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Impact of the 1990 Hong Kong Legislation for Restriction on Sulfur Content in Fuel

Chit-Ming Wong, Ari Rabl, Thuan Q. Thach,
Yuen Kwan Chau, King Pan Chan, Benjamin J. Cowling,
Hak Kan Lai, Tai Hing Lam, Sarah M. McGhee,
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Includes a Commentary by the Institute's Health Review Committee

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Research Report 170

ABOUT HEI

The Health Effects Institute is a nonprofit corporation chartered in 1980 as an independent research organization to provide high-quality, impartial, and relevant science on the effects of air pollution on health. To accomplish its mission, the institute

- Identifies the highest-priority areas for health effects research;
- Competitively funds and oversees research projects;
- Provides intensive independent review of HEI-supported studies and related research;
- Integrates HEI's research results with those of other institutions into broader evaluations; and
- Communicates the results of HEI's research and analyses to public and private decision makers.

HEI typically receives half of its core funds from the U.S. Environmental Protection Agency and half from the worldwide motor vehicle industry. Frequently, other public and private organizations in the United States and around the world also support major projects or research programs. HEI has funded more than 280 research projects in North America, Europe, Asia, and Latin America, the results of which have informed decisions regarding carbon monoxide, air toxics, nitrogen oxides, diesel exhaust, ozone, particulate matter, and other pollutants. These results have appeared in the peer-reviewed literature and in more than 200 comprehensive reports published by HEI.

HEI's independent Board of Directors consists of leaders in science and policy who are committed to fostering the public–private partnership that is central to the organization. The Health Research Committee solicits input from HEI sponsors and other stakeholders and works with scientific staff to develop a Five-Year Strategic Plan, select research projects for funding, and oversee their conduct. The Health Review Committee, which has no role in selecting or overseeing studies, works with staff to evaluate and interpret the results of funded studies and related research.

All project results and accompanying comments by the Health Review Committee are widely disseminated through HEI's Web site (*www.healtheffects.org*), printed reports, newsletters and other publications, annual conferences, and presentations to legislative bodies and public agencies.

ABOUT THIS REPORT

Research Report 170, *Impact of the 1990 Hong Kong Legislation for Restriction on Sulfur Content in Fuel*, presents a research project funded by the Health Effects Institute and conducted by Chit-Ming Wong of the Department of Community Medicine, School of Public Health, The University of Hong Kong, China, and his colleagues. This report contains three main sections.

The HEI Statement, prepared by staff at HEI, is a brief, nontechnical summary of the study and its findings; it also briefly describes the Health Review Committee's comments on the study.

The Investigators' Report, prepared by Wong and his colleagues, describes the scientific background, aims, methods, results, and conclusions of the study.

The Commentary is prepared by members of the Health Review Committee with the assistance of HEI staff; it places the study in a broader scientific context, points out its strengths and limitations, and discusses remaining uncertainties and implications of the study's findings for public health and future research.

This report has gone through HEI's rigorous review process. When an HEI-funded study is completed, the investigators submit a draft final report presenting the background and results of the study. This draft report is first examined by outside technical reviewers and a biostatistician. The report and the reviewers' comments are then evaluated by members of the Health Review Committee, an independent panel of distinguished scientists who have no involvement in selecting or overseeing HEI studies. During the review process, the investigators have an opportunity to exchange comments with the Review Committee and, as necessary, to revise their report. The Commentary reflects the information provided in the final version of the report.

PREFACE

HEI's Outcomes Research Program

The goal of most air quality regulations is to protect the public's health by implementing regulatory actions or providing economic incentives that help reduce the public's exposure to air pollutants. If this goal is met, air pollution should be reduced, and indicators of public health should improve or at least not deteriorate. Evaluating the extent to which air quality regulations succeed in protecting public health is part of a broader effort — variously termed *outcomes research*, *accountability research*, or *research on regulatory effectiveness* — designed to assess the performance of environmental regulatory policies in general. In recent decades, air quality in the United States and Western Europe has improved substantially, and this improvement is attributable to a number of factors, including increasingly stringent air quality regulations. However, the cost of the pollution-control technologies and mechanisms needed to implement and enforce these regulations is often high. It is therefore prudent to ask whether the regulations have in fact yielded demonstrable improvements in public health, which will provide useful feedback to inform future efforts.

Several U.S. government agencies have concluded that direct evidence about the extent to which air quality regulations have improved health (measured as a decrease in premature mortality and excess morbidity) is lacking. This finding is well documented by the National Research Council (NRC) in its report *Estimating the Public Health Benefits of Proposed Air Pollution Regulations* (NRC 2002), as well as by the California Air Resources Board, the U.S. Environmental Protection Agency (EPA), the U.S. Centers for Disease Control and Prevention (CDC), and other agencies.

In 2003, the Health Effects Institute published a monograph on outcomes research, Communication 11, *Assessing Health Impact of Air Quality Regulations: Concepts and Methods for Accountability Research* (HEI 2003). This monograph was written by the members of HEI's multidisciplinary Accountability Working Group after a 2001 workshop on the topic. Communication 11 set out a conceptual framework for outcomes research and identified the types of evidence required and the methods by which the evidence should be obtained. It has also guided the development of the HEI Health Outcomes Research program, which is discussed below.

Between 2002 and 2004, HEI issued four requests for applications (RFAs) for studies to evaluate the effects of actions taken to improve air quality. The study by Dr. Chit-Ming Wong and colleagues described in this Research Report (Wong et al. 2012) was funded under RFA 04-4, "Measuring the Health Impact of Actions Taken to Improve Air Quality." HEI funded eight additional outcomes studies resulting from this and other RFAs (see Preface Table).

This preface describes both the framework of outcomes research as it relates to air quality regulations and HEI's Outcomes Research program.

BACKGROUND

The first step in assessing the effectiveness of air quality regulations is to measure emissions of the targeted pollutants to see whether they have in fact decreased as intended. A series of intermediate assessments, described in detail below, are needed in order to accurately measure the adverse health effects associated with air pollution to see whether they, too, decreased in incidence or severity relative to emissions. Some outcomes studies to date have used hypothetical scenarios (comparing estimated outcomes under existing and more stringent regulations) and risk estimates obtained from epidemiologic studies in an attempt to quantify past effects on health and to predict future effects (U.S. EPA 1999). However, more extensive validation of these estimates with data on actual outcomes would be helpful.

The long-term improvements in U.S. air quality have been associated with improved health in retrospective epidemiologic studies (Chay and Greenstone 2003;

HEI's Outcomes Research Program[a]

RFA / Investigator (Institution)	Study or Report Title	Intervention
RFA 02-1		
Douglas Dockery (Harvard School of Public Health, Boston, MA)	"Effects of Air Pollution Control on Mortality and Hospital Admissions in Ireland" (in press)	Coal ban in Irish cities
Annette Peters (GSF–National Research Center for Environment and Health, Neuherberg, Germany[b])	The Influence of Improved Air Quality on Mortality Risks in Erfurt, Germany (published as Research Report 137, 2009)	Switch from brown coal to natural gas for home heating and power plants, changes in motor vehicle fleet after reunification of Germany
RFA 04-1		
Frank Kelly (King's College London, London, U.K.)	The Impact of the Congestion Charging Scheme on Air Quality in London: Part 1. Emissions Modeling and Analysis of Air Pollution Measurements. Part 2. Analysis of the Oxidative Potential of Particulate Matter (published as Research Report 155, 2011)	Measures to reduce traffic congestion in the inner city of London
RFA 04-4		
Frank Kelly (King's College London, London, U.K.)	The London Low Emission Zone Baseline Study (published as Research Report 163, 2011)	Measures to exclude most polluting vehicles from entering greater London
Richard Morgenstern (Resources for the Future, Washington, DC)	"Accountability Assessment of Title IV of the Clean Air Act Amendments of 1990" (in press)	Measures to reduce sulfur emissions from power plants east of the Mississippi River
Curtis Noonan (University of Montana, Missoula, MT)	Assessing the Impact of a Wood Stove Replacement Program on Air Quality and Children's Health (published as Research Report 162, 2011)	Woodstove change-out program
Jennifer Peel (Colorado State University, Fort Collins, CO)	Impact of Improved Air Quality During the 1996 Summer Olympic Games in Atlanta on Multiple Cardiovascular and Respiratory Outcomes (published as Research Report 148, 2010)	Measures to reduce traffic congestion during the Atlanta Olympics
Chit-Ming Wong (University of Hong Kong, Hong Kong)	"Impact of the 1990 Hong Kong Legislation for Restriction on Sulfur Content in Fuel" (published as Research Report 170, 2012)	Measures to reduce sulfur content in fuel for motor vehicles and power plants
RFPA 05-3		
Junfeng (Jim) Zhang (University of Medicine and Dentistry of New Jersey, Piscataway, NJ)	"Molecular and Physiological Responses to Drastic Changes in PM Concentration and Composition" (in press)	Measures to improve air quality during the Beijing Olympics

[a] Abbreviations: RFA, Request for Applications; RFPA, Request for Preliminary Applications.

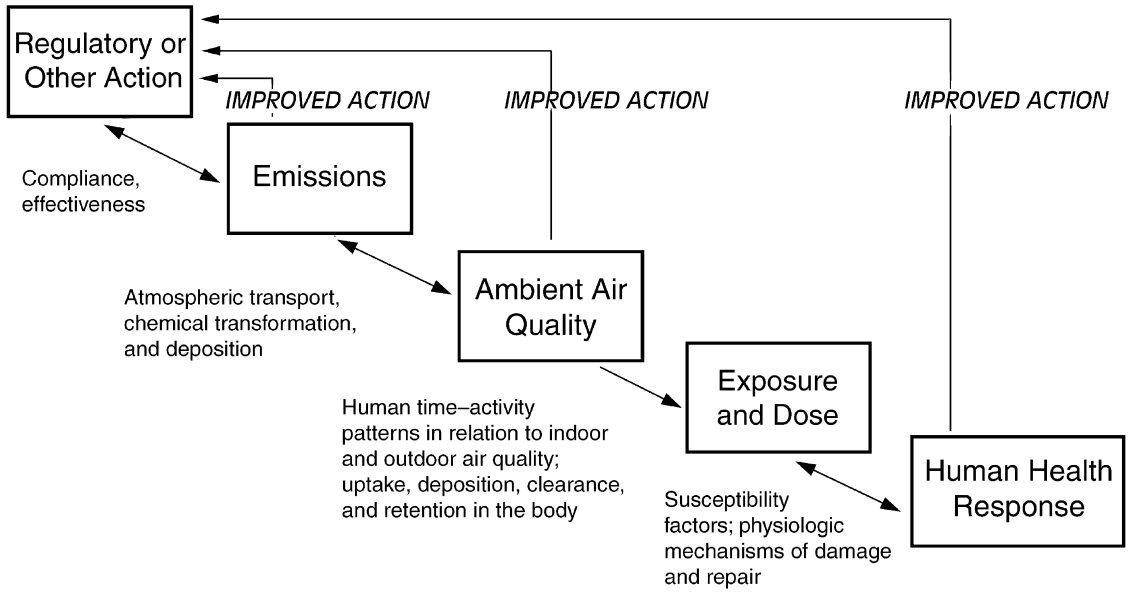
[b] As of 2008, this institution has been called the Helmholtz Zentrum München–German Research Center for Environmental Health.

Laden et al. 2006; Pope et al. 2009). Considerable challenges, however, are inherent in the assessment of the health effects of air quality regulations. Different regulations go into effect at different times, for example, and may be implemented at different levels of government (e.g., national, regional, or local). Their effectiveness therefore needs to be assessed in ways that take into account the varying times of implementation and levels of regulation. In addition, other changes at the same time and place might confound an apparent association between pollution reduction and improved health, such as economic trends (e.g., changes in employment), improvements in health care, and behavioral changes (e.g., staying indoors when government warnings indicate pollution concentrations are high). Moreover, adverse health effects that might have been caused by exposure to air pollution can also be caused by other environmental risk factors (some of which may have changed over the same time periods as the air pollution concentrations). These challenges become more pronounced when regulations are implemented over long periods and when changes in air quality and health outcomes are not seen immediately, thus increasing the chance for confounding by other factors. For these reasons, scenarios in which regulations are expected to have resulted in rapid changes in air quality tend to be among the first, and most likely, targets for investigation, rather than evaluations of complex regulatory programs implemented over multiple years. Studies in Ireland by Clancy and colleagues (2002) and in Hong Kong by Hedley and colleagues (2002) are examples of such scenarios.

These inherent challenges are well documented in Communication 11 (HEI 2003), which was intended to advance the concept of outcomes research and to foster the development of methods and studies throughout the relevant scientific and policy communities. In addition, recent advances in data collection and analytic techniques provide an unprecedented opportunity to improve our assessments of the effects of air quality interventions.

THE OUTCOMES EVALUATION CYCLE

The NRC's Committee on Research Priorities for Airborne Particulate Matter set out a conceptual framework for linking air pollution sources to adverse health effects (NRC 1998). This framework can be used to identify factors along an "outcomes evaluation cycle" (see Figure below), each stage of which affords



Outcomes Evaluation Cycle. Each box represents a stage in the process between regulatory action and human health responses to air pollution. Arrows connecting the stages indicate possible directions of influence. The text below the arrows identifies factors affecting the effectiveness of regulatory actions at each stage. At several of the stages, knowledge gained from studies on outcomes can provide valuable feedback for improving regulatory or other actions.

its own opportunities for making quantitative measurements of the intended improvements.

At the first stage (regulatory action), one can assess whether controls on source emissions have in fact been put into place. At the second stage (emissions), one can determine whether controls on sources have indeed reduced emissions, whether emitters have changed their practices, and whether there have been unintended consequences. At the third stage (ambient air quality), one can assess whether controls on sources and reductions in emissions have resulted in improved air quality. At the fourth stage (personal or population exposure), one can assess whether the improvement in air quality has reduced people's actual exposure and whether susceptible subpopulations (those most likely to experience adverse health effects) have benefited. At this stage, it is important to take into account changes in time–activity patterns that could either increase or reduce exposure. The actual dose that an individual's organs may be exposed to should also be considered (i.e., whether reductions in exposure have led to reductions in concentrations in body tissues such as the lung). Finally, at the fifth stage (human health response), one can assess whether risks to health have declined, given the evidence about changes in health outcomes such as morbidity and mortality that have resulted from changes in exposure. The challenge at this stage is to investigate the health outcomes that are most directly related to exposure to air pollution.

At each stage in the outcomes evaluation cycle, the opportunity exists to collect evidence that either validates the assumptions that motivated the intervention or points to ways in which the assumptions were incorrect. The collection of such evidence can thus ensure that future interventions are maximally effective.

Ultimately, the framework for outcomes research will need to encompass investigations of the broader consequences of regulations, not just the intended consequences. Unintended consequences should also be investigated, along with the possibility that risks to public health in fact increased, as discussed by Wiener (1998) and others who have advanced the concept of a portfolio of effects of a regulation.

HEI'S OUTCOMES RESEARCH PROGRAM

HEI's Outcomes Research program currently includes nine studies. The study by Dr. Chit-Ming Wong and colleagues presented in this report is the sixth to be published. The remaining three studies are in press and are expected to be published in 2012 and 2013.

These studies involve the measurement of indicators along the entire outcomes evaluation cycle, from regulatory or other interventions to human health outcomes. Some of the studies focused on interventions that are implemented over relatively short periods of time, such as a ban on the sale of coal, the replacement of old wood stoves with more efficient, cleaner ones, reductions in the sulfur content of fuels, and measures to reduce traffic. Other groups focused on longer-term, wider-ranging interventions or events; for instance, one study assessed complex changes associated with the reunification of the former East and West Germany, including a switch from brown coal to natural gas for fueling power plants and home-heating systems and an increase in the number of modern diesel-powered vehicles in eastern Germany. HEI is also supporting research, including the development of methods, in an especially challenging area, namely, assessment of the effects of regulations implemented incrementally over extended periods of time, such as those resulting from Title IV of the 1990 Clean Air Act Amendments (U.S. EPA 1990), which aimed at reducing sulfur dioxide emissions from power plants by requiring compliance with prescribed emission limitations. Studies on health outcomes funded by HEI to date are summarized in the Preface Table and described in more detail in an interim evaluation of the HEI Outcomes Research program (van Erp and Cohen 2009).

FUTURE DIRECTIONS

As a part of its Strategic Plan for 2010 through 2015 (HEI 2010a), HEI has looked closely at opportunities for unique new contributions to health outcomes research. Key recommendations for future research were made at a December 2009 planning workshop (HEI 2010b), which led to HEI issuing a new Request for Applications in January 2011 for a second wave of outcomes research. RFA 11-1, "Health Outcomes Research — Assessing the Health Outcomes of Air Quality Actions," solicited applications for studies designed to assess the health effects of actions to improve air quality and to develop methods required for, and specifically suited to, conducting such research. Recently, HEI approved two studies that will evaluate

regulatory and other actions at the national or regional level implemented over multiple years; one study will evaluate complex sets of actions targeted at improving air quality in large urban areas and major ports with well-documented air quality problems and programs to address them, and the other study will develop methods to support such health outcomes research. These studies will be starting in 2012.

In addition, HEI has funded the development of two Web sites intended to enhance transparency and provide other researchers with access to extensive data and software from HEI-funded studies:

1. Data and software from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), as described by Zeger and colleagues (2006) (data available at the Johns Hopkins Bloomberg School of Public Health Web site *www.ihapss.jhsph.edu*); and
2. Data from the National Particle Component Toxicity Initiative (NPACT) on concentrations of components of particulate matter with an aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$) collected at or near the 54 sites in the EPA's $PM_{2.5}$ Chemical Speciation Trends Network (STN) (data available at the Atmospheric and Environmental Research Web site *https://hei.aer.com*).

The data on pollution and health from a large number of U.S. cities, as documented by the NMMAPS team and made available on the Internet-Based Health and Air Pollution Surveillance System (iHAPSS) Web site, constitute a valuable resource that allows other researchers to undertake additional analyses, possibly including further outcomes studies. The STN Web site provides scientists an opportunity to investigate specific questions about concentrations of $PM_{2.5}$ components and their association with adverse health effects in regions covered by the STN network and to address questions related to outcomes research when interventions in these regions are being planned.

In January 2008, HEI co-organized and cosponsored, with the CDC's National Environmental Public Health Tracking Program and the EPA, a workshop titled "Methodologic Issues in Environmental Public Health Tracking of Air Pollution Effects." The workshop was part of an effort to implement the initiative outlined in HEI's Strategic Plan for 2005 through 2010 (HEI 2005) to "build networks with the U.S. Centers for Disease Control and Prevention and state public health tracking programs to facilitate accountability research."

The workshop built on the work of the CDC's National Environmental Public Health Tracking Program (see the CDC Web site *www.cdc.gov/nceh/tracking/*) in the development of standardized measures of air pollution–related effects on health at the state and local levels in the United States. It brought together representatives of state and federal agencies and academic researchers to discuss methodologic issues in developing standardized measures and made recommendations for their further development and application in assessing the health impacts of air pollution, including the impacts of actions taken to improve air quality. The recommendations were provided in a September 2008 report to the CDC, and the proceedings were published in the journal *Air Quality, Atmosphere & Health* in December 2009 (Matte et al. 2009). The CDC has subsequently funded a pilot project under the National Environmental Public Health Tracking Program to implement the recommendations of the workshop in selected states and metropolitan areas.

HEI will continue to seek opportunities to work with the CDC and the EPA to apply methods newly developed for tracking public health and assessing the effectiveness of environmental regulations.

Investigators who have identified a distinctive opportunity to evaluate the effects of environmental regulations on air pollution and human health are encouraged to contact HEI.

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HEI STATEMENT

Synopsis of Research Report 170

Impact of the 1990 Hong Kong Legislation for Restriction on Sulfur Content in Fuel

INTRODUCTION

On July 1, 1990, the government in Hong Kong implemented a new restriction on sulfur in fuel, mandating a limit of 0.5% sulfur by weight. After the full impact of this regulation was realized, airborne sulfur dioxide (SO_2) concentrations were reduced by 45% on average and by as much as 80% in some districts, although other components of the pollutant mixture, including particle mass, measured as particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}), did not decline. The reductions in the SO_2 concentrations were estimated in previous studies to have resulted in health improvements, including decreases in mortality rates and improvements in life expectancy.

The current study by Dr. Chit-Ming Wong of The University of Hong Kong and colleagues aimed to extend this earlier work in two ways: First, the investigators proposed to explore the role that specific chemical constituents of particulate air pollution may have played in the effects on mortality of the 1990 Hong Kong restriction of sulfur in fuels, hereafter referred to as "the intervention." They also proposed to develop methods for estimating the impact on life expectancy of an improvement in air quality after the imposition over a brief interval of a change in fuel quality, and to apply these methods in the context of the intervention. These objectives entailed evaluating the effects on mortality due to short-term changes in air quality after the intervention (specifically changes in levels of particle and gaseous pollutants and in particular chemical components or "species" of particulate air pollution); developing new and improved methods for assessing the health impact, in terms of the change in life expectancy, resulting from interventions taken to improve air quality; and determining whether or not improvements in air quality had any relation to short-term and long-term health benefits.

APPROACH

The investigators obtained counts of daily deaths from specific causes for the Hong Kong population between January 1, 1985, and June 30, 1995, a period that extends from 5 years before to 5 years after the imposition of the sulfur reduction. They also obtained measured ambient concentrations of air pollutants from the Hong Kong Environmental Protection Department from a network of 13 stations that operated in Hong Kong over the 10-year period of the study. Daily average concentrations of the gaseous pollutants nitrogen dioxide (NO_2), SO_2, and ozone (O_3) were derived from hourly measurements. Data on PM_{10} and associated chemical species were measured on every third and sixth day, respectively. Concentrations of specific chemical species were estimated by chemical analysis of the PM_{10} collected in filters using the gravimetric method. The investigators obtained daily mean temperature (°C) and relative humidity (%) data from the Hong Kong Observatory.

The investigators compared mean levels of pollutants between pre- and post-intervention periods using standard statistical methods (*t* tests and analysis of variance) to assess mean differences in concentrations. The investigators used Poisson regression methods to estimate the effects of short-term exposure to air pollution on daily mortality rates for all natural causes and for cardiovascular and respiratory causes, at all ages and in the 65-years-or-older age group, taking into account long-term trends and seasonal variation in daily mortality rates and variation in temperature and relative humidity. They also compared alternative models using both statistical tests and visual inspection of results.

The regression models were used to estimate the effects of gaseous pollutants, PM_{10}, and various associated chemical species on daily mortality rates for the periods 5 years before and 5 years after the

This Statement, prepared by the Health Effects Institute, summarizes a research project funded by HEI and conducted by Dr. Chit-Ming Wong at The University of Hong Kong, Hong Kong, China, and colleagues. Research Report 170 contains both the detailed Investigators' Report and a Commentary on the study prepared by the Institute's Health Review Committee.

intervention, as well as in the 10-year period pre- and post-intervention. They also conducted sensitivity analyses in which the pre-intervention time periods were shortened and ran analyses in which indicator variables were used to represent the intervention. All effects were reported as excess risks (ERs), defined as the percentage change in mortality per 10-$\mu g/m^3$ and 10-ng/m^3 increase in the concentrations of the four criteria pollutants and PM-associated chemical species, respectively. ERs were estimated for exposure cumulated over the day of death and the preceding day and for exposure to chemical species on the day of death.

