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Susceptibility to air pollution effects on mortality in Seoul, Korea: A case-crossover analysis of individual-level effect modifiers

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Abstract

Air pollution's mortality effects may differ by subpopulation; however, few studies have investigated this issue in Asia. We investigated susceptibility to air pollutants on total, cardiovascular, and respiratory mortality in Seoul, Korea for the period 2000 – 2007. We applied time-stratified case-crossover analysis, which allows direct modeling of interaction terms, to estimate susceptibility based on sex, age, education, marital status, and occupation. An interquartile range increase in pollution was associated with odds ratios of 0.94 (95% confidence interval, 0.25 – 1.62), 2.27 (1.03–3.53), 1.94 (0.80 – 3.09), and 2.21 (1.00 – 3.43) for total mortality and 1.95 (0.64 – 3.27), 4.82 (2.18 – 7.54), 3.64 (1.46 – 5.87), and 4.32 (1.77 – 6.92) for cardiovascular mortality for PM_{10}, nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO), respectively. Ozone effect estimates were positive, but not statistically significant. Results indicate that some populations are more susceptible than others. For total or cardiovascular mortality, associations were higher for males, those 65 – 74 years, and those with no education or manual occupation for some pollutants. For example, the odds ratio for SO_2 and cardiovascular mortality was 1.19 (1.03 – 1.37) times higher for those with manual occupations than professional occupations. Our findings provide evidence that some populations are more susceptible to the effects of air pollution than others, which has implications for public policy and risk assessment for susceptible subpopulations.

Keywords

air pollution; effect modifiers; mortality; time-stratified case–crossover analysis; susceptible subpopulations

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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INTRODUCTION

Numerous epidemiological studies have demonstrated short-term effects of ambient air pollution on daily mortality in many parts of the world.[1,2] Some studies indicated that certain populations are more vulnerable than others, based on individual or community level characteristics including socioeconomic status (SES).[3 – 5] Which populations are most vulnerable to air pollution's effects on health may vary by region. However, in Asia including Korea, only a limited number of studies have examined how air pollution's effects differ among subpopulations. Most epidemiological studies addressing this issue were conducted in Europe or North America.[6] Thus, more research about the susceptibility of various populations to ambient air pollution for other regions is needed, especially as the characteristics of air pollution and of populations may differ by country.

In Korea, research on which populations are susceptible to air Pollution's health effects is limited, and only some a few potential effect modifiers have been investigated. Yi et al.[7] found higher risk for mortality and morbidity among women and the elderly in Seoul, Korea in relation to exposure to particulate matter with aerodynamic diameter ≤10 μm (PM_{10}). Other studies in Korea explored effect modification of air pollution's associations with mortality by age for respiratory, stroke, and total mortality[8,9] and by sex for stroke mortality.[9] Few previous studies in Korea investigated whether some populations are more susceptible to cardiovascular mortality from air pollution. Further, no studies of Korea examined effect modification by marital status or socio-economic characteristics, although risk estimates could differ by SES due to baseline health care status, occupational exposure, and related factors. Previous studies suggest that marital status may be related to SES[10] and groups with lower SES generally have increased vulnerability to air pollution due to several factors such as poor nutrition, inadequate access to medical care, high prevalence of preexisting disease, and higher exposure to air pollution.[11,12]

To date, many studies have used time-series and case-crossover analyses to investigate the acute effects of ambient air pollution on daily mortality.[13,14] Most studies examining susceptibility used stratification methods to explore effect modifiers.[3,15,16] These approaches are useful, but have limitations including reduced statistical power and the inability to detect interaction effects directly. Such methods may be problematic due to differences in the distributions of other covariates across strata. Relatively few studies have applied a case-crossover analysis with the direct modeling of an interaction term rather than multiple stratified analyses.

We conducted a time-stratified case-crossover analysis to examine the association between ambient air pollution and mortality (total, cardiovascular, and respiratory) in Seoul, Korea for the period 2000 – 2007. We evaluated whether individual characteristics (sex, age, education, marital status, and occupation) modify the air pollution and mortality relationship.

METHODS

Study Subjects

Individual-level mortality data for Seoul between 1 January 2000 and 31 December 2007 were obtained from the National Statistical Office, Republic of Korea. We restricted study subjects to adults (≥ 35 years) as our covariates of interest include education, marital status, and occupation. A similar approach was applied in previous studies.[4,17] Mortality data included date of death, cause of death, sex, age, educational level, marital status (never married, married, divorced, widowed, and unknown), and occupation. This information was used to investigate effect modification of the associations between air pollution and

mortality. We classified mortality data into all-causes of death except external causes (International Classification of Diseases, ICD-10, A00-R99), cardiovascular causes (ICD-10, I00-I99), and respiratory causes (ICD-10, J00-J99). Analyses were stratified by cause of death. Data were categorized for age (35 – 64, 65 – 74, and ≥ 75 years); educational level (none, ≤ 12, > 12 years, and unknown); and occupation (professional as executive/manager, expert, engineer, office worker, or service job; manual as agriculture/fishery/forestry, technical service, mechanic, or physical labor; unemployed including housewife; and unknown).

Pollution and Weather Data

Hourly ambient air pollution levels in Seoul were obtained for 27 monitoring stations operated by the Department of Environment, Republic of Korea. Each monitor has routine measurements for PM_{10}, NO_2, SO_2, ozone (O_3), and CO using standardized reference methods for the whole study period.[18] We used 24-h averages as the exposure index, by first averaging hourly values across all monitors for each day and then calculating 24-h values, except for O_3 and CO, for which we calculated the maximum daily 8-h moving average. The daily 8-hour metric for O_3 and CO was chosen based on previous research.[19] We assigned air pollution exposure as the average value across all monitors for the given lag period for each individual.

The National Meteorological Administration, Republic of Korea, provided hourly measurements of ambient temperature and relative humidity, and 3-h measurements of barometric pressure for Seoul during the study period. We converted weather data into 24-h values by averaging the 24 hourly values for temperature and relative humidity or averaging the eight 3-hour values for barometric pressure for each day.

Statistical Analysis

We applied a case-crossover design that was first proposed by Maclure[20] and has been used to analyze acute health effects of air pollution. This approach is a special type of case-control design in which each case acts as his or her own control and control days are chosen as days on which the event did not occur. This method has the advantage of controlling for potential confounding from fixed individual-level characteristics (e.g., physiological status).[13] There are several referent selection strategies in case-crossover design, and recently, time-stratified referent selection was developed to avoid selection bias.[21] In this method, time is divided into fixed strata and the days in each stratum are considered for referents. We selected control days matched on the same rounded daily temperature value in the same month of the same year when a death (i.e., case) occurred.

