are sometimes not statistically significant. The 2016 Health Canada conclusion was that "Overall, the association between coarse particles and mortality is clearly not as strong as the association between fine particles and mortality. Uncertainty remains due to the different methods utilized to estimate coarse PM concentrations across studies and the potential for confounding by the fine PM fraction and gaseous co-pollutants".

Research on the health impacts of coarse particulate has increased since the 2016 evaluation. A further 2020 study (Tian et al., 2020) conducted a time-series analysis to explore the effects of  $PM_{2.5}$ , coarse particulate, and  $PM_{10}$  on mortality from ischemic heart disease (IHD) and chronic obstructive pulmonary disease (COPD) in 96 Chinese cities during 2013–2016. The study found significant effects of coarse particulate on mortality from IHD and COPD, but the magnitudes of effects were weaker than those of  $PM_{2.5}$ . The results were robust when adjusting for co-pollutants and altering model parameters. They concluded that the role of coarse particulate in triggering in cardiopulmonary mortality was not negligible.

A recent study (Liu et al., 2022) evaluated daily mortality (total, cardiovascular, and respiratory) and PM<sub>10</sub>-PM<sub>2.5</sub> data from 205 cities in 20 countries/regions. "Two-pollutant models were used to test the independent effect of  $PM_{10}$  -PM<sub>2.5</sub> from co-pollutants (fine PM, nitrogen dioxide, sulfur dioxide, ozone, and carbon monoxide). A 10 µg/m3 increase in coarse particulate concentrations (lag 1 day) were associated with increments of 0.51% (95% confidence interval [CI], 0.18%-0.84%), 0.43% (95% CI, 0.15%-0.71%), and 0.41% (95% CI, 0.06%-0.77%) in total, cardiovascular, and respiratory mortality, respectively. These associations were robust to adjustment by all co-pollutants in two-pollutant models, especially for  $PM_{2.5}$ . The exposure-response curves for total, cardiovascular, and respiratory mortality were positive, with steeper slopes at lower exposure ranges and without discernible thresholds."

In 2022 Jonathan Samet, a well-known expert in health impacts of particulate pollution, reviewed literature on health impacts of the coarse-mode (Samet, 2022). He concluded that the study by Liu et al., 2022 "adds robust epidemiological evidence on coarse particulate and daily mortality, but—by itself—it does not shift the weight of evidence towards causation of adverse effects by coarse mass  $PM_{10}$ ". Samet (2022) goes on to note that "certainty as to the causation of adverse health effects by coarse mass PM would be bolstered by advancing understanding of toxicity to complement the epidemiological findings". Samet (2022) notes that the prevalence of coarse particulate may be amplified by desertification brought on by drought from climate change.

Fussell & Kelly, (2021) reviewed 52 experimental studies looking at mechanisms and intermediate endpoints underlying epidemiological evidence of an impact of desert dust on cardiovascular and respiratory health. They concluded the experimental research of desert dust on respiratory endpoints "go some way in clarifying the mechanistic effects of atmospheric desert dust on the upper and lower human respiratory system" and "in doing

<sup>&</sup>lt;sup>6</sup> case-crossover designs compare individuals to themselves at different times. This parallels the randomized crossover trial approach that compares individuals to themselves as they are going on and off treatment <a href="https://www.publichealth.columbia.edu/research/population-health-methods/case-crossover-study-design">https://www.publichealth.columbia.edu/research/population-health-methods/case-crossover-study-design</a>.

so, they provide support for biological plausibility of epidemiological associations between this particulate air pollutant and events including exacerbation of asthma, hospitalization for respiratory infections and seasonal allergic rhinitis."

Health Canada's (2016) conclusion on the causality of the relationship is "the epidemiology data are **suggestive** of a causal relationship between short-term exposure to the coarse PM fraction and mortality."

USEPA (2019) undertook causality assessments for PM<sub>2.5</sub>, PM<sub>10</sub>-PM<sub>2.5</sub> and ultrafine particles. They note several variables that contribute to poorer information on health impacts of the coarse particulate fraction including greater spatial variability (making it more problematic to characterise exposure and consequently determine associations) and greater measurement uncertainties<sup>7</sup>. For short term mortality impacts of PM<sub>10</sub>-PM<sub>2.5</sub> they conclude that the association is *suggestive of but not sufficient to infer*.

The WHO 2021Report includes "*PM (all size fractions)*" in the causality assessment for PM<sub>2.5</sub> and PM<sub>10</sub> (Table 2.1 WHO, 2021) short term mortality impacts.

#### 3.2.1 Short term exposures (acute effects) and morbidity impacts

The main health endpoint for which there appears consistent evidence for increases in coarse particulate exposure is hospital admissions and emergency room visits for respiratory conditions especially in children with asthma. In particular, both epidemiological studies and panel studies show significant impacts for asthma in children, with panel studies demonstrating increases in respiratory symptoms and medication use, and epidemiological studies showing significant associations with increased hospitalisations for asthma. The populations most susceptible to suffering the short-term effects of suspended particulates are considered to be older persons, individuals with chronic cardiopulmonary disorders, and children (Goudie, 2014).

There is plausibility around the health endpoints from the viewpoint of biological mechanisms and deposition locations as the coarse particles are associated with upper respiratory conditions (and will deposit higher owing to size) whereas the fine particles are associated with lower respiratory conditions.

A toxicological review of 67 experimental studies commissioned by WHO concluded that sand and dessert storm dust (coarse-mode) may be a significant risk factor for inflammatory and allergic lung diseases such as child and adult asthma (World Health Organization, 2021). The studies demonstrated that sand dust particles collected from surface soils and dust-storm particles sampled at remote locations away from the source (and as such, mixed with industrial pollutants and microorganisms) induce inflammatory lung injury and aggravate allergen-induced tissue eosinophilia (World Health Organization, 2021).

The toxicological review by Fussell & Kelly, (2021) concludes that "In vitro findings suggest that the significant amounts of suspended desert dust during storm periods may provide a platform to intermix with chemicals on its surfaces, thereby increasing the bioreactivity of  $PM_{2.5}$  during dust storm episodes, and that mineral dust surface reactions are an unrecognized source of toxic organic chemicals in the atmosphere, enhancing toxicity of aerosols in urban environments."

Health Canada concludes that "the epidemiology data and the limited results from controlled human exposure and toxicological studies are **suggestive of a causal relationship** between short-term exposure to the coarse PM fraction and respiratory effects."

For short term exposures morbidity impacts of PM<sub>10</sub>-PM<sub>2.5</sub> USEPA, (2019) conclude:

- Respiratory effects Suggestive of, but not sufficient to infer.
- Cardiovascular effects Suggestive of, but not sufficient to infer.
- Metabolic effects inadequate.

 $<sup>^{7}</sup>$  The relationship between the cut point at 10 microns for PM<sub>10</sub> and the particulate distribution frequency increases measurement uncertainty.

- Nervous system effects inadequate.
- Male and female reproduction and fertility (not presented by exposure duration) inadequate.
- Birth outcomes (not presented by exposure duration) inadequate.
- Cancer (not presented by exposure duration) Suggestive of, but not sufficient to infer.

#### 3.2.2 Long term exposures and mortality impacts

There are a few cohort studies that have evaluated long term exposures and mortality impacts. In 2016 Health Canada evaluated these and concluded that "the results do not provide significant insight into the role, if any, played by the coarse PM fraction." They concluded that the epidemiological data are "*inadequate to infer a causal relationship* between chronic exposure to the coarse PM fraction and mortality".

The USEPA (2019) conclude that the association between long term exposures to PM<sub>10</sub>-PM<sub>2.5</sub> and premature mortality is *suggestive of, but not sufficient to infer* causality. This represents a change from "inadequate" in the previous USEPA, (2009) evaluation.

The WHO (2021) include "*PM (all size fractions*)" in the causality assessment for  $PM_{2.5}$  and  $PM_{10}$  (Table 2.1 WHO, 2021, see Appendix C) for long term mortality impacts.

More recently, a large cohort study of cardiovascular mortality and long-term particulate exposure was carried out in China (Zhang et al., 2023). The study included 580,757 participants who were examined from 2009–2015 and followed up through 2020. For cardiovascular mortality, the hazard ratios and 95% confidence interval for each 1  $\mu$ g/m<sup>3</sup> increase in the annual average concentration of PM<sub>2.5</sub>, PM<sub>10</sub>, and coarse particulate were 1.033 (1.028–1.037), 1.028 (1.024–1.032), and 1.022 (1.012–1.033), respectively. All three size fractions were linked to a higher mortality risk for myocardial infarction and ischemic heart disease (IHD). The mortality risk of chronic IHD and hypertension was linked to PM<sub>2.5</sub> and PM<sub>10</sub>. Significant association between coarse particulate and other heart disease mortality was also observed. They did not observe a statistically significant association of coarse particulate with hypertension and chronic ischemic heart disease mortality. A significant association between mortality from other forms of heart disease (IRE: 1.061, 95% CI: 1.012–1.114) and coarse particulate was observed which was not significant for PM<sub>2.5</sub> and PM<sub>10</sub>.

#### 3.2.3 Long term exposures and morbidity impacts

There have been relatively few studies that examined the respiratory and cardiovascular effects of long-term exposure to air pollutants. In their 2016 review, Health Canada concluded that epidemiological data are *inadequate to infer a causal relationship* between chronic exposure to the coarse PM fraction and respiratory and cardiovascular health effects, as well as with the incidence of developmental outcomes.

For long term exposures and morbidity impacts of PM<sub>10</sub>-PM<sub>2.5</sub> USEPA, (2019 - Table 1.4) conclude:

- Respiratory effects inadequate.
- Cardiovascular effects suggestive of, but not sufficient to infer.
- Metabolic effects suggestive of, but not sufficient to infer.
- Nervous system effects suggestive of, but not sufficient to infer.
- Male and female reproduction and fertility (not presented by exposure duration) inadequate.
- Birth outcomes (not presented by exposure duration) inadequate.
- Cancer (not presented by exposure duration) Suggestive of, but not sufficient to infer.

The pool of studies has not grown significantly since 2016. A number of experimental studies have been carried out. These include Davis et al., (2020) who examined a range of inflammatory and haemostatic markers in a

cohort of midlife women and found significant increases in markers that remained unchanged with adjustment for PM<sub>2.5</sub>, ozone, nitrogen dioxide, and carbon monoxide. They concluded long-term coarse particulate exposure may be associated with changes in coagulation independently from PM<sub>2.5</sub> and thus, contribute to cardiovascular disease risk in midlife women.

