RESEARCH REPORT

HEALTH EFFECTS INSTITUTE

Number 154
November 2010

Public Health and Air Pollution in Asia (PAPA): Coordinated Studies of Short-Term Exposure to Air Pollution and Daily Mortality in Four Cities

HEI Public Health and Air Pollution in Asia Program

Part 3

Includes Commentaries by the Institute's Health Review Committee

Part 3

Estimating the Effects of Air Pollution on Mortality in Bangkok, Thailand

Nuntavarn Vichit-Vadakan, Nitaya Vajanapoom, and Bart Ostro

with a Commentary by the HEI Health Review Committee

Publishing history: The Web version of this document was posted at *www.healtheffects.org* in November 2010.

Citation for whole document:

Vichit-Vadakan N, Vajanapoom N, Ostro B. 2010. Part 3. Estimating the effects of air pollution on mortality in Bangkok, Thailand. In: Public Health and Air Pollution in Asia (PAPA): Coordinated Studies of Short-Term Exposure to Air Pollution and Daily Mortality in Four Cities. HEI Research Report 154. Health Effects Institute, Boston, MA.

When specifying a section of this report, cite it as a chapter of the whole document.

 Asterisk Typographics, Barre, Vt., Compositor. Printed by Recycled Paper Printing, Boston, Mass. Library of Congress Catalog Number for the HEI Report Series: WA 754 R432.

Cover paper: made with at least 55% recycled content, of which at least 30% is post-consumer waste; free of acid and elemental chlorine. Text paper: made with 100% post-consumer waste recycled content; acid free; no chlorine used in processing. The book is printed with soy-based inks and is of permanent archival quality.

CONTENTS

Research Report 154, Part 3

INVESTIGATORS' REPORT

Part 3. Estimating the Effects of Air Pollution on Mortality in Bangkok, Thailand

Nuntavarn Vichit-Vadakan, Nitaya Vajanapoom, and Bart Ostro

Faculty of Public Health, Thammasat University, Klongluang, Pathumthani, Thailand (N. V-V., N. V.); Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Oakland, California (B.O.)

ABSTRACT

While the effects of particulate matter (PM*) on mortality have been well documented in North America and Western Europe, considerably less is known about its effects in developing countries in Asia. Existing air pollution data in Bangkok, Thailand, indicate that airborne concentrations of PM ≤ 10 μm in aerodynamic diameter (PM_{10}) are as high or higher than those experienced in most cities in North America and Western Europe. At the same time, the demographics, activity patterns, and background health status of the population, as well as the chemical composition of PM, are different in Bangkok. It is important, therefore, to determine whether the effects of PM_{10} on mortality occurring in this large metropolitan area are similar to those in Western cities.

The quality and completeness of Bangkok mortality data have been recently enhanced by the completion of a few mortality studies and through input from monitors currently measuring daily PM_{10} in Bangkok. In this analysis, we examined the effects of PM_{10} and several gaseous pollutants on daily mortality for the years 1999 through 2003. Our results suggest strong associations between several different mortality outcomes and levels of PM_{10} and several of the gaseous pollutants, including nitrogen dioxide (NO_2), nitric oxide (NO), and ozone (O_3). In many cases, the effect estimates were higher than the approximately 6% per 10 μg/m^3 typically reported in Western industrialized nations — based on reviews by the U.S. Environmental Protection Agency (U.S. EPA) and the World Health Organization (WHO) (Anderson et al. 2004). For example, the excess risk (ER) for mortality due to all natural causes was 1.3% (95% confidence interval [CI], 0.8 to 1.7), with higher ERs for cardiovascular and respiratory mortality of 1.9% (95% CI, 0.8 to 3.0) and 1.0% (95% CI, −0.4 to 2.4), respectively. Of particular note, for this warm, tropical city of approximately 6 to 10 million people, is that there is no covariation between pollution and cold weather, with its associated adverse health problems. Multiday averages of PM_{10} generated even higher effect estimates. Our analysis of age- and disease-specific mortality indicated elevated ERs for young children, especially infants with respiratory illnesses, children less than 5 years of age with lower respiratory infections (LRIs), and people with asthma. Age-restricted analyses showed that the associations between mortality due to all natural causes and PM_{10} concentration increased with age, with the strongest effects among people aged 75 years and older. However, associations between increases in PM_{10} concentration and mortality were observed for all of the other age groups. With a few exceptions, relatively similar results were observed for several of the other pollutants — sulfur dioxide (SO_2), NO_2, O_3, and NO, which were highly correlated with PM_{10}. However, many of the effects from gaseous pollutants were attenuated in multipollutant models, while effects from PM_{10} appeared to be most consistent. In addition, there was some evidence of an independent effect of O_3 for certain health outcomes.

We conducted substantial sensitivity analyses to examine whether our results were robust. The results indicated that our core model was generally robust to the choice of model specification, spline model, degrees of freedom (df) of time-smoothing functions, lags for temperature, adjustment for autocorrelation, adjustment for epidemics, and adjustment for missing values using centered data (see the description of the centering method used in the Common Protocol found at the end of this volume). Finally, the

This Investigators' Report is one part of Health Effects Institute Research Report 154, which also includes a Commentary by the Health Review Committee. Correspondence concerning the Investigators' Report may be addressed to Dr. Nuntavarn Vichit-Vadakan, Faculty of Public Health, Thammasat University, Rangsit Campus, Klongluang, Pathumthani 12121, Thailand.

The PAPA Program was initiated by the Health Effects Institute in part to support the Clean Air Initiative for Asian Cities (CAI-Asia), a partnership of the Asian Development Bank and the World Bank to inform regional decisions about improving air quality in Asia. Additional funding was obtained from the U.S. Agency for International Development and the William and Flora Hewlett Foundation. The contents of this document have not been reviewed by private party institutions, including those that support the Health Effects Institute; therefore, it may not reflect the views or policies of these parties, and no endorsement by them should be inferred.

*A list of abbreviations and other terms appears at the end of the Investigators' Report.

concentration–response functions for most of the pollutants appear to be linear. Thus, our sensitivity analyses results suggest an impact of pollution on mortality in Bangkok that is fairly consistent. They also provide support for the extrapolation of results from health effects studies conducted in North America and Western Europe to other parts of the world, including developing countries in Asia.

INTRODUCTION

Compelling epidemiologic evidence indicates that exposure to current levels of ambient, airborne PM in North American and Western European cities is associated with premature mortality and a wide range of morbidity outcomes (WHO 2000; U.S. EPA 2004). Existing air pollution monitoring information and recent exposure assessments suggest that airborne PM concentrations in Bangkok and other major Asian cities are as high as or higher than those in North American and Western European cities. A recent review of Asian cities, mostly undertaken in developing countries, suggests that PM may also be associated with both mortality and morbidity (HEI International Scientific Oversight Committee 2004). However, PM chemical composition and relevant population characteristics, such as activity patterns, background health status, and other factors related to lower socioeconomic status, all may contribute to differential risks in developing countries such as Thailand, China, and India. More specifically, it has not been clearly documented whether or to what extent PM-associated human health effects are occurring in Bangkok, the capital and largest city in Thailand. In addition, most of the studies in developing countries have relied on incomplete data of unknown quality. Therefore, decision makers seeking replication of North American and Western European results in their own cities and countries may be hesitant to draw inferences from previous studies. As a result, the Health Effects Institute embarked on the Public Health and Air Pollution in Asia (PAPA) project in order to examine the effects of PM_{10} and other pollutants in Bangkok and three Chinese cities (Hong Kong, Shanghai, and Wuhan).

Three previous studies have been conducted in Bangkok (Ostro 1998, 2000; Vajanapoom 2002). In an initial effort, Ostro and colleagues (1998, 1999) examined counts of daily mortality in Bangkok from 1992 through 1995 and reported a consistent association between PM_{10} and total cardiovascular and respiratory mortality, as well as mortality among certain age groups. For that analysis, however, daily data on PM_{10} were limited and of questionable quality. As a result, concentrations of PM_{10} were estimated from daily measurements of airport visibility. Since then, the air quality monitoring efforts for the Bangkok Metropolitan Region have been greatly expanded and improved, making them better able to address exposure issues for epidemiologic research. The Pollution Control Department within the Ministry of Natural Resources and Environment currently has five ambient and seven roadside air pollution monitoring stations that measure daily PM_{10} and gaseous pollutants. These monitors are located throughout the metropolitan area and have been operating since 1996. The resulting air quality data show that PM_{10} concentrations in Bangkok remain high and exceed both the annual and 24-hour standards set by Thailand, the United States, and WHO. Although PM_{10} concentrations decreased during the economic recession of 1997 and trended downward through 2001, they have risen during the last few years as the economy has recovered.

The daily mortality data used in the study by Ostro and colleagues (1999) were gathered and processed through a newly computerized system, and large periods of data were lost or of questionable quality because of the transition from a manual to on-line computerized system, which required adapting to new procedures and training of staff. Currently, the death reporting system is completely online, and the transitional period ended 3 years before our study period, so the data quality is assured. An online reporting system links each death notification, which is registered at the local registrar's office by the deceased's relative, to the Central Registrar Office. There all death notifications from local offices throughout Thailand are compiled into a usable database. Therefore, daily mortality data are complete, quality controlled, and readily available.

The 6 to 10 million residents of Bangkok are potentially exposed to high concentrations of PM_{10}. Two questions remain: first, whether they are adversely affected by the existing concentration levels of PM_{10} and, second, whether the impact per unit of PM_{10} concentration (μ/m^3) is similar to what is experienced in developed Western countries. The improvement in the collection system for mortality data and the expanded air monitoring program provided an excellent opportunity to reexamine the effects of PM_{10} and several gaseous pollutants on daily mortality for the years 1997 through 2003. In undertaking this project, we sought to expand the literature documenting the effects on mortality of ambient air pollution by adding a study in an Asian city. Our work in Bangkok may be particularly important since Bangkok is a warm, tropical city, and there is no covariation and potential confounding between pollution and cold weather, with its associated adverse respiratory problems. Therefore, we were able to test the association between PM and health essentially without the confounding effects of most weather factors.

Of equal importance, we sought to add to the small body of literature documenting the health effects of air pollution in developing countries, particularly on the Asian continent.

Since our research team included investigators from both Thailand and the United States, an additional objective of our work was the transfer of technical knowledge from American to Thai investigators regarding data collection, data analysis, and the quantitative assessment of health outcomes. Ultimately, we hope this effort will be useful in fostering additional epidemiologic inquiries into air pollution in Thailand and will aid Thai decision makers in determining priorities among many competing environmental and nonenvironmental issues that affect public health.

SPECIFIC AIMS

Our study took advantage of the improved sources of data available to test several specific hypotheses. First, using improved data on PM_{10} (as well as data on gases such as O_3, NO_2, and SO_2) and mortality, we tested for an association between ambient air pollution and mortality in Bangkok. For this effort, both the mortality and air pollution data went through a rigorous quality assurance and control program administered by HEI. Second, we examined associations between air pollution and both disease- and age-specific mortality, in order to identify potential subgroups at particular risk. Finally, we conducted extensive sensitivity analyses of the results in order to examine the influence of model specification, smoothing methods, lag structure, and copollutants.

DATA AND METHODS

DATA

In this study, three basic categories of data were collected: (1) mortality data, (2) air pollution data, and (3) meteorologic data. All three databases were compiled electronically by the responsible government agencies, as described in the next few sections.

Mortality Data

Currently, under the administration of the Bureau of Registration Administration, the reporting system for deaths is entirely online and computerized with no manual recording of death certifications or cause of death. The Bangkok Metropolitan Region has a population of around 6 to 10 million people, with about 100 deaths per day. Available data used in this study for each individual death included date of death, age and sex of deceased, and cause of death. We compiled the data for individual deaths from June 1, 1997, to May 31, 2003, in order to determine the total number of deaths each day in Bangkok, as well as the number of deaths in various disease- and age-specific categories.

During evaluation of the data quality, we observed significantly lower death counts for 1997 and 1998 than for the rest of the study period (1999 to 2003). In order to determine whether the variations we found were systematic or random, we examined the distribution of deaths by district and validated the results with the local hard copy of death confirmation records, which is a carbon copy of the printout of the death certificate kept at the local registrar's office. The results show that 4 out of 50 districts (districts 4, 8, 17, and 37) had death counts that were different from those recorded in the 1997 and 1998 databases we received from the Ministry of Public Health, with a much higher number of deaths in the local hard copy database. To avoid these discrepancies, we used only data from 1999 to 2003, thereby deviating from our original study plan.

The Ministry of Public Health currently uses the tenth revision of the International Classification of Diseases (ICD-10) to categorize cause of death. All deaths must be certified by a physician, and the primary cause and underlying cause of death are recorded on a temporary death certificate by attending physicians at hospitals. Relatives of the deceased must submit the temporary death certificate to the local Registrar Office, and in turn, the Registrar Office issues an official death certificate.

As mentioned earlier, the system for reporting deaths is entirely computerized, and both the primary causes and underlying causes of death are recorded as they appeared on the official death certificate. The database is then sent to the Ministry of Public Health for assignment of the ICD-10 code. Thus, consistency in the classification of causes and underlying causes of death is maintained.

We subtracted from the total mortality count accidents and homicides, leaving deaths from "natural/nonaccidental" causes (ICD-10 codes A00–R99) — more specifically, deaths due to respiratory-specific causes (J00–J98) and cardiovascular-specific causes (I00–I99); and deaths falling into some additional subcategories, including deaths from ischemic heart disease (I20–I25), conduction disorders (I44–I49), LRIs (J10–J22), chronic obstructive pulmonary disease (COPD) (J40–J47), asthma (J45–J46), and senility (R54). We also examined two control groups: (1) those who died from natural, non-cardiopulmonary causes; and (2) those who died from accidental causes. Although senility is not usually considered in time-series mortality

studies, we examined this cause of death because our preliminary analysis showed a relatively low number of daily deaths from cardiovascular diseases. We hypothesized that this might have been a result of mislabeling the cause of death as being senility rather than cardiovascular disease, especially when an elderly person died outside the hospital. In those cases, the cause of death may have been diagnosed as senility by a nonphysician coroner. We included this hypothesis of possible misdiagnosis in our investigation. We also examined mortality stratified by sex and by age for the following age groups: 0 to 4 years, 5 to 44 years, 18 to 50 years, 45 to 64 years, more than 50 years, more than 65 years, and more than 75 years.

Air Pollution Data

The Pollution Control Department manages the air pollution monitoring in Thailand. Standard methods recommended by the U.S. EPA are used in measuring air pollution. In Bangkok, there are five ambient and seven roadside monitoring stations that have been measuring hourly ambient levels of PM_{10} since 1996; ten stations that have been measuring hourly ambient NO_2, SO_2, and NO since 1995; and eight stations that have been measuring hourly ambient O_3 since 1997. We carefully reviewed the monitoring data and picked sites based on consistency, lack of outliers, and completeness. In addition, we used only those monitors that met the site selection criteria, specifically, those that were not in the immediate vicinity of traffic or industrial sources and were, therefore, not overinfluenced by local sources (e.g., highways, industries, and open burning). As a result of these criteria, designed to avoid mismeasurement and overestimation of PM_{10} exposure, we did not use any of the available roadside monitoring stations since their measurements are unlikely to be representative of general population exposure.

To ensure the representativeness of the daily air pollution data, days with fewer than 18 hourly readings were considered ineligible and therefore excluded from the analyses. We calculated 24-hour averages for NO_2, NO (using the difference between NO_X and NO_2), SO_2, and PM_{10}, with the requirement that at least 75% of the 1-hour values be available on that particular day. For the 8-hour average value of O_3, at least six hourly values from 10 am to 6 pm had to be available, since the maximum O_3 levels always occur during daylight (Mikkelsen et al. 2000; Mair et al. 2002; Tao et al. 2004; Reddy 2008). The daily concentrations for each pollutant used in the analysis were calculated by taking the mean of the measurements from all available monitoring stations. We used only the stations with a completion rate of at least 75% of the measurements over the study period. With this criterion, the data completion rate for all pollutants was 100%, except for PM_{10}, which had 4 ineligible days due to missing data.

Averaging the pollutant concentrations over all monitoring stations provided the best representation of population exposure to air pollution for several reasons: First, the Pollution Control Department strategically placed the monitoring stations (five stations for ambient PM_{10}; ten for NO_2, SO_2, and NO; and eight for O_3) over an area of 1600 km^2 throughout Bangkok in order to capture exposures over the entire metropolitan area. However, two-thirds of these monitoring stations were situated within the inner city area, comprising approximately 300 km^2, where most of Bangkok's 6 to 10 million residents live; this area has a greater population density than the outer rim of the city. Second, many Bangkok residents work outside of their homes. Many offices are located in the inner city, and residents must travel to their workplaces on a daily basis. Therefore, averaging pollutant concentration measurements across various stations rather than measuring concentrations only at selected stations can capture the variation in population exposure over time and space. Moreover, most time-series studies of major metropolitan areas have adopted a similar approach.

Meteorologic Data

Daily weather data collected at the Don Muang Airport weather station and the Bangkok metropolitan weather station (at the Queen Sirikit National Convention Center) were available for the study period. The data obtained included average daily temperature and average daily relative humidity (RH). Daily weather variables at the two Bangkok locations were found to be highly correlated with each other (Ostro et al. 1999). We chose to use the data from the Bangkok metropolitan weather station, which were more complete (100%).

METHODS

To assess the short-term effects of PM_{10} and gaseous pollutants on daily mortality, we followed the Common Protocol developed by participants in the PAPA project, which included research teams representing Bangkok, Hong Kong, Shanghai, and Wuhan (see the Common Protocol at the end of this volume). We applied generalized linear models using a Poisson regression, conditional on several independent variables, in order to control for temporal trends and meteorologic conditions. In the basic analytic approach, we employed natural cubic spline models using the statistical software package R, version 2.5, with mgcv, version 1.3-24 (R Development Core Team 2007, Vienna, Austria). A *natural spline model* uses a parametric approach that fits cubic functions together by joining them

at *knots*, which are typically placed evenly throughout the distribution of the variable of concern (e.g., time). The number of knots used determines the overall fit for the temporal smoothing function. Before entering measurements of an air pollutant into the model, we used this technique to determine the best core model for all mortalities attributed to natural causes, while controlling for time, season, temperature, RH, day of the week, and whether or not it was a public holiday. This was undertaken to control for factors, besides air pollutants, that vary on a daily basis and that might explain variations in daily mortality. Same-day exposure to the air pollutants, single-day lags up to five days, and moving averages of up to five days were examined.

All studies in the PAPA project examined a core analytic model, which employed 4 to 6 df per year for the smooth function of time and 3 df for the whole study period for the smooth function of the same-day lag of daily mean temperature and daily mean RH. The number of degrees of freedom used has been shown in many previous studies to provide adequate control for these potential confounders (HEI 2003). Nevertheless, we conducted a sensitivity analysis by considering as many as 15 df for time per year. The best core model and number of degrees of freedom for the smooth function of time were chosen to minimize serial correlation. Specifically, we used the criterion of minimizing the absolute magnitude of partial autocorrelation function (PACF) values, with an additional requirement that the first-order autocorrelation (AR1) and the second-order autocorrelation be less than 0.1. The PACF measures the absolute value of autocorrelation for lags from 1 to 30 days. We used a quasi-Poisson option in the regression models to correct for any overdispersion in the data.

In the sensitivity analysis, we assessed the possibility that an influenza epidemic could be a potential confounder of the associations. Unfortunately, daily death counts for influenza in Bangkok were likely to be underreported, so we defined an influenza epidemic as existing when the weekly respiratory mortality counts were greater than the 90th percentile of the mean frequency (count) of respiratory mortality for the given year. We also used sensitivity analyses to assess the impact of different model specifications on our results. We included models with various sets of degrees of freedom for time and weather and with different lags for temperature and RH, using penalized spline smoothing functions for time and weather in place of natural splines with the same degrees of freedom. We included autoregressive terms in the model where appropriate. We also fit copollutant models assessing the effects of PM_{10} with adjustments for gaseous pollutants, and vice versa.

RESULTS

DESCRIPTIVE ANALYSIS

Tables 1 to 3 summarize the daily mortality data in Bangkok from January 1, 1999, to December 31, 2003. There was an average of 95 deaths per day from mortality due to all natural causes. About 8% and 14% of the total consisted of mortality from respiratory and cardiovascular diseases, respectively, and about half of the total involved those aged 65 and older. This study showed that males made up about 64% of the total mortality in Bangkok, although a lower proportion of men (48%) were living in Bangkok during the study period (Bangkok Metropolitan Administration 2003). The mean number of deaths per day for various causes of death did not change much when stratified by seasons (Table 3), indicating that season is unlikely to be an effect modifier for the Bangkok data. The long-term trends of daily mortality from all natural causes (for all ages and among those aged 65 and older), cardiovascular causes, and respiratory diseases over the study period are shown in Figure 1. We observed extremely low death counts on December 31 in 1999 and 2000. It is likely that these outliers were caused by an error in the data recording process.

The mortality data for Bangkok during this time period shows slightly increasing trends without apparent seasonal patterns. This evidence is quite different from that usually observed in temperate regions, where mortality peaks during the winter months, suggesting that time and seasonality may not be strong confounding factors for the acute effects

Table 1. Average Daily Mortality by Sex and Age in Bangkok from January 1, 1999, to December 31, 2003[a]

	Deaths per Day Mean (SD)
Sex	
Male	61 (8.9)
Female	43 (7.6)
Age (yr)	
< 5	3 (1.8)
5–44	29 (5.9)
18–50	34 (6.4)
45–64	27 (5.4)
50+	66 (9.9)
65+	45 (7.9)
75+	21 (5.2)

[a] Definition: SD indicates standard deviation.

Table 2. Distribution of Daily Mortality by Cause of Death in Bangkok from January 1, 1999, to December 31, 2003[a]

Cause of Death (All Ages)	ICD-10 Codes	Mean	Minimum	Maximum	SD	Percentiles 25	50	75	100
All natural	A00–R99	95	29	147	12.1	87	95	103	147
Cardiopulmonary	I00–I99, J00–J98	22	5	47	5.7	18	21	25	47
Cardiovascular	I00–I99	14	1	28	4.3	10	13	16	28
Ischemic heart diseases	I20–I25	4	1	16	2.3	3	4	6	16
Stroke	I60–I69	5	1	17	2.5	4	5	7	17
Conduction disorder	I44–I49	1	1	4	0.5	1	1	1	4
Respiratory	J00–J98	8	1	20	3.1	6	8	10	20
COPD	J40–J47	2	1	6	1.0	1	2	2	6
LRI	J10–J22	4	1	13	2.3	3	4	6	13
LRI < 5 yr	J10–J22	1	1	4	0.4	1	1	1	4
Respiratory < 1 yr	J00–J98	0.1	0	2	0.4	0	0	0	2
Asthma	J45–J46	1	1	4	0.4	1	1	1	4
Senility	R54	14	1	30	4.2	9	12	15	30
Non-cardiopulmonary and natural (others)	A00–H95, K00–R99	76	22	116	10.4	67	73	80	116
Accidental	V01–V99, W00–X59	3	0	11	1.8	1	2	4	11

[a] Definitions: COPD indicates chronic obstructive pulmonary disease; LRI indicates lower respiratory infection; SD indicates standard deviation.

Table 3. Average Daily Mortality by Cause of Death and Seasons in Bangkok from January 1, 1999, to December 31, 2003[a]

Cause of Death (All Ages)	Season: Summer Mean (SD)	Winter Mean (SD)	Rainy Mean (SD)
All natural	97 (12.0)	95 (10.9)	94 (13.4)
Cardiopulmonary	21 (5.5)	22 (5.6)	22 (5.5)
Cardiovascular	13 (4.3)	14 (4.2)	14 (4.2)
Ischemic heart diseases	4 (2.1)	4 (2.4)	5 (2.2)
Stroke	5 (2.5)	5 (2.5)	6 (2.5)
Conduction disorder	1 (0.5)	1 (0.5)	1 (0.4)
Respiratory	8 (3.0)	8 (3.2)	8 (3.1)
COPD	2 (1.0)	2 (1.1)	2 (0.9)
LRI	4 (2.2)	5 (2.3)	4 (2.3)
LRI < 5 yr	1 (0.3)	1 (0.5)	1 (0.2)
Respiratory < 1 yr	0.1 (0.3)	0.1 (0.4)	0.1 (0.4)
Asthma	1 (0.4)	1 (0.4)	1 (0.4)
Senility	14 (3.9)	11 (4.2)	12 (4.2)
Non-cardiopulmonary and natural (others)	76 (10.3)	73 (9.6)	72 (11.0)
Accidental	3 (1.8)	3 (1.7)	3 (1.7)

[a] Definitions: COPD indicates chronic obstructive pulmonary disease; LRI indicates lower respiratory infection; SD indicates standard deviation.