We examined the effect of air pollutants with single-day lags (lag0, lag1, and lag2) and multi-day lags (lag01, lag12, and lag02) by cause-specific mortality (total, cardiovascular, and respiratory). Lag0 represents exposure on the same day, lag1 represents the previous day, and multi-day lags represent averages across the same day and previous days. For example, lag01 represents the average across the same day and previous day. For O_3, we generated year-round and warm season (May – September) estimates, separately.

To examine effect modification, we separately fitted models with an interaction term for each air pollutant and potential effect modifier (sex, age, education, marital status, or occupation). We then linearly combined the coefficients for reference group and interaction terms to assess the associations by the subcategories of each modifier. We reported statistical significance of the comparisons between the effect modifiers and their reference groups. Each effect modifier was analyzed separately. We also examined multiple susceptibilities (age and education, age and sex, and education and sex) by stratifying by one

group and performing analysis with an interaction model for the other group. Relative humidity, barometric pressure, and day of the week were included in the models. We fitted conditional logistic regression models to estimate associations between ambient air pollution and mortality and examine effect modification using PROC PHREG in SAS 9.2 (SAS Institute, Cary, NC, USA). Results were expressed as the percentage change in risk of mortality per interquartile range increase of each pollutant.

RESULTS

Table 1 shows summary statistics for study subjects, pollutants, and weather. Analysis included 261,952 deaths including 15,523 from respiratory disease and 73,356 from cardiovascular disease. Supplementary Table 1 provides the distribution of population characteristics by sex and age. Patterns by cause of death were similar in males, females, and each age group. Older persons and females were less educated. The primary marital status of older deceased females was widowed, whereas older males were married. On average, females lived longer than males, with an average age of death of 75.2 years, compared with 66.5 years. Air pollutant concentrations were not highly correlated with each other (range of the Pearson correlation coefficients: −0.19 to 0.74).

We first estimated the association between air pollution and mortality for the overall population for multiple lag structures (Supplementary Table 2). For subsequent analysis on susceptibility, we selected the lag with the most certain effect estimates (largest *t*-statistics), separately for each pollutant and cause of death. The lagged effect of each pollutant differed by cause of death. The results for year-round O_3 and warm-season O_3 were similar, although most warm-season central estimates were slightly higher. Table 2 shows results for the selected lag structures. All pollutants exhibited statistically significant effects for total or cardiovascular mortality, except O_3. For respiratory mortality, associations were positive but not statistically significant. The effects of O_3 were not statistically significant with any mortality outcomes. For subsequent analysis for effect modification, we investigated the pollutants exhibiting associations in Table 2; however, results for year-round O_3 are presented in Supplementary Tables 3 – 5 and Supplementary Figure 1.

For pollutants that demonstrated associations (PM_{10}, NO_2, SO_2, and CO), we investigated whether individual-level characteristics (sex, age, education, marital status, or occupation) modified associations with cause-specific mortality (Table 3, Supplementary Table 3 presents results for O_3). Effect estimates were higher among men in all analyses except PM_{10} in total mortality, although the 95% intervals of estimates for men and women overlapped. Effects of pollutants for those 65 □ 74 years were larger than for other age groups for all results except for SO_2 and CO for cardiovascular mortality for which the highest risk was for those 35 – 64 years. The effects of all four air pollutants on both total and cardiovascular mortality for those 65 – 74 years were statistically significant. We did not find consistent gradients of effect with educational for any mortality outcomes. The highest effect was observed in the unknown education category for many analyses, including all pollutants for total mortality. Consistent patterns of effect by marital status were not observed; however, divorced persons had more risk than other marital status groups for cardiovascular mortality for NO_2, SO_2, or CO. Effects on total and cardiovascular mortality were higher in the manual occupation group for NO_2, SO_2, and CO, and in the unknown occupation category for PM_{10}. We tested the significance of the interaction terms (see footnotes in Table 3). These results indicate whether the air pollution effect on mortality for one subpopulation is statistically different than for another group (e.g., males versus females), indicating effect modification. For example, the odds ratio for SO_2 cardiovascular mortality was 1.05 (1.01, 1.09) times higher for males than females (Table 3). Other

susceptibilities with statistically significant results were higher risk estimates for SO_2 or CO and total mortality for those with manual occupations than professional occupations.

Because of differences in the demographic distributions for combinations of sex, age, and education, we examined effect modification by multiple susceptibilities (age and sex, age and education, and sex and education). Figure 1 and Supplementary Table 4 show associations between air pollution and mortality by age, for each sex (see Supplementary Figure 1 for O_3 results). For both men and women, similar patterns by age were observed, with the highest effect generally for those 65 – 74 years, although not in all cases.

Supplementary Table 4 provides associations by educational categories for each sex. For both sexes, the highest effect estimates were most often in the unknown education group compared with other education categories. Of those with known education levels, the highest effects were for those with no education or < 12 years of education for both sexes.

Table 4 provides risk effect estimates by education stratified by age group (see Supplementary Table 5 for O_3 results). The highest association remains for the no or unknown education group compared with other education categories in the 35 – 64 year age group, except for associations for PM_{10} and respiratory mortality. Effect estimates for NO_2 and total mortality were 1.13 (1.01, 1.26) times higher for those with no education than those with > 12 years education for those 35 – 64 years. In the oldest age category, effect estimates for the most educated group were higher than for other education groups for some pollutants (e.g., NO_2 and SO_2).

DISCUSSION

We investigated whether some populations are more susceptible to mortality impacts from air pollutants in Seoul, Korea using a temperature-matched case-crossover analysis. Our results suggest that individual factors may modify air pollution's mortality effects in this region. Specifically, associations for total or cardiovascular mortality were higher for males, those 65 – 74 years, and those with indicators of low SES such as no education or manual occupation for some pollutants.

Our findings of associations between air pollution and total and cardiovascular mortality are consistent with several previous studies.[1,22] Guo et al.[23] applied case-crossover and time-series analyses in China, finding that both methods produced positive associations between pollutants (PM_{10}, SO_2, and NO_2) and cardiovascular mortality. The US studies found associations between O_3 and daily death,[4,24] whereas our results were positive, but not statistically significant. We did not find statistically significant effects of any pollutant on respiratory mortality; however, results should be viewed in the context of the smaller number of respiratory deaths. Our findings are similar to a previous Korean study examining PM_{10} and mortality from non-accidental causes and cardiovascular and respiratory disease, which reported positive and statistically significant effects except for respiratory mortality.[7]

We found that males were more susceptible to pollution-mortality effects than females except for the relationship between PM_{10} and total mortality. The findings of previous studies on sex-specific effects were inconsistent. Chen et al.[2] observed that estimates for PM_{10} and CO on cardiovascular mortality in males were statistically significant and slightly higher than in females in China. Other work found higher risk of mortality with PM_{10} among women compared with men in the United States.[15] Higher effects of air pollution (PM_{10}, NO_2, SO_2, and O_3) on total mortality were observed in females than males in Shanghai, China.[25] However, Ren et al.[4] did not find evidence that the associations were significantly modified by sex in eastern MA, USA. Different effects by sex may relate to smoking rates in females,[26] smaller airway and greater airway reactivity in females,[27]

greater deposition of particles in lungs of women,[28] and lower SES for females.[25] A recent review found that most studies observed higher $PM_{2.5}$ and NO_2 effects in women for adults, with higher effects for males in early childhood.[29]

