Air pollution and mortality in New Zealand: cohort study

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Statistics New Zealand Security Statement The New Zealand Census Mortality Study is a study of the relation between social factors and mortality in New Zealand, based on the integration of anonymised population census data from Statistics New Zealand and mortality data from the New Zealand Health Information Service. This project was approved by Statistics New Zealand as a data laboratory project under the Microdata Access Protocols in 1997. The datasets created by the integration process are covered by the Statistics Act and can be used for statistical purposes only. Only approved researchers who have signed Statistics New Zealand's declaration of secrecy can access the integrated data in the data laboratory. Access to the data used in this study was provided by Statistics New Zealand under conditions designed to give effect to the security and confidentiality provisions of the Statistics Act, 1975. The results presented in this study are the work of the author, not Statistics New Zealand. The authors take full responsibility for the paper and Statistics New Zealand will not be held accountable for any error or inaccurate findings.

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ABSTRACT

Background Few cohort studies of the health effects of urban air pollution have been published. There is evidence, most consistently in studies with individual measurement of social factors, that more deprived populations are particularly sensitive to air pollution effects.

Methods Records from the 1996 New Zealand census were anonymously and probabilistically linked to mortality data, creating a cohort study of the New Zealand population followed up for 3 years. There were 1.06 million adults living in urban areas for which data were available on all covariates. Estimates of exposure to air pollution (measured as particulate matter with an aerodynamic diameter less than 10 μm, PM_{10}) were available for census area units from a previous land use regression study. Logistic regression analyses were conducted to investigate associations between cause-specific mortality rates and average exposure to PM_{10} in urban areas, with control for confounding by age, sex, ethnicity, social deprivation, income, education, smoking history and ambient temperature.

Results The odds of all-cause mortality in adults (aged 30–74 years at census) increased by 7% per 10 $\mu g/m^3$ increase in average PM_{10} exposure (95% CI 3% to 10%) and 20% per 10 $\mu g/m^3$ among Maori, but with wide CI (7% to 33%). Associations were stronger for respiratory and lung cancer deaths.

Conclusions An association of PM_{10} with mortality is reported in a country with relatively low levels of air pollution. The major limitation of the study is the probable misclassification of PM_{10} exposure. On balance, this means the strength of association was probably underestimated. The apparently greater association among Maori might be due to different levels of co-morbidity.

Anthropogenic air pollution is thought to cause a substantial global burden of disease, even if conservative assumptions are used for the analysis.[1] Most epidemiological studies of air pollution effects use a time series design, in which day-to-day changes in morbidity or mortality (usually in a single city) are related to day-to-day changes in air pollution exposure. These studies have the advantage of controlling for time invariant (or slowly varying) confounding factors by design. Time series studies provide extensive and detailed evidence of short-term air pollution effects, but are not the best basis for public health policy on air pollution.[2] This is because cohort studies suggest that long-term exposures have much greater public health impact than short-term exposures. Because it is difficult to estimate air pollution exposure for large populations over long periods, relatively few cohort studies have been reported and few such studies have been conducted outside north America and Europe.[3–18]

We report here the findings from a longitudinal study in New Zealand, based on the national census. In New Zealand, major sources of urban air pollution are home heating and motor vehicle emissions and levels of pollution are low by international standards. Typical annual average levels of particulate air pollution (particulate matter with an aerodynamic diameter less than 10 μm, PM_{10}) at monitoring stations in the main urban centres are in the range 15–25 $\mu g/m^3$. In this study, we analyse the spatial association between average PM_{10} exposure and mortality over the years 1996–9. We also investigate potential modification by social factors (age, sex, ethnicity, income, education and area level deprivation).

We hypothesise a priori that effects of PM_{10} on mortality will be stronger for cardiorespiratory causes of death than for all diseases, because these causes are most sensitive to air pollution effects.