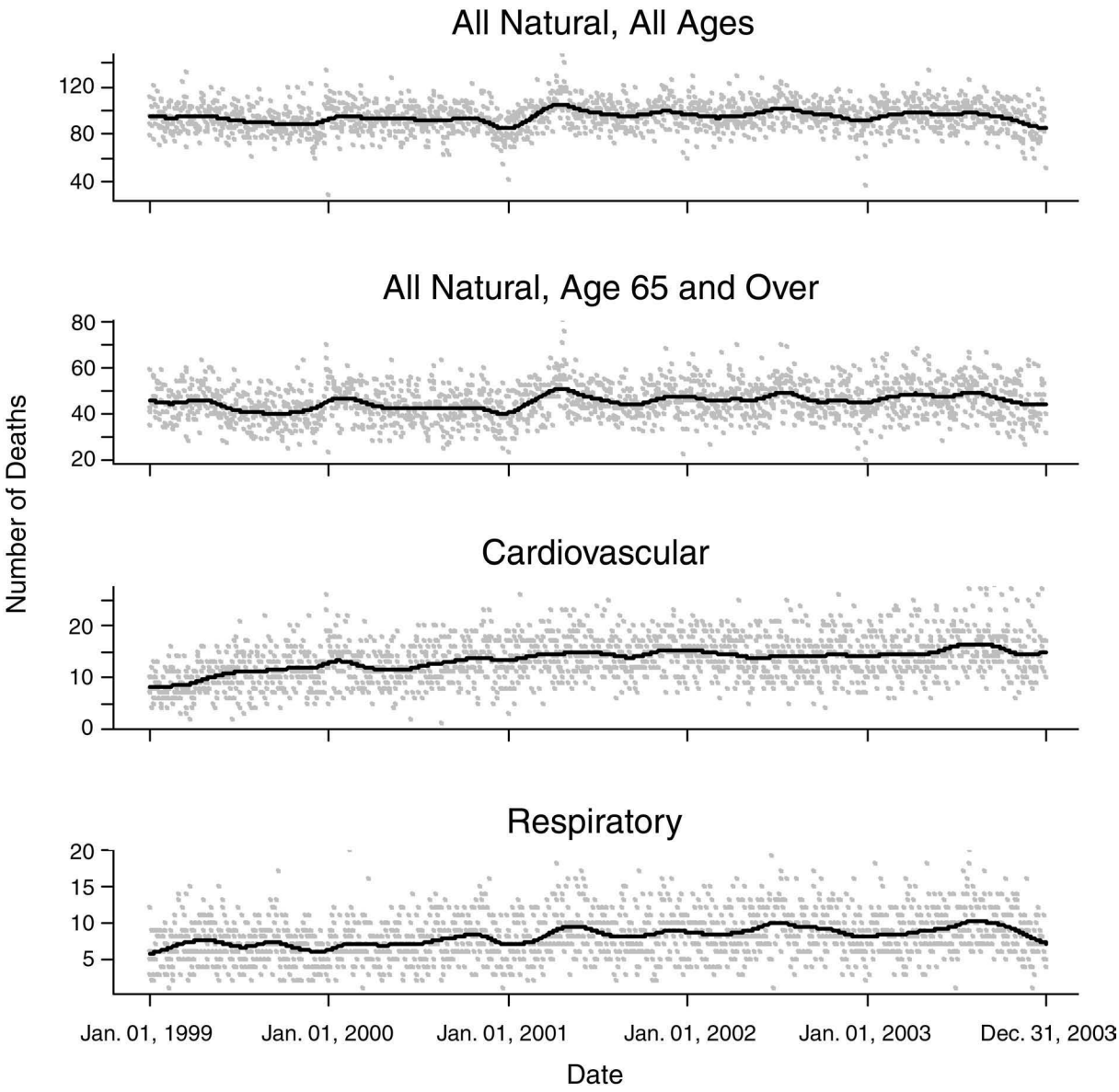


Figure 1. Smooth function plots of daily mortality due to several causes in Bangkok from 1999 to 2003.

of PM_{10} on mortality in Bangkok. A tropical country, Thailand has three seasons, all warm: "winter" from mid-October to mid-February; "summer" from mid-February to mid-May; and a rainy season from mid-May to mid-October. Generally, there is not much variation in the temperature across the three seasons, which may explain the lack of any large seasonal pattern in mortality.

Tables 4, 5, and 6 summarize the pollution data. Table 4 indicates the percentage of valid measurements at each of the monitors. For example, four of the five PM_{10} monitors collected data on 90% or more of the possible days. Except for station 7, the data for the gaseous pollutants were fairly complete. The locations of the air monitoring sites are shown in Figure 2, which indicates that they are fairly evenly distributed throughout Bangkok. The correlations between the air pollutants are shown in Table 5. We observed a high correlation between PM_{10} and NO_2 (r = 0.71), and a relatively high correlation of O_3 with NO_2 (r = 0.62) and PM_{10} (r = 0.55). The correlation patterns between the air pollutants were relatively similar when stratified by season (see Table B.1 in Appendix B) or by monitor (see Tables B.2 and B.3 in Appendix B). Generally, the correlations between stations for each air pollutant were relatively high, except for SO_2 (see Table B.3). This indicates that

Table 4. Percentage of Valid Measures of Air Pollutants in Each Monitoring Station Over the Five-Year Study Period (1999–2003)[a]

	General Monitoring Stations (Station Number)									
Pollutant	1	2	3	5	7	9	10	11	12	15
PM_{10}	—	—	—	—	—	82	95	94	90	91
SO_2	87	95	95	82	84	70	83	84	86	86
NO_2	77	94	94	71	62	79	86	87	81	81
O_3	—	—	94	59	61	83	88	90	86	85
NO	77	93	93	72	65	82	86	87	81	81

[a] Definition: — indicates station not set up to monitor this pollutant.

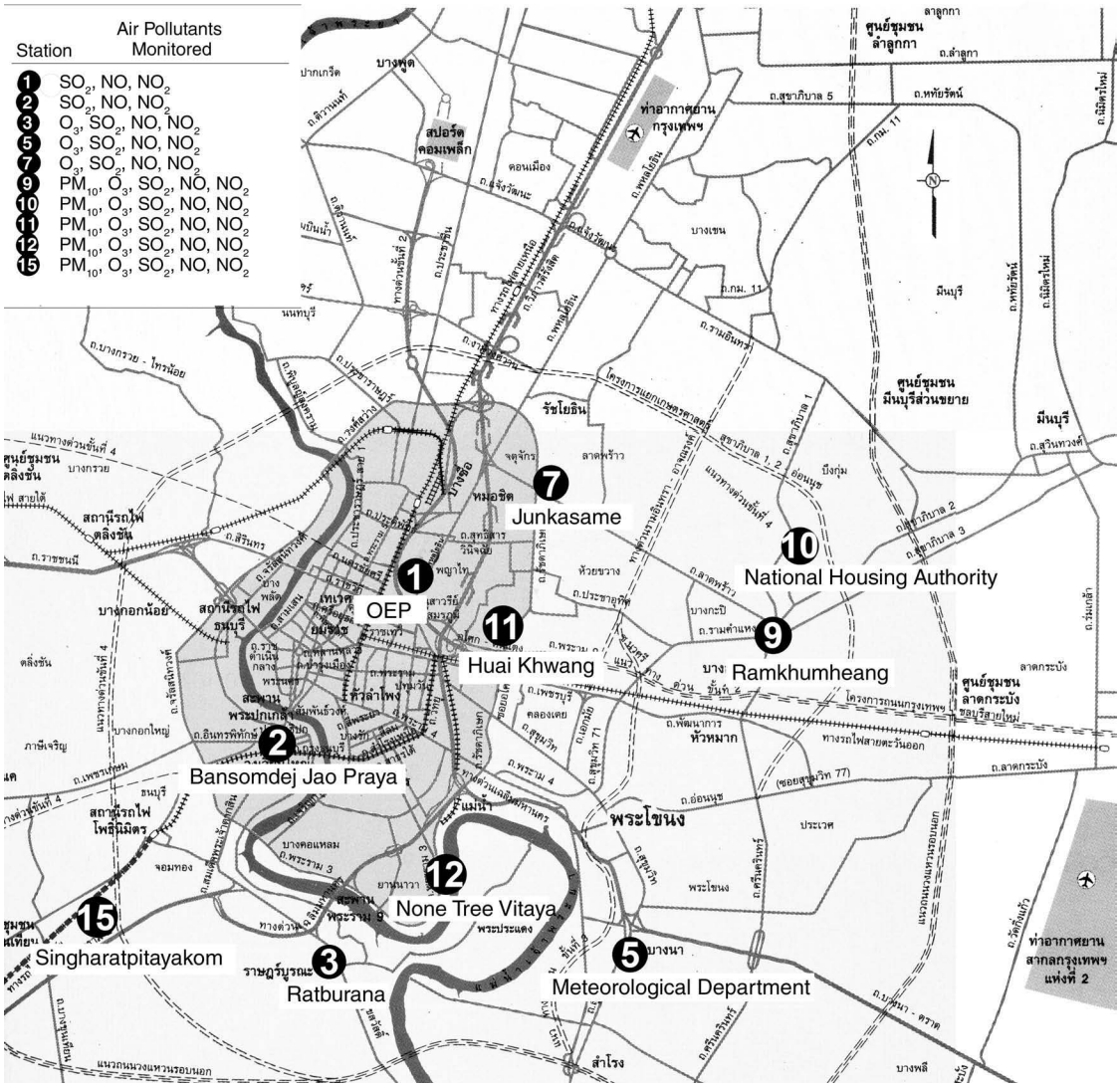


Figure 2. Location of air monitoring stations in Bangkok and list of pollutants monitored. (OEP indicates Office of Environmental Policy and Planning.)

Table 5. Spearman Correlation among PM_{10} and Specific Gaseous Pollutants from January 1, 1999, to December 31, 2003

Pollutant	PM_{10}	SO_2	NO_2	O_3	NO
PM_{10}	1.00	0.24	0.71	0.55	0.22
SO_2		1.00	0.27	0.18	0.38
NO_2			1.00	0.62	0.36
O_3				1.00	−0.07
NO					1.00

most of the pollutants tend to move together across the city over time and that production of SO_2 is most likely more local. Table 6 provides the statistical distributions of the air pollutant and weather data used in this analysis. Over the five-year study period, there were four missing days for PM_{10}, and no missing days for the other pollutants. Figure 3 shows the evidence of seasonal patterns of daily PM_{10}, NO_2, O_3, and NO concentrations, with peaks in winter. However, SO_2 does not show clear evidence of a seasonal pattern. Similar evidence was also observed for station-specific plots (see Figures C.1–C.5 in Appendix C). PM_{10} concentration levels in Bangkok decreased from 1999 to 2001 (in fact, the decline started in the third quarter of 1997 concurrent with a severe economic downtown) and then increased again from 2001 to 2003 (Figures C.1–C.5). The mean and median of PM_{10} were 52.1 and 46.8 μg/m^3, respectively, and the interquartile (75th to 25th percentile) values were 59.9 and 38.9 μg/m^3, respectively, with a maximum value of 169.2 μg/m^3 (see Table 6). This illustrates that daily PM_{10} levels in Bangkok were higher than those in many cities in Western countries (Anderson et al. 2004) and exceeded the 24-hour and annual average ambient air quality standards for Thailand and the United States (Thailand Pollution Control Dept. 2007; U.S. EPA 2007). The median concentrations of SO_2, NO_2, O_3, and NO were 12.5 μg/m^3, 39.7 μg/m^3, 59.4 μg/m^3, and 28.0 μg/m^3, respectively (Table 6). The weather in Bangkok was generally hot and humid for the 5-year study. The median 24-hour temperature was 29.1°C, and the median daily average humidity was 73.1%.

ANALYTIC RESULTS

Initially, we examined several core models for all natural mortality counts as a function of natural spline smoothing for time, unlagged (lag 0 day) temperature, and RH at lag 0 day, along with dummy variables for the day of week and for public holidays. In the core model, we used an average of zero- and 1-day lag (lag 0–1 day) for the pollution term. The results of this core model are displayed

Table 6. Distribution of Air Pollutant and Meteorologic Data in Bangkok from January 1, 1999, to December 31, 2003[a]

Pollutant / Meteorologic Measurement	Days (*n*)	Mean	Minimum	Maximum	SD	Percentiles				
						5	25	50	75	95
PM_{10} (μg/m^3)	1822	52.1	21.3	169.2	20.1	29.6	38.9	46.8	59.9	93.2
SO_2 (μg/m^3)	1826	13.2	1.5	61.2	4.8	7.1	10.1	12.5	15.6	21.0
NO_2 (μg/m^3)	1826	44.7	15.8	139.6	17.3	24.4	31.7	39.7	54.8	79.3
O_3 (μg/m^3)	1826	59.4	8.2	180.6	26.4	25.3	39.1	59.4	75.3	109.8
NO (μg/m^3)	1826	28.0	3.7	126.9	14.2	11.4	18.1	28.0	34.9	56.0
Temperature[b] (°C)	1826	28.9	18.7	33.6	1.7	25.8	28.1	29.1	29.9	31.3
RH[c] (%)	1826	72.8	41.0	95.0	8.3	58.0	67.8	73.1	78.3	86.0

[a] Definitions: RH indicates relative humidity; SD indicates standard deviation.

[b] Average daily temperature.

[c] Average relative humidity.

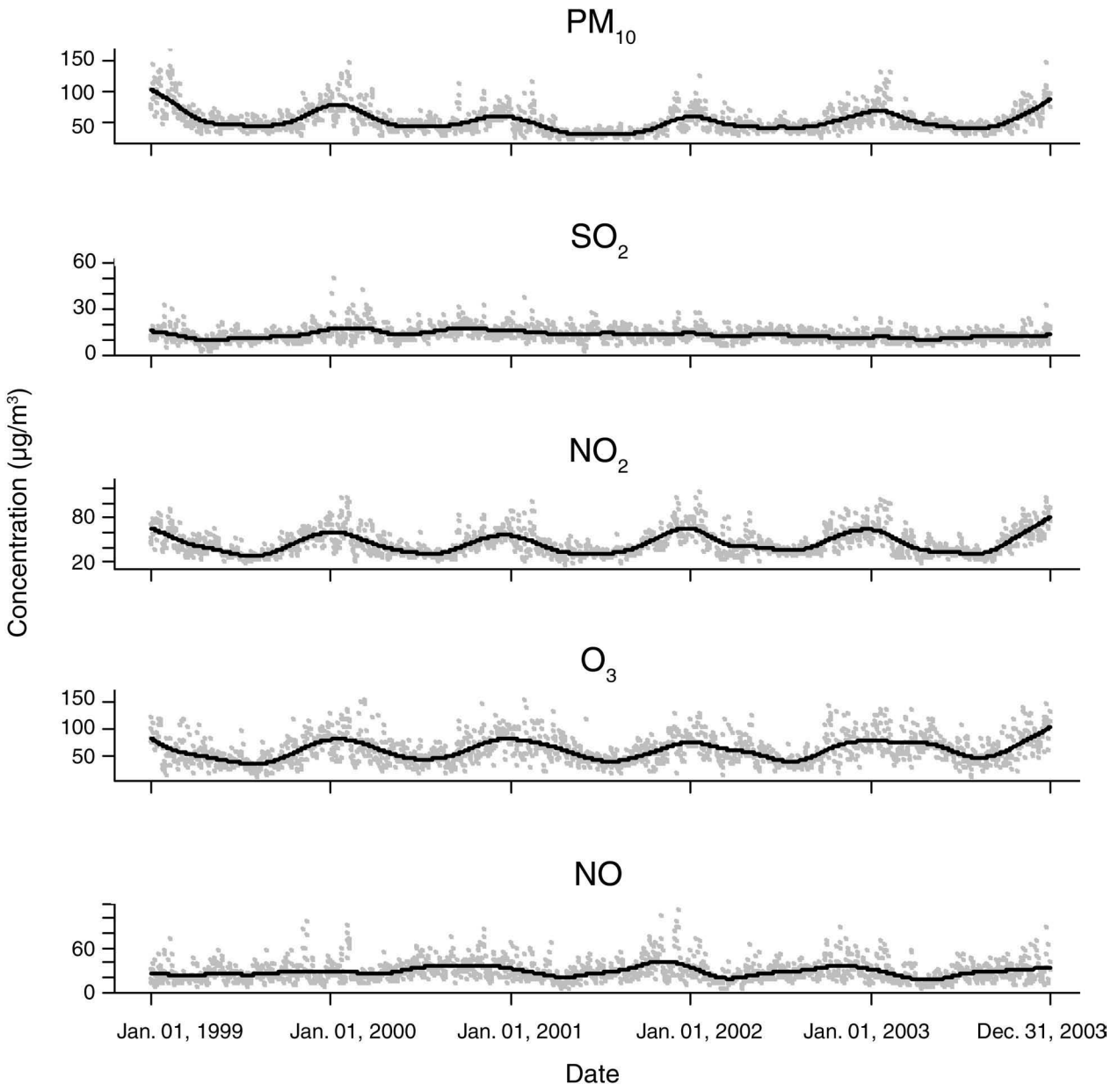


Figure 3. Smooth function plots of daily concentrations of air pollution in Bangkok from 1999 to 2003.

in Table 7a (showing the effects per 10-µg/m^3 increase in pollutants for various disease-specific causes of mortality as well as age- and sex-specific mortality) and Table 7b (showing the effects per interquartile range [IQR] of increases in pollutants). (Details of the results for other lags are shown in Table 8 and Appendix D). In developing the core model, as part of the PAPA Common Protocol, we examined the PACF plots of the models with 4 to 6 df for time per year of data and 3 df for temperature and RH for the entire study period. The results indicated that the models with 4 and 5 df for time provided PACF values with an AR1 of greater than 0.1. In contrast, the first- and second-order autocorrelations of the model with 6 df per year of data for the smooth function of time were less than 0.1 (Figure 4A), which thereby met the requirements of the PAPA protocol. As a result, we selected this model as the core model for most of our analyses.

As shown in Figure 4A, the AR1 was statistically significant, suggesting that the residuals may indicate that there could be some underestimation of the standard errors

Table 7a. Percentage of Excess Risk in Mortality and Confidence Interval for a 10-μg/m^3 Increase in Air Pollutants with Lag 0–1[a]

Mortality Class	PM_{10} % ER (95% CI)	SO_2 % ER (95% CI)	NO_2 % ER (95% CI)	O_3 % ER (95% CI)	NO % ER (95% CI)
Cause-Specific (All Ages)					
All natural	1.3 (0.8 to 1.7)	1.6 (0.1 to 3.2)	1.4 (0.9 to 1.9)	0.6 (0.3 to 0.9)	1.1 (0.6 to 1.6)
Cardiopulmonary	1.6 (0.7 to 2.5)	1.1 (−1.91 to 4.2)	1.5 (0.5 to 2.6)	0.9 (0.2 to 1.5)	1.0 (0.0 to 2.0)
Cardiovascular	1.9 (0.8 to 3.0)	0.8 (−3.0 to 4.7)	1.8 (0.5 to 3.1)	0.8 (0.0 to 1.6)	2.0 (0.7 to 3.2)
Ischemic heart disease	1.5 (−0.4 to 3.5)	1.9 (−4.7 to 9.0)	1.5 (−0.7 to 3.8)	0.0 (−1.4 to 1.4)	2.8 (0.6 to 5.0)
Stroke	2.3 (0.6 to 4.0)	0.8 (−5.1 to 7.1)	2.8 (0.7 to 4.9)	2.2 (1.0 to 3.5)	1.9 (−0.1 to 3.9)
Conduction disorders	−0.3 (−5.9 to 5.6)	−5.0 (−23.1 to 17.3)	1.3 (−5.4 to 8.4)	−3.5 (−7.5 to 0.6)	3.8 (−2.7 to 10.9)
Respiratory	1.0 (−0.4 to 2.4)	1.7 (−3.1 to 6.6)	1.0 (−0.6 to 2.7)	0.9 (−0.1 to 1.9)	−0.7 (−2.3 to 0.9)
Respiratory ≤ 1 yr	14.6 (2.9 to 27.6)	97.7 (23.2 to 217.2)	10.7 (−2.0 to 25.2)	3.4 (−4.0 to 11.3)	3.4 (−7.90 to 16.0)
LRI	0.5 (−1.4 to 2.5)	2.2 (−4.6 to 9.4)	1.3 (−0.9 to 3.7)	1.2 (−0.2 to 2.6)	0.1 (−2.1 to 2.3)
LRI < 5 yr	7.7 (−3.6 to 20.3)	14.4 (−22.6 to 69.3)	8.2 (−4.9 to 23.2)	4.9 (−3.0 to 13.4)	6.9 (−5.3 to 20.7)
COPD	1.3 (−1.8 to 4.4)	3.0 (−7.1 to 14.1)	−1.4 (−5.0 to 2.4)	−0.8 (−3.0 to 1.5)	−1.7 (−5.1 to 1.8)
Asthma	7.4 (1.1 to 14.1)	−1.5 (−20.4 to 21.9)	3.9 (−3.5 to 12.0)	1.3 (−3.3 to 6.0)	−2.8 (−9.5 to 4.3)
Senility	1.8 (0.7 to 2.8)	3.2 (−0.5 to 7.1)	2.7 (1.3 to 4.0)	1.6 (0.8 to 2.4)	1.8 (0.6 to 3.1)
Others[b]	1.2 (0.8 to 2.0)	1.8 (0.1 to 3.5)	1.4 (0.8 to 2.0)	0.6 (0.2 to 0.9)	1.1 (0.5 to 1.7)
Accidental	0.1 (−2.3 to 2.6)	0.0 (−8.0 to 8.8)	−0.1 (−3.0 to 2.8)	0.0 (−1.8 to 1.8)	0.8 (−2.0 to 3.6)
Age-Specific (All Natural) (yr)					
0–4	0.2 (−2.0 to 2.4)	3.4 (−4.1 to 11.5)	−0.4 (−3.1 to 2.2)	−0.8 (−2.4 to 0.8)	0.9 (−1.6 to 3.5)
5–44	0.9 (0.2 to 1.7)	0.2 (−2.4 to 2.8)	0.9 (0.0 to 1.8)	0.4 (−0.2 to 1.0)	0.6 (−0.2 to 1.5)
18–50	1.2 (0.5 to 1.9)	0.9 (−1.5 to 3.3)	1.4 (0.5 to 2.2)	0.6 (0.1 to 1.1)	0.7 (−0.1 to 1.5)
45–64	1.1 (0.4 to 1.9)	0.9 (−1.7 to 3.5)	1.3 (0.4 to 2.2)	0.8 (0.3 to 1.4)	0.8 (0.0 to 1.7)
50+	1.4 (0.9 to 1.9)	2.3 (0.5 to 4.2)	1.7 (1.1 to 2.3)	0.8 (0.5 to 1.2)	1.2 (0.6 to 1.8)
65+	1.5 (0.9 to 2.1)	2.8 (0.7 to 5.0)	1.8 (1.1 to 2.6)	0.8 (0.4 to 1.3)	1.3 (0.6 to 2.0)
75+	2.2 (1.3 to 3.0)	4.6 (1.7 to 7.7)	2.9 (1.9 to 4.0)	1.5 (0.9 to 2.1)	1.6 (0.7 to 2.6)
Sex-Specific (All Natural)					
Male	1.2 (0.7 to 1.7)	0.8 (−1.0 to 2.7)	1.2 (0.6 to 1.9)	0.6 (0.2 to 1.0)	0.8 (0.2 to 1.4)
Female	1.3 (0.7 to 1.9)	2.6 (0.4 to 4.8)	1.6 (0.9 to 2.4)	0.7 (0.3 to 1.2)	1.2 (0.5 to 1.9)

[a] Definitions: CI indicates confidence interval; COPD indicates chronic obstructive pulmonary disease; ER indicates excess risk; LRI indicates lower respiratory infection.

[b] Non-cardiopulmonary and natural.

(SEs) of the parameter estimates. To address this problem, an autoregressive model was implemented. As shown in Figure 4B, the AR1 was removed with the autoregressive model, with little impact on the effect estimate or SE (shown among other sensitivity analyses in Table 9). Additionally, there was no evidence of seasonal patterns in and overdispersions of the Pearson residual plots of the core models for major outcomes (i.e., all natural, cardiovascular-related, and respiratory-related deaths, and deaths among those aged 65 and older; Figure 5).