Shin et al., (2020) examined the associations between exposure to particulate matter and changes in fasting glucose and lipid profiles. They concluded "fine particulate matter exposure affects worsening fasting glucose and low-density lipoprotein cholesterol levels, with no evidence of an association for coarse particulate matter".

### 3.3 Comparison of CRFs for long-term and short-term exposures

Short-term exposure to particulate matter (PM<sub>10</sub> and PM<sub>2.5</sub>), nitrogen dioxide (NO<sub>2</sub>), and ozone (O<sub>3</sub>) and allcause and cause-specific mortality were reviewed by Orellano et al., (2020) in a systematic review and metaanalysis for the WHO 2021 guideline review. Similarly, the long-term exposure to PM and all-cause and causespecific mortality were reviewed by Chen & Hoek, (2020) in a systematic review and meta-analysis for the WHO 2021 guideline review. The pooled effect estimates (relative risks RR<sup>8</sup>) reported in these studies are shown in Table 3.2.

Premature mortality - Long term Chen & Hoek, (2020)	PM <sub>2.5</sub> RR 10 µg/m <sup>3</sup> increase	PM₁₀ RR 10 µg/m³ increase
All cause	1.08	1.04
Circulatory	1.11	1.04
ICD	1.16	1.06
Stroke	1.11	1.01
Respiratory	1.10	1.12
COPD	1.11	1.19
Lung cancer	1.12	1.08
Short term premature mortality - Orellano et al., (2020)	PM <sub>2.5</sub> RR	PM₁₀ RR
All cause	1.0065	1.0041
Cardiovascular	1.0092	1.006
Respiratory	1.0073	1.0091
Cerebrovascular mortality	1.0072	1.0044
	NO <sub>2</sub> (24-hour average)	NO <sub>2</sub> (hourly average)
All-cause mortality	1.0072	1.0024

#### Table 3.2: Comparison of CRFs for PM<sub>2.5</sub> and PM<sub>10</sub>

\*bold indicates a significant effect across the meta analysis (p<0.05)

Chen & Hoek, (2020) also analysed the shape of the CRF to investigate the potential for higher effects at lower concentrations (a nonlinear relationship). They found an all-cause premature mortality combined effect estimate of 1.17 for the five studies with average exposures less than 10  $\mu$ g/m<sup>3</sup>. This compares with the overall combined RR of 1.08 supporting the possibility of a higher relative risk at lower concentrations. This point is noted because in New Zealand annual average PM<sub>2.5</sub> concentrations are typically less than 10  $\mu$ g/m<sup>3</sup> and thus higher CRFs might be expected (as illustrated in Hales 2021).

Table 3.2 shows that the magnitude of the associations for short term exposures are a factor of ten lower than the associations between mortality and the exposure to these same air pollutants in the long-term. Orellano et

<sup>&</sup>lt;sup>8</sup> In epidemiology the relative risk is the ratio of the probability of an event occurring with an exposure versus the probability of the event occurring without the exposure. A relative risk of 1.08 per 10 μg/m<sup>3</sup> increase is expressed as a CRF of 0.08.

al., (2020) with respect to the magnitude of these relationships note that "*in epidemiology small risks applied to large populations are likely to represent a major health problem.*"

An additional observation from Table 3.3 is that the relative risks for  $PM_{10}$  are typically lower than for  $PM_{2.5}$ . Exceptions are for respiratory and chronic obstructive pulmonary disease (COPD) which are higher for  $PM_{10}$ . The lower relative risk for  $PM_{10}$  might imply that size of impact from the coarse-mode particulate is not as great as for  $PM_{2.5}$ . There may be other variables influencing the differences in effect size, however, as the base studies for each size fraction differ.

#### 3.3.1 Conclusions

The most consistent evidence of coarse-mode impacts is with premature mortality from short term exposures and hospital admissions and emergency room visits for respiratory conditions especially in children with asthma.

The populations most susceptible to suffering the short-term effects of suspended particulates are considered to be older persons, individuals with chronic cardiopulmonary disorders, and children (Goudie, 2014).

Causality assessments for coarse particles include Health Canada (2016) and USEPA (2019) both of which conclude that studies were *suggestive of a causal relationship* (*USEPA wording "suggestive but insufficient to infer"*) between coarse particles and acute premature mortality and morbidity endpoints. USEPA (2019) also conclude that studies are suggestive but insufficient to infer a causal relationship between long term exposures and mortality and long-term exposures and cardiovascular, metabolic and nervous system effects. WHO (2021) do not exclude coarse particulate from the causality assessment, and refer to PM (all size fractions) as being causal for premature mortality health endpoints for short and long term exposures. Thus, the approach of WHO is to integrate the coarse size fraction into the PM<sub>10</sub> assessment for which they determine causality and have set guidelines based on minimum observed health impacts.

With respect to desert dust the WHO concludes that studies indicate an overall effect of desert dust on cardiovascular mortality and respiratory morbidity but note that the evidence is still inconsistent when accounting for sources of PM in different geographical areas. They also note indications from experimental studies that coarse-mode particles may increase the toxicity of PM<sub>2.5</sub>.

There is less certainty around the contribution of the coarse particulate to long-term premature mortality and morbidity, the main health endpoints used in the ESR report, than there is for  $PM_{2.5}$ . Because the coarse particulate fraction is lower in the MMA than average (0.32 versus 0.53) there is the potential that the  $PM_{10}$  CRFs used in the risk assessment might overestimate these impacts. This will depend on whether the coarse fraction is causing health impacts and if so whether the size of the impact is as great as it is for  $PM_{2.5}$ .

There are uncertainties around these issues. The causality assessments suggest that the coarse size fraction may be causal in short term mortality and morbidity effects and potentially also for long term mortality.

The size of the effects for  $PM_{10}$  are generally smaller than for  $PM_{2.5}$  suggesting that the impact of coarse-mode particulate is less than for  $PM_{2.5}$ . This is also demonstrated in Zhang et al., (2023) with relative risks of cardiovascular mortality for  $PM_{2.5}$ ,  $PM_{10}$ , and coarse particulate of 1.033, 1.028 and 1.022 respectively.

It is noted that the increased coarse particulate may result in greater prevalence of upper respiratory impacts such as asthma in the MMA and that the risks to children of these exposures may be greater.

## 4 REVIEW OF AIR POLLUTION: HEALTH RISK ASSESSMENT MOUNT MAUNGANUI

Risk assessment methodology is a desktop exercise to provide statistical estimates of the scale of health impacts of exposure to air contaminants. The basis for the method is relationships between contaminant concentrations and health endpoints established using epidemiological studies. The risk assessment methodology utilises a relative risk (RR) for each contaminant established based on epidemiological studies, baseline health data and contaminant concentration data (as a proxy for exposure).

The ESR report implements a quantitative risk assessment of exposure to  $PM_{2.5}$  and  $NO_2$  and to  $PM_{10}$ . A qualitative assessment of other contaminants including sulphur dioxide (SO<sub>2</sub>), benzene and hydrogen sulphide (H<sub>2</sub>S) is also made.

The risk assessment in the ESR report follows the methodology of HAPINZ 3, which has been extensively reviewed with changes to:

- Baseline mortality statistics (updated for 2019).
- Exposure data estimates for each CAU.

The main components of HAPINZ 3 that have been used for this study therefore are the health endpoints and associated CRFs, the cost data and assumptions and the use of the spatial distribution functionality (assessment of data at the CAU level).

Additionally, a comparison is made to a neighbouring residential area to highlight the health risks associated with exposures to air contaminants in the Mount Maunganui area.

This review focuses on the accuracy and reliability of the health impacts evaluation in the ESR report and the extent to which it can be relied on in decision making.

## 4.1 Concentration response function (CRF)

The ESR report acknowledges the difference in the composition of air pollutants in the MMA relative to urban areas of New Zealand and notes that the selected CRFs may not accurately estimate the effects of air pollution in and around the Mount Maunganui areas. To better understand whether this aspect is likely to undermine the conclusions of the risk assessment we have undertaken a more critical review of the applicability of the CRFs and associated interpretation issues for health risks in Mount Maunganui.

The CRFs in the HAPINZ 3 model and used in the ESR report are from a two-pollutant model (for PM<sub>2.5</sub> and NO<sub>2</sub>) reported in Hales (2021) and a single pollutant model for PM<sub>10</sub> used in HAPINZ 3:

PM<sub>2.5</sub>

- Premature mortality 1.105 (10.5% increase in baseline mortality (adults 30+) per 10 μg/m<sup>3</sup> increase in PM<sub>2.5</sub>).
- Cardiovascular hospitalisations all ages 1.115
- Respiratory hospitalisations all ages 1.07

 $NO_2$ 

- Premature mortality 1.105 (10.5% increase in baseline mortality (adults 30+) per 10 μg/m<sup>3</sup> increase in PM<sub>2.5</sub>)
- Cardiovascular hospitalisations (adults 30+ years) 1.047
- Respiratory hospitalisations 1.13

- Asthma wheeze hospitalisations (0-18 years) 1.182
- Asthma prevalence (0-18 years) 1.050

The CRF for PM<sub>10</sub> used in HAPINZ 3 based on a pollutant model – PM<sub>10</sub>

 Premature mortality – 1.111 (11.1% increase in baseline mortality (adults 30+) per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>)

In the MMA airshed there is a large difference between the annual average  $PM_{10}$  and  $PM_{2.5}$  concentrations used in the health risk assessment (20 µg/m<sup>3</sup> and around 6 µg/m<sup>3</sup> respectively) and there is minimal difference in the CRFs for  $PM_{10}$  and  $PM_{2.5}$  used (RR 1.111 vs 1.105). As a result, the ESR report estimates for premature allcause mortality (30 years + adults) are 26 and 9 respectively.

The models used to derive the CRFs have impact on the interpretation of the results, however. In both cases, single or two pollutant models the effects demonstrated are not limited to the pollutant or pollutants specified, with effects just best accounted for the contaminant specified. Two pollutant models for PM<sub>2.5</sub> and NO<sub>2</sub> have been found to give much lower RR for PM<sub>2.5</sub> than single pollutant models. For example, Chen & Hoek, (2020) found a RR for PM<sub>2.5</sub> of 1.02 for two pollutant models versus 1.07 in the single pollutant model. Thus, the single pollutant model outputs for PM<sub>10</sub> should not be directly compared with the two pollutant model for PM<sub>2.5</sub> to infer coarse-mode impacts (i.e., it is not appropriate to estimate 17 premature deaths from PM<sub>10</sub>-PM<sub>2.5</sub> as might be implied in the previous paragraph). Chen & Hoek, (2020) further note that two pollutant models can be difficult to interpret when the correlation between pollutants is high or exposure for pollutants is assessed with different methods or at a different spatial resolution. We note in the case of the Hales (2021) two pollutant model that the exposure to PM<sub>2.5</sub> and NO<sub>2</sub> is assessed with different methods (PM<sub>2.5</sub> monitored and NO<sub>2</sub> modelled) and at different spatial resolutions and thus falls into this "difficult to interpret" category.