METHODS

The New Zealand Census-Mortality Study is a population-wide cohort study, in which the cohort consists of the entire 1996 resident population (N=3 732 000) and the outcome of interest is mortality. For this analysis, records from the 1996 census were anonymously linked to 3 years of subsequent mortality data, creating a cohort study of the New Zealand population followed up for 3 years.[19 20] In New Zealand, urban areas are defined as cities having a population of at least 30 000 people; about half of the approximately 2000 census areas are classified as urban. Because the definition of 'urban areas' in New Zealand includes small towns and suburbs, many urban census areas have low levels of air pollution.

Record linkage

The method of record linkage has been described in detail elsewhere.[19] Briefly, probabilistic record linkage is a process used to link two files of records, in which records in one file have a corresponding record in the other file. Records from the first file are compared with records from the second file in order to find 'matching' record pairs (ie, two records belonging to the same individual). Census and mortality records were linked using date of birth (day, month and year as separate matching variables), country of birth, sex, ethnicity and address of usual residence. (Geocoded addresses were the most discriminatory matching variable.) Linkage was restricted to individuals aged 74 years or below at the time of the census. The proportion of mortality records linked overall was 78%,[20] and varied by sex, age, ethnicity and the deprivation index.[19] Weights were therefore applied to adjust for linkage bias, by strata of sex, age,

ethnicity, deprivation, rurality and cause of death.[21] For example, if 20 of 30 Maori men who died aged 45–64 years and living in moderately deprived (see below) rural areas of New Zealand were linked to a census record, each of the 20 linked records received a weight of 1.5 (30/20). The estimated proportion of links being true links was 97%.

Air pollution exposure estimation

Air pollution monitoring data are typically only available for a few sites and may not be representative of exposure in other areas. Detailed atmospheric dispersion modelling can be used to provide estimates of air pollution exposure for small areas, but these models are difficult to apply to large areas due to data and computer processing limitations. The method of air pollution exposure assessment has been described in detail elsewhere.[22] Briefly, the approach to modelling long-term average PM_{10} was as follows. Atmospheric dispersion modelling results were available for one city (Christchurch, population 300 000). These data were assumed to be representative of the spatial pattern of annual average PM_{10} exposures for small areas (census area units) in Christchurch.[23] Data on meteorological variables and indicators of air pollutant emissions for these areas were used as predictors of PM_{10} exposure in Christchurch, using regression models.

The predictors for the regression models were: census data on domestic heating; estimates of industrial emissions; and vehicle kilometres travelled within small areas. Because data for these predictor variables were available for all of New Zealand, we were able to extrapolate the empirical results for the Christchurch regression model to urban census area units throughout the country.

The resulting exposure estimates agreed well with multiyear averages of PM_{10} based on routine monitoring data for urban centres (1995–2001) (N=43; r^2=0.87). While we acknowledge that there may be considerable exposure misclassification, we assume that the PM_{10} estimates are representative of long-term average spatial patterns of exposure during the 1990s.

Ideally, for analyses with the New Zealand Census Mortality Study data we would have used the continuous PM_{10} estimates for individual census areas. However, because the New Zealand Census Mortality Study data contain census information at unit record level, access to the data is restricted and additional data can only be added if confidentiality is not compromised as a result. Assigning continuous PM_{10} estimates at census area level would have permitted many census areas to be uniquely identified. In order to maintain confidentiality, it was necessary to aggregate the PM_{10} estimates into quintiles before they could be merged with the census data. Quintiles of exposure were calculated based on the estimates for individual census areas. For this purpose, we assigned a PM_{10} estimate of 0 $\mu g/m^3$ to rural census areas where PM_{10} estimates were unavailable. The average PM_{10} level for all New Zealand census areas was 8.3 $\mu g/m^3$ (SD 8.4 $\mu g/m^3$) and the cut-off values between PM_{10} quintiles were 0.0, 0.5, 12.5 and 15.4 $\mu g/m^3$.