In this study, the 65 – 74 year age group was at a greater risk for total mortality from air pollution than other age groups. Our findings by age differ from most previous studies from other regions. Zeka et al.[3] found that those > 75 years were significantly more affected by PM_{10} for all-cause mortality, compared with those 0 – 65 or 56 – 75 years in the United States. An increasing proportion of the population at ≥ 75 years was associated with a greater PM_{10} effect in the United States and Europe.[30] Regarding our finding, one possible explanation is a healthy survivor effect. According to some studies, when individuals reach their 70 s, many of the ill persons have died or been placed under institutionalized care, and therefore, the surviving community cohort is healthier.[31,32] Thus, the older population includes people who were healthy enough to have survived to later ages because the most susceptible persons have already died. Consistent with our finding, Serinelli et al.[33] examined effects of outdoor PM_{10} on out-of-hospital coronary deaths in Italy by age (35 – 64, 65 – 74, 75 – 84, and > 85 years), reporting the strongest effect for those 65 – 74 years.

We also found the highest risk for cardiovascular mortality in the 35 – 64 year age group for some pollutants (e.g., SO_2, CO). Within this age group and cause of death, effects were higher and more significant among males than females. This finding may be related to more physical activity causing higher exposure to ambient pollutants, or a higher possibility of an occupation associated with cardiovascular disease among younger males.[26] Yi et al.[7] found that those 16 – 65 years had the greatest risk for PM_{10} and cardiovascular hospital admissions in Seoul.

We identified susceptibilities to air pollution's association with mortality by SES. The highest effect was observed in the unknown education category for total mortality compared with other education groups. For cardiovascular mortality, we found higher effects for those with no or unknown education for all air pollutants except CO. Effects of all air pollutants on total and cardiovascular mortality were higher in the manual or unknown occupation groups. Higher effects in the unknown education or occupation category may relate to lower SES. People with low SES are more likely to report their status as unknown.[34] Those with no education, manual occupation, unknown education, or unknown occupation are more likely to have lower SES. Increased vulnerability due to low SES may result from several factors such as limited access to health care, poor nutrition, poor baseline health status, and high prevalence of preexisting disease.[3,11] Persons with lower SES are more likely to live in poorer and more disadvantaged neighborhoods, possibly with higher exposure as a result of living closer to busy roadways with increased traffic density and poor housing conditions.[25] Those with higher educational levels may have improved knowledge and ability to manage personal health and obtain access to health care, higher income, better jobs, and stronger social connections that enhance health. Educational attainment may also relate to differences in occupational exposure, living conditions, or baseline health status.[35]

Overall, our findings for susceptibility by SES are consistent with previous results. The effects of particulate matter and SO_2 on mortality were reported to be more pronounced among those with low educational attainment or SES.[3,36] Ou et al.[37] found that the health effects of air pollutants (PM_{10}, NO_2, SO_2, and O_3) were significantly associated with occupational class and that the greatest mortality risk from each pollutant was found in blue-collar workers compared with the white-collar and never-employed groups in Hong Kong. A study in France reported that blue-collar workers had a greater risk of death associated with black smoke.[16]

Little research and no previous studies in Korea have been conducted to assess how marital status affects susceptibility to ambient air pollution. Our study found that widowed or divorced people had higher risk to air pollution for cardiovascular mortality than other marital status groups. Higher effects in the widowed or divorced groups may be related to SES. In Korea, women who experienced marital disruption were more likely to have lower income.[10] Krewski et al.[38] found that the risk of death associated with fine particles was less for married persons than others in the United States. On the other hand, Ren et al.[4] did not find evidence that the association between O_3 and non-accidental mortality was modified by marital status (never married, married or separated, widowed, and divorced) in eastern MA, USA.

We evaluated susceptibility using individual-level education and occupation as indicators of SES. Only a few studies have investigated effect modification of air pollution effects by SES indicators such as occupation,[39] although several studies have used education attainment SES measures and also studied race, which relates to SES.[15,35] Some of the potential effect modifiers are related (e.g., sex and education). We considered multiple susceptibilities, by sex, age, and education. Although we considered multiple indicators of SES at the individual level, this may not fully reflect complete socioeconomic conditions. Actual SES is related to many other factors such as income, housing type, the type and age of vehicles, the air conditioning system at the residence, and previous history of socioeconomic conditions. Further, true socioeconomic conditions are also related to community-level SES. Bell and Dominici[5] found that community-level SES modified the association between ambient O_3 and mortality in the United States. Similarly, in Korea, area-level SES modified the association between air pollution and health outcomes.[40] Thus, further studies based on more precise exposure estimation (e.g., personal exposures as opposed to community-wide averages) and considering both individual and community-level SES are needed to investigate the overall effects of SES as effect modifiers of air pollution-mortality associations.

A further limitation in this study is that the occupational information collected from death certificate did not represent lifetime work histories as the information was based on the job when the death occurred. The occupation data in this study were based on specific categories and may not represent the full range of possible occupations. Thus, we categorized occupation into four groups (professional, manual, unemployed, and unknown) to minimize the possibility of misclassification. Further work using data with full occupational history and detailed categories would provide more understanding of how occupation affects susceptibility to the impacts of air pollution.

The strengths of this study include the use of a case-crossover design with interaction terms to identify populations susceptible to air pollution's mortality impacts, by examining effect modification with individual-level data. An advantage of this approach is that all time-invariant variables were controlled by the study design, and the method allows for the direct modeling of effect modification through an interaction term rather than with multiple stratified analyses. In the stratification studies, the different effect estimates across subpopulations might be interpreted as differences in the distributions of the risk factors in subpopulations. Moreover, the stratified analysis design does not directly examine an interactive effect but simply main effects in terms of statistical theory.[4] Our method may not be beneficial in all situations. For example, a limitation of the case-crossover approach is difficulty in examining associations over longer time periods than a few days. This hinders the ability to examine the issues of harvesting or associations with longer-term exposure.[41,42]

In conclusion, we found that ambient air pollution was associated with mortality from all-cause and cardiovascular disease in Seoul, Korea and identified potential susceptibilities based on individual characteristics such as sex, age, and indicators of SES. Our findings have implications for public policy making and risk assessment to the susceptible subpopulations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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REFERENCES