In any model, the effects are just best accounted for by using the concentrations of the pollutants described (in the CRF e.g.,  $PM_{10}$ ,  $PM_{2.5}$  or  $NO_2$ ) as a proxy for all co-emitted pollutants. Interpretation is therefore also nuanced by the contaminant mix making comparisons between risk assessment outputs for specific contaminants and risk fractions even more problematic.

The PM<sub>10</sub> CRF from the single pollutant model is based on epidemiology from New Zealand including urban areas which typically have higher PM<sub>2.5</sub> to PM<sub>10</sub> ratios than the MMA. If the PM<sub>2.5</sub> component has a greater contribution in PM<sub>10</sub> premature mortality impacts this may mean impacts of the PM<sub>10</sub> size fraction in the MMA are overestimated. The average PM<sub>2.5</sub> to PM<sub>10</sub> ratio from the HAPINZ study is around 0.53 and the PM<sub>2.5</sub> to PM<sub>10</sub> ratio at Totara Street is around 0.32. Conversely, the contaminant mix in the MMA may contribute to additional impacts owing to higher exposures of pollutants that do not form a substantive part of the urban exposures underpinning the CRFs.

The morbidity estimates in the ESR report are based on  $PM_{2.5}$  concentrations as the HAPINZ model includes no  $PM_{10}$  CRFs for morbidity, noting that in the NZ study the  $PM_{10}$  associations with morbidity were weaker than for  $PM_{2.5}$  and NO<sub>2</sub>. The morbidity estimates for restricted activity days are based on a single 1987 study. This appears to be the only study of its type in the literature and is dated and difficult to validate in the absence of supporting studies. Additionally, we note that it is based on  $PM_{2.5}$  concentrations. The health literature indicates acute impacts (respiratory including asthma) of exposure to coarse particulate and given the larger coarse particulate component in the Mount Maunganui a  $PM_{2.5}$  metric is likely to result in an underestimate of impact.

It is our view that:

- The CRFs used are based on robust studies and represent the best available information for New Zealand.
- There are differences in the urban environments used to derive the CRFs and the MMA. Some of these may serve to increase impacts in the MMA (e.g., more complex pollutant mix) and others to decrease impacts (e.g., higher proportion of PM<sub>10</sub> in the coarse (PM<sub>10</sub>-PM<sub>2.5</sub>) size fraction). The impact of these differences is not presently quantifiable.

- There are complexities with single and two pollutant models in attributing impacts to contaminants.
- Any resulting risk assessment will provide an indicative of scale rather than exact to the number output.
- Estimates of restricted activity days should be treated with a high degree of uncertainty.

## 4.2 Baseline health data

The risk assessment model notes that the baseline health data used for HAPINZ 3 (which is referred to as the basis for the method) was updated using a 2018-2019 baseline dataset that utilises incidence rates from a broader range of years. The approach to baseline data for the 2018-2019 dataset is detailed in Kuschel & Metcalfe, (2023). The two-year average particularly at the CAU level and for cardiovascular and respiratory hospitalisations (all ages) will be slightly more susceptible to temporal variability as it relies on baseline data for only two years.

Whilst the prevalence of disease (relative to the population used to derive the CRF) is accounted for in the baseline health statistics, the application of a CRF based on a large population dataset to a small population is not ideal in that it requires the assumption of the same distribution in health impacts within the smaller baseline health data as in the larger population baseline health data. The baseline mortality (adults 30+ years) statistic used for the Mount Maunganui risk assessment area in the HAPINZ 3 model is 142 premature deaths per year (0.88% of population) and compares with 152 premature deaths per year for Ōtūmoetai (also 0.88% of population).

It is our view that:

- The approach to baseline health data used as detailed in Kuschel & Metcalfe, (2023) is appropriate.
- Some variations in actual versus estimated impacts may occur as a result of the application of the risk assessment to a relatively small population area.

## 4.3 Misclassification of exposure

In risk assessments and in epidemiology, concentrations measured at monitoring sites are used a proxy for exposure to a contaminant. To do this, data from monitoring sites are typically extrapolated to be representative of exposures over many tens of kilometres.

The approach of use of monitoring data to represent exposure is used in both the underlying epidemiology (where the CRFs come from) and in the subsequent risk assessments (calculations of the burden of disease). If the concentration data were not correlated with exposure, significant associations between health endpoints and concentrations would not be observed in the epidemiology. If the concentration data overestimates exposure (as could potentially be expected in some locations in New Zealand where monitoring is limited to worst case areas) then the relative risks (concentrations response relationships) will be lower per  $\mu$ g/m<sup>3</sup> of contaminant. Thus, subsequent estimates of impact (via risk assessment using those concentration response relationships) will be balanced out.

#### 4.3.1 Spatial representation

The potential for spatial representation misclassification was considered and is detailed in Appendix D. Note that any variations in suggested exposures are just differences in expert opinion and do not constitute issues with the ESR report. We note that coarse particulate is typically more spatially variable than  $PM_{2.5}$  (USEPA, 2019) and thus there is greater potential for variability in the  $PM_{10}$  assessments. The assessment in Appendix D shows generally good agreement with the evaluation in the ESR report. The one exception is for the Arataki CUA for which we considered a slightly lower exposure of around 17  $\mu$ g/m<sup>3</sup> to likely be more representative.

#### 4.3.2 Temporal representation

The analysis uses the 2019 annual average concentration for  $PM_{10}$  at Rata Street of 20 µg/m<sup>3</sup>. The Bay of Plenty air quality monitoring report for 2023 (Iremonger, 2023) gives annual average concentrations at Rata Street ranging between 18 and 21 µg/m<sup>3</sup> from 2019 to 2022. The average across these years is 20 µg/m<sup>3</sup> with the most recent annual average for 2022 being 21 µg/m<sup>3</sup>. In our view, use of the 2019 annual average concentration of 20 µg/m<sup>3</sup> is appropriate as representative of this site.

The temporal representation of the PM<sub>2.5</sub> concentrations is a bit more complex. The Bay of Plenty air quality monitoring report for 2023 (Iremonger, 2023) shows an annual average PM<sub>2.5</sub> concentrations for 2019 of 8  $\mu$ g/m<sup>3</sup> followed by annual averages of 6  $\mu$ g/m<sup>3</sup> (for 2020, 2021 and 2022). Whilst is unclear if 2019 data represented baseline PM<sub>2.5</sub> or a high pollution year for PM<sub>2.5</sub>, some improvements in concentrations in the airshed from 2020 may have occurred as a result of MARPOL requirements on the shipping sector, as was observed for SO<sub>2</sub> at this site.

Whilst the ESR report provides an estimate of impact for 2019 only, this review considers the implications of the report for air quality management and thus needs to consider any temporal or trend issues.

#### 4.3.3 Impacts on quantitative health impact assessment

This section considers the significance of potential misclassification of exposure on premature mortality and other health endpoints detailed in the ESR report.

• <u>PM<sub>10</sub></u>

If the Arataki exposure to  $PM_{10}$  assumption was reduced from 20 µg/m<sup>3</sup> to 17 µg/m<sup>3</sup> the impact on the premature mortality (adults 30 years +) would be a reduction of two deaths per year (from 26 down to 24).

• <u>PM<sub>2.5</sub></u>

If the Totara 2019 PM<sub>2.5</sub> concentrations were used for exposure across the airshed, the impact of this on the health estimates for PM<sub>2.5</sub> would be an increase of around 25% (e.g., the premature mortality (adults 30 years +) would increase from 9 to 11). The 2020 onwards Totara PM<sub>2.5</sub> concentrations (~6  $\mu$ g/m<sup>3</sup>) would give health impact estimates similar to those in the ESR report.

<u>NO<sub>2</sub></u>

In our view, the two CAUs most likely impacted by shipping NO<sub>2</sub> concentrations are Mount Maunganui North and Omanu. The assumed annual NO<sub>2</sub> exposures from motor vehicles in the risk assessment for these areas are 8.3 and 6.6  $\mu$ g/m<sup>3</sup> and the estimated premature mortality is four premature deaths per year. We are uncertain what the impact of shipping NO<sub>2</sub> might be. If we assume an upper limit potential impact being a factor of two then this would equate to a further four deaths per year.

#### 4.3.4 Conclusion – misclassification of exposure

There is potential for variability in  $PM_{10}$  and  $PM_{2.5}$  concentrations in the study areas as there is in the studies used to derive the CRFs. Experts may have differing opinions on what likely exposures will be in different areas. However, the airshed is extremely well monitored for  $PM_{10}$  and more than adequately monitored for  $PM_{2.5}$  (sampler ratio of 1:10km<sup>2</sup>). Exposure misclassification is therefore likely to be significantly less than for most risk assessments where ratios of 1:100 or higher are common.

Notwithstanding this, we calculated the impact on risk assessment if different exposure assumptions were used. For  $PM_{10}$  we would have estimated two fewer deaths per year and for  $PM_{2.5}$  we would have estimated two additional deaths per year for 2019. The size of this variability is small in the context of risk assessment methodology. With respect to  $PM_{10}$  and  $PM_{2.5}$ , we conclude that different assumptions or approaches could be used to estimate concentrations in the residential areas, but these are unlikely to impact on the risk assessment in any meaningful way.

In the case of NO<sub>2</sub>, the health risk assessment is not based on monitoring and consequently there is potential for misclassification of exposure owing to the model not adequately accounting for sources. In this case the main source of NO<sub>2</sub> in the airshed has been excluded. We consider it likely that the risk assessment could substantively underestimates NO<sub>2</sub> premature mortality and other health endpoints of NO<sub>2</sub> exposure.

It is our view that:

- The approach to exposure classification in the ESR report is generally robust. There could be an
  exception with NO<sub>2</sub> exposure owing to the model relied on for NO<sub>2</sub> concentrations not including
  shipping emissions.
- The impacts of this on the analysis are uncertain.
- The impacts of any potential misclassification of exposure for PM<sub>10</sub> or PM<sub>2.5</sub> are likely to be small.
- The quantified impacts are likely to be indicative of the scale of impact for present day exposures and thus can be used for air quality management purposes without further trend evaluations.