Individuals were assigned to quintiles of PM_{10} exposure depending on their census area of residence on census night. In order to avoid bias affecting analyses in smaller rural areas due to migration to cities following the development of disease, we restricted the analyses to the urban population. This resulted in 1 065 645 observations[i] with complete data. Following restriction to the urban population, the lowest two quintiles of exposure had relatively few observations (table 1). For this reason, and given the small variation in PM levels between quintiles 1 and 2, we combined the two lowest quintiles of PM_{10} to produce four categories in total. The estimated mean PM_{10} levels for these categories were 0.1, 7, 14 and 19 $\mu g/m^3$ (long-term averages).

Ethnicity and socioeconomic position are strong predictors of mortality in New Zealand, and potential confounders of the association of air pollution with mortality. We assigned each respondent to a mutually exclusive ethnic group using a prioritisation system commonly used in New Zealand: Maori, if any one of the responses was Maori; Pacific, if any one response was Pacific but not Maori; and the remainder non-Maori non-Pacific (mostly New Zealand European). Smoking status was reported in the census using the categories: never smoker, ex-smoker, or current smoker.[24] Socioeconomic position was characterised as: total household income, with adjustment for the number of children and adults in the household to allow for economies of scale, log-transformed having first set all values of less than NZ $1000 to equal NZ$1000;[20] highest educational qualification (higher than school, school, or none); and neighbourhood deprivation measured by the NZDep index (in quintiles).[25] This index of deprivation within small geographical areas was calculated using census data on socioeconomic characteristics (eg, car access, tenure and receipt of benefits) at aggregations of approximately 100 people, and assigned to mortality data by use of address.

As climate is correlated with PM_{10}, and associated with mortality (independently of PM_{10}), temperature has the potential to confound air pollution effects. Therefore, we also included estimates of long-term average minimum temperature (in addition to the above sociodemographic factors) in the models, also in quintiles based on place of residence. These data were derived from an interpolated temperature surface.[26] Mean values for the minimum temperature quintiles were 0.2, 2, 4, 5 and 7°C.

Logistic regression analyses were conducted to investigate associations between all-cause and cause-specific mortality rates and average exposure to PM_{10}, with control for confounding by age, sex, ethnicity, social deprivation, income, education, smoking history and average minimum temperature. As death is a rare outcome over 3 years for the age groups included in the analysis, logistic regression results differ very little from those from either Poisson or Cox proportional hazards modelling. Initial models incorporated PM_{10} categories as dummy variables, and given a reasonably linear dose–response, final models used the estimated mean PM_{10} for these four categories (see above). In order to facilitate comparison with overseas findings, we report results for adults aged 30–74 years. In final models, age was included as a linear plus a squared term, and the income variable was natural-log transformed. All other covariates were included as dummy variables (table 2).

Any association of air pollution with mortality might be modified by social and environmental factors, due to different vulnerability or intensity of exposure to air pollution. We tested for interaction both by stratification and by inclusion of interaction terms.

Finally, we conducted a sensitivity analysis by restricting the dataset for analysis to those people who had lived in the same geographical unit at the 1991 census as they did at the time of exposure assignment (1996) in this cohort study. This restriction should reduce exposure misclassification by excluding those people who have not been exposed to the same level of PM_{10} for at least 5 years.

[i]All counts were randomly rounded to a multiple of 3 in order to preserve confidentiality, as per Statistics New Zealand protocol. For this reason, subtotals may not match across categories. Note that regression analyses, however, were undertaken on actual unit record data.