1. Touloumi G, Samoli E, Quenel P, Paldy A, Anderson RH, Zmirou D, et al. Short-term effects of air pollution on total and cardiovascular mortality: the confounding effect of influenza epidemics. Epidemiology. 2005; 16:49–57. [PubMed: 15613945]
2. Chen R, Pan G, Kan H, Tan J, Song W, Wu Z, et al. Ambient air pollution and daily mortality in Anshan, China: a time-stratified case-crossover analysis. Sci Total Environ. 2010; 408(24):6086–6091. [PubMed: 20889186]
3. Zeka A, Zanobetti A, Schwartz J. Individual-level modifiers of the effects of particulate matter on daily mortality. Am J Epidemiol. 2006; 163(9):849–859. [PubMed: 16554348]
4. Ren C, Melly S, Schwartz J. Modifiers of short-term effects of ozone on mortality in eastern Massachusetts–a case-crossover analysis at individual level. Environ Health. 2010; 9:3–12. [PubMed: 20092648]
5. Bell ML, Dominici F. Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 US communities. Am J Epidemiol. 2008; 167:986–997. [PubMed: 18303005]
6. Health Effects Institute. Health Effects of Outdoor Air Pollution in Developing Countries of Asia: A Literature Review. Boston, MA, USA: Health Effects Institute; 2004.
7. Yi O, Hong YC, Kim H. Seasonal effect of PM_{10} concentrations on mortality and morbidity in Seoul, Korea: a temperature-matched case-crossover analysis. Environ Res. 2010; 110(1):89–95. [PubMed: 19819431]
8. Ha EH, Lee JT, Kim H, Hong YC, Lee BE, Park HS, et al. Infant susceptibility of mortality to air pollution in Seoul, South Korea. Pediatrics. 2003; 111(2):284–290. [PubMed: 12563052]
9. Hong YC, Lee JT, Kim H, Ha EH, Schwartz J, Christiani DC. Effects of air pollutants on acute stroke mortality. Environ Health Perspect. 2002; 110(2):187–191. [PubMed: 11836148]
10. Yoon HS. The role of public and private income transfers to the income status of women who experienced marital disruption. Korean J Soc Welfare. 2004; 56(2):5–27.
11. O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia J, et al. Health, wealth, and air pollution: advancing theory and methods. Environ Health Perspect. 2003; 111:1861–1870. [PubMed: 14644658]
12. Forastiere F, Stafoggia M, Tasco C, Picciotto S, Agabiti N, Cesaroni G, Perucci CA. Socioeconomic status, particulate air pollution, and daily mortality: differential exposure or differential susceptibility. Am J Ind Med. 2007; 50:208–216. [PubMed: 16847936]
13. Lee JT, Schwartz J. Reanalysis of the effects of air pollution on daily mortality in Seoul, Korea: a case-crossover design. Environ Health Perspect. 1999; 107:633–636. [PubMed: 10417360]
14. Katsouyanni K, Touloumi G, Samoli E, Gryparis A, Le Tertre A, Monopolis Y, et al. Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 project. Epidemiology. 2001; 12:521–531. [PubMed: 11505171]

15. Zanobetti A, Schwartz J. Race, gender, and social status as modifiers of the effects of PM_{10} on mortality. J Occup Environ Med. 2000; 42(5):469–474. [PubMed: 10824299]
16. Filleul L, Rondeau V, Cantagrel A, Dartigues JF, Tessier JF. Do subject characteristics modify the effects of particulate air pollution on daily mortality among the elderly? J Occup Environ Med. 2004; 46:1115–1122. [PubMed: 15534498]
17. Forastiere F, Stafoggia M, Berti G, Bisanti L, Cernigliaro A, Chiusolo M, et al. Particulate matter and daily mortality: a case-crossover analysis of individual effect modifiers. Epidemiology. 2008; 19:571–580. [PubMed: 18467959]
18. Lee JT, Shin DC, Chung Y. Air pollution and daily mortality in Seoul and Ulsan, Korea. Environ Health Persp. 1999; 107(2):149–154.
19. Mann JK, Tager IB, Lurmann F, Segal M, Quesenberry CP Jr, Lugg MM, et al. Air pollution and hospital admissions for ischemic heart disease in persons with congestive heart failure or arrhythmia. Environ Health Perspect. 2002; 110:1247–1252. [PubMed: 12460805]
20. Maclure M. The case-crossover design: a method for studying transient effects on the risk of acute events. Am J Epidemiol. 1991; 133:144–153. [PubMed: 1985444]
21. Levy D, Lumley T, Sheppard L, Kaufman J, Checkoway H. Referent selection in case-crossover analyses of acute health effects of air pollution. Epidemiology. 2001; 12(2):186–192. [PubMed: 11246579]
22. Bateson TF, Schwartz J. Who is sensitive to the effects of particulate air pollution on mortality? A case-crossover analysis of effect modifiers. Epidemiology. 2004; 15:143–149. [PubMed: 15127905]
23. Guo Y, Barnett AG, Zhang Y, Tong S, Yu W, Pan X. The short-term effect of air pollution on cardiovascular mortality in Tianjin, China: comparison of time series and case-crossover analyses. Sci Total Environ. 2010; 409(2):300–306. [PubMed: 21055792]
24. Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Ozone and short-term mortality in 95 US urban communities, 1987 – 2000. JAMA. 2004; 292(19):2372–2378. [PubMed: 15547165]
25. Kan H, London SJ, Chen G, Zhang Y, Song G, Zhao N, et al. Season, sex, age, and education as modifiers of the effects of outdoor air pollution on daily mortality in Shanghai, China: the public health and air pollution in Asia (PAPA) study. Environ Health Perspect. 2008; 116:1183–1188. [PubMed: 18795161]
26. Kunzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, et al. Ambient air pollution and atherosclerosis in Los Angeles. Environ Health Perspect. 2005; 113:201–206. [PubMed: 15687058]
27. Yunginger JW, Reed CE, O'Connell EJ, Melton LJ III, O'Fallon WM, Silverstein MD. A community-based study of the epidemiology of asthma. Incidence rates, 1964 – 1983. Am Rev Respir Dis. 1992; 146(4):888–894. [PubMed: 1416415]
28. Kohlhäufl M, Brand P, Scheuch G, Meyer TS, Schulz H, Häussinger K, et al. Increased fine particle deposition in women with asymptomatic nonspecific airway hyperresponsiveness. Am J Respir Crit Care Med. 1999; 159(3):902–906. [PubMed: 10051270]
29. Clougherty JE. A growing role for gender analysis in air pollution epidemiology. Environ Health Perspect. 2010; 118(2):167–176. [PubMed: 20123621]
30. Samoli E, Peng R, Ramsay T, Pipikou M, Touloumi G, Dominici F, et al. Acute effects of ambient particulate matter on mortality in Europe and North America: results from the APHENA study. Environ Health Perspect. 2008; 116:1480–1486. [PubMed: 19057700]
31. Levkoff SE, Cleary PD, Wetle T. Differences in the appraisal of health between aged and middle-aged adults. J Gerontol. 1987; 42(1):114–120. [PubMed: 3794190]
32. Ware JE, Davies AR. Choosing measures of health status for individuals in general populations. Am J Public Health. 1981; 71:620–625. [PubMed: 7235100]
33. Serinelli M, Vigotti MA, Stafoggia M, Berti G, Bisanti L, Mallone S, et al. Particulate matter and out-of-hospital coronary deaths in eight Italian cities. Occup Environ Med. 2010; 67(5):301–306. [PubMed: 19819851]
34. Pietilä AM, Rantakallio P, Läärä E. Background factors predicting non-response in a health survey of Northern Finnish young men. Scand J Soc Med. 1995; 23(2):129–136. [PubMed: 7676219]