Regarding PM_{10}, we observed statistically significant associations with most of the outcomes, including all natural and cardiovascular mortality (Table 7a). A positive, but not-significant association was observed for respiratory mortality. The ER—calculated as (relative risk − 1) × 100—for mortality due to all natural causes was 1.3% (95% CI, 0.8 to 1.7), with a higher ER for cardiovascular mortality of 1.9% (95% CI, 0.8 to 3.0). With respect to subgroups of cardiovascular disease, many were associated with PM_{10} (and other pollutants), with mortality from stroke demonstrating

Table 7b. Percentage of Excess Risk in Mortality and Confidence Interval for an Interquartile Range[a] Increase in Air Pollutants with Lag 0–1[b]

Mortality Class	PM_{10} % ER (95% CI)	SO_2 % ER (95% CI)	NO_2 % ER (95% CI)	O_3 % ER (95% CI)	NO % ER (95% CI)
Cause-Specific (All Ages)					
All natural	2.6 (1.7 to 3.6)	0.9 (0.0 to 1.7)	3.3 (2.1 to 4.6)	2.3 (1.1 to 3.5)	1.8 (0.9 to 2.7)
Cardiopulmonary	3.3 (1.5 to 5.2)	0.6 (−1.1 to 2.3)	3.5 (1.1 to 6.0)	3.1 (0.8 to 5.5)	1.7 (0.0 to 3.4)
Cardiovascular	4.0 (1.7 to 6.4)	0.4 (−1.7 to 2.5)	4.2 (1.1 to 7.3)	3.0 (0.1 to 6.0)	3.3 (1.3 to 5.5)
Ischemic heart disease	3.2 (−0.9 to 7.4)	1.0 (−2.6 to 4.9)	3.6 (−1.7 to 9.1)	0.1 (−4.8 to 5.2)	4.7 (1.04 to 8.6)
Stroke	4.8 (1.2 to 8.6)	0.5 (−2.8 to 3.9)	6.5 (1.7 to 11.7)	8.3 (3.6 to 13.2)	3.1 (−0.1 to 6.5)
Conduction disorders	−0.6 (−11.9 to 12.2)	−2.8 (−13.5 to 9.2)	3.0 (−12.0 to 20.6)	−12.2 (−24.6 to 2.3)	6.5 (−4.6 to 18.9)
Respiratory	2.1 (−0.7 to 5.1)	0.9 (−1.7 to 3.6)	2.4 (−1.4 to 6.4)	3.3 (−0.4 to 7.0)	−1.2 (−3.8 to 1.4)
Respiratory ≤ 1 yr	33.0 (6.2 to 66.5)	50.2 (21.2 to 86.1)	26.6 (−4.6 to 68.2)	12.7 (−13.8 to 47.3)	5.7 (−12.9 to 28.2)
LRI	1.1 (−2.8 to 5.3)	1.2 (−2.5 to 5.0)	3.1 (−2.2 to 8.7)	4.5 (−0.6 to 9.8)	0.2 (−3.4 to 3.9)
LRI < 5 yr	16.8 (−7.3 to 47.1)	7.7 (−13.1 to 33.5)	20.1 (−11.0 to 62.0)	18.7 (−10.5 to 57.4)	11.9 (−8.7 to 37.1)
COPD	2.7 (−3.7 to 9.5)	1.6 (−3.9 to 7.5)	−3.2 (−11.2 to 5.6)	−2.9 (−10.5 to 5.4)	−2.8 (−8.4 to 3.1)
Asthma	16.1 (2.4 to 31.8)	−0.8 (−11.8 to 11.5)	9.3 (−8.0 to 29.9)	4.6 (−11.3 to 23.4)	−4.7 (−15.5 to 7.4)
Senility	3.7 (1.5 to 6.0)	1.8 (−0.3 to 3.8)	6.2 (3.1 to 9.5)	5.8 (2.8 to 8.9)	3.1 (1.0 to 5.3)
Others[c]	2.5 (1.4 to 3.5)	1.0 (0.0 to 1.9)	3.2 (1.9 to 4.6)	2.0 (0.7 to 3.3)	1.8 (0.9 to 2.8)
Accident	0.3 (−4.7 to 5.5)	0.0 (−4.5 to 4.7)	−0.3 (−6.9 to 6.7)	−0.1 (−6.3 to 6.6)	1.3 (−3.3 to 6.1)
Age-Specific (All Natural) (yr)					
0–4	0.4 (−4.1 to 5.0)	1.9 (−2.3 to 6.1)	−1.0 (−6.9 to 5.3)	−3.0 (−8.5 to 2.8)	1.6 (−2.6 to 5.9)
5–44	2.0 (0.4 to 3.6)	0.1 (−1.3 to 1.6)	2.2 (0.1 to 4.3)	1.5 (−0.5 to 3.5)	1.0 (−0.4 to 2.5)
18–50	2.5 (1.1 to 3.9)	0.5 (−0.8 to 1.8)	3.2 (1.2 to 5.1)	2.2 (0.4 to 4.1)	1.2 (−0.1 to 2.5)
45–64	2.4 (0.8 to 4.0)	0.5 (−0.9 to 1.9)	3.1 (1.0 to 5.2)	3.0 (1.0 to 5.0)	1.4 (0.0 to 2.9)
50+	3.0 (1.9 to 4.1)	1.3 (0.3 to 2.3)	4.0 (2.5 to 5.5)	3.1 (1.7 to 4.5)	2.1 (1.1 to 3.1)
65+	3.2 (2.0 to 4.5)	1.5 (0.4 to 2.7)	4.3 (2.6 to 6.0)	3.1 (1.4 to 4.7)	2.2 (1.0 to 3.4)
75+	4.6 (2.8 to 6.4)	2.5 (0.9 to 4.2)	6.9 (4.5 to 9.5)	3.1 (1.4 to 4.7)	2.8 (1.1 to 4.4)
Sex-Specific (All Natural)					
Male	2.5 (1.4 to 3.6)	0.5 (−0.6 to 1.5)	2.8 (1.3 to 4.4)	2.2 (0.8 to 3.7)	1.4 (0.3 to 2.4)
Female	2.7 (1.4 to 4.1)	1.4 (0.2 to 2.6)	3.8 (2.1 to 5.6)	2.7 (1.0 to 4.4)	2.1 (0.9 to 3.3)

[a] IQR: PM_{10} = 20.94 μg/m^3, SO_2 = 5.50 μg/m^3, NO_2 = 23.14 μg/m^3, O_3 = 36.16 μg/m^3, NO = 16.77 μg/m^3.

[b] Definitions: CI indicates confidence interval; COPD indicates chronic obstructive pulmonary disease; ER indicates excess risk; IRQ indicates interquartile range; LRI indicates lower respiratory infection.

[c] Non-cardiopulmonary and natural.

Table 8. Lag Effects for a 10-μg/m^3 Increase of PM_{10} and Gaseous Pollutants with Various Lags on Major Causes of Death[a]

Mortality Class / Pollutant	Lag 0 % ER (95% CI)	Lag 1 % ER (95% CI)	Lag 2 % ER (95% CI)	Lag 3 % ER (95% CI)	Lag 4 % ER (95% CI)	Lag 0–1 (Mean) % ER (95% CI)	Lag 0–4 (Mean) % ER (95% CI)
All Natural							
PM_{10}	1.2 (0.8 to 1.6)	0.9 (0.6 to 1.3)	0.9 (0.5 to 1.3)	0.8 (0.4 to 1.2)	0.3 (−0.1 to 0.7)	1.3 (0.8 to 1.7)	1.4 (0.9 to 1.9)
SO_2	1.6 (0.2 to 2.9)	1.0 (−0.4 to 2.3)	0.4 (−0.9 to 1.7)	0.5 (−0.8 to 1.8)	−0.2 (−1.5 to 1.1)	1.6 (0.1 to 3.2)	1.3 (−0.6 to 3.3)
NO_2	1.3 (0.8 to 1.8)	1.1 (0.6 to 1.5)	1.0 (0.5 to 1.4)	0.7 (0.2 to 1.1)	0.3 (−0.1 to 0.8)	1.4 (0.9 to 1.9)	1.6 (0.9 to 2.2)
O_3	0.5 (0.2 to 0.8)	0.4 (0.1 to 0.6)	0.3 (0.0 to 0.5)	0.4 (0.2 to 0.7)	0.2 (0.0 to 0.5)	0.6 (0.3 to 0.9)	0.9 (0.5 to 1.3)
NO	0.9 (0.5 to 1.4)	0.7 (0.3 to 1.2)	0.7 (0.2 to 1.1)	0.4 (0.0 to 0.8)	−0.2 (−0.6 to 0.2)	1.1 (0.6 to 1.6)	1.1 (0.4 to 1.7)
Cardiovascular							
PM_{10}	1.5 (0.5 to 2.6)	1.7 (0.7 to 2.7)	1.6 (0.6 to 2.6)	0.8 (−0.1 to 1.8)	−0.1 (−1.1 to 0.9)	1.9 (0.8 to 3.0)	1.9 (0.6 to 3.2)
SO_2	1.6 (1.8 to 5.0)	−0.3 (−3.6 to 3.1)	1.0 (−2.3 to 4.4)	−0.8 (−4.0 to 2.6)	−0.6 (−3.9 to 2.7)	0.8 (−3.0 to 4.7)	0.3 (−4.4 to 5.3)
NO_2	1.2 (−0.1 to 2.4)	1.7 (0.6 to 2.9)	1.4 (0.3 to 2.6)	0.5 (−0.6 to 1.7)	0.0 (−1.2 to 1.1)	1.8 (0.5 to 3.1)	1.8 (0.2 to 3.4)
O_3	0.3 (−0.4 to 1.0)	0.8 (0.2 to 1.5)	0.5 (−0.1 to 1.2)	0.2 (−0.4 to 0.8)	−0.3 (−0.9 to 0.4)	0.8 (0.0 to 1.6)	0.8 (−0.2 to 1.8)
NO	1.6 (0.5 to 2.7)	1.5 (0.4 to 2.6)	1.5 (0.5 to 2.6)	0.8 (−0.3 to 1.8)	−0.4 (−1.5 to 0.6)	2.0 (0.7 to 3.2)	2.1 (0.5 to 3.7)
Respiratory							
PM_{10}	1.0 (−0.3 to 2.3)	0.8 (−0.5 to 2.0)	1.1 (−0.1 to 2.3)	1.3 (0.1 to 2.6)	0.7 (−0.6 to 1.9)	1.0 (−0.4 to 2.4)	1.7 (0.1 to 3.4)
SO_2	1.4 (−2.8 to 5.7)	1.2 (−2.9 to 5.6)	2.9 (−1.3 to 7.3)	4.1 (−0.1 to 8.5)	2.2 (−2.0 to 6.5)	1.7 (−3.1 to 6.6)	5.1 (−1.1 to 11.7)
NO_2	1.0 (−0.5 to 2.6)	0.7 (−0.7 to 2.2)	0.7 (−0.8 to 2.1)	1.1 (−0.4 to 2.5)	0.3 (−1.1 to 1.7)	1.0 (−0.6 to 2.7)	1.4 (−0.6 to 3.4)
O_3	1.0 (0.2 to 1.9)	0.3 (−0.5 to 1.1)	0.2 (−0.5 to 1.0)	0.8 (0.0 to 1.6)	0.4 (−0.4 to 1.2)	0.9 (−0.1 to 1.9)	1.3 (0.1 to 2.6)
NO	−0.5 (−1.9 to 0.9)	−0.6 (−1.9 to 0.8)	−0.4 (−1.7 to 0.9)	0.0 (−1.4 to 1.3)	0.7 (−0.7 to 2.0)	−0.7 (−2.3 to 0.9)	−0.3 (−2.3 to 1.7)
Age 65+							
PM_{10}	1.5 (0.9 to 2.0)	1.1 (0.6 to 1.7)	1.1 (0.6 to 1.6)	1.2 (0.6 to 1.7)	0.7 (0.2 to 1.2)	1.5 (0.9 to 2.1)	1.9 (1.2 to 2.6)
SO_2	2.3 (0.5 to 4.2)	2.0 (0.2 to 3.9)	0.2 (−1.6 to 2.1)	1.8 (0.0 to 3.7)	0.2 (−1.6 to 2.0)	2.8 (0.7 to 5.0)	2.8 (0.1 to 5.5)
NO_2	1.7 (1.0 to 2.4)	1.3 (0.7 to 2.0)	1.2 (0.5 to 1.8)	1.1 (0.5 to 1.8)	0.9 (0.3 to 1.6)	1.8 (1.1 to 2.6)	2.3 (1.4 to 3.2)
O_3	1.7 (0.2 to 1.0)	0.6 (0.2 to 0.9)	0.4 (0.1 to 0.8)	0.8 (0.4 to 1.1)	0.4 (0.0 to 0.7)	0.8 (0.4 to 1.3)	1.4 (0.8 to 1.9)
NO	1.2 (0.6 to 1.8)	0.8 (0.2 to 1.4)	0.7 (0.1 to 1.3)	0.6 (0.0 to 1.1)	0.1 (−0.5 to 0.7)	1.3 (0.6 to 2.0)	1.4 (0.5 to 2.3)

[a] Definitions: CI indicates confidence interval; ER indicates excess risk.

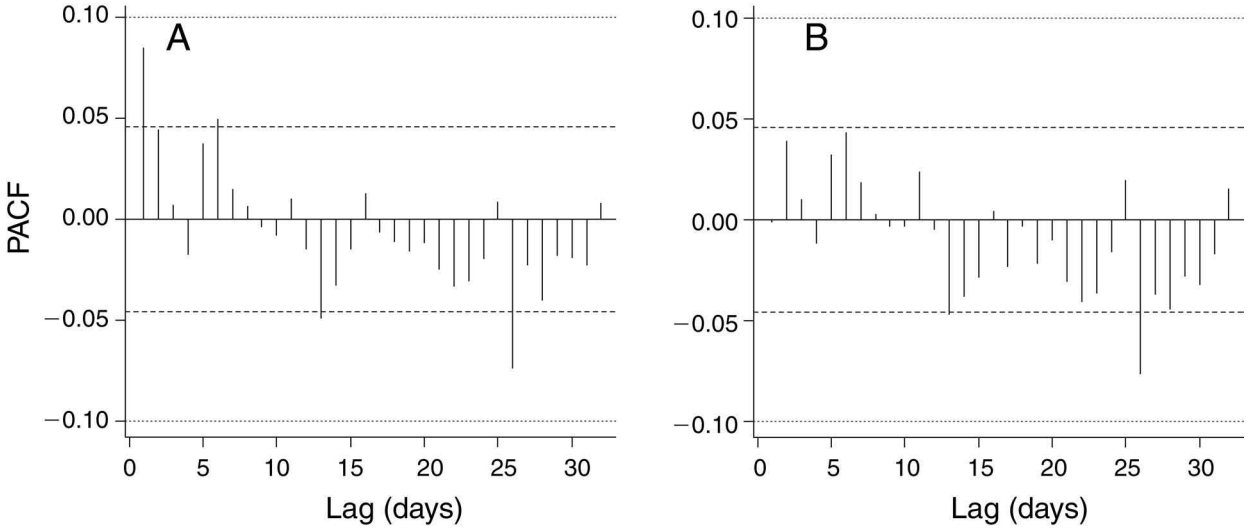


Figure 4. A: PACF plots of the model for all natural mortality with natural spline smoothing and 6 df for time per year of data; B: PACF plots of the AR1 for all natural mortality with natural spline smoothing and 6 df for time per year of data.

a particularly elevated risk. Among the subgroups of respiratory mortality, we observed elevated ERs in association with PM_{10} for young children (especially mortality among infants from respiratory causes) and for people with asthma. Some of these estimates had very wide CIs, probably because of the small number of mortality counts for these outcomes. Associations were also found with the "control" outcome of mortality due to non-cardiopulmonary causes (shown as "Others" in Table 7a).

As shown in Table 2, the average deaths per day in Bangkok from cardiovascular causes were low relative to what are usually observed in Western cities (Anderson et al. 2004) and in other cities in the PAPA project. This may have been a result of misdiagnosis, because deaths occurring outside the hospitals were diagnosed by nonphysician coroners, and deaths attributed to senility in reports may have actually been due to cardiovascular causes. We assessed this possibility, which was based on anecdotal information, by examining the ER of mortality from senility. We observed strong effects of PM_{10} on the risk of death from senility, with an ER of 1.8% (CI, 0.7 to 2.8) (Table 7a). These findings are consistent with a previous report by Vajanapoom and associates (2002) and suggest that mortality from cardiovascular diseases might have been systematically misdiagnosed as being due to senility, leading to an erroneously small percentage of daily mortality being attributed to cardiovascular causes in Bangkok.

In general, the results of age-specific all natural mortality counts showed that the effects of PM_{10} increased with age, with the strongest effects among those 75 years and older (Table 7a). However, associations were observed for all of the other age groups, including, as indicated earlier, for respiratory mortality for children less than 1 year of age. Our analysis by sex demonstrated similar effects for males and females.

Relatively similar results regarding associations with PM_{10} were also observed for several of the other pollutants, including SO_2, NO_2, O_3, and NO, with a few exceptions. For example, fewer associations were observed for SO_2 than for the other pollutants, and it was not associated with cardiopulmonary or respiratory mortality, asthma, or stroke. Regarding NO_2 and NO, no association was observed for respiratory mortality, LRI, or asthma. However, unlike with any of the other pollutants, exposure to NO was associated with mortality from ischemic heart disease. Finally, both NO_2 and NO demonstrated larger effects for cardiovascular mortality than all natural and respiratory mortality, while O_3 had larger effects on respiratory mortality than cardiovascular and all natural mortality.

Table 7b allows comparison between the effects of air pollutants on the ER of daily mortality per IQR increase in pollutant concentration. For most of the outcomes, PM_{10} and NO_2 demonstrated the highest ERs for the IQR. However, for stroke and all respiratory and LRI deaths, O_3 had the highest ER.

Table 8 shows the effects of different lags of PM_{10} and gaseous pollutants on several mortality outcomes. For PM_{10} and mortality due to all natural causes, lag 0 day generated the highest single-day effect estimate compared with other single-day lags. Estimated effects increased in size when multiday average lags were used. Generally, similar results were observed for the other pollutants. For

Table 9. Sensitivity Analyses: Natural Spline Models for Major Causes of Death with Different Model Specifications[a]

Models	PM_{10} % ER (95% CI)	SO_2 % ER (95% CI)	NO_2 % ER (95% CI)	O_3 % ER (95% CI)	NO % ER (95% CI)
All Natural					
ns	1.3 (0.8 to 1.7)	1.6 (0.1 to 3.2)	1.4 (0.9 to 1.9)	0.6 (0.3 to 0.9)	1.1 (0.6 to 1.6)
ps	1.2 (0.8 to 1.7)	1.5 (0.0 to 3.0)	1.3 (0.8 to 1.8)	0.6 (0.3 to 0.9)	1.0 (0.5 to 1.5)
AR1	1.2 (0.8 to 1.7)	1.5 (0.0 to 3.1)	1.4 (0.9 to 1.9)	0.6 (0.3 to 0.9)	1.0 (0.5 to 1.5)
Respiratory epidemic adjustment	1.2 (0.8 to 1.7)	1.7 (0.2 to 3.3)	1.4 (0.9 to 2.0)	0.6 (0.3 to 1.0)	1.1 (0.6 to 1.6)
Centering values of pollutant	1.2 (0.8 to 1.6)	1.1 (−0.7 to 2.9)	1.6 (1.0 to 2.2)	0.7 (0.4 to 1.1)	1.1 (0.6 to 1.7)
Cardiovascular					
ns	1.9 (0.8 to 3.0)	0.8 (−3.0 to 4.7)	1.8 (0.5 to 3.1)	0.8 (0.0 to 1.6)	2.0 (0.7 to 3.2)
ps	1.8 (0.8 to 2.8)	1.8 (−1.7 to 5.5)	1.2 (0.1 to 2.4)	0.7 (0.0 to 1.4)	2.0 (0.8 to 3.1)
AR1	1.9 (0.8 to 3.0)	0.4 (−3.3 to 4.3)	1.7 (0.4 to 3.0)	0.8 (0.1 to 1.7)	1.9 (0.7 to 3.2)
Respiratory epidemic adjustment	1.9 (0.8 to 3.0)	0.8 (−3.0 to 4.7)	1.8 (0.5 to 3.1)	0.8 (0.0 to 1.6)	2.0 (0.8 to 3.3)
Centering values of pollutant	1.8 (0.8 to 2.9)	−0.7 (−5.0 to 3.8)	1.6 (0.2 to 3.0)	0.8 (0.0 to 1.6)	2.1 (0.7 to 3.4)
Respiratory					
ns	1.0 (−0.4 to 2.4)	1.7 (−3.1 to 6.6)	1.0 (−0.6 to 2.7)	0.9 (−0.1 to 1.9)	−0.7 (−2.3 to 0.9)
ps	0.8 (−0.5 to 2.1)	1.1 (−3.4 to 5.9)	0.7 (−0.8 to 2.3)	0.9 (−0.1 to 1.9)	−0.8 (−2.3 to 0.7)
AR1	0.8 (−0.7 to 2.2)	0.5 (−4.4 to 5.6)	0.7 (−1.0 to 2.4)	0.7 (−0.4 to 1.7)	−0.7 (−2.3 to 0.9)
Respiratory epidemic adjustment	0.7 (−0.6 to 2.1)	2.8 (−1.9 to 7.7)	1.1 (−0.5 to 2.7)	1.0 (0.1 to 2.0)	−0.4 (−1.9 to 1.2)
Centering values of pollutant	0.7 (−0.6 to 2.1)	0.2 (−5.2 to 5.8)	1.8 (0.0 to 3.6)	1.0 (−0.1 to 2.0)	−0.7 (−2.4 to 1.0)
Age 65+[b]					
ns	1.5 (0.9 to 2.1)	2.8 (0.7 to 5.0)	1.8 (1.1 to 2.6)	0.8 (0.4 to 1.3)	1.3 (0.6 to 2.0)
ps	1.5 (1.0 to 2.1)	2.7 (0.6 to 4.9)	1.8 (1.1 to 2.5)	0.8 (0.4 to 1.3)	1.4 (0.7 to 2.0)
AR1	1.5 (0.9 to 2.1)	2.8 (0.6 to 4.9)	1.8 (1.1 to 2.5)	0.8 (0.4 to 1.3)	1.2 (0.5 to 1.9)
Respiratory epidemic adjustment	1.5 (0.9 to 2.1)	2.9 (0.8 to 5.1)	1.8 (1.1 to 2.6)	0.9 (0.4 to 1.3)	1.3 (0.6 to 2.0)
Centering values of pollutant	1.4 (0.9 to 2.0)	1.8 (−0.6 to 4.3)	1.9 (1.1 to 2.7)	1.0 (0.5 to 1.5)	1.2 (0.4 to 1.9)

[a] Definitions: AR1 indicates model with first-order autoregressive term; ER indicates excess risk; ns indicates natural spline; ps indicates penalized spline.

[b] For all natural mortality.

cardiovascular mortality and PM_{10}, lag 1 day showed the strongest single-day effect compared with lags of other lengths, and the cumulative averages over 5 days again demonstrated the strongest overall effects for most of the pollutants. For respiratory mortality, a slightly different pattern emerged. For PM_{10}, a 3-day lag gave the strongest single-day effect, while a 5-day moving average generated the largest overall effect estimate. None of the other pollutant-lag combinations were associated with respiratory mortality, with the exception of O_3, which showed associations at lag 0 day and lag 3 days and at the 5-day moving average. The results of lags in other mortality categories (sex and age) are shown in Tables D.1 and D.2 in Appendix D.

SENSITIVITY ANALYSES

Tables 9 to 11 present the results of the sensitivity analyses for the major mortality groups including all natural, cardiovascular, and respiratory mortality. Specifically, we examined the impacts of (1) alternative model specifications and assumptions; (2) copollutant models: (3) different amounts of smoothing (degrees of freedom) for time; and

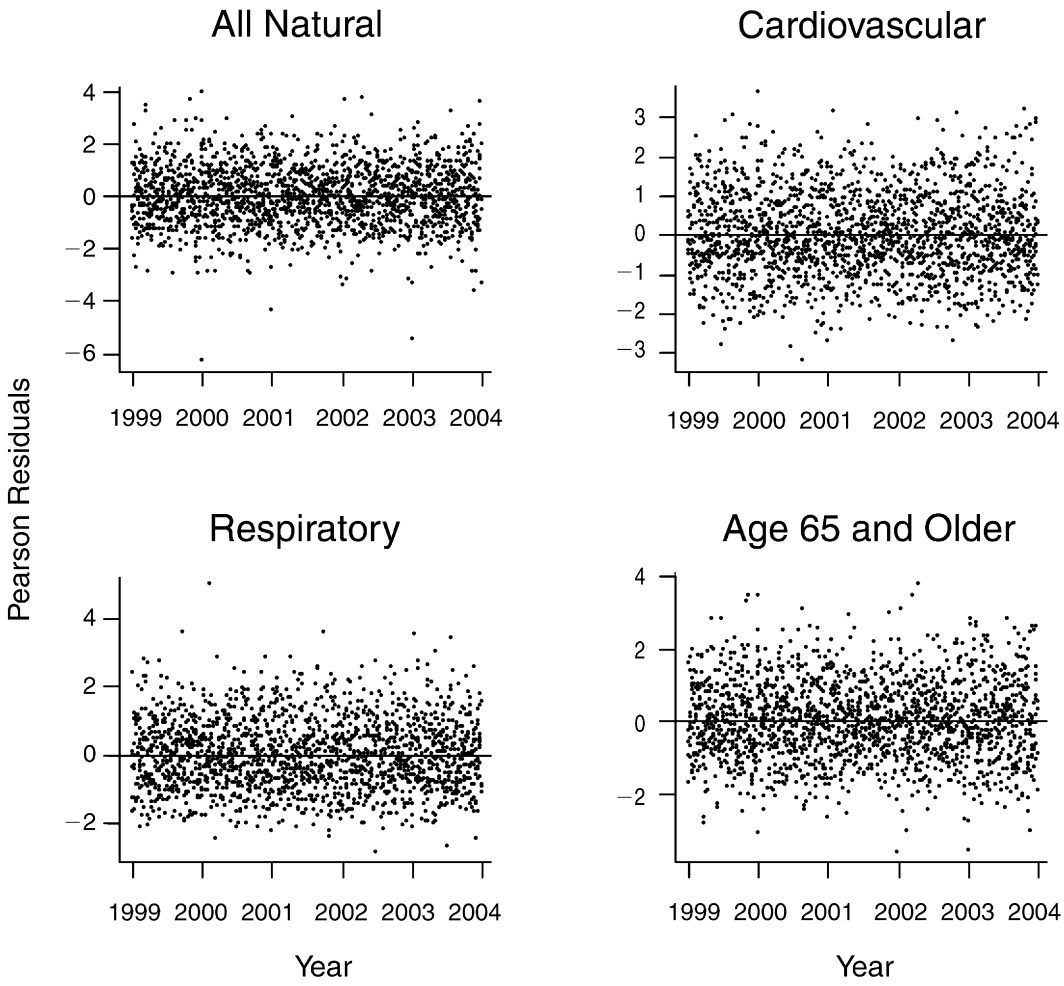


Figure 5. Residual plots of core models for major outcomes.

(4) different lags for temperature and humidity. Recall that our core model used natural spline smoothing with 6 df for time per year, and 3 df for temperature and RH for the entire study period, both with lag 0 day, along with dummy variables for days of the week and public holidays.

We assessed the impacts of various model functions and specifications (Table 9) including (1) a penalized spline model with the same degrees of freedom for time and weather as in the natural spline model; (2) models with an AR1; (3) models with an adjustment for influenza; and (4) models using centered values of air pollutants. To develop centered values, we used the same process as that reported by Wong and coworkers (2001). For each pollutant, the average was developed using the following method: (1) calculation of the mean value for each monitor across the study period; (2) subtraction of each monitor's mean concentration from the daily values available for that monitor (i.e., centering the data); (3) calculation of the daily mean of the available centered data across all monitors; and (4) for each day, addition of the grand mean (the mean of all unadjusted daily values of all of the monitors) back into the calculation.

Table 12 presents the results for gaseous pollutants with PM_{10} included in the model. A lag 0–1 day was used for all pollutants. Not surprisingly, because of the high degree of correlation among the pollutants, many of the effects of gases were attenuated and often became insignificant after adjusting for PM_{10}. Of note, NO_2 was no longer associated with any of the outcomes examined, except senility (which, as discussed earlier, may have been a misdiagnosis of cardiovascular disease). O_3 remained associated with stroke and senility, and with those older than age 75, while NO remained associated with ischemic heart disease.