The investigators developed two methods to estimate the effect of the intervention on life expectancy, which differ with regard to the outcome variable. In the first method, they used the daily age-standardized mortality rate, and in the second, the daily mortality count. Both methods were used to evaluate the relation between outcome variables and daily air pollution concentrations in the current day back to all previous days in the past 3 to 4 years.

RESULTS

The investigators included 275,254 deaths between 1985 and 1995 in the study, after excluding records that did not meet quality control specifications. They reported that the average daily number of deaths from all natural causes for all ages was 69 in the 5-year pre-intervention period and 76 in the 5-year post-intervention period. Cardiovascular and respiratory mortality comprised approximately 30% and 18% of total mortality, respectively, in both periods.

The investigators reported decreases in NO_2 and SO_2 concentrations between pre-intervention and post-intervention periods, which were particularly pronounced in the more heavily polluted industrial areas, but no consistent changes in PM_{10} concentrations after the intervention. The investigators reported decreases in each of seven PM_{10}-associated chemical species: aluminum, iron, manganese, nickel, vanadium, lead, and zinc between pre- and post-intervention periods. Of these, the reductions in nickel and vanadium were the most consistent and statistically significant.

The investigators analyzed the relation between short-term exposure to air pollution and daily mortality using data for the entire 10-year period from 1985 to 1995, and reported increased ERs for mortality due to all natural causes for both SO_2 and NO_2, and for mortality from cardiovascular causes for SO_2 and respiratory causes for NO_2. O_3 was also associated with an increase in deaths due to all natural causes and respiratory disease. Neither PM_{10} nor most PM_{10}-associated chemical species were consistently or statistically associated with increased ER. However, the investigators reported that both nickel and vanadium were associated with an increased ER from respiratory causes, especially in those older than 65 years.

The investigators reported that only NO_2 was associated with increased daily mortality in the pre-intervention period. This association was particularly pronounced for respiratory causes of death in those older than 65 years. Post-intervention, they reported that the various gaseous pollutants were associated with an increased ER of mortality from all natural causes and from cardiovascular and respiratory diseases. However, the investigators reported no statistically significant associations between PM_{10} and mortality in either the pre- or post-intervention periods, and little evidence of an association in the pre-intervention period between mortality and exposure to most of the chemical species. Nickel and vanadium, however, were associated with mortality from both all natural causes and respiratory disease, especially in those older than 65 years. In the post-intervention period, the investigators reported that most ERs for the individual chemical species were lower than in the pre-intervention period, though the ER of nickel for mortality due to all natural causes was higher in the 5-year pre-intervention period for both age groups.

The investigators reported that most gaseous pollutants were associated with increases in the ER of mortality after the intervention. The most pronounced increases were associated with NO_2 and SO_2 on cardiovascular mortality and for NO_2 and O_3 on respiratory mortality. They also observed that neither PM_{10} nor most chemical species were consistently associated with changes in ER, although zinc and manganese showed some evidence of associations with cardiovascular mortality. However, both nickel and vanadium were associated with a decline in ER of mortality, a finding that was replicated in a sensitivity analyses.

The investigators reported estimates of effects on life expectancy due to air pollution exposure over a 21-year period from 1985 through 2005. They stated that although some analyses suggested a relatively rapid but unsustained increase in life expectancy

associated with the intervention, they were unable to reliably estimate the effect of the intervention on life expectancy using approaches based on either the daily age-standardized mortality rate or the daily mortality count, because they could not adequately adjust for the effects of season on mortality or for the effects of long-term trends in mortality rates that had occurred in the population of Hong Kong.

INTERPRETATION AND CONCLUSIONS

The reduction in the sulfur content of fuel in Hong Kong, implemented over a relatively brief period in 1990, and the health effects associated with it have been the subject of several epidemiologic studies. The current study by Wong and colleagues explored the effects of the reduction in the sulfur content of fuel on mortality in more detail, taking advantage of the marked step change in a major source of air pollution, an extended 10-year period of observation of both air pollution and mortality in Hong Kong, and additional, detailed measurements of specific chemical constituents of PM_{10} and gaseous pollutants. The extended period of observation also afforded Wong and colleagues the opportunity to develop and apply innovative methods to estimate the effects of reduction in the sulfur content of fuel on life expectancy using daily mortality rates, a controversial approach with potential applications to similar interventions. Dr. Wong's team was particularly well suited to carry out this ambitious research agenda, building on their extensive prior work on air quality and health in Hong Kong. However, they met with, at best, limited success.

The investigators applied conventional analytic methods for the analysis of daily time-series data and reported associations over a 10-year period from 1985 to 1995. They reported estimates for the effects of the gaseous pollutants and PM_{10} that are broadly consistent, both in direction and magnitude if not statistical precision, with many previous studies. The analyses of PM-associated chemical species indicated particularly strong associations between mortality and exposure to the specific chemical components nickel and vanadium, constituents derived mainly from the combustion of bunker fuels with high sulfur content used in marine shipping. However, the current study has several important weaknesses that limit its contribution to knowledge about the effects of this much-studied intervention.

Although the investigators observed reductions in PM-associated nickel and vanadium concentrations associated with the intervention and reported adverse effects of short-term exposure to both of these components, they were unable to reliably link changes in their concentrations associated with the intervention to changes in the effects of short-term exposure in the pre- and post-intervention periods. The investigators acknowledge this and attribute their inability to demonstrate any effects of the intervention per se to the fact that nickel and vanadium concentrations were measured only on every sixth day. But other weaknesses may also be responsible.

The comparison of estimated pollution effects between models (in particular between pre- and post-intervention models) was complicated by different specifications of model components for time trends and temperature, and it is not clear how sensitive the results were to these alternative specifications. Moreover, the monitoring data reveal considerable spatial variation in concentrations of the various pollutants across Hong Kong and also considerable spatial differences in the temporal variation of air pollution levels among them. These uncertainties suggest that only tentative conclusions can be drawn from these time-series analyses.

In addition, the investigators were unable to disentangle the effects of individual pollutants, both gases and components in the particle fraction, on mortality over the 10-year period of the study. After adjustment for levels of nickel and vanadium, the associations with SO_2, which had provided the strongest evidence for an effect of the intervention, became statistically unstable. These analyses make clear that the effects of SO_2, nickel, and vanadium could not be separated in the current study and likely cannot be separated using the current set of data and analytic methods given the correlations among these pollutants. Perhaps this is not surprising given the common combustion sources of these pollutants (e.g., sulfur-rich bunker fuel used in marine shipping). The investigators conclude that they "cannot exclude the possibility that decreases in their concentrations were responsible for some of the observed health benefits due to the intervention." However, based on these results, it is not possible to confidently attribute such beneficial effects on mortality as may have occurred due to the intervention to any specific component of the air pollution mixture.

Wong and colleagues did conduct the first rigorous effort to estimate from daily time-series data the

effects of long-term exposure on life expectancy. The theoretical basis for such estimates had been explored previously and appeared to offer some promise, and the long-term time-series data associated with the Hong Kong intervention seemed to present an ideal opportunity for practical application. Unfortunately, because of the inability to control for the effects of potential confounding factors correlated over the long term with air pollution, the results appear to offer little hope for estimating the effects on life expectancy from daily time-series data for use in scientific or policy applications.

INVESTIGATORS' REPORT

Impact of the 1990 Hong Kong Legislation for Restriction on Sulfur Content in Fuel

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ABSTRACT

INTRODUCTION

After the implementation of a regulation restricting sulfur to 0.5% by weight in fuel on July 1, 1990, in Hong Kong, sulfur dioxide (SO_2*) levels fell by 45% on average and as much as 80% in the most polluted districts (Hedley et al. 2002). In addition, a reduction of respiratory symptoms and an improvement in bronchial hyperresponsiveness in children were observed (Peters et al. 1996; Wong et al. 1998). A recent time-series study (Hedley et al. 2002) found an immediate reduction in mortality during the cool season at six months after the intervention, followed by an increase in cool-season mortality in the second and third years, suggesting that the reduction in pollution was associated with a delay in mortality. Proportional changes in mortality trends between the 5-year periods before and after the intervention were measured as relative risks and used to assess gains in life expectancy using the life table method (Hedley et al. 2002). To further explore the relation between changes in pollution-related mortality before and after the intervention, our study had three objectives: (1) to evaluate the short-term effects on mortality of changes in the pollutant mix after the Hong Kong sulfur intervention, particularly with changes in the particulate matter (PM) chemical species; (2) to improve the methodology for assessment of the health impact in terms of changes in life expectancy using linear regression models; and (3) to develop an approach for analyzing changes in life expectancy from Poisson regression models. A fourth overarching objective was to determine the relation between short- and long-term benefits due to an improvement in air quality.

METHODS

For an assessment of the short-term effects on mortality due to changes in the pollutant mix, we developed Poisson regression Core Models with natural spline smoothers to control for long-term and seasonal confounding variations in the mortality counts and with covariates to adjust for temperature (T) and relative humidity (RH). We assessed the adequacy of the Core Models by evaluating the results against the Akaike Information Criterion, which stipulates that, at a minimum, partial autocorrelation plots should be between –0.1 and 0.1, and by examining the residual plots to make sure they were free from patterns. We assessed the effects for gaseous pollutants (NO_2, SO_2, and O_3), PM with an aerodynamic diameter $\leq$ 10 μm (PM_{10}), and its chemical species (aluminum [Al], iron [Fe], manganese [Mn], nickel [Ni], vanadium [V], lead [Pb], and zinc [Zn]) using the Core Models, which were developed for the periods 5 years (or 2 years in the case of the sensitivity analysis) before and 5 years after the intervention, as well as in the

This Investigators' Report is one part of Health Effects Institute Research Report 170 which also includes a Commentary by the Health Review Committee and an HEI Statement about the research project. Correspondence concerning the Investigators' Report may be addressed to Dr. C-M Wong, The University of Hong Kong, School of Public Health, Department of Community Medicine, 5/F William MW Mong Block, 21 Sasson Road, Hong Kong, hrmrwcm@hku.hk.

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

10-year (or 7-year in the case of the sensitivity analysis) period pre- and post-intervention. We also included an indicator to separate the pre- and post-intervention periods, as well as the product of the indicator with an air pollution concentration variable. The health outcomes were mortality for all natural causes and for cardiovascular and respiratory causes, at all ages and in the 65 years or older age group.

To assess the short- and long-term effects, we developed two methods: one using linear regression models reflecting the age-standardized mortality rate $D(j)$ at day j, divided by a reference D_{ref}; and the other using Poisson regression models with daily mortality counts as the outcome variables. We also used both models to evaluate the relation between outcome variables and daily air pollution concentrations in the current day up to all previous days in the past 3 to 4 years.

In the linear regression approach, we adjusted the data for temperature and relative humidity. We then removed season as a potential confounder, or *deseasonalized* them, by calculating a standard seasonal mortality rate profile, normalized to an annual average of unity, and dividing the mortality rates by this profile. Finally, to correct for long-term trends, we calculated a reference mortality rate $D_{ref}(j)$ as a moving average of the corrected and deseasonalized $D(j)$ over the observation window. Then we regressed the outcome variable $D(j)/D_{ref}$ on an entire exposure sequence $\{c(i)\}$ with lags up to 4 years in order to obtain impact coefficient $f(i)$ from the regression model shown below:

$$\frac{\Delta D(j)}{D_{ref}} = \sum_{i=0}^{i_{\max}} f(i)\, c(j - i)$$

The change in life expectancy (LE) for a change of units (Δc) in the concentration of pollutants on T_{day} — representing the short interval (i.e., a day) — was calculated from the following equation (ΔL_{pop} = average loss in life expectancy of an entire population):

$$\Delta L_{pop} = -\Delta c\, T_{day} \sum_{j=0}^{\infty} \sum_{i=0}^{j} f(i)$$

In the Poisson regression approach, we fitted a distributed-lag model for exposure to previous days of up to 4 years in order to obtain the cumulative lag effect $\Sigma\hat{\beta}_i$. We fit the linear regression model of $\log(LE^*/LE) = \gamma(SMR - 1) + \alpha$ to estimate the parameter γ by $\hat{\gamma}$, where LE* and LE are life expectancy for an exposed and an unexposed population, respectively, and SMR represents the standardized mortality ratio. The life expectancy change per Δc increase in concentration is

$$LE\{\exp[\hat{\gamma}\Delta c(\textstyle\sum\hat{\beta}_j)] - 1\}$$

RESULTS

In our assessment of the changes in pollutant levels, the mean levels of SO_2, Ni, and V showed a statistically significant decline, particularly in industrial areas. Ni and V showed the greatest impact on mortality, especially for respiratory diseases in the 5-year pre-intervention period for both the all-ages and 65+ groups among all chemical species. There were decreases in excess risks associated with Ni and V after the intervention, but they were nonsignificant.

Using the linear regression approach, with a window of 1095 days (3 years), the losses in life expectancy with a 10-μg/m^3 increase in concentrations, using two methods of estimation (one with adjustment for temperature and RH before the regression against pollutants, the other with adjustment for temperature and RH within the regression against pollutants), were 19.2 days (95% CI, 12.5 to 25.9) and 31.4 days (95% CI, 25.6 to 37.2) for PM_{10}; and 19.7 days (95% CI, 15.2 to 24.2) and 12.8 days (95% CI, 8.9 to 16.8) for SO_2. The losses in life expectancy in the current study were smaller than the ones implied by Elliott and colleagues (2007) and Pope and colleagues (2002) as expected since the observation window in our study was only 3 years whereas these other studies had windows of 16 years. In particular, the coefficients used by Elliott and colleagues (2007) for windows of 12 and 16 years were non-zero, which suggests that our window of at most 3 years cannot capture the full life expectancy loss and the effects were most likely underestimated.

Using the Poisson regression approach, with a window of 1461 days (4 years), we found that a 10-μg/m^3 increase in concentration of PM_{10} was associated with a change in life expectancy of −69 days (95% CI, −140 to 1), and a change of −133 days (95% CI, −172 to −94) for the same increase in SO_2. The effect estimates varied as expected according to most variations in the sensitivity analysis model, specifically in terms of the Core Model definition, exposure windows, constraint of the lag effect pattern, and adjustment for smoking prevalence or socioeconomic status.

CONCLUSIONS

Our results on the excess risks of mortality showed exposure to chemical species to be a health hazard. However, the statistical power was not sufficient to detect the differences between the pre- and post-intervention periods in Hong Kong due to the data limitations (specifically, the chemical species data were available only once every 6 days, and data were not available from some monitoring stations). Further work is needed to develop methods for maximizing the information from the data in order to assess any changes in effects due to the intervention.

With complete daily air pollution and mortality data over a long period, time-series analysis methods can be applied to assess the short- and long-term effects of air pollution, in terms of changes in life expectancy. Further work is warranted to assess the duration and pattern of the health effects from an air pollution pulse (i.e., an episode of a rapid rise in air pollution) so as to determine an appropriate length and constraint on the distributed-lag assessment model.

INTRODUCTION

On July 1, 1990, the government of Hong Kong enacted a regulation restricting the sulfur content of solid and liquid fuels (hereafter referred to as the "intervention" in this report). The intervention was first called the "Air Pollution Control Ordinance (Fuel Restriction) Regulation 1989," and it prohibited the use of sulfur-bearing fuels in specified areas of Hong Kong. In 1990 it was renamed the "Air Pollution Control (Fuel Restriction) Regulation 1990" and was expanded to include a restriction on conventional solid and liquid fuels throughout all the territories of Hong Kong. Under the intervention, liquid fuels could not have a sulfur content of more than 0.5% by weight or a viscosity of more than 6 centistokes at 40°C. The term *liquid fuels* covered kerosene, diesel oil, petroleum, and any equivalent liquid fuel. For solid fuels, the sulfur content could not exceed 1% by weight, and the regulation covered coal, coke, charcoal, and wood. The intervention applied to any relevant plants in Hong Kong except those in or on premises used solely as dwellings, as well as vessels, motor vehicles, railway locomotives, aircraft, and licensed electricity generation plants. The regulation did not apply to gaseous fuel (Hong Kong Legal Information Institute 1990).

Compared with the baseline period of 2 years before the implementation of the July 1990 regulation, the annual average reduction in ambient SO_2 concentration across all monitoring stations was from 35% to 53% in the 5 subsequent years, with the greatest reduction occurring in the first year (Hedley et al. 2002). The level rose in the third year and then dropped again to a mean concentration of 22 $\mu g/m^3$ in the fifth year. In industrial areas, the SO_2 levels were the highest in the 2-year period before the intervention and the decrease was the greatest compared with other areas. For example, at the station in Kwai Chung the mean concentration was over 100 $\mu g/m^3$ and decreased by 80% immediately after the intervention (Hedley et al. 2002).

None of the other criteria pollutants, such as nitrogen dioxide (NO_2), ozone (O_3), and PM_{10}, showed a pattern of decreases similar to that of ambient SO_2. In fact, conversely, mean O_3 concentration increased annually by 22% to 70% for all 5 years after the intervention, while NO_2 and PM_{10} concentrations did not show any significant differences between the pre- and post-intervention periods (Hedley et al. 2002).

The 1990 intervention was associated with health gains in children and adults. Decreases in respiratory symptoms were shown among primary school children and their nonsmoking mothers (Wong et al. 1999). Great improvement in the bronchial responsiveness of the school children was also found (Wong et al. 1998).

In a time-series trend study, Hedley and colleagues (2002) assessed the effects of the intervention on mortality risks. The models were based on the intervention as a turning point and used data on mortality and pollutants for 5 years before and 5 years after the fuel restriction. There was an immediate reduction in mortality during the cool season at six months followed by an increase in cool-season mortality in the second and third years. The pattern suggested that the reduction in pollution was associated with a delay in mortality. Proportional changes in mortality trends between the 5-year periods before and after the intervention were measured as relative risks and used to assess gains in life expectancy using the life table method (Brunekreef 1997; Leksell and Rabl 2001; Miller and Hurley 2003). Relative risks were not directly related to pollutant concentrations.

Estimates from time-series studies for the association between short-term exposure to air pollution in Asia and mortality have been accumulating in recent years (see the PAPA-SAN database, Health Effects Institute 2008), but as of yet, there are no published results from local cohort studies. With the acceleration of economic development in Asia, air quality has been deteriorating rapidly in the region. Public health advocates are aware of an urgent need to communicate the risk of poor air quality to the public, to legislators, and to the government, with a view to strengthening the standards of control and the measures for the abatement of poor air quality (Hedley et al. 2008). The excess risks of mortality derived from short-term time-series studies can be used as a basis for computing the numbers of deaths brought forward and the economic costs associated with air pollution on a daily basis. However, the results of measuring excess daily deaths due to air pollution may not adequately convey the message that air pollution is a threat to health and life among those who are generally healthy. A better instrument for communicating risk is *life expectancy*, which can more graphically illustrate how changes are needed for accountability and environmental justice.

In general, changes in life expectancy due to air pollution exposure are assessed by comparing the survival experience of cohorts residing in areas with different air pollution levels. However, a difficulty with cohort design relates to the potential for confounding and misclassification of retrospective exposure and the cost of data collection (Künzli and Tager 2000). There are many short-term time-series studies but a paucity of cohort studies. Attempts have been made either to improve cohort study design or to use other methods to estimate life expectancy. With relatively readily available data, time-series studies offer a design that is the simplest to implement and is not subject to the limitations of cohort studies in the assessment of population health risks related to air pollution.

Daily air pollution time-series studies are designed to assess short-term effects and are generally regarded as unable to provide information on changes in life expectancy (McMichael et al. 1998; Künzli et al. 2001; Rabl 2003). However, effect estimates from daily time-series studies are mathematically equivalent to those derived from time-dependent covariates of Cox regression models, under the assumption that there is no systematic variation in the hazard rates among cohort members after adjustment for time-independent covariates or inclusion of some smooth function of time. Under these conditions, the effect estimates could be exploited for estimation of life expectancy (Burnett et al. 2003).

A mathematical model to assess the loss of years of life expectancy attributable to air pollution, with a homogeneous dynamic cohort, has recently been developed (Rabl 2006). The model postulates that, after a pulse of air pollution, effects on mortality last for a long period and vary as a function of time and that observed variations in mortality are affected by the effects of advanced deaths. As a result, sign reversals among the time-series coefficients may be observed, and it is necessary to look at exposure over a wide window of time in order to assess the long-term effects. We, therefore, decided that it would be interesting to know whether the impacts of air pollution could be estimated using a distributed-lag model, and whether the change in life expectancy could be assessed using a summation of the coefficients — that is, the impact functions — of the regression model for daily mortality. These questions formed the basis of one of our research objectives (see Objective 2 in "Specific Objectives"). However, in that linear model, the assumption that the short duration of time taken for an increase in mortality rate is equal to a decrease in individual life expectancy is not assessable.

With an adjustment for expected deaths derived from a standard set of age-specific death rates (ASDRs), the excess risk estimate from a daily time-series Poisson regression model can be interpreted as a change in SMR from the reference, per unit change in air pollution concentration. On the one hand, the SMR can be regarded as an age-adjusted measure of the hazard ratio (relative risk) when constructing post-exposure survival curves from life tables and, therefore, can be used to assess the change in life expectancy, under the assumption that there is no systematic variation in the hazard rates among cohort members, corresponding to a change in air pollution concentration. On the other hand, the SMR has been shown to possess a simple relationship, mathematically and empirically, with the life expectancy change (Lai et al. 1996). However, it remains a contentious question as to whether time-series studies can be legitimately used for the estimation of life expectancy changes, attributable to air pollution, for a time scale longer than a few months. This question is captured in Objective 3 (see "Specific Objectives").

To assess the effects of air pollution and of the air quality intervention, we investigated the impact of exposure to pollutants on acute daily deaths before and after the intervention. In Hong Kong after the implementation of the sulfur fuel restriction, there were substantial changes in SO_2 levels and particulate matter (PM) chemical species. The effect estimates of chemical species on mortality have been assessed in several studies (Burnett et al. 2000; Lippmann et al. 2006; Dominici et al. 2007; Ostro et al. 2007; Schlesinger 2007). The effect estimates of chemical species on mortality are still uncertain, because the effects among lags of different lengths are not consistent. A reanalysis of the same data, with adjustment for differences among geographic areas, showed them to be sensitive to confounding adjustment (Dominici et al. 2007). The issues of biologic plausibility and consistency remain problems, among others, in establishing causality. The Hong Kong intervention led to large reductions of SO_2, sulfate (SO_4), Ni, and V in PM_{10} for varying periods, but no sustained comparable changes in PM_{10} or nitrogen dioxide (NO_2). We wondered whether the population health effects that followed the 1990 Hong Kong intervention could be used to quantify any associations between individual chemical species of special interest and daily mortality — a research question we assess in Objective 1 (see "Specific Objectives").

In order to estimate short-term (acute) and long-term (chronic) effects of air pollution on human mortality, Künzli and colleagues (2001) outlined three categories of pathways through which air pollution contributes to

increased mortality risks: (A) "air pollution increases both the risk of underlying diseases leading to frailty and the short term risk of death among the frail"; (B) "air pollution increases the risk of chronic diseases leading to frailty but is unrelated to timing of death"; and (C) "air pollution is unrelated to risk of chronic diseases but short term exposure increases mortality among persons who are frail." Since time-series studies cannot assess the second category (B), the use of only time-series studies necessarily leads to an incomplete assessment of any health effects. In this study we postulate that with a lag of 0 to 2 days, the excess risk of death under the pathway category C can be assessed and that with a longer lag of several years, the change in life expectancy associated with air pollution can also be assessed. The short- and long-term effects of air pollution subject to the pathways described under (A) and (C) are depicted in Figure 1.

