In the Environment Court of New Zealand
Auckland Registry

I Mua I Te Kōti Taiao O Aotearoa
Ki Tāmaki Makaurau

ENV-2023-AKL-160

Under	the Resource Management Act 1991
In the matter of	An application for a direct referral to the Environment Court under section 87G of the Act for an order granting the applicant's resource consent applications to construct and operate a new asphalt plant at 54 Aerodrome Road, Mt Maunganui, together with an application for consent to authorise the continued operation of the existing asphalt plant on the site pending construction of the new plant
Between	**Allied Asphalt Limited**
	Applicant
And	**Bay of Plenty Regional Council and Tauranga City Council**
	Consent Authorities

Statement of Evidence in Reply of Dr Lynette Denison

Date: 26 April 2024

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Qualifications and experience

1. My full name is Dr Lynette Susan Denison.

2. I hold the position of Technical Director – Health Risk Assessment at Tonkin & Taylor Pty Ltd. My qualifications and experience are as set out in my evidence in chief dated 29 February 2024.

3. In addition to the summary of my experience presented in my evidence dated 29 February 2024 I would like to add the following experience. This wasn't included in my primary evidence as I didn't believe it was relevant to this hearing at that time. However, issues raised in the evidence of Mx Wickham and Dr Wilton have made this information relevant.

4. I have been a co-investigator on several epidemiological studies on air pollution and health in Australia. A list of relevant publications in peer reviewed journals is attached – Attachment 1.

5. In addition, I have been co-investigator on several Government Publications on air pollution and health including the Australian Air Pollution and Children's Health Study and Multicity Mortality and Morbidity Study, funded by the Environment Protection and Heritage Council, the Melbourne Air Pollution Mortality Study and Air Pollution and Hospital Admissions Study in Melbourne for EPA Victoria.

6. I was also a member of the NEPC Working Group that established a methodology for setting air quality standards in Australia. This guidance provides a methodology for assessing epidemiological studies for use in air pollution risk assessments to support the development of risk-based air quality standards.

7. I confirm that in preparing this reply evidence I have complied with the Code of Conduct for expert witnesses contained in the Environment Court of New Zealand Practice Note 2023.

Correction to Primary Evidence

8. In preparing this reply evidence I found an error in the risk estimate tables for carcinogenic risk and the hazard quotients that were included in the HRA and in my primary evidence.

9. This error was a transcription error from the risk calculation spreadsheet. These have been corrected and are attached to this reply evidence – Attachment 2.

10. This revised data does not affect the conclusions of the HRA, or my opinions presented in my primary evidence. The calculated risks in the revised tables are lower than those presented in my primary evidence.

Scope of evidence

11. Further technical information was requested at the air quality expert conferencing on:

    (a) Health statistics used in the health risk assessment (HRA);

    (b) Assessment of risk to workers in adjacent industrial locations; and

    (c) Justification of use of OEHHA unit risk factors

12. This further information is presented in this reply evidence.

13. This reply evidence responds to the evidence of:

    (a) Mx Lou Wickham

    (b) Dr Emily Wilton

14. I have structured my reply under the following topic headings:

    (a) Approach to the health risk assessment – incremental impacts

    (b) Health statistics used

    (c) Concentration response functions

    (d) Trade-offs of health effects for individual pollutants

    (e) Choice of unit risk factors for cancer risk assessment

    (f) Acceptable risk levels

    (g) Cumulative risks

    (h) Assessment of risk using non-continuous exposure scenario

    (i) Assessment of risk for workers in industrial area

    (j) Assessment of risk to potential residents in Airport precinct (Aerodrome Road and Dakota Way)

Incremental Approach to the HRA

15. The approach taken in the HRA was to assess the incremental impact of the emissions from the Allied Plants – both existing and proposed. This is appropriate and standard practice for a consent application for an individual industry.

16. In the health expert caucusing and in the evidence of Dr Wilton[1] there appears to be some confusion about the purpose of the HRA in estimating the effects of air quality in the whole airshed on the health of the population of Mount Maunganui. The HRA assessed the localised effects of the Allied Plants at specific receptors.

17. There was discussion in the health conferencing that the HRA should drive a policy response for the whole airshed. This was not the purpose of the HRA done for the Allied consent application. The ESR HRA for the Mount Maunganui airshed was conducted for that purpose.

18. In the Joint Witness Statement of the health risk assessment experts, Dr Wilton and Mx Wickham raised concerns that the incremental risk approach used in the HRA did not consider cumulative impacts in a polluted airshed. They were of the view that the application should be considered in terms of overall benefits to health for the whole airshed.

19. The cumulative effects of the emissions from the existing and proposed Allied Plants on air quality considering existing background data where available, has been addressed in the Air Quality Assessment (Tonkin & Taylor, 2024) and the evidence of Ms Simpson.

20. The HRA was undertaken to estimate the potential health risks from the existing and proposed Allied Plants. The predicted health risks show that with the proposed Plant there will be reductions in the potential health risk in the exposed community compared to the existing Plant. These estimates reflect the benefits of the new Allied Plant in the airshed. This is also shown in the air quality assessment.[2]

21. Although the predicted health risk for the proposed Plant it is important to note that the predicted health risks associated with the existing Plant are also below acceptable risk levels.

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[1] Evidence of Dr Emily Wilton, paragraph 25

[2] Air Quality Assessment for Consent Application Allied Asphalt Mount Maunganui, Tonkin & Taylor (2024)

22. Dr Wilton in her evidence states that "*The HRA undertaken by Dr Denison for the applicant considers only the impact of the discharge (existing plant) and proposed discharge (new plant) on health impacts in the MMA. This uses the same risk assessment approach as for the ESR report in that estimates of health impacts are made based on multiplying the CRF by baseline mortality by concentration but adds an additional divided by 100,000 so the unit is estimated health impact per 100,000 people rather than estimated health impact for a specific population.*" This is incorrect.

Health Statistics

23. Mx Wickham in their evidence[3] questioned the use of the Bay of Plenty District Health Board statistics in the HRA. They noted that census area unit (CAU) data was available from the HAPINZ 3.0 study.

24. The HAPINZ 3.0 methodology report identifies the source of the health data used in HAPINZ as unit record data from the New Zealand Mortality Collection (MoH 2021a), extracted by the Ministry of Health in August 2021. Advice from Health New Zealand (Te Whatu Ora) is that the mortality collection data is only available at District Health Board level. Their database only holds District Health Board level data. This is the same data as used in the Allied HRA.

25. The CAU data used in HAPINZ 3.0 was derived by the HAPINZ team from the District Health Board level data. The process used to derive the CAU data is unclear from the HAPINZ documentation.

26. One key difference between HAPINZ 3.0 and the Allied Health Statistics is the form of the health data. In undertaking the Allied HRA I have used the age standardised mortality/morbidity rates, eg deaths per 100,000 population, provided by Te Whatu Ora. The data reported by Te Whatu Ora is presented in this format and is recommended by WHO in their risk assessment guidance[4].

27. The numbers of deaths per 100,000 population are influenced by the age distribution of the population. Two populations with the same age-specific mortality rates for a particular cause of death will have different overall death rates if the age distributions of their populations are different. Age-standardized mortality rates adjust for differences in the age distribution of

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[3] Evidence of Lou Wickham, paragraph 65.

[4] WHO (2016) Health Risk of Air Pollution – General Principles. Health risk assessment of air pollution: general principles (who.int)

the population by applying the observed age-specific mortality rates for each population to a standard population.

28. Almost all diseases or health outcomes occur at different rates in different age groups. Most chronic diseases, including most cancers, occur more often among older people. Other outcomes, such as many types of injuries, occur more often among younger people.

29. The age-standardized mortality rate is a weighted average of the age-specific mortality rates per 100,000 persons, where the weights are the proportions of persons in the corresponding age groups of the WHO standard population[5].

30. Since age structure varies between countries and in the same country over time, this adjustment allows us to see how mortality and morbidity vary without age differences.

31. The data reported by Te Whatu Ora (Health New Zealand) is reported as age standardised rates. The Te Whatu Ora data is only available at District Health Board level.

32. The data analysed for HAPINZ 3.0 were supplied by the Environmental Health (EHINZ) Indicators programme, Centre for Public Health Research, Massey University by Statistics New Zealand and the Ministry of Health. The data sources are the Census Population data, the National Minimum Dataset (Hospital Inpatient Events), the Mortality Collection Data, and the New Zealand Cancer Registry Data[6].

33. According to the EHINZ website the data reported is for age standardised and non-adjusted rates which is only available at District Health Board Level. The source of their data is Te Whatu Ora which only has data at District Health Board Level.

34. The CAU level data has been derived by the HAPINZ team by scaling for the population in each CAU. However, the base data used in HAPINZ 3.0 and subsequently in the ESR Mount Maunganui HRA is DHB level data which is what has been used in the Allied HRA but scaled according to population. HAPINZ 3.0 has converted mortality/morbidity rates to actual

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[5] WHO Age-standardized mortality rate (per 100 000 population) (who.int)

[6] For more information on the data source see http://www.stats.govt.nz/Census.aspx and http://www.health.govt.nz/nz-health-statistics. The Environmental Health Indicators team makes no warranty, express or implied, nor assumes any legal liability or responsibility for the use of this spreadsheet or its contents by any person or organisation.

numbers of deaths/hospital admissions. However, the mortality rates per 100,000 for CAU are the same as that used in the HRA.

35. In my opinion the use of the age standardised mortality/morbidity rate per 100,000 will not change the conclusions of the HRA compared with the use of CAU derived for HAPINZ 3.0 as it is the same source data at the DHB level.

36. Mx Wickham[7] also raised concerns about the use of the 2020 health data in the HRA as it was COVID affected. I extracted the 2019 health data and reran some of the mortality calculations for Allied using this data. The differences in the mortality rates were minimal and did not change the conclusions of the Allied HRA.

37. Although similar methodologies have been used in both the Allied HRA and the ESR HRA one of the key differences is the health statistics used. The Allied HRA used age standardised mortality rates (deaths/100,000) in the calculations where the ESR HRA used numbers of deaths at a CAU level as derived from HAPINZ 3.0.

38. Use of the age standardised rates provides risk estimates per 100,000 as this is baseline health data used. The health risk estimates were not calculated as number of deaths, as per HAPINZ 3.0 and the ESR HRA, and divided by a factor of 100,000 as suggested by Dr Wilton.

39. Although this is a difference in approach between the Allied HRA and the ESR HRA it does not affect the conclusion of the HRA that all predicted risks are below acceptable risk levels.

Concentration response functions

40. Concentration-response functions have been established by epidemiological studies and represent the relationship between the concentration of an air pollutant to which a population is exposed and the change in a health outcome with changes in air pollution levels. As such, concentration-response functions quantify the health impact per concentration unit of air pollutant (WHO, 2016).

41. Mx Wickham's evidence[8] notes that *It is important to note that CRFs are relative (to non-exposure). This means that to understand a CRF, the range of exposure needs to be clearly stated.* A CRF is derived within a given

[7] Evidence of Lou Wickham, paragraph 65.

[8] Evidence Lou Wickham paragraph 58

population and is not a measure of differences between two populations. It is measure of a change in a health outcome with a unit change in air pollutant concentration within the exposed population.

42. In the HRA both the WHO and New Zealand concentration response functions (CRFs) derived for the HAPINZ 3.0 study were used. This was acknowledged in the Health Joint Witness Statement.

43. Mx Wickham provided a discussion on CRFs in their evidence[9] and how they were derived. They importantly acknowledged that the HAPINZ 3.0 CRFs were used for a sensitivity analysis but considered it should be the other way around (i.e., use the WHO CRFs for sensitivity analysis).

44. It is important to note that the order in which the CRFs are used does not affect the outcomes of the HRA. Irrespective of the CRFs used (WHO or HAPINZ 3.0) the incremental increase in risk due to emissions from the existing and proposed Allied Plants are below acceptable risk levels.

45. There are a number of issues raised by Mx Wickham[10] relating to the interpretation of the CRFs and the epidemiological evidence that in my opinion are not correct. Although many do not impact on the conclusions of the HRA for the Allied Consent application, there are several specific issues that warrant a response here. These are discussed in the following paragraphs.