The results for PM_{10} with gaseous pollutants included in the model are shown in Table 13. In this analysis, several of the earlier associations remained, including effects on

Table 10. Sensitivity Analyses: Natural Spline Models for Major Causes of Death with Different Degrees of Freedom for Time[a]

Degrees of Freedom	PM_{10} % ER (95% CI)	SO_2 % ER (95% CI)	NO_2 % ER (95% CI)	O_3 % ER (95% CI)	NO % ER (95% CI)
All Natural					
3	1.3 (0.9 to 1.8)	2.1 (0.6 to 3.6)	1.3 (0.8 to 1.8)	0.6 (0.3 to 0.9)	1.2 (0.7 to 1.7)
4	1.2 (0.8 to 1.7)	1.4 (−0.1 to 2.9)	1.2 (0.7 to 1.7)	0.6 (0.3 to 0.9)	1.1 (0.6 to 1.6)
6	1.3 (0.8 to 1.7)	1.6 (0.1 to 3.2)	1.4 (0.9 to 1.9)	0.6 (0.3 to 0.9)	1.1 (0.6 to 1.6)
9	1.1 (0.7 to 1.6)	2.2 (0.6 to 3.8)	1.3 (0.7 to 1.8)	0.6 (0.3 to 0.9)	1.0 (0.5 to 1.5)
12	1.1 (0.6 to 1.5)	1.8 (0.2 to 3.4)	1.2 (0.6 to 1.7)	0.6 (0.2 to 0.9)	0.9 (0.4 to 1.4)
15	1.2 (0.7 to 1.6)	1.5 (−0.2 to 3.2)	1.3 (0.7 to 1.8)	0.5 (0.2 to 0.9)	0.9 (0.4 to 1.5)
Cardiovascular					
3	2.0 (0.9 to 3.1)	1.6 (−2.1 to 5.4)	1.4 (0.2 to 2.7)	0.7 (0.0 to 1.5)	2.1 (0.9 to 3.3)
4	1.9 (0.8 to 3.0)	0.8 (−2.8 to 4.7)	1.4 (0.2 to 2.7)	0.8 (0.0 to 1.5)	2.1 (0.9 to 3.3)
6	1.9 (0.8 to 3.0)	0.8 (−3.0 to 4.7)	1.8 (0.5 to 3.1)	0.8 (0.0 to 1.7)	2.0 (0.7 to 3.2)
9	1.7 (0.6 to 2.8)	1.4 (−2.5 to 5.5)	1.7 (0.3 to 3.0)	0.7 (0.0 to 1.5)	2.0 (0.7 to 3.2)
12	1.8 (0.7 to 3.0)	0.8 (−3.1 to 4.8)	1.5 (0.2 to 2.9)	0.8 (0.0 to 1.6)	1.7 (0.4 to 3.0)
15	2.2 (0.9 to 3.4)	1.2 (−2.9 to 5.5)	2.0 (0.6 to 3.5)	1.0 (0.2 to 1.8)	1.9 (0.6 to 3.2)
Respiratory					
3	1.1 (−0.3 to 2.4)	2.5 (−2.2 to 7.3)	1.2 (−0.3 to 2.8)	0.9 (−0.1 to 1.9)	−0.4 (−1.9 to 1.2)
4	1.0 (−0.4 to 2.4)	1.3 (−3.3 to 6.2)	0.9 (−0.6 to 2.6)	0.9 (−0.1 to 1.9)	−0.4 (−1.9 to 1.2)
6	1.0 (−0.4 to 2.4)	1.7 (−3.1 to 6.6)	1.0 (−0.6 to 2.7)	0.9 (−0.1 to 1.9)	−0.7 (−2.3 to 0.9)
9	0.7 (−0.7 to 2.1)	2.1 (−2.8 to 7.2)	0.7 (−1.0 to 2.4)	0.8 (−0.2 to 1.8)	−0.9 (−2.5 to 0.7)
12	0.3 (−1.1 to 1.8)	1.6 (−3.3 to 6.8)	0.5 (−1.2 to 2.2)	0.7 (−0.3 to 1.7)	−1.3 (−2.8 to 0.3)
15	0.3 (−1.1 to 1.9)	1.1 (−4.1 to 6.6)	0.6 (−1.1 to 2.4)	0.7 (−0.4 to 1.7)	−1.3 (−2.9 to 0.3)
Age 65+[b]					
3	1.6 (1.1 to 2.2)	3.4 (1.3 to 5.5)	1.5 (0.8 to 2.3)	0.7 (0.3 to 1.2)	1.5 (0.8 to 2.1)
4	1.5 (0.9 to 2.1)	2.6 (0.5 to 4.7)	1.4 (0.7 to 2.1)	0.8 (0.3 to 1.2)	1.3 (0.6 to 2.0)
6	1.5 (0.9 to 2.1)	2.8 (0.7 to 5.0)	1.8 (1.1 to 2.6)	0.8 (0.4 to 1.3)	1.3 (0.6 to 2.0)
9	1.4 (0.8 to 2.0)	3.9 (1.7 to 6.1)	1.7 (0.9 to 2.4)	0.9 (0.4 to 1.3)	1.2 (0.5 to 1.9)
12	1.4 (0.8 to 2.0)	3.2 (1.1 to 5.5)	1.6 (0.8 to 2.3)	0.8 (0.4 to 1.3)	1.1 (0.4 to 1.8)
15	1.5 (0.8 to 2.1)	3.0 (0.8 to 5.4)	1.7 (0.9 to 2.5)	0.8 (0.4 to 1.3)	1.1 (0.4 to 1.8)

[a] Definitions: CI indicates confidence interval; ER indicates excess risk.

[b] For all natural mortality.

nonaccidental cardiopulmonary and asthma deaths, mortality among those older than age 65, and mortality among males and females. In addition, the effect estimates remained relatively similar to those of the single-pollutant model for PM_{10}.

Table 10 summarizes the results (for single-pollutant models) using different degrees of freedom in the specification of time in natural spline models. Specifically, we assessed the results of the models with 3 to 15 df for time per year, while keeping 3 df for temperature and RH for the entire study period. With the exception of respiratory mortality, the results for PM_{10}, SO_2, NO_2, O_3, and NO were robust to the degrees of freedom used in the models, as the ER was generally similar. For respiratory mortality, increases in the degrees of freedom to 12 or more per year significantly attenuated the effect estimate, except for O_3. Respiratory mortality effect estimates for this pollutant were robust to the degrees of freedom used to control for time.

Next, we examined the results of the models with alternative lags for temperature and humidity, including single-day lags of 0 to 3 days and cumulative lags of 1 to 2 and 3 to 7 days. As shown in Table 11, we observed that for respiratory mortality, the air pollutant effects were robust

Table 11. Sensitivity Analyses Using Natural Spline Models for Major Causes of Death with Different Lags of Temperature and Relative Humidity[a]

Temperature and Humidity Lag	PM_{10} % ER (95% CI)	SO_2 % ER (95% CI)	NO_2 % ER (95% CI)	O_3 % ER (95% CI)	NO % ER (95% CI)
All Natural					
Lag 0	1.3 (0.8 to 1.7)	1.6 (0.1 to 3.2)	1.4 (0.9 to 1.9)	0.6 (0.3 to 0.9)	1.1 (0.6 to 1.6)
Lag 1	1.3 (0.8 to 1.7)	1.9 (0.4 to 3.5)	1.2 (0.6 to 1.7)	0.7 (0.4 to 1.0)	1.1 (0.6 to 1.6)
Lag 2	1.2 (0.7 to 1.7)	1.9 (0.4 to 3.5)	0.7 (0.2 to 1.3)	0.8 (0.4 to 1.1)	0.7 (0.1 to 1.2)
Lag 3	1.0 (0.5 to 1.4)	1.6 (0.1 to 3.2)	0.4 (−0.2 to 0.9)	0.7 (0.4 to 1.0)	0.5 (0.0 to 1.0)
1–2 (mean)	1.3 (0.8 to 1.7)	2.1 (0.5 to 3.6)	1.0 (0.5 to 1.6)	0.7 (0.4 to 1.1)	0.9 (0.4 to 1.5)
3–7 (mean)	0.8 (0.3 to 1.3)	1.7 (0.2 to 3.3)	0.3 (−0.3 to 0.8)	0.7 (0.4 to 1.0)	0.4 (−0.1 to 0.9)
Cardiovascular					
Lag 0	1.9 (0.8 to 3.0)	0.8 (−3.0 to 4.7)	1.8 (0.5 to 3.1)	0.8 (0.0 to 1.6)	2.0 (0.7 to 3.2)
Lag 1	1.9 (0.8 to 3.0)	1.2 (−2.5 to 5.1)	1.8 (0.4 to 3.1)	0.8 (0.0 to 1.6)	2.1 (0.8 to 3.4)
Lag 2	1.7 (0.5 to 2.9)	0.9 (−2.9 to 4.7)	1.2 (−0.1 to 2.6)	0.6 (−0.2 to 1.4)	1.8 (0.6 to 3.1)
Lag 3	1.4 (0.3 to 2.6)	0.5 (−3.2 to 4.3)	1.0 (−0.3 to 2.3)	0.5 (−0.2 to 1.3)	1.7 (0.5 to 2.9)
1–2 (mean)	1.8 (0.6 to 3.0)	1.2 (−2.5 to 5.1)	1.5 (0.1 to 2.9)	0.7 (−0.1 to 1.5)	1.9 (0.7 to 3.2)
3–7 (mean)	1.2 (0.0 to 2.3)	0.4 (−3.3 to 4.2)	0.8 (−0.5 to 2.1)	0.4 (−0.3 to 1.2)	1.5 (0.3 to 2.8)
Respiratory					
Lag 0	1.0 (−0.4 to 2.4)	1.7 (−3.1 to 6.6)	1.0 (−0.6 to 2.7)	0.9 (−0.1 to 1.9)	−0.7 (−2.3 to 0.9)
Lag 1	1.0 (−0.5 to 2.4)	2.1 (−2.7 to 7.0)	0.8 (−0.9 to 2.5)	0.9 (−0.1 to 1.9)	−0.4 (−2.0 to 1.2)
Lag 2	1.5 (0.1 to 3.0)	2.7 (−2.0 to 7.6)	1.0 (−0.7 to 2.7)	1.3 (0.3 to 2.3)	−0.8 (−2.4 to 0.7)
Lag 3	1.3 (−0.1 to 2.8)	2.5 (−2.2 to 7.4)	0.5 (−1.1 to 2.2)	1.3 (0.4 to 2.3)	−1.2 (−2.7 to 0.4)
1–2 (mean)	1.3 (−0.1 to 2.8)	2.5 (−2.3 to 7.4)	1.1 (−0.6 to 2.8)	1.1 (0.1 to 2.1)	−0.4 (−2.0 to 1.2)
3–7 (mean)	1.1 (−0.4 to 2.5)	2.5 (−2.2 to 7.5)	0.3 (−1.3 to 2.0)	1.2 (0.3 to 2.2)	−1.0 (−2.5 to 0.5)
Age 65+[b]					
Lag 0	1.5 (0.9 to 2.1)	2.8 (0.7 to 5.0)	1.8 (1.1 to 2.6)	0.8 (0.4 to 1.3)	1.3 (0.6 to 2.0)
Lag 1	1.6 (0.9 to 2.2)	3.1 (1.0 to 5.3)	1.6 (0.8 to 2.3)	1.0 (0.5 to 1.4)	1.4 (0.7 to 2.1)
Lag 2	1.5 (0.8 to 2.1)	3.0 (0.9 to 5.2)	0.9 (0.2 to 1.7)	1.0 (0.6 to 1.5)	0.7 (0.0 to 1.5)
Lag 3	1.2 (0.5 to 1.8)	2.7 (0.6 to 4.9)	0.5 (−0.2 to 1.3)	0.9 (0.5 to 1.4)	0.6 (−0.1 to 1.3)
1–2 (mean)	1.6 (0.9 to 2.2)	3.3 (1.2 to 5.4)	1.4 (0.6 to 2.2)	1.0 (0.6 to 1.5)	1.2 (0.4 to 1.9)
3–7 (mean)	0.9 (0.3 to 1.6)	2.8 (0.7 to 5.0)	0.3 (−0.4 to 1.0)	0.9 (0.5 to 1.4)	0.3 (−0.4 to 1.0)

[a] Definitions: CI indicates confidence interval; ER indicates excess risk.

[b] For all natural mortality.

to the use of different lags of temperature and RH. In general, the results of the models for the all natural and cardiovascular categories were robust to the choices of lag days used for temperature and RH. However, for NO_2 for all natural and cardiovascular cases, the cumulative lag temperature of 3 to 7 days resulted in an attenuation of the pollution effect. Note, however, that overall, a temperature and humidity smoothing term for lag 0 day provided the best model fit, based on the percentage of the explained deviation or the Akaike information criterion.

Finally, for interested parties, a summary of technical details pertaining to the study is presented in Appendix A, including such information as the software package and specific version used and the explicit codes employed in the main statistical model. Other technical details of the main regression model — degrees of freedom per year used for the smooth function of time, the value of the overdispersion parameter, and whether the deviance parameter was scaled or unscaled — are also included in Appendix A.

The results indicate that most of the estimated ERs for all natural, cardiovascular, and respiratory mortality did not change. The results of the sensitivity analyses indicate that our core model was generally robust to choices of model specifications, spline model, degrees of freedom of

Table 12. Percentage of Excess Risk in Mortality and Confidence Interval for a 10-μg/m^3 Increase in Gases with Lag 0–1 with PM_{10} Adjustment[a]

Mortality Class	SO_2 % ER (95% CI)	NO_2 % ER (95% CI)	O_3 % ER (95% CI)	NO % ER (95% CI)
Cause-Specific, All Ages				
All natural	0.1 (−1.5 to 1.7)	0.4 (−0.5 to 1.4)	0.2 (−0.2 to 0.5)	0.4 (−0.2 to 1.0)
Cardiopulmonary	−1.0 (−4.2 to 2.3)	−0.2 (−2.1 to 1.7)	0.3 (−0.5 to 1.1)	0.1 (−1.1 to 1.2)
Cardiovascular	−2.0 (−6.0 to 2.1)	−0.5 (−2.9 to 1.9)	0.1 (−0.9 to 1.0)	1.1 (−0.3 to 2.6)
Ischemic heart disease	−0.8 (−7.9 to 6.8)	−0.4 (−4.5 to 3.9)	−0.9 (−2.5 to 0.8)	2.6 (0.0 to 5.2)
Stroke	−2.6 (−8.9 to 4.1)	1.6 (−2.2 to 5.5)	1.9 (0.4 to 3.5)	0.6 (−1.6 to 3.0)
Conduction disorders	−4.4 (−24.0 to 20.4)	6.3 (−6.1 to 20.3)	−5.0 (−9.7 to 0.0)	6.0 (−1.9 to 14.5)
Respiratory	0.7 (−4.4 to 6.0)	0.3 (−2.7 to 3.4)	0.7 (−0.5 to 1.9)	−1.8 (−3.6 to 0.0)
Respiratory ≤ 1 yr	98.8 (26.0 to 213.8)	−6.5 (−25.5 to 17.3)	−2.8 (−11.2 to 6.5)	−4.8 (−16.8 to 9.0)
LRI	1.5 (−5.6 to 9.1)	1.9 (−1.7 to 5.7)	1.4 (−0.2 to 3.1)	−0.3 (−2.8 to 2.2)
LRI < 5 yr	6.3 (−31.0 to 63.8)	4.1 (−17.2 to 31.0)	2.8 (−6.4 to 12.9)	4.0 (−9.8 to 19.9)
COPD	2.7 (−7.9 to 14.6)	−7.9 (−14.0 to −1.3)	−1.9 (−4.6 to 0.8)	−3.2 (−7.1 to 0.9)
Asthma	−12.1 (−30.7 to 11.5)	−11.0 (−22.5 to 2.3)	−2.6 (−7.8 to 2.9)	−9.5 (−16.8 to −1.7)
Senility	1.5 (−2.3 to 5.5)	3.0 (0.6 to 5.5)	1.2 (0.3 to 2.2)	1.1 (−0.3 to 2.6)
Others[b]	0.4 (−1.4 to 2.2)	0.6 (−0.5 to 1.7)	0.1 (−0.3 to 0.5)	0.5 (−0.2 to 1.1)
Accidental	−0.2 (−8.7 to 9.2)	−0.8 (−6.0 to 4.8)	−0.1 (−2.2 to 2.1)	0.9 (−2.3 to 4.2)
Age-Specific (All Natural) (yr)				
0–4	3.4 (−4.5 to 12.0)	−2.1 (−6.7 to 2.8)	−1.3 (−3.1 to 0.6)	1.1 (−1.8 to 4.2)
5–44	−1.0 (−3.7 to 1.8)	−0.1 (−1.7 to 1.6)	0.0 (−0.6 to 0.7)	0.0 (−1.0 to 1.0)
18–50	−0.6 (−3.0 to 2.0)	0.5 (−1.0 to 2.0)	0.2 (−0.4 to 0.8)	−0.1 (−1.0 to 0.9)
45–64	−0.6 (−3.4 to 2.2)	0.5 (−1.2 to 2.1)	0.5 (−0.1 to 1.2)	0.1 (−0.9 to 1.1)
50+	0.7 (−1.2 to 2.6)	0.8 (−0.3 to 2.0)	0.4 (−0.1 to 0.8)	0.5 (−0.2 to 1.2)
65+	1.1 (−1.1 to 3.4)	0.9 (−0.4 to 2.3)	0.3 (−0.2 to 0.8)	0.5 (−0.3 to 1.3)
75+	2.4 (−0.8 to 5.6)	2.5 (0.6 to 4.5)	0.9 (0.1 to 1.6)	0.4 (−0.7 to 1.6)
Sex-Specific (All Natural)				
Male	−0.7 (−2.6 to 1.3)	0.0 (−1.1 to 1.2)	0.2 (−0.3 to 0.6)	0.1 (−0.6 to 0.8)
Female	1.1 (−1.1 to 3.5)	1.0 (−0.4 to 2.4)	0.3 (−0.3 to 0.8)	0.6 (−0.3 to 1.4)

[a] Definitions: CI indicates confidence interval; COPD indicates chronic obstructive pulmonary disease; ER indicates excess risk; LRI indicates lower respiratory infection.

[b] Non-cardiopulmonary and natural.

time-smoothing functions, lags for temperature, adjustment for autocorrelation, adjustment for influenza epidemics, and adjustment for missing values using centered data.

In our final analysis of the effects of pollution on mortality, we examined the shape of the concentration–response function. All of our previous analyses (and those of most other previous studies) assumed linear models of exposure to PM_{10}, SO_2, NO_2, O_3, and NO. We investigated the possibility of nonlinear functions given the relatively high concentrations of pollution observed in the PAPA cities. This is important since it is likely that at some high concentrations, the concentration–response function will tend to level off. To assess this question, we examined the smoothed relation between pollution and mortality, after controlling for other factors in the analysis. Again, we used a lag 0–1 day for pollutants. Residuals of mortality — variations of daily mortality obtained after adjusting for time and weather — were created after fitting our core model, omitting the pollution term. As shown in Figure 6, the concentration–response relation between each air pollutant and all natural mortality appears to be fairly linear, except for NO_2, which appears to show an increasing effect at higher concentrations, based on a limited number of observations at the higher levels. We conducted a specific test for linearity (see Appendix A), which was rejected only for NO_2. The findings indicated that our models were reasonably appropriate for assessing the effects of air pollutants on mortality.

Table 13. Percentage of Excess Risk in Mortality and Confidence Interval for a 10-μg/m^3 Increase in PM_{10} with Lag 0–1 with Gaseous Adjustment[a]

Mortality Class	PM_{10} % ER (95% CI)	With SO_2 % ER (95% CI)	With NO_2 % ER (95% CI)	With O_3 % ER (95% CI)	With NO % ER (95% CI)
Cause-Specific, All Ages					
All natural	1.3 (0.8 to 1.7)	1.2 (0.8 to 1.7)	1.0 (0.2 to 1.8)	1.1 (0.6 to 1.7)	1.1 (0.6 to 1.6)
Cardiopulmonary	1.6 (0.7 to 2.5)	1.7 (0.7 to 2.6)	1.7 (0.1 to 3.3)	1.3 (0.3 to 2.4)	1.5 (0.5 to 2.6)
Cardiovascular	1.9 (0.8 to 3.0)	2.1 (0.9 to 3.3)	2.3 (0.2 to 4.3)	1.8 (0.5 to 3.2)	1.4 (0.1 to 2.7)
Ischemic heart disease	1.5 (−0.4 to 3.5)	1.6 (−0.5 to 3.7)	1.8 (−1.8 to 5.4)	2.2 (−0.2 to 4.6)	0.3 (−2.0 to 2.6)
Stroke	2.3 (0.6 to 4.0)	2.6 (0.7 to 4.5)	1.2 (−2.0 to 4.4)	0.8 (−1.3 to 2.9)	2.0 (−0.1 to 4.1)
Conduction disorders	−0.3 (−5.9 to 5.6)	0.2 (−5.9 to 6.7)	−4.5 (−13.9 to 6.0)	3.8 (−3.2 to 11.2)	−2.9 (−9.3 to 3.9)
Respiratory	1.0 (−0.4 to 2.4)	0.9 (−0.5 to 2.4)	0.8 (−1.7 to 3.3)	0.5 (−1.2 to 2.2)	1.9 (0.2 to 3.5)
Respiratory ≤1 yr	14.6 (2.9 to 27.6)	4.0 (−8.2 to 17.8)	20.4 (−1.3 to 46.9)	17.3 (2.9 to 33.8)	17.2 (3.6 to 32.7)
LRI	0.5 (−1.4 to 2.5)	0.4 (−1.7 to 2.5)	−1.2 (−4.6 to 2.3)	−0.6 (−2.9 to 1.7)	0.7 (−1.5 to 3.0)
LRI < 5 yr	7.7 (−3.6 to 20.3)	7.0 (−5.1 to 20.6)	4.7 (−13.9 to 27.2)	5.4 (−7.7 to 20.3)	5.7 (−7.1 to 20.3)
COPD	1.3 (−1.8 to 4.4)	1.0 (−2.2 to 4.4)	7.2 (1.3 to 13.4)	2.8 (−0.9 to 6.7)	2.8 (−0.8 to 6.6)
Asthma	7.4 (1.1 to 14.1)	8.9 (2.0 to 16.3)	16.4 (3.9 to 30.3)	9.5 (1.9 to 17.8)	12.4 (4.8 to 20.6)
Senility	1.8 (0.7 to 2.8)	1.6 (0.5 to 2.7)	−0.3 (−2.2 to 1.7)	0.9 (−0.4 to 2.1)	1.2 (0.0 to 2.5)
Others[b]	1.2 (0.8 to 2.0)	1.1 (0.6 to 1.6)	0.8 (−0.1 to 1.6)	1.1 (0.5 to 1.7)	1.0 (0.4 to 1.5)
Accidental	0.1 (−2.3 to 2.6)	0.2 (−2.4 to 2.8)	0.7 (−3.7 to 5.3)	0.2 (−2.6 to 3.2)	−0.3 (−3.1 to 2.6)
Age-Specific (All Natural) (yr)					
0–4	0.2 (−2.0 to 2.4)	−0.1 (−2.4 to 2.2)	1.6 (−2.3 to 5.7)	1.1 (−1.4 to 3.8)	−0.3 (−2.8 to 2.2)
5–44	0.9 (0.2 to 1.7)	1.0 (0.3 to 1.8)	1.0 (−0.4 to 2.4)	0.9 (0.0 to 1.8)	0.9 (0.1 to 1.8)
18–50	1.2 (0.5 to 1.9)	1.2 (0.5 to 2.0)	0.8 (−0.4 to 2.1)	1.0 (0.2 to 1.9)	1.2 (0.4 to 2.0)
45–64	1.1 (0.4 to 1.9)	1.2 (0.4 to 2.0)	0.8 (−0.5 to 2.2)	0.8 (−0.1 to 1.6)	1.1 (0.2 to 2.0)
50+	1.4 (0.9 to 1.9)	1.4 (0.8 to 1.9)	0.9 (−0.1 to 1.8)	1.1 (0.5 to 1.8)	1.2 (0.6 to 1.8)
65+	1.5 (0.9 to 2.1)	1.4 (0.8 to 2.1)	0.9 (−0.2 to 2.0)	1.3 (0.6 to 2.0)	1.3 (0.6 to 2.0)
75+	2.2 (1.3 to 3.0)	1.9 (1.0 to 2.8)	0.4 (−1.1 to 2.0)	1.5 (0.5 to 2.5)	2.0 (1.0 to 3.0)
Sex-Specific (All Natural)					
Male	1.2 (0.7 to 1.7)	1.2 (0.7 to 1.8)	1.1 (0.2 to 2.1)	1.0 (0.4 to 1.7)	1.1 (0.5 to 1.8)
Female	1.3 (0.7 to 1.9)	1.2 (0.5 to 1.8)	0.6 (−0.5 to 1.7)	1.1 (0.3 to 1.8)	1.0 (0.3 to 1.8)

[a] Definitions: CI indicates confidence interval; COPD indicates chronic obstructive pulmonary disease; ER indicates excess risk; LRI indicates lower respiratory infection.

[b] Non-cardiopulmonary and natural.