The effects of air pollution that are longer than a few days have been examined using distributed-lag models (Zanobetti et al. 2002; Goodman et al. 2004). Any effects captured in a short- or middle-term model may be due to the increasing susceptibility of subjects after long-term exposure or to the prolonged effects from pulses of air pollution (Goldberg et al. 2001; Schwartz et al. 2008). The biologic mechanisms explaining these associations are not well understood. Associations in a long distributed-lag model could, in theory, be explained by a critical short-term event that manifested only after a much longer period of time. (The relation between long- and short-term events will be addressed under our Objective 4 listed below.) For example, for individuals who are vulnerable to coronary thrombosis, there is an increased association between acute myocardial infarction and air pollution. Once this air-pollution–induced myocardial event has occurred, there may be long-term vascular insufficiency and reduced cardiac performance, which will further increase the risk of other episodes and shorten life expectancy.

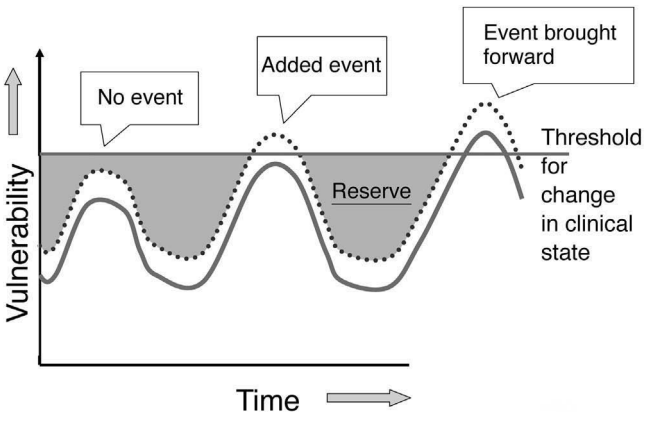

Figure 1. Hypothetical variations of the vulnerability of individuals taking into account trends and seasonality shown through time before (solid line) and after (broken line) exposure to air pollution. At the early stage when there is enough reserve capacity (shaded), there is no clinical event, for example, death (No event). At the intermediate stage, those who do not have enough reserve are added (become vulnerable) to the event (Added event). At the end stage during the peak season, all are vulnerable to the event (Event brought forward).

SPECIFIC OBJECTIVES

In this project, the overall aim was to develop the methodology and analytical approaches for estimating the impact on life expectancy of a sudden improvement in air quality after the implementation of a fuel regulation. We set out to achieve this aim in the context of the 1990 Hong Kong restriction of sulfur in fuels ("the intervention"). The objectives arising from the issues and research questions described in the Introduction are as follows:

1. to evaluate the short-term effects on mortality due to changes in pollutants after the intervention, particularly changes in the PM chemical species;
2. to improve the current methodology for assessing the health impact, in terms of the change in life expectancy, resulting from interventions taken to improve air quality, using linear regression models;
3. to develop a methodology using Poisson regression specifically suited to quantify the benefits of the intervention in terms of years of life gained; and
4. to determine any relation between short-term and long-term health benefits due to an improvement in air quality.

SHORT-TERM EFFECTS OF AIR POLLUTION BEFORE AND AFTER THE 1990 HONG KONG INTERVENTION (OBJECTIVE 1)

In the Introduction, we briefly outlined the health gains from the Hong Kong intervention. However, the assessments (Wong et al. 1998, 1999; Hedley et al. 2002) of the health effects were based mainly on a comparison between periods before and after the intervention. Although there were reductions in health effects, the findings did not specify which pollutants contributed to the change in health

effects and which did not. The effects were not targeted for specific pollutants. In this section, we pursue Objective 1 focusing particularly on the concentrations of gaseous and particulate pollutants and PM chemical species. If we found a real change in any pollutant concentrations, the next question we posed was whether the change after the intervention was associated with a reduction in the excess risk of mortality. All analyses were performed using R statistical software, version 2.5.1 (R Development Core Team 2007, Vienna, Austria). Statistical significance refers to results with P values ≤ 0.05.

METHODS AND DESIGN

The study period was from July 1, 1985, to June 30, 1995. The pre- and post-intervention periods were defined as 5 years before and 5 years after the intervention date (July 1, 1990).

Mortality Data

We obtained daily mortality counts from January 1, 1985, to June 30, 1995, from the Immigration Department (IMMD) in Hong Kong. Diagnoses of mortality were coded according to the *International Classification of Diseases,* 9th revision (ICD-9). In our study, the disease rubrics were all natural causes (ICD-9 001–799), and the subcategories cardiovascular diseases (ICD-9 390–459), respiratory diseases (ICD-9 460–519), and accidental causes (ICD-9 800–999).

For each mortality outcome, we extracted population-based daily counts. For all natural causes, cardiovascular diseases, and respiratory diseases, daily death counts for those aged 65 years or older (65+) were also extracted.

The data collection process involved three government departments (see also Figure 2):

- The Hong Kong IMMD, which provided individual death records;
- The University of Hong Kong School of Public Health, Environmental Health Research Group (HKU), which provided data entry; and
- The Census and Statistics Department (C&SD) of the Hong Kong Special Administrative Region, which linked the data we entered to the existing computerized database.

Since there was no computerized mortality database containing information on date of death before 1994, the IMMD provided hard copies of the death certificates of individuals whose deaths were registered between January 1985 and December 1994. These hard copies were originally in a microfilm format with personal identifiers. Because of data confidentiality regulations, the IMMD concealed any identification information, such as names and Hong Kong Identification Numbers, on the hard copies and then photocopied the microfilm for our study. These photocopies were delivered to us in boxes in batches. From a total of 33 boxes in 11 batches, we received 59,941 hard copies. After setting up a Microsoft Access data-entry template, we entered each record from the hard copies at our HKU unit.

When the batches arrived, each hard copy was assigned a sheet number (see Figure C.1 in Appendix C available on the HEI Web site). We entered into our database the date of death, the death registry at which the death was registered, and the registration number, age, and sex of the deceased individual. In addition, we assigned a variable

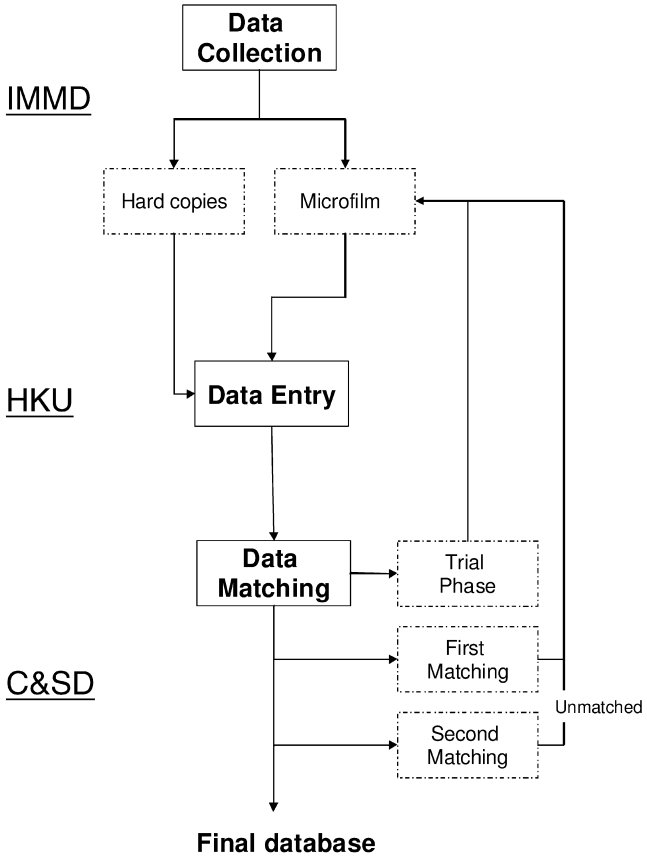

Figure 2. Flow chart of mortality data collection. (Note that Trial Phase refers to 1985 data only.)

to qualify the clarity of data on each hard copy, indicating if it was (1) clearly readable, (2) traceable by conjecture, or (3) unclear. To verify data quality, we sent the data entered for year 1985 to the C&SD to match against their records as a test (labeled "Trial Phase" in Figure 2). The C&SD responded by recommending that we include the date of registration in each unique identifier to facilitate matching the records. We then negotiated with the IMMD for special permission to enter the immigration record unit and read the microfilms. We entered the date of registration for every deceased person, as well as additional records from January 1995 through June 1995 to ensure that no registration records around the end of 1994 would be missed. At the same time, we rechecked the information that was already keyed in for each record as a countercheck against data-entry errors.

We sent our completed database to the C&SD to link to its database. (We used deterministic linkage by including the following data: date of registration, place of registration, date of death, sex, and age to create a unique identifier to do the linking.) The C&SD database included additional information not obtainable from the IMMD, such as place of residence and occupation. The C&SD used our unique identifier (consisting of the registry name, date of registration, and year of death) to match the records and then sent us a master database. We returned to the IMMD and checked any unmatched records to ensure that the information was entered correctly. The matching process was repeated (shown as the "Second Matching" step in Figure 2). Our analyses of mortality were based on data from the master database after this linkage process.

As confirmation of the completeness of these retrieved data, the distribution of the data from the master database and from the C&SD database was calculated to check whether there were systematic discrepancies in the master database.

Pollutants

Air pollutant concentrations were provided by the Hong Kong Environmental Protection Department (EPD). During the 10-year study period, there were 13 monitoring stations operating: Causeway Bay, Central/West (CW), Hong Kong South, Junk Bay, Kwai Chung (KC), Kwun Tong (KT), Mong Kok, Sha Tin, Sham Shui Po (SSP), Tai Po, Tsim Sha Tsui, Tsuen Wan (TW), and Yuen Long. We included only the monitoring stations at CW, KC, KT, SSP, and TW because data were available at these stations across the period of the intervention, especially including the 5 years after the intervention (see Table B.1 in Appendix B).

The five monitoring stations included in the study were located in two different types of areas. Stations CW and SSP were in residential and commercial areas, in which vehicles were the major source of emissions. Stations KC, KT, and TW were located in industrial areas in which commercial vehicles, manufacturing plants, and service industries were the major sources of emissions. We obtained quality assurance/control and standard operating procedure documents and reviewed the processes for data measurement and collection. We did our own calculation of values and checked them against those reported by the government to see if they were equivalent. The details of our procedure to validate monitor measurements have been published in our study in the Public Health and Air Pollution in Asia (PAPA) project (Wong et al. 2010).

Gaseous Pollutants Hourly measurements of NO_2 (by chemiluminescence), SO_2 (by fluorescence), and O_3 (by UV absorption) were available. Daily mean pollutant concentrations for each station were derived from 24 hourly measurements of NO_2 and SO_2, as well as 8 hourly measurements of O_3 from 10:00–18:00 on any day for which at least 75% of the number of hourly measurements were available. Daily concentrations for the whole territory of Hong Kong were estimated as the arithmetic mean over five stations. The diurnal patterns of pollutant concentrations (see Figure B.1 in Appendix B) were consistent with those in the annual government report. Cumulative concentrations at the average of a same-day and 1-day lag (lag 0–1 day) were also calculated as the simple average of concentrations from the current day to lag 1 day.

PM_{10} and Its Chemical Species PM_{10} and chemical species in PM_{10} were measured approximately once every 3 or 6 days, respectively. The gravimetric method was used to collect filtered samples of PM_{10}. The EPD sent the chemical species samples to government chemists to measure their concentrations by different chemical analyses.

Chemical species data from monitors for which less than 75% of days had complete measurements in the study period were not included in our study. Those chemical species for which measurements before the intervention period were not available were also excluded from our study. In our study, there were data on 16 out of 28 chemical species (about 57% of all chemical species):

Al, bromide (Br^-), carbon (C), calcium (Ca), chloride (Cl), Fe, magnesium (Mg), Mn, sodium (Na), ammonium (NH_4^+), Ni, nitrate (NO_3), Pb, SO_4, V, and Zn.

Al, Ca, Fe, Mg, Mn, Ni, Pb, V, and Zn were measured by inductively coupled plasma-atomic emission spectrometry. Ion chromatography was used to measure Na, NH_4^+, Br, Cl, NO_3, and SO_4. For the measurement of organic and elemental carbon, thermal/optical carbon analysis was applied. Values below the detection limit for all chemical analyses were notated as 0. (The proportion of nondetectable values for each pollutant ranged from 0% to 10% [see Table B.13 in Appendix B].) Concentrations of PM chemical species were aggregated over the five stations to represent the exposure for the whole territory of Hong Kong.

Meteorologic Variables

Daily mean temperature (°C) and RH (%) were obtained from the Hong Kong Observatory.

DATA ANALYSIS

Mortality and Pollutant Concentrations

We produced time-series plots of daily mortality and pollutant concentrations. We also reported summary statistics by time period.

Our team used independent *t* tests to assess mean differences in concentrations between pre- and post-intervention periods. We used analysis of variance (ANOVA) tests with Tukey's Honestly Significant Differences (Tukey's HSD) to assess the mean differences between the average concentration in the pre-intervention period and each of the 5 years in the post-intervention period adjusted for multiple comparisons. The same tests were also applied on the mean differences among years in the post-intervention period in a sequential order.

Short-Term Effects of Air Pollution

In order to assess the short-term effects of air pollution for time i (AP_i) from 1, 2, 3 . . . days, a Core Model for each outcome (Y_i) was developed to eliminate any confounding effects (C_i). Then, we added daily pollutants into the Core Model separately to obtain the ER (%) of mortality due to daily pollutants.

$$\log(Y_i) = C_i + AP_i \qquad \text{for } i = 1, 2, 3 \ldots \tag{1}$$

For simplicity, the coefficients associated with variables were not specified in this section.

Core Model Development We developed a Core Model to assess how mortality was associated with exposure to air pollution, with an adjustment for confounding factors. For gaseous pollutants, we used the daily mortality count as the outcome; for chemical species, we used total mortality, which summed up the number of deaths from the current day to lag 1 day, as the outcome (Roberts and Martin 2006).

We employed a Poisson regression model with a natural spline smooth function of confounding factors. We added a smooth function term for time and an indicator of the month to remove confounding effects due to trend and seasonality. In addition, we added smooth function terms for temperature and RH. The confounding factors were put into the Core Model in sequential order.

We selected the Core Model based on the minimum Akaike Information Criterion. First, we added monthly indicators since they showed a better fit than the indicators for days of weeks. Then, we selected the model by changing the degrees of freedom (df) for the smoothing of time trend from 3 to 12 df per year, the T at current day from 0 to 12 df, and the RH at current day from 0 to 12 df sequentially. Furthermore, we selected another smooth function for T with 3 df by averaging various numbers of days from lag l day to lag 12 days.

We plotted the residuals of the Core Model based on the minimum Akaike Information Criterion (that the partial autocorrelation plots should be between −0.1 and 0.1) to check if there was a discernible pattern. We checked the partial auto-correlation of the residuals to ensure the absence of auto-correlation. We also applied the Ljung-Box test for overall randomness in the first 15 lag days of residuals. We had the option of adding auto-regressive terms into the model if auto-correlations were detected.

For each outcome, three Core Models were developed covering the pre-intervention, post-intervention, and whole study period. We applied the same selection method for all Core Models. We recorded the model specifications for all periods (see Table C.1 in Appendix C available on the HEI Web site).

For the sensitivity analysis, we defined the pre-intervention period as the 2-year period before the intervention and defined the whole assessment period as covering 7 years (2 years before and 5 years after the intervention). We chose a relatively short time period for the pre-intervention period because all five stations were operating during the 2 years before the intervention.

Table 1. Matched Cases Between HKU Database and C&SD Master Database by Year

Year	No. of Records in C&SD Master Database	No. of Records Matched to HKU Database	Percentage of Matched Cases
1985	25,248	23,725	93.97
1986	25,902	25,237	97.43
1987	26,916	26,125	97.06
1988	27,659	27,141	98.13
1989	28,745	28,045	97.56
1990	29,136	28,581	98.10
1991	28,429	28,020	98.56
1992	30,550	29,854	97.72
1993	30,571	29,494	96.48
1994	29,905	29,032	97.08
Total	283,061	275,254	97.24[a]

[a] Average.

Assessment of Short-Term Effects After developing the Core Models, we put data regarding each of the gaseous pollutants and PM_{10} at lag 0–1 day and regarding the PM chemical species at the current day into the model separately to assess the ER. We considered the ER to be the percentage change in mortality per 10-μg/m^3 and 10-ng/m^3 increase in the concentrations of four criteria pollutants and PM chemical species, respectively.

To assess the change in the short-term effects due to air pollution before and after the intervention, we created a time indicator of period p (I_p) to separate pre- and post-intervention periods. Then, we added the main effect of each air pollutant (AP_i), the time indicator, and an interaction (product) term expressing the interaction between the air pollutant and the indicator into the model.

$$\log(Y_i) = C_i + I_p + AP_i + I_p \times AP_i \qquad \text{for } i = 1, 2, 3 \ldots \quad (2)$$

We considered the coefficient estimate of AP_i to be the ER of mortality due to the air pollutant before the intervention, while we interpreted the coefficient estimate of $I_p \times AP_i$ in the interaction model to be the change in ER after the implementation of the Hong Kong intervention.

RESULTS

Mortality Data

We received 263,779 unique death records from the IMMD (as of August 23, 2007) covering from 1985 to 1995. After retrieving the information from the microfilm, we entered 305,356 records, excluding 22,334 records in the study period 1985–1994. In total, we matched 275,254 cases with records in the C&SD master databases. More than 96% of the individual death records were matched except those in 1985 (Table 1).

Comparison with the C&SD Database The difference in the proportion of disease groups represented in our HKU database and the master C&SD database was within 2% each year (Table 2). The other factors, such as area of residence, sex, marital status, and occupation, were within 0.5% (see Tables C.2–C.5 in Appendix C available on the HEI Web site)

Daily and Cumulative Counts The average daily number of deaths from all natural causes (with the standard deviation) for all ages was 69 (13) in the 5-year pre-intervention period and 76 (14) in the 5-year post-intervention period. On average, cardiovascular, respiratory, and accidental deaths contributed to 30%, 17%, and 6% of all-cause mortality, respectively, in the pre-intervention period. The percentages for these three disease groups were very similar to those in the post-intervention period (30%, 18%, and 5%, respectively) (Table 3).

Over the 10-year study period, the daily number of deaths due to all natural causes, cardiovascular disease, and respiratory disease showed a seasonal pattern, with higher numbers of deaths occurring in the cool season (Figure 3). However, mean daily number of deaths did not show the same change (Table 3 and Table C.6 in Appendix C available on the HEI Web site).

Variations in the number of daily deaths and in the cumulative number of deaths (based on aggregated daily numbers from current day to lag 1 day) were similar (Figure 4). The correlations between daily deaths and cumulative deaths for the same outcomes were all > 0.75 (data not shown).

Table 2. Percentage of Matched Cases and Numbers in C&SD Database by Disease Category

Disease Category	1985		1986		1987		1988		1989	
	Matched (n = 23,725)	C&SD (n = 25,248)	Matched (n = 25,237)	C&SD (n = 25,902)	Matched (n = 26,125)	C&SD (n = 26,916)	Matched (n = 27,141)	C&SD (n = 27,659)	Matched (n = 28,045)	C&SD (n = 28,745)
Unknown	0.00	0.24	0.00	0.44	0.00	0.56	0.00	0.69	0.01	0.93
Infectious and parasitic diseases	3.09	3.04	3.05	3.00	3.11	3.04	3.17	3.13	3.52	3.49
Neoplasms	30.36	29.86	31.58	31.10	31.15	30.68	30.10	29.80	30.31	29.95
Endocrine, nutritional and metabolic diseases, and immunity disorders	0.98	0.99	1.02	1.00	0.94	0.93	0.87	0.86	0.92	0.91
Diseases of the blood and blood-forming organs	0.13	0.12	0.16	0.15	0.09	0.09	0.10	0.10	0.12	0.12
Mental disorders	0.01	0.01	0.00	0.00	0.01	0.01	0.00	0.00	0.00	0.00
Diseases of the nervous system and sense organs	0.79	0.78	0.69	0.68	0.87	0.86	0.69	0.68	0.78	0.77
Diseases of the circulatory system	28.76	29.13	28.95	28.88	29.07	29.10	29.04	28.92	28.16	27.96
Diseases of the respiratory system	16.25	16.29	15.71	15.79	16.28	16.31	17.55	17.45	17.52	17.38
Diseases of the digestive system	4.42	4.36	4.15	4.07	4.08	4.03	4.16	4.10	4.38	4.33
Diseases of the genitourinary system	4.32	4.37	4.74	4.66	4.58	4.52	4.56	4.51	4.46	4.42
Complications of pregnancy, childbirth and the puerperium	0.01	0.02	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Diseases of the skin and subcutaneous tissues	0.05	0.04	0.03	0.03	0.03	0.03	0.03	0.03	0.04	0.04
Diseases of the musculoskeletal system and connective tissue	0.21	0.21	0.24	0.24	0.21	0.20	0.16	0.16	0.19	0.18
Congenital anomalies	0.95	0.92	0.90	0.88	0.91	0.89	0.83	0.82	0.76	0.75
Certain conditions originating in the prenatal period	1.15	1.11	1.08	1.06	0.88	0.87	0.91	0.90	0.82	0.81
Symptoms, signs and ill-defined conditions	2.48	2.48	2.36	2.38	2.33	2.33	2.38	2.38	2.43	2.40
Injury and poisoning	6.05	6.03	5.34	5.63	5.45	5.55	5.47	5.46	5.58	5.55

(*Table continues on next page*)

Table 2 (*Continued*). Percentage of Matched Cases and Numbers in C&SD Database by Disease Category

Disease Category	1990		1991		1992		1993		1994	
	Matched (n = 28,581)	C&SD (n = 29,136)	Matched (n = 28,020)	C&SD (n = 28,429)	Matched (n = 29,854)	C&SD (n = 30,550)	Matched (n = 29,494)	C&SD (n = 30,571)	Matched (n = 29,032)	C&SD (n = 29,905)
Unknown	0.00	1.05	0.00	0.69	0.00	1.43	0.00	1.69	0.00	1.69
Infectious and parasitic diseases	3.59	3.54	3.30	3.28	3.05	2.99	3.37	3.32	3.95	3.86
Neoplasms	30.03	29.70	31.26	31.03	30.32	29.79	31.17	30.66	32.36	31.62
Endocrine, nutritional and metabolic diseases, and immunity disorders	0.98	0.97	1.02	1.01	1.51	1.49	1.58	1.56	1.38	1.35
Diseases of the blood and blood-forming organs	0.07	0.07	0.13	0.13	0.21	0.21	0.21	0.20	0.21	0.20
Mental disorders	0.00	0.00	0.01	0.01	0.04	0.04	0.01	0.01	0.01	0.01
Diseases of the nervous system and sense organs	0.77	0.78	0.76	0.75	1.01	1.00	0.81	0.79	0.88	0.86
Diseases of the circulatory system	28.63	28.32	28.85	28.61	28.87	28.35	28.34	27.72	28.74	28.13
Diseases of the respiratory system	17.22	17.10	16.67	16.54	18.49	18.19	18.85	18.44	17.55	17.19
Diseases of the digestive system	4.20	4.15	4.23	4.20	4.15	4.09	4.53	4.45	4.42	4.35
Diseases of the genitourinary system	4.70	4.62	4.55	4.56	3.67	3.62	3.52	3.46	3.73	3.66
Complications of pregnancy, childbirth and the puerperium	0.00	0.00	0.01	0.01	0.01	0.01	0.01	0.01	0.02	0.02
Diseases of the skin and subcutaneous tissues	0.00	0.00	0.02	0.02	0.09	0.09	0.09	0.09	0.08	0.08
Diseases of the musculoskeletal system and connective tissue	0.22	0.22	0.10	0.10	0.12	0.11	0.17	0.17	0.16	0.15
Congenital anomalies	0.63	0.62	0.59	0.58	0.46	0.45	0.48	0.47	0.45	0.44
Certain conditions originating in the prenatal period	0.63	0.62	0.77	0.76	0.56	0.55	0.50	0.49	0.53	0.53
Symptoms, signs and ill-defined conditions	2.79	2.76	2.41	2.41	2.57	2.56	1.77	1.73	1.31	1.30
Injury and poisoning	5.54	5.48	5.32	5.30	4.85	5.03	4.58	4.73	4.22	4.57