35. O'Neill MS, Bell ML, Ranjit N, Cifuentes LA, Loomis D, Gouveia N, et al. Air pollution and mortality in Latin America: the role of education. Epidemiology. 2008; 19(6):810–819. [PubMed: 18633327]

36. Finkelstein MM, Jerrett M, DeLuca P, Finkelstein N, Verma DK, Chapman K, et al. Relation between income, air pollution and mortality: a cohort study. CMAJ. 2003; 169:397–402. [PubMed: 12952800]

37. Ou CQ, Hedley AJ, Chung RY, Thach TQ, Chau YK, Chan KP, et al. Socioeconomic disparities in air pollution-associated mortality. Environ Res. 2008; 107(2):237–244. [PubMed: 18396271]

38. Krewski D, Burnett RT, Goldberg M, Hoover K, Siemiatycki J, Abrahamowicz M, et al. Reanalysis of the Harvard Six Cities Study, part II: sensitivity analysis. Inhal Toxicol. 2005; 17(7–8):343–353. [PubMed: 16020033]

39. Jerrett M, Burnett RT, Brook J, Kanaroglou P, Giovis C, Finkelstein N, et al. Do socioeconomic characteristics modify the short term association between air pollution and mortality? Evidence from a zonal time series in Hamilton, Canada. J Epidemiol Community Health. 2004; 58:31–40. [PubMed: 14684724]

40. Yi O, Kim H, Ha E. Does area level socioeconomic status modify the effects of PM(10) on preterm delivery? Environ Res. 2010; 110(1):55–61. [PubMed: 19878932]

41. Bateson TF, Schwartz J. Control for seasonal variation and time trend in case-crossover studies of acute effects of environmental exposures. Epidemiology. 1999; 10:539–544. [PubMed: 10468428]

42. Maclure M, Mittleman MA. Should we use a case-crossover design? Annu Rev Public Health. 2000; 21:193–221. [PubMed: 10884952]

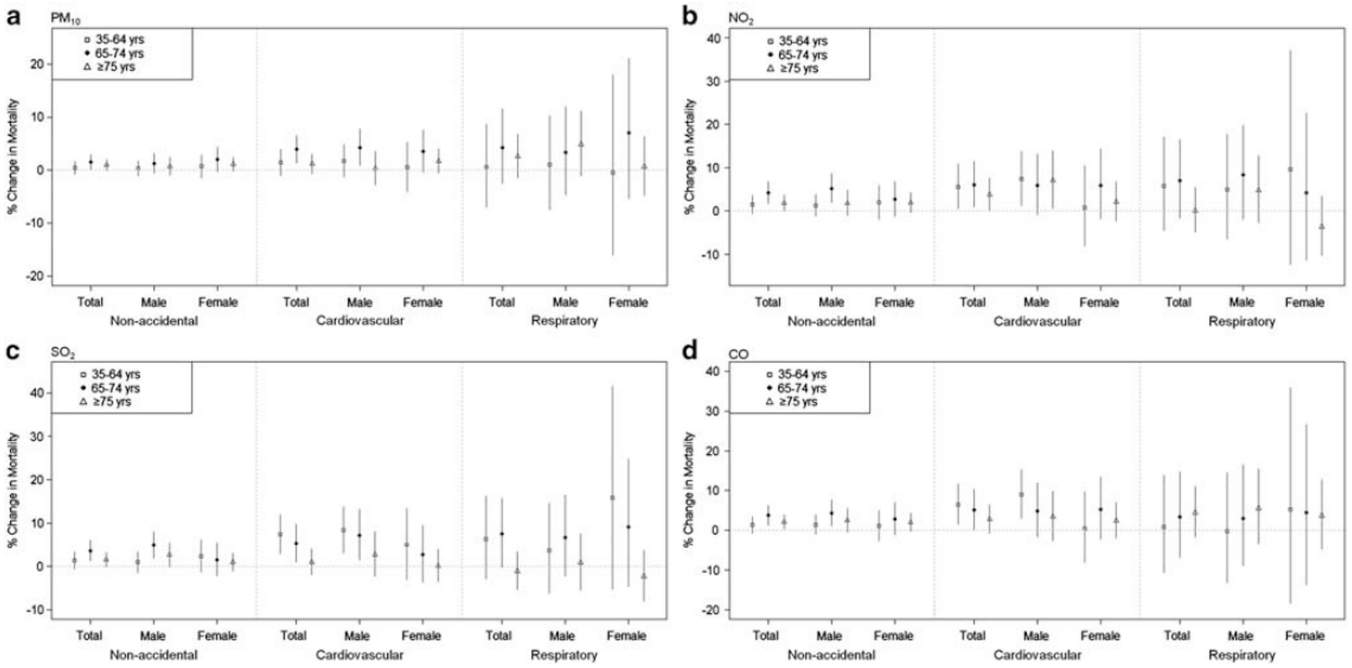

Figure 1.

Percent change in cause-specific mortality per interquartile range increase in air pollutant concentrations by sex and age (**a**) PM_{10}, (**b**) NO_2, (**c**) SO_2, and (**d**) CO. The point represents the central estimate; the vertical line represents the 95% confidence interval. See Supplementary Figure 1 for results for ozone.

Table 1

Summary statistics of the study population, daily air pollutant concentrations, and weather variables for the study period 2000 – 2007.

	N (%)
All observations	261,952
Cause of death	
Respiratory	15,523 (5.9)
Cardiovascular	73,356 (28.0)
Other non-accidental	173,073 (66.1)
Sex	
Male	140,094 (53.5)
Female	121,723 (46.5)
Age (years)	
35 – 64	85,215 (32.5)
65 – 74	59,967 (22.9)
≥ 75	116,770 (44.6)
Education	
None	65,032 (24.8)
≤ 12 years	164,171 (62.7)
> 12 years	30,667 (11.7)
Unknown	2,082 (0.8)
Marital status	
Never married	9340 (3.6)
Married	132,509 (50.6)
Divorced	12,583 (4.8)
Widowed	103,600 (39.6)
Unknown	3920 (1.5)
Occupation	
Professional	20,221 (12.7)
Manual	8376 (5.2)
Housewife/unemployed	127,043 (79.6)
Unknown	4056 (2.5)
Air pollutant concentration (mean ± SD)	
PM_{10} (μg/m^3)	66.08 ± 46.26
NO_2 (p.p.b.)	36.91 ± 12.36
SO_2 (p.p.b.)	5.41 ± 2.37
CO (p.p.m.)	0.86 ± 0.40
O_3 (p.p.b.)	27.73 ± 15.47
Meteorological measures (mean ± SD)	
Temperature (°C)	12.87 ± 10.10
Humidity (%)	61.98 ± 14.48
Barometric pressure (hPa)	1016.05 ± 8.16

Table 2

Percent change (95% confidence intervals) of mortality outcomes associated with an IQR increase in air pollutant concentrations.