46. Both Mx Wickham and Dr Wilton refer to the CRF for PM_{10} derived in Hales (2021) and that it was used in HAPINZ 3.0. The publication by Hales (2021) does not derive a CRF for PM_{10} only $PM_{2.5}$ and NO_2 (see Attachment 3). The focus of HAPINZ 3.0 was $PM_{2.5}$ and NO_2 as these were the pollutants considered to be of main concern in New Zealand.

47. According to the HAPINZ 3.0 Methodology Report (Volume 2) "*while PM_{10} based on a single pollutant model is a good measure (proxy) for the effects of all air pollution, much of its association with mortality can be explained by $PM_{2.5}$ and NO_2. Consequently, we opted for the two pollutant ($PM_{2.5}$ and NO_2) model mortality risks*".[11] This is important when considering whether the predicted health effects can be added or traded off. This is discussed further at paragraphs 54-62 of this reply evidence.

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[9] Evidence Lou Wickham p66

[10] Evidence Lou Wickham paragraphs 55-61.

[11] HAPINZ 3.0, Volume 2 Methodology, p85

48. Mx Wickham refers to the HAPINZ 3.0 CRF for PM_{10} as applying to PM_{10} only. In my opinion and supported by the conclusion from HAPINZ 3.0 referred to in paragraphs 46 and 47 of this reply evidence, the opposite is true. The one pollutant PM_{10} CRF represents the health effects of exposure to the mixture of pollution that we breathe every day, including the effects of $PM_{2.5}$ and NO_2. It does not represent PM_{10} "alone". Therefore, the use of the one pollutant PM_{10} CRF will overestimate the health effects attributable to PM_{10}.

49. The one pollutant CRF from HAPINZ 3.0 was used in the sensitivity analysis in the HRA. The predicted risk estimates using this CRF reflect, to a large extent, the effects of $PM_{2.5}$ and NO_2 and is an overestimate of the effects attributable to PM_{10}. The predicted risks are still below the acceptable risk criteria.

50. Dr Wilton (paragraph 36 of her evidence) notes that there are "*uncertainties in using a CRF from a two-pollutant model when considering individual pollutant impacts. The two-pollutant model used for New Zealand integrates contaminants which have different exposure classification methods"*. A two-pollutant model does not integrate the effects of contaminants but attempts to separate out the individual pollutant effects.

51. In a two-pollutant regression model, the relationship between the health effect and the pollutant of interest (for example, NO_2) is estimated while the other pollutant (for example, $PM_{2.5}$) is held constant in an attempt to identify any independent effect of NO_2 itself. This is particularly important when the pollutants are from the same sources and are highly correlated[12].

52. For the HRA the New Zealand CRFs for $PM_{2.5}$ and NO_2 from two pollutant models were used in in the sensitivity analysis. This is consistent with HAPINZ 3.0 and limits, so far as possible, double counting of health effects.

53. In summary, with respect to the HRA for the Allied Consent Application, while there are some differences in opinion between the health experts regarding the interpretation of the epidemiological data and the CRFs from these studies, neither Mx Wickham or Dr Wilton disagreed with the CRFs used and noted that the New Zealand (HAPINZ 3.0) CRFs had been used. The incremental risks from the Allied Asphalt Plants, existing and proposed, were predicted to be below acceptable risk levels regardless of which CRFs are used.

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[12] $PM_{2.5}$ and NO_2 are usually highly correlated as they are both from combustion sources.

Trade-offs of health effects for different pollutants

54. Mx Wickham and Dr Wilton both raised the issue of trading off the impacts of the individual pollutants in particular PM_{10}, $PM_{2.5}$, NO_2 and SO_2. This was discussed in the Health Expert Conferencing and was raised in their evidence.

55. As discussed above the pollutants are highly correlated and the CRFs for each pollutant incorporates to some extent the effects of the other pollutants. Aggregation of these risk together into a singular estimate is inappropriate without a method to control for the inherent correlation between them. It will lead to a significant overestimate of the attributed health effects.

56. This is acknowledged to some extent in Dr Wilton's evidence[13] "*There are difficulties in making comparisons between health impacts of different pollutants and in evaluating whether improvements in concentrations of one contaminant will offset increases in concentrations of another. This is because impacts occur because of a pollutant mix."*

57. As noted in HAPINZ 3.0 Methodology Report (Volume 2) the one pollutant PM_{10} model CRF is a good measure (proxy) for the effects of all air pollution. Much of its association with mortality can be explained by $PM_{2.5}$ and NO_2. Therefore, the predicted health effects attributed to PM_{10} already contain the health effects of $PM_{2.5}$ and NO_2. Adding the predicted health effects from all pollutants together for each scenario assessed in the HRA will double count the effects of both $PM_{2.5}$ and NO_2.

58. The use of the WHO CRFs limits that to some extent as they are derived from multiple studies from around the world where the correlations between the pollutants will differ.

59. According to the Committee on the Medical Effects of Air Pollution (COMEAP) in the UK, even when attempting to control for other pollutants in two pollutant models, there is some residual contribution from the other pollutants.

60. Table 1 below is a Table from COMEAP (2018) that shows the potential contribution of NO_2 to $PM_{2.5}$ health effects using CRFs from both one and two pollutant models.

[13] Evidence of Dr Emily Wilton Paragraph 45.

Table 1: Types of coefficients that might be used to represent associations between long-term average concentrations of $PM_{2.5}$ and NO_2 and mortality, and their possible interpretations[14].

Coefficient	Possible Interpretation
Unadjusted coefficient for PM2.5	Reflects the effect of $PM_{2.5}$ and, to some extent, the effect of other pollutants with which $PM_{2.5}$ is correlated. These include other fractions of PM, NO_2 and other components of the air pollution mixture
Unadjusted coefficient for PM2.5	Reflects any causal effect of NO_2 and, to some extent, the effects of other pollutants with which NO_2 is correlated. These include $PM_{2.5}$, other fractions of PM, and other components of the air pollution mixture (e.g., ultrafine particles, Black Carbon, Volatile Organic Compounds, etc.)
Coefficient for PM2.5 adjusted for NO2	Reflects the effect of $PM_{2.5}$ and, to some extent, the effects of other pollutants with which $PM_{2.5}$ is most closely correlated but excludes (as far as possible) effects associated with NO_2, and other components of the air pollution mixture which are more closely correlated with NO_2 concentrations than with $PM_{2.5}$ concentrations. Given the good evidence and plausibility of causality, it is reasonable to regard most of this effect as likely to be causally correlated to $PM_{2.5}$.
Coefficient for NO2 adjusted for PM2.5	Reflects any effect of NO_2 and, to some extent, other pollutants with which NO_2 is closely correlated but excludes (as far as possible) effects associated with $PM_{2.5}$ concentrations and other components of the air pollution mixture that are more closely correlated with $PM_{2.5}$ concentrations than with NO_2 concentrations. Given the weaker evidence for plausibility and causality, the extent to which this effect is likely to be causally related to NO_2 is unclear. It is unlikely to be zero, but also unlikely to be 100%.

[14] Source Committee on the Medical Effects of Air Pollution (COMEAP) (2018) available at Associations of long term average concentrations of nitrogen dioxide with mortality (publishing.service.gov.uk)

61. The data shown in Table 1 indicates that even when controlling for the other pollutant, in particular controlling for $PM_{2.5}$ in estimating the NO_2 CRF, part of that relationship is still due to $PM_{2.5}$.

62. The proposed new Allied Plant leads to a reduction in incremental health risks for PM_{10}, $PM_{2.5}$ and SO_2 compared to the existing plant For NO_2 there is no change in risk between the existing and proposed Plants when the new Plant is running on natural gas. There is a minimal increase with the proposed Plant running on diesel – the increase in risk is $0.1x10^{-5}$ for long-term mortality.

Benzene and Dioxins

63. Dr Wilton in her evidence[15] commented that the health risks of benzene and dioxins should be assessed. The health effects of benzene were assessed in the HRA Sections 4.7.1 and 4.7.2. They are also presented in my primary evidence paragraphs 73-87 and in Tables 6 and 7.

64. All risks associated with benzene were below acceptable risk levels.

65. Dioxins were not assessed in the HRA as the air quality assessment (Tonkin & Taylor, 2024) showed that the predicted ground level concentrations were very low (0.00018% of the OEHHA chronic REL).

66. To address the issue raised by Dr Wilton I have calculated the incremental cancer risk due to dioxin emissions from the existing and proposed Plants. The incremental cancer risk estimates for the most impacted residential receptors are shown in Table 2.

67. The approach to calculating the cancer risks is presented in paragraphs 78-82 of my primary evidence.

Table 2 Carcinogenic Risks from Dioxin emissions from the Existing and Proposed Allied Plants

Receptor	Incremental Cancer risk	
	Existing Plant (ULO)	Proposed Plant (NG/Diesel)
Whareroa Marae	$1x10^{-10}$	$3x10^{-10}$
Most affected receptor Maunganui Road	$1x10^{-9}$	$2x10^{-9}$
Mt Maunganui Intermediate	$2x10^{-11}$	$5x10^{-11}$

[15] Evidence Dr Emily Wilton paragraph 47.

Receptor	Incremental Cancer risk	
	Existing Plant (ULO)	Proposed Plant (NG/Diesel)
Gwen Rogers Kindergarten	$2x10^{-10}$	$4x10^{-10}$
Omanu Primary	$1x10^{-10}$	$2x10^{-10}$
Little Einsteins	$1x10^{-10}$	$2x10^{-10}$
Best Start MacDonald St	$1x10^{-10}$	$2x10^{-10}$
Most affected receptor DeHavilland Way	$1x10^{-9}$	$2x10^{-9}$
Mt Maunganui college	$2x10^{-10}$	3E-10
Most affected receptor Aerodrome Road	$2x10^{-9}$	$3x10^{-9}$
Most affected receptor Dakota Way	$2x10^{-9}$	$2x10^{-9}$

68. The results in Table 2 show that at all locations the predicted cancer risks attributable to dioxins from the existing and proposed Allied Plants for adults and children are below the negligible risk criterion of $1x10^{-6}$ by 3-5 orders of magnitude.

Choice of Unit Risk Factor for Carcinogenic Risk Assessment

69. During the health caucusing there was discussion on the selection of the unit risk factors for carcinogens in the HRA. I provided a table that compared the unit risk factors (URFs) from OEHHA and the US EPA which showed the OEHHA values were typically more up to date and health protective. It was agreed that I add a rationale for the use of OEHHA[16] risk factors, including the table presented at the caucusing, in my reply evidence.

70. As described in paragraph 78 of my primary evidence, a URF is the increase in risk of cancer due to a 1 $\mu g/m^3$ increase in pollutant concentration in air.

71. There are limited sources for URFs, and each agency uses different methodologies in their derivation. Therefore, it is important to choose as many URFs for the same source as possible to ensure consistency for differing pollutants in their derivation.

72. The main sources for URFs are OEHHA and the US EPA. Both sources are commonly used in New Zealand.

[16] Office of Environmental Health Hazard Assessment, a Division of the Californian EPA

73. In determining the relevant URFs for use in the Allied HRA, I reviewed the current URFs published by each agency to determine the most recently reviewed values to ensure that they represent the most recent health data for each pollutant. The comparison of the values from both agencies for each pollutant being assessed in the Allied HRA are shown in Table 3.

Table 3: Comparison of Unit Risk Factors OEHHA and US EPA

Pollutant	URF OEHHA	URF US EPA	Notes	Year of Review
BaP	0.0011	0.0006	USEPA used Bench Mark Dose approach - assumes 10% increase in cancer OEHHA – linear low dose extrapolation	OEHHA 2009; USEPA 2017
Benzene	0.000029	0.0000022 to 0.0000078		OEHHA 2009; USEPA 1995
Formaldehyde	0.000006	0.000013	OEHHA - known human carcinogen - linear no threshold effect US EPA – probable human carcinogen – is based on residual risk - assumes threshold and only applies above that threshold	OEHHA 2011 USEPA 1991
Arsenic	0.0033	0.0043		OEHHA 2009; USEPA 1995
Cadmium	0.0042	0.0018	USEPA assumes two stage carcinogenicity - probable human carcinogen; OEHHA known human carcinogen – genotoxic	OEHHA 2009; USEPA 1985

Pollutant	URF OEHHA	URF US EPA	Notes	Year of Review
			carcinogen no threshold	
Nickel	0.00026	no URF	OEHHA consistent with IARC classifications; not assessed under IRIS program	OEHHA 2004
Chromium VI	0.15	0.012		OEHHA 2011; USEPA 1998
Beryllium	0.0024	0.0024		OEHHA 2009; USEPA 1998
Dioxins (TCDD)	38	No URF	not assessed under IRIS program	OEHHA 2011; USEPA 2012

74. The information in Table 3, shows that the OEHHA URFs are, in general, more recent than the US EPA values. In addition, they are more conservative in most cases and are available for all the pollutants being considered in the Allied HRA.