Table 3. Averages of Daily and Cumulative Mortality Counts from Current Day to Lag 1 Day

	Whole Period July 1985–June 1995 Mean ± SD	Pre-Intervention Period		Post-Intervention Period July 1990–June 1995 Mean ± SD
		July 1985–June 1990 Mean ± SD	July 1988–June 1990 Mean ± SD	
Daily				
All natural causes				
All ages	72 ± 14	69 ± 13	72 ± 13	76 ± 14
65+	50 ± 12	46 ± 11	49 ± 11	55 ± 12
Cardiovascular				
All ages	22 ± 6	21 ± 6	22 ± 6	23 ± 7
65+	17 ± 6	16 ± 5	17 ± 5	18 ± 6
Respiratory				
All ages	13 ± 5	12 ± 5	13 ± 5	14 ± 5
65+	11 ± 5	10 ± 4	11 ± 4	12 ± 5
Accidental				
All ages	4 ± 2	4 ± 2	4 ± 2	4 ± 2
Cumulative				
All natural causes				
All ages	145 ± 24	138 ± 22	145 ± 22	152 ± 24
65+	101 ± 22	92 ± 19	99 ± 19	109 ± 21
Cardiovascular				
All ages	44 ± 11	42 ± 10	43 ± 10	46 ± 11
65+	34 ± 10	32 ± 9	33 ± 9	37 ± 10
Respiratory				
All ages	27 ± 9	24 ± 8	27 ± 8	29 ± 9
65+	22 ± 8	20 ± 7	22 ± 7	24 ± 8
Accidental				
All ages	8 ± 3	8 ± 3	8 ± 3	8 ± 4

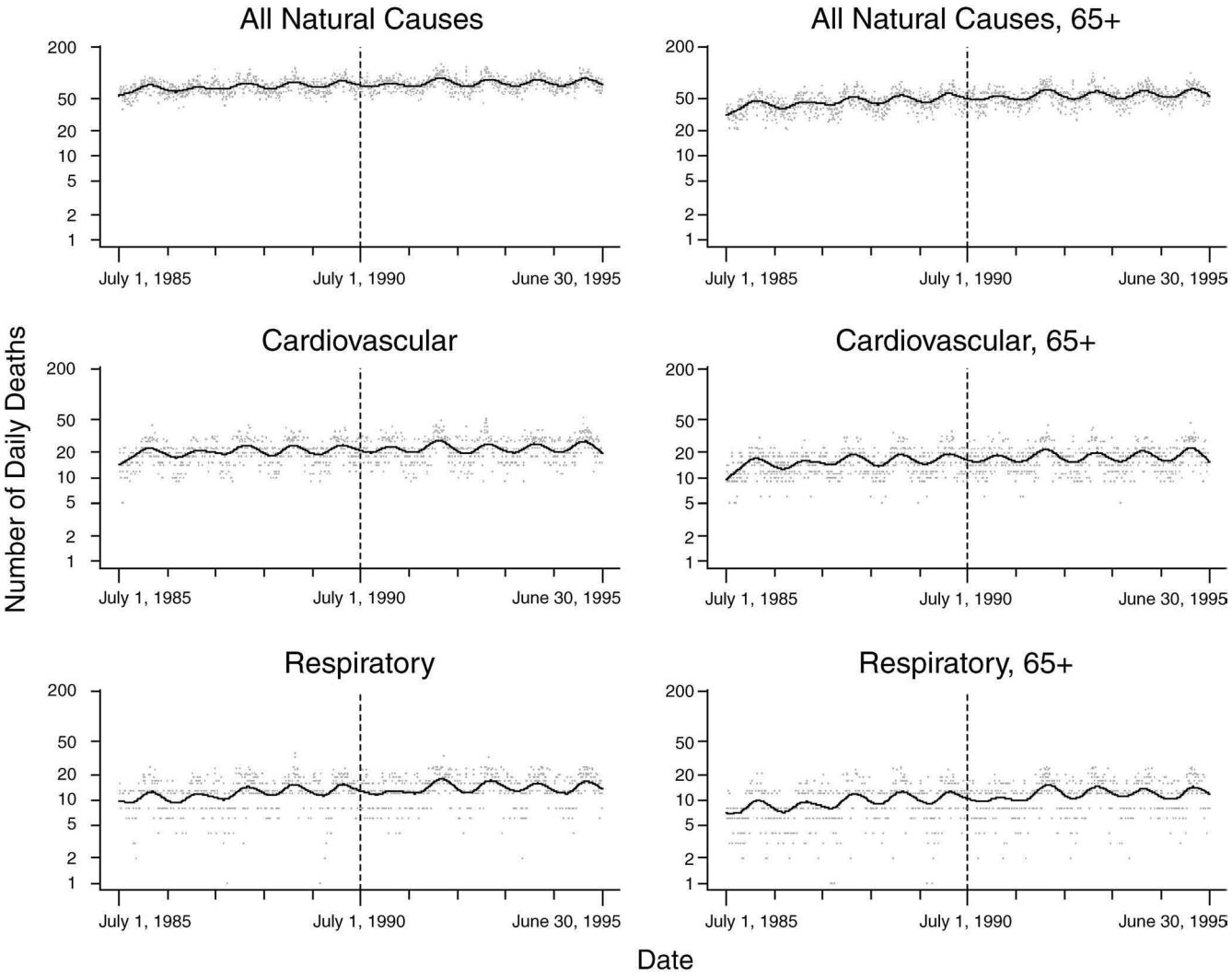

Figure 3. Time-series plot of daily mortality before and after the intervention. The solid line represents the smoothed curve of the observed daily number of mortalities. The dotted line marks the beginning of the sulfur fuel intervention.

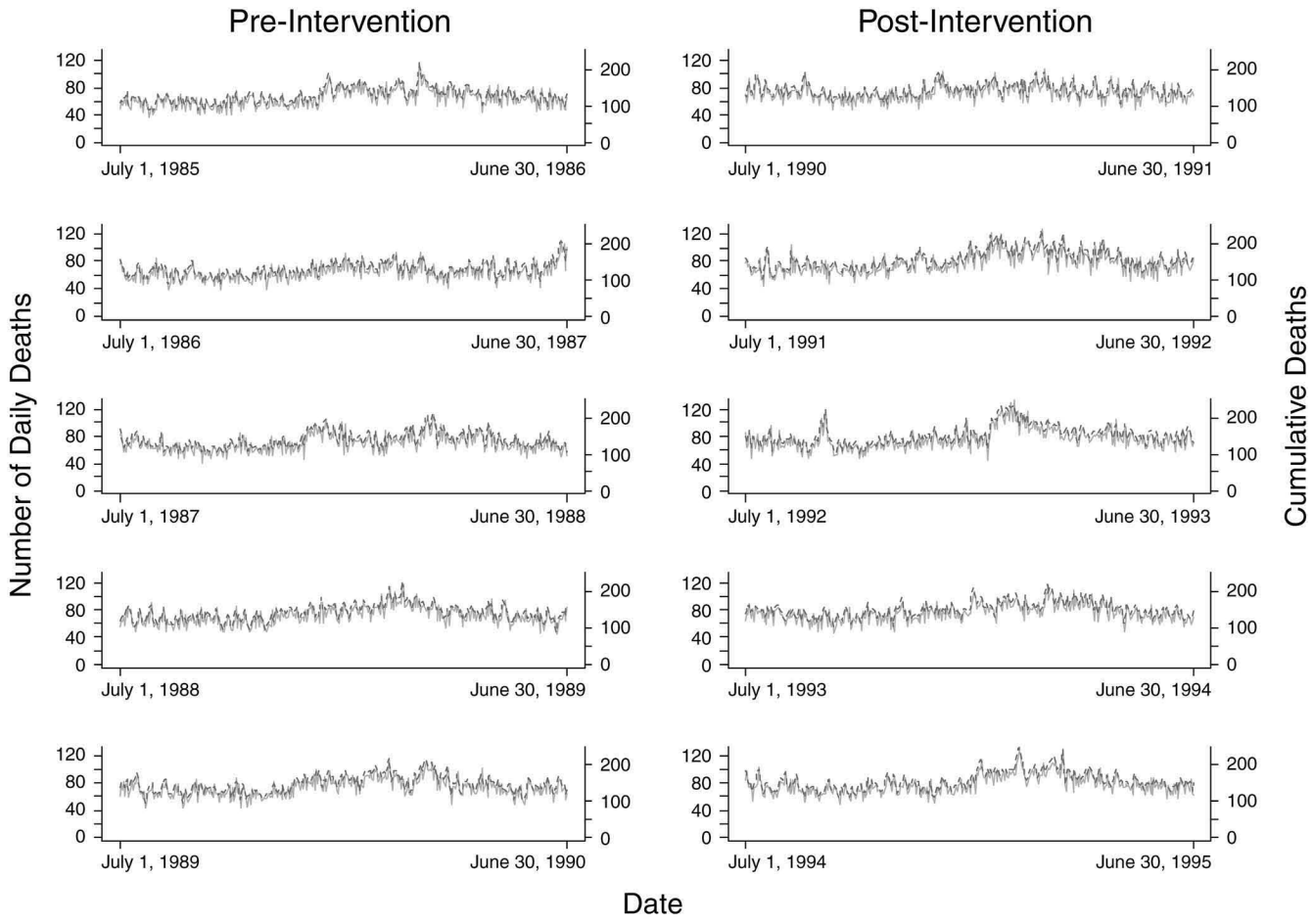

Figure 4. Time-series plots of daily mortality and total mortality due to all causes pre- and post-intervention.

Pollutant Data

NO_2 The mean concentration of NO_2 ranged from 36 μg/m^3 at station KC to 90 μg/m^3 at station KT in the pre-intervention period. At stations CW, KT, and TW, NO_2 concentrations decreased in all five years of the post-intervention period. Significant differences in mean were shown between the pre- and post-intervention period at these three stations (Table 4 and Figure 5).

Dispersion of data at each station varied, with the standard deviation ranging from 15 μg/m^3 at station KC to 59 μg/m^3 at station KT in the pre-intervention period. After the intervention, the dispersion at each station became similar with an average value of 21 μg/m^3 (Tables 5 and 6).

SO_2 SO_2 showed a drop of 43% in the first year of the intervention compared with the level of 36 μg/m^3 in the pre-intervention period. In the pre-intervention period, the mean level at station CW and SSP in mixed areas was 17 μg/m^3. The other stations, which were in industrial areas, had much higher levels, with the highest mean level at 114 μg/m^3 (KC). After the intervention, mean concentrations in two of the industrial areas (KC and KT) declined sharply by as much as 78%, with the resulting mean level at 24 μg/m^3 for all stations (Table 5) (Figure 6).

The distribution of daily SO_2 in mixed areas (stations CW and SSP) showed a narrower range than in industrial areas (stations KC, KT, and TW) in both pre- and post-intervention periods. Standard deviations of stations in industrial areas decreased to a level close to mixed areas (Table C.7 in Appendix C available on the HEI Web site).

Table 4. Mean Pollutant Concentration at Each Monitoring Station and *t* Test Results Between Pre- and Post-Intervention Periods

	Pre-Intervention Period			Post-Intervention Period			*t* Test		
	n[a]	Mean	SD	n[a]	Mean	SD	*t* Value	df	*P* Value
Central/Western									
NO_2	1703	56.05	33.25	1601	48.59	21.26	7.73	2917	0.000
SO_2	1740	16.95	14.75	1665	20.27	15.78	−6.34	3362	0.000
PM_{10}	535	58.01	33.86	317	56.89	29.04	0.51	744	0.608
O_3	1552	30.72	20.10	1529	38.39	28.53	−8.62	2742	0.000
Kwai Chung									
NO_2	630	36.07	14.86	1692	44.65	18.71	−11.50	1408	0.000
SO_2	628	113.85	61.78	1721	24.96	20.04	35.38	676	0.000
PM_{10}	186	60.85	28.74	348	53.15	27.28	3.00	361	0.003
O_3	490	23.28	31.58	1688	35.52	26.59	−7.82	702	0.000
Kwun Tong[b]									
NO_2	1689	89.61	59.00	1762	63.22	23.00	17.17	2172	0.000
SO_2	1723	50.25	75.36	1781	23.49	21.89	14.17	2002	0.000
PM_{10}	520	61.53	31.48	349	64.68	28.71	−1.52	791	0.128
O_3	—	—	—	—	—	—	—	—	—
Sham Shui Po[b]									
NO_2	491	56.59	26.85	1559	58.42	21.26	−1.38	694	0.169
SO_2	1567	16.74	14.34	1711	18.20	17.29	−2.64	3245	0.008
PM_{10}	517	59.21	28.83	344	64.71	28.24	−2.78	745	0.006
O_3	—	—	—	—	—	—	—	—	—
Tsuen Wan									
NO_2	667	62.57	24.63	1605	58.54	20.67	3.72	1075	0.000
SO_2	666	35.90	36.60	1623	35.09	28.52	0.52	1012	0.606
PM_{10}	211	57.27	29.30	340	58.30	25.38	−0.42	398	0.673
O_3	635	29.25	17.72	899	32.39	27.17	−2.74	1523	0.006

[a] n = number of measurements.

[b] O_3 readings from stations KT and SSP were excluded from the study because < 75% of hourly data were available.

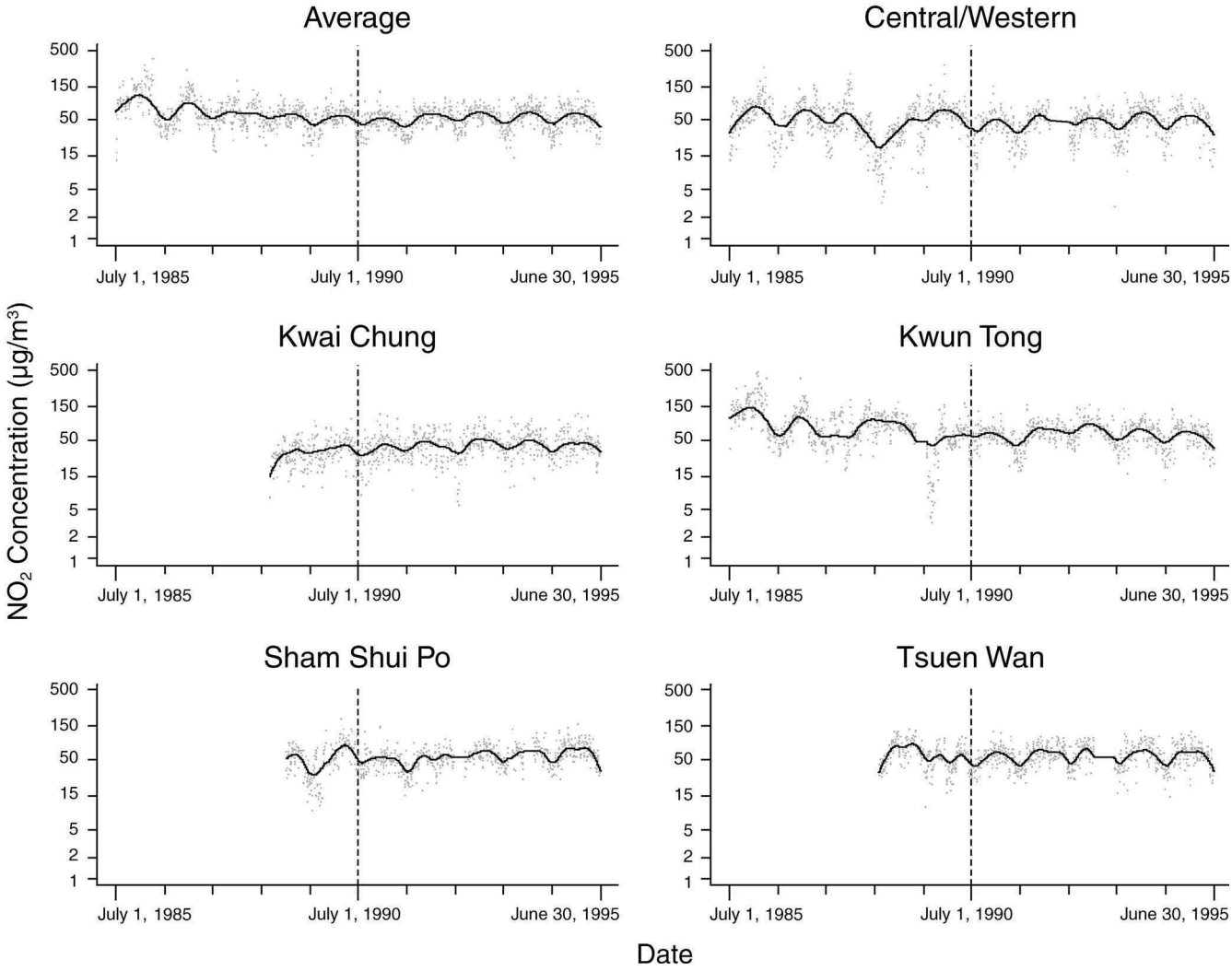

Figure 5. Time-series plot of the average daily concentrations of NO_2 ($\mu g/m^3$) from all stations in the study. The solid line represents the smoothed curve of the observed concentrations of the pollutant. The dotted line marks the beginning of the sulfur fuel intervention.

Table 5. Mean and Standard Deviation of Pollutant Concentration (μg/m^3) at Each Monitoring Station

	Pre-Intervention Period				Post-Intervention Period											
	5-Year		2-Year		5-Year		July 1990–June 1991		July 1991–June 1992		July 1992–June 1993		July 1993–June 1994		July 1994–June 1995	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
NO_2																
CW	56.05	33.25	50.89	32.46	48.59	21.26	41.47	19.10	53.04	21.22	47.85	18.66	52.43	23.83	50.12	21.23
KC	36.07	14.86	36.07	14.86	44.65	18.71	39.44	16.85	44.59	17.02	47.50	19.90	45.95	19.45	46.05	19.25
KT	89.61	59.00	69.73	32.13	63.22	23.00	55.31	18.00	67.97	21.22	73.42	24.45	61.50	23.96	58.20	22.27
SSP	56.59	26.85	56.59	26.85	58.42	21.26	50.98	17.08	51.75	18.05	61.32	17.89	62.50	24.07	66.63	23.75
TW	62.57	24.63	62.57	24.63	58.54	20.67	53.73	17.84	61.82	19.41	59.26	21.30	58.54	22.34	59.64	21.82
Overall	70.65	39.15	54.71	17.60	55.01	19.18	48.06	15.96	56.21	17.21	57.44	18.66	56.63	21.72	56.71	20.25
SO_2																
CW	16.95	14.75	18.17	15.62	20.27	15.78	17.39	12.63	20.93	12.85	23.45	19.56	21.10	16.89	18.77	14.26
KC	113.80	61.78	113.80	61.78	24.96	20.04	22.49	14.09	21.01	13.73	29.48	26.16	29.60	23.17	22.01	17.94
KT	50.25	75.36	42.27	60.27	23.49	21.89	25.00	18.42	21.12	17.75	27.80	23.35	25.72	29.41	17.93	16.75
SSP	16.74	14.34	15.29	15.25	18.20	17.29	7.75	8.72	14.03	10.59	16.90	18.52	28.00	21.43	25.60	15.91
TW	35.90	36.60	35.90	36.60	35.09	28.52	31.25	19.46	37.91	26.58	43.26	41.98	38.96	34.34	27.00	16.43
Overall	36.33	31.55	44.41	23.33	24.29	15.70	20.76	11.27	23.00	11.73	27.09	19.19	28.41	19.72	22.19	13.04
PM_{10}																
CW	58.01	33.86	54.94	27.84	56.89	29.04	58.11	27.51	53.30	29.18	59.16	27.78	56.49	31.36	54.49	31.68
KC	60.85	28.74	60.85	28.74	53.15	27.28	53.55	24.77	52.54	29.59	54.32	23.09	54.91	31.24	49.96	29.81
KT	61.53	31.48	62.59	33.71	64.68	28.71	65.34	25.95	67.38	36.20	72.95	26.74	58.16	26.40	57.56	26.16
SSP	59.21	28.83	60.76	30.92	64.71	28.24	64.59	26.38	65.55	34.69	73.27	27.96	63.38	26.98	55.00	22.20
TW	57.27	29.30	57.27	29.30	58.30	25.38	54.43	21.98	59.02	30.36	59.27	20.12	62.17	25.94	60.66	28.59
Overall	58.87	27.57	58.65	25.91	60.11	26.60	59.31	22.04	59.46	30.07	64.07	24.79	60.73	30.16	57.75	29.08
O_3[a]																
CW	30.72	20.10	27.37	20.07	38.39	28.53	33.53	22.84	45.67	34.62	39.55	27.22	40.56	32.07	35.76	26.63
KC	23.28	31.58	23.28	31.58	35.52	26.59	30.16	18.81	27.88	19.88	30.59	24.57	45.41	29.72	43.62	32.13
TW	29.25	17.72	29.25	17.72	32.39	27.17	32.44	25.17	32.85	29.16	35.23	28.64	14.48	5.82	—	—
Overall	30.46	18.87	26.89	17.19	35.79	25.48	31.76	20.27	31.83	24.31	34.31	23.14	41.09	28.86	40.07	28.41

[a] O_3 readings from stations KT and SSP were excluded from the study because <75% of hourly data were available.