Table 1 Summary data

	Census counts		Deaths*						
	All	Complete data†	All causes	Natural causes‡	Cardiovascular	Respiratory	Lung cancer	Injury	Other
Age (decade)									
30	443 610	347 772	1152	552	168	48	27	453	483
40	375 699	291 885	1941	1482	567	156	96	294	927
50	257 274	199 044	3645	3183	1155	474	309	186	1830
60	199 599	157 185	7794	7056	2814	1410	759	186	3387
70	88 275	69 750	6237	5664	2574	1125	495	111	2421
Sex									
Male	661 314	515 007	12 468	10 656	4881	1917	1044	903	4770
Female	703 137	550 635	8301	7281	2397	1296	639	327	4281
Ethnicity									
Maori	121 026	87 825	2253	1908	861	414	243	180	801
Pacific	59 574	35 916	771	651	315	84	54	42	327
European	1 183 851	941 904	17 745	15 378	6096	2715	1389	1011	7923
Income (tertiles)									
Missing	255 492								
Lower	375 462	356 853	11 973	10 476	4410	2115	1038	537	4908
Middle	363 870	350 490	5571	4779	1878	774	435	375	2547
Upper	369 627	358 302	3225	2679	990	318	210	318	1596
NZDep (quintiles)									
Missing	768								
Rich	352 656	289 071	3918	3387	1278	477	252	234	1926
	278 199	225 192	3729	3240	1254	531	291	204	1740
	257 613	204 690	4014	3477	1389	612	333	237	1779
	247 107	189 117	4473	3867	1608	774	405	267	1827
Poor	228 111	157 572	4638	3963	1746	819	402	288	1779
Education									
Missing	66 492								
None	443 736	353 280	10 446	9150	3789	1860	981	480	4317
School	344 745	286 479	4584	3915	1536	660	330	300	2091
Higher	509 478	425 883	5742	4869	1953	693	372	450	2646
Smoking									
Missing	100 926								
Current	283 992	232 995	5559	4713	1998	1317	789	450	1794
Past	328 548	283 470	7812	6975	2784	1425	690	300	3300
Never	650 991	549 180	7401	6246	2493	471	207	477	3957
Temperature (°C)									
0.2	168 261	137 571	3003	2625	1041	480	267	150	1335
2	224 265	176 730	3849	3300	1329	648	315	234	1638
3	249 363	199 971	4131	3576	1491	636	351	255	1752
5	354 780	275 739	5172	4419	1809	807	414	330	2223
7	367 788	275 634	4614	4014	1611	639	333	264	2100
Particulate ($\mu g/m^3$)									
0.1	72 102	57 213	999	861	348	135	69	51	462
7	319 914	251 109	4665	3975	1659	702	375	312	1992
14	506 913	388 134	7083	6132	2472	1050	549	417	3141
19	465 522	369 189	8022	6969	2799	1323	693	444	3453
Totals	1 364 451	1 065 645	20 769	17 937	7278	3210	1686	1224	9048

*Weighted counts, for deaths occurring among the census respondents with complete data.
†The complete dataset is that used for final analyses in this paper.
‡All causes of death excluding accidents and injury.

RESULTS

When PM_{10} was modelled using dummy variables, there was an approximately linear increase in mortality with increasing average PM_{10} exposure (figure 1). Three models are shown: (1) adjusting for sex, age and ethnicity on all observations (N=1 364 454); (2) the same model, but restricted to observations with non-missing data on other covariates (N=1 065 645; a test of any selection bias compared with model 1); (3) fully adjusted model (N=1 065 645; a test of possible confounding compared with model 2). There is no meaningful difference between models 1 and 2, suggesting no selection bias when restricting analyses to those with full data on covariates. Adjusting for potential confounders in model 3 attenuated the OR for all non-referent groups, although the relative differences between quintiles 3, 4 and 5 were not much reduced. That is, adjusting for confounders mostly closed the gap between the referent group and quintile 3.

Considering the preferred model 3, the OR increased monotonically and linearly with increasing PM_{10} level, and the 95% CI for the two highest quintiles excluded 1.0. Given the approximately linear association of PM_{10} with mortality described above, subsequent models incorporated PM_{10} as a linear term,

Table 2 Logistic regression model including all variables listed as independent variables (all deaths, all ethnicities: N= 1 065 645)*