75. Based on this information I used the OEHHA URFs, as discussed in paragraph 78 of my primary evidence, in calculating the carcinogenic risks associated with the emissions from the existing and proposed Allied Plants.

76. Neither Mx Wickham nor Dr Wilton disagreed with the use of these values.

Acceptable risk criteria

77. Mx Wickham in their evidence[17] questions the use of the international acceptable risk criteria in the HRA and recommends the more stringent value of 1 in a million incremental cancer risk. This is inconsistent with international guidance from agencies such as the WHO and US EPA and New Zealand guidance for other environmental media such as contaminated land.

78. The acceptable risk criteria used in the HRA are described in paragraphs 53-54 of my primary evidence.

[17] Evidence Mx Lou Wickham, paragraphs 69-70

79. According to the US EPA an acceptable exposure/risk level is the "concentration level of a contaminant to which the human population, including sensitive subgroups, may be exposed without adverse effect during a lifetime or part of a lifetime..." For known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent lifetime cancer risk to an individual of between 10^{-4} (1 in 10,000) and 10^{-6} (1 in 1,000,000).

80. In assessing excess cancer risk the US EPA has established the following definition - *The additional risk of cancer from exposure to a contaminant beyond an individual's risk of cancer from everyday life. Excess cancer risk is described in terms of the probability that an exposed individual will develop cancer because of that exposure by age 70. In general, EPA considers excess cancer risks that are below about 1 chance in 1,000,000 (1×10-6) to be so small as to be negligible, and risks above 1 in 1,000 (1×10-4) to be sufficiently large that some sort of remediation is desirable. Excess cancer risks that range between 10-6 and 10-4 are generally considered to be "acceptable".*

81. The Ministry of Primary Industries[18] states that *'Acceptable' intakes of non-threshold contaminants are based on intakes over a lifetime that leading to an acceptable increased likelihood of cancer. An 'Acceptable risk level' is used to define the acceptable increased risk, with an acceptable increased risk level of 1 in 100,000 used in New Zealand.*

82. This position is consistent with the Ministry for the Environment guidelines for contaminated soil.

83. Use of the more stringent 1 in a million incremental risk criteria is inconsistent with other NZ guidance and international guidance. In my opinion this is not justifiable and would mean that air quality is treated differently to other environmental media in New Zealand.

84. It is important to note that all predicted cancer risks are below the 1 in a million criteria. All predicted risks for all pollutants are below the 1 in 100,000 criteria used more broadly in New Zealand and internationally. This means that all potential risks are below acceptable risk criteria.

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[18] Ministry of Primary Industries, Working towards New Zealand risk-based soil guideline values for the management of cadmium accumulation on productive land Contract: LC 965 MPI Technical Paper No: 2012/06, p3

85. Mx Wickham in their evidence[19] questions the use of the acceptable risk criteria used in the hazard quotients for metals and VOCs. The main issue is that background data is not available and therefore the total risk cannot be assessed.

86. The approach used to calculate the HQs for the Allied Plants is described in paragraphs 85-86 of my primary evidence.

87. While I acknowledge the point Mx Wickham makes, all hazard quotients for all metals and VOCs assessed are several orders of magnitude below the acceptable hazard quotient of 1. This means even if the measured background was at the public health criteria used in the assessment the addition of the incremental risk from the Allied Plants would not affect the overall risk.

88. In my opinion the potential non-carcinogenic risks assessed for the existing and propped Allied Plants using the HQ approach are well below the negligible risk criteria of 0.1 and even with the inclusion of background, if it was available, would not change that conclusion.

Cumulative assessment

89. The changes in emissions and associated health risks attributable to the existing and proposed Allied Plants being considered in this consent application are independent of emissions to the airshed from other industries. The cumulative effects of emissions on the airshed are addressed in AQ assessment and the evidence of Ms Simpson.

90. Dr Wilton in her evidence[20] states that because the approach taken in the Allied assessment does not consider the cumulative impacts the risks presented seem very small. I disagree with this statement. The risk from Allied is the same irrespective of what happens in the rest of the airshed. Emissions from other sources in the airshed do not impact on the risk arising attributable to Allied.

91. Dr Wilton[21] further notes that "*Advantages of the Denison approach are that it enables the impact of spatial variability in concentrations of contaminants from the discharge to be assessed*." This was the intent of the HRA to

[19] Evidence Mx Lou Wickham, paragraph 73

[20] Evidence Dr Emily Wilton, paragraph 28

[21] Evidence Dr Emily Wilton, paragraph 31

assess the risks associated with emissions from the existing and proposed Allied Plants to inform decisions in relation to the consent application.

Assessment of Risk for non-continuous Plant operation

92. The HRA has assessed the potential health risk for the existing and proposed Allied Plants operating continuously at maximum production capacity. This is not a realistic operational scenario and presents the worst-case impacts.

93. To provide a more 'realistic' estimate of potential impacts the air dispersion model results having been adjusted to account for the existing and proposed plants not operating continuously as described in Ms Simpson's reply evidence. For the proposed plant this represents a more realistic estimate of the effects if the plant were to operate at the maximum consented annual production of 300,000 TPA.

94. As discussed in Ms Simpson's reply evidence, it is important to note that for the proposed plant these estimates represent the maximum envelope of effects that would be authorised by the consent being sought. In this sense they are still highly conservative rather than "realistic" because, as explained in Mr Palmer's evidence, annual production is expected to increase incrementally with demand. Therefore, for most of the term of the consent the effects will be much lower.

95. I have undertaken the risk calculations using the adjustment factors referred to in Ms Simpson's reply evidence. The risk estimates for the most affected residential receptor are shown in Tables 4 to 6.

Table 4: Adjusted Incremental Risk Estimates for non-continuous operational scenario

Receptor	Pollutant	Health Outcome	Increase in risk due to PM_{10} , $PM_{2.5}$ and NO_2		
			Existing Plant	Proposed Plant	
Most affected residential receptor	PM_{10}	Long-term mortality 30 + years (all cause non-accidental)	$0.08x10^{-5}$	$0.02x10^{-5}$	
	$PM_{2.5}$	Long-term mortality 30 + years (all cause non-accidental)	$0.08x10^{-5}$	$0.05x10^{-5}$	
	NO_2	Long-term mortality 30 + years (all cause non-accidental)	$0.01x10^{-5}$	Diesel $0.03x10^{-5}$	Natural Gas $0.02x10^{-5}$

Table 5: Adjusted Incremental Cancer Risk Estimates for non-continuous operational scenario

Receptor	Pollutant	Incremental lifetime cancer risk	
		Existing Plant	Proposed Plant
		ULO	Diesel/Natural Gas
Most affected residential receptor	Arsenic	$2x10^{-7}$	$3x10^{-9}$
	Cadmium	$8x10^{-8}$	$6x10^{-9}$
	Chromium VI	$2x10^{-7}$	$2x10^{-8}$
	Benzene	$8x10^{-9}$ *$8x10^{-8}$*	$2x10^{-8}$ *$2x10^{-7}$*
	Formaldehyde	$1x10^{-8}$	$1x10^{-8}$
	BaP	$1x10^{-10}$ *$1x10^{-9}$*	$8x10^{-12}$ *$8x10^{-11}$*
	Lead	$6x10^{-9}$	$2x10^{-11}$

Table 6: Adjusted Incremental Hazard Quotients for non-continuous operational scenario

Receptor	Pollutant	Chronic Hazard Quotients		Acute Hazard Quotients	
		Existing	Proposed	Existing	Proposed
		Used Oil	Diesel/Natural Gas	Used Oil	Diesel/Natural Gas
Most affected residential receptor	Arsenic	0.003	0.00007	0.004	0.0001
	Cadmium	0.001	0.00007	na	na
	Chromium VI	0.000006	0.0000005	na	na
	Benzene	0.00009	0.0002	0.00004	0.00002
	Formaldehyde	0.0002	0.0002	0.0007	0.0006
	Chromium III	0.0008	0.00002	0.002	0.00005
	Copper	na	na	0.00009	0.000001
	Lead	na	na	0.003	0.00001

96. The risk estimates shown in Tables 4 to 6 are approximately an order of magnitude lower than those predicted for the continuous operation scenario assessed in the HRA.

97. The risks for all other receptors are lower than those shown in Tables 4 to 6 for the most affected residential receptor.

98. All risks for all health outcomes are well below the acceptable and negligible risk criteria for both the existing and proposed Allied Plants.

Risk assessment for workers on adjacent industrial locations.

99. During the health expert caucusing Dr Wilton and Mx Wickham raised the issue of the potential impacts of the Allied emissions on adjacent industrial workers. There was discussion around the appropriate criteria to be applied in the assessment and it was agreed that I would undertake an assessment of the potential risks and include in this reply evidence.

100. Typically, the WorkSafe New Zealand workplace exposure standards (WES) are used to assess the risk to worker health. They apply to the workplace where the emissions are produced and are not typically used to assess other industrial sites that are adjacent to source.

101. Mx Wickham and Dr Wilton were of the view that the ambient air quality standards/guidelines should be applied at the adjacent sites. In my opinion it is not appropriate to apply the ambient air quality standards/guidelines as they have been derived to protect sensitive subgroups within the population namely children and people over the age of 65 years. Neither of these sensitive groups are not present within industrial workplaces.

102. Another reason that, in my opinion, the ambient air quality standards/guidelines do not apply in an industrial setting is that the ambient air quality guidelines have been set assuming exposure of the exposed population for 24 hours per day, 7 days per week, 365 days per year. This is a conservative assumption that provides an additional level of protection for sensitive groups within the population.

103. Workplace exposures are assumed to be 8 hours per day, 5 days per week, 240 days per year. This is a much lower level of exposure than that assumed in the derivation of the ambient air quality guidelines. As exposure is directly related to the risk this means that the application of ambient air quality guidelines in an industrial location is overly conservative and does not represent the risk to worker health

104. In the health expert caucusing the application of the same methodology used in the community health risk assessment was discussed. Post the caucusing and joint witness statement I have reviewed the information required to undertake such an assessment and have concluded that this is not possible.

105. There are several reasons for this conclusion. The first is that the CRFs used in the assessment have been derived from epidemiological studies

where entire populations have been exposed to the pollutants of concern, e.g. PM_{10}, NO_2 etc. These populations include sensitive groups and therefore reflect the response of these groups. These groups, such as children and people over 65 years of age, are not typically present on industrial sites.

106. A second reason is that the available mortality data is for the total population over the age of 30 years. This does not reflect the working population, ages 15 – 64 years, alone. The mortality data is strongly influenced by people in the older age groups – 65+ years – as the mortality rates are higher in this age group. Therefore, the baseline health data currently available will overestimate the risk to the worker population.

107. As discussed above the exposure of people in a workplace differs from the general population. The epidemiological studies that have been conducted to derive the CRFs use daily 24 hour and annual average data for every day of the year. These exposure scenarios are not applicable in an industrial setting where the emissions from the site in many cases are only generated for a limited period when the Plant is in operation. This is certainly the case for the existing and proposed Allied Plants. This is another reason why using the CRFs for the general population are not appropriate in an industrial setting.

108. According to WorkSafe NZ[22] WES are guidance values provided by WorkSafe that refer to the airborne concentration of substances at which it is believed that nearly all workers can be repeatedly exposed day after day without coming to harm. The WES are intended to be used as guidelines for health risk management.

109. I acknowledge that the use of the WES to assess the potential risk to adjacent industrial sites is not consistent with the application of the WES intended in the Workplace legislation, however in the absence of other applicable guidelines, I believe they can be used to provide an assessment of potential risk in the adjacent industrial sites.

110. In the assessment of risk to workers in adjacent sites I have not applied them as compliance standards but as levels that may pose a risk to workers in an occupational setting. As such, they can be used as a guide to screen the potential risk posed to the workers from exposure to emissions from the Allied Plant.