Table 6. Absolute and Relative Change in Pollutant Concentration at Each Monitoring Station

		Absolute Change ($\mu g/m^3$)[a]					Relative Change (%)[b]				
	Pre-Intervention Mean ± SD	July 1990–June 1991	July 1991–June 1992	July 1992–June 1993	July 1993–June 1994	July 1994–June 1995	July 1990–June 1991	July 1991–June 1992	July 1992–June 1993	July 1993–June 1994	July 1994–June 1995
Central/Western											
NO_2	56.05 ± 33.25	−14.58	−3.01	−8.20	−3.62	−5.93	−26.01	−5.36	−14.64	−6.46	−10.58
SO_2	16.95 ± 14.75	0.44	3.98	6.50	4.14	1.82	2.61	23.46	38.33	24.45	10.74
PM_{10}	58.01 ± 33.86	0.10	−4.71	1.15	−1.52	−3.52	0.17	−8.11	1.98	−2.62	−6.07
O_3	30.72 ± 20.10	2.81	14.96	8.83	9.84	5.04	9.14	48.69	28.75	32.04	16.40
Kwai Chung											
NO_2	36.07 ± 14.86	3.37	8.52	11.43	9.89	9.98	9.34	23.63	31.68	27.41	27.67
SO_2	113.80 ± 61.78	−91.35	−92.84	−84.37	−84.25	−91.84	−80.24	−81.55	−74.10	−74.00	−80.67
PM_{10}	60.85 ± 28.74	−7.30	−8.31	−6.53	−5.94	−10.89	−12.00	−13.65	−10.72	−9.76	−17.89
O_3	23.28 ± 31.58	6.88	4.61	7.31	22.13	20.34	29.56	19.79	31.40	95.09	87.38
Kwun Tong[c]											
NO_2	89.61 ± 59.00	−34.31	−21.64	−16.20	−28.12	−31.42	−38.28	−24.15	−18.07	−31.38	−35.06
SO_2	50.25 ± 75.36	−25.26	−29.14	−22.45	−24.53	−32.33	−50.26	−57.98	−44.68	−48.81	−64.33
PM_{10}	61.53 ± 31.48	3.81	5.85	11.42	−3.37	−3.98	6.20	9.50	18.55	−5.48	−6.46
O_3	—	—	—	—	—	—	—	—	—	—	—
Sham Shui Po[c]											
NO_2	56.59 ± 26.85	−5.61	−4.84	4.73	5.91	10.04	−9.91	−8.55	8.35	10.45	17.75
SO_2	16.74 ± 14.34	−8.99	−2.70	0.16	11.26	8.87	−53.69	−16.16	0.98	67.27	52.97
PM_{10}	59.21 ± 28.83	5.39	6.34	14.06	4.17	−4.21	9.10	10.70	23.75	7.05	−7.11
O_3	—	—	—	—	—	—	—	—	—	—	—
Tsuen Wan											
NO_2	62.57 ± 24.63	−8.84	−0.75	−3.31	−4.03	−2.93	−14.12	−1.20	−5.29	−6.44	−4.68
SO_2	35.90 ± 36.60	−4.65	2.01	7.36	3.05	−8.90	−12.96	5.60	20.50	8.50	−24.79
PM_{10}	57.27 ± 29.30	−2.84	1.75	2.00	4.90	3.39	−4.95	3.06	3.49	8.56	5.92
O_3	29.25 ± 17.72	3.19	3.60	5.99	−14.77	—	10.91	12.32	20.47	−50.50	—
Overall											
NO_2	70.65 ± 39.15	−22.59	8.15	1.23	−0.81	0.08	−31.97	16.96	2.19	−1.41	0.14
SO_2	36.33 ± 31.55	−15.57	2.24	4.09	1.32	−6.22	−42.86	10.79	17.78	4.87	−21.89
PM_{10}	58.87 ± 27.57	0.44	0.59	5.20	1.86	−1.12	0.76	1.00	8.84	3.16	−1.90
O_3	30.46 ± 18.87	1.30	0.07	2.48	6.78	−1.02	4.27	0.22	7.79	19.76	−2.48

[a] Absolute change = annual mean level in post-intervention period − mean in pre-intervention period.

[b] Relative change = (annual mean level in post-intervention period − mean in pre-intervention period)/mean level in pre-intervention period × 100.

[c] O_3 readings from stations KT and SSP were excluded from the study because < 75% of hourly data were available.

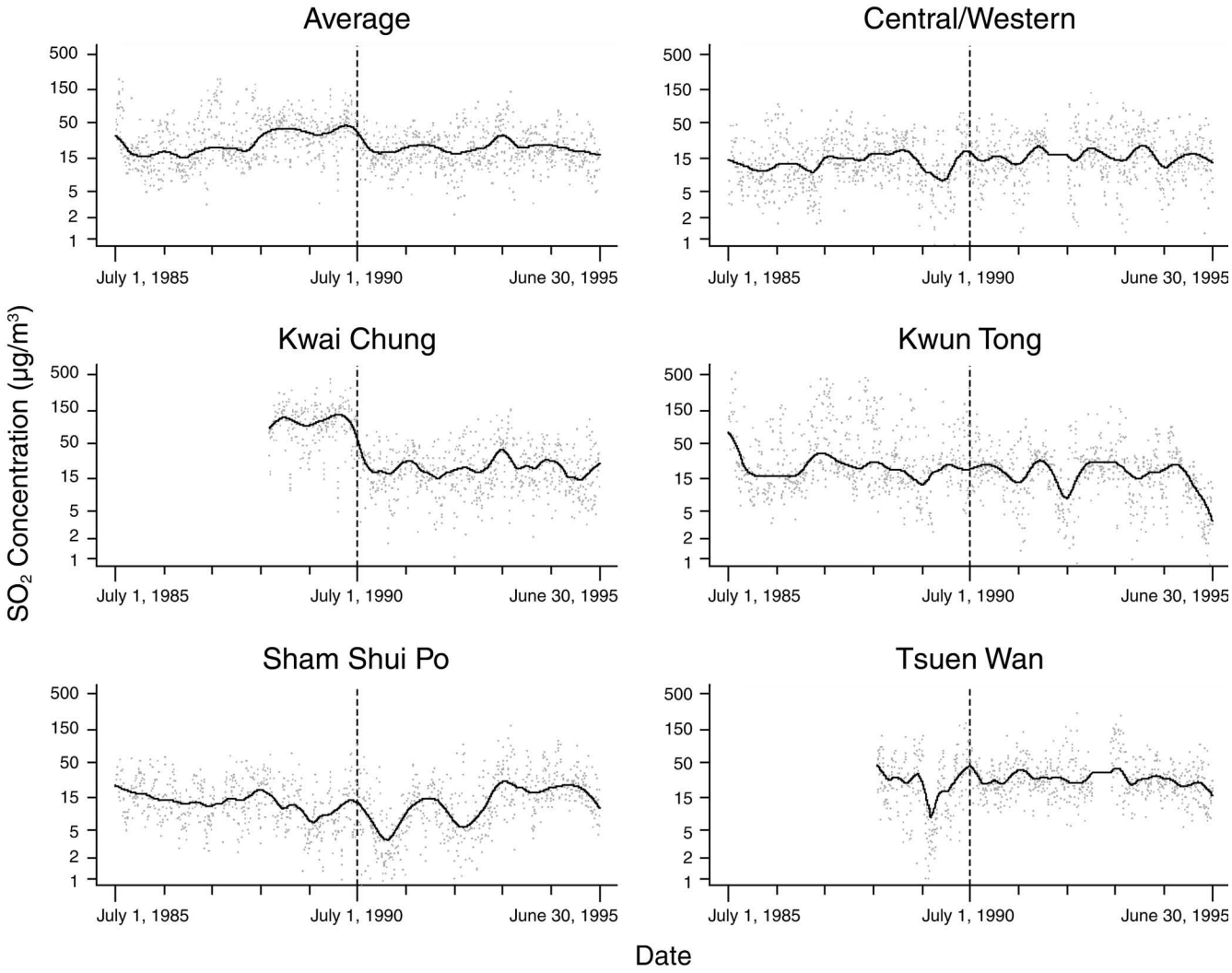

Figure 6. Time-series plot of the average daily concentrations of SO_2 ($\mu g/m^3$) from all stations in the study. The solid line represents the smoothed curve of the observed concentrations of the pollutant. The dotted line marks the beginning of the sulfur fuel intervention.

PM_{10} Mean PM_{10} concentrations at all stations did not decrease in all five years after the post-intervention period. There was little variation across stations in the pre-intervention period (ranging from 57 μg/m^3 at TW to 62 μg/m^3 at KT) (Tables 5 and 6). After the intervention, KC (in an industrial area) was the only station to show a consistent decrease (13% on average) for all 5 years (Table 6) (Figure 7).

O_3 O_3 showed a different pattern compared with the other gaseous pollutants in that its concentration increased after the intervention. The relative change in O_3 concentrations increased from 0.2% to 20% until the fifth year after the intervention. The standard deviation also increased after the intervention; however, not all stations showed the same pattern (Table 6).

PM_{10} Species

Al Mean ambient Al concentrations at all stations ranged from 311 ng/m^3 to 435 ng/m^3 in the pre-intervention period (Tables 7 and 8). Mean levels declined at all stations in the post-intervention period (Table 8). At stations CW and KT, there were significant mean differences between pre- and post-intervention periods (Table 8).

In the first year after the intervention, mean levels of Al at all stations decreased, ranging from a drop of 78 ng/m^3 at station SSP to 215 ng/m^3 at station KT, compared with those in the pre-intervention period (Table 7) (Figure 8). At stations CW and KT, the decreases in annual means observed during each of the five post-intervention years ranged from 8% to 31% and 31% to 49%, respectively.

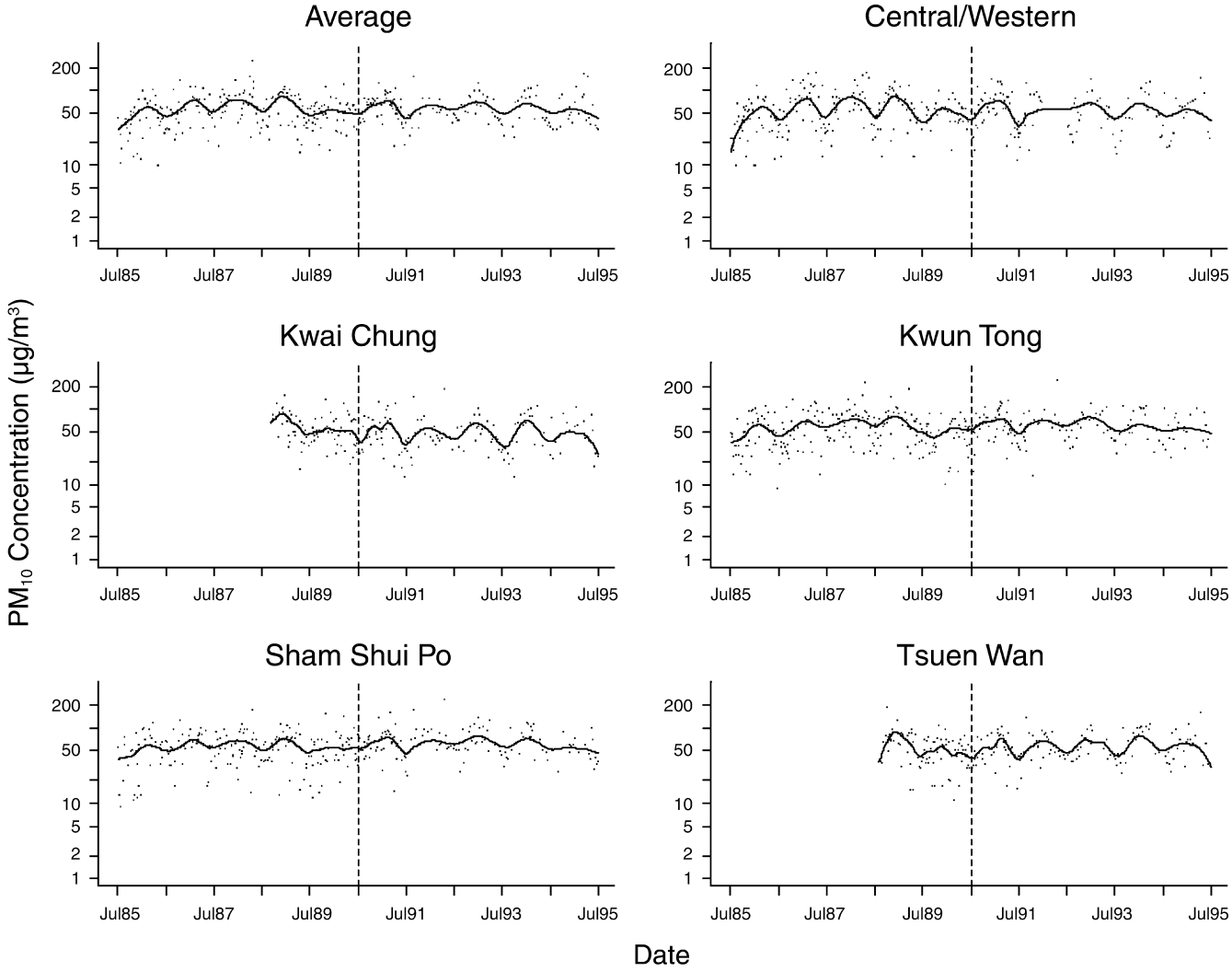

Figure 7. Time-series plot of the average daily concentrations of PM_{10} (μg/m^3) from the five stations, as well as the average of all five. The solid line represents the smoothed curve of the observed concentrations of the pollutants. The dotted line marks the beginning of the sulfur fuel intervention.

Table 7. Absolute and Relative Change in PM Chemical Species Concentration at Each Monitoring Station

		Absolute Change (ng/m^3)[a]					Relative Change (%)[b]				
	Pre-Intervention Mean ± SD	July 1990–June 1991	July 1991–June 1992	July 1992–June 1993	July 1993–June 1994	July 1994–June 1995	July 1990–June 1991	July 1991–June 1992	July 1992–June 1993	July 1993–June 1994	July 1994–June 1995
Central/Western											
Al	427.47 ± 399.83	−132.22	−78.81	−36.26	−52.70	−62.39	−30.93	−18.44	−8.48	−12.33	−14.59
Fe	595.78 ± 423.72	−178.33	−119.78	−71.20	6.80	−1.46	−29.93	−20.10	−11.95	1.14	−0.25
Mn	21.58 ± 15.70	−8.26	−3.67	−2.54	−0.98	−2.32	−38.29	−16.99	−11.77	−4.53	−10.73
Ni	5.47 ± 3.50	−4.11	−0.93	−1.96	−2.32	−2.32	−75.20	−16.96	−35.81	−42.41	−42.43
V	10.77 ± 6.58	−8.25	1.56	2.71	1.56	−2.08	−76.56	14.50	25.13	14.50	−19.29
Pb	69.62 ± 35.82	−15.16	−26.82	−13.70	−6.60	−8.25	−21.77	−38.53	−19.67	−9.47	−11.85
Zn	118.86 ± 80.11	−33.17	−30.56	−25.74	−15.86	−12.40	−27.90	−25.71	−21.66	−13.34	−10.43
Kwai Chung											
Al	311.22 ± 234.78	−99.58	−35.86	−2.34	30.83	7.72	−32.00	−11.52	−0.75	9.91	2.48
Fe	511.41 ± 290.53	−170.94	−61.41	−63.68	72.96	50.59	−33.43	−12.01	−12.45	14.27	9.89
Mn	15.14 ± 10.94	−5.63	−0.70	1.22	3.51	2.07	−37.22	−4.59	8.07	23.19	13.66
Ni	21.60 ± 10.42	−17.29	−16.24	−14.88	−15.53	−18.02	−80.07	−75.18	−68.90	−71.92	−83.43
V	60.30 ± 27.36	−53.10	−48.62	−41.27	−41.19	−50.29	−88.06	−80.63	−68.43	−68.30	−83.40
Pb	59.16 ± 29.40	0.23	−6.46	−3.79	14.49	5.50	0.40	−10.92	−6.40	24.50	9.29
Zn	196.75 ± 129.17	−33.28	−45.66	−94.90	−91.56	−93.38	−16.92	−23.21	−48.23	−46.54	−47.46
Kwun Tong											
Al	435.05 ± 488.23	−214.91	−140.48	−132.69	−157.51	−173.48	−49.40	−32.29	−30.50	−36.21	−39.88
Fe	653.15 ± 582.94	−72.30	−28.51	−55.06	−88.50	−85.11	−11.07	−4.37	−8.43	−13.55	−13.03
Mn	24.94 ± 28.52	−14.15	−5.98	−3.89	−6.95	−9.61	−56.75	−23.98	−15.61	−27.86	−38.54
Ni	9.15 ± 14.89	−6.12	−4.99	−4.01	−5.97	−5.80	−66.88	−54.47	−43.83	−65.20	−63.36
V	18.65 ± 35.35	−15.49	−7.71	−4.52	−6.49	−9.30	−83.04	−41.34	−24.21	−34.77	−49.87
Pb	75.14 ± 36.88	−3.11	−14.22	10.29	10.18	−7.76	−4.14	−18.92	13.69	13.55	−10.33
Zn	194.57 ± 180.33	−105.12	−98.50	−77.19	−66.15	−92.12	−54.03	−50.62	−39.67	−34.00	−47.34

(*Table continues on next page*)

[a] Absolute change = annual mean level in post-intervention period − mean in pre-intervention period.

[b] Relative change = (annual mean level in post-intervention period − mean in pre-intervention period)/mean level in pre-intervention period × 100.

Table 7 (*Continued*). Absolute and Relative Change in PM Chemical Species Concentration at Each Monitoring Station

		Absolute Change (ng/m^3)[a]					Relative Change (%)[b]				
	Pre-Intervention Mean ± SD	July 1990–June 1991	July 1991–June 1992	July 1992–June 1993	July 1993–June 1994	July 1994–June 1995	July 1990–June 1991	July 1991–June 1992	July 1992–June 1993	July 1993–June 1994	July 1994–June 1995
Sham Shui Po											
Al	370.91 ± 473.36	−78.49	−52.28	87.23	−13.72	−82.31	−21.16	−14.10	23.52	−3.70	−22.19
Fe	552.67 ± 527.13	−99.05	67.58	185.78	134.67	−0.22	−17.92	12.23	33.62	24.37	−0.04
Mn	19.03 ± 14.79	−5.55	10.41	11.58	5.86	−2.53	−29.18	54.71	60.85	30.77	−13.27
Ni	5.82 ± 4.14	−3.12	−0.85	−0.50	−1.19	−2.28	−53.56	−14.53	−8.58	−20.37	−39.12
V	8.52 ± 7.14	−6.04	1.05	4.59	5.26	1.34	−70.94	12.28	53.93	61.82	15.70
Pb	76.14 ± 48.34	−11.79	−18.94	−3.85	10.27	−14.88	−15.49	−24.87	−5.05	13.49	−19.55
Zn	110.89 ± 86.69	−35.57	−10.82	45.23	66.54	−14.94	−32.07	−9.76	40.79	60.01	−13.47
Tsuen Wan											
Al	310.66 ± 246.56	−129.98	13.95	−43.04	15.43	21.38	−41.84	4.49	−13.85	4.97	6.88
Fe	517.52 ± 347.21	−2.97	−78.49	−58.58	94.09	92.66	−0.57	−15.17	−11.32	18.18	17.91
Mn	16.03 ± 13.00	−7.21	−1.56	0.68	3.16	2.15	−45.00	−9.76	4.22	19.74	13.44
Ni	10.80 ± 6.42	−8.43	−5.07	−4.60	−5.79	−7.76	−78.03	−46.93	−42.55	−53.58	−71.87
V	28.32 ± 17.33	−25.18	−14.63	−8.48	−12.14	−19.66	−88.91	−51.65	−29.94	−42.86	−69.43
Pb	67.20 ± 33.94	−5.46	−6.31	2.67	10.12	3.78	−8.12	−9.38	3.97	15.05	5.62
Zn	94.82 ± 58.63	−34.60	−7.07	−1.00	0.69	9.29	−36.49	−7.45	−1.06	0.73	9.80
Overall											
Al	407.80 ± 461.69	−169.71	−122.57	−59.84	−59.13	−76.39	−41.62	−30.06	−14.67	−14.50	−18.73
Fe	598.50 ± 519.45	−134.74	−88.65	−41.00	27.42	3.78	−22.51	−14.81	−6.85	4.58	0.63
Mn	21.43 ± 16.81	−10.24	−2.85	−0.61	−0.76	−3.31	−47.80	−13.28	−2.82	−3.55	−15.45
Ni	8.57 ± 6.67	−5.70	−3.59	−3.19	−3.53	−5.35	−66.56	−41.93	−37.21	−41.14	−62.40
V	17.87 ± 16.51	−13.92	−6.27	−2.20	−1.75	−8.73	−77.92	−35.10	−12.33	−9.81	−48.85
Pb	74.43 ± 32.91	−12.43	−18.11	−7.57	5.91	−8.15	−16.70	−24.33	−10.17	7.93	−10.95
Zn	146.00 ± 88.61	−52.18	−41.02	−33.07	−18.06	−41.76	−35.74	−28.10	−22.65	−12.37	−28.60

[a] Absolute change = annual mean level in post-intervention period − mean in pre-intervention period.

[b] Relative change = (annual mean level in post-intervention period − mean in pre-intervention period)/mean level in pre-intervention period × 100.

Table 8. Mean Differences in PM Chemical Species Concentrations Between Pre- and Post-Intervention Periods at Each Monitoring Station

	Pre-Intervention Period		Post-Intervention Period		*t* Test		
	n[a]	Mean ± SD	n[a]	Mean ± SD	*t* Value	df	*P* Value
Central/Western							
Al	229	427.47 ± 399.83	232	356.21 ± 389.91	1.94	458	0.027
Fe	235	595.78 ± 423.72	232	527.49 ± 522.36	1.55	444	0.061
Mn	235	21.58 ± 15.70	232	17.98 ± 16.99	2.38	461	0.009
Ni	235	5.47 ± 3.50	232	2.90 ± 2.64	8.97	435	0.000
V	235	10.77 ± 6.58	232	9.52 ± 9.29	1.69	416	0.046
Pb	234	69.62 ± 35.82	232	57.44 ± 47.27	3.13	431	0.001
Zn	231	118.86 ± 80.11	232	96.00 ± 62.21	3.43	433	0.000
Kwai Chung							
Al	87	311.22 ± 234.78	260	292.93 ± 370.72	0.54	235	0.296
Fe	87	511.41 ± 290.53	260	478.35 ± 502.69	0.75	258	0.227
Mn	87	15.14 ± 10.94	260	15.31 ± 16.09	−0.11	218	0.545
Ni	87	21.60 ± 10.42	260	5.25 ± 5.47	14.01	102	0.000
V	87	60.30 ± 27.36	260	13.74 ± 15.06	15.13	104	0.000
Pb	87	59.16 ± 29.40	260	61.85 ± 53.66	−0.59	272	0.721
Zn	87	196.75 ± 129.17	260	122.80 ± 87.38	4.97	113	0.000
Kwun Tong							
Al	229	435.05 ± 488.23	263	270.13 ± 345.91	4.26	404	0.000
Fe	236	653.15 ± 582.94	263	585.54 ± 507.34	1.37	469	0.085
Mn	236	24.94 ± 28.52	263	16.74 ± 17.09	3.84	376	0.000
Ni	236	9.15 ± 14.89	263	3.77 ± 4.18	5.37	268	0.000
V	235	18.65 ± 35.35	263	9.91 ± 8.92	3.69	261	0.000
Pb	236	75.14 ± 36.88	263	75.18 ± 64.95	−0.01	423	0.504
Zn	231	194.57 ± 180.33	263	107.46 ± 106.43	6.42	362	0.000
Sham Shui Po							
Al	223	370.91 ± 473.36	261	347.49 ± 362.87	0.60	412	0.274
Fe	230	552.67 ± 527.13	261	613.22 ± 516.69	−1.28	479	0.900
Mn	230	19.03 ± 14.79	261	22.88 ± 19.65	−2.47	478	0.993
Ni	230	5.82 ± 4.14	261	4.22 ± 3.62	4.53	458	0.000
V	229	8.52 ± 7.14	261	9.80 ± 8.53	−1.81	487	0.964
Pb	230	76.14 ± 48.34	261	69.56 ± 58.50	1.36	487	0.087
Zn	223	110.89 ± 86.69	261	124.11 ± 94.70	−1.60	480	0.945
Tsuen Wan							
Al	96	310.66 ± 246.56	259	283.41 ± 381.19	0.79	262	0.216
Fe	96	517.52 ± 347.21	259	534.16 ± 739.58	−0.29	335	0.613
Mn	96	16.03 ± 13.00	259	15.41 ± 15.82	0.37	205	0.354
Ni	96	10.80 ± 6.42	259	4.31 ± 4.74	9.03	135	0.000
V	96	28.32 ± 17.33	259	11.86 ± 12.43	8.53	133	0.000
Pb	96	67.20 ± 33.94	259	68.45 ± 53.59	−0.26	268	0.602
Zn	96	94.82 ± 58.63	259	87.80 ± 63.27	0.98	182	0.164

[a] n = number of measurements.