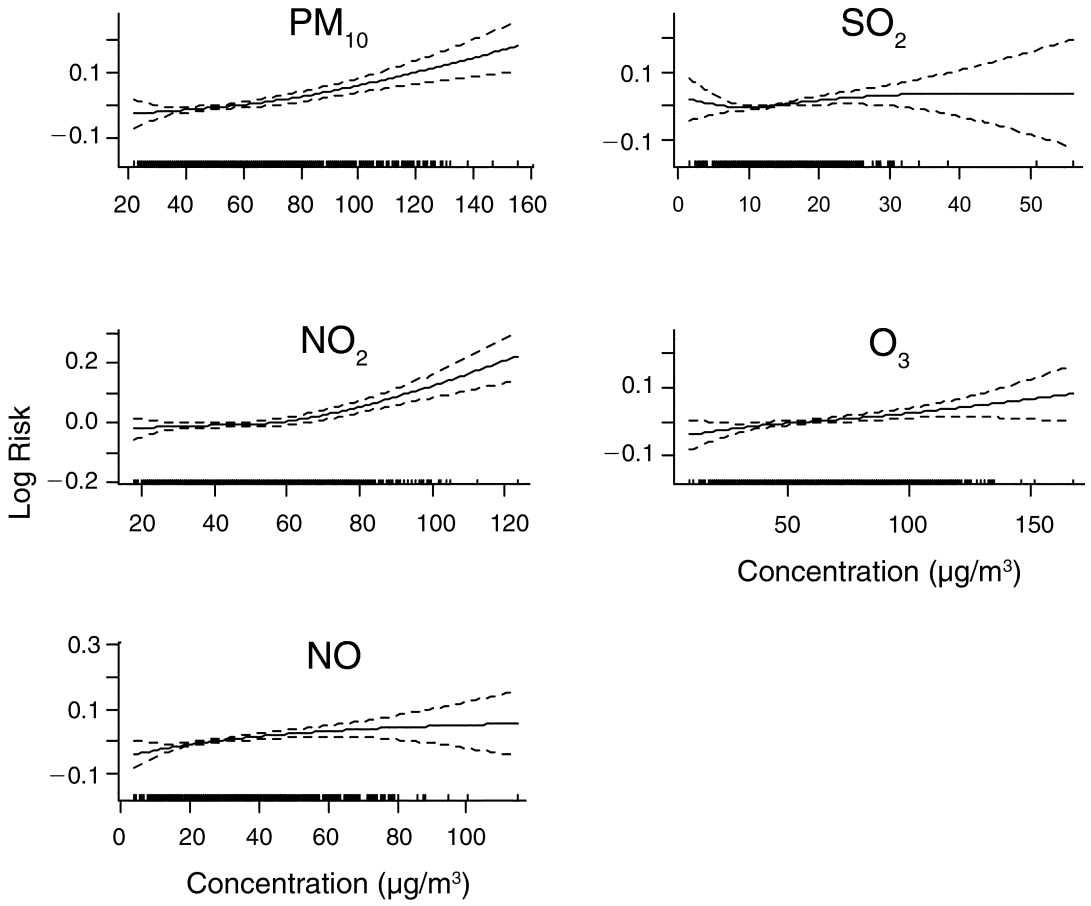


Figure 6. Curves showing the concentration–response relation between air pollutants and all natural mortality. Small bar graphs along the *x*-axes show areas of sparse data; there were many fewer days with high concentrations of air pollutants as shown by wide spacing between bars.

DISCUSSION

The results of our analysis of five years of data from Bangkok, Thailand, indicate a statistically significant association between daily mortality and daily concentrations of PM_{10}, NO_2, NO, and O_3. More modest associations were found with SO_2. For PM_{10}, the effect estimates for all natural mortality, cardiovascular-related and respiratory-related mortality, and mortality among those over age 65 years are generally similar to those found in other studies, although high in that range (U.S. EPA 2004). A 10-μg/m^3 change in PM_{10} was associated with an ER of death of 1.3%, 1.9%, 1.0%, and 1.5% in the categories of all natural, cardiovascular-related, and respiratory-related deaths, and among those over age 65, respectively (Table 7a). The CI for respiratory mortality included zero, however (indicating nonsignificant association). These estimates are generally similar to those reported by Ostro and colleagues (1998, 1999) and Vajanapoom and associates (2002) in previous studies in Bangkok covering earlier years. However, these older studies largely utilized PM_{10} data from airport visibility measurements rather than direct measurements of PM_{10}. Nevertheless, these previous results are supported by the current analysis, which used mortality and air pollution data that was subjected to significant quality assurance and quality control procedures. As such, they may rank among the highest quality and most complete mortality and pollution data from less-developed countries.

Our results also found some disease- and age-specific subgroups that exhibit ERs of death. ERs from PM_{10} were observed for many of the cardiovascular- and respiratory-disease subclasses of mortality, with particularly high risks related to respiratory diseases in those less than 1 year of age, LRI for those under 5 years, asthma, stroke, and senility, although many of the deaths attributed to senility may have actually been misclassified cardiovascular mortalities. Analysis by age indicated associations with PM_{10} for all of the subgroups with the exception of those under age 5. For that group, no associations were observed for all natural mortality, although, as indicated earlier, associations were

observed for that group for respiratory mortality. Our examination of lags indicated that among single-day lags, a zero- or one-day lag (lag 0 day or lag 1 day, respectively) was important, but that multiday averages of 5 days generated the largest effect estimates. We did not find associations with COPD, possibly due to low daily death counts (about 2 per day) or misclassification of diseases. It should be noted that the daily counts for some of the outcomes, such as asthma-related and infant-respiratory-related mortality, were low, so caution is suggested in interpreting these results.

Many of the PM_{10} associations with mortality were retained in multipollutant models. However, notably there was a high correlation between PM_{10} and several of the other pollutants, particularly NO_2 ($r = 0.71$). This makes it difficult to separate out the independent effects of PM_{10} from those of NO_2 and leads to an attenuation of the pollution effect when both of these pollutants are included in the same model. Associations between the other pollutants (SO_2, NO_2, NO, and O_3) and mortality were often observed in single-pollutant models. However, the effects were greatly attenuated and often insignificant when PM_{10} was also included in the model. Since air pollution in Bangkok is dominated by traffic, it is not surprising that PM_{10} would be highly correlated with traffic-based pollutants such as NO_2 and NO. Of particular note is the potential independent effect of O_3. For example, while most of the pollutants exhibited larger effects for cardiovascular mortality, O_3 had larger effects on respiratory mortality (Table 7a). In contrast, SO_2, NO_2, and NO were not associated with respiratory mortality. In addition, in models that included PM_{10}, O_3 remained associated with stroke and with deaths among those over the age of 75 (Table 12), and NO_2 was associated only with deaths among those over the age of 75, whereas those results were not observed for SO_2 or NO. These findings suggest a plausible independent effect of O_3.

We conducted extensive sensitivity analyses to investigate whether the results were robust to various assumptions. The results indicated that most of the estimated ERs for all natural, cardiovascular-related, and respiratory-related mortality were not sensitive to our core modeling assumptions. Specifically, the results of our core models were not significantly affected by the choice of model specifications, spline model (natural versus penalized), degrees of freedom of time smoothing (from 3 to 15 df), lags for temperature (zero- to 3-day), adjustment for autocorrelation, adjustment for epidemics, and adjustment for missing values using centered data. Of note, however, is that when longer cumulative lags of temperature were used (lags of 3 to 7 days), the effect estimates for PM_{10} were significantly reduced. However, the best overall model fit was observed for a lag 0 day for temperature. Therefore, it is most likely that the attenuation of the pollution effect with a longer cumulative lag in temperature (specifically, the mean of temperature from lag 3–7 days) was due to its correlation with lags in pollution (see Tables B.4–B.8 in Appendix B) coupled with the greater effect of cumulative averages (relative to single-day averages) of PM_{10} on mortality. Finally, our analysis showed a fairly linear concentration–response relation between each air pollutant (except for NO_2) and all natural mortality (Figure 6).

Generally, our analysis of short-term changes in daily mortality per 10-μg/m^3 increase in PM_{10} in Bangkok generated effect estimates that are higher than most previously reported. For example, our estimate for all natural mortality is 1.3% (95% CI, 0.8 to 1.7). As a comparison, an analysis of 75 single-city time-series analyses generated an estimate of 0.6% (95% CI, 0.5 to 0.7) (Anderson et al. 2005). A study of the 90 largest cities in the United States gave an estimate of 0.2% (95% CI, 0.1 to 0.4) (Dominici et al. 2003), while a study of 29 European cities provided an estimate of 0.6% (95% CI, 0.4 to 0.7) (Katsouyanni et al. 2003). A study of 14 cities in the United States using a case-crossover approach generated an estimate of 0.35% (95% CI, 0.2 to 0.5) (Schwartz 2004). A meta-analysis of Asian studies using a random-effects estimate gave an estimate of 0.49% (95% CI, 0.23 to 0.76) based on four cities: Bangkok, Seoul, Inchon, and Hong Kong (HEI International Scientific Oversight Committee 2004). Thus, it is clear that the results for Bangkok are at the upper end of the range of estimates. It is also of note that some high estimates have been reported in other developing countries. For example, a study in Mexico City reported an ER of 1.8% (95% CI, 0.9 to 2.7), while an analysis of Santiago, Chile, generated an ER of 1.1% (95% CI, 0.9 to 1.4) (Ostro et al. 1996; Castillejos et al. 2000).

We can speculate on several possible reasons for the finding of high effect estimates in Bangkok relative to most other cities, including (1) differences in particle chemistry in Bangkok; (2) the proximity of a large portion of the population to roads and traffic congestion; (3) the likely high penetration rates due to the low prevalence of air conditioning and the presence of open ventilation between indoors and outdoors; (4) the greater duration of exposure due to the amount of time spent outdoors; (5) factors related to lower economic development and socioeconomic status, such as background health status, use of health care, smoking rates, and comorbidity; and (6) stochastic variability. Several of these factors (although only anecdotal in nature) may explain why the effective exposure to any given concentration measured from a fixed-site outdoor monitor is also greater than that observed in the Western industrialized countries. Although there is limited empiric evidence about these factors, one study in Bangkok demonstrated high correlations

between outdoor and indoor PM_{10} and $PM_{2.5}$ (particulate matter $\leq$ 2.5 μm in aerodynamic diameter), particularly in homes with limited indoor sources (Tsai et al. 2000).

Most previous studies of air pollution and mortality have been conducted in cities in the United States and Western Europe — areas with relatively cold winters and strong seasonal patterns in daily mortality. Our replication of previous time-series studies showing associations between PM_{10} and mortality in a city with a very different seasonal pattern, and no cold season, is notable. Specifically, we have found an association between mortality and air pollution in a location where there is very little likelihood of confounding due to seasonality or epidemics related to cold weather.

For the most part, policy makers in Asia have had to draw from studies that have been conducted in North America and Western Europe. For example, the WHO effort to estimate the global burden of disease from outdoor air pollution relied primarily on studies conducted in the Western, industrialized nations (Cohen et al. 2005). Similarly, previous guidance from WHO (Ostro 2004) and the World Bank (Ostro 1994) for developing estimates of the health effects of air pollution in developing countries also focused largely on studies from the industrialized West. The current study suggests that it is reasonable to extrapolate the findings from the North American and Western European regions to other parts of the world. However, our study also suggests the possibility that the per-unit effects may be higher in certain developing countries. Additional studies undertaken in developing countries in Asia and other parts of the world may serve to validate our findings and help determine the factors that might modify the effect estimate.

To date, studies of daily mortality in Asia have been conducted in Inchon, Korea (Hong et al. 1999); Seoul and Ulsan, Korea (Lee et al. 1999; Lee and Schwartz 1999; Kwon et al. 2001); Shenyang, China (Xu et al. 2000); seven cities in South Korea (Lee et al. 2000); and New Delhi, India (Cropper et al. 1997). These studies generally report associations consistent with those found in studies undertaken in Western, industrialized nations. To further examine this issue, HEI has provided support for an analysis of three Chinese cities as part of the PAPA project — Hong Kong, Shanghai, and Wuhan — in addition to Bangkok.

Given the relatively few studies conducted to date in developing countries in Asia, particularly for mortality, our findings for Bangkok lend critical support to the extrapolation of empirical findings from Western, industrialized nations. Therefore, the exposure of hundreds of millions of people in Asia to high concentrations of PM_{10} and other ambient air pollutants most likely represents a significant and preventable public health burden.

ACKNOWLEDGMENTS

This study would not have been possible without contributions from and the support of many people. We wish to thank the staff at the Thailand Ministry of Public Health, the Pollution Control Department of the Thailand Ministry of Natural Resources and Environment, and the Thailand Meteorological Department, who provided us with the core data for our study. We also appreciate their patience in answering our questions and meeting our requests. A special thanks goes to Dr. Supat Wangwongwatana, Deputy Director of the Pollution Control Department, who provided us with easy and timely access to air quality data.

We were quite fortunate to be surrounded by those who provided us with valuable comments and suggestions. Much of the improvement of our study can be attributed to their advice. One such group of mentors was the International Scientific Oversight Committee. Their feedback on all our progress reports and workshops contributed to the depth and comprehensiveness of our approach and analysis. Additionally, our thanks go to Kristin Hoover, Linda Calisti, and Warren White, who taught us that data auditing is not just a scrutinizing process, in which detection of error is the only objective, but also a way to review and revisit data in order to be confident of their quality. Their suggestions and advice exceeded our expectations. Their relaxed and affable attitudes reassured us that auditing could be a learning experience.

We wish to extend our sincere appreciation to Dr. Wei Huang and Dr. Sumi Mehta for their earnest guidance and efforts in keeping us on track, both administratively and academically, throughout the two years. We wish to thank them for being our friends. Also, our study would not have run smoothly without administrative assistance from Jacqueline Rutledge and Francine Marmenout, as well as other HEI staff members.

We have gained much more than just scientific knowledge from this study from working closely with our colleagues in the PAPA project. The friendship and bonds that have developed are valuable by-products that extend beyond this project. We had the opportunity to come to know our colleagues, not only as professionals, but also as people who have many facets to their lives.

None of this would have been possible without HEI's support from the beginning to the end. We truly appreciate Daniel Greenbaum and Robert O'Keefe for including us in this important endeavor. Their commitment to, support of, and confidence in the PAPA project and the researchers involved greatly contributed to its success. We are thankful for the opportunity to be a part of this important research landmark in environmental epidemiology in Asia.

REFERENCES

Anderson HR, Atkinson RW, Peacock JL, Sweeting MJ, Marston L. 2005. Ambient particulate matter and health effects: Publication bias in studies of short-term associations. Epidemiol 16:155–163.

Anderson HR, Atkinson RW, Peacock JL, Marston L, Konstantinou K. 2004. Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (O_3): Report of a WHO task group. *http://www.euro.who.int/document/e82792.pdf*

Bangkok Metropolitan Administration. 2003. Population and household statistics in Bangkok by sub-district and districts in December 2003. *www.bma.go.th/info/.* Accessed 10/9/2003.

Castillejos M, Borja-Aburto VH, Dockery DW, Gold DR, Loomis D. 2000. Airborne coarse particles and mortality. In: Inhalation Toxicology: Proceedings of the Third Colloquium on Particulate Air Pollution and Human Health (first special issue) June 1999 (Phalen RF, ed.). Durham, NC. Inhal Toxicol 12(S1):61–72.

Cohen AJ, Anderson RH, Ostro B, Pandey KD, Krzyzanowski M, Kunzli N, Gutschmidt K, Pope A, Romieu I, Samet JM, Smith K. 2005. The global burden of disease due to outdoor air pollution. J Toxicol Environ Health A 68: 1301–1307.

Cropper ML, Simon NB, Alberini A, Arora S, Sharma PK. 1997. The health benefits of air pollution control in Delhi. Am J Agric Econ 79:1625–1629.

Dominici F, McDermott A, Daniels M, Zeger SL, Samet JM. 2003. Mortality among residents of 90 cities. In: Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute, Boston MA.

Health Effects Institute. 2003. Revised Analysis of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute, Boston, MA.

HEI International Scientific Oversight Committee. 2004. Health Effects of Outdoor Air Pollution in Developing Countries of Asia: A Literature Review. Special Report 15. Health Effects Institute, Boston, MA.

Hong YC, Leem JH, Ha EH, Christiani DC. 1999. PM_{10} exposure, gaseous pollutants, and daily mortality in Inchon, South Korea. Environ Health Perspect 107:873–878.

Katsouyanni K, Touloumi G, Samolu E, Petasakis Y, Analitis A, Le Tertre A, Rossi G, Zmirou D, Ballester F, Boumghar A, Anderson HR, Wojtyniak B, Paldy A, Braustein R, Pekkanen J, Schindler C, Schwartz J. 2003. Sensitivity analysis of various models of short-term effects of ambient particles on total mortality in 29 cities in APHEA2. In: Revised Analyses of Time-Series of Air Pollution and Health. Special Report. Health Effects Institute, Boston, MA.

Kwon HJ, Cho SH, Nyberg F, Pershagen G. 2001. Effects of ambient air pollution on daily mortality in a cohort of patients with congestive heart failure. Epidemiology 12:413–419.

Lee JT, Kim H, Hong YC, Kwon HJ, Schwartz J, Christiani DC. 2000. Air pollution and daily mortality in seven major cities of Korea, 1991–1997. Environ Res 84:247–254.

Lee JT, Schwartz J. 1999. Reanalysis of the effects of air pollution on daily mortality in Seoul, Korea: A case-crossover design. Environ Health Perspect 107:633–636.

Lee JT, Shin D, Chung Y. 1999. Air pollution and daily mortality in Seoul and Ulsan, Korea. Environ Health Perspect 107:149–154.

Mikkelsen TN, Ro-Poulsen H, Pilegaard K, Hovmand MF, Jensen NO, Christensen CS, Hummelshoej P. 2000. Ozone uptake by an evergreen forest canopy: Temporal variation and possible mechanisms. Environ Pollut 109:423–429.

Nair PR, Chand D, Lal S, Modh KS, Naja M, Parameswaran K, Ravindran S, Venkataramani S. 2002. Temporal variations in surface ozone at Thumba (8.6°N, 77°E): A tropical coastal site in India. Atmos Environ 36:603–610.

Ostro B. 1994. Estimating the health effects of air pollution: A method with an application to Jakarta. Policy research working paper 1301. World Bank, Washington, DC.

Ostro B. 2004. Outdoor Air Pollution: Assessing the Environmental Burden of Disease at National and Local Levels. Environmental Burden of Disease Series, No. 5. World Health Organization, Geneva, Switzerland.

Ostro B, Chestnut L, Vichit-Vadakan N, Laixuthai A. 1998. The impact of fine particulate matter on mortality in Bangkok, Thailand. In: $PM_{2.5}$: A Fine Particle Standard, Volume II (Chow J, Koutrakis P, eds.). Air and Waste Management Association, Pittsburgh, PA.

Ostro BD, Chestnut L, Vichit-Vadakan N, Laixuthai A. 1999. The impact of particulate matter on daily mortality in Bangkok, Thailand. J Air Waste Manag Assoc 49:100–107.

Ostro B, Sanchez JM, Aranda C, Eskeland GS. 1996. Air pollution and mortality: Results from a study of Santiago, Chile. J Expo Anal Environ Epidemiol 6:97–114.

Pollution Control Department (Thailand). 2007. Air Quality and Noise Standards. *www.pcd.go.th/info_serv/*. Accessed 12/5/2007.

Reddy RR, Gopal KR, Narasimhulu K, Reddy LSS, Kumar KR, Ahammed YN, Rao TVR, Azeem PA. 2008. Diurnal and seasonal variabilities in surface ozone and its precursor gases at a semi-arid site Anantapur (14.62° N, 77.65° E, 331 m asl) in India. Internat J Environ Stud 65:247–265.

Schwartz J. 2004. The effects of particulate air pollution on daily deaths: A multi-city case-crossover analysis. Occup Environ Med 61:956–961.

Tao Z, Larson SM, Williams A, Caughey M, Wuebbles DJ. 2004. Sensitivity of regional ozone concentrations to temporal distribution of emissions. Atmos Environ 38:6279–6285.

Tsai FC, Smith KR, Vichit-Vadakan N, Ostro BD, Chestnut LG, Kungskulniti N. 2000. Indoor/outdoor PM_{10} and $PM_{2.5}$ in Bangkok, Thailand. J Exp Anal Environ Epidemiol 10: 15–26.

U.S. Environmental Protection Agency. 2004. Review of the National Ambient Air Quality Standards for Particulate Matter. EPA-452/R-96-013. Office of Air Quality Planning and Standards, Research Triangle Park, NC.

U.S. Environmental Protection Agency. 2007. National Ambient Air Quality Standards (NAAQS). *www.epa.gov/air*. Accessed 12/5/2007.

Vajanapoom N, Shy C, Neas L, Loomis D. 2002. Associations of particulate matter and daily mortality in Bangkok, Thailand. Southeast Asian J Trop Med Public Health 33: 389–399.

Wong CM, Ma S, Hedley AJ, Lam TH. 2001. Effect of air pollution on daily mortality in Hong Kong. Environ Health Perspect 109:335–340.

World Health Organization. 2000. WHO Air Quality Guidelines for Europe. European Series, No. 91. 2nd edition. WHO Regional Office for Europe, WHO Regional Publications, Copenhagen, Denmark.

Xu Z, Yu D, Jing L, Xu X. 2000. Air pollution and daily mortality in Shenyang, China. Arch Environ Health 55: 115–120.

APPENDIX A. Technical Summary

STATISTICAL METHODS

To assess the short-term effects of PM_{10} and gaseous pollutants on daily mortality, we followed the Common Protocol of the PAPA project (found at the end of this volume). In all analyses, we used natural cubic spline models using the statistical software package R, version 2.5, with mgcv, version 1.3–24 (R Development Core Team 2007, Vienna, Austria). We used the quasi-Poisson structure in the generalized additive model option. This method accounts for the overdispersion in the confidence intervals and *P* values. The general form of the model is as follows:

$$\log E[Y_t] = \beta_0 + \beta_1 x_t + \sum_i s_i(z_{it}, k_i) + [others]$$

Where $E[Y_t]$ = expected count of the health outcome on day t; β_1 = regression coefficient of the pollutant; x_t = air pollution level at time t; s = smooth functions using a natural spline model; z_{it} = covariates (i.e., time [day], temperature [tmean], and RH); k_i = degrees of freedom (4–6 df per year for time and 3 df for tmean and RH for the entire study period); *others* = dummy variables of covariates (i.e., day of the week [dow] and public holiday).

DEVELOPMENT OF THE CORE MODEL

1. We determined the best core model for all deaths due to all natural causes, controlling for time, temperature, RH, the dow (Monday through Saturday), and whether or not it was a public holiday, before entering an air pollutant into the model.
2. We examined the model with 4 to 6 df per year for the smooth function of time and 3 df for the whole study period for the smooth function of same-day lag of daily mean temperature and daily mean RH, along with dummy variables for the dow (Monday through Saturday) and a dummy variable for public holidays.
3. Based on a criterion of minimizing the absolute magnitude of PACF values, with an additional requirement that the first- and second-order autocorrelation be less than |0.1|, the model with 6 df for time was chosen as the best core model and was used for most of our analyses. The specific codes for the selected core model are as follows:

   gam.model<-gam(allcause~ s(day, m=2, fx=TRUE, bs= "cr", k=30+1) + as.factor(dow) + holiday +s(tmean, m=2, fx=TRUE, bs="cr", k=3+1) + s(rh, fx=TRUE, bs="cr", k=3+1), data=dati2, family=quasipoisson)

CONCENTRATION–RESPONSE CURVES AND LINEARITY TEST

The concentration–response curves were plotted by the plot function under the mgcv GAM function with 3 df for each pollutant variable in the core model. The linearity of the concentration–response curves was tested by comparing the difference in the model deviances between the smoothed model and the linear model (with a linear term for the pollutant variable). The difference in model deviances follows a chi-square distribution with 2 df.

APPENDIX B. Correlations of Pollutants

Table B.1. Spearman Partial Correlation of Pollutants Averaged Across Monitors Corrected for Seasons

	PM_{10}	SO_2	NO_2	O_3	NO
PM_{10}	1.00000	0.36959	0.75434	0.48650	0.48763
SO_2		1.00000	0.38780	0.22161	0.38546
NO_2			1.00000	0.56489	0.48781
O_3				1.00000	0.09995
NO					1.00000

Table B.2. Spearman Correlation Between Specific Pollutants by Monitoring Stations[a]

	PM_{10}	SO_2	NO_2	O_3	NO
ST 1					
PM_{10}	NA	NA	NA	NA	NA
SO_2	—	1.00	0.42	NA	0.16
NO_2	—	—	1.00	NA	0.25
O_3	NA	NA	NA	NA	NA
NO	—	—	—	—	1.00
ST2					
PM_{10}	NA	NA	NA	NA	NA
SO_2	—	1.00	0.33	NA	0.25
NO_2	—	—	1.00	NA	0.38
O_3	NA	NA	NA	NA	NA
NO	—	—	—	—	1.00
ST3					
PM_{10}	NA	NA	NA	NA	NA
SO_2	—	1.00	0.70	0.36	0.61
NO_2	—	—	1.00	0.50	0.75
O_3	—	—	—	1.00	0.20
NO	—	—	—	—	1.00
ST5					
PM_{10}	NA	NA	NA	NA	NA
SO_2	—	1.00	NA	NA	NA
NO_2	NA	NA	NA	NA	NA
O_3	NA	NA	NA	NA	NA
NO	NA	NA	NA	NA	NA
ST7					
PM_{10}	NA	NA	NA	NA	NA
SO_2	—	1.00	NA	NA	NA
NO_2	NA	NA	NA	NA	NA
O_3	NA	NA	NA	NA	NA
NO	NA	NA	NA	NA	NA

[a] Definitions: NA indicates not applicable; ST indicates station.

Table B.2 (*Continued*). Spearman Correlation Between Specific Pollutants by Monitoring Stations[a]

	PM_{10}	SO_2	NO_2	O_3	NO
ST9					
PM_{10}	1.00	NA	0.65	0.50	0.24
SO_2	—	NA	NA	NA	NA
NO_2	—	—	1.00	0.39	0.46
O_3	—	—	—	1.00	−0.12
NO	—	—	—	—	1.00
ST10					
PM_{10}	1.00	0.10	0.63	0.53	0.30
SO_2	—	1.00	0.19	0.11	0.25
NO_2	—	—	1.00	0.48	0.44
O_3	—	—	—	1.00	−0.04
NO	—	—	—	—	1.00
ST11					
PM_{10}	1.00	0.37	0.49	0.13	0.29
SO_2	—	1.00	0.15	−0.22	0.42
NO_2	—	—	1.00	0.28	0.24
O_3	—	—	—	1.00	−0.53
NO	—	—	—	—	1.00
ST12					
PM_{10}	1.00	0.04	0.74	0.40	0.28
SO_2	—	1.00	−0.03	−0.12	0.25
NO_2	—	—	1.00	0.39	0.29
O_3	—	—	—	1.00	−0.35
NO	—	—	—	—	1.00
ST15					
PM_{10}	1.00	0.13	0.74	0.57	0.57
SO_2	—	1.00	0.28	0.03	0.33
NO_2	—	—	1.00	0.59	0.69
O_3	—	—	—	1.00	0.21
NO	—	—	—	—	1.00

[a] Definitions: NA indicates not applicable; ST indicates station.