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[22] Regulations, legal requirements and rights | WorkSafe

111. Table 7 below shows the hazard quotient (HQ) for the maximum offsite concentration within the industrial area. In these calculations the WES 8-hour time weighted average (TWA) for all pollutants apart from SO_2 have been used. For SO_2 the 15 min short term exposure limit (STEL) has been used.

Table 7: Hazard Quotients for Most Affected Industrial Receptor – Continuous Operation and more realistic scenario.

Receptor	Pollutant	Hazard Quotients	
		Existing	Proposed
		Used Oil	Diesel/Natural Gas
Most affected industrial receptor	PM_{10}	0.01	0.001
	$PM_{2.5}$	0.01	0.001
	SO_2		
	NO_2	0.03	0.02 (D)/0.009 (NG)
	Arsenic	0.08	0.0003
	Cadmium	0.01	0.0001
	Chromium VI	0.1	0.002
	Benzene	0.003	0.001
	BaP	0.000001	0.00000001
	Lead	0.02	0.00001

112. As discussed in paragraph 85 of my primary evidence a HQ of 1 is considered an acceptable risk and 0.1 a negligible risk. All the HQs shown in Table 7 are below the acceptable risk level and for most below the negligible risk level. The HQs for the proposed Plant are lower than those for the existing Plant.

113. For Chromium VI the HQ is 0.1 for the existing Plant – the negligible risk level. For the proposed Plant the HQ is 2 orders of magnitude lower. I understand from Ms Simpson that the estimated concentration of chromium VI is a conservative estimate based on AP42 emission factors. Laboratory analysis of the used oil showed that for all, but one sample analysed total chromium, which includes chromium VI, were below detectable limits. Therefore, the use of the AP42 factors is likely to overestimate the chromium VI emissions from the use of ULO for the existing Plant.

114. In addition to comparison with the WES I have also calculated the incremental cancer risk. For this calculation I have applied the same approach used in the HRA for the community receptors adjusted for the worker exposure scenario. These results are shown in Table 8 for both the continuous operation of the Plants as well as the more realistic non-continuous scenario described in the evidence of Ms Simpson.

Table 8: Incremental Carcinogenic Risks for Most Impacted Industrial Receptor

Receptor	Pollutant	Incremental lifetime cancer risk	
		Existing Plant	Proposed Plant
		ULO	Diesel/Natural Gas
Most impacted industrial receptor – continuous operation	Arsenic	$5x10^{-7}$	$2x10^{-9}$
	Cadmium	$2x10^{-7}$	$3x10^{-9}$
	Chromium VI	$6x10^{-7}$	$9x10^{-9}$
	Benzene	$2x10^{-8}$	$1x10^{-8}$
	BaP	$4x10^{-10}$	$4x10^{-12}$
	Lead	$2x10^{-8}$	$1x10^{-11}$
Most impacted industrial receptor – more realistic scenario	Arsenic	$5x10^{-8}$	$3x10^{-10}$
	Cadmium	$2x10^{-8}$	$5x10^{-10}$
	Chromium VI	$5x10^{-8}$	$2x10^{-9}$
	Benzene	$2x10^{-9}$	$2x10^{-9}$
	BaP	$4x10^{-11}$	$7x10^{-13}$
	Lead	$2x10^{-9}$	$2x10^{-12}$

115. The results shown in Table 8 show that for both the continuous operation and more realistic scenarios all cancer risks are below the acceptable risk criteria of $1x10^{-5}$ and the negligible risk criteria of $1x10^{-6}$.

116. Based on the results shown in Tables 4 and 5 above it is my opinion that the emissions from the existing and proposed Allied Plants pose a negligible risk to the workers at adjacent industrial locations.

Assessment of Risk to Residential Receptors on Aerodrome Road and Dakota Way.

117. In the Joint Witness Statement of the Air Quality Experts, Mx Wickham identified additional receptors that needed to be assessed. These are hangars located in the Airport precinct where people can stay for a period of time. These receptors are on Dakota Way and Kittyhawk Way.

118. These receptors have been modelled by Ms Simpson and are discussed in her reply evidence.

119. In addition to these receptors, it was identified that there are hangars on Aerodrome Road that can also be used for residential use. These have also been assessed in the reply evidence of Ms Simpson.

120. I have undertaken the health risk calculations for the most affected receptors which include Dakota Way and Aerodrome Road. The results

are presented in Tables 9 – 11. Results have been presented for continuous and non-continuous exposure scenarios.

121. As there are no known restrictions on who can live in these locations or for how long, I have assessed the risk to these residents as for other residential locations.

Table 9: Incremental Risks for PM_{10}, $PM_{2.5}$ and NO_2 Dakota Way and Aerodrome Road

Receptor	Pollutant	Health Outcome	Increase in risk due to PM_{10} - continuous operation		
			Existing Plant	Proposed Plant	
Most affected residential receptor Dakota Way	PM_{10}	Long-term mortality 30 + years (all cause non-accidental)	$1x10^{-5}$	$0.2x10^{-5}$	
	$PM_{2.5}$	Long-term mortality 30 + years (all cause non-accidental)	$1x10^{-5}$	$0.07x10^{-5}$	
	NO_2	Long-term mortality 30 + years (all cause non-accidental)	$0.2x10^{-5}$	Diesel	Natural Gas
				$0.2x10^{-5}$	$0.1x10^{-5}$
Most affected residential receptor Aerodrome Road	PM_{10}	Long-term mortality 30 + years (all cause non-accidental)	$1x10^{-5}$	$0.2x10^{-5}$	
	$PM_{2.5}$	Long-term mortality 30 + years (all cause non-accidental)	$1x10^{-5}$	$0.1x10^{-5}$	
	NO_2	Long-term mortality 30 + years (all cause non-accidental)	$0.2x10^{-5}$	Diesel	Natural Gas
				$0.3x10^{-5}$	$0.2x10^{-5}$
Receptor	**Pollutant**	**Health Outcome**	**Increase in risk - non-continuous operation**		
			Existing Plant	**Proposed Plant**	
Most affected residential receptor Dakota Way	PM_{10}	Long-term mortality 30 + years (all cause non-accidental)	$0.1x10^{-5}$	$0.01x10^{-5}$	
	$PM_{2.5}$	Long-term mortality 30 + years (all cause non-accidental)	$0.1x10^{-5}$	$0.02x10^{-5}$	
	NO_2	Long-term mortality 30 +	$0.02x10^{-5}$	Diesel	Natural Gas
				$0.04x10^{-5}$	$0.02x10^{-5}$

Receptor	Pollutant	Health Outcome	Increase in risk due to PM_{10} - continuous operation	
			Existing Plant	Proposed Plant
		years (all cause non-accidental)		
Most affected residential receptor Aerodrome Road	PM_{10}	Long-term mortality 30 + years (all cause non-accidental)	$0.1x10^{-5}$	$0.01x10^{-5}$
	$PM_{2.5}$	Long-term mortality 30 + years (all cause non-accidental)	$0.1x10^{-5}$	$0.02x10^{-5}$
	NO_2	Long-term mortality 30 + years (all cause non-accidental)	$0.02x10^{-5}$	Diesel: $0.05x10^{-5}$ / Natural Gas: $0.03x10^{-5}$

Table 10: Incremental Cancer Risks Dakota Way and Aerodrome Road

Receptor	Pollutant		
		Existing Plant	Proposed Plant
		ULO	Diesel/Natural Gas
Dakota Way	Arsenic	$2x10^{-6}$	$2x10^{-8}$
	Cadmium	$1x10^{-6}$	$3x10^{-8}$
	Chromium VI	$3x10^{-6}$	$9x10^{-8}$
	Benzene	$1x10^{-7}$	$1x10^{-7}$
		$1x10^{-6}$	*$1x10^{-6}$*
	Formaldehyde	$2x10^{-7}$	$6x10^{-8}$
	BaP	$2x10^{-9}$	$4x10^{-11}$
		$2x10^{-8}$	*$4x10^{-10}$*
	Lead	$8x10^{-8}$	$1x10^{-10}$
Aerodrome Road	Arsenic	$2x10^{-6}$	$4x10^{-8}$
	Cadmium	$1x10^{-6}$	$4x10^{-8}$
	Chromium VI	$3x10^{-6}$	$2x10^{-8}$
	Benzene	$1x10^{-7}$	$1x10^{-7}$
		$1x10^{-6}$	*$1x10^{-6}$*
	Formaldehyde	$2x10^{-7}$	$9x10^{-8}$
	BaP	$2x10^{-9}$	$6x10^{-11}$
		$2x10^{-8}$	*$6x10^{-10}$*
	Lead	$8x10^{-8}$	$2x10^{-10}$

Receptor	Pollutant		
		Existing Plant	Proposed Plant
		ULO	Diesel/Natural Gas
Receptor	**Pollutant**	**Incremental lifetime cancer risk – non-continuous Operation**	
		Existing Plant	**Proposed Plant**
		ULO	**Diesel/Natural Gas**
Dakota Way	Arsenic	$2x10^{-7}$	$4x10^{-9}$
	Cadmium	$1x10^{-7}$	$6x10^{-9}$
	Chromium VI	$2x10^{-7}$	$2x10^{-8}$
	Benzene	$1x10^{-8}$	$2x10^{-8}$
		$1x10^{-7}$	*$2x10^{-7}$*
	Formaldehyde	$2x10^{-7}$	$6x10^{-8}$
	BaP	$2x10^{-10}$	$8x10^{-12}$
		$2x10^{-9}$	*$8x10^{-11}$*
	Lead	$8x10^{-9}$	$2x10^{-11}$
Aerodrome Road	Arsenic	$2x10^{-7}$	$4x10^{-9}$
	Cadmium	$1x10^{-7}$	$7x10^{-9}$
	Chromium VI	$3x10^{-7}$	$2x10^{-8}$
	Benzene	$1x10^{-8}$	$2x10^{-8}$
		$1x10^{-7}$	*$2x10^{-7}$*
	Formaldehyde	$2x10^{-8}$	$1x10^{-8}$
	BaP	$2x10^{-10}$	$1x10^{-11}$
		$2x10^{-9}$	*$1x10^{-10}$*
	Lead	$8x10^{-9}$	$3x10^{-11}$

Table 11: Chronic Hazard Quotients Dakota Way and Aerodrome Road

Receptor	Pollutant	Chronic Hazard Quotients – Continuous Operation		Chronic Hazard Quotients – non Continuous Operation	
		Existing	Proposed	Existing	Proposed
		Used Oil	Diesel/Natural Gas	Used Oil	Diesel/Natural Gas
Most affected residential receptor Dakota Way	Arsenic	0.04	0.0004	0.004	0.00007
	Cadmium	0.01	0.0004	0.0001	0.00007
	Chromium VI	0.0001	0.000003	0.000008	0.0000005
	Benzene	0.001	0.001	0.0001	0.0002
	Formaldehyde	0.003	0.001	0.0003	0.0001
Receptor	**Pollutant**	**Chronic Hazard Quotients – Continuous Operation**		**Chronic Hazard Quotients – non Continuous Operation**	
		Existing	**Proposed**	**Existing**	**Proposed**

Receptor	Pollutant	Chronic Hazard Quotients – Continuous Operation		Chronic Hazard Quotients – non Continuous Operation	
		Existing	Proposed	Existing	Proposed
		Used Oil	Diesel/Natural Gas	Used Oil	Diesel/Natural Gas
		Used Oil	Diesel/Natural Gas	Used Oil	Diesel/Natural Gas
Most affected residential receptor Aerodrome Road	Arsenic	0.04	0.0005	0.004	0.00009
	Cadmium	0.01	0.0005	0.002	0.00009
	Chromium VI	0.0001	0.000004	0.000008	0.0000007
	Benzene	0.001	0.001	0.0001	0.0003
	Formaldehyde	0.003	0.001	0.0003	0.0002

122. The data presented in Tables 9 – 12 show that all risks are below the acceptable risk levels for both the existing and proposed Allied Plants. The risks are lower for the proposed Plant and the non-continuous operation scenario.

Conclusion

123. I have reconsidered the overall conclusions I set out in my primary evidence considering the matters raised in the evidence of other witnesses and my responses to those matters as set out in this reply evidence.

124. My opinions and conclusions have not changed and the Allied proposal, for both the existing and proposed Plants, do not pose an unacceptable risk to the health of the surrounding community or workers within the Mount Maunganui industrial area.