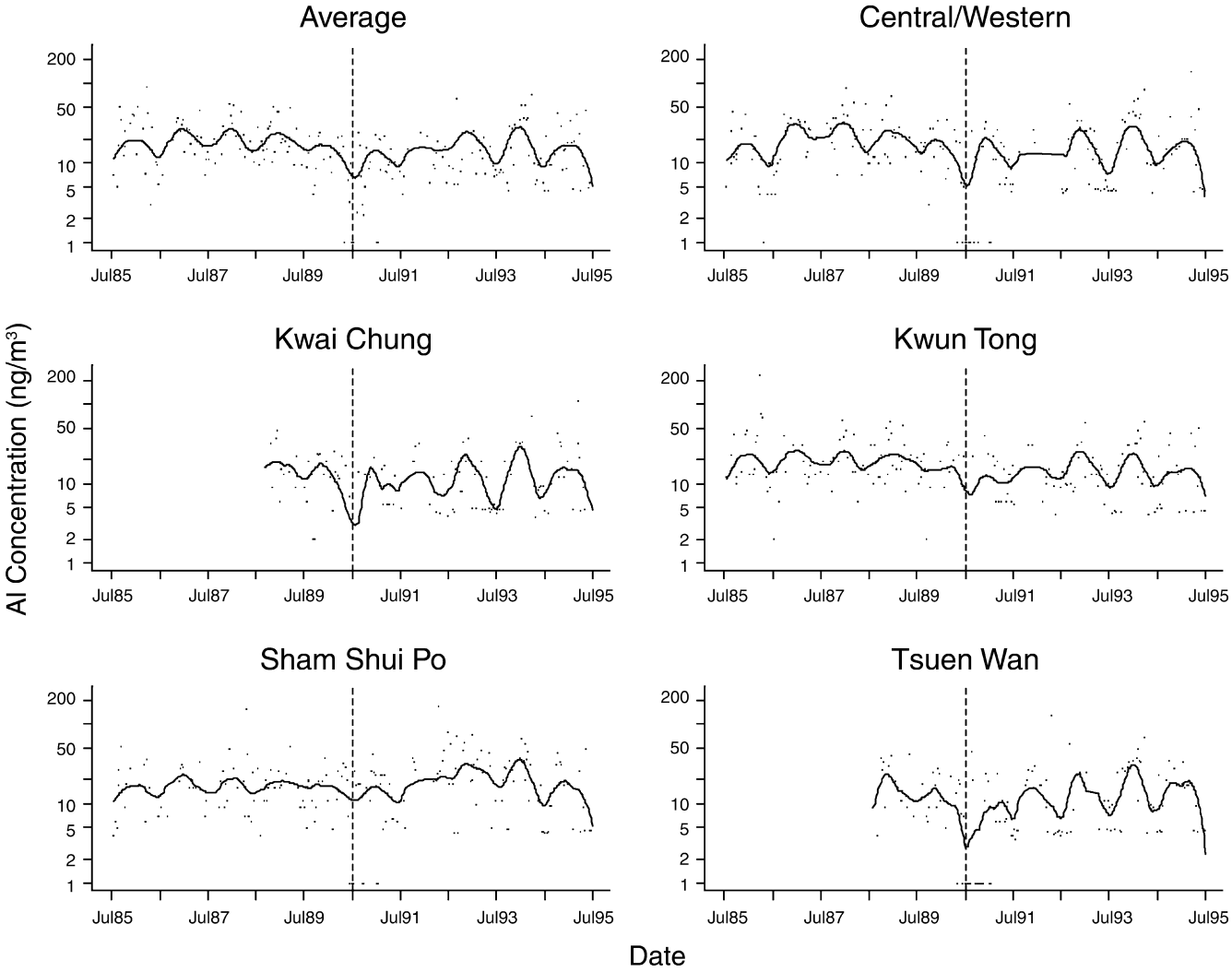

Figure 8. Time-series plot of the average daily concentrations of Al (ng/m^3) from the five stations, as well as the average of all five. The dotted line marks the beginning of the sulfur fuel intervention.

In analyses based on multiple comparisons using Tukey's HSD, only station KT showed a significant mean difference in the first year of the post-intervention period compared with that in the pre-intervention period. There were no significant mean differences in the sequential comparisons in all 5 years during the post-intervention periods (Table 9).

Fe The mean ambient Fe concentrations at all stations ranged from 511 ng/m^3 to 653 ng/m^3 in the pre-intervention period (Table 7) (Figure 9). The mean concentrations dropped at stations CW, KC, and KT; however, no statistically significant mean differences were detected in mean concentrations at any stations between the pre- and post-intervention periods (Table 8).

When compared with values in the pre-intervention period, the annual mean concentrations of Fe declined over a wide range, from 3 ng/m^3 at station TW to 178 ng/m^3 at station CW in the first year of the post-intervention period (Table 7). Stations KC and TW showed a rebound to a higher mean level 3 years after the intervention. Station SSP showed an increase in mean concentration after a year of the intervention, peaking in 1992 (Table 7). No significant mean differences were found in multiple comparisons at all stations as a result of Tukey's HSD analyses (Table 9).

Table 9. Multiple Comparisons Using Tukey's HSD of Mean Concentrations of PM Chemical Species (CS) for Pre-Intervention Period (B) Versus Each of the 5 Years (A_1, A_2, A_3, A_4, A_5) in Post-Intervention Period at Each Monitoring Station[a,b]

PM CS/ Comparison of Years	Central/Western		Kwai Chung		Kwun Tong		Sham Shui Po		Tsuen Wan	
	Difference	*P* Value	Difference	*P* Value	Difference	*P* Value	Difference	*P* Value	Difference	*P* Value
Al										
$A_1 - B$	−132.22	0.221	−99.58	0.531	−214.91	**0.008**	−78.49	0.797	−129.98	0.215
$A_2 - B$	−78.81	0.976	−35.86	0.995	−140.48	0.358	−52.28	0.978	13.95	1.000
$A_3 - B$	−36.26	0.988	−2.34	1.000	−132.69	0.262	87.23	0.714	−43.04	0.983
$A_4 - B$	−52.70	0.954	30.83	0.995	−157.51	0.120	−13.72	1.000	15.43	1.000
$A_5 - B$	−62.39	0.920	7.72	1.000	−173.48	0.083	−82.31	0.832	21.38	0.999
Fe										
$A_1 - B$	−178.33	0.118	−170.94	0.243	−72.30	0.947	−99.05	0.784	−2.97	1.000
$A_2 - B$	−119.78	0.934	−61.41	0.985	−28.51	1.000	67.58	0.974	−78.49	0.988
$A_3 - B$	−71.20	0.903	−63.68	0.962	−55.06	0.983	185.78	0.144	−58.58	0.996
$A_4 - B$	6.80	1.000	72.96	0.933	−88.50	0.885	134.67	0.471	94.09	0.958
$A_5 - B$	−1.46	1.000	50.59	0.989	−85.11	0.915	−0.22	1.000	92.66	0.960
Mn										
$A_1 - B$	−8.26	**0.009**	−5.63	0.229	−14.15	**0.001**	−5.55	0.221	−7.21	**0.040**
$A_2 - B$	−3.67	0.959	−0.70	1.000	−5.98	0.647	10.41	**0.005**	−1.56	0.993
$A_3 - B$	−2.54	0.888	1.22	0.996	−3.89	0.861	11.58	**0.000**	0.68	1.000
$A_4 - B$	−0.98	0.999	3.51	0.722	−6.95	0.333	5.86	0.160	3.16	0.801
$A_5 - B$	−2.32	0.947	2.07	0.969	−9.61	0.079	−2.53	0.942	2.15	0.955
Ni										
$A_1 - B$	−4.11	**0.000**	−17.29	**0.000**	−6.12	**0.002**	−3.12	**0.000**	−8.43	**0.000**
$A_2 - B$	−0.93	0.862	−16.24	**0.000**	−4.99	0.067	−0.85	0.789	−5.07	**0.000**
$A_3 - B$	−1.96	**0.000**	−14.88	**0.000**	−4.01	0.110	−0.50	0.949	−4.60	**0.000**
$A_4 - B$	−2.32	**0.000**	−15.53	**0.000**	−5.97	**0.003**	−1.19	0.268	−5.79	**0.000**
$A_5 - B$	−2.32	**0.000**	−18.02	**0.000**	−5.80	**0.007**	−2.28	**0.004**	−7.76	**0.000**
V										
$A_1 - B$	−8.25	**0.000**	−53.10	**0.000**	−15.49	**0.000**	−6.04	**0.000**	−25.18	**0.000**
$A_2 - B$	1.56	0.970	−48.62	**0.000**	−7.71	0.456	1.05	0.961	−14.63	**0.000**
$A_3 - B$	2.71	0.121	−41.27	**0.000**	−4.52	0.823	4.59	**0.000**	−8.48	**0.004**
$A_4 - B$	1.56	0.750	−41.19	**0.000**	−6.49	0.506	5.26	**0.000**	−12.14	**0.000**
$A_5 - B$	−2.08	0.497	−50.29	**0.000**	−9.30	0.158	1.34	0.872	−19.66	**0.000**
Pb										
$A_1 - B$	−15.16	0.148	0.23	1.000	−3.11	0.999	−11.79	0.667	−5.46	0.985
$A_2 - B$	−26.82	0.158	−6.46	0.986	−14.22	0.615	−18.94	0.310	−6.31	0.983
$A_3 - B$	−13.70	0.208	−3.79	0.997	10.29	0.776	−3.85	0.997	2.67	1.000
$A_4 - B$	−6.60	0.909	14.49	0.488	10.18	0.793	10.27	0.774	10.12	0.824
$A_5 - B$	−8.25	0.816	5.50	0.988	−7.76	0.935	−14.88	0.531	3.78	0.997
Zn										
$A_1 - B$	−33.17	**0.025**	−33.28	0.346	−105.12	**0.000**	−35.57	0.061	−34.60	**0.009**
$A_2 - B$	−30.56	0.600	−45.66	0.179	−98.50	**0.001**	−10.82	0.978	−7.07	0.989
$A_3 - B$	−25.74	0.129	−94.90	**0.000**	−77.19	**0.005**	45.23	**0.006**	−1.00	1.000
$A_4 - B$	−15.86	0.703	−91.56	**0.000**	−66.15	**0.030**	66.54	**0.000**	0.69	1.000
$A_5 - B$	−12.40	0.886	−93.38	**0.000**	−92.12	**0.001**	−14.94	0.898	9.29	0.945

[a] Boldface indicates statistically significant; *P* value ≤ 0.05.

[b] B: 1 July 1985–30 June 1990; A_1: 1 July 1990–30 June 1991; A_2: 1 July 1991–30 June 1992; A_3: 1 July 1992–30 June 1993; A_4: 1 July 1993–30 June 1994; A_5: 1 July 1994–30 June 1995.

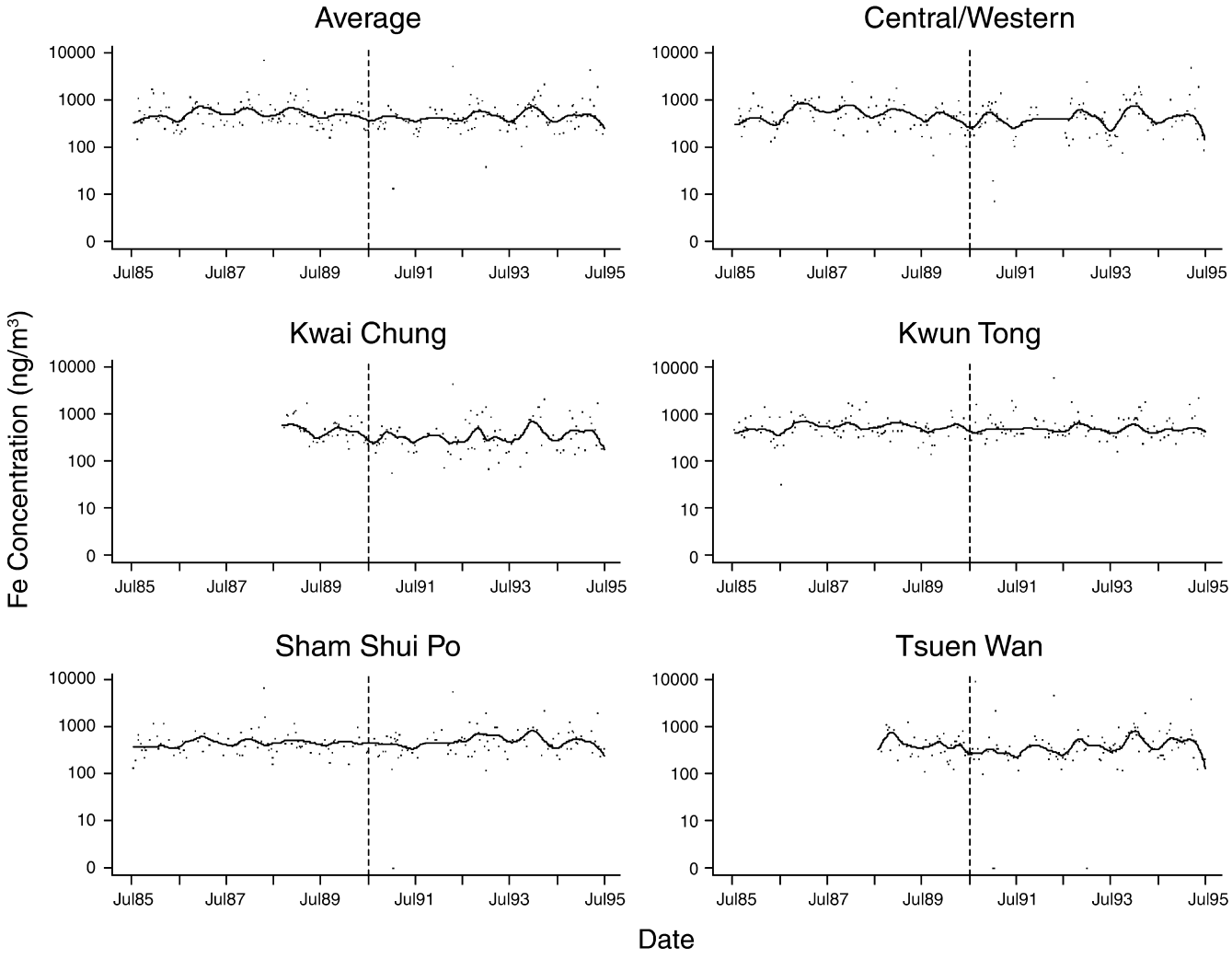

Figure 9. Time-series plot of the average daily concentrations of Fe (ng/m^3) from the five stations, as well as the average of all five. The dotted line marks the beginning of the sulfur fuel intervention.

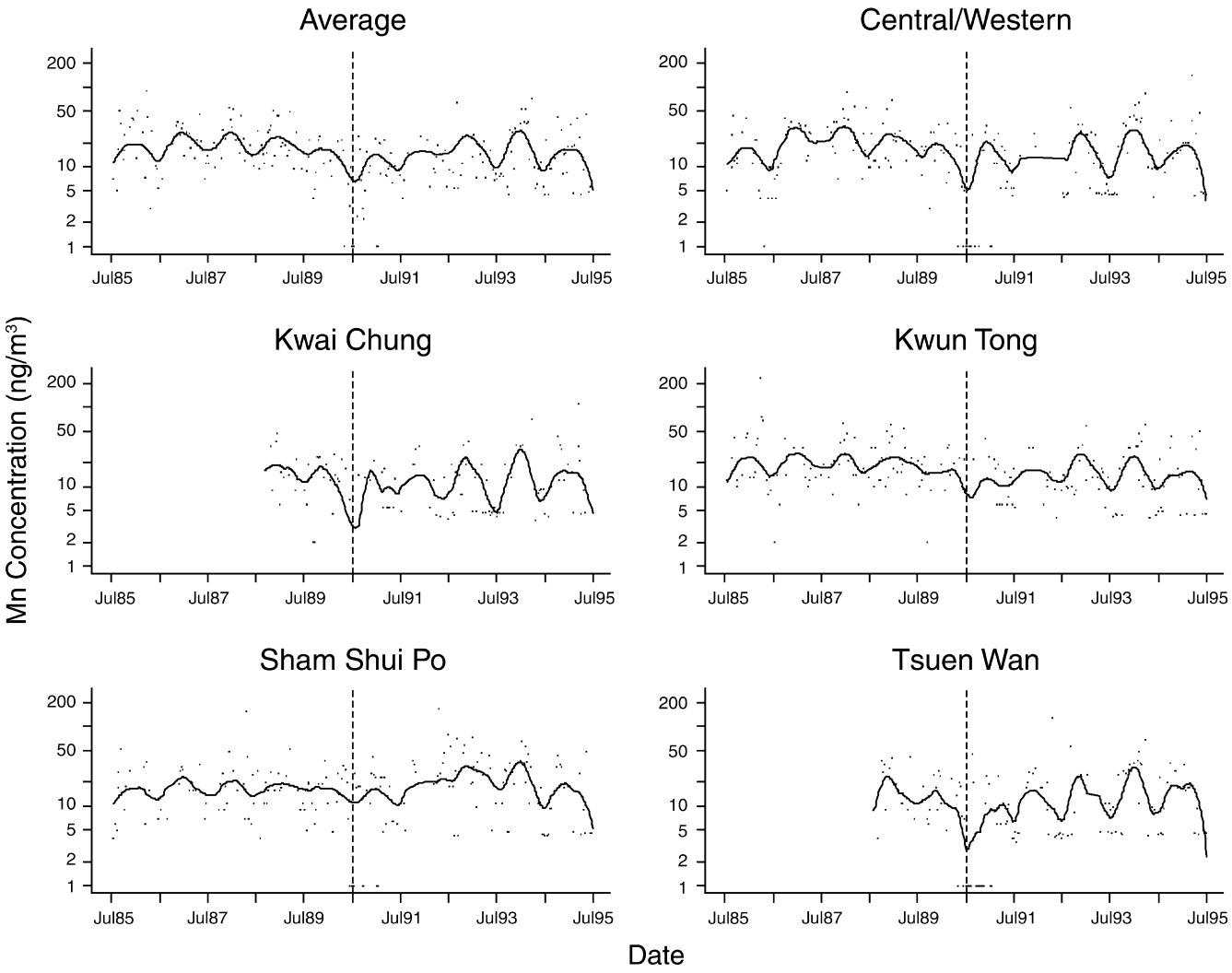

Figure 10. Time-series plot of the average daily concentrations of Mn (ng/m^3) from the five stations, as well as the average of all five. The dotted line marks the beginning of the sulfur fuel intervention.

Mn Mean ambient Mn concentrations at all stations ranged from 15 ng/m^3 to 25 ng/m^3 in the pre-intervention period and from 15 ng/m^3 to 23 ng/m^3 in the post-intervention period (Table 7) (Figure 10). Stations CW and KT showed statistically significant differences in mean concentrations between the pre- and post-intervention periods (Table 8).

With reference to the pre-intervention period, the greatest absolute decrease in Mn mean concentration was in the first year of the post-intervention period at all stations (Table 7). However, only stations CW, KT, and TW showed statistically significant mean differences in the first year of the post-intervention period (Table 9). Both station CW and station KT maintained lower annual mean levels over 5 years after the intervention. Station SSP showed a biphasic pattern with a significant peak in 1992. No significant comparisons were found in sequential tests on the means, except at station SSP, between the first and second year in the post-intervention period (Table 9).

Ni Mean ambient Ni concentrations at all stations ranged from 5 ng/m^3 to 22 ng/m^3 in the pre-intervention period (Tables 7 and 8) (Figure 11). All stations demonstrated statistically significant decreases in means in the 5-year post-intervention period compared with the 5-year pre-intervention period (Table 8).

The smallest drop in means occurred in July 1992–June 1993 except at station CW in 1991 (Table 7). The annual

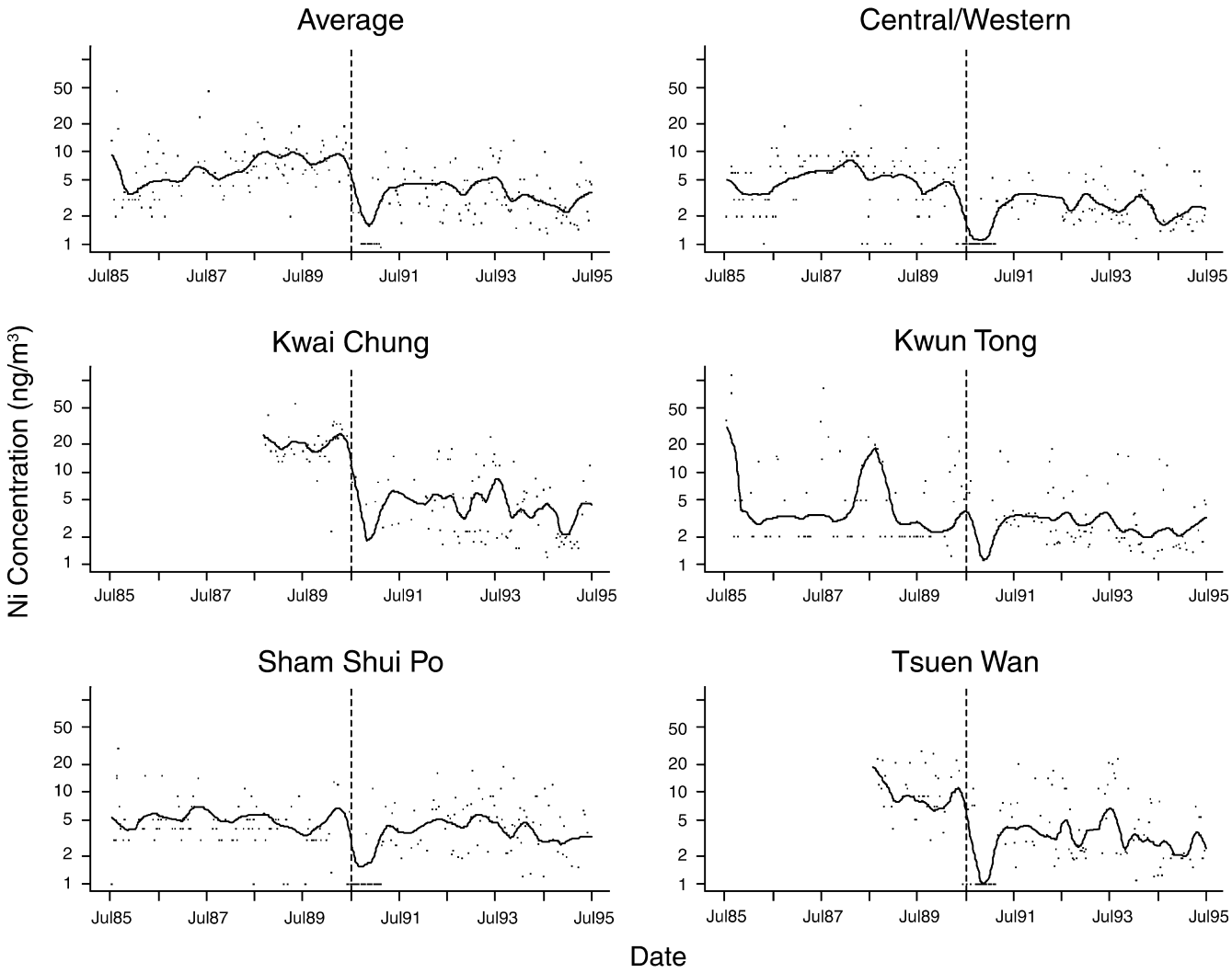

Figure 11. Time-series plot of the average daily concentrations of Ni (ng/m^3) from the five stations, as well as the average of all five. The dotted line marks the beginning of the sulfur fuel intervention.

means at all stations showed a decrease in all 5 years of the post-intervention period compared with the pre-intervention period, but only stations KC and TW showed significant mean differences across all 5 years of the post-intervention period (Table 9). The annual mean levels of Ni at stations CW, SSP, and KT decreased significantly in the first year after the intervention. In the second year of the post-intervention period, there were significant increases in means compared with the first year of the post-intervention at stations CW, SSP, and TW (Table 10).