	Percentage change in risk of mortality				
	PM_{10}	**NO_2**	**SO_2**	**CO**	**O_3**
Total	**0.94 (0.25, 1.62)**	**2.27 (1.03, 3.53)**	**1.94 (0.80, 3.09)**	**2.21 (1.00, 3.43)**	0.51 (−0.44, 1.46)
Cardiovascular	**1.95 (0.64, 3.27)**	**4.82 (2.18, 7.54)**	**3.64 (1.46, 5.87)**	**4.32 (1.77, 6.92)**	1.26 (−1.32, 3.92)
Respiratory	2.61 (−0.59, 5.91)	2.44 (−1.69, 6.74)	1.80 (−1.68, 5.41)	3.64 (−1.32, 8.85)	2.04 (−1.91, 6.15)

Abbreviations: CO, carbon monoxide; IQR, interquartile range; NO_2, nitrogen dioxide; O_3, ozone; SO_2, sulfur dioxide.

Total PM_{10} (lag0) NO_2, SO_2, CO (lag01) O_3 (lag2); cardiovascular PM_{10} (lag0) NO_2, CO (lag02), SO_2, O_3 (lag01); respiratory PM_{10}, CO (lag01) NO_2 (lag1) SO_2, O_3 (lag2). See Supplementary Table 2 for results for all lag structures.

The model included following variables; relative humidity, barometric pressure, and day of the week.

Bold values represent statistically significant results.

Table 3

Percent change (95% CIs) in mortality risk associated with an IQR increase in air pollutant concentrations, by sex, age, education, marital status, and occupations.

	Percentage change in risk of mortality			
	PM_{10}	**NO_2**	**SO_2**	**CO**
Total mortality				
Sex				
Male	0.7 (−0.2, 1.6)	**2.5 (0.8, 4.2)**	**2.5 (1.0, 4.1)**	**2.5 (0.9, 4.2)**
Female	**1.2 (0.2, 2.2)**	**2.1 (0.3, 3.9)**	1.3 (−0.3, 3.0)	**1.9 (0.2, 3.6)**
Age (years)				
35 – 64	0.4 (−0.7, 1.6)	1.5 (−0.6, 3.6)	1.3 (−0.6, 3.3)	1.3 (−0.7, 3.4)
65 – 74	**1.6 (0.1, 3.0)**	**4.3 (1.7, 6.8)**	**3.6 (1.2, 6.0)**	**3.7 (1.2, 6.3)**
≥ 75 years	**1.0 (0.0, 2.0)**	**1.9 (0.1, 3.7)**	1.5 (−0.1, 3.2)	**2.1 (0.3, 3.9)**
Education				
None	0.9 (−0.4, 2.3)	1.2 (−1.2, 3.6)	1.1 (−1.1, 3.4)	1.5 (−0.9, 3.8)
≤ 12 years	**0.9 (0.0, 1.8)**	**3.0 (1.5, 4.6)**	**2.2 (0.7, 3.6)**	**2.7 (1.2, 4.3)**
> 12 years	0.9 (−1.1, 3.0)	0.3 (−3.0, 3.8)	2.3 (−0.9, 5.6)	0.8 (−2.6, 4.3)
Unknown	6.4 (−1.5, 14.9)	4.2 (−8.7, 18.9)	3.2 (−8.0, 15.8)	8.7 (−5.2, 24.7)
Marital status				
Never married	0.3 (−3.3, 4.0)	2.2 (−3.8, 8.7)	3.8 (−2.1, 10.0)	4.5 (−1.8, 11.2)
Married	0.6 (−0.3, 1.6)	**3.3 (1.6, 5.1)**	**2.5 (0.9, 4.1)**	**2.5 (0.8, 4.2)**
Divorced	1.1 (−2.0, 4.2)	−3.1 (−8.2, 2.2)	−0.4 (−5.3, 4.7)	−1.1 (−6.3, 4.3)
Widowed	**1.4 (0.3, 2.5)**	1.8 (−0.1, 3.8)	1.5 (−0.3, 3.3)	**2.3 (0.4, 4.2)**
Unknown	−1.3 (−6.8, 4.5)	−4.3 (−13.1, 5.3)	−1.2 (−9.1, 7.5)	−4.8 (−13.7, 5.1)
Occupation				
Professional	0.1 (−2.3, 2.6)	1.0 (−3.3, 5.4)	−2.0 (−5.7, 1.9)[a]	0.3 (−3.8, 4.5)[b]
Manual	0.2 (−3.5, 4.0)	**7.1 (0.2, 14.5)**	**8.4 (1.9, 15.3)**[a]	**9.8 (3.0, 17.0)**[b]
Housewife/unemployed	**1.3 (0.4, 2.2)**	**2.8 (1.0, 4.6)**	**2.3 (0.8, 3.9)**[a]	**2.8 (1.1, 4.5)**
Unknown	2.7 (−2.8, 8.6)	−1.5 (−10.4, 8.3)	−3.2 (−10.7, 4.9)	−0.7 (−9.8, 9.3)
Cardiovascular mortality				
Sex				
Male	**2.1 (0.3, 4.0)**	**7.1 (3.3, 11.0)**	**6.1 (3.0, 9.3)**[c]	**6.1 (2.5, 9.8)**
Female	**1.8 (0.0, 3.7)**	2.5 (−1.1, 6.2)	1.2 (−1.8, 4.3)[c]	2.5 (−1.0, 6.1)
Age (years)				
35 – 64	1.4 (−1.1, 4.0)	**5.5 (0.5, 10.8)**	**7.4 (3.0, 11.9)**[d]	**6.4 (1.5, 11.6)**
65 – 74	**3.9 (1.4, 6.6)**	**6.0 (0.8, 11.4)**	**5.3 (1.0, 9.7)**	**5.1 (0.1, 10.4)**
≥ 75	1.2 (−0.7, 3.1)	**3.8 (0.1, 7.7)**	1.0 (−2.0, 4.1)[d]	2.8 (−0.8, 6.5)
Education				
None	1.7 (−0.8, 4.3)	**5.8 (0.8, 11.1)**	2.1 (−2.0, 6.3)	4.7 (−0.1, 9.7)
≤ 12 years	**2.1 (0.4, 3.8)**	**4.6 (1.3, 8.0)**	**4.7 (1.9, 7.5)**	**5.0 (1.8, 8.4)**
> 12 years	0.6 (−3.3, 4.6)	4.1 (−3.4, 12.2)	1.3 (−4.8, 7.8)	−0.3 (−7.4, 7.3)