125. All health risks assessed are below acceptable risk levels established by international agencies such as the WHO and US EPA as well as in New Zealand.

Dr Lynette Denison

Dated this 26th day of April 2024

List of Attachments:

1. List of Publications Dr Lyn Denison
2. Corrected Cancer Risk and Hazard Quotient Tables
3. Publication Hales et al (2021)

Attachment One: Relevant Publications of Dr Lyn Denison

1. Veivers D, Williams GM, Toelle BG, Waterman AMC, Guo Y, Denison L, Yang BY, Dong GH, Jalaludin B, Marks GB, Knibbs LD. The Indoor Environment and Otitis Media among Australian Children: A National Cross-Sectional Study. Int J Environ Res Public Health. 2022 Jan 29;19(3):1551. doi: 10.3390/ijerph19031551. PMID:35162576; PMCID: PMC8835613.
2. Li S, Baker PJ, Jalaludin BB, Marks GB, Denison LS, Williams GM. Ambient temperature and lung function in children with asthma in Australia. Eur RespirJ. 2014 Apr;43(4):1059-66. doi: 10.1183/09031936.00079313. Epub 2013 Dec 5.P MID: 24311765.
3. Tu Y, Williams GM, Cortés de Waterman AM, Toelle BG, Guo Y, Denison L, Babu GR, Yang BY, Dong GH, Jalaludin B, Marks GB, Knibbs LD. A national cross-sectional study of exposure to outdoor nitrogen dioxide and aeroallergen sensitization in Australian children aged 7-11 years. Environ Pollut. 2021 Feb 15;271:116330. doi: 10.1016/j.envpol.2020.116330. Epub 2020 Dec 16. PMID:33383426.
4. Li S, Baker PJ, Jalaludin BB, Guo Y, Marks GB, Denison LS, Williams GM. Are children's asthmatic symptoms related to ambient temperature? A panel study in Australia. Environ Res. 2014 Aug;133:239-45. doi: 10.1016/j.envres.2014.05.032. Epub 2014 Jun 28. PMID: 24981821.
5. Li S, Baker PJ, Jalaludin BB, Guo Y, Marks GB, Denison LS, Williams GM. an Australian national panel study of diurnal temperature range and children's respiratory health. Ann Allergy Asthma Immunol. 2014 Apr;112(4):348-53.e1-8. doi: 10.1016/j.anai.2014.01.007. Epub 2014 Jan 31. PMID: 24485873.
6. Knibbs LD, Cortés de Waterman AM, Toelle BG, Guo Y, Denison L, Jalaludin B,- Marks GB, Williams GM. The Australian Child Health and Air Pollution Study (ACHAPS): A national population-based cross-sectional study of long-term exposure to outdoor air pollution, asthma, and lung function. Environ Int. 2018 Nov;120:394-403. doi: 10.1016/j.envint.2018.08.025. Epub 2018 Aug 17. PMID:30125857.
7. Hinwood AL, Rodriguez C, Runnion T, Farrar D, Murray F, Horton A, Glass D, Sheppeard V, Edwards JW, Denison L, Whitworth T, Eiser C, Bulsara M, Gillett RW, Powell J, Lawson S, Weeks I, Galbally I. Risk factors for increased BTEX exposure in four Australian cities. Chemosphere. 2007 Jan;66(3):533-41. doi:10.1016/j.chemosphere.2006.05.040. Epub 2006 Jul 11. PMID: 16837022.
8. Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A, Neville G. The short-term effects of air pollution on hospital admissions in four Australian cities. Aust N Z J Public Health. 2005 Jun;29(3):213-21. PMID: 15991768.

9. Simpson R, Denison L, Petroeschevsky A, Thalib L, Wilams G. Effects of ambient particle pollution on daily mortality in Melbourne, 1991-1996. J Expo Anal Environ Epidemiol. 2000 Sep-Oct;10(5):488-96. doi: 10.1038/sj.jea.7500137. PMID: 11051538.
10. Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, Hinwood A, Neville G, Neller A. The short-term effects of air pollution on daily mortality in four Australian cities. Aust N Z J Public Health. 2005 Jun;29(3):205-12. PMID: 15991767.

Attachment Two: Corrected Cancer Risk and Hazard Quotient Tables

Table 0.1: Predicted incremental lifetime cancer risk attributable to emissions from the existing and proposed Allied Asphalt Plants – Worst Case Scenario

Receptor	Pollutant	Incremental lifetime cancer risk – Worst Case Scenario		
		Existing Plant	Proposed Plant	
		ULO	Diesel	Natural Gas
Whareroa Marae	Arsenic	$2x10^{-7}$	$3x10^{-9}$	$3x10^{-9}$
	Cadmium	$9x10^{-7}$	$5x10^{-9}$	$5x10^{-9}$
	Chromium VI	$2x10^{-7}$	$1x10^{-8}$	$1x10^{-8}$
	Benzene	$9x10^{-9}$	$1x10^{-8}$	$1x10^{-8}$
		$9x10^{-8}$	*$1x10^{-7}$*	*$1x10^{-7}$*
	Formaldehyde	$2x10^{-8}$	$8x10^{-9}$	$8x10^{-9}$
	BaP	$2x10^{-10}$	$6x10^{-12}$	$6x10^{-12}$
		$2x10^{-9}$	*$6x10^{-11}$*	*$6x10^{-11}$*
	Lead	$7x10^{-9}$	$2x10^{-11}$	$2x10^{-11}$
Most affected residential receptor	Arsenic	$2x10^{-6}$	$2x10^{-8}$	$2x10^{-8}$
	Cadmium	$9x10^{-7}$	$3x10^{-8}$	$3x10^{-8}$
	Chromium VI	$2x10^{-6}$	$9x10^{-8}$	$9x10^{-8}$
	Benzene	$8x10^{-8}$	$1x10^{-7}$	$1x10^{-7}$
		$8x10^{-7}$	*$1x10^{-6}$*	*$1x10^{-6}$*
	Formaldehyde	$1x10^{-7}$	$6x10^{-8}$	$6x10^{-8}$
	BaP	$2x10^{-9}$	$4x10^{-11}$	$4x10^{-11}$
		$2x10^{-8}$	*$4x10^{-10}$*	*$4x10^{-10}$*
	Lead	$6x10^{-8}$	$1x10^{-10}$	$1x10^{-10}$
Most affected receptor -De Havilland Way	Arsenic	$1x10^{-6}$	$2x10^{-8}$	$2x10^{-8}$
	Cadmium	$7x10^{-7}$	$3x10^{-8}$	$3x10^{-8}$
	Chromium VI	$2x10^{-6}$	$8x10^{-8}$	$8x10^{-8}$
	Benzene	$7x10^{-8}$	$9x10^{-8}$	$9x10^{-8}$
		$7x10^{-7}$	*$9x10^{-7}$*	*$9x10^{-7}$*
	Formaldehyde	$1x10^{-7}$	$5x10^{-8}$	$5x10^{-8}$
	BaP	$1x10^{-9}$	$4x10^{-11}$	$4x10^{-11}$
		$1x10^{-8}$	*$4x10^{-10}$*	*$4x10^{-10}$*
	Lead	$5x10^{-8}$	$1x10^{-10}$	$1x10^{-10}$
Most affected receptor childcare	Arsenic	$2x10^{-6}$	$2x10^{-8}$	$2x10^{-8}$
	Cadmium	$1x10^{-6}$	$4x10^{-8}$	$4x10^{-8}$
	Chromium VI	$2x10^{-6}$	$1x10^{-7}$	$1x10^{-7}$
	Benzene	$1x10^{-9}$	$1x10^{-7}$	$1x10^{-7}$
		$1x10^{-8}$	*$1x10^{-6}$*	*$1x10^{-6}$*
	Formaldehyde	$2x10^{-7}$	$7x10^{-8}$	$7x10^{-8}$

Receptor	Pollutant	Incremental lifetime cancer risk – Worst Case Scenario		
		Existing Plant	Proposed Plant	
		ULO	Diesel	Natural Gas
	BaP	$2x10^{-9}$	$6x10^{-11}$	$6x10^{-11}$
		$2x10^{-8}$	*$6x10^{-10}$*	*$6x10^{-10}$*
	Lead	$7x10^{-8}$	$1x10^{-10}$	$1x10^{-10}$
Mount Maunganui College	Arsenic	$2x10^{-6}$	$2x10^{-8}$	$2x10^{-8}$
	Cadmium	$8x10^{-7}$	$3x10^{-8}$	$3x10^{-8}$
	Chromium VI	$2x10^{-6}$	$8x10^{-8}$	$8x10^{-8}$
	Benzene	$8x10^{-8}$	$8x10^{-8}$	$8x10^{-8}$
		$8x10^{-7}$	*$8x10^{-7}$*	*$8x10^{-7}$*
	Formaldehyde	$1x10^{-7}$	$5x10^{-8}$	$5x10^{-8}$
	BaP	$1x10^{-9}$	$4x10^{-11}$	$4x10^{-11}$
		$1x10^{-8}$	*$4x10^{-10}$*	*$4x10^{-10}$*
	Lead	$6x10^{-8}$	$1x10^{-10}$	$1x10^{-10}$
Mount Maunganui Intermediate	Arsenic	$1x10^{-6}$	$1x10^{-8}$	$1x10^{-8}$
	Cadmium	$5x10^{-7}$	$2x10^{-8}$	$2x10^{-8}$
	Chromium VI	$1x10^{-6}$	$6x10^{-8}$	$6x10^{-8}$
	Benzene	$5x10^{-8}$	$7x10^{-8}$	$7x10^{-8}$
		$5x10^{-7}$	*$7x10^{-7}$*	*$7x10^{-7}$*
	Formaldehyde	$8x10^{-8}$	$4x10^{-8}$	$4x10^{-8}$
	BaP	$9x10^{-10}$	$3x10^{-11}$	$3x10^{-11}$
		$9x10^{-9}$	*$3x10^{-10}$*	*$3x10^{-10}$*
	Lead	$4x10^{-8}$	$9x10^{-11}$	$9x10^{-11}$
Gwen Rogers Kindergarten	Arsenic	$1x10^{-6}$	$1x10^{-8}$	$1x10^{-8}$
	Cadmium	$6x10^{-7}$	$2x10^{-8}$	$2x10^{-8}$
	Chromium VI	$1x10^{-6}$	$5x10^{-8}$	$5x10^{-8}$
	Benzene	$5x10^{-8}$	$6x10^{-8}$	$6x10^{-8}$
		$5x10^{-7}$	*$6x10^{-7}$*	*$6x10^{-7}$*
	Formaldehyde	$9x10^{-8}$	$3x10^{-8}$	$3x10^{-8}$
	BaP	$1x10^{-9}$	$3x10^{-11}$	$3x10^{-11}$
		$1x10^{-8}$	*$3x10^{-10}$*	*$3x10^{-10}$*
	Lead	$4x10^{-8}$	$8x10^{-11}$	$8x10^{-11}$
Omanu Primary	Arsenic	$1x10^{-6}$	$1x10^{-8}$	$1x10^{-8}$
	Cadmium	$6x10^{-7}$	$2x10^{-8}$	$2x10^{-8}$
	Chromium VI	$1x10^{-6}$	$6x10^{-8}$	$6x10^{-8}$
	Benzene	$6x10^{-8}$	$6x10^{-8}$	$6x10^{-8}$
		$6x10^{-7}$	*$6x10^{-7}$*	*$6x10^{-7}$*
	Formaldehyde	$9x10^{-8}$	$4x10^{-8}$	$4x10^{-8}$
	BaP	$1x10^{-9}$	$3x10^{-11}$	$3x10^{-11}$
		$1x10^{-8}$	*$3x10^{-10}$*	*$3x10^{-10}$*
	Lead	$4x10^{-8}$	$8x10^{-11}$	$8x10^{-11}$