## 4.4 Comparison to Ōtūmoetai

The ESR report compares the risk assessment estimates for MMA to  $\bar{O}t\bar{u}moetai$ . The only contaminant that is monitored in  $\bar{O}t\bar{u}moetai$  is PM<sub>10</sub>. An estimate is made for PM<sub>2.5</sub> concentrations based on an average ratio of 0.53 from the HAPINZ model. As with the Mount Maunganui NO<sub>2</sub> assessment, the concentrations of this contaminant estimated for  $\bar{O}t\bar{u}moetai$  are based on a modelling.

In our view, the differences between  $\overline{O}t\overline{u}moetai$  and MMA for NO<sub>2</sub> and PM<sub>2.5</sub> concentrations are likely to be within the uncertainties of the contaminant estimation methods. Sulphur point and Tauranga City-Marinas for NO<sub>2</sub> exposure are likely exceptions, as there is a greater difference in the estimated NO<sub>2</sub> concentrations. As these two areas have very low baseline health data owing to small populations and they have minimal impact on the analysis.

Because the objective of the comparison is to estimate differences in health endpoints as a result of exposure to air contaminants, the baseline health statistics should be adjusted for differences in age between the populations. This is because baseline health data will be higher for an older population. Whilst this has not been done, the health statistics in the HAPINZ model for baseline premature mortality is 0.87% of the population for both Ōtūmoetai and Mount Maunganui suggesting that the impact of any age differences between the areas is minimal or is being offset by other variables. We therefore assume that the impact of not having age adjusted the data is minimal.

The comparison does not take into account the exposures of over 11,000 workers in the MMA. The workers in the MMA whilst potentially younger overall may also have differing levels of underlying disease e.g., if there were a greater prevalence of smokers then there may be higher underlying cardiovascular disease. Additionally, the pollutant mix in the Mount Maunganui area is more complex and this is likely to have impacts on the health of Mount Maunganui residents. Ōtūmoetai residents will not be subject to the same complexity in contaminant exposures.

In our view the quantitative comparison should be on a  $PM_{10}$  only basis (noting any potential limitations) with a qualitative evaluation to accompany it that highlights the different nature of impacts and exposures.

In summary it is our view that:

- The NO<sub>2</sub> and PM<sub>2.5</sub> comparison is compromised by the absence of monitoring data.
- To compare the impact of air quality in the areas the baseline health statistics should have been adjusted for age.
- Residents in the Mount Maunganui area will be exposed to a more complex pollutant mix and increased overall exposures compared to Ōtūmoetai which is not accounted for in the quantified comparison.

- Whilst the two areas have the same number of residents, over 11,000 workers in the Mount Maunganui airshed will be exposed to air contaminants in that airshed during their working day. This is not accounted for in the quantified comparison.
- The Ōtūmoetai comparison should be limited to impacts of PM<sub>10</sub>.

### 4.5 Qualitative assessment – SO<sub>2</sub>, benzene, hydrogen sulphide

The qualitative assessment of impacts of exposure to  $SO_2$  concludes that people living at Whareroa Marae and the Tauranga Bridge Marina may have been and continue to be adversely affected by  $SO_2$  emissions. The analysis compares  $SO_2$  concentrations in these areas to health impacts associated with short term exposures In our view the qualitative analysis undertaken is robust and we concur with the conclusion drawn. It would be beneficial if references were included for the 10-minute average health impacts reported and if an explanation was provided as to why the concentration response relationships in the Orellano et al (2021) reference given were not used to estimate impacts. We consider the prevalence of high daily  $SO_2$  concentrations to add to the health burden at Whareroa Marae and Tauranga Bridge Marina in a way that it is not captured by the quantitative analysis, given that concentrations of  $SO_2$  in urban areas of New Zealand are relatively low. We note, however, that the CRFs for  $SO_2$  exposures in Orellano (2021) are low (i.e., 1.0059 per 10 µg/m<sup>3</sup> increase for all cause mortality) as is typically observed with the acute impacts assessments.

The  $H_2S$  assessment focuses on odour impacts. It would have been helpful if the ESR report had noted, in the qualitative assessment, that concentrations measured were below the guideline used for health impacts but above the odour threshold guideline. Otherwise, the qualitative assessment for  $H_2S$  and conclusions are appropriate in our view.

The review suggests that owing to the presence of the oil refinery and storage tanks the concentrations of benzene in ambient air could be significant. We note historical monitoring of benzene by BOPRC indicated concentrations at the Totara Street site below ambient air quality guidelines (Iremonger, pers comm, 2024). The Totara Street site is in the vicinity of the storage tanks but further away from the oil refinery than Whareroa Marae. These sources of benzene will add to the pollutant mix. Whilst "significant" is likely to overstate ambient benzene concentrations in our view, some updated monitoring including at the Whareroa Marae would assist with understanding benzene exposures.

#### 4.6 Sensitivity analysis, uncertainty and confidence assessment

A sensitivity analysis is undertaken on the  $PM_{2.5}$  and  $NO_2$  health endpoint estimates for only the difference between  $\bar{O}t\bar{u}$ moetai and Mount Maunganui. This reports the variation as a cost and provides a percentage difference to the base case. We could see no issues with the calculation. In our view sensitivity on the Mount Maunganui results alone and the difference to  $\bar{O}t\bar{u}$ moetai based on  $PM_{10}$  would have been useful.

The uncertainty assessment in the ESR report provides qualitative and quantitative assessments of uncertainty for different components of the risk assessment.

We generally concur with the uncertainty assessment with the exception that we do not consider the CRF confidence intervals include consideration of uncertainties with the application of the CRFs to the Mount Maunganui Area. In our view there is additional uncertainty associated with this application owing to the character of the MMA. This includes both a more complex pollution mix and a higher proportion of coarse particulate matter. Whilst we note that the issue of coarse particulate matter health impacts is addressed in the ESR report through the statement of WHO position being one of treating all PM as causative, this does not mean it does not impact on the CRF.

We also note insufficient evidence to support the statement that the exposure classification approach erred on the side of underestimation of exposure. In our view the estimates do not clearly represent a bias in either direction.

The ESR report appears to conclude that the overall uncertainty is around  $\pm 30\%$  and refers to it as a moderate degree of confidence in the model estimates. This seems reasonable in our view.

Our evaluation of the potential impacts on premature mortality of variations in exposure assumptions gave two less for  $PM_{10}$  and an increase of up to four for  $NO_2$ .

## 4.7 General

In the ESR report  $PM_{10}$  is referred to as "being known as coarse particulate". As detailed in Section 2.3 the term coarse particulate has evolved from the formation mechanism or mode and is thus aligned with the  $PM_{10}$ - $PM_{2.5}$  size fraction.  $PM_{10}$  is either referred to as  $PM_{10}$  or historically in New Zealand it was referred to as suspended particulate.

The source of the NO<sub>2</sub> exposure estimates in the ESR Report appears to be the HAPINZ 3 model. The model used does not appear to have included NO<sub>2</sub> from shipping in its methodology. The ESR report author notes that roadside monitoring in the MMA gives rise to much higher NO<sub>2</sub> concentrations than assumed for the residential area exposures and having viewed roadside NO<sub>2</sub> concentrations in the MMA we concur with this view. We consider it likely that the report underestimates health impacts from exposure to NO<sub>2</sub>.

The ESR report refers to sources of  $PM_{10}$  and  $PM_{2.5}$  and  $NO_2$  noting that  $PM_{10}$  is dominated by emissions from the Port and industrial activities and that  $PM_{2.5}$  and  $NO_2$  are likely to be dominated by emissions from ships and transport with some influence from industry. We disagree with this assessment noting that  $NO_2$  is likely dominated by those sources, but that industry and the port are the significant sources of  $PM_{2.5}$ . We also note that marine aerosol is a likely contributor to both  $PM_{10}$  and  $PM_{2.5}$ .

## 4.8 Conclusions

The method used in the ESR analysis is commonly used to quantify the burden of disease, typically in larger populations, associated with exposure to air contaminants.

We find no significant issues or errors with the analysis of health impacts in the Mount Maunganui area. In our view, the comparison to  $\tilde{O}t\bar{u}$ moetai should be limited to  $PM_{10}$  given the uncertainty in the concentrations estimates for the other contaminants overlaps with the concentrations for the same contaminants in Mount Maunganui. Whilst there is potential for less reliable outputs for applications to smaller populations this does not detract from the general conclusion.

In our view, the estimates of impact do not err on the side of underestimation of impact, however, except to the extent that they do not consider impacts in the workers or children exposed at early childhood education centres that do not reside in the Mount Maunganui Area.

Because PM<sub>10</sub> in the MMA contains a greater proportion of coarse-mode particulate than the average for New Zealand we have evaluated health literature to assess the possibility that the PM<sub>10</sub> CRF might overestimate impacts in the MMA. From that we concluded that the literature supports the coarse particulate size fraction being causal or suggestive of causal for a range of adverse health impacts including most of those used in the ESR risk assessment. There is the possibility that the size of impact is less than for PM<sub>2.5</sub> and if so the PM<sub>10</sub> CRF could overestimate impacts in the MMA. We do not anticipate this to be of enough significance to change the conclusions of the report except that we do not consider the estimates to err on the side of underestimation of impact. We note also that the MMA has a more comprehensive pollution mix than the urban areas used to derive the CRFs and that this could have the opposite effect of greater impact in the MMA.

In our view, the results of the report should be considered in the context of the purpose which is to provide information on the potential scale of adverse health outcomes. We concur with the report outcomes at this higher level. Some of the sub analysis undertaken, for example, area differences and health benefits of achieving guidelines, imply a level of accuracy that goes beyond the "scale of adverse health outcomes" in our view.

## **5 IMPLICATIONS FOR AIR QUALITY MANAGEMENT**

The MMA includes a complex mix of pollutants that collectively are likely to result in significant adverse health impacts. Epidemiological studies in New Zealand have attributed air quality impacts to PM<sub>2.5</sub>, NO<sub>2</sub> and PM<sub>10</sub> as the contaminants that were best able to explain relationships between health endpoints and exposures. Other epidemiological studies illustrate health impacts of exposures to SO<sub>2</sub> at levels of existing exposure in the MMA.

The implications for air quality management depend on the sources that are contributing to degraded air quality. The main sources of  $NO_2$  are shipping and motor vehicles. Whilst neither of these sources are readily manageable at a Regional or District level, the introduction of shore power would decrease  $NO_2$  from shipping.

Sources of  $PM_{2.5}$  in the MMA likely include industrial combustion process, shipping, cargo handling equipment, motor vehicles, handling and storage of bulk solid materials and logs and marine aerosol. Sources of  $PM_{10}$  in the MMA include bulk materials handling, log storage and handling, industrial activities, marine aerosol with smaller contributions from motor vehicles and shipping. In the case of  $SO_2$ , there are a limited number of sources with a small number of industrial activities and shipping contributing to concentrations.