Table B.3. Spearman Correlation Between Monitoring Stations by Specific Pollutant[a]

	ST1	ST2	ST3	ST5	ST7	ST9	ST10	ST11	ST12	ST15
PM_{10}										
ST1	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ST2	—	NA	NA	NA	NA	NA	NA	NA	NA	NA
ST3	—	—	NA	NA	NA	NA	NA	NA	NA	NA
ST5	—	—	—	NA	NA	NA	NA	NA	NA	NA
ST7	—	—	—	—	NA	NA	NA	NA	NA	NA
ST9	—	—	—	—	—	1.00	0.86	0.48	0.84	0.77
ST10	—	—	—	—	—	—	1.00	0.66	0.81	0.77
ST11	—	—	—	—	—	—	—	1.00	0.58	0.45
ST12	—	—	—	—	—	—	—	—	1.00	0.78
ST15	—	—	—	—	—	—	—	—	—	1.00
SO_2										
ST1	1.00	0.30	0.25	0.15	0.21	NA	0.08	0.08	0.08	0.09
ST2	—	1.00	0.45	0.24	0.16	NA	0.10	0.02	−0.01	0.16
ST3	—	—	1.00	0.41	0.17	NA	0.00	−0.09	−0.12	0.32
ST5	—	—	—	1.00	0.02	NA	0.18	−0.04	0.03	0.06
ST7	—	—	—	—	1.00	NA	0.13	0.19	0.13	0.19
ST9	—	—	—	—	—	NA	NA	NA	NA	NA
ST10	—	—	—	—	—	—	1.00	0.16	0.15	0.14
ST11	—	—	—	—	—	—	—	1.00	0.36	0.15
ST12	—	—	—	—	—	—	—	—	1.00	0.14
ST15	—	—	—	—	—	—	—	—	—	1.00
NO_2										
ST1	1.00	0.66	0.49	NA	NA	0.52	0.60	0.63	0.64	0.63
ST2	—	1.00	0.69	NA	NA	0.59	0.62	0.61	0.78	0.80
ST3	—	—	1.00	NA	NA	0.70	0.60	0.31	0.61	0.88
ST5	—	—	—	NA	NA	NA	NA	NA	NA	NA
ST7	—	—	—	—	NA	NA	NA	NA	NA	NA
ST9	—	—	—	—	—	1.00	0.72	0.45	0.60	0.62
ST10	—	—	—	—	—	—	1.00	0.56	0.60	0.62
ST11	—	—	—	—	—	—	—	1.00	0.63	0.41
ST12	—	—	—	—	—	—	—	—	1.00	0.64
ST15	—	—	—	—	—	—	—	—	—	1.00
O_3										
ST1	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
ST2	—	NA	NA	NA	NA	NA	NA	NA	NA	NA
ST3	—	—	1.00	NA	NA	0.67	0.68	0.62	0.61	0.81
ST5	—	—	—	NA	NA	NA	NA	NA	NA	NA
ST7	—	—	—	—	NA	NA	NA	NA	NA	NA
ST9	—	—	—	—	—	1.00	0.89	0.73	0.67	0.79
ST10	—	—	—	—	—	—	1.00	0.82	0.72	0.80
ST11	—	—	—	—	—	—	—	1.00	0.81	0.76
ST12	—	—	—	—	—	—	—	—	1.00	0.71
ST15	—	—	—	—	—	—	—	—	—	1.00
NO										
ST1	1.00	0.73	0.66	NA	NA	0.78	0.74	0.23	0.30	0.62
ST2	—	1.00	0.65	NA	NA	0.56	0.60	0.40	0.51	0.77
ST3	—	—	1.00	NA	NA	0.68	0.68	−0.02	0.20	0.80
ST5	—	—	—	—	NA	NA	NA	NA	NA	NA
ST7	—	—	—	—	—	NA	NA	NA	NA	NA
ST9	—	—	—	—	—	1.00	0.87	0.05	0.15	0.62
ST10	—	—	—	—	—	—	1.00	0.13	0.25	0.64
ST11	—	—	—	—	—	—	—	1.00	0.62	0.05
ST12	—	—	—	—	—	—	—	—	1.00	0.32
ST15	—	—	—	—	—	—	—	—	—	1.00

[a] Definitions: NA indicates not applicable; ST indicates station.

Table B.4. Spearman Correlation Between Lags of Temperature and Lags of PM_{10}

	PM_{10}					
Temperature	Lag 0	Lag 1	Lag 2	Lag 3	Lag 4	Lag 0–1 (Mean)
Lag 0	−0.24	−0.21	−0.21	−0.20	−0.19	−0.23
Lag 1	−0.30	−0.24	−0.21	−0.21	−0.20	−0.28
Lag 2	−0.31	−0.30	−0.24	−0.21	−0.21	−0.31
Lag 3	−0.32	−0.31	−0.30	−0.24	−0.21	−0.32
Lag 4	−0.31	−0.31	−0.31	−0.30	−0.24	−0.32
Lag 5	−0.31	−0.31	−0.31	−0.30	−0.30	−0.31
Lag 6	−0.31	−0.30	−0.30	−0.31	−0.30	−0.31
Lag 7	−0.29	−0.31	−0.30	−0.30	−0.31	−0.31
Lag 1–2 (mean)	−0.32	−0.28	−0.23	−0.21	−0.21	−0.31
Lag 3–7 (mean)	−0.35	−0.36	−0.35	−0.34	−0.31	−0.36

Table B.5. Spearman Correlation Between Lags of Temperature and Lags of SO_2

	SO_2					
Temperature	Lag 0	Lag 1	Lag 2	Lag 3	Lag 4	Lag 0–1 (Mean)
Lag 0	−0.06	−0.03	−0.04	−0.05	−0.07	−0.05
Lag 1	−0.12	−0.06	−0.03	−0.04	−0.05	−0.09
Lag 2	−0.14	−0.12	−0.06	−0.03	−0.04	−0.14
Lag 3	−0.15	−0.14	−0.12	−0.06	−0.03	−0.16
Lag 4	−0.17	−0.15	−0.14	−0.12	−0.06	−0.17
Lag 5	−0.17	−0.17	−0.15	−0.14	−0.12	−0.19
Lag 6	−0.16	−0.17	−0.17	−0.15	−0.14	−0.19
Lag 7	−0.16	−0.16	−0.17	−0.17	−0.15	−0.18
Lag 1–2 (mean)	−0.13	−0.09	−0.04	−0.03	−0.04	−0.12
Lag 3–7 (mean)	−0.18	−0.18	−0.17	−0.14	−0.11	−0.20

Table B.6. Spearman Correlation Between Lags of Temperature and Lags of NO_2

	NO_2					
Temperature	Lag 0	Lag 1	Lag 2	Lag 3	Lag 4	Lag 0–1 (Mean)
Lag 0	−0.44	−0.38	−0.35	−0.32	−0.30	−0.43
Lag 1	−0.43	−0.44	−0.38	−0.35	−0.32	−0.45
Lag 2	−0.40	−0.43	−0.44	−0.38	−0.35	−0.43
Lag 3	−0.38	−0.40	−0.43	−0.44	−0.38	−0.41
Lag 4	−0.36	−0.38	−0.40	−0.43	−0.44	−0.38
Lag 5	−0.35	−0.36	−0.38	−0.40	−0.43	−0.36
Lag 6	−0.33	−0.35	−0.36	−0.38	−0.40	−0.35
Lag 7	−0.33	−0.33	−0.34	−0.36	−0.38	−0.34
Lag 1–2 (mean)	−0.44	−0.46	−0.43	−0.38	−0.35	−0.46
Lag 3–7 (mean)	−0.41	−0.43	−0.45	−0.47	−0.47	−0.43

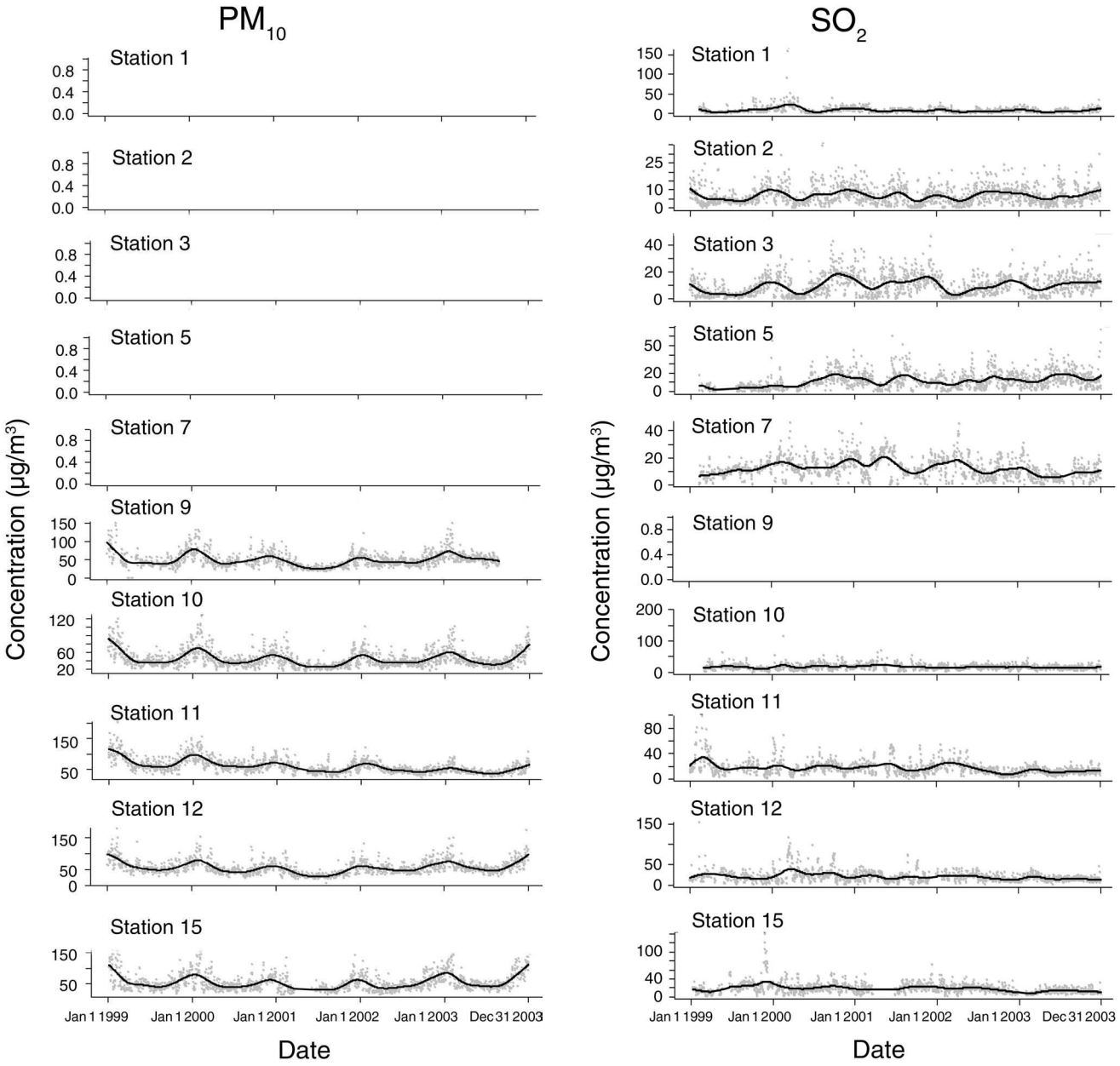
Table B.7. Spearman Correlation Between Lags of Temperature and Lags of O_3

	O_3					
Temperature	Lag 0	Lag 1	Lag 2	Lag 3	Lag 4	Lag 0–1 (Mean)
Lag 0	−0.01	−0.05	−0.09	−0.09	−0.09	−0.02
Lag 1	−0.11	−0.01	−0.05	−0.09	−0.09	−0.06
Lag 2	−0.15	−0.11	−0.01	−0.05	−0.09	−0.14
Lag 3	−0.16	−0.15	−0.11	−0.01	−0.05	−0.16
Lag 4	−0.16	−0.16	−0.15	−0.10	−0.06	−0.17
Lag 5	−0.17	−0.16	−0.16	−0.15	−0.11	−0.18
Lag 6	−0.17	−0.17	−0.16	−0.16	−0.14	−0.18
Lag 7	−0.17	−0.17	−0.17	−0.16	−0.16	−0.18
Lag 1–2 (mean)	−0.13	−0.05	−0.02	−0.07	−0.09	−0.10
Lag 3–7 (mean)	−0.20	−0.19	−0.17	−0.13	−0.12	−0.20

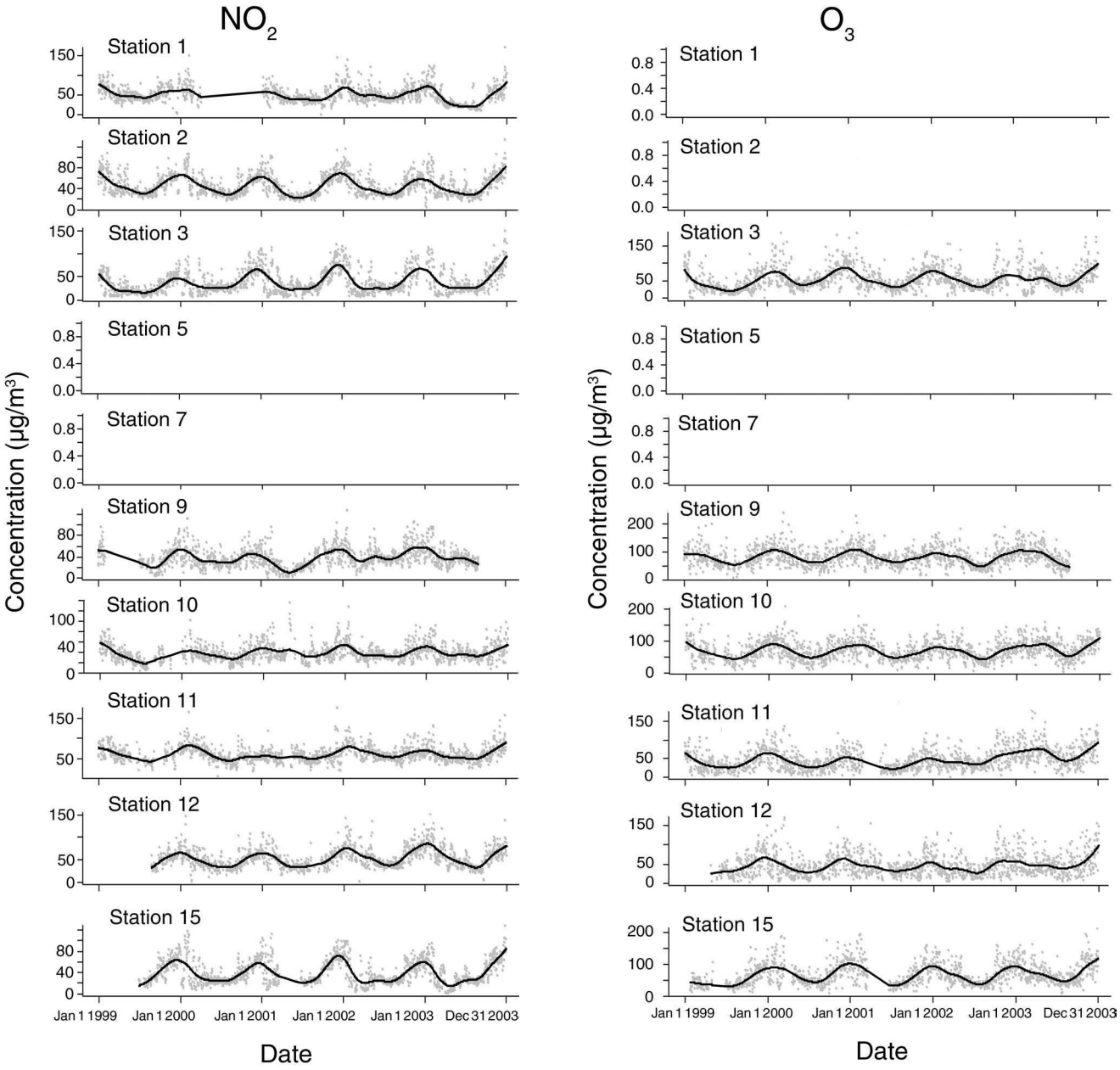
Table B.8. Spearman Correlation Between Lags of Temperature and Lags of NO

	NO					
Temperature	Lag 0	Lag 1	Lag 2	Lag 3	Lag 4	Lag 0–1 (Mean)
Lag 0	−0.37	−0.25	−0.22	−0.21	−0.21	−0.35
Lag 1	−0.39	−0.36	−0.25	−0.22	−0.21	−0.41
Lag 2	−0.33	−0.38	−0.36	−0.25	−0.23	−0.40
Lag 3	−0.30	−0.33	−0.38	−0.36	−0.26	−0.35
Lag 4	−0.28	−0.30	−0.33	−0.38	−0.37	−0.32
Lag 5	−0.26	−0.28	−0.30	−0.33	−0.39	−0.30
Lag 6	−0.25	−0.25	−0.28	−0.30	−0.33	−0.28
Lag 7	−0.24	−0.25	−0.25	−0.28	−0.30	−0.27
Lag 1–2 (mean)	−0.38	−0.39	−0.32	−0.25	−0.23	−0.43
Lag 3–7 (mean)	−0.30	−0.33	−0.36	−0.38	−0.37	−0.35

APPENDIX C. Time-Series Plots of Air Pollution



Figures C.1 and C.2. Time-series plots of PM_{10} and SO_2 concentration by monitoring station. Blank plots indicate stations with data that were less than 75% complete and were not used in the analysis.



Figures C.3 and C.4. Time-series plots of NO_2 and O_3 concentration by monitoring station. Blank plots indicate stations with data that were less than 75% complete and were not used in the analysis. O_3 was not measured at all at stations 1 and 2.

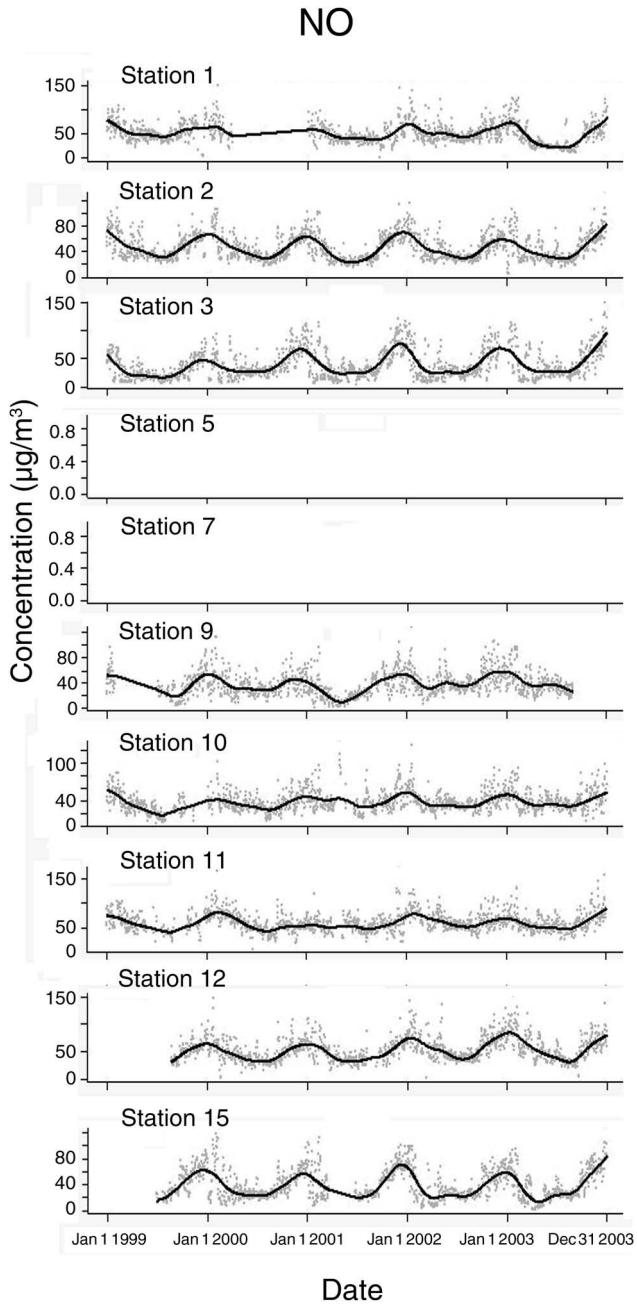


Figure C5. Time-series plots of NO concentration by monitoring station. Blank plots indicate stations with data that were less than 75% complete and were not used in the analysis.

APPENDIX D. Lag Effects of PM_{10} and Gaseous Pollutants

Table D.1. Percentage of Excess Risk and Confidence Interval in All Natural Mortality for a 10-μg/m^3 Increase in Air Pollutants by Sex[a]

Sex / Pollutant	Lag 0 % ER (95% CI)	Lag 1 % ER (95% CI)	Lag 2 % ER (95% CI)	Lag 3 % ER (95% CI)	Lag 4 % ER (95% CI)	Lag 0–1 (Mean) % ER (95% CI)	Lag 0–4 (Mean) % ER (95% CI)
Male							
PM_{10}	1.1 (0.6 to 1.6)	0.9 (0.4 to 1.4)	0.8 (0.3 to 1.3)	0.8 (0.3 to 1.3)	0.3 (−0.2 to 0.8)	1.2 (0.7 to 1.7)	1.4 (0.7 to 2.0)
SO_2	1.0 (−0.7 to 2.6)	0.3 (−1.3 to 2.0)	1.0 (−0.7 to 2.6)	0.6 (−1.1 to 2.2)	−0.2 (−1.8 to 1.4)	0.8 (−1.0 to 2.7)	1.1 (−1.3 to 3.5)
NO_2	1.2 (0.6 to 1.8)	0.8 (0.3 to 1.4)	0.9 (0.3 to 1.4)	0.7 (0.1 to 1.2)	0.3 (−0.2 to 0.9)	1.2 (0.6 to 1.9)	1.4 (0.6 to 2.2)
O_3	0.6 (0.3 to 0.9)	0.3 (0.0 to 0.6)	0.3 (0.0 to 0.6)	0.4 (0.1 to 0.7)	0.4 (0.1 to 0.7)	0.6 (0.2 to 1.0)	1.0 (0.5 to 1.5)
NO	0.8 (0.2 to 1.3)	0.5 (0.0 to 1.0)	0.6 (0.0 to 1.1)	0.3 (−0.2 to 0.9)	−0.2 (−0.7 to 0.4)	0.8 (0.2 to 1.4)	0.8 (0.1 to 1.6)
Female							
PM_{10}	1.2 (0.6 to 1.8)	1.0 (0.4 to 1.5)	1.1 (0.6 to 1.7)	1.1 (0.6 to 1.7)	0.5 (0.0 to 1.1)	1.3 (0.7 to 1.9)	1.7 (1.0 to 2.5)
SO_2	2.1 (0.2 to 4.0)	1.9 (0.0 to 3.9)	−0.1 (−2.0 to 1.8)	0.8 (−1.1 to 2.7)	−0.5 (−2.3 to 1.4)	2.6 (0.4 to 4.8)	1.7 (−1.0 to 4.6)
NO_2	1.5 (0.8 to 2.2)	1.2 (0.5 to 1.9)	1.2 (0.6 to 1.9)	0.9 (0.3 to 1.6)	0.7 (0.0 to 1.3)	1.6 (0.9 to 2.4)	2.0 (1.1 to 2.9)
O_3	0.4 (0.0 to 0.8)	0.6 (0.2 to 1.0)	0.4 (0.0 to 0.7)	0.5 (0.2 to 0.9)	0.3 (−0.1 to 0.7)	0.7 (0.3 to 1.2)	1.1 (0.5 to 1.7)
NO	1.0 (0.4 to 1.7)	0.9 (0.3 to 1.5)	0.8 (0.2 to 1.4)	0.7 (0.1 to 1.3)	0.0 (−0.6 to 0.6)	1.2 (0.5 to 2.0)	1.4 (0.5 to 2.4)

[a] Definitions: CI indicates confidence interval; ER indicates excess risk.