V Mean ambient V concentrations at all stations ranged from 9 ng/m^3 to 60 ng/m^3 in the pre-intervention period (Tables 7 and 8) (Figure 12). Mean concentrations of V at all stations declined statistically significantly after the intervention except at station SSP.

All stations showed a convex curve at peak in 1992 or 1993 (Table 7). Mean V concentrations at stations KC, KT, and TW were all lower in all 5 years of the post-intervention period compared with the pre-intervention period. Stations KC and TW showed significant mean differences in all 5 years of the post-intervention period, while stations CW, KT, and SSP showed significant mean differences in the first year of the post-intervention period compared with the pre-intervention period (Table 10).

Table 10. Multiple Comparisons Using Tukey's HSD of Mean Concentrations of PM Chemical Species (CS) Between Two Sequential Years in the 5-Year (A_1, A_2, A_3, A_4, A_5) Post-Intervention Period at Each Monitoring Station[a,b]

PM CS/ Comparison of Years	Central/Western		Kwai Chung		Kwun Tong		Sham Shui Po		Tsuen Wan	
	Difference	*P* Value	Difference	*P* Value	Difference	*P* Value	Difference	*P* Value	Difference	*P* Value
Al										
$A_2 - A_1$	53.42	0.997	63.71	0.954	74.43	0.954	26.21	1.000	143.93	0.326
$A_3 - A_2$	42.55	0.999	33.52	0.997	7.79	1.000	139.51	0.579	−56.99	0.973
$A_4 - A_3$	−16.44	1.000	33.17	0.995	−24.83	1.000	−100.95	0.776	58.47	0.958
$A_5 - A_4$	−9.69	1.000	−23.11	0.999	−15.97	1.000	−68.58	0.961	5.95	1.000
Fe										
$A_2 - A_1$	58.55	0.998	109.54	0.874	43.79	0.999	166.63	0.622	−75.52	0.993
$A_3 - A_2$	48.57	0.999	−2.27	1.000	−26.55	1.000	118.20	0.877	19.91	1.000
$A_4 - A_3$	78.00	0.953	136.64	0.578	−33.44	1.000	−51.11	0.995	152.67	0.850
$A_5 - A_4$	−8.26	1.000	−22.37	1.000	3.40	1.000	−134.89	0.774	−1.43	1.000
Mn										
$A_2 - A_1$	4.60	0.928	4.94	0.632	8.17	0.517	15.97	**0.000**	5.65	0.420
$A_3 - A_2$	1.13	1.000	1.92	0.990	2.09	0.998	1.17	0.999	2.24	0.981
$A_4 - A_3$	1.56	0.996	2.29	0.960	−3.05	0.981	−5.72	0.440	2.49	0.958
$A_5 - A_4$	−1.34	0.999	−1.44	0.996	−2.66	0.991	−8.38	0.121	−1.01	0.999
Ni										
$A_2 - A_1$	3.19	**0.005**	1.06	0.982	1.14	0.995	2.27	**0.045**	3.36	**0.017**
$A_3 - A_2$	−1.03	0.848	1.36	0.944	0.97	0.998	0.35	0.998	0.47	0.998
$A_4 - A_3$	−0.36	0.989	−0.65	0.996	−1.96	0.926	−0.69	0.925	−1.19	0.847
$A_5 - A_4$	0.00	1.000	−2.49	0.431	0.17	1.000	−1.09	0.697	−1.98	0.318
V										
$A_2 - A_1$	9.81	**0.000**	4.48	0.871	7.78	0.655	7.09	**0.000**	10.55	**0.001**
$A_3 - A_2$	1.15	0.995	7.35	0.426	3.20	0.989	3.55	0.172	6.15	0.236
$A_4 - A_3$	−1.15	0.965	0.08	1.000	−1.97	0.998	0.67	0.996	−3.66	0.714
$A_5 - A_4$	−3.64	0.148	−9.11	0.106	−2.82	0.992	−3.93	0.072	−7.53	**0.029**
Pb										
$A_2 - A_1$	−11.66	0.931	−6.69	0.988	−11.11	0.912	−7.14	0.987	−0.85	1.000
$A_3 - A_2$	13.13	0.887	2.67	1.000	24.50	0.216	15.09	0.746	8.97	0.957
$A_4 - A_3$	7.10	0.947	18.28	0.321	−0.11	1.000	14.12	0.710	7.45	0.973
$A_5 - A_4$	−1.65	1.000	−9.00	0.928	−17.94	0.507	−25.15	0.166	−6.34	0.984
Zn										
$A_2 - A_1$	2.60	1.000	−12.38	0.992	6.63	1.000	24.75	0.731	27.54	0.230
$A_3 - A_2$	4.82	1.000	−49.24	0.169	21.31	0.980	56.05	**0.021**	6.06	0.997
$A_4 - A_3$	9.89	0.978	3.34	1.000	11.04	0.999	21.31	0.763	1.69	1.000
$A_5 - A_4$	3.46	1.000	−1.82	1.000	−25.97	0.941	−81.48	**0.000**	8.60	0.976

[a] Boldface indicates statistically significant; *P* value ≤ 0.05.

[b] A_1: 1 July 1990–30 June 1991; A_2: 1 July 1991–30 June 1992; A_3: 1 July 1992–30 June 1993; A_4: 1 July 1993–30 June 1994; A_5: 1 July 1994–30 June 1995.

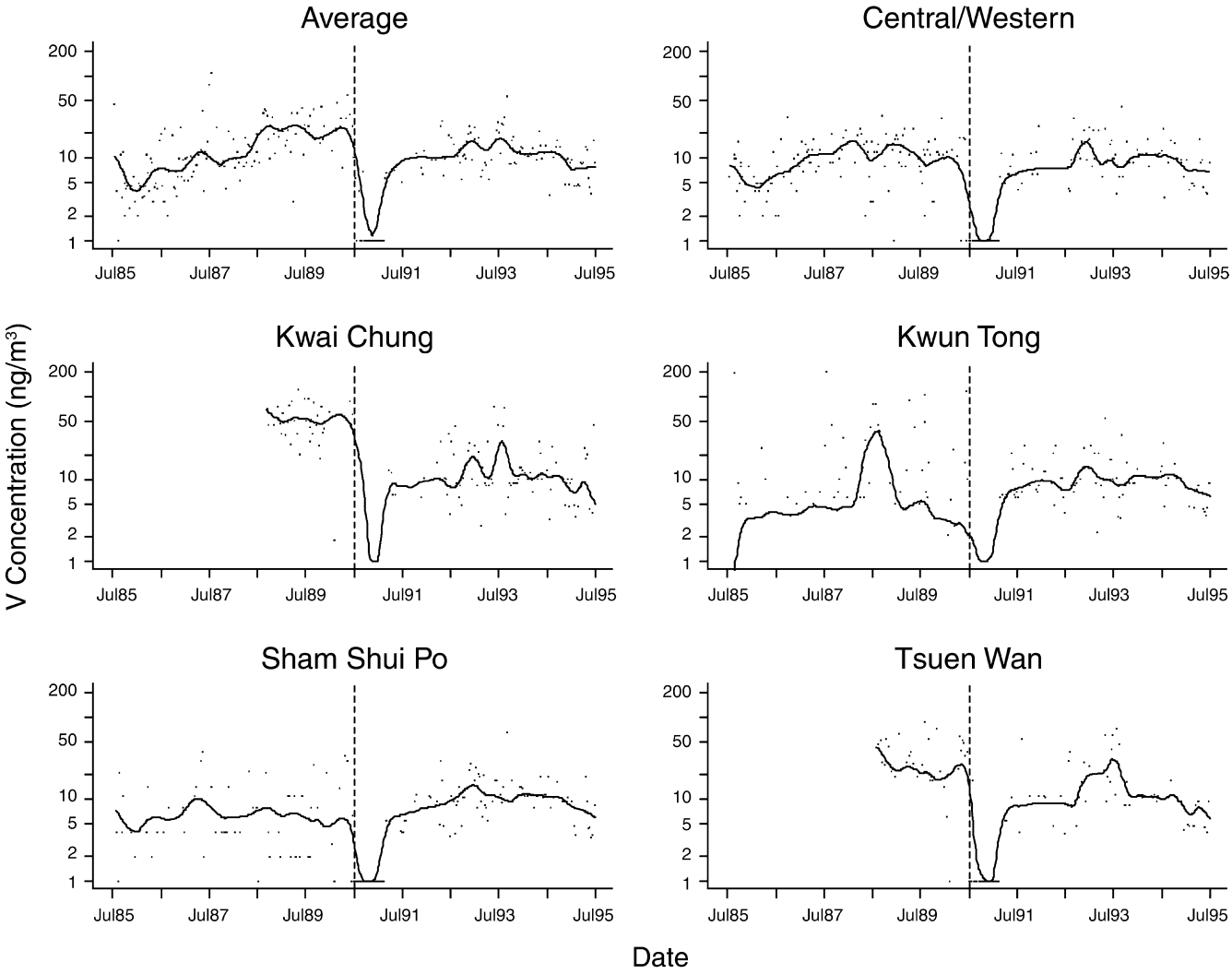

Figure 12. Time-series plot of the average daily concentrations of V (ng/m^3) from the five stations, as well as the average of all five. The dotted line marks the beginning of the sulfur fuel intervention.

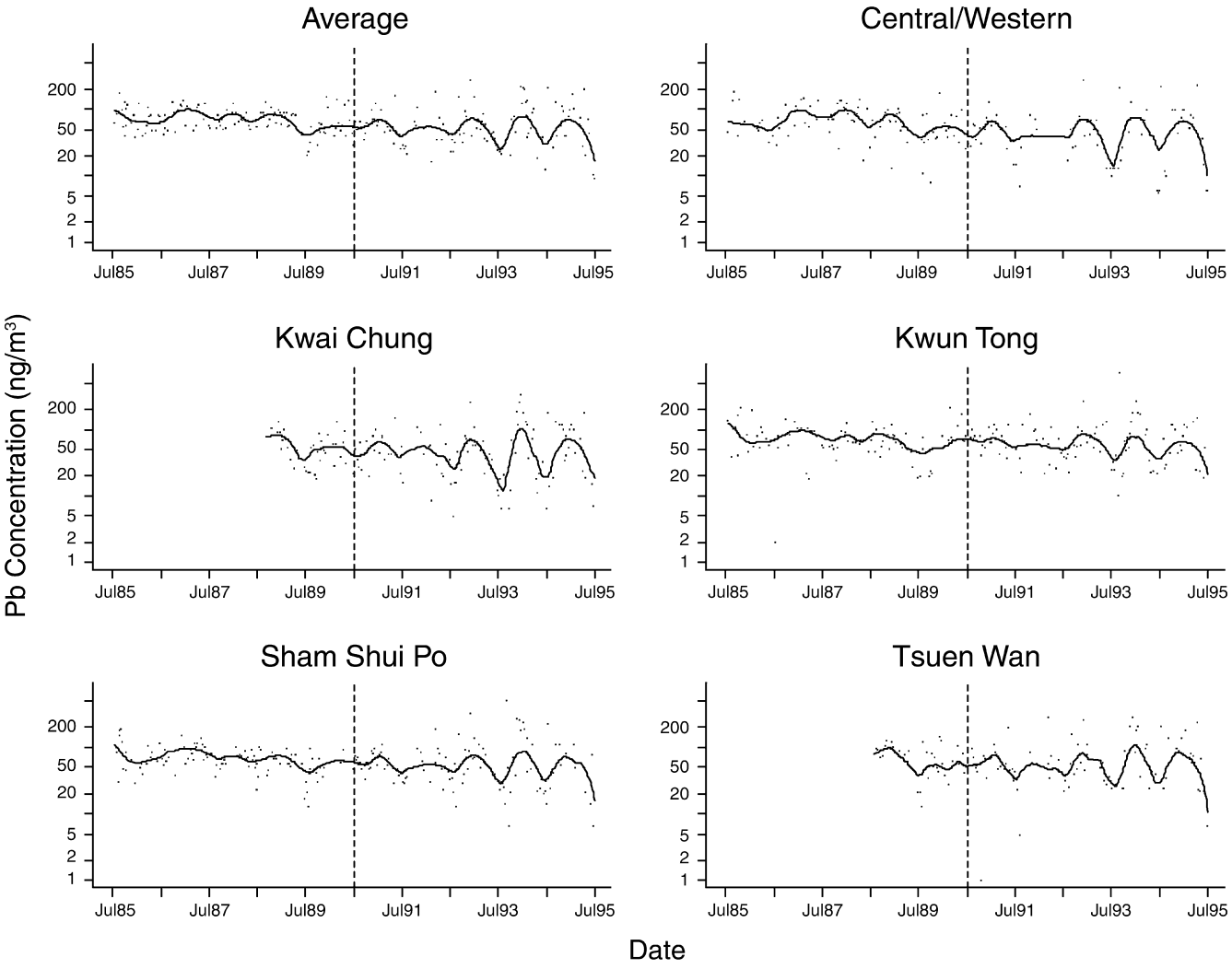

Figure 13. Time-series plot of the average daily concentrations of Pb (ng/m^3) at five stations, as well as the average of all five. The dotted line marks the beginning of the sulfur fuel intervention.

Pb Mean ambient Pb concentration at all stations ranged from 60 ng/m^3 to 76 ng/m^3 in the pre-intervention period. The means increased at stations KC, KT, and TW; however, none were significant (Tables 7 and 8) (Figure 13).

Mean levels at all stations dropped in the first two years of the post-intervention period except at station KC (Table 9). Only the annual mean at station CW was persistently lower across all 5 years in the post-intervention period compared with the pre-intervention period. None of the stations showed significant mean differences from the results of all multiple comparisons.

Zn Mean ambient Zn concentrations at all stations in the pre-intervention period ranged from 95 ng/m^3 to 197 ng/m^3 (Tables 7 and 8) (Figure 14). Mean levels at stations CW, KC, and KT dropped significantly by 19%, 38%, and 45%, respectively, after the intervention.

At all stations, the mean Zn concentrations decreased in the first 2 years of the post-intervention period (Table 9). However, significant mean differences were detected only at CW, KT, and TW in the first year of the post-intervention period. At station KT, Zn mean levels were significantly lower for all 5 years of the post-intervention period. Station KC showed significant decreases in annual means in the last three years of the post-intervention periods.

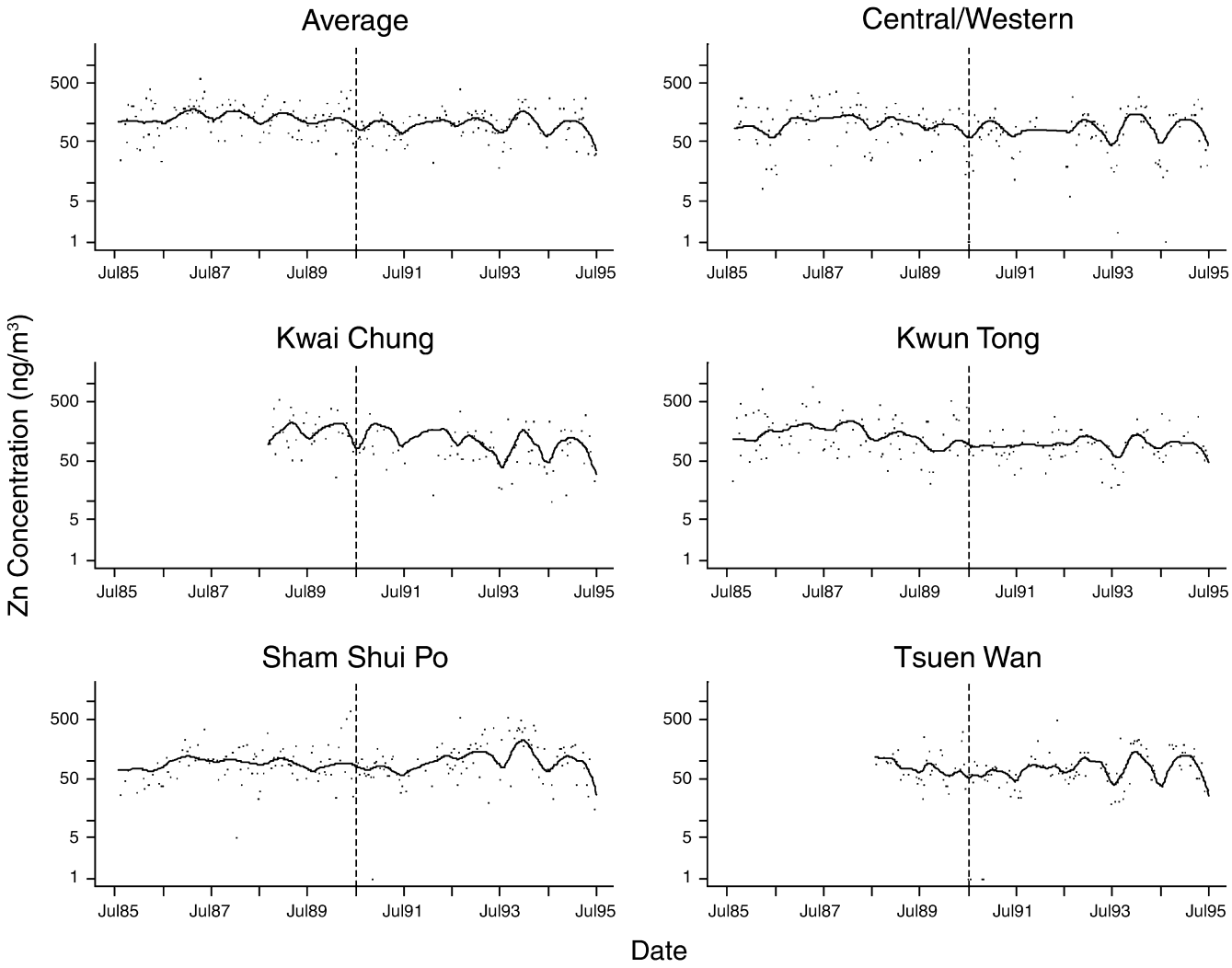

Figure 14. Time-series plot of the average daily concentrations of Zn (ng/m^3) from the five stations, as well as the average of all five. The dotted line marks the beginning of the sulfur fuel intervention.

Health Effects of Air Pollutants

Whole Assessment Period

Gaseous Pollutants In different assessment periods, impacts on mortality due to all natural causes, cardiovascular disease, and respiratory disease were positive for all three gaseous pollutants. In the 10-year assessment period for all ages (per 10-μg/m^3 unit change at lag 0–1 day), the mortality outcomes associated with NO_2 were a 0.85% ER for mortality due to all natural causes and 1.51% for respiratory disease; for SO_2 there was an ER of 0.42% for all natural causes and 1.31% for cardiovascular disease; for O_3 there was an ER of 0.64% for all natural causes and 1.01% for respiratory disease. In the 7-year assessment period (the sensitivity analysis), all excess risks were higher compared with those from the 10-year assessment period, except for SO_2 for cardiovascular disease at all ages and for O_3 for cardiovascular disease for those 65+ (Table 11).

PM_{10} and Its Chemical Species In general PM_{10} and most of its species were not significantly associated with an ER of mortality. However, in the 10-year assessment period, Ni and V were associated with ERs per 10 ng/m^3 of respiratory mortality of 3.1% and 1.4%, respectively, for all ages. There were also significant associations of Ni and V for the 65+ age group. Most ER estimates were higher in the 7-year (as a sensitivity analysis) than in the 10-year assessment period (Tables 11 and 12).

Table 11. Excess Risk of Mortality Due to Air Pollution from Criteria Pollutants in Assessment Periods of Different Lengths[a]

Disease / Age Group / Pollutant	7 Years ER (95% CI)	10 Years ER (95% CI)
All Natural Causes		
All ages		
NO_2	1.45 (1.04 to 1.85)	0.85 (0.46 to 1.25)
SO_2	0.52 (0.15 to 0.89)	0.42 (0.06 to 0.77)
PM_{10}	0.14 (−0.16 to 0.43)	0.09 (−0.12 to 0.30)
O_3	0.76 (0.42 to 1.10)	0.64 (0.35 to 0.94)
Cardiovascular		
All ages		
NO_2	1.69 (0.99 to 2.40)	0.61 (−0.20 to 1.41)
SO_2	1.22 (0.58 to 1.86)	1.31 (0.60 to 2.02)
PM_{10}	−0.16 (−0.76 to 0.46)	−0.26 (−0.67 to 0.18)
O_3	0.57 (−0.01 to 1.16)	0.08 (−0.53 to 0.70)
65+		
NO_2	1.74 (0.95 to 2.55)	1.34 (0.42 to 2.27)
SO_2	1.53 (0.80 to 2.26)	0.22 (−0.61 to 1.05)
PM_{10}	−0.06 (−0.70 to 0.59)	−0.23 (−0.71 to 0.25)
O_3	0.45 (−0.21 to 1.11)	1.04 (0.37 to 1.72)
Respiratory		
All ages		
NO_2	2.98 (2.03 to 3.93)	1.51 (0.52 to 2.51)
SO_2	0.89 (0.01 to 1.77)	0.63 (−0.27 to 1.54)
PM_{10}	0.38 (−0.12 to 0.87)	0.02 (−0.33 to 0.36)
O_3	1.97 (1.20 to 2.75)	1.01 (0.28 to 1.74)
65+		
NO_2	3.23 (2.22 to 4.26)	0.40 (−0.26 to 1.07)
SO_2	1.33 (0.39 to 2.29)	0.04 (−0.54 to 0.63)
PM_{10}	0.37 (−0.18 to 0.91)	−0.09 (−0.46 to 0.29)
O_3	1.82 (0.99 to 2.66)	0.50 (0.00 to 1.00)
Accidental		
All ages		
NO_2	0.50 (−1.12 to 2.14)	0.78 (0.18 to 1.37)
SO_2	1.53 (0.11 to 2.97)	0.51 (−0.08 to 1.11)
PM_{10}	0.73 (−0.35 to 1.82)	0.53 (−0.28 to 1.34)
O_3	0.64 (−0.66 to 1.95)	0.63 (−0.01 to 1.27)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day, except PM_{10} at current day.