Improving air quality and reducing the population exposures to these and other contaminants, particularly for sensitive individuals, would result in health benefits in Mount Maunganui.

The main mechanism available to Regional Councils for improving air quality in the MMA is the Regional Air Plan. Plan Change 13 (Air Quality) requires that bulk solid materials handling and log handling and storage activities obtain resource consents for their discharges. A 2023 Environment Court decision on Plan Change 13 (PC13) provides the opportunity for improvements in PM<sub>10</sub> in the airshed over time with affected industry required to submit dust management protocols and carry out monitoring prior to the expiry of rule R22A (three years from the Court decision) when resource consents are required under rule R22B. Other options such as classifying an unsealed yard as a discretionary activity encourages activities to seal their yards to avoid the resource consent process. In the MMA this approach is being implemented as an additional measure to control fugitive dusts.

Land use planning can be used to both reduce exposures and prevent further degradation of the airshed. Mechanisms for reducing exposures include locating industrial areas further from residential areas and not allowing sensitive activities such as childcare centres, schools, hospitals and retirement villages near to these areas. In an existing industrial area, mechanisms may be more limited. Zoning rules could assist with preventing further degradation by ensuring new or existing industry that wishes to relocate premises are not readily able to establish in the MMA and similarly new sensitive activities may not be permitted to locate in areas where degradation exists. There may also be opportunities to create new recreational areas such as sport fields further from the MMA.

An alternative mechanism for preventing further degradation in theory is through Regulation 17 of the National Environmental Standards for Air Quality. This requires new industry in a "polluted" airshed to offset the impact of their emissions. Whilst preventing further degradation by requiring offsets for new discharges, Regulation 17 does not provide for improvements in the Airshed. In an airshed where reductions in concentrations are needed to improve health, allowing a new industry to effectively "claim" a potential source of improvement that would otherwise result in a benefit to the airshed may not be desirable. Relying on Section 17 is therefore not preferable for preventing further degradation at present.

## 6 CONCLUSIONS

The ESR report assesses the health impacts in the MMA including quantification for the pollutants  $PM_{2.5}$ ,  $PM_{10}$  and  $NO_2$  for a selection of health endpoints and a qualitative assessment for  $SO_2$ , benzene and  $H_2S$ . The approach to quantification of the burden of disease associated with exposure to air contaminants used is accepted internationally.

In our view, the airshed has been extremely well characterised for  $PM_{10}$  and  $SO_2$  and has more than adequate monitoring of  $PM_{2.5}$  for its size. Following the most recent BOPRC air quality monitoring report (Iremonger, 2023), planned monitoring of  $PM_{2.5}$  at Rata Street, the establishment of a monitoring site in the residential area to the north-east of the MMA and monitoring of  $NO_2$  at Whareroa Marae have now been implemented. The additional monitoring will assist with an improved understanding of the airshed. Updated benzene monitoring will assist with airshed characterisation but is unlikely to result in substantive changes to the health risk assessment.

The risk assessment contributes to the understanding of health impacts of air quality in the MMA and provides an indication of the scale of impact using premature mortality and hospital admissions. We found no issues of substance and concur with the findings that air quality in the Mount Maunganui area will result in premature mortality and hospital admissions. We consider the calculations of numbers for these health endpoints likely to be indicative of the scale of impact.

In reaching this conclusion, we evaluated the nature of the airshed and undertook a review of the literature on health impacts of the coarse particulate fraction ( $PM_{10}-PM_{2.5}$ ) as  $PM_{10}$  concentrations in the MMA include a higher proportion of coarse particulate than most urban areas of New Zealand. The health evidence, whilst weaker than for the  $PM_{2.5}$  size fraction did indicate that the coarse particulate would likely contribute to adverse health impacts including some of the more significant endpoints quantified (e.g., long term exposures and premature mortality). The size of the effect is probably less than for  $PM_{2.5}$ , however, although the impact of this may be offset to some degree by the higher exposures in the MMA to a more complex pollutant mix.

For non-residents that are exposed for prolonged periods there is a health risk that is not able to be charactered by the risk assessment approach. This includes over 11,000 workers and children in childcare centres. Children are particularly susceptible to acute impacts of coarse particulate exposure. Other susceptible groups include the elderly and those with underlying cardiopulmonary disease (a risk factor for smoking).

The assessment of restricted activity days associated with exposure to air quality in the MMA is useful in that it highlights that there is a range of less severe health endpoints that impact on a greater proportion of the population. However, we consider the estimated impacts characterised by restricted activity days to be highly uncertain owing to reliance on a single study.

In our view, the results of the report should be considered in the context of the purpose which is to provide information on the potential scale of adverse health outcomes. We concur with the report outcomes at this higher level. Some of the sub analysis undertaken, for example, area differences and health benefits of achieving guidelines, imply a level of accuracy that goes beyond the "scale of adverse health outcomes" in our view.

In our view, the report supports the need to manage and minimise emissions of all contaminants in the MMA but with specific attention to  $PM_{10}$ ,  $PM_{2.5}$ ,  $NO_2$  and  $SO_2$ . The main sources of  $PM_{10}$ ,  $PM_{2.5}$  and  $SO_2$  are industrial activities, port activities and shipping. Motor vehicles and shipping are main sources of  $NO_2$ . Resource consents and land use planning are tools that can be used to improve air quality from industrial activities and minimise exposures.

We do not consider additional monitoring is needed to improve our understanding of the scale or significance of the health impacts of air quality in the Mount Maunganui area. There may be value in monitoring  $NO_2$  in an area where annual exposures will be most impacted on by shipping emissions (e.g., Rata Street) but this is unlikely to impact on scale significantly<sup>9</sup>.

<sup>&</sup>lt;sup>9</sup> The single model PM<sub>10</sub> outputs currently exceed the two pollutant (NO<sub>2</sub> + PM<sub>2.5</sub>) model

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## APPENDIX A: DATA ANALYSIS MOUNT MAUNGANUI AIRSHED

#### Correlation between RYS and Rata Street PM<sub>10</sub>

To evaluation the hypothesis that elevated PM<sub>10</sub> concentrations in the MMA are localised and do not have significant impacts beyond the vicinity of the discharge an evaluation of the correlation in PM<sub>10</sub> concentrations between Rata Street and Rail Yard South was carried out. Figure A1 shows the directional line from Rata to RYS is at around 170 degrees and that both are upwind of the BSM unloading area and the log handling area under a south south west (SSW) wind direction.

#### Methodology

- Data input Rata Street and Rail Yard South 10-minute average concentrations of PM<sub>10</sub>, wind direction and wind speed (both sites) from 2019 to 2022.
- Analysis method polar and polar frequency plots (weighted by wind direction to give contributions to annual average) and the dilution lines and pollutant ratios function in Openair R software (Carslaw & Ropkins, 2012).
- Data thresholds a coefficient of determination (r<sup>2</sup>) value of 0.85 was used to select "correlated" data periods. An additional concentration threshold of 50 µg/m<sup>3</sup> (ten-minute average) based on RYS data (average across sampler period) was used to remove low concentration correlations.
- The evaluation was set based on a rolling three-hour average (18 data points of 10 minutes)
- Polar Plot wind direction using NWR technique. The Openair manual described this method as "In NWR, smoothing is achieved using nonparametric kernel smoothers that weight concentrations on a surface according to their proximity to defined wind speed and direction intervals". It notes also that this method provides similar results to the default method but has advantages when there is insufficient data available to use a Generalised Additive Model (GAM).

#### **Results**

Figure A2 shows the slope of the correlation between  $PM_{10}$  concentrations measured at Rata Street and RYS from 2019 to 2022 for the methodology described above for the periods meeting the R<sup>2</sup> >0.85 and PM<sub>10</sub> (RYS) > 50 µg/m<sup>3</sup> criteria. The colour of the slope reflects the magnitude of the background concentrations at RYS. For example, in Figure A2 the cluster with the lowest slope (around 0.25) and highest RYS concentrations (reaching 250 µg/m<sup>3</sup>) are in a light blue to green shades indicating that when this correlation occurs (a source or correlating factor impacts on both RYS and Rata Street) background concentrations from other sources are low at around 10 µg/m<sup>3</sup>.



Figure A1: Location of historical air quality monitoring sites in the Mount Maunganui Airshed.

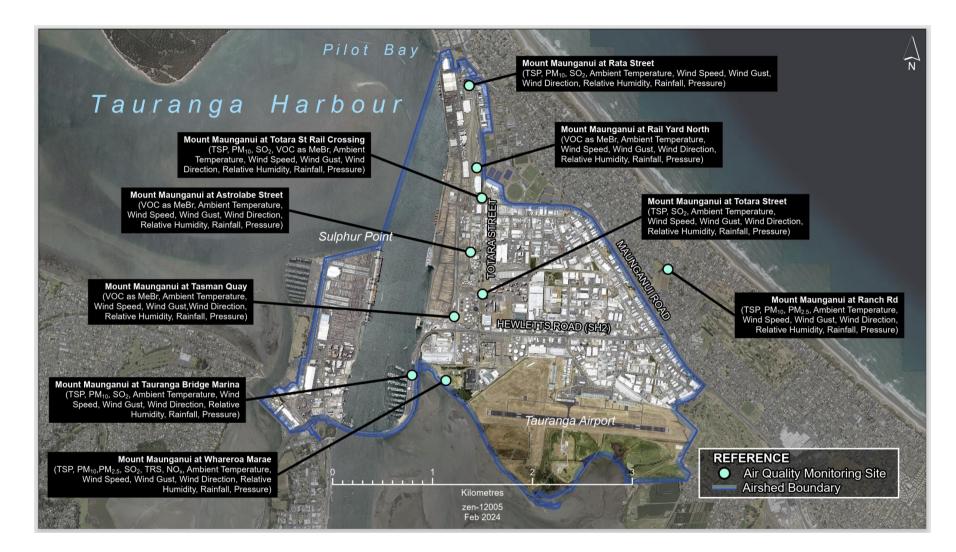


Figure A2: Location of existing air quality monitoring sites in the Mount Maunganui Airshed

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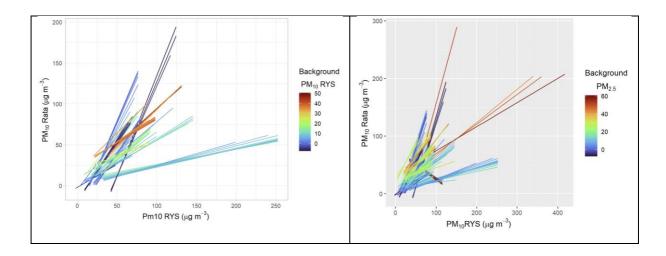


Figure A2: Slope of relationships between PM<sub>10</sub> at Rata and RYS from 2019 to 2022 for R<sup>2</sup> >0.85 (left) and 0.7 (right) and PM<sub>10</sub> at RYS > 50  $\mu$ g/m<sup>3</sup> (10 minute average).