	OR	95% CI lower	95% CI upper
Particulate			
PM_{10} ($\mu g/m^3$)	1.007	1.003	1.010
Temperature quintile (base: lowest)			
2	0.967	0.915	1.022
3	0.987	0.932	1.046
4	0.957	0.908	1.010
5	0.908	0.861	0.959
Age	1.088	1.071	1.105
Age squared	1.000	1.000	1.000
Natural log of income	0.819	0.799	0.839
Sex (base: male)			
Female	0.598	0.579	0.618
Ethnicity (base: Māori)			
Pacific	0.813	0.731	0.905
European	0.500	0.472	0.529
Deprivation quintile (base: least deprived)			
2	1.078	1.025	1.134
3	1.173	1.116	1.234
4	1.330	1.265	1.399
5	1.548	1.469	1.632
Smoking (base: current smoker)			
past	0.706	0.677	0.736
never	0.498	0.477	0.519
Education (base: none)			
school	0.933	0.895	0.971
higher	0.860	0.828	0.894

*Rounded; for logit form of model, constant −6.528 (−7.036 to −6.020).

using the average PM_{10} for each category) (tables 2 and 3). Table 2 shows the same model as in figure 1, except for the continuous treatment of PM_{10}. The OR of 1.007 corresponds to a 1 unit increase in PM_{10}, which equates to an increase of 7% (95% CI 3% to 10%) in the odds of all-cause mortality in adults (aged between age 30 and 74 years at census) per 10 $\mu g/m^3$ increase in long-term average PM_{10} exposure.

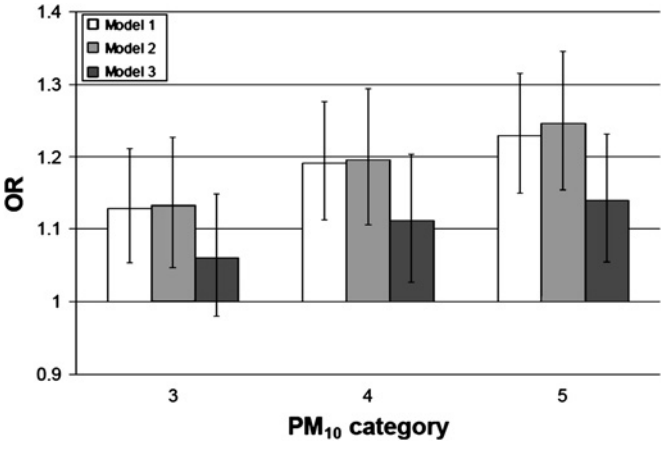

Figure 1 OR and 95% CI of all-cause mortality for people living in the three non-referent PM_{10} categories, compared with quintiles 1 and 2 combined. Model is for sexes combined, restricted to adults aged 30–74 years on census night, urban population, with covariates as follows: model 1: age, sex, ethnicity, all data, N=1 364 454; model 2: age, sex, ethnicity, data with non-missing values for covariates in model 3, N=1 065 645; model 3: age, sex, ethnicity, deprivation, income, education, smoking, temperature, N=1 065 645.

Table 3 Findings by subgroups of ethnicity and cause of death (model 3, fully adjusted)

	N*	OR (per 1 $\mu g/m^3$ PM_{10})	Lower 95% CI	Upper 95% CI
Model				
All deaths, all ethnicities	1 065 645	1.007	1.003	1.010
All deaths, living in the same census area as for the 1991 census	601 401	1.008	1.004	1.011
All natural causes, all ethnicities (excludes accidents and injury)	1 063 563	1.007	1.003	1.010
All natural causes, European	940 107	1.006	1.003	1.010
All natural causes, Maori	87 615	1.018	1.007	1.029
Analyses by specific cause of death, based on ICD9 codes†				
Lung cancers (ICD 162)	1 050 222	1.015	1.004	1.026
Respiratory disease (ICD 162, 470–478, 490–519)	1 051 464	1.013	1.005	1.021
Cardiovascular disease (ICD 393–438)	1 054 731	1.006	1.001	1.011
Accidents and injury (ICD 800–949)	1 049 646	1.004	0.990	1.018
Other, includes unspecified‡	1 056 288	1.005	1.001	1.010

*Rounded.
†Note: Models included persons dying from the specified causes (coded 1) and people presumed alive at the end of follow-up (coded 0), but excluded persons dying from other causes. Respiratory disease includes lung cancers.
‡'Other' deaths are all those deaths (including unspecified) that are not included among lung cancer, respiratory, cardiovascular disease or accidents.
ICD, International Classification of Diseases.