Receptor	Pollutant	Incremental lifetime cancer risk – Worst Case Scenario		
		Existing Plant	Proposed Plant	
		ULO	Diesel	Natural Gas
Best Start MacDonald Street	Arsenic	$8x10^{-7}$	$1x10^{-8}$	$1x10^{-8}$
	Cadmium	$4x10^{-7}$	$2x10^{-8}$	$2x10^{-8}$
	Chromium VI	$9x10^{-7}$	$5x10^{-8}$	$5x10^{-8}$
	Benzene	$4x10^{-8}$	$5x10^{-8}$	$5x10^{-8}$
		$4x10^{-7}$	*$5x10^{-7}$*	*$5x10^{-7}$*
	Formaldehyde	$6x10^{-8}$	$3x10^{-8}$	$3x10^{-8}$
	BaP	$7x10^{-10}$	$2x10^{-11}$	$2x10^{-11}$
		$7x10^{-9}$	*$2x10^{-10}$*	*$2x10^{-10}$*
	Lead	$3x10^{-8}$	$7x10^{-11}$	$7x10^{-11}$

Receptor	Pollutant	Chronic Hazard Quotients – Worst Case Scenario			Acute Hazard Quotients – Worst Case Scenario		
		Existing	Proposed		Existing	Proposed	
		Used Oil	Diesel	Natural Gas	Used Oil	Diesel	Natural Gas
Whareroa Marae	Arsenic	0.004	0.00005	0.00005	0.02	0.0002	0.0002
	Cadmium	0.001	0.00005	0.00005	na	na	na
	Chromium VI	0.000007	0.0000004	0.0000004	na	na	na
	Benzene	0.0001	0.0002	0.0002	0.00005	0.00002	0.00002
	Formaldehyde	0.0003	0.0001	0.0001	0.003	0.001	0.001
	Chromium III	0.0009	0.00002	0.00002	0.007	0.0001	0.0001
	Copper	na	na	na	0.0004	0.000002	0.000002
	Lead	na	na	na	0.006	0.00002	0.00002
Most affected residential receptor	Arsenic	0.03	0.0004	0.0004	0.04	0.001	0.001
	Cadmium	0.01	0.0004	0.0004	na	na	na
	Chromium VI	0.00007	0.000003	0.000003	na	na	na
	Benzene	0.001	0.001	0.001	0.0004	0.0001	0.0001
	Formaldehyde	0.003	0.001	0.001	0.007	0.003	0.003
	Chromium III	0.009	0.0001	0.0001	0.02	0.0003	0.0003
	Copper	na	na	na	0.0009	0.000007	0.000007
	Lead	na	na	na	0.03	0.00007	0.00007
Most affected receptor – De Havilland Way	Arsenic	0.03	0.0003	0.0003	0.06	0.0006	0.0006
	Cadmium	0.009	0.0003	0.0003	na	na	na
	Chromium VI	0.00005	0.000003	0.000003	na	na	na
	Benzene	0.0008	0.001	0.001	0.0004	0.0001	0.0001

Receptor	Pollutant	Chronic Hazard Quotients – Worst Case Scenario			Acute Hazard Quotients – Worst Case Scenario		
		Existing	Proposed		Existing	Proposed	
		Used Oil	Diesel	Natural Gas	Used Oil	Diesel	Natural Gas
	Formaldehyde	0.002	0.0009	0.0009	0.009	0.003	0.003
	Chromium III	0.007	0.0001	0.0001	0.02	0.0003	0.0003
	Copper	na	na	na	0.001	0.000007	0.000007
	Lead	na	na	na	0.05	0.00005	0.00005
Little Einstein's Childcare Centre	Arsenic	0.04	0.0005	0.0005	0.05	0.0006	0.0006
	Cadmium	0.01	0.0005	0.0005	na	na	na
	Chromium VI	0.00008	0.000004	0.000004	na	na	na
	Benzene	0.001	0.001	0.001	0.0005	0.0002	0.0002
	Formaldehyde	0.003	0.001	0.001	0.008	0.003	0.003
	Chromium III	0.01	0.0002	0.0002	0.02	0.0003	0.0003
	Copper	na	na	na	0.001	0.000007	0.000007
	Lead	na	na	na	0.03	0.0001	0.0001
Mount Maunganui College	Arsenic	0.03	0.0003	0.0003	0.05	0.0006	0.0006
	Cadmium	0.009	0.0003	0.0003	na	na	na
	Chromium VI	0.00006	0.000002	0.000002	na	na	na
	Benzene	0.0009	0.001	0.001	0.0004	0.0001	0.0001
	Formaldehyde	0.002	0.0009	0.0009	0.008	0.003	0.003
	Chromium III	0.008	0.0001	0.0001	0.02	0.0003	0.0003
	Copper	na	na	na	0.001	0.000007	0.000007
	Lead	na	na	na	0.02	0.00005	0.00005

Receptor	Pollutant	Chronic Hazard Quotients – Worst Case Scenario			Acute Hazard Quotients – Worst Case Scenario		
		Existing	Proposed		Existing	Proposed	
		Used Oil	Diesel	Natural Gas	Used Oil	Diesel	Natural Gas
Mount Manganui Intermediate	Arsenic	0.02	0.0003	0.0003	0.04	0.0004	0.0004
	Cadmium	0.006	0.0003	0.0003	na	na	na
	Chromium VI	0.00004	0.000002	0.000002	na	na	na
	Benzene	0.0006	0.0008	0.0008	0.0003	0.00009	0.00009
	Formaldehyde	0.001	0.0007	0.0007	0.006	0.002	0.002
	Chromium III	0.005	0.00008	0.00008	0.02	0.0002	0.0002
	Copper	na	na	na	0.0007	0.000004	0.000004
	Lead	na	na	na	0.02	0.00006	0.00006
Gwen Rogers Kindergarten	Arsenic	0.02	0.0002	0.0002	0.04	0.001	0.001
	Cadmium	0.007	0.0002	0.0002	na	na	na
	Chromium VI	0.00004	0.000002	0.000002	na	na	na
	Benzene	0.0006	0.0007	0.0007	0.002	0.003	0.003
	Formaldehyde	0.002	0.0006	0.0006	0.007	0.004	0.004
	Chromium III	0.006	0.00007	0.00007	0.01	0.0002	0.0002
	Copper	na	na	na	0.00003	0.000007	0.000007
	Lead	na	na	na	0.03	0.00004	0.00004
Omanu Primary	Arsenic	0.02	0.0002	0.0002	0.05	0.0006	0.0006
	Cadmium	0.007	0.0002	0.0002	na	na	na
	Chromium VI	0.00005	0.000002	0.000002	na	na	na
	Benzene	0.0007	0.0007	0.0007	0.002	0.003	0.003

Receptor	Pollutant	Chronic Hazard Quotients – Worst Case Scenario			Acute Hazard Quotients – Worst Case Scenario		
		Existing	Proposed		Existing	Proposed	
		Used Oil	Diesel	Natural Gas	Used Oil	Diesel	Natural Gas
	Formaldehyde	0.003	0.0006	0.0006	0.008	0.003	0.003
	Chromium III	0.006	0.00008	0.00008	0.01	0.0002	0.0002
	Copper	na	na	na	0.00004	0.000007	0.000007
	Lead	na	na	na	0.03	0.00005	0.00005
Best Start MacDonald Street	Arsenic	0.02	0.0002	0.0002	0.03	0.0003	0.0003
	Cadmium	0.005	0.0002	0.0002	na	na	na
	Chromium VI	0.00003	0.000002	0.000002	na	na	na
	Benzene	0.0004	0.0006	0.0006	0.001	0.002	0.002
	Formaldehyde	0.001	0.0005	0.0005	0.005	0.002	0.002
	Chromium III	0.004	0.00006	0.00006	0.01	0.0002	0.0002
	Copper	na	na	na	0.0006	0.000004	0.000004
	Lead	na	na	na	0.05	0.00005	0.00005

Attachment Three: Paper Hales (2021)

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Long term exposure to air pollution, mortality and morbidity in New Zealand: Cohort study

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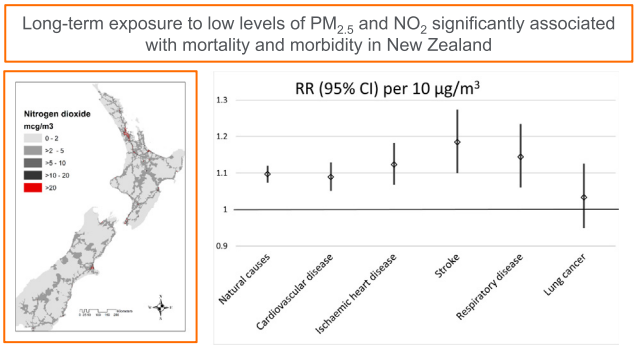
HIGHLIGHTS

- Cohort study of long term exposure to air pollution in a low-exposure setting.
- Effect estimates for NO_2 were higher than reported in many previous studies.
- There was no evidence of a threshold.

GRAPHICAL ABSTRACT



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ABSTRACT

Objectives: To investigate associations between long-term exposure to $PM_{2.5}$, NO_2, mortality and morbidity in New Zealand, a country with low levels of exposure.
Design: Retrospective cohort study.
Setting: The New Zealand resident population.
Method: The main analyses included all adults aged 30 years and over with complete data on covariates: $N = 2{,}223{,}507$. People who died, or were admitted to hospital, (2013–2016) were linked anonymously to the 2013 census, and to estimates of ambient $PM_{2.5}$, and NO_2 concentration. We fitted Poisson regression models of mortality and morbidity in adults (≥30) for all natural causes of death, and by sub- group of major cause. Person-time of exposure, censored at the time of death, was included as an offset. We adjusted for confounding by age, sex, ethnicity, income, education, smoking status and ambient temperature. Further analyses stratified by ethnic group, and investigated respiratory hospital admissions in children.
Results: There were statistically significant positive associations between pollutants and natural causes of death: RR (per 10 µg/m^3) for $PM_{2.5}$ 1.11 (1.07 to 1.15) and for NO_2 1.10 (1.07 to 1.12). For morbidity, the strongest associations were for $PM_{2.5}$ and ischaemic heart disease in adults, RR: 1.29 (1.23 to 1.35) and for NO_2 and asthma in children, RR: 1.18 (1.09 to 1.28). In models restricted to specific ethnic groups, we found no consistent differences in any of the associations.
Conclusions: The results for NO_2 are higher than those published previously. Other studies have reported that the dose-response for $PM_{2.5}$ may be higher at low concentrations, but less is known about NO_2. It is possible NO_2 is acting as a proxy for other traffic-related pollutants that are causally related to health impacts. This study underlines the importance of controlling pollution caused by motor vehicles.

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1. Introduction

The health effects of exposure to air pollution are increasingly well established, but there is debate about the importance of individual pollutants and of exposure at low levels. Many epidemiological studies have linked short-term air pollution exposure to mortality and morbidity in daily time series studies (Héroux et al., 2015) and long-term air pollution exposure is now established as the most substantial global environmental health risk (Burnett et al., 2018; Evangelopoulos et al., 2020). Evidence of health effects at levels well below regulatory guidelines has been strengthened by improved methods of exposure assessment and the findings from large cohort studies based on administrative data. In many countries, including New Zealand, it is possible to link air pollution exposure with routinely collected individual data on health outcomes, as well as information on other variables of interest in air pollution studies, such as smoking and socio-economic position.

A recent systematic review concluded that there was clear evidence of associations between particulate matter (PM) and mortality (Chen and Hoek, 2020). The evidence of health effects in relation to other pollutants is less definitive. A meta-analysis found that associations between long-term exposure to NO_2 and mortality were broadly similar in strength to those of $PM_{2.5}$, and in four studies, were not substantially altered following adjustment for $PM_{2.5}$ (Faustini et al., 2014). Meta-analyses reported statistically significant associations between NO_2 and mortality, but with substantial heterogeneity of the effect size (Hoek et al., 2013; Huangfu and Atkinson, 2020). Huang et al. considered that: *"… long term exposure to NO_2, a proxy for traffic-sourced air pollutants, is associated with higher risk of all-cause, cardiovascular and respiratory mortality that might be independent of other pollutants"* (Huang et al., 2021).

Relatively few studies have analysed associations between long-term air pollution exposure and morbidity (Kloog et al., 2012; Yitshak-Sade et al., 2018; Williams et al., 2019; Yazdi et al., 2021). As with mortality, associations with long-term exposure are typically stronger than those for short-term exposure (Héroux et al., 2015). For example, a study of both long- and short-term $PM_{2.5}$ exposure and hospital admissions reported significantly stronger associations with long-term exposure (Kloog et al., 2012).

Previously, we reported associations between PM_{10} and mortality in a cohort based on anonymous linkage of the 1996 New Zealand census (Hales et al., 2012). In the present study, we investigate associations between long-term exposure to $PM_{2.5}$, NO_2, mortality and morbidity. As the previous study provided suggestive evidence of effect modification by ethnicity, we were particularly interested to explore this in the present study.