Table D.2. Percentage of Excess Risk and Confidence Interval in All Natural Mortality for a 10-μg/m^3 Increase in Air Pollutants by Age[a]

Age / Pollutant	Lag 0 % ER (95% CI)	Lag 1 % ER (95% CI)	Lag 2 % ER (95% CI)	Lag 3 % ER (95% CI)	Lag 4 % ER (95% CI)	Lag 0–1 (Mean) % ER (95% CI)	Lag 0–4 (Mean) % ER (95% CI)
0–4 yr							
PM_{10}	0.2 (−1.7 to 2.3)	0.1 (−1.9 to 2.0)	−0.6 (−2.5 to 1.3)	−0.3 (−2.2 to 1.6)	−1.1 (−3.0 to 0.8)	0.2 (−2.0 to 2.4)	−0.7 (−3.2 to 2.0)
SO_2	5.2 (−1.4 to 12.3)	0.1 (−6.3 to 7.0)	2.5 (−4.0 to 9.5)	6.7 (0.0 to 13.8)	2.4 (−4.1 to 9.3)	3.4 (−4.1 to 11.5)	7.2 (−2.5 to 18.0)
NO_2	0.3 (−2.2 to 2.8)	−0.9 (−3.2 to 1.4)	−0.7 (−2.9 to 1.7)	−0.8 (−3.1 to 1.5)	−1.7 (−4.0 to 0.6)	−0.4 (−3.1 to 2.2)	−1.4 (−4.5 to 1.8)
O_3	−1.0 (−2.4 to 0.4)	−0.2 (−1.5 to 1.1)	−0.4 (−1.7 to 0.9)	0.6 (−0.6 to 1.9)	−0.2 (−1.5 to 1.0)	−0.8 (−2.4 to 0.8)	−0.6 (−2.6 to 1.5)
NO	0.9 (−1.3 to 3.2)	0.5 (−1.6 to 2.7)	1.5 (−0.6 to 3.7)	0.5 (−1.7 to 2.6)	−1.0 (−3.1 to 1.1)	0.9 (−1.6 to 3.5)	1.1 (−2.1 to 4.3)
5–44 yr							
PM_{10}	0.8 (0.1 to 1.5)	0.7 (0.1 to 1.4)	0.8 (0.2 to 1.5)	0.8 (0.2 to 1.5)	0.2 (−0.4 to 0.9)	0.9 (0.2 to 1.7)	1.2 (0.3 to 2.1)
SO_2	0.1 (−2.2 to 2.4)	0.2 (−2.0 to 2.5)	0.1 (−2.2 to 2.4)	−1.2 (−3.4 to 1.1)	−0.9 (−3.1 to 1.4)	0.2 (−2.4 to 2.8)	−0.7 (−3.9 to 2.6)
NO_2	1.1 (0.2 to 1.9)	0.5 (−0.3 to 1.3)	0.6 (−0.2 to 1.4)	0.5 (−0.3 to 1.3)	0.1 (−0.7 to 0.9)	0.9 (0.0 to 1.8)	1.0 (−0.1 to 2.1)
O_3	0.5 (0.0 to 1.0)	0.1 (−0.3 to 0.6)	0.1 (−0.3 to 0.5)	0.2 (−0.2 to 0.7)	0.3 (−0.1 to 0.8)	0.4 (−0.2 to 1.0)	0.6 (−0.1 to 1.3)
NO	0.5 (−0.2 to 1.3)	0.4 (−0.3 to 1.2)	0.6 (−0.1 to 1.3)	0.2 (−0.5 to 0.9)	−0.2 (−1.0 to 0.5)	0.6 (−0.2 to 1.5)	0.6 (−0.5 to 1.7)
45–64 yr							
PM_{10}	1.0 (0.3 to 1.7)	0.9 (0.2 to 1.6)	1.0 (0.4 to 1.7)	0.8 (0.1 to 1.4)	0.2 (−0.5 to 0.9)	1.1 (0.4 to 1.9)	1.3 (0.4 to 2.2)
SO_2	1.1 (−1.2 to 3.4)	0.3 (−2.0 to 2.6)	1.2 (−1.1 to 3.5)	−0.2 (−2.4 to 2.1)	−1.1 (−3.3 to 1.2)	0.9 (−1.7 to 3.5)	0.6 (−2.7 to 4.0)
NO_2	0.9 (0.1 to 1.8)	1.2 (0.4 to 2.0)	1.4 (0.6 to 2.2)	0.6 (−0.2 to 1.4)	0.3 (−0.5 to 1.1)	1.3 (0.4 to 2.2)	1.7 (0.6 to 2.8)
O_3	0.6 (0.1 to 1.1)	0.5 (0.1 to 1.0)	0.5 (0.1 to 1.0)	0.1 (−0.3 to 0.6)	0.3 (−0.1 to 0.7)	0.8 (0.3 to 1.4)	1.0 (0.3 to 1.7)
NO	0.7 (−0.1 to 1.5)	0.6 (−0.1 to 1.4)	0.6 (−0.1 to 1.4)	0.7 (0.0 to 1.4)	−0.3 (−1.00 to 0.5)	0.8 (0.0 to 1.7)	1.0 (−0.1 to 2.1)
75+ yr							
PM_{10}	2.0 (1.2 to 2.8)	1.7 (0.9 to 2.4)	1.5 (0.8 to 2.3)	1.3 (0.5 to 2.0)	0.6 (−0.2 to 1.3)	2.2 (1.3 to 3.0)	2.5 (1.4 to 3.5)
SO_2	3.6 (1.0 to 6.3)	3.6 (1.0 to 6.3)	0.3 (−2.2 to 2.9)	1.4 (−1.1 to 4.1)	−1.1 (−3.6 to 1.5)	4.6 (1.7 to 7.7)	3.3 (−0.5 to 7.3)
NO_2	2.5 (1.5 to 3.4)	2.4 (1.5 to 3.3)	1.8 (0.9 to 2.7)	1.4 (0.6 to 2.3)	0.7 (−0.2 to 1.6)	2.9 (1.9 to 4.0)	3.3 (2.0 to 4.5)
O_3	1.0 (0.5 to 1.6)	1.0 (0.5 to 1.5)	0.7 (0.2 to 1.2)	1.1 (0.7 to 1.6)	0.3 (−0.2 to 0.8)	1.5 (0.9 to 2.1)	2.1 (1.3 to 2.9)
NO	1.4 (0.6 to 2.3)	1.1 (0.3 to 2.0)	1.0 (0.1 to 1.8)	0.5 (−0.4 to 1.3)	−0.1 (−1.0 to 0.7)	1.6 (0.7 to 2.6)	1.6 (0.4 to 2.9)

[a] Definitions: CI indicates confidence interval; ER indicates excess risk.

Table D.3 Percentage of Excess Risk and Confidence Interval in Mortality for a 10-μg/m^3 Increase in Gaseous Pollutants with PM_{10} Adjustment by Causes of Death[a]

Mortality Class / Pollutant	Lag 0 % ER (95% CI)	Lag 1 % ER (95% CI)	Lag 2 % ER (95% CI)	Lag 3 % ER (95% CI)	Lag 4 % ER (95% CI)	Lag 0–1 (Mean) % ER (95% CI)	Lag 0–4 (Mean) % ER (95% CI)
All Natural							
SO_2	0.1 (−1.3 to 1.6)	−0.3 (−1.7 to 1.2)	−0.7 (−2.2 to 0.7)	−0.5 (−1.9 to 1.0)	−0.5 (−1.9 to 0.9)	0.1 (−1.5 to 1.7)	0.0 (−2.0 to 2.0)
NO_2	0.3 (−0.6 to 1.1)	0.4 (−0.4 to 1.2)	0.4 (−0.4 to 1.2)	−0.2 (−1.0 to 0.6)	0.3 (−0.5 to 1.1)	0.4 (−0.6 to 1.4)	0.5 (−0.7 to 1.7)
O_3	0.1 (−0.2 to 0.5)	0.1 (−0.2 to 0.4)	−0.1 (−0.4 to 0.2)	0.2 (−0.1 to 0.5)	0.2 (−0.1 to 0.5)	0.2 (−0.2 to 0.6)	0.4 (−0.1 to 0.9)
NO	0.2 (−0.3 to 0.8)	0.3 (−0.2 to 0.8)	0.3 (−0.2 to 0.8)	0.0 (−0.5 to 0.5)	−0.4 (−0.9 to 0.1)	0.4 (−0.2 to 1.0)	0.3 (−0.4 to 1.0)
Cardiovascular							
SO_2	−0.6 (−4.2 to 3.1)	−3.0 (−6.5 to 0.6)	−1.2 (−4.7 to 2.5)	−2.1 (−5.6 to 1.5)	−0.4 (−3.9 to 3.2)	−2.0 (−6.0 to 2.1)	−1.8 (−6.7 to 3.3)
NO_2	−1.1 (−3.2 to 1.0)	0.4 (−1.6 to 2.4)	−0.2 (−2.1 to 1.8)	−0.6 (−2.5 to 1.4)	0.4 (−1.5 to 2.4)	−0.5 (−2.9 to 1.9)	−0.4 (−3.3 to 2.7)
O_3	−0.3 (−1.1 to 0.5)	0.3 (−0.4 to 1.1)	0.0 (−0.8 to 0.7)	−0.1 (−0.9 to 0.6)	−0.3 (−1.0 to 0.5)	0.1 (−0.9 to 1.0)	−0.1 (−1.4 to 1.2)
NO	0.9 (−0.4 to 2.2)	0.8 (−0.4 to 2.0)	0.9 (−0.3 to 2.2)	0.5 (−0.7 to 1.7)	−0.4 (−1.6 to 0.8)	1.1 (−0.3 to 2.6)	1.3 (−0.5 to 3.2)
Respiratory							
SO_2	0.1 (−4.3 to 4.8)	0.6 (−3.9 to 5.3)	2.0 (−2.5 to 6.7)	3.2 (−1.3 to 7.9)	1.7 (−2.7 to 6.4)	0.7 (−4.3 to 6.0)	4.1 (−2.3 to 11.0)
NO_2	0.1 (−2.6 to 2.8)	0.4 (−2.1 to 2.9)	−0.9 (−3.3 to 1.6)	−0.1 (−2.5 to 2.3)	−0.8 (−3.2 to 1.6)	0.3 (−2.7 to 3.4)	−0.7 (−4.3 to 3.1)
O_3	0.9 (−0.1 to 1.9)	0.0 (−0.9 to 1.0)	−0.2 (−1.1 to 0.8)	0.5 (−0.4 to 1.4)	0.3 (−0.6 to 1.2)	0.7 (−0.5 to 1.9)	0.9 (−0.6 to 2.5)
NO	−1.6 (−3.2 to 0.1)	−1.3 (−2.8 to 0.3)	−1.1 (−2.6 to 0.4)	−0.8 (−2.2 to 0.7)	0.5 (−0.9 to 2.0)	−1.8 (−3.6 to 0.0)	−1.6 (−3.8 to 0.7)
Age 65+							
SO_2	0.7 (−1.3 to 2.7)	0.7 (−1.2 to 2.7)	−1.2 (−3.2 to 0.7)	0.6 (−1.3 to 2.6)	−0.7 (−2.6 to 1.3)	1.1 (−1.1 to 3.4)	1.0 (−1.7 to 3.9)
NO_2	0.8 (−0.4 to 2.0)	0.7 (−0.4 to 1.9)	0.3 (−0.8 to 1.4)	0.2 (−0.9 to 1.3)	0.8 (−0.2 to 1.9)	0.9 (−0.4 to 2.3)	1.3 (−0.4 to 3.0)
O_3	0.1 (−0.3 to 0.6)	0.2 (−0.2 to 0.7)	0.0 (−0.4 to 0.4)	0.5 (0.1 to 0.9)	0.2 (−0.2 to 0.6)	0.3 (−0.2 to 0.8)	0.8 (0.1 to 1.5)
NO	0.4 (−0.4 to 1.1)	0.3 (−0.4 to 1.0)	0.2 (−0.5 to 0.8)	0.0 (−0.7 to 0.6)	−0.3 (−0.9 to 0.4)	0.5 (−0.3 to 1.3)	0.4 (−0.6 to 1.4)

[a] Definitions: CI indicates confidence interval; ER indicates excess risk.

Table D.4. Percentage of Excess Risk in Mortality and Confidence Interval for a 10-μg/m^3 Increase in PM_{10} with Gaseous Adjustment by Causes of Death[a]

Mortality Class / Adjusted Pollutant	Lag 0 PM_{10} % ER (95% CI)	Lag 1 PM_{10} % ER (95% CI)	Lag 2 PM_{10} % ER (95% CI)	Lag 3 PM_{10} % ER (95% CI)	Lag 4 PM_{10} % ER (95% CI)	Lag 0–1 (Mean) PM_{10} % ER (95% CI)	Lag 0–4 (Mean) PM_{10} % ER (95% CI)
All Natural							
SO_2	1.2 (0.7 to 1.6)	1.0 (0.6 to 1.4)	1.0 (0.6 to 1.4)	0.9 (0.4 to 1.3)	0.3 (−0.1 to 0.7)	1.2 (0.8 to 1.7)	1.4 (0.9 to 2.0)
NO_2	1.0 (0.3 to 1.7)	0.7 (0.0 to 1.4)	0.6 (0.0 to 1.3)	0.9 (0.3 to 1.6)	0.1 (−0.6 to 0.7)	1.0 (0.2 to 1.8)	1.1 (0.1 to 2.0)
O_3	1.1 (0.6 to 1.5)	0.9 (0.4 to 1.3)	1.0 (0.5 to 1.4)	0.6 (0.2 to 1.1)	0.1 (−0.4 to 0.6)	1.1 (0.6 to 1.7)	1.1 (0.5 to 1.8)
NO	1.0 (0.6 to 1.5)	0.8 (0.4 to 1.3)	0.8 (0.3 to 1.2)	0.8 (0.4 to 1.2)	0.4 (0.0 to 0.9)	1.1 (0.6 to 1.6)	1.3 (0.7 to 1.9)
Cardiovascular							
SO_2	1.6 (0.5 to 2.7)	2.0 (0.9 to 3.1)	1.7 (0.6 to 2.8)	1.1 (0.0 to 2.1)	−0.1 (−1.1 to 1.0)	2.1 (0.9 to 3.3)	2.0 (0.6 to 3.4)
NO_2	2.3 (0.5 to 4.2)	1.4 (−0.3 to 3.1)	1.7 (0.0 to 3.4)	1.3 (−0.4 to 3.0)	−0.4 (−2.1 to 1.3)	2.3 (0.2 to 4.3)	2.1 (−0.4 to 4.7)
O_3	1.8 (0.6 to 3.0)	1.4 (0.2 to 2.6)	1.6 (0.4 to 2.8)	0.9 (−0.3 to 2.1)	0.1 (−1.0 to 1.3)	1.8 (0.5 to 3.2)	2.0 (0.3 to 3.6)
NO	1.1 (−0.1 to 2.3)	1.3 (0.2 to 2.4)	1.2 (0.1 to 2.3)	0.6 (−0.5 to 1.7)	0.1 (−1.0 to 1.2)	1.4 (0.1 to 2.7	1.3 (−0.2 to 2.9)
Respiratory							
SO_2	1.0 (−0.4 to 2.4)	0.7 (−0.6 to 2.0)	0.9 (−0.4 to 2.2)	1.0 (−0.3 to 2.3)	0.5 (−0.8 to 1.8)	0.9 (−0.5 to 2.4)	1.4 (−0.3 to 3.1)
NO_2	0.9 (−1.3 to 3.2)	0.5 (−1.6 to 2.7)	1.7 (−0.4 to 3.9)	1.4 (−0.7 to 3.5)	1.2 (−0.8 to 3.3)	0.8 (−1.7 to 3.3)	2.2 (−0.9 to 5.4)
O_3	0.3 (−1.2 to 1.8)	0.7 (−0.7 to 2.2)	1.2 (−0.2 to 2.7)	0.9 (−0.5 to 2.4)	0.4 (−1.0 to 1.9)	0.5 (−1.2 to 2.2)	1.0 (−1.0 to 3.1)
NO	1.8 (0.2 to 3.3)	1.3 (−0.1 to 2.8)	1.6 (0.2 to 3.0)	1.6 (0.3 to 3.0)	0.4 (−0.9 to 1.8)	1.9 (0.2 to 3.5)	2.4 (0.5 to 4.3)
Age 65+							
SO_2	1.4 (0.8 to 2.0)	1.0 (0.5 to 1.6)	1.2 (0.7 to 1.8)	1.1 (0.5 to 1.7)	0.8 (0.2 to 1.4)	1.4 (0.8 to 2.1)	1.8 (1.1 to 2.6)
NO_2	0.9 (0.0 to 1.9)	0.6 (−0.3 to 1.6)	0.9 (0.0 to 1.8)	1.0 (0.1 to 1.9)	0.1 (−0.8 to 1.1)	0.9 (−0.2 to 2.0)	1.0 (−0.3 to 2.4)
O_3	1.4 (0.7 to 2.0)	0.9 (0.3 to 1.6)	1.1 (0.5 to 1.7v	0.7 (0.1 to 1.3)	0.5 (−0.1 to 1.2)	1.3 (0.6 to 2.0)	1.3 (0.5 to 2.2)
NO	1.3 (0.6 to 1.9)	1.0 (0.4 to 1.6)	1.0 (0.4 to 1.7)	1.2 (0.6 to 1.8)	0.8 (0.2 to 1.4)	1.3 (0.6 to 2.0)	1.8 (0.9 to 2.6)

[a] Definitions: CI indicates confidence interval; ER indicates excess risk.

APPENDIX E. HEI Quality Assurance Statement

The conduct of this study was subjected to periodic, independent audits by a team from Hoover Consultants. This team consisted of auditors with experience in toxicology, epidemiology, and air quality data. The audits included in-process monitoring of study activities for conformance to the study protocol and examination of records and supporting data. The dates of each audit are listed in the table below with the phase of the study examined.

QUALITY ASSURANCE AUDITS

Date	Phase of Study
May 26–27, 2005	Data for mortality, air quality parameters, and meteorology were audited. Documentation was examined for personnel qualifications and experience. During this site visit, data were audited in the 5- and 10-Month Progress reports supplied by HEI. This study was conducted in accordance with an individual study protocol that covered the unique features of the Bangkok study and a combined protocol for the coordinated time-series analysis. Study documentation was reviewed to determine that it met the requirements of both protocols. Statements in the text of the reports were compared to the subsequent tables, figures, and data files. Reports were reviewed for internal consistency. Air pollution data were provided by the investigators as electronic copies of the original zipped text files of 1997–2003 hourly data delivered by the Pollution Control Department. The investigators provided a spreadsheet of daily data used to produce daily citywide averages for the epidemiologic analysis and the summary statistics in the 10-Month Progress Report. Dr. Patcharawadee Suwanathada of the Pollution Control Department was present to answer questions and provide background. The audit team made a series of database queries designed to validate all of the numbers presented in the 10-Month Progress Report (with the exception of Table 3.1, death counts by district, for the years 1997 and 1998 since these years were to be dropped from the study). In addition, the auditors visited District 10 (Mean-Buri) to compare a sample of 50 death certificates to 12 different variables per certificate in the study database. The underlying cause of death was verbally translated by study personnel and compared to the ICD-10 code that had been assigned. The audit team then compared these assigned codes to the written descriptions of the underlying cause of death in the ICD-10 Classification System.
April 7, 2008	A draft of the final study report was examined for internal consistency and conformance with the study proposal. Comments were provided to HEI via e-mail.

A written report of the first inspection was provided to the Director of Science of the Health Effects Institute who transmitted these findings to the Principal Investigator. These quality assurance audits demonstrated that the study was conducted by experienced professionals in accordance with the study protocols. The report appears to be an accurate representation of the study.

B. Kristin Hoover
Hoover Consultants

ABOUT THE AUTHORS

Nuntavarn Vichit-Vadakan, Dr. PH., has been involved in air pollution research in Thailand for many years. Her work in environmental epidemiology in Thailand has led to policy changes and the development of mitigation measures (environmental management interventions). She was the lead author on three labor- and data-intensive panel studies examining the effects of PM_{10} and $PM_{2.5}$ in Thailand and has led many training sessions for Thai students and faculty in epidemiologic methods.

Nitaya Vajanapoom has a Ph.D. in epidemiology from the University of North Carolina and is an environmental epidemiologist at Thammasat University in Bangkok. She has published studies on air pollution and mortality in Thailand (Vajanapoom et al. 2002) and is one of the lead epidemiologists on a five-year study that examines the health

effects of air pollution from a lignite power plant located in Lampang province, Thailand. Her work has contributed to the Thai government's efforts in setting $PM_{2.5}$ standards.

Bart Ostro, Ph.D., has published more than 70 studies linking air pollution to adverse health, including studies in Thailand (Ostro et al. 1999), in other developing countries including Chile, and in developed countries outside the United States. He has also worked with researchers in China, India, Indonesia, Malaysia, Mexico, Nepal, and Sri Lanka in collecting or evaluating data, developing epidemiologic studies, or assessing health effects. Finally, he has led several training programs for Thai students and faculty.

OTHER PUBLICATIONS RESULTING FROM THIS RESEARCH

Vichit-Vadakan N, Vajanapoom N, Ostro B. 2008. The Public Health and Air Pollution in Asia (PAPA) project: Estimating the mortality effects of particulate matter in Bangkok, Thailand. Environ Health Perspect 116:1179–1182.

Wong CM, Vichit-Vadakan N, Kan H, Qian Z, the PAPA Project Team. 2008. Public Health and Air Pollution in Asia (PAPA): A multicity study of short-term effects of air pollution on mortality. Environ Health Perspect 116: 1195–1202.

ABBREVIATIONS AND OTHER TERMS

AR1	model with first-order autocorrelation term
CI	confidence interval
COPD	chronic obstructive pulmonary disease
df	degrees of freedom
dow	day of week
ER	excess risk
ICD-10	*International Classification of Diseases*, 10th revision
IQR	interquartile range
LRI	lower respiratory infection
NO	nitric oxide
NO_2	nitrogen dioxide
O_3	ozone
OEP	Office of Environmental Policy and Planning
PACF	partial autocorrelation function
PAPA	Public Health and Air Pollution in Asia
PM	particulate matter
$PM_{2.5}$	particulate matter $\leq$ 2.5 μm in aerodynamic diameter
PM_{10}	particulate matter $\leq$ 10 μm in aerodynamic diameter
RH	relative humidity
RR	relative risk
SO_2	sulfur dioxide
U.S. EPA	U.S. Environmental Protection Agency
WHO	World Health Organization

Research Report 154, Part 3. *Estimating the Effects of Air Pollution on Mortality in Bangkok, Thailand*, N. Vichit-Vadakan et al.

BACKGROUND

The study by Dr. Vitchit-Vadakan of Thammasat University, Pathumthani, Thailand, and colleagues, titled *Estimating the Effects of Air Pollution on Mortality in Bangkok, Thailand*, was conducted as part of a coordinated suite of time-series studies of the health effects of short-term exposure to air pollution in major Asian cities, a major component of HEI's Public Health and Air Pollution in Asia (PAPA*) program. Information on the origins, objectives, and scope of the health and environmental conditions in Bangkok, Thailand, and a brief review of previous epidemiologic research on the health effects of air pollution in Bangkok are presented in the Overview for the four PAPA time-series studies in this volume.

Previous time-series studies of daily pollutant levels and mortality conducted predominantly in North America and Europe have found that increasing levels of air pollution were associated with increasing mortality from varying causes (Kinney and Ozkaynak 1991; Anderson et al. 1996; Sunyer et al. 1996; Wietlisbach et al. 1996; Touloumi et al. 1997; Saez et al. 1999). Thailand is one of several Asian countries that has adopted national ambient air quality standards for pollutants, including nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and particulate matter (PM) with an aerodynamic diameter $\leq$ 10 μm (PM_{10}). Monitoring studies conducted in Bangkok have shown that ambient levels of PM_{10} frequently exceed that of the Thai government's national daily standards of 120 $\mu g/m^3$ (Jinsart et al. 2002).

SPECIFIC AIMS

In response to a request by the HEI Research Committee, Dr. Vitchit-Vadakan submitted an application to investigate the impact of air pollution on daily mortality in Bangkok using six years of data (1996–2001). That time period included an economic recession (June 1996–December 1997). Hence, the study also sought to evaluate the impact of economic recession on traffic-related air pollution and any possible effects on mortality rates. The Committee considered these aims responsive to the objective of the PAPA program and recommended the study for funding. The Committee also recommended funding time-series studies in three other Asian cities: Hong Kong, Shanghai, and Wuhan.

The originally proposed specific aims of the present study were as follows:

1. Using PM_{10} monitoring data, quantify the association between ambient air pollution and mortality in Bangkok.
2. Using stratified analyses, examine the potential effect modification by sex and age in the association between air pollution and mortality.
3. Examine disease-specific associations between mortality and air pollution.
4. Examine whether reductions in local traffic levels during an economic recession affected mortality rates and the resultant concentration–response functions.

After the study had been completed and the report submitted, the HEI Review Committee was not convinced that the economic analyses were informative and suggested that the revised final report exclude this aim. Thus, the final report accepted by HEI had the following specific aims:

1. To evaluate the association between levels of ambient particulate and gaseous air pollutants (PM_{10}, O_3, NO_2, and SO_2) and daily mortality due to all natural causes (nonaccidental) in Bangkok.
2. To evaluate both disease-specific and age-specific mortality in the identification of subgroups at particular risk for the adverse health effects of air pollution.
3. To conduct extensive sensitivity analyses of the results in order to examine the influence of model specification, smoothing methods, lag structure, and copollutants.

Dr. Nuntavarn Vichit-Vadakan's 2-year study "Estimating the Mortality Effects of Air Pollution in Bangkok, Thailand" began in June 2004. Total expenditures were $119,055. The draft Investigators' Report from Vichit-Vadakan and colleagues was received for review in September 2006. The revised report received in October 2007 was accepted for publication in December 2007. During the review process, the HEI Health Review Committee and the investigators had the opportunity to exchange comments and to clarify issues in both the Investigators' Report and the Review Committee's Commentary.

*A list of abbreviations and other terms appears at the end of the Investigators' Report.

DATA SOURCES

The investigators used three sets of data — mortality, air pollution, and meteorologic — which were compiled electronically by the responsible government agency.

Mortality data were obtained from the Bureau of Registration Administration for the Bangkok Metropolitan Region for the period June 1, 1997, to May 31, 2003. Data consistency checks comparing the electronic mortality data to death certificates revealed that a significantly lower number of deaths were recorded for the period June 1, 1997, to December 31, 1998. Thus this period was excluded from the analyses, and the study period was changed to January 1, 1999, to December 31, 2003. Causes of death were classified using the tenth revision of the *International Classification of Diseases* (ICD-10).

Air pollution data were collected from several sets of monitors, set up by the Pollution Control Department of the Thai Ministry of Natural Resources and Environment to measure air pollution in Bangkok. Hourly measurements of PM_{10} levels were available for 5 ambient and 7 roadside monitoring stations; 10 stations measured hourly ambient NO_2, SO_2, and nitric oxide (NO), while 8 stations measured hourly ambient O_3. Since there was substantial road traffic around the 7 roadside monitors, the researchers used only the PM_{10} data from the 5 ambient monitors, believing that these measurements best represented actual population exposures in Bangkok. As with all of the PAPA studies, carbon monoxide was not included in the analyses. Individual monitors were included in the study if data from the monitor were determined to be internally consistent, were not heavily influenced by a local air pollution source, and met the inclusion criteria for individual pollutants described in the Common Protocol (found at the end of this volume) and Integrated Discussion (found in Part 5 of this volume).

Daily meteorologic data were obtained from the Bangkok metropolitan weather station for the study period. The data included average daily temperature and average daily relative humidity (RH).

DATA ANALYSIS

To assess the short-term effect of PM_{10} and gaseous pollutants on daily mortality, the investigators used the statistical analyses that were developed by all participants in the PAPA project as part of the Common Protocol (see the Integrated Discussion in Part 5 of this volume).