We found stronger effects of PM_{10} among people who lived in the same census area in 1991 (at the time of the previous census), 8% (4% to 12%) per 10 $\mu g/m^3$ increase in PM_{10}. By cause of death, the association was similar for all natural causes, 7% (3% to 10%), but substantially stronger for respiratory deaths (including lung cancers), 14% (5% to 23%) and for lung cancers, 16% (4% to 29%). For cardiovascular disease, 6% (1% to 12%) and for 'other and unspecified' causes of death, 5% (1% to 10%), the association was marginally significant; while for accidental deaths and injuries, the association was non-significant, 4% (−9% to 20%) (table 3).

Considering interaction with social variables, there was an apparently stronger association of PM_{10} with all-cause mortality among Maori, 20% (7% to 33%). However, the 95% CI for Europeans overlapped that for Maori, 7% (3% to 10%). A Wald test for interaction between ethnicity and PM_{10} was not significant (p=0.12). There were no statistically significant interactions with age, sex, income, deprivation, educational status or average temperature. Mortality was lower among people living in warmer census areas, and the difference between the lowest and the highest quintile of annual average temperature was statistically significant.

DISCUSSION

Using linked mortality and census data, we report a significant positive association between estimated long-term exposure to air pollution (PM_{10}) and mortality in New Zealand urban areas. This setting includes approximately 75% of the New Zealand population, who are exposed to relatively low levels of PM_{10} compared with other countries.

Selection bias and confounding seem unlikely to explain our results. There is no evidence of selection bias (figure 1). The results persist after controlling for plausible confounders, including multiple measures of socioeconomic position and smoking. It seems unlikely that mismeasured or unknown confounders might explain the remaining association.

There is likely to be substantial misclassification of the air pollution exposure. Most of this misclassification was probably

non-differential by mortality risk (meaning we have probably significantly underestimated the true strength of association). There is potential differential misclassification of PM_{10} by mortality risk in our study, because our assessment was based on modelling in one city using proxies, including domestic heating, estimates of industrial emissions and vehicle kilometres travelled within small areas. If these proxies are not such reliable predictors of PM_{10} in other cities, and are (say) correlated with socioeconomic position, then it may be that our PM_{10} estimates are also capturing aspects of socioeconomic exposure. However, the fact that an association remained after extensive control of socioeconomic factors, including individual level income and education, makes this an unlikely explanation of the results. The use of modelled estimates of PM_{10} exposure will tend to smooth the data and reduce the resulting CI. However, this should not affect the central effect estimates.

It is possible that less healthy people might migrate towards health services (or other service amenities) that happen to be in more polluted areas—a form of reverse causation or endogeneity. However, we think this is unlikely to be an important factor as New Zealand cities are relatively small (maximum 1.4 million), and most suburbs in New Zealand's main cities have relatively good access to hospitals.

The odds of all-cause mortality in adults (aged between 30 and 74 years at census) increased by 7% (95% CI 3% to 10%) per 10 $\mu g/m^3$ increase in average PM_{10} exposure. Our observations are consistent with an increasing number of studies of long-term exposure to particulate matter and mortality.[3–10 13–18]

The original US Six Cities Study reported an adjusted mortality rate ratio of 1.27 (95% CI 1.08 to 1.48) for the most polluted compared with the least polluted city, corresponding to 18.2 and 46.5 $\mu g/m^3$ PM_{10}, respectively[3]—equivalent to an increase in mortality of approximately 10% per 10 $\mu g/m^3$ PM_{10}. In the US Nurses Health Study, there was a 16% (5% to 28%) increase in all-cause mortality per 10 $\mu g/m^3$ PM_{10}.[14] It is not yet clear to what extent the heterogeneity in reported dose response in those studies is related to differences in the accuracy of exposure measurement, to differences in the toxicity of complex mixtures of pollutants at differing levels of exposure, and/or differences in the sensitivity of exposed populations.