2. Method

2.1. Data sources

Analyses relied on the Integrated Data Infrastructure (IDI) in the Statistics New Zealand data laboratory, a national dataset covering the entire New Zealand population (4.2 million people in 2013). The IDI spine aims to capture all New Zealand residents. Routinely collected administrative datasets including the census and health system data are linked separately to the spine. These links are not perfect: in June 2020 when these analyses were conducted, the linkage rate with the 2013 census was 94%; and with health datasets, 85%. The false positive linkage rate was estimated to be 0.8% in each case.

The 2013 New Zealand census population included 2,531,841 people aged 30 and over, who were eligible to be included in the main analyses. Of these, 2,347,467 participants (92%) were linked to the census in the Integrated Data Infrastructure. Participants who died, or were admitted to hospital, between March 2013 and December 2016 were linked anonymously at individual level to the 2013 census, using data from two health datasets, the mortality data collection (Ministry of Health, 2021a) and the National Minimum Dataset of publicly-funded hospital discharges (Ministry of Health, 2021b). As health effects of NO_2 and potential effect modification by ethnicity were of particular interest, we excluded individuals with missing data on ethnicity (N = 119,742) or NO_2 exposure (N = 414). A further 3822 people had missing data on $PM_{2.5}$ exposure. The main analyses were two pollutant models in adults aged 30 years and over with complete data on covariates: N = 2,223,507; 95% of participants; 88% of the eligible population (Table 1). The age, sex and ethnicity profile matched that of the census population, with the exception of people identifying as European/other, who were slightly over-represented in our study (Table 1). Main analyses included 77,394 deaths from natural causes and 274,992 hospital admissions (Table 2).

Participants were spatially referenced at the level of meshblocks (the smallest administrative unit, containing approximately 100 people) and larger census area units (CAU, containing several hundred to several thousand people). We used annual estimates of air pollution for small areas as proxies for long-term average exposure (years to decades) for individuals. Estimates of fine particulate matter ($PM_{2.5}$) concentration were available, nominally for 2006 and 2016, at CAU scale from ambient air quality monitoring data extrapolated to cover New Zealand (Kuschel et al., 2021). Estimates of NO_2 concentrations, nominally for the year 2016, at 50 m spatial resolution, were developed from a vehicle emissions modelling tool (Tonkin and Taylor, 2021) and

Table 1
Number of participants by variable, and comparison with age, sex, ethnicity distribution in the 2013 census.[1]

Variable	Label	Included in main analysis (n)	%	Census (%)
Age	All ≥ 30 years	2,227,332		
	30–34 years	226,719	10.2	10.1
	35–39 years	238,755	10.7	10.6
	40–44 years	273,264	12.3	12.1
	45–49 years	269,601	12.1	11.9
	50–54 years	267,078	12.0	11.8
	55–59 years	231,270	10.4	10.3
	60–64 years	206,646	9.3	9.2
	65–69 years	170,805	7.7	7.7
	70–74 years	129,495	5.8	5.9
	75–79 years	90,669	4.1	4.2
	80–84 years	66,735	3.0	3.2
	≥ 85 years	56,298	2.5	2.9
Sex	Male	1,054,254	47.3	47.4
	Female	1,173,075	52.7	52.6
Ethnicity	Māori	224,505	10.1	10.0
	Pacific Peoples	92,643	4.2	4.2
	Asian	225,081	10.1	9.4
	European/other	1,685,097	75.7	71.2
	Missing	0		5.2
Education	Primary and lower secondary	730,464	32.8	
	Upper secondary and post-secondary non-Tertiary	704,067	31.6	
	Tertiary	683,913	30.7	
	Missing	108,885	4.9	
Income	Lowest	727,932	32.7	
	Middle	786,141	35.3	
	Highest	713,256	32.0	
Smoking	Smoker	303,534	13.6	
	Ex-Smoker	592,287	26.6	
	Never smoked regularly	1,261,383	56.6	
	Not specified	70,125	3.1	
Temperature	Missing	0		
	Present	2,227,332	100.0	
NO_2	Missing	0		
	Present	2,227,332	100.0	
$PM_{2.5}$	Missing	3822	0.2	
	Present	2,223,507	99.8	

[1] Participants included in main analyses, aged 30 and over with complete data on ethnicity and NO_2 exposure, (counts rounded to base 3 to maintain confidentiality, meaning that category totals do not match exactly).

Table 2
Number of participants by disease outcome.[1]

Hospital discharges	Disease category	Included in main analysis (n)
	Cardiovascular disease	140,358
	Ischaemic heart disease	41,670
	Stroke	30,591
	Respiratory	52,269
	Lung cancer	5214
	Asthma (adults)	4890
	All adults	274,992
	Asthma (children)	6249
Mortality		
	Natural causes	77,394
	Cardiovascular disease	26,766
	Ischaemic heart disease	13,314
	Stroke	6195
	Respiratory disease	5856
	Lung cancer	4806
	Asthma	195

[1] Participants aged 30 and over with complete data on ethnicity and NO_2 exposure, rounded to base 3 to maintain confidentiality.

averaged within meshblock boundaries in a geographic information system. The meshblock scale estimates were averaged at CAU scale with population weighting for sensitivity analyses. Further details of the air pollution estimation methods are in the supplementary material. Long term average annual temperature estimates by CAU were derived from the ERA5 land dataset (Muñoz Sabater, 2019). Ambient temperature is a potential confounder of the relationship between air pollution exposure and health.

The study was approved by the University of Otago Ethics Committee (ref HD20/022); the National Health and Disability Ethics Committee determined that their approval was not required.

2.2. Statistical analysis

2.2.1. Mortality

We fitted Poisson regression models of mortality in adults (aged 30 and above) for all natural causes and by sub-group of major cause: cardiovascular diseases, ischaemic heart disease, stroke, respiratory diseases, lung cancer and asthma, based on reported ICD10 codes: for mortality from natural causes (all codes, excluding V00-V05, V09-V10, V12-V18, V193, V20-V804, V809-X85, X88-Y8); cardiovascular disease (G45, I011, I012, I05-I13, I159, I20-I51, I60-I99, M30-M31); ischaemic heart disease (I20-I25); stroke (G45, I60-I69); respiratory causes (J22-J65, J668, J67-J98); lung cancer (C33-C34) and asthma (J45-J46). Person-time of exposure, censored at the time of death, was included as an offset.

Initial models were analysed in relation to the estimated annual average exposure to air pollution in 2016, at the place of usual residence at the time of the 2013 Census. All models used estimates of $PM_{2.5}$ concentration at CAU scale. For models including NO_2, we carried out analyses at meshblock scale.

We adjusted for confounding by age, sex, prioritized ethnicity: Māori then Pacific Peoples then Asian then European/other, (the largest group, over 95% of whom are European); personal income (lowest, middle, highest incomes); education ('European Education ISCED 97 3-level grouping' Primary and Lower Secondary; Upper Secondary and Post-Secondary non-Tertiary; Tertiary); smoking status (current smoker; ex-smoker; never smoked regularly; missing) and ambient temperature.

All models included adjustment for age (in 5-year groups), sex and prioritized ethnicity. Initially the effects of all pollutants were assessed in single pollutant models (step 1). Next, a series of adjustments were applied. In step 2, we added individual income, education and smoking status to the model. In step 3, the models included NO_2 along with $PM_{2.5}$ and finally, in step 4, we added annual mean temperature.

2.2.2. Morbidity

We repeated the main analyses described above for mortality, using data on public hospital discharges for cardiovascular diseases, ischaemic heart disease, stroke, respiratory diseases, lung cancer and asthma in adults ages 30 years and above, and for asthma in children aged 0 to 14 years inclusive. Participants who had been admitted to hospital for the same condition prior to the 2013 census were not excluded from these analyses. For the analysis of asthma in children, educational and smoking status were not available; we substituted equivalized household income (lowest, middle, highest) for personal income.

2.3. Further analyses

Potential effect modification by ethnicity was assessed in subgroup analyses. We also restricted the analysis of mortality to people who lived in the same CAU in 2013 and at least five years previously, using the average of estimated annual $PM_{2.5}$ concentrations by CAU in 2006 and 2016. We ran a model at the spatial scale of CAUs, using population weighted estimates of NO_2 exposure at CAU level. We estimated the annual number of premature deaths attributable to annual $PM_{2.5}$ concentrations in 2016 based on population attributable fractions multiplied by the observed national counts of mortality.

3. Results

3.1. Air pollution exposure

Summary statistics for air pollution exposure by ethnicity are provided in Table 3. Estimated population weighted annual average air pollutant concentrations in 2016 were low by international standards: $PM_{2.5}$ 6.5 μg/m^3 and NO_2 7.6 μg/m^3; (comparable figures for the UK were $PM_{2.5}$ 10.1 μg/m^3 and NO_2 27.4 μg/m^3). Average concentrations of $PM_{2.5}$ were slightly lower in 2016 (6.5 μg/m^3) than in 2006 (7.5 μg/m^3), but highly correlated ($R^2 = 0.92$). In 2016, $PM_{2.5}$ and NO_2 were weakly correlated ($R^2 = 0.30$). Concentrations of NO_2 were higher among Pacific Peoples (9.9 μg/m^3) and lower among Māori (6.9 μg/m^3) than European/other ethnicities (7.6 μg/m^3). Concentrations of $PM_{2.5}$ were similar among ethnicities (Table 3).

3.2. Mortality

In single pollutant models, with adjustment for age, sex and ethnicity only, the rate ratio (RR) for $PM_{2.5}$ was 1.288 per 10 μg/m^3 (1.255 to 1.322) and for NO_2 1.100 (1.081 to 1.120). The RR for NO_2 was reduced only slightly when income, education and smoking were included, fell slightly with the addition of $PM_{2.5}$ to the model and

Table 3
Estimated air pollution concentrations, adults aged >30 years, population weighted, by year and ethnicity.

	Pollutant	Mean	SD	Min	Max
		μg/m^3			
All ethnicities	$PM_{2.5}$ 2016	6.5	2.2	4.1	15.4
	$PM_{2.5}$ 2006	7.5	2.6	4.0	19.1
	NO_2	7.6	3.9	2.6	19.8
Māori	$PM_{2.5}$ 2016	6.4	2.0	4.1	15.4
	$PM_{2.5}$ 2006	7.5	2.7	4.0	19.1
	NO_2	6.9	3.6	2.6	19.8
Pacific peoples	$PM_{2.5}$ 2016	6.2	1.5	4.1	15.4
	$PM_{2.5}$ 2006	7.4	1.8	4.0	19.1
	NO_2	9.9	3.6	2.6	19.8
European/Asian/other[1]	$PM_{2.5}$ 2016	6.6	2.3	4.1	15.4
	$PM_{2.5}$ 2006	7.6	2.6	4.0	19.1
	NO_2	7.6	3.9	2.6	19.8

[1] In this table, this category also includes Asian people. All other analyses were carried out separately for Asian ethnicity.

Table 4
Change in effect size (RR per 10 μg/m^3) at each step of the analysis: natural causes of mortality.

Model (analysis step)	$PM_{2.5}$	NO_2
Single pollutant		
1. Adjusted for age, sex, ethnicity	1.29	1.10
2. Adjusted for age, sex, ethnicity, income, education, smoking	1.20	1.09
Two pollutant		
3. Adjusted for age, sex, ethnicity, income, education, smoking	1.18	1.07
4. Adjusted for age, sex, ethnicity, income, education, smoking, temperature	1.10	1.10

increased slightly on addition of temperature. The RR for $PM_{2.5}$ was attenuated at each of these steps (Table 4).

Results for the final two pollutant models are shown in Table 5 and Tables S1, S2. There were statistically significant positive associations between pollutants and natural causes of death: RR for $PM_{2.5}$ 1.105; (1.065 to 1.145) and for NO_2 1.097; (1.074 to 1.120, Table 5). Applying these results to observed mortality and estimates of exposure to $PM_{2.5}$ (all sources) and NO_2 (from road traffic), approximately 2000 premature deaths were attributable to each pollutant in 2016.