	Percentage change in risk of mortality			
	PM_{10}	**NO_2**	**SO_2**	**CO**
Unknown	**15.2 (0.2, 32.5)**	−0.3 (−25.1, 32.6)	9.4 (−11.4, 35.2)	1.7 (−23.6, 35.6)
Marital status				
Never married	−1.1 (−8.6, 7.0)	4.7 (−9.8, 21.6)	2.3 (−9.8, 16.1)	3.9 (−10.4, 20.5)
Married	**1.9 (0.0, 3.8)**	**6.2 (2.4, 10.1)**	**5.9 (2.8, 9.2)**	**5.4 (1.7, 9.1)**
Divorced	2.1 (−4.0, 8.6)	**14.3 (1.1, 29.3)**	**16.8 (5.4, 29.4)**	**18.0 (4.3, 33.4)**
Widowed	**2.3 (0.3, 4.3)**	2.7 (−1.2, 6.7)	0.2 (−3.0, 3.4)	2.5 (−1.2, 6.4)
Unknown	−1.4 (−11.3, 9.6)	−1.7 (−19.6, 20.3)	1.9 (−12.7, 18.8)	−9.2 (−25.8, 11.1)
Occupation				
Professional	2.4 (−2.5, 7.5)	6.5 (−3.4, 17.3)	1.0 (−6.6, 9.2)[e]	6.6 (−2.8, 16.9)
Manual	1.9 (−5.6, 9.9)	**26.1 (8.7, 46.3)**	**19.7 (6.2, 35.0)**[e]	**24.6 (8.3, 43.4)**
Housewife/Unemployed	**2.2 (0.4, 4.0)**	**5.3 (1.6, 9.2)**	**3.0 (0.1, 6.1)**	3.2 (−0.3, 6.8)
Unknown	7.2 (−3.7, 19.4)	9.5 (−10.6, 34.2)	1.7 (−12.8, 18.7)	9.9 (−9.9, 34.1)
Respiratory mortality				
Sex				
Male	3.2 (−1.0, 7.6)	**5.6 (0.1, 11.3)**	3.1 (−1.6, 7.9)	3.6 (−2.9, 10.5)
Female	2.0 (−2.8, 7.0)	−1.5 (−7.4, 4.8)	0.3 (−4.9, 5.7)	3.7 (−3.7, 11.6)
Age (years)				
35 – 64	0.5 (−7.0, 8.7)	5.7 (−4.5, 17.1)	6.2 (−2.9, 16.2)	0.8 (−10.7, 13.8)
65 – 74	4.3 (−2.5, 11.6)	7.0 (−1.7, 16.5)	7.5 (−0.2, 15.7)	3.4 (−6.8, 14.7)
≥ 75	2.6 (−1.4, 6.8)	0.1 (−4.8, 5.4)	−1.1 (−5.3, 3.3)	4.4 (−1.8, 11.0)
Education				
None	0.9 (−4.6, 6.7)	−2.1 (−8.8, 5.1)	−1.7 (−7.6, 4.6)	2.6 (−5.8, 11.6)
≤ 12 years	3.5 (−0.6, 7.9)	4.9 (−0.5, 10.6)	4.2 (−0.4, 9.1)	4.2 (−2.2, 11.1)
> 12 years	4.1 (−6.6, 15.9)	2.9 (−9.6, 17.2)	−0.5 (−11.2, 11.4)	1.0 (−14.0, 18.6)
Unknown	−6.8 (−31.9, 27.4)	2.2 (−33.1, 56.1)	−1.9 (−30.2, 37.8)	39.9 (−16.3, 133.8)
Marital status				
Never married	9.8 (−9.0, 32.4)	17.7 (−6.7, 48.5)	18.1 (−4.2, 45.7)[f]	12.8 (−16.4, 52.1)
Married	1.7 (−3.0, 6.7)	1.7 (−4.3, 8.0)	**5.5 (0.1, 11.2)**	0.9 (−6.2, 8.6)
Divorced	3.7 (−11.9, 22.2)	−0.1 (−19.0, 23.1)	−11.7 (−26.4, 6.0)[f]	−9.4 (−28.8, 15.3)
Widowed	3.7 (−0.9, 8.5)	2.6 (−3.2, 8.8)	−0.5 (−5.4, 4.6)	**7.5 (0.3, 15.3)**
Unknown	−15.9 (−34.4, 7.9)	−1.9 (−26.6, 31.1)	−16.4 (−36.8, 10.4)	−10.4 (−37.3, 27.9)
Occupation				
Professional	5.2 (−9.1, 21.7)	2.2 (−14.9, 22.8)	**22.2 (5.2, 41.8)**[g]	2.9 (−16.9, 27.5)
Manual	−17.8 (−32.6, 0.1)	−1.5 (−24.0, 27.7)	**50.2 (17.7, 91.5)**	−0.8 (−27.7, 35.9)
Housewife/Unemployed	3.2 (−0.9, 7.6)	1.3 (−4.2, 7.2)	−1.9 (−6.2, 2.7)[g]	5.3 (−1.3, 12.4)
Unknown	6.0 (−16.6, 34.8)	−3.3 (−28.7, 31.3)	3.5 (−21.3, 36.2)	−7.5 (−37.0, 35.7)

Abbreviations: CIs, confidence intervals; CO, carbon monoxide; IQR, interquartile range; NO_2, nitrogen dioxide; OR, odds ratio; SO_2, sulfur dioxide.

See Supplementary Table 3 for results for O_3.

Below are the comparisons of OR that were statistically significant:

[a] SO_2 total mortality OR were 1.11 (1.03, 1.19), 1.04 (1.00, 1.09) times higher for manual and housewife/unemployed occupation than professional occupation, respectively.

[b] CO total mortality OR was 1.09 (1.01, 1.18) times higher for manual occupation than professional occupation.

[c] SO_2 cardiovascular mortality OR was 1.05 (1.01, 1.09) times higher for male than female.

[d] SO_2 cardiovascular mortality OR was 1.06 (1.01, 1.12) times higher for 35 – 64 years than ≥ 75 years.

[e] SO_2 cardiovascular mortality OR was 1.19 (1.03, 1.37) times higher for manual occupation than professional occupation.

[f] SO_2 respiratory mortality OR was 1.34 (1.01, 1.77) times higher for never married than divorced.

[g] SO_2 respiratory mortality OR was 1.24 (1.07, 1.45) times higher for professional occupation than housewife/unemployed.

Bold values represent statistically significant results.

Table 4

Percent change in mortality risk associated with an IQR increase in air pollutant concentrations by educational categories, for each age group.