The exposure measures in other studies are not directly comparable. Recent studies use the more specific measure $PM_{2.5}$ (particulate matter with an aerodynamic diameter less than 2.5 μm) rather than PM_{10}, whereas several European studies use black smoke or total suspended particulates as the exposure measure. The association between particulate air pollution exposure and mortality is usually found to be strongest for finer fractions, such that the dose response for $PM_{2.5}$ is greater than for PM_{10}, which in turn is greater than for total suspended particulates. An extended follow-up of the US Six Cities Study reported a 14% (6% to 22%) increase in mortality per 10 $\mu g/m^3$ $PM_{2.5}$,[9] whereas the American Cancer Society Study reported 6% (2% to 11%) and the Nurses Health Study 26% (2% to 54%), while coarse particulate matter exposure ($PM_{10-2.5}$) was not associated with an increase in mortality in that study.[27] An analysis based on electoral wards in the UK found a 1.3% (1% to 1.6%) increase in all-cause mortality per 10 $\mu g/m^3$ increase in black smoke.[10] In 18 regions in France, the PAARC study reported a 7% (3% to 10%) increase per 10 $\mu g/m^3$ increase in black smoke and a 5% (2% to 8%) increase for total suspended particulates.[7] In The Netherlands, there was a 5% (0% to 10%) increase in mortality per 10 $\mu g/m^3$ increase in black smoke.

Our study assessed the association of PM_{10} and mortality over 3 years. In the US Nurses Health Study, mortality was most strongly associated with average PM_{10} exposures in the 24 months before death. In the UK, the association, although weak, was stronger for exposure in the 4 years before death. However, a re-analysis of the American Cancer Society Study found no clear effect of exposure period.[17]

A priori, we hypothesised that the association of PM_{10} with mortality in our study would be stronger for a cohort restricted to those census respondents who lived in the same census area at the time of the 1991 census. We also hypothesised that the association of PM_{10} with mortality would be stronger for cardiorespiratory deaths. Our results were consistent with these a priori hypotheses, strengthening the ability to make causal inference.

There is some evidence, most consistently in studies with individual measurements of social factors, that more deprived populations are particularly sensitive to air pollution effects.[5 11 12 28 29] Our ability to detect a true difference by ethnicity in sensitivity to air pollution was limited by the relatively small Maori population. Although not significant, the difference in our central effect estimates for European and Maori was substantial: 7% versus 20%. The reasons for this difference, if real, are not clear. This might reflect a higher prevalence of pre-existing cardiorespiratory disease among Maori, or a difference in the toxicity of air pollution to which different ethnic groups are typically exposed. Alternatively, this finding may reflect biases in exposure estimates. We found no other suggestion of interaction of social factors with PM_{10} in the association with mortality.

CONCLUSION

In this longitudinal study we report an association of PM_{10} with mortality, consistent with that reported elsewhere, in a country with low levels of air pollution. The major limitation of our study is the probable misclassification of the PM_{10} exposure. On balance, this means we have probably underestimated the strength of association. The study design has several strengths, including national population coverage and good control of confounding. We found that the association was, as hypothesised a priori, stronger for cardiorespiratory deaths and people with less residential mobility.

What is already known on this subject

Relatively few cohort studies of the health effects of urban air pollution have been published, particularly outside north America and Europe. We report here findings from a longitudinal study in New Zealand, based on the national census. There is evidence, most consistently in studies with individual measurement of social factors, that more deprived populations are particularly sensitive to air pollution effects.

What this study adds

We found an association of PM_{10} with mortality in a country with relatively low levels of air pollution. There was some evidence that Maori may have greater susceptibility to life-shortening effects of air pollution. This might reflect a higher prevalence of pre-existing cardiorespiratory disease among Maori.

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Competing interests None.

Contributors TB conceived the analysis and contributed to the design, interpretation and revision of drafts. SH performed the analyses, drafted the text of the paper and contributed to the design and interpretation of results. SH had full access to the data, and takes responsibility for the integrity of the analysis and the accuracy of the results. AW contributed to study design, the interpretation of results and revision of drafts.

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