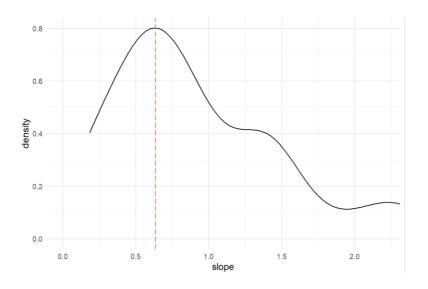




Figure A3 shows the frequency of different slopes with the mode (most common slope) illustrated by a red dashed line at around 0.65 with a relatively normal distribution for slope frequencies in the 0.2 to 1.2 range. The mode slope indicates concentrations are around 35% higher at RYS than at Rata Street. Although less frequent than the 0.2 - 1.2 slope range, correlations also occur in the range of 1.2 - 2.3. These higher slopes indicate PM<sub>10</sub> concentrations are higher at Rata Street than RYS.

An evaluation of slope by wind direction and speed shows that the high frequency slopes occur predominantly from the south south west (SSW) to southwest (SW) wind sectors with a narrow band width at the North (N) also in this range (Figure A4). The SW directions are also the directions of the highest  $PM_{10}$  concentrations at Rata Street (Figure A5). These wind directions carry sources from the Port area to RYS and Rata Street with the Rata Street concentrations ranging from 0.6 to 0.8 of the RYS concentrations for wind speeds up to 3 ms<sup>-1</sup> and 0.4 to 0.6 for higher wind speeds.

The northeast (NE) and north west (NW) wind quadrants both reflect higher concentrations at Rata Street than at RYS (slope >1) and likely indicate marine aerosol contributions. It is important to note that Figure A4

represents average slopes for each wind direction/speed for which correlations exist. Thus, inferences about prevalence cannot be made from Figure A4 (left) unless considered in conjunction with Figure A4 (fight). It is also a relatively loose inference in that the contribution to A4 right might occur where there is no correlation.

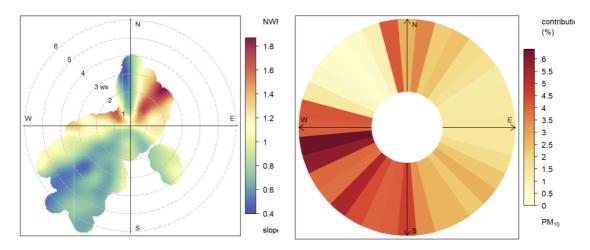
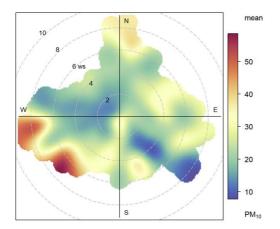


Figure A4: (Left) Polar plot illustrating the slope of correlated  $PM_{10}$  concentrations at Rata Street and RYS and right polar frequency plot for Rata Street 2019.



### Figure A5: Polar plot for Rata Street 2019.

These graphical outputs and associated analysis provide an indication of the dilution effect by comparing concentrations at RYS and Rata Street during periods where there are correlated and examining the extent of dilution and for different wind speeds. This can be useful in understanding the extent to which emissions sources are having localised impacts or if they are contributing significantly to concentrations across a longer distance.

# APPENDIX B: CORRELATION BETWEEN TOTARA STREET PM<sub>10</sub> AND PM<sub>2.5</sub>

An evaluation of the relationship between  $PM_{2.5}$  and  $PM_{10}$  at Totara Street was carried out to assist with understanding of sources of  $PM_{2.5}$  and  $PM_{10}$ .

## Methodology

- Data input Totara Street –10 minute average concentrations of PM<sub>10</sub>, wind direction and wind speed with a focus on 2019 data but evaluations also made for larger datasets. The focus on 2019 is because of the increasing storage of containers at the Totara Street monitoring site and the potential interference of these with airflows around the site (see Iremonger, 2023).
- Analysis method polar and polar frequency plots (weighted by wind direction to give contributions to annual average) and the dilution lines and pollutant ratios function in Openair R software (Carslaw & Ropkins, 2012).
- Data thresholds a coefficient of determination (r<sup>2</sup>) value of 0.70 was used to select "correlated" data periods. An additional concentration threshold of 10 µg/m<sup>3</sup> (ten-minute average) based on PM<sub>10</sub> data (average across sampler period) was used to remove low concentration correlations.
- The evaluation was set based on a rolling three hour average (18 data points of 10 minutes)
- Polar Plot wind direction using NWR technique. The Openair manual described this method as "In NWR, smoothing is achieved using nonparametric kernel smoothers that weight concentrations on a surface according to their proximity to defined wind speed and direction intervals". It notes also that this method provides similar results to the default method but has advantages when there is insufficient data available to use a Generalised Additive Model (GAM).

Results

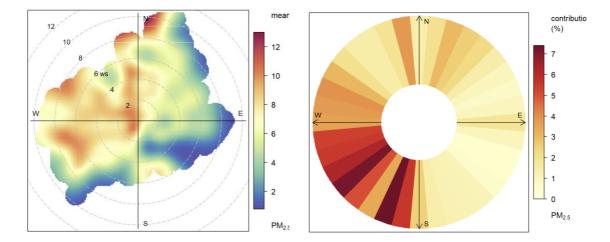


Figure B1: Polar plots for Totara  $PM_{2.5}$  (left) and  $PM_{2.5}$  polar frequency (right) weighted by wind direction to illustrate percentage contributions

Figure B1 (left) shows the  $PM_{2.5}$  polar plot (average  $PM_{2.5}$  concentration for each wind direction and speed) for the 10min data at Totara Street for 2019. Highest  $PM_{2.5}$  concentrations occur when the wind is blowing between SW and NNE. Some high  $PM_{2.5}$  concentrations occur under calm conditions (wind speeds less than 2 ms<sup>-1</sup>) whilst other elevated  $PM_{2.5}$  concentrations occur when the wind speed is greater than 6 ms<sup>-1</sup>. To the north there appears to be a source of  $PM_{2.5}$  that results in higher concentrations across a broad range of wind speeds including those greater than 8 ms<sup>-1</sup>.

While the polar plot can highlight the wind directions and wind speed associated with the different  $PM_{2.5}$  concentrations it is not helpful in assessing relative contribution directions because of variability in the prevalence of different wind directions. The polar frequency plot on the right in Figure B1 shows the  $PM_{2.5}$  concentrations weighted by wind direction prevalence to illustrate directions with the greatest impact on  $PM_{2.5}$  measured at Totara Street. Figure B2 shows the similar graphical presentations for  $PM_{10}$  data at Totara for 2019. Concentrations of  $PM_{10}$  are significantly higher than those of  $PM_{2.5}$  at Totara Street. The highest  $PM_{10}$  concentrations occur from the SW to W under higher wind speeds (typically between 6 and 10 ms<sup>-1</sup>).

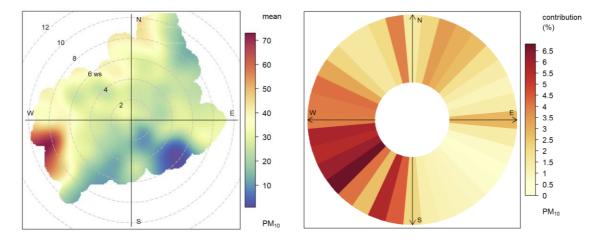
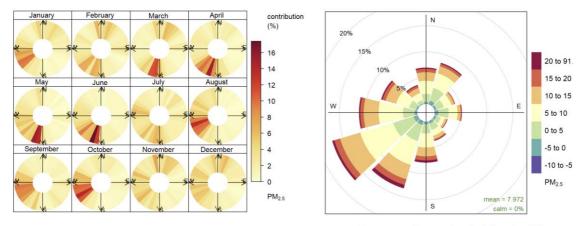


Figure B2: Polar plots for Totara  $PM_{10}$  (left) and  $PM_{10}$  polar frequency (right)weighted by wind direction to illustrate percentage contributions



Frequency of counts by wind direction (%)

Figure B3: Polar frequency plots for  $PM_{2.5}$  concentrations at Totara for 2019 (10 minute data) by month (left) and  $PM_{2.5}$  pollution rose.

Figure B3 (left) illustrates significant monthly variations in contributions to  $PM_{2.5}$  concentrations at Totara Street. The extent to which variations occur as a result of variability in wind direction as opposed to sources

may be inferred by comparison to Figure B3 (right) which shows the frequency of each wind direction by PM<sub>2.5</sub> concentration range. Figure B3 (right) shows that the higher PM<sub>2.5</sub> concentrations occur across a range of wind directions but occur slightly more frequently from the south an SSW quadrants.

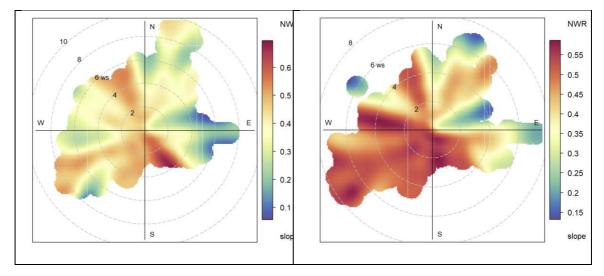


Figure B4: Polar plot illustrating the slope of correlated  $PM_{2.5}$  and  $PM_{10}$  concentrations at Totara Street for different levels of correlation (r2 = 0.7 - left and r2 = 0.8 - right).

Figure B4 shows the slope of the correlations between PM<sub>2.5</sub> and PM<sub>10</sub> averaged for each wind direction and speed for data with correlations of  $r^2 = 0.7$  (left) and  $r^2 = 0.8$  (right). The higher correlation requirement ( $r^2 = 0.8$ ) delivers a smaller dataset and results in a higher slope value. This is likely because when the ratio between PM<sub>2.5</sub> and PM<sub>10</sub> is low (e.g., 0.3) it is easier for the correlation to be negated by the presence of other PM<sub>2.5</sub> sources. The lower correlation dataset (left) is a better representation of the average situation for correlated data, whereas the higher correlation dataset (right) shows that within the broader dataset there are periods where the ratios are much higher.