Education	Effect (95% CI)								
	Total			Cardiovascular			Respiratory		
	35 – 64 years	65 – 74	≥ 75	35 – 64	65 – 74	≥ 75	35 – 64	65 – 74	≥ 75
PM_{10}									
None	1.5 (−4.0, 7.4)	2.5 (−1.1, 6.1)	0.5 (−1.0, 2.1)	−2.7 (−13.7, 9.8)	5.7[a] (−0.3, 12.1)	1.1 (−1.8, 3.9)	−13.4 (−37.4, 19.6)	4.1 (−12.1, 23.2)	0.8 (−5.1, 7.1)
≤ 12 years	1.0 (−0.4, 2.3)	0.9 (−0.8, 2.6)	0.8 (−0.6, 2.3)	**3.0 (0.1, 5.9)**	2.9 (−0.2, 6.1)	0.9 (−1.7, 3.5)	3.0 (−5.7, 12.4)	5.6 (−2.4, 14.3)	2.8 (−2.9, 8.9)
> 12 years	−2.0 (−4.8, 0.9)	3.0 (−0.7, 6.9)	**4.5 (0.4, 8.9)**	−3.2 (−9.1, 3.0)	4.0 (−3.1, 11.6)	2.8 (−4.6, 10.7)	−3.3 (−21.8, 19.6)	−2.3 (−21.6, 21.7)	11.6 (−4.3, 30.1)
Unknown	5.5 (−9.4, 22.9)	9.0 (−8.0, 29.2)	5.7 (−4.7, 17.3)	4.6 (−22.7, 41.5)	**48.2[a] (8.1, 103.2)**	9.6 (−8.7, 31.5)	−25.3 (−75.8, 130.7)	7.9 (−57.4, 172.9)	−6.1 (−33.8, 33.2)
NO_2									
None	8.3[b] (−1.9, 19.5)	4.6 (−1.7, 11.2)	0.1 (−2.5, 2.9)	10.6 (−11.7, 38.5)	6.1 (−5.7, 19.3)	5.1 (−0.6, 11.1)	35.1 (−6.2, 94.4)	5.1 (−15.3, 30.3)	−4.9 (−12.0, 2.8)
≤ 12 years	2.3 (−0.2, 4.7)	**4.6 (1.5, 7.8)**	**2.8 (0.2, 5.5)**	**7.8 (1.9, 14.0)**	**6.9 (0.6, 13.7)**	0.5 (−4.5, 5.8)	9.1 (−3.1, 22.9)	8.5 (−2.0, 20.0)	2.1 (−5.1, 9.8)
> 12 years	−4.0[b] (−8.6, 0.9)	3.1 (−3.4, 10.1)	5.8 (−1.1, 13.2)	0.6 (−10.6, 13.2)	0.1 (−13.0, 15.0)	14.3 (−0.3, 31.1)	−3.8 (−27.5, 27.6)	−2.5 (−24.8, 26.4)	8.9 (−8.9, 30.1)
Unknown	−3.3 (−24.8, 24.4)	15.8 (−12.8, 53.7)	3.8 (−13.8, 25.0)	0.3 (−46.8, 88.9)	−7.9 (−49.8, 68.7)	2.3 (−29.8, 49.1)	−15.7 (−83.3, 326.7)	65.5 (−39.0, 349.2)	−8.2 (−44.2, 51.2)
SO_2									
None	8.6 (−1.6, 19.8)	1.7 (−4.1, 7.9)	0.6 (−1.9, 3.2)	8.8 (−11.1, 33.3)	1.3 (−8.3, 11.8)	1.7 (−2.9, 6.5)	10.5 (−22.6, 57.6)	15.7 (−3.8, 39.1)	−4.1 (−10.4, 2.5)
≤ 12 years	1.6 (−0.6, 3.9)	**3.5 (0.7, 6.5)**	1.8 (−0.6, 4.2)	**10.2 (5.1, 15.7)**	**5.9 (0.6, 11.4)**	−0.2 (−4.3, 4.1)	8.4 (−2.1, 20.0)	6.4 (−2.4, 16.0)	1.4 (−4.8, 8.0)
> 12 years	−2.3 (−6.7, 2.4)	**6.4 (0.2, 13.0)**	**6.4 (0.0, 13.3)**	−1.7 (−10.9, 8.5)	4.6 (−6.5, 17.1)	2.6 (−8.4, 14.9)	−7.7 (−27.8, 18.0)	−6.3 (−26.3, 19.1)	5.0 (−9.8, 22.3)
Unknown	6.6 (−14.4, 32.7)	8.1 (−15.3, 37.9)	−0.5 (−15.4, 17.0)	38.9 (−11.0, 116.7)	−0.2 (−39.4, 64.3)	2.5 (−22.3, 35.1)	63.2 (−52.6, 461.6)	**118.7 (3.6, 361.9)**	−28.9 (−53.9, 9.7)
CO									
None	2.8 (−6.8, 13.3)	4.7 (−1.5, 11.3)	0.8 (−1.8, 3.5)	3.8 (−16.3, 28.6)	2.1 (−9.1, 14.6)	5.1 (−0.4, 10.8)	31.7 (−14.6, 103.1)	8.6 (−16.1, 40.7)	0.5 (−8.4, 10.2)
≤ 12 years	2.2 (−0.1, 4.7)	**3.4 (0.3, 6.5)**	**2.8 (0.2, 5.4)**	**8.6 (2.9, 14.7)**	**7.7 (1.5, 14.3)**	0.3 (−4.7, 5.5)	3.6 (−9.9, 19.2)	2.6 (−9.3, 16.0)	5.8 (−3.2, 15.5)
> 12 years	−3.9 (−8.6, 1.0)	4.3 (−2.3, 11.5)	6.5 (−0.6, 14.1)	0.7 (−10.3, 12.9)	−5.0 (−17.1, 8.8)	3.8 (−9.5, 19.0)	−22.9 (−45.1, 8.2)	−0.6 (−28.8, 38.8)	14.5 (−8.1, 42.7)
Unknown	12.3 (−13.7, 46.0)	21.9 (−7.8, 61.3)	0.9 (−17.1, 22.9)	24.4 (−35.6, 140.3)	−1.0 (−42.6, 70.6)	−4.1 (−35.3, 42.3)	233.8 (−67.3, 3302.3)	60.0 (−53.1, 445.6)	28.8 (−28.9, 133.2)

Abbreviations: CIs, confidence intervals: CO, carbon monoxide; IQR, interquartile range; NO_2, nitrogen dioxide; O_3, ozone; OR, odds ratio; SO_2, sulfur dioxide.

Below are the comparisons of OR that were statistically significant:

[a] PM_{10} cardiovascular mortality OR was 1.40 (1.02, 1.93) times higher for unknown education than no education for those 65 – 74 years.

[b] NO_2 total mortality OR was 1.13 (1.01, 1.26) times higher for no education than > 12 years education for those 35 – 64 years.

See Supplementary Table 5 for results for O_3.

Bold values represent statistically significant results.