The investigators determined the best core model for all natural mortality in accordance with the agreed-upon Common Protocol: a model using 6 degrees of freedom (df) minimized the partial autocorrelation, and so the investigators used this specification in all subsequent analyses. The core model used a two-day average of lag 0 and lag 1 (lag 0–1 day), but the investigators also examined lags of up to 5 days and moving averages of up to 5 days. A day-of-the-week term was also included. This core model for all deaths due to natural causes was used for each of the subcategories of cause of death, specifically, cardiopulmonary (cardiovascular and respiratory), cardiovascular separately (including subcategories of ischemic heart disease, stroke, and conduction disorders), respiratory separately (including subcategories of chronic obstructive pulmonary disorder [COPD] and asthma), "senility," non-cardiopulmonary (natural), and accidental.

They also performed several sensitivity analyses in order to test their modeling assumptions, including the fitting of models with different lags for PM_{10}; different sets of degrees of freedom for time and weather; different lags of temperature and RH; and penalized splines in place of natural splines for time and weather. They also fitted copollutant models that assessed the effects of PM_{10}, adjusted for gaseous pollutants. In addition, they performed a sensitivity analysis that considered an influenza epidemic as a potential confounder of pollutant effects, defining "influenza epidemic" as a weekly respiratory mortality count greater than the 90th percentile of the mean frequency (count) of respiratory mortality for each year.

RESULTS

Commentary Tables 1–3 and the following paragraphs summarize the key findings. All analysis results are presented as excess risk (ER) per 10 $\mu g/m^3$ of pollutant, calculated from relative risk (RR) as ER = (RR − 1) × 100.

DAILY MORTALITY AND POLLUTANT INFORMATION

Commentary Table 1, which summarizes information from Table 2 of the Vichit-Vadakan Investigators' Report, shows that during the study period (1999–2003) Bangkok had an average of 95 deaths per day from all natural causes. An average of 13 deaths per day were attributed to cardiovascular causes, 8 deaths per day were due to respiratory causes, and 14 deaths were attributed to "senility." Dr. Vitchit-Vadakan included "senility" as an endpoint because there were lower-than-expected daily counts of deaths in the elderly that were attributed to cardiovascular diseases and a correspondingly high number attributed to "senility." Bangkok recorded an average of 45 deaths per

Commentary Table 1. Average Daily Mortality in Bangkok During Study Period for Total Nonaccidental Mortality and Selected Other Categories of Cause of Death[a]

Cause of Death	Deaths per Day (SD)
All natural (nonaccidental)	95 (12.1)
Cardiovascular	14 (4.3)
Respiratory	8 (3.1)
"Senility"	14 (4.2)

[a] Definition: SD indicates standard deviation.

day in persons age 65 or older (see Vichit-Vadakan Investigators' Report Table 1).

Commentary Table 2 shows the mean 24-hour concentrations of PM_{10}, SO_2, NO_2, O_3, and NO and mean temperature and RH during the study period.

The investigators reported relatively high correlations between daily levels of PM_{10} and NO_2 ($r = 0.71$), PM_{10} and O_3 ($r = 0.55$), and O_3 and NO_2 ($r = 0.62$) (Vichit-Vadakan Investigators' Report Table 5). Mean temperature and humidity were provided for the entire year as the relatively low standard deviation demonstrated the relatively minimal seasonal variation in Bangkok's weather due to its tropical location and local weather conditions (see Vichit-Vadakan Investigators' Report Table 6).

ASSOCIATIONS BETWEEN DAILY MORTALITY AND INDIVIDUAL POLLUTANTS

Commentary Table 3 summarizes the main findings of associations between individual pollutants and daily deaths by cause of mortality in single-pollutant models (shown in Table 7a of the Vichit-Vadakan Investigators' Report).

Commentary Table 2. Mean 24-Hour Pollutant Concentrations, Temperature, and Relative Humidity in Bangkok During Study Period[a]

Pollutant / Meteorologic Measurement	Mean (SD)	Maximum
PM_{10} (μg/m^3)	52.1 (20.1)	169.2
SO_2 (μg/m^3)	13.2 (4.8)	61.2
NO_2 (μg/m^3)	44.7 (17.3)	139.6
O_3 (μg/m^3)	59.4 (26.4)	180.6
NO (μg/m^3)	28.0 (14.2)	126.9
Temperature (°C)	28.9 (1.7)	33.6
RH (%)	72.8 (8.3)	95.0

[a] Definitions: RH indicates relative humidity; SD indicates standard deviation.

Findings from the main analysis indicate that short-term increases in the concentrations of all of the air pollutants (PM_{10}, NO_2, SO_2, O_3, and NO), using the mean pollutant concentrations at lag 0–1 day, were associated with increased daily nonaccidental all-cause mortality. Associations between increases in the pollutant concentrations and respiratory mortality were positive (except for NO) but quite imprecise, and a few statistically significant associations were observed for some pollutants in mortality subgroups. While no pollutant was associated with increases in accidental mortality, all of the pollutants were significantly associated with increases in natural, non-cardiopulmonary mortality. Effect estimates (excess risk) were largest in people 65 or older and, except for SO_2, in those who died of cardiovascular causes and in those whose cause of death was termed "senility" (Vichit-Vadakan Investigators' Report Table 7a).

Commentary Table 3. Excess Risk of Mortality per 10-μg/m^3 Increase in Pollutants at Lag 0–1 Day[a]

Cause of Death (All Ages Unless Specified)	PM_{10} % ER (95% CI)	SO_2 % ER (95% CI)	NO_2 % ER (95% CI)	O_3 % ER (95% CI)	NO % ER (95% CI)
All natural	1.3 (0.8 to 1.7)	1.6 (0.1 to 3.2)	1.4 (0.9 to 1.9)	0.6 (0.3 to 0.9)	1.1 (0.6 to 1.6)
Cardiovascular	1.9 (0.8 to 3.0)	0.8 (−3.0 to 4.7)	1.8 (0.5 to 3.1)	0.8 (0.0 to 1.6)	2.0 (0.7 to 3.2)
Respiratory	1.0 (−0.4 to 2.4)	1.7 (−3.1 to 6.6)	1.0 (−0.6 to 2.7)	0.9 (−0.1 to 1.9)	−0.7 (−2.3 to 0.9)
Non-cardiopulmonary (nonaccidental)	1.2 (0.8 to 2.0)	1.8 (0.1 to 3.5)	1.4 (0.8 to 2.0)	0.6 (0.2 to 0.9)	1.1 (0.5 to 1.7)
Accidental	0.1 (−2.3 to 2.6)	0.0 (−8.0 to 8.8)	−0.1 (−3.0 to 2.8)	0.0 (−1.8 to 1.8)	0.8 (−2.0 to 3.6)
Age 65+ yr	1.5 (0.9 to 2.1)	2.8 (0.7 to 5.0)	1.8 (1.1 to 2.6)	0.8 (0.4 to 1.3)	1.3 (0.6 to 2.0)

[a] Definitions: CI indicates confidence interval; ER indicates excess risk.

Results from sensitivity analyses demonstrated that positive associations between individual pollutants and daily mortality in single-pollutant models were largely robust to differences in model specifications for the degree of smoothing for time, choice of regression spline (natural or penalized), and adjustment for confounders including "influenza." There was greater sensitivity, however, to inclusion of alternative lags for humidity and temperature on the effect on total (all natural) mortality (Vichit-Vadakan Investigators' Report Table 11), especially for NO_2 and NO. The effect of PM_{10} was also substantially reduced with longer-term weather lags but was still greater than zero, while those of SO_2 and O_3 were largely unaffected. In two-pollutant models (Vichit-Vadakan Investigators' Report Table 13), the association of PM_{10} with daily total (all natural) and cardiovascular mortality was relatively robust to adjustment for gaseous copollutants. However, the associations between the gaseous pollutants and daily mortality were generally sensitive to PM_{10} adjustment (Vichit-Vadakan Investigators' Report Table 12).

HEI EVALUATION OF THE STUDY

ASSESSMENT OF HEALTH OUTCOMES

The mortality health endpoints commonly used in time-series studies were also used in the Bangkok study. Hence, total mortality (after excluding "accidental" deaths) and the large strata of cardiovascular and respiratory deaths were included as endpoints. In addition, partly because of the large number of daily deaths in Bangkok, effects in a large number of additional cause-of-death strata were examined, specifically, ischemic heart disease, lower respiratory infection, COPD, and asthma. While the classification of cause of death into cardiopulmonary and noncardiopulmonary categories should be relatively valid, finer cause-of-death strata would be expected to be less so, complicating the discernment of ischemic heart disease from other causes of cardiovascular deaths.

The findings in this Bangkok study showing that the effect estimates were generally larger in the elderly (age 65 or older) and in those who died of cardiovascular causes were similar to those in most air pollution time-series studies. However, complicating the disease-specific findings in Bangkok, especially those involving cardiovascular causes of death, is the use of "senility" as a cause of death. The authors of the report speculate that the "senility" coding may be essentially capturing a category of cardiovascular deaths; their point is supported by the unusually low percentage of deaths due to cardiovascular disease captured by the coding of only "cardiovascular." Also, the pattern and size of the pollution effect estimates in the senility cause-of-death stratum mirror those in the cardiovascular stratum, further supporting the authors' speculation about the misclassification of cardiovascular deaths in the oldest age categories.

POLLUTANT CONCENTRATION MONITORING AND EXPOSURE ESTIMATION IN BANGKOK

Similar to the other PAPA studies, the Bangkok study relied on using routine monitoring data from the government-operated network, which is based on the U.S. Environmental Protection Agency (EPA) standard methods. The monitors are assessed, and the data are routinely checked by the local agency, though no additional evaluation (e.g., using collocated sampling for a limited period) was done for this study. Only four days were found to not have sufficient observations to calculate a daily average, and that was only for PM_{10}. For the main results, the investigators did not "center" concentrations when calculating daily means, which would have limited noise caused by variation in the pattern of missing values. The Committee considered this a limitation, though when centered values were used in the sensitivity analyses in this study (Vichit-Vadakan Investigators' Report Table 9), only the results for SO_2 changed appreciably. Similar to studies in many other cities, the Bangkok study found that observations of O_3 and PM_{10} at different monitor sites (Vichit-Vadakan Investigators' Report Table B.3 in Appendix B) tended to be highly correlated (Spearman correlations typically above 0.5, often around 0.8), while NO_2 was somewhat less so. SO_2 and NO tended to have low, and sometimes negative, correlations across monitors. This would suggest that assessment of area-wide exposures to SO_2 and NO, even using averages, was more likely to contain meaningful exposure measurement error. On the other hand, ambient exposures to O_3 and PM_{10} are likely to be well represented by the calculated averages because site-to-site correlations were high.

No additional calculations were done to help assess the potential exposure error for any of the pollutants or to see if the correlations between stations were dependent on distance or were influenced by sources of pollution and other land-use features near the monitors. The average calculated is used to represent exposure across the entire study area, and there is no consideration of the extent of or lack of agreement between the calculated averages and the observations at each monitor, as the investigators do not show how the averages used correlate with individual station data. Showing the statistical comparisons between area averages and individual monitor observations could have shed light on which stations are most representative

of the region and if one monitor had excessive influence on the results.

Correlations between the pollutant concentrations (Vichit-Vadakan Investigators' Report Table B.1) show that NO_2 and PM_{10} have the highest correlation (0.71), suggesting a common source (e.g., automotive emissions). O_3–NO_2 and O_3–PM_{10} concentrations are moderately correlated (0.62 and 0.55, respectively), suggesting the photochemical formation of a fraction of the $PM_{2.5}$ and the potential for limited confounding. NO and SO_2 are not highly correlated with any of the other pollutants, suggesting unique sources, local sources or conditions, and/or different atmospheric dynamics.

Unlike in most time series, NO was included as a pollutant of interest in Bangkok. In general, it is not known how the estimates of the health effects associated with NO are best interpreted. NO concentrations, much like carbon monoxide, are spatially very heterogeneous in urban areas due in large part to the much higher concentrations in proximity to roadways and to the fact that NO is rapidly converted to NO_2 in the presence of O_3. One would expect that averaging the daily concentrations of NO and NO_2 would result in considerable exposure measurement error, certainly to a greater extent than with the more spatially homogeneous pollutants such as PM and O_3.

Also similar to the other PAPA studies, this study had only a very brief characterization of the regional sources and dynamics of the pollutants. Further, there was no discussion of how specific sources may have impacted specific monitoring.

STATISTICAL METHODS

Aspects of the statistical methods that were common to all four PAPA studies in this phase are discussed in the Integrated Discussion (in Part 5 of this volume). Here we comment on the specifics of the statistical analysis in the Bangkok study.

The results of the sensitivity analyses of key results to the degrees of freedom (up to 15 df/year) in the time smoothers (see Vichit-Vadakan Investigators' Report Table 10) were reassuring, with little sensitivity identified. There was greater sensitivity to the inclusion of alternative lags (lags 1, 2, 3, 1–2 mean, or 3–7 mean) for humidity and temperature (Vichit-Vadakan Investigators' Report Table 11). For example, the excess risk from NO_2 was reduced from 1.4% (95% confidence interval [CI], 0.9–1.9) in the core model to 0.3% (95% CI, −0.3–0.8) with lag 3–7 day mean weather terms. The PM_{10} effect was more robust, but was also substantially reduced, from 1.3% (95% CI, 0.8–1.7) to 0.8% (95% CI, 0.3–1.3) with the inclusion of the longer weather lags. The effects of SO_2 and O_3 were largely unchanged.

This sensitivity to weather terms in the model was somewhat surprising, given the absence of much temperature variation in this tropical city. It may be that other aspects of weather patterns influence both mortality and pollution in the rainy season. A limitation of these sensitivity analyses was that the investigators replaced the lag 0 weather term with longer-lag terms, rather than adding the longer-lag terms to the model with the lag 0 term. We note that several investigators have found that distributed lag models (i.e., those that include several lags simultaneously) are needed to capture weather effects well (Braga et al. 2000; McMichael et al. 2008). Thus the Committee is concerned that even if a 0-day lag for temperature fits best out of the single-lag models considered, this does not exclude the possibility that other lags could additionally confound the pollution effects. The investigators also suggest that the longer lags for weather variables may be proxies for longer lags for pollution levels. This is plausible, but no more so than the possibility that there are longer-lag weather effects that confound the short-lag pollution effects. A way to address this would be to fit distributed lag models for both pollution and weather, which was not attempted in this report.

We agree that the Bangkok study team's other investigations of sensitivity showed broad robustness to their model choices. These included replacing the natural by a penalized spline, adding a first-order autocorrelation term (though there is no detail given on the method used for this), adjusting for influenza, and using a "centering" method to combine monitoring station daily concentrations. These sensitivity analyses provide confidence in the reported associations.

EPIDEMIOLOGIC ASPECTS

Features of Bangkok

As described by Vichit-Vakakan and coworkers, Bangkok is a relatively large city and therefore experiences a large number of deaths per day — on average, 95. This study, which included 5 years of data, has good statistical power consistent with all of the Asian cities in PAPA to detect statistically significant associations. This allows a realistic examination of stratified associations, such as those stratified by cause of death (including "control" causes of death) and age category. Relative to most cities in the West, the age distribution of the Bangkok population is young, and consequently so is the age distribution of deaths. However, a younger age distribution may reduce the size of the population at risk of dying from exposure to air pollution, especially if death is caused by cardiovascular disease, thereby effectively reducing the power to detect effects relative to

similarly sized Western cities. This may be particularly relevant when focusing on specific categories of cause of death where the daily counts are much smaller relative to mortality counts due to all natural causes.

Concentrations of air pollutants in Bangkok were high relative to those typically measured in Western cities, although not as high as in two of the three cities in China (Wuhan and Shanghai) also included in this first wave of PAPA studies. According to the investigators' report, the principal source of PM in Bangkok is vehicular emissions. The chemical composition of PM is largely determined by source contributions. To the extent that PM composition determines the type and severity of health effects attributed to PM exposure, PM effect estimates in Bangkok likely reflect the composition of motor-vehicle–related PM. Issues regarding the interpretation and relevance of health effects estimates from high-concentration settings are discussed in the Integrated Discussion, which covers all five PAPA reports and is found in Part 5.

Weather in Bangkok is characterized by temperatures and humidity that is high relative to that in Western cities, and the temperature and humidity in Bangkok are among the most extreme of the PAPA cities studied in this report. While high temperature is itself a risk factor for higher daily mortality (McMichael et al. 2008), there is also some evidence that temperature modifies the effect of air pollution on daily mortality, with higher temperatures enhancing the pollution mortality effect (Hu et al. 2008; Stafoggia et al. 2008). The findings from Wuhan in this set of PAPA studies provide some further support for such an interaction.

Pollutant-specific Effects and Robustness

The primary pollutant-related findings in the Bangkok time-series study were that associations with mortality from various causes were observed for all of the air pollutants analyzed (PM_{10}, NO_2, SO_2, NO, and O_3 in single-pollutant models [not adjusted for other pollutants]). For all of these pollutants, the estimates of relative risk per unit of pollutant concentration were higher than the averages in other cities in the United States (Dominici et al. 2005) and Europe (Katsouyanni 2003; HEI International Scientific Oversight Committee 2004) and in the Asian cities in the other PAPA studies (see Part 5. Combined Analysis in this volume). This was most pronounced for PM_{10}, for which the ER was 1.3% (95% CI, 0.8–1.7) in this analysis.

The effects of PM_{10} were largely insensitive to controlling for the gaseous pollutants in two-pollutant models. In contrast, the effects of NO_2 and SO_2 were largely sensitive to the adjustment for PM_{10}. However, even the effects of PM_{10}, as noted earlier in the discussion of the statistical methods, were sensitive to control for long temperature lags. These findings indicate that the mortality effects of short-term pollutants in Bangkok are dominated by effects from PM_{10}.

As discussed more fully in the accompanying Integrated Discussion (in Part 5 of this volume), the intent of multiple regression modeling is to allow the estimated effects of any single variable (e.g., the concentration of one pollutant) to be interpreted as the effect of that variable, controlled for the effects of the other variables (e.g., concentrations of other pollutants) included in the model. There are, however, obstacles to this interpretation of multiple regression effect estimates, including problems of model specification, collinearity resulting from strong correlations between concentrations of different air pollutants, differential impacts of exposure measurement error, and residual confounding across the different pollutants. Therefore, the extent to which multipollutant models, even two-pollutant models as used here, serve their intended purpose of allowing pollutant effect estimates to be interpreted independent of the effects of other pollutants is limited. If PM_{10} concentrations provide better exposure measures in the time-series study design than concentrations of the other pollutants (as they do in this study when compared with NO_2 and SO_2), the robustness of the PM_{10} effect to the inclusion of other pollutants may indicate less error in the exposure measurements rather than a dominant effect of PM_{10}.

Health Endpoints

Unlike in most time-series air pollution studies, there was no strong evidence for associations between exposure to the pollutants and overall respiratory mortality in the Bangkok study. It is not clear why there were no associations for overall respiratory mortality, but there are some possible explanations. Most respiratory mortality in Western countries is due to COPD (Kung et al. 2008). Since coded COPD deaths in Bangkok make up a much smaller proportion of respiratory deaths (only 25%) than in the other three cities, an association with overall respiratory mortality might not be seen if air pollution affected mainly this specific disease. The same situation would result if COPD deaths were miscoded as another category, such as "senility." The relatively small proportion of deaths coded as due to respiratory causes may also have led to imprecision in the resulting effect estimate. Substantial effects were seen for the subcategories of death due to asthma and for respiratory mortality in children less than 1 year old in this study, especially those associated with PM_{10}. It is also striking to note that the percentage of average daily deaths due to cardiovascular or respiratory causes in Bangkok is considerably lower than it is in the three Chinese cities

studied in the PAPA program, and the number of deaths attributed to these causes in the older ages is proportionately lower. Because of the very small number of daily deaths in these subcategories, one must be especially cautious in placing too much weight on these specific findings.

Analyzing the causes of death not anticipated to be due to short-term exposure to air pollution might be useful for assessing the adequacy of this study's methods, such as its pollutant exposure estimation or statistical modeling. Specifically, if pollutant effects are found in these cause-of-death strata, one might be more skeptical of the findings for causes of death for which such effects were anticipated. In this study, both "accidental" deaths and total non-cardiopulmonary deaths after excluding accidental deaths were used as "control" causes of death. No pollutant was associated with accidental causes of death, but increases in all of the pollutants were associated with increased non-cardiopulmonary deaths.

How are we to interpret the findings in the Bangkok study of associations for non-cardiopulmonary mortality with increases in all the pollutants? In general, such findings should prompt consideration of features of the study methods. First, are the data derived from the pollutant monitoring network adequate for the purpose of exposure estimation in the context of a time-series study? This question is relevant for all time-series studies; the Bangkok network does not appear to have unique features that set it apart from other networks that have been used for time-series studies. Second, is there evidence for residual confounding in the health models, or are the models poorly specified? It is possible, in this regard, that these effects do not persist after more aggressive control for meteorology, in which case it could be argued that residual confounding cannot be ruled out as the explanation for these effects. Finally, misclassification of the cause of death might have allowed a substantial number of cardiopulmonary deaths to have been incorrectly coded as due to a non-cardiopulmonary cause, in which case some effects on non-cardiopulmonary causes might be expected. This is the most likely explanation, particularly given the atypical distribution of cause of death exemplified by the large number of deaths attributed to senility.

CONCLUSION

Given the rigorous approach used by the investigators in carrying out the Bangkok study, together with the data collection and analysis protocol shared among these PAPA studies, these pollutant effect estimates for Bangkok are among the most reliable estimates currently available for Southeast Asia. The finding of an elevated risk per unit of PM_{10} is consistent with observations in most cities worldwide. Although the methods could still be improved on, and although residual uncertainties persist as outlined earlier, local policymakers now have more assurance that the estimation of pollutant health impacts in Bangkok is on a sound footing. The larger relative excess risk reported by Vichit-Vadakan and colleagues compared with the other PAPA cities is currently unexplained, and it would be premature to assume that this result reflects real differences in risk for the population of Bangkok rather than effects of data quality or analytic approach. As elaborated earlier, and in the Integrated Discussion, uncertainty in risk estimates is considerably greater than that reflected in the confidence intervals. Building on the strengths of this study, a better appreciation of these uncertainties will help focus future research in Bangkok and elsewhere.

ACKNOWLEDGMENTS

The Health Review Committee thanks the ad hoc reviewers for their help in evaluating the scientific merit of the Investigators' Report. The Committee is also grateful to Wei Huang and Sumi Mehta for their oversight of the study, to Kate Adams, Amara Ezeamama, Geoffrey Sunshine, and Marian Berkowitz for their assistance in preparing its Commentary, to Hilary Selby Polk for science editing of this Report and its Commentary, and to Flannery Carey McDermott, Fred Howe, and Bernard Jacobson for their roles in preparing this Research Report for publication.

REFERENCES

Anderson HR, Ponce de Leon A, Bland JM, Bower JS, Strachan DP. 1996. Air pollution and daily mortality in London: 1987–92. BMJ 312:665–669.

Braga A, Zanobetti A, Schwartz J. 2000. Do respiratory epidemics confound the association between air pollution and daily deaths? Eur Respir J 16:732–728.

Dominici F, McDermott A, Daniels M, Zeger SL, Samet JM. 2005. Revised analyses of the National Morbidity, Mortality, and Air Pollution Study: Mortality among residents of 90 cities. J Toxicol Environ Health A 68:1071–1092.

HEI International Scientific Oversight Committee. 2004. Health Effects of Outdoor Air Pollution in Developing Countries of Asia: A Literature Review. Special Report 15. Health Effects Institute, Boston, MA.

Hu W, Mengersen K, McMichael A, Tong S. 2008. Temperature, air pollution and total mortality during summers in Sydney, 1994–2004. Int J Biometeorol 52:689–696.

Jinsart W, Tamura K, Loetkamonwit S, Thepanondh S, Karita K, Yano E. 2002. Roadside particulate air pollution in Bangkok. J Air Waste Manag Assoc 52:1102–1110.

Katsouyanni K. 2003. Ambient air pollution and health. Br Med Bull 68:143–156.

Kinney PL, Ozkaynak H. 1991. Associations of daily mortality and air pollution in Los Angeles County. Environ Res 54:99–120.

Kung HC, Hoyert DL, Xu J, Murphy SL; Division of Vital Statistics. 2008. Deaths: Final Data for 2005. National Vital Statistics Reports, Vol. 56. U.S. Department of Health and Human Services. Available at *www.cdc.gov/nchs/data/nvsr/nvsr56/nvsr56_10.pdf.* Accessed 7/29/10.

McMichael AJ, Wilkinson P, Kovats RS, Pattenden S, Hajat S, Armstrong B, Vajanapoom N, Niciu EM, Mahomed H, Kingkeow C, Kosnik M, O'Neill MS, Romieu I, Ramirez-Aguilar M, Barreto ML, Gouveia N, Nikiforov B. 2008. International study of temperature, heat and urban mortality: The "ISOTHURM" project. Int J Epidemiol 37:1121–1131.

Sáez M, Tobias A, Muñoz P, Campbell MJ. 1999. A GEE moving average analysis of the relationship between air pollution and mortality for asthma in Barcelona, Spain. 30:2077–2086.

Stafoggia M, Schwartz J, Forastiere F, Perucci CA; SISTI Group. 2008. Does temperature modify the association between air pollution and mortality? A multicity case-crossover analysis in Italy. Am J Epidemiol 167:1476–1485.

Sunyer J, Castellsagué J, Sáez M, Tobias A, Antó JM. 1996. Air pollution and mortality in Barcelona. 50 Suppl 1:s76–s80.

Touloumi G, Katsouyanni K, Zmirou D, Schwartz J, Spix C, de Leon AP, Tobias A, Quennel P, Rabczenko D, Bacharova L, Bisanti L, Vonk JM, Ponka A. 1997. Short-term effects of ambient oxidant exposure on mortality: A combined analysis within the APHEA project. Air Pollution and Health: A European Approach. 146:177–185.

Wietlisbach V, Pope CA III, Ackermann-Liebrich U. 1996. Air pollution and daily mortality in three Swiss urban areas. 41:107–115.

HEALTH
EFFECTS
INSTITUTE

101 Federal Street, Suite 500
Boston, MA 02110, USA
+1-617-488-2300
www.healtheffects.org

RESEARCH
REPORT

Number 154
November 2010