Figure B4 (left) shows that sources of  $PM_{10}$  and  $PM_{2.5}$  concentrations to the south (S), SW, NW and SE of Totara Street have the highest ratio of  $PM_{2.5}$  to  $PM_{10}$ . The  $PM_{2.5}$  to  $PM_{10}$  ratio from the SW direction is around 0.5 and this correlation occurs for a range of wind speeds. This could reflect a contribution from a fertiliser plant located around 700m to the SW of Totara Street. The ratio of  $PM_{2.5}$  to  $PM_{10}$  from the NW/NNW wind direction is around 0.5. This may reflect the contribution from BSM loading at the Port of Tauranga as berths 7 and 8 where this activity occurs are located to the NW of Totara Street. The source that bands around the S has a ratio of  $PM_{2.5}$  to  $PM_{10}$  of 0.7 or greater and likely represents a combustion source. Lawters is a significant combustion source in the Mount Maunganui airshed and is located directly south of Totara Street.

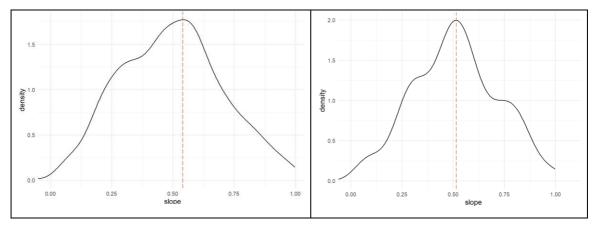
Two other high ratio sources apparent in Figure B4 (right) occur to the SW under a wind speed of between 6 and 8 ms<sup>-1</sup> and to the WNW under a wind speed of 2-5 ms<sup>-1</sup>. Further analysis not illustrated here shows the high wind SW datapoints occurred only in October (12 October 2019). The WNW ratio occurred in July and potentially February.

It is noted from figures B1 and B2 that the WSW to W quadrant includes both  $PM_{10}$  and  $PM_{2.5}$  concentrations at the higher end of the scale. Figure B4 shows that the slope of  $PM_{2.5}$  to  $PM_{10}$  for these concentrations as well as other concentrations in the WNW to NW quadrant is low at around 0.3 to 0.4. This suggests that the source of  $PM_{10}$  to the west of Totara Street has a lower proportion of  $PM_{2.5}$ . The main source of  $PM_{10}$  from this wind direction is likely to be log handling and storage at the POT.

The NE quadrant is dominated by correlations with ratios around 0.3 to 0.4  $PM_{2.5}$  to  $PM_{10}$ . As indicated previously (with  $PM_{10}$  concentrations higher than and correlated with RYS concentrations) this wind direction is likely to give rise to marine aerosol contributions to  $PM_{10}$  and  $PM_{2.5}$ . As the majority of the marine aerosol is typically in the coarse mode and this ratio is sensible for this source.

A further point of interest from Figure B4 is the prevalence of a source of PM<sub>10</sub> to the east of Totara Street with a low proportion of PM<sub>2.5</sub> around 0.2 to 0.3. The area to the east of Totara contains a number of BSM warehouses. It is possible that the PM<sub>2.5</sub> to PM<sub>10</sub> ratio for BSM handling in a warehouse differs for ship unloading owing to the increased abrasion caused by ship unloading mechanisms or it may be that the ratio is being confounded by other sources. Additionally, the proportion of PM<sub>10</sub> that is in the PM<sub>2.5</sub> size fraction for the handling of BSM will depend on the materials being handled and the mitigation and operation of the warehouses and operators. Further BSM warehouses located to the north of Totara, may be the source of similar PM<sub>2.5</sub> to PM<sub>10</sub> ratios from the north. Whilst the sources to the east with these ratios do not appear to result in significant elevated PM<sub>2.5</sub>, the sources to the north do give rise to elevated PM<sub>2.5</sub> concentrations are illustrated in Figure B1 (polar plot PM<sub>2.5</sub>) although it is noted that the very high PM<sub>2.5</sub> concentrations from Figure B1 occur under winds greater than 7 ms<sup>-1</sup> and these are absent from the slope polar plot indicating that they do not represent a period of correlation with PM<sub>10</sub>.

Figure B5 shows the slopes density plot for data fitting the correlation criteria of 0.7 ( $r^2$ ) at Totara with a mode of around 0.55 indicating that the most frequent correlation was for a slope of .55 for PM<sub>2.5</sub> to PM<sub>10</sub>. Figure B6 shows that for the majority of the correlations the background PM<sub>2.5</sub> (i.e., concentrations of PM<sub>2.5</sub> present that are not explained by the slope) are low. This is indicated by the colour of the slope, with the background concentrations able to be extrapolated by visualising where the line might cross the y axis. There are a very small number of slopes illustrated whereby a decrease in PM<sub>2.5</sub> corresponds with an increase in PM<sub>10</sub>. These may be data anomalies or may represent a wind related phenomena such as increased wind speed causing more coarse mode particulate (with only a small proportion of PM<sub>2.5</sub>) but increasing dispersion of another source with a higher PM<sub>2.5</sub> to PM<sub>10</sub> ratio.



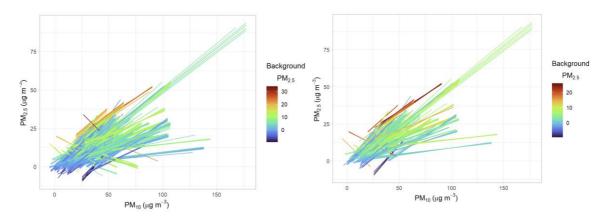
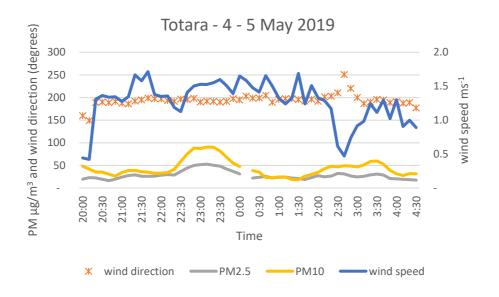


Figure B5: Slope density plot for  $PM_{2.5}$  and  $PM_{10}$  at Totara for 2019 for  $R^2 > 0.70$  (left) and  $R^2 = 0.8$  (right).

Figure B6: Slope plots for  $PM_{2.5}$  and  $PM_{10}$  at Totara for 2019 for  $R^2 > 0.70$  (left) and  $R^2 = 0.8$  (right).

Figure B6 shows only a handful of days when very high  $PM_{2.5}$  concentrations occur and a slope of around 0.5 with low background (other source) contributions on these days. These episodes occur for relatively short periods of time and occur when the wind is from the SW quadrant with winds less than 1 ms<sup>-1</sup> (13 March, ratio 0.5,  $PM_{2.5}$  maximum 90 µg/m<sup>3</sup>, around 7am, WD – SW -variable owing to low velocity), between 1-2 ms<sup>-1</sup> (5 April,  $PM_{2.5}$  maximum 55 µg/m<sup>3</sup>, ratio 0.5 around 6:30am, WD around 205 degrees). An example of a higher  $PM_{2.5}$  concentrations with a high slope event is 4 May (1-2 ms<sup>-1</sup>, ratio 0.7, maximum  $PM_{2.5}$  52 µg/m<sup>3</sup>, 8pm to 4:30am, WD around 190 degrees). Daily variations in  $PM_{2.5}$  and  $PM_{10}$  concentrations and meteorological variables for the latter incident are shown in Figure B7. This likely is a combustion source and is located to the south of the Totara Street monitoring site, most likely Lawter.



# Figure B7: Daily variations in $PM_{2.5}$ and $PM_{10}$ at Totara for 4-5 May 2019 illustrating elevated $PM_{2.5}$ concentrations occurring overnight.

This analysis indicates that the largest contributions to  $PM_{2.5}$  concentrations at Totara Street are from the south west quadrant. Whilst it is difficult to draw conclusions regarding sources from monitoring data, the correlation method used here for  $PM_{2.5}$  to  $PM_{10}$  ratios provides examples of when measured concentrations are likely to reflect the majority of concentrations coming from a single source. There may be instances where the correlation is a consequence of increased emissions for several sources as a result of changes in wind speed, however. Results suggest that industrial sources including fugitive dust sources are likely contributing to  $PM_{2.5}$  concentrations as well as  $PM_{10}$ .

# APPENDIX C: WHO GUIDELINE (2021) SUMMARY OF HEALTH ENDPOINTS

Pollutant	Health outcomes used in Global update 2005	Health outcomes selected for updating in the 2021 air quality guidelines	Justification for health outcome selection
PM <sub>2.5</sub> and PM <sub>10</sub>	Total, cardiopulmonary and	All-cause mortality	CAUSALITY DETERMINATION (REFERENCE)
	lung cancer mortality	<ul> <li>Cardiovascular mortality (all, cerebrovascular, IHD)</li> <li>Respiratory mortality (any, COPD, acute lower respiratory infections)</li> <li>Lung cancer mortality</li> </ul>	<ul> <li>PM<sub>2.5</sub></li> <li>Causal for cardiovascular and respiratory mortality (US EPA, 200</li> <li>Causal for total and cardiovascular mortality (Health Canada, 200</li> <li>PM</li> <li>Causal for total mortality in relation to PM (Health Canada, 2013)</li> <li>Group 1<sup>b</sup> lung cancer for PM (Straif et al., 2013)</li> <li>Likely causal for lung cancer mortality in relation to PM (Health Canada, 2013)</li> </ul>
			SUPPORTING CONSIDERATIONS
			<ul> <li>PM<sub>10</sub></li> <li>Health outcome supported by evidence from PM<sub>10</sub> and PM<sub>2.5</sub></li> </ul>
			OTHER RELEVANT CAUSAL DETERMINATIONS (REFERENCE)
			<ul> <li>PM<sub>2.5</sub></li> <li>Likely causal for respiratory effects (US EPA, 2009)</li> <li>Likely causal for respiratory effects (Health Canada, 2013)</li> </ul>

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 PM2.5 and PM10
 COHb levels of below
 · All-cause mortality
 CAUSALI

 2% in nonsmokers' blood (also protective for longterm exposure) (WHO
 · Cardiovascular mortality
 PM2.5

 Regional Office for Europe, 2000a, 2010)
 · Respiratory mortality
 · Causal (US EPA)

#### CAUSALITY DETERMINATION (REFERENCE)

- Causal for all-cause, cardiovascular and respiratory mortality (US EPA, 2009)
- Causal for all-cause, respiratory and cardiovascular mortality (Health Canada, 2013)

#### SHORT-TERM EXPOSURE

Pollutant	Health outcomes used in Global update 2005	Health outcomes selected for updating in the 2021 air quality guidelines	Justification for health outcome selection
PM <sub>2.5</sub> and PM <sub>10</sub>			PM (any size fraction)
(contd)			Causal for all-cause mortality (Health Canada, 2013)
			SUPPORTING CONSIDERATIONS
			<ul> <li>Cardiovascular and respiratory mortality also considered in causa determination of respiratory/cardiovascular effects (US EPA, 2009) (see other relevant causal determinations)</li> </ul>
			+ $PM_{10}$ , supported by evidence from $PM_{2.5}$
			OTHER RELEVANT CAUSAL DETERMINATIONS (PM <sub>2.5</sub> ) (REFERENCE)
			Likely causal for respiratory effects (US EPA, 2009)
			Causal for cardiovascular effects (US EPA, 2009)
			Causal for respiratory effects (Health Canada, 2013)
			Causal for cardiovascular effects (Health Canada, 2013)

# APPENDIX D: MISCLASSIFICATION OF EXPOSURE – SPATIAL EVALUATION

Population health data in the ESR report were based on census area unit (CAU) 2013 boundaries. The areas included and the exposure assumptions used in the ESR report for annual average concentrations in the Mount Maunganui area are shown in Table D1.