There were significant associations between $PM_{2.5}$ exposure and both cardiovascular diseases and respiratory diseases, but associations with sub-categories were not significant. The strongest associations, though with wide confidence intervals, were for $PM_{2.5}$ and respiratory diseases: RR 1.142; (1.004 to 1.298) and for NO_2 and asthma: RR 1.535; (1.018 to 2.315). Positive associations with lung cancer were not significant.

Results for natural causes of mortality were substantially unchanged in a single pollutant model using the averages of $PM_{2.5}$ concentration by census area in 2006 and 2016, restricted to people who lived in the same CAU for at least 5 years prior to the 2013 census. In models restricted to specific ethnic groups, we found no consistent differences in any of the associations (Table S2).

3.3. Morbidity

In two-pollutant models, there were statistically significant positive associations between both pollutants and cardiovascular diseases and respiratory diseases (Table 6). There were stronger associations for $PM_{2.5}$ and cardiovascular diseases, and for NO_2 and respiratory diseases. The strongest associations were for $PM_{2.5}$ and ischaemic heart disease in adults, RR: 1.289; (1.227 to 1.353) and for NO_2 and asthma in children, RR: 1.18 (1.09 to 1.28; Table 6). Associations with lung cancer were not significant. In models restricted to specific ethnic groups, we found no consistent differences in any of the associations (Table S3).

Table 5
Mortality (adults aged 30 and above) by cause and pollutant, two pollutant models.[1]

Mortality	RR (per 10 μg/m^3)	Lower 95% CI	Upper 95% CI
$PM_{2.5}$			
Natural causes	1.105	1.065	1.145
Cardiovascular disease	1.089	1.024	1.158
Ischaemic heart disease	1.082	0.992	1.179
Stroke	1.121	0.986	1.274
Respiratory disease	1.142	1.004	1.298
Lung cancer	1.014	0.875	1.176
Asthma	0.560	0.267	1.176
NO_2			
Natural causes	1.097	1.074	1.120
Cardiovascular disease	1.089	1.051	1.129
Ischaemic heart disease	1.123	1.068	1.182
Stroke	1.184	1.100	1.274
Respiratory disease	1.144	1.060	1.234
Lung cancer	1.033	0.949	1.126
Asthma	1.535	1.018	2.315

[1] Main analyses included participants aged 30 years and over with complete data on ethnicity and NO_2 exposure. Models included age, sex, ethnicity, personal income, education, smoking, temperature, $PM_{2.5}$ and NO_2.

Table 6
Morbidity by cause and pollutant, in two pollutant models.[1]

Morbidity	RR (per 10 μg/m^3)	Lower 95% CI	Upper 95% CI
$PM_{2.5}$			
Cardiovascular disease	1.115	1.084	1.146
Ischaemic heart disease	1.289	1.227	1.353
Stroke	1.128	1.064	1.197
Respiratory disease	1.070	1.021	1.122
Lung cancer	0.989	0.860	1.137
Asthma (adults)	0.954	0.810	1.123
Asthma (children)	1.103	0.952	1.278
NO_2			
Cardiovascular disease	1.047	1.031	1.064
Ischaemic heart disease	0.972	0.944	1.001
Stroke	1.041	1.006	1.077
Respiratory disease	1.130	1.102	1.159
Lung cancer	1.011	0.930	1.098
Asthma (adults)	1.169	1.075	1.271
Asthma (children)	1.182	1.094	1.276

[1] Main analyses included adults aged ≥ 30 years (or children aged 0–14) with complete data on ethnicity and NO_2 exposure. Models in adults included age, sex, ethnicity, personal income, education, smoking, temperature, $PM_{2.5}$ and NO_2; models in children included age, sex, ethnicity, household income, temperature, $PM_{2.5}$ and NO_2.

4. Discussion

4.1. Main findings

In two-pollutant models, there were statistically significant associations between both $PM_{2.5}$ and NO_2 and mortality from natural causes, and both mortality and morbidity from cardiovascular diseases and respiratory diseases (Tables 5, 6). For mortality from specific causes of death except for asthma, effect sizes were similar for $PM_{2.5}$ and NO_2. For morbidity, effects of $PM_{2.5}$ were stronger for cardiovascular disease, while effects of NO_2 were stronger for respiratory disease.

4.2. Strengths and weaknesses

The strengths of this study are the fine spatial scale of the exposure assessment and the inclusion of the whole New Zealand population linked to the census. We did not adjust for persons not linked to the census, but linkage rates were high (92%). Exposure levels were assessed with robust methods and at fine spatial scale. Both actual and modelled exposure contrasts may be relatively well defined in NZ, compared to other settings, due to lack of long-distance transport of air pollution, coupled with very low concentrations in rural areas.

We were able to control for effects of age, sex, ethnicity, education, income and smoking at individual level, based on census data linkage. We carried out a stepwise analysis which allowed assessment of potential confounding at each step. The effects of NO_2 were not substantially attenuated by control for confounding. The associations between ambient air pollution and health could be biased if concentrations change over time. We can be reasonably confident that this does not affect results for $PM_{2.5}$, given that they were substantially unchanged when estimating exposure over a 10-year period (2006 to 2016) in participants who were in the same census area unit for at least 5 years (2009 to 2013). This analysis could not be repeated for NO_2, as estimates of NO_2 concentrations prior to 2016 were unavailable at fine geographic scale.

While fine-scale data are not available for 2006, NO_2 has been monitored at a series of locations across the New Zealand state highway network since 2007 using passive diffusion tubes (NZTA, 2017). The percentage change in measured annual averages between 2007 and

2016 was calculated for the 34 sites that have been operating since 2007 and have at least 75% valid data for 2007, 2011, 2012 and 2016. The annual NO_2 concentrations for these sites are higher by an average of 14% in 2016 than in 2007. Whilst results vary spatially, most of these sites (26/34) show at least some increase in measured NO_2, with 12 sites recording increases of 20% or more. Therefore the results reported here, based on NO_2 concentrations nominally for 2016, might be biased downwards.

An unmeasured factor which is associated with both air pollution exposure and mortality could bias the results. This type of bias can never be completely excluded. For example, NO_2 might be acting as a proxy for exposure to traffic, and the true causal factor might be another traffic-related exposure such as noise or coarse PM ($PM_{2.5–10}$) from road dust. We assessed the risk of bias in our study using a tool developed by the World Health Organization (WHO, 2020). We consider the risk of bias to be low in all domains except for confounding. WHO recommends controlling for body mass index (BMI) for cohort studies of long term air pollution exposure (WHO, 2020). We were unable to do this as BMI data are not available at individual or small area level in New Zealand. For this reason, we assessed the risk of confounding as low to moderate. On the other hand, we controlled for ethnicity in the multivariate analysis, which would be expected to control for BMI indirectly, since BMI is strongly associated with ethnicity in New Zealand (Ministry of Health, 2020). Studies of air pollution and health which control for noise have reported mixed results (Stieb et al., 2021). Ideally, future air pollution studies should also control for noise.

4.3. Comparison with previous studies

4.3.1. Mortality

The results for $PM_{2.5}$ are comparable with the findings from a recent meta-analysis: for natural causes, RR 1.08 (1.06 to 1.09) per 10 μg/m^3 in single pollutant models (Chen and Hoek, 2020). In a subset of five studies that ran two-pollutant models with $PM_{2.5}$ and NO_2, a reduction in $PM_{2.5}$ effect was observed, to 1.02 (1.00 to 1.04) (Chen and Hoek, 2020).

An Australian study reported that "$PM_{2.5}$ and NO_2 had detrimental but non statistically significant associations with all-cause mortality at low concentrations"(Hanigan et al., 2019). In Canada, a country with relatively low levels of ambient air pollution, Zhang et al. carried out a prospective cohort study. These authors reported hazard ratios (HR) for mortality of 1.037 (1.018, 1.057) per 1 μg/m^3 increase for $PM_{2.5}$ and 1.027 (1.021, 1.034) per 1 ppb NO_2: equivalent to HR 1.438 per 10 μg/m^3 for $PM_{2.5}$ and 1.151 per 10 μg/m^3 NO_2 (Zhang et al., 2021). A recent analysis from the ELAPSE study reported HRs for mortality, in single pollutant models, of 1.134, (1.109 to 1.159) per 5 μg/m^3 increase for $PM_{2.5}$ and 1.087, (1.071 to 1.103) per 10 μg/m^3 for NO_2 (Chen et al., 2021). The latter results are comparable with ours, but 2-to-5-fold higher than recent meta-analyses (Table 5). Huangfu and Atkinson (2020) reported a pooled RR estimate of 1.02 (1.01 to 1.04) per 10 μg/m^3 NO_2. Another recent study examined the effect of using different methods of exposure assessment in the Netherlands. In that study, results for exposure based on a dispersion model, the most comparable approach to that used in the present study, were: 1.015 (1.005 to 1.024) per 10 μg/m^3 NO_2 (Klompmaker et al., 2021).

Most studies report higher effect sizes for specific cardiovascular and respiratory causes of death than for natural causes. For mortality from cardiovascular disease, our results are lower than reported by Faustini (Faustini et al., 2014), but higher than the other meta-analyses. For mortality from respiratory disease, the results are much higher than reported in most previous studies (Huangfu and Atkinson, 2020). For mortality from lung cancer, the results are slightly lower than reported in the meta-analyses.

4.3.2. Morbidity

The associations reported here are stronger than those in previous studies of long-term exposure (Table 6). For example, Kloog et al. reported a 4.22% increase in respiratory admissions per 10 μg/m^3 long-term exposure to $PM_{2.5}$ (Kloog et al., 2012). Yitshak-Sade et al. reported a 6.58% increase in cardiovascular admissions for a 2.3 μg/m^3 (interquartile range) increase in $PM_{2.5}$ long-term exposure (Yitshak-Sade et al., 2018). Both of these studies adjusted for temperature, and short-term changes in air pollution, but did not have access to individual data on income, education or smoking status. For asthma morbidity, our findings are comparable to those of a recent large study from the ELAPSE project (Liu et al., 2020). Notably, in that study as in ours, the effect of NO_2 remained when $PM_{2.5}$ was included in the model, while that of $PM_{2.5}$ was attenuated and non-significant after inclusion of NO_2.

4.3.3. Results by ethnicity

Socioeconomically disadvantaged communities are often exposed to higher levels of ambient air pollution (Fairburn et al., 2019). Whether or not social factors modify the effects of air pollution is less clear (Hajat et al., 2021). This is perhaps not surprising, given different patterns of concurrent exposures and health status within and between countries. In the present study, population-weighted average exposure to NO_2 was higher among Pacific Peoples than for other ethnicities. In models restricted to specific ethnic groups, we found no consistent differences in any of the associations (Table 3; Table S3). However, health impacts among Māori and Pacific populations will be relatively high, due to higher prevalence of underlying cardiorespiratory diseases in these groups.

4.4. Implications

Pollution levels in New Zealand are generally lower than in Europe and North America, where much of the research in this field has been conducted so far. There is evidence that the dose-response for PM may be higher (supra-linear) at low doses (Chen and Hoek, 2020), but less is known about NO_2 (Huangfu and Atkinson, 2020).

It is possible that an unmeasured confounding factor, associated with air pollution and health outcomes, but unrelated to traffic, is the true cause of the associations reported here; however, we consider this unlikely. More plausibly, NO_2 may act as a proxy for other traffic-related pollutants that are causally related to health impacts. The strength of the association of NO_2 with childhood asthma stands out for local policy and clinical practice, given that the prevalence of this condition in New Zealand is high by international standards (Lai et al., 2009). The findings of this study underline the importance of controlling pollution caused by motor vehicles in New Zealand. Special emphasis should be given to transport interventions that will reduce the burden of pollution-related ill-health that is experienced by Māori and Pacific populations, including measures to promote active transportation modes.

Disclaimer

These results are not official statistics. They have been created for research purposes from the Integrated Data Infrastructure (IDI) which is carefully managed by Stats NZ. For more information about the IDI please visit https://www.stats.govt.nz/integrated-data/.

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CRediT authorship contribution statement

Simon Hales: Conceptualization, Investigation, Methodology, Writing – original draft, Writing – review & editing. **June Atkinson:**

Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Writing – review & editing. **Jayne Metcalfe:** Data curation, Investigation, Methodology, Writing – review & editing. **Gerda Kuschel:** Funding acquisition, Investigation, Methodology, Writing – review & editing. **Alistair Woodward:** Methodology, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scitotenv.2021.149660.

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