Table D1: Ar	reas and exposure a	assumptions used in	n ESR report
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	PM <sub>10</sub> exposure assumption and basis	PM <sub>2.5</sub> exposure assumption and basis	NO <sub>2</sub> exposure*
Mount Maunganui North	20 μg/m³ (Rata St 2019)	6.4 µg/m <sup>3</sup> (0.32 x PM <sub>10</sub> )	7.8 µg/m³
Omanu	20 μg/m³ (Rata St 2019)	6.4 µg/m <sup>3</sup> (0.32 x PM <sub>10</sub> )	6.2 µg/m³
Arataki	20 μg/m³ (Rata St 2019)	6.4 µg/m <sup>3</sup> (0.32 x PM <sub>10</sub> )	8.1 µg/m³
Sulphur Point	14 µg/m <sup>3</sup> (Sulphur Point - 2019	4.5 μg/m <sup>3</sup> (0.32 x PM <sub>10</sub> )	12 µg/m³
Tauranga City-Marina	16 µg/m³ (Bridge Marina – 2019)	5.1 μg/m <sup>3</sup> (0.32 x PM <sub>10</sub> )	12 µg/m³

\* NO<sub>2</sub> exposure assumptions are concentration estimates made for HAPINZ 3 for 2016 adjusted downwards (0.9354) for vehicle technology improvements for a 2019 base year.

The spatial distribution of reference method samplers across polluted airsheds in New Zealand varies with area. In Christchurch the ratio of samplers per square kilometer is around 1:470 and in Napier it is 1:100. The Mount Maunganui Airshed is around 5.5 kilometers squared and has seven reference method sampling sites for  $PM_{10}$ . This is the most spatially comprehensive monitoring for  $PM_{10}$  in New Zealand with many reference method samplers within a few kilometers of each other.

The potential for misclassification of exposure for  $PM_{10}$  is therefore low in the context of a risk assessment and the anticipated exposure variability that underpins both epidemiology and risk assessment. This is because the furthest distance being extrapolated is less than six kilometers. Risk assessments generally extrapolate from a single monitoring site to a much wider area often in excess of 30 kilometers. Thus, it is reasonable to expect that the variability (in exposures) that you would get across the six kilometers will be less than the variability you would get when extrapolating across the more typical distances and importantly the variability that might exist across the epidemiological studies underpinning the risk assessment.

Spatial variability in PM<sub>2.5</sub> concentrations tends to be lower than for PM<sub>10</sub>. In the case of the MMA there is only one PM<sub>2.5</sub> sampler and seven PM<sub>10</sub> samplers so the spatial characterisation is less. In the ESR report PM<sub>10</sub> data from Rata Street are used to estimate PM<sub>2.5</sub> concentrations based on the PM<sub>2.5</sub> to PM<sub>10</sub> ratio at Totara Street. The ratio is 0.32 PM<sub>2.5</sub>:PM<sub>10</sub> and is at the lower end of the scale for industrial areas (typical range in New Zealand is 0.3 - 0.5). In our view use of the annual average PM<sub>2.5</sub> concentration at Totara Street for PM<sub>2.5</sub> exposure across the airshed would have been suitable for assessing airshed exposures.

The potential for misclassification of exposure for NO<sub>2</sub> is higher however, as the concentration estimates is based on a modelled output which doesn't include a significant source of NO<sub>2</sub> in the airshed. The emission rate for NO<sub>x</sub> and SO<sub>x</sub> from shipping pre MARPOL in the inventory were similar (Wilton, 2019) although conversions to NO<sub>2</sub> and SO<sub>2</sub> will vary. Improvements in annual SO<sub>2</sub> concentrations in the MMA since MARPOL range from 5  $\mu$ g/m<sup>3</sup> at Whareroa Marae to 19  $\mu$ g/m<sup>3</sup> at RYS. A comparison of emissions inventory data from 2018 to 2022 (Wilton, 2019, Wilton, 2023) shows industrial sources of SO<sub>2</sub> remain unchanged over this period. It seems reasonable that the impact of NO<sub>2</sub> from shipping is not insignificant. Thus, the ESR report which uses concentrations of 6 – 12  $\mu$ g/m<sup>3</sup> with variability linked to vehicle flows likely underestimates NO<sub>2</sub> exposure.

Further evaluation of exposure assessment for  $PM_{10}$  has been undertaken for each CAU included in the Mount Maunganui health impacts assessment.

## Mount Maunganui North

Figure D1 shows the Rata Street monitoring site location within the census area unit of Mount Maunganui North and an illustration of the relative contribution of  $PM_{10}$  from different wind directions to annual average  $PM_{10}$ concentrations at Rata Street for 2019. The graphs illustrate that the predominant winds are from the west, south west and north and that the majority of the  $PM_{10}$  concentrations measured at Rata Street originate from the southwest (SW) the west (W), the south east (SE) and the north (N). A yard to the south and in close proximity to the Rata Street may contribute when the wind is in the S to SW quadrant. This contribution appears less than 20% of the annual average  $PM_{10}$ . Appendix A also shows that some of these contributions occur when there is a correlation between  $PM_{10}$  at Rata Street and RYS (see Figure A4 left). Contributions from the yard site to annual average concentrations at Rata Street should not be detrimental to the use of this site as an indicator of exposure<sup>10</sup>.

The applicability of the Rata Street monitoring site in Mount Maunganui North to the residential areas within the same CAU is realistic even outside of the generalised nature of risk assessments. The distance from Rata Street to most residential areas is less than a kilometre, the exception being the area to the NE of Rata Street. The majority of the CAU is downwind of the industrial areas and PM<sub>10</sub> sources within the MMA under prevailing winds. The exception is the area adjacent to Mount Maunganui to the far NW of Rata Street.

Some dilution of sources of PM<sub>10</sub> from the MMA may occur over this distance although it is also likely that sources within the residential area will also contribute to exposures. Appendix A shows correlations in PM<sub>10</sub> concentrations between RYS and Rata Street (distance = 1.2 kilometers) and indicates that when data at the sites are correlated and from the SW the Rata Street sites are about 0.68 of the RYS concentrations. The correlation in the data indicates a consistent relationship in the impact of a source or sources on the two sites and thus it seems a reasonable indication of the dilution of sources from the Port over this distance under the conditions giving rise to the correlation. If the dilution continued at the same rate the area to the north beyond Rata Street may experience some improvements in contribution from the MMA but this is unlikely to have significant impact on annual average concentrations. There are also portions of residential areas within the CAU that are closer to the MMA industrial area. In our view use of the Rata Street monitoring site to estimate exposure in Mount Maunganui North is appropriate and will provide a closer estimate of exposure than one would normally expect with a risk assessment methodology.



# Figure D1: Wind rose for Rata Street monitoring site for 2019 and location of monitoring site within Mount Maunganui North CAU (left) and PM<sub>10</sub> concentration contributions by wind direction (right).

<sup>&</sup>lt;sup>10</sup> If the contribution of this site were around 20% (worst case assumption given RYS correlations for this wind direction) and the dilution across the residential area were similar to that from the port area to Rata Street (as indicated by RYS to Rata slope) then exposure would reduce to around 19  $\mu$ g/m<sup>3</sup> (20% x (1-0.68) x 20  $\mu$ g/m<sup>3</sup>).

## <u>Omanu</u>

The Omanu CAU includes the airport, the Mount Maunganui industrial area and a residential area to the east of the industrial area. The main residential area in Omanu is to the east of the industrial area with most dwellings located between 1-3 km from the Port and is downwind of the Port area when the wind has a westerly trajectory. Between the Port and the residential area there are numerous industrial sources of particulate which will have additional impact on this area.

A small number of residents in the Omanu CAU reside at the Whareroa Marae. The exposure of this population is more likely represented by concentrations of  $PM_{10}$  measured at the Marae. As baseline mortality statistics are available only for the whole CAU assessing for subsets is not possible, nor appropriate.

Use of an annual average  $PM_{10}$  concentration of 20  $\mu$ g/m<sup>3</sup> for the Omanu CAU seems reasonable considering prevailing wind directions and distances from major sources within the Port area.

### <u>Arataki</u>

The Arataki CAU is located to the east of the Tauranga Airport and is downwind of the POT on a NW wind trajectory which is not a particularly prevalent wind at around 10% at Whareroa Marae (Iremonger, 2023). There are a few particulate sources located to the west including bulk solid material (BSM) warehouses at De Havilland Way. The Arataki area is relatively close to the De Havilland Way monitoring site which has annual average concentrations of around 19  $\mu$ g/m<sup>3</sup> (average 2019 to 2022). Peak concentrations at the monitoring site are influenced by the warehouse facilities and it is unclear to what extent they might reflect chronic exposures in the Arataki area.

Use of an annual average  $PM_{10}$  concentration of 20  $\mu$ g/m<sup>3</sup> for the Arataki CAU could be too high given the prevailing wind direction and locations of sources. In our view an annual average concentration of around 17  $\mu$ g/m<sup>3</sup> might be more realistic for this area.

Notwithstanding this, the approach taken in the ESR report (using data from a monitoring site) is better practice than making estimates based on judgement. The authors have been relatively cautious in selecting the monitoring site with the lowest annual average concentrations of the sites within the main industrial area, i.e., excluding the sites to the south west which appear less impacted by the industrial area for longer term averages owing to the prevalence of the SW wind. In the case of Arataki, however, of the Whareroa Marae monitoring site may have been a closer approximation for annual average PM<sub>10</sub> concentrations.