Table 12. Excess Risk of Mortality Due to Air Pollution from PM Chemical Species in Assessment Periods of Different Lengths[a]

Disease / Age Group / Pollutant	7 Years ER (95% CI)	10 Years ER (95% CI)
All Natural Causes		
All ages		
Al	0.064 (−0.034 to 0.161)	0.154 (0.046 to 0.261)
Fe	0.006 (−0.013 to 0.024)	−0.001 (−0.015 to 0.013)
Mn	0.351 (−0.268 to 0.974)	0.032 (−0.421 to 0.488)
Ni	3.071 (0.999 to 5.185)	1.237 (−0.282 to 2.778)
V	0.864 (0.092 to 1.641)	0.455 (−0.170 to 1.084)
Pb	0.078 (−0.140 to 0.296)	−0.007 (−0.201 to 0.188)
Zn	−0.078 (−0.216 to 0.060)	−0.030 (−0.137 to 0.076)
65+		
Al	0.115 (−0.027 to 0.257)	0.055 (−0.010 to 0.119)
Fe	0.015 (−0.008 to 0.037)	0.012 (−0.003 to 0.027)
Mn	0.620 (−0.116 to 1.362)	0.513 (0.006 to 1.023)
Ni	3.992 (1.467 to 6.581)	2.139 (0.432 to 3.875)
V	1.008 (0.056 to 1.969)	0.605 (−0.105 to 1.319)
Pb	0.058 (−0.208 to 0.325)	−0.023 (−0.237 to 0.190)
Zn	−0.083 (−0.256 to 0.090)	−0.020 (−0.135 to 0.096)
Cardiovascular		
All ages		
Al	0.088 (−0.116 to 0.294)	0.037 (−0.078 to 0.153)
Fe	−0.034 (−0.072 to 0.005)	−0.013 (−0.040 to 0.013)
Mn	−0.867 (−2.152 to 0.435)	−0.196 (−1.094 to 0.711)
Ni	2.098 (−2.182 to 6.566)	0.069 (−2.913 to 3.141)
V	0.487 (−1.117 to 2.117)	−0.289 (−1.539 to 0.976)
Pb	−0.227 (−0.693 to 0.240)	−0.266 (−0.654 to 0.123)
Zn	−0.291 (−0.582 to 0.000)	−0.262 (−0.471 to −0.053)
65+		
Al	−0.028 (−0.189 to 0.133)	0.069 (−0.057 to 0.195)
Fe	−0.035 (−0.078 to 0.007)	−0.012 (−0.042 to 0.018)
Mn	−1.145 (−2.550 to 0.281)	−0.098 (−1.106 to 0.922)
Ni	1.944 (−2.552 to 6.648)	1.086 (−2.313 to 4.604)
V	0.502 (−1.223 to 2.258)	−0.023 (−1.442 to 1.416)
Pb	−0.265 (−0.742 to 0.215)	−0.310 (−0.743 to 0.124)
Zn	−0.351 (−0.655 to −0.047)	−0.249 (−0.483 to −0.014)

(*Table continues on next page*)

[a] Data are expressed in % per 10-ng/m^3 unit change.

Table 12 (*Continued*). Excess Risk of Mortality Due to Air Pollution from PM Chemical Species in Assessment Periods of Different Lengths[a]

Disease / Age Group / Pollutant	7 Years ER (95% CI)	10 Years ER (95% CI)
Respiratory		
All ages		
Al	−0.030 (−0.196 to 0.136)	0.016 (−0.117 to 0.149)
Fe	0.008 (−0.024 to 0.040)	−0.002 (−0.023 to 0.020)
Mn	0.125 (−0.953 to 1.215)	−0.011 (−0.732 to 0.714)
Ni	5.006 (1.203 to 8.952)	3.112 (0.588 to 5.699)
V	1.634 (0.239 to 3.049)	1.423 (0.407 to 2.449)
Pb	0.056 (−0.332 to 0.445)	−0.078 (−0.395 to 0.240)
Zn	−0.101 (−0.348 to 0.147)	−0.088 (−0.256 to 0.080)
65+		
Al	−0.063 (−0.199 to 0.074)	−0.015 (−0.118 to 0.088)
Fe	0.021 (−0.012 to 0.055)	0.012 (−0.011 to 0.035)
Mn	0.302 (−0.838 to 1.454)	0.191 (−0.584 to 0.972)
Ni	3.740 (−0.139 to 7.769)	3.206 (0.525 to 5.958)
V	1.476 (0.020 to 2.952)	1.536 (0.435 to 2.649)
Pb	−0.002 (−0.394 to 0.391)	−0.108 (−0.440 to 0.226)
Zn	−0.096 (−0.347 to 0.157)	−0.040 (−0.220 to 0.141)
Accidental		
All ages		
Al	−0.058 (−0.368 to 0.252)	0.175 (−0.084 to 0.435)
Fe	0.023 (−0.048 to 0.093)	0.019 (−0.034 to 0.073)
Mn	0.574 (−1.797 to 3.002)	0.481 (−1.224 to 2.215)
Ni	1.191 (−5.462 to 8.312)	1.506 (−3.259 to 6.506)
V	−0.532 (−3.107 to 2.112)	−0.024 (−2.054 to 2.048)
Pb	0.009 (−0.787 to 0.812)	−0.025 (−0.719 to 0.675)
Zn	−0.088 (−0.571 to 0.398)	−0.034 (−0.381 to 0.315)

[a] Data are expressed in % per 10-ng/m^3 unit change.

Pre- and Post-intervention Periods

Gaseous Pollutants In the pre-intervention assessment, we used two periods, the full 5-year period before the intervention and the last 2 years (as a sensitivity analysis). Only NO_2 showed significant associations in the pre-intervention assessment for both periods. In the 5-year pre-intervention assessment, NO_2 was associated with respiratory disease for both age groups with an ER of around 0.7% to 0.8%; and in the 2-year period (sensitivity analysis), a significant association between NO_2 and respiratory disease was found only for the 65+ age group, with an ER of 2.6% (Table 13).

In the post-intervention period, for both age groups, gaseous pollutants were significantly associated with an ER of mortality from all natural causes, and cardiovascular and respiratory diseases, with some exceptions: SO_2 was not significantly associated with respiratory deaths for all ages (but was for the 65+ group). NO_2, SO_2, and O_3 were not significantly associated with accidental deaths for all ages (Table 13).

PM_{10} and Its Chemical Species There was no significant association of PM_{10} with mortality due to all natural causes, cardiovascular disease, and respiratory disease for both the all-ages and 65+ groups in both the pre- and post-intervention periods (Table 13). With the exception of Ni and V, all chemical species in PM_{10} were not significantly associated with mortality in the 5-year pre-intervention period. In this period, both Ni and V were associated with mortality due to all natural causes for the 65+ age group, with an ER per 10 ng/m^3 of 3% for Ni and 1% for V; and with respiratory mortality for both age groups with an ER of 4% for Ni and 2% for V. In the 2-year pre-intervention period (sensitivity analysis), Ni and V showed a significant

Table 13. Short-Term Effects on Mortality Due to Air Pollution from Criteria Pollutants for Pre- and Post-Intervention Periods[a]

Disease / Age Group / Pollutant	Pre-Intervention (5-Year) ER (95% CI)	Pre-Intervention (2-Year) ER (95% CI)	Post-Intervention (5-Year) ER (95% CI)
All Natural Causes			
All ages			
NO_2	−0.19 (−0.50 to 0.13)	0.74 (−0.13 to 1.61)	1.30 (0.81 to 1.80)
SO_2	−0.08 (−0.37 to 0.21)	0.50 (−0.08 to 1.08)	0.98 (0.44 to 1.53)
PM_{10}	−0.01 (−0.27 to 0.25)	−0.22 (−0.67 to 0.22)	0.26 (−0.12 to 0.64)
O_3	−0.34 (−0.89 to 0.21)	0.47 (−0.34 to 1.27)	0.56 (0.21 to 0.92)
65+			
NO_2	−0.09 (−0.48 to 0.31)	0.22 (−0.75 to 1.20)	1.40 (0.83 to 1.98)
SO_2	0.04 (−0.32 to 0.40)	0.27 (−0.37 to 0.91)	1.29 (0.65 to 1.93)
PM_{10}	−0.11 (−0.43 to 0.21)	−0.35 (−0.90 to 0.21)	0.39 (−0.05 to 0.84)
O_3	−0.98 (−1.69 to −0.26)	0.19 (−0.73 to 1.13)	0.52 (0.11 to 0.94)
Cardiovascular			
All ages			
NO_2	−0.67 (−1.17 to −0.16)	0.04 (−1.52 to 1.61)	1.26 (0.40 to 2.12)
SO_2	−0.12 (−0.61 to 0.37)	0.86 (−0.18 to 1.91)	1.68 (0.72 to 2.65)
PM_{10}	−0.35 (−0.92 to 0.22)	−0.34 (−1.19 to 0.52)	−0.09 (−0.84 to 0.67)
O_3	−0.48 (−1.41 to 0.45)	0.56 (−0.93 to 2.08)	0.19 (−0.40 to 0.79)
65+			
NO_2	−0.50 (−1.08 to 0.09)	−0.07 (−1.86 to 1.76)	1.34 (0.37 to 2.33)
SO_2	0.16 (−0.41 to 0.73)	1.13 (−0.08 to 2.35)	1.94 (0.85 to 3.05)
PM_{10}	−0.28 (−0.90 to 0.35)	0.04 (−0.92 to 1.01)	−0.06 (−0.90 to 0.78)
O_3	−0.97 (−2.04 to 0.12)	0.13 (−1.60 to 1.89)	0.12 (−0.63 to 0.87)
Respiratory			
All ages			
NO_2	0.72 (0.06 to 1.39)	2.01 (−0.02 to 4.08)	1.24 (0.15 to 2.33)
SO_2	0.09 (−0.54 to 0.74)	0.49 (−0.85 to 1.85)	1.00 (−0.20 to 2.21)
PM_{10}	−0.10 (−0.52 to 0.33)	−0.15 (−0.94 to 0.64)	0.38 (−0.25 to 1.01)
O_3	−0.49 (−1.64 to 0.68)	0.92 (−0.98 to 2.86)	0.73 (−0.02 to 1.50)
65+			
NO_2	0.80 (0.06 to 1.54)	2.62 (0.39 to 4.90)	1.57 (0.39 to 2.77)
SO_2	0.31 (−0.40 to 1.02)	1.02 (−0.45 to 2.51)	1.62 (0.32 to 2.93)
PM_{10}	−0.26 (−0.74 to 0.23)	−0.12 (−1.04 to 0.80)	0.49 (−0.19 to 1.18)
O_3	−0.57 (−1.86 to 0.73)	1.12 (−0.96 to 3.24)	0.68 (−0.14 to 1.50)
Accidental			
All ages			
NO_2	−0.25 (−1.31 to 0.82)	−0.94 (−4.08 to 2.29)	0.93 (−1.00 to 2.89)
SO_2	0.15 (−0.84 to 1.16)	1.11 (−1.04 to 3.31)	1.38 (−0.73 to 3.54)
PM_{10}	0.28 (−0.70 to 1.27)	0.45 (−1.24 to 2.16)	1.98 (0.18 to 3.81)
O_3	−0.26 (−2.17 to 1.69)	0.16 (−3.08 to 3.50)	1.29 (−0.17 to 2.77)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day, except PM_{10} at current day.

Table 14. Short-Term Effects on Mortality Due to Air Pollution from PM Chemical Species for Pre- and Post-Intervention Periods[a]

Disease / Age Group / Pollutant	Pre-Intervention (5-Year) ER (95% CI)	Pre-Intervention (2-Year) ER (95% CI)	Post-Intervention (5-Year) ER (95% CI)
All Natural Causes			
All ages			
Al	0.033 (−0.081 to 0.147)	0.042 (−0.124 to 0.209)	0.060 (−0.048 to 0.168)
Fe	−0.011 (−0.032 to 0.010)	−0.006 (−0.071 to 0.059)	0.011 (−0.008 to 0.029)
Mn	−0.041 (−0.714 to 0.637)	−1.076 (−2.848 to 0.728)	0.449 (−0.172 to 1.073)
Ni	1.573 (−0.217 to 3.395)	5.178 (2.266 to 8.174)	2.157 (−0.645 to 5.038)
V	0.690 (−0.069 to 1.456)	1.711 (0.649 to 2.783)	0.378 (−0.721 to 1.489)
Pb	−0.066 (−0.434 to 0.303)	−0.099 (−0.743 to 0.550)	0.045 (−0.186 to 0.277)
Zn	−0.051 (−0.180 to 0.078)	−0.235 (−0.456 to −0.012)	0.001 (−0.181 to 0.182)
65+			
Al	0.029 (−0.067 to 0.125)	0.056 (−0.145 to 0.258)	0.096 (−0.030 to 0.223)
Fe	−0.009 (−0.034 to 0.017)	−0.035 (−0.114 to 0.045)	0.027 (0.007 to 0.047)
Mn	0.056 (−0.772 to 0.891)	−1.605 (−3.689 to 0.523)	0.945 (0.254 to 1.641)
Ni	2.641 (0.443 to 4.888)	6.071 (2.313 to 9.968)	2.874 (−0.326 to 6.176)
V	1.004 (0.065 to 1.951)	1.694 (0.293 to 3.114)	0.404 (−0.869 to 1.694)
Pb	−0.152 (−0.617 to 0.314)	0.086 (−0.697 to 0.875)	0.117 (−0.146 to 0.381)
Zn	0.061 (−0.101 to 0.224)	−0.119 (−0.404 to 0.167)	0.009 (−0.200 to 0.218)
Cardiovascular			
All ages			
Al	−0.030 (−0.205 to 0.145)	−0.135 (−0.376 to 0.107)	0.197 (−0.032 to 0.427)
Fe	−0.017 (−0.059 to 0.025)	0.000 (−0.101 to 0.101)	−0.028 (−0.064 to 0.007)
Mn	0.241 (−1.132 to 1.633)	−0.579 (−3.307 to 2.226)	−0.900 (−2.098 to 0.313)
Ni	0.633 (−3.019 to 4.423)	4.273 (−0.903 to 9.719)	−2.888 (−8.255 to 2.793)
V	−0.091 (−1.674 to 1.518)	1.000 (−0.830 to 2.863)	−1.058 (−3.225 to 1.157)
Pb	−0.059 (−0.848 to 0.736)	0.131 (−1.078 to 1.355)	−0.352 (−0.829 to 0.128)
Zn	−0.166 (−0.441 to 0.110)	−0.336 (−0.755 to 0.084)	−0.262 (−0.631 to 0.109)
65+			
Al	0.038 (−0.156 to 0.232)	−0.072 (−0.334 to 0.191)	0.219 (−0.028 to 0.466)
Fe	−0.012 (−0.060 to 0.037)	−0.053 (−0.177 to 0.071)	−0.026 (−0.065 to 0.014)
Mn	0.461 (−1.116 to 2.062)	−2.427 (−5.708 to 0.969)	−0.788 (−2.107 to 0.549)
Ni	1.775 (−2.457 to 6.191)	6.724 (0.404 to 13.442)	−1.582 (−7.520 to 4.736)
V	0.197 (−1.626 to 2.053)	1.627 (−0.603 to 3.908)	−0.619 (−3.009 to 1.831)
Pb	−0.542 (−1.451 to 0.376)	−0.686 (−2.174 to 0.825)	−0.229 (−0.750 to 0.294)
Zn	−0.088 (−0.406 to 0.231)	−0.320 (−0.831 to 0.195)	−0.299 (−0.705 to 0.109)

(*Table continues on next page*)

[a] Data are expressed in % per 10-ng/m^3 unit change.

association with mortality due to all natural causes in the all-ages and 65+ groups. The excess risks were higher in the 2-year period compared with the 5-year pre-intervention period (Table 14).

In the post-intervention period, only Fe and Mn showed a significant association with mortality due to all natural causes for the 65+ age group; and most excess risks were lower than in the pre-intervention period. A notable exception was that the ER of Ni for mortality due to all natural causes for both age groups was higher in the 5-year pre-intervention period (Table 14).

Change in ER After the Intervention

Gaseous Pollutants In the 10-year assessment period, most gaseous pollutants were associated with increases in the ER of mortality after the intervention (Table 15). These increases

Table 14 (*Continued*). Short-Term Effects on Mortality Due to Air Pollution from PM Chemical Species for Pre- and Post-Intervention Periods[a]

Disease / Age Group / Pollutant	Pre-Intervention (5-Year) ER (95% CI)	Pre-Intervention (2-Year) ER (95% CI)	Post-Intervention (5-Year) ER (95% CI)
Respiratory			
All ages			
Al	−0.107 (−0.339 to 0.126)	−0.008 (−0.266 to 0.251)	0.023 (−0.165 to 0.211)
Fe	0.009 (−0.028 to 0.046)	−0.032 (−0.153 to 0.089)	0.009 (−0.021 to 0.040)
Mn	0.275 (−0.904 to 1.467)	−2.390 (−5.571 to 0.899)	0.050 (−0.983 to 1.093)
Ni	3.695 (0.629 to 6.855)	6.252 (0.795 to 12.004)	3.383 (−1.529 to 8.540)
V	1.531 (0.257 to 2.820)	1.904 (−0.009 to 3.853)	1.256 (−0.696 to 3.246)
Pb	0.061 (−0.570 to 0.696)	−0.281 (−1.435 to 0.888)	−0.141 (−0.538 to 0.259)
Zn	−0.058 (−0.282 to 0.166)	−0.248 (−0.667 to 0.173)	−0.138 (−0.449 to 0.174)
65+			
Al	−0.075 (−0.296 to 0.147)	−0.069 (−0.295 to 0.157)	0.049 (−0.106 to 0.204)
Fe	0.000 (−0.039 to 0.039)	0.014 (−0.122 to 0.150)	0.026 (−0.006 to 0.057)
Mn	0.076 (−1.178 to 1.346)	−2.041 (−5.680 to 1.737)	0.502 (−0.590 to 1.606)
Ni	4.171 (0.803 to 7.650)	5.866 (−0.902 to 13.097)	2.317 (−2.704 to 7.596)
V	1.884 (0.470 to 3.318)	2.144 (−0.242 to 4.586)	0.951 (−1.131 to 3.077)
Pb	−0.350 (−1.030 to 0.334)	0.129 (−1.229 to 1.505)	−0.104 (−0.509 to 0.303)
Zn	0.054 (−0.189 to 0.298)	−0.166 (−0.664 to 0.333)	−0.143 (−0.446 to 0.160)
Accidental			
All ages			
Al	0.104 (−0.078 to 0.286)	−0.118 (−0.532 to 0.298)	0.247 (−0.153 to 0.649)
Fe	0.039 (−0.030 to 0.108)	0.028 (−0.219 to 0.275)	0.072 (−0.019 to 0.163)
Mn	1.013 (−1.065 to 3.134)	−1.335 (−7.768 to 5.547)	2.461 (−0.756 to 5.782)
Ni	1.373 (−3.341 to 6.316)	−0.777 (−10.183 to 9.614)	6.430 (−6.224 to 20.790)
V	0.142 (−1.895 to 2.222)	−0.785 (−4.322 to 2.882)	−0.253 (−5.550 to 5.342)
Pb	0.234 (−0.889 to 1.369)	−0.585 (−2.766 to 1.645)	−0.288 (−1.408 to 0.845)
Zn	0.203 (−0.157 to 0.565)	0.169 (−0.564 to 0.907)	−0.156 (−0.972 to 0.667)

[a] Data are expressed in % per 10-ng/m^3 unit change.

were significant for NO_2 on cardiovascular and respiratory mortality for both age groups, with increases in ER ranging between 1.3% and 1.5% per 10-μg/m^3 change; for SO_2 on cardiovascular mortality for both age groups, with increases in ER between 1.1% and 1.2%; and for O_3 on mortality due to all natural causes and respiratory mortality for all ages with increases in ER between 0.6% and 1.6% (Table 15). In the 7-year assessment period (sensitivity analysis), most changes in ER were positive, but none of them were statistically significant except NO_2 on mortality due to all natural causes for all ages (Table B.3 in Appendix B).

PM_{10} and Its Chemical Species There was no consistent pattern of change for all PM chemical species, except Ni and V after the intervention in the 7- or 10-year assessment periods. In the 10-year assessment period, Zn showed a significant change in ER for mortality due to all natural causes in the all-ages group and for cardiovascular mortality in both age groups; and Mn also showed a significant change for cardiovascular mortality in the 65+ group (Table 16). However, no pollutant in the PM_{10} and chemical species category showed a significant change in ER for mortality in the 7-year assessment period (Table B.3 and B.4 in Appendix B).

In the 10-year assessment period, both Ni and V were associated with a decline in ER of mortality in all health outcomes except cardiovascular mortality for all ages and for 65+ (Table 16). In the 7-year assessment period (sensitivity analysis), both Ni and V were associated with declines in ER of mortality after the intervention for all health outcomes under study (Table B.3 in Appendix B).

Table 15. Excess Risk of Mortality Due to Air Pollution from Criteria Pollutants in 5-Year Pre-Intervention Period and Change in Excess Risk After the Intervention[a]

Disease / Age Group / Pollutant	Main Effects (5-Year Pre-Intervention Period) ER (95% CI)	Change in Effects (vs. 5-Year Pre-Intervention Period) ER (95% CI)
All Natural Causes		
All ages		
NO_2	−0.09 (−0.37 to 0.19)	0.25 (−0.25 to 0.75)
SO_2	0.08 (−0.19 to 0.34)	0.27 (−0.28 to 0.82)
PM_{10}	−0.08 (−0.52 to 0.36)	0.21 (−0.28 to 0.71)
O_3	−0.40 (−0.88 to 0.09)	0.62 (0.07 to 1.18)
Cardiovascular		
All ages		
NO_2	−0.64 (−1.11 to −0.16)	1.49 (0.61 to 2.37)
SO_2	−0.07 (−0.53 to 0.39)	1.20 (0.23 to 2.18)
PM_{10}	−0.37 (−1.28 to 0.55)	0.16 (−0.87 to 1.20)
O_3	−0.29 (−1.10 to 0.51)	0.36 (−0.59 to 1.31)
65+		
NO_2	−0.50 (−1.05 to 0.05)	1.41 (0.42 to 2.41)
SO_2	0.28 (−0.25 to 0.81)	1.12 (0.03 to 2.23)
PM_{10}	−0.24 (−1.25 to 0.78)	0.01 (−1.12 to 1.16)
O_3	−0.65 (−1.57 to 0.28)	0.66 (−0.43 to 1.76)
Respiratory		
All ages		
NO_2	−0.34 (−0.98 to 0.31)	1.29 (0.15 to 2.43)
SO_2	0.53 (−0.08 to 1.14)	−0.30 (−1.53 to 0.95)
PM_{10}	−0.02 (−0.74 to 0.71)	0.05 (−0.76 to 0.86)
O_3	−0.61 (−1.71 to 0.50)	1.62 (0.35 to 2.91)
65+		
NO_2	0.33 (−0.37 to 1.02)	1.53 (0.33 to 2.75)
SO_2	0.41 (−0.25 to 1.07)	0.48 (−0.82 to 1.81)
PM_{10}	−0.23 (−1.03 to 0.57)	0.19 (−0.71 to 1.09)
O_3	−0.29 (−1.44 to 0.88)	1.27 (−0.09 to 2.65)
Accidental		
All ages		
NO_2	−0.09 (−1.10 to 0.93)	0.60 (−1.39 to 2.63)
SO_2	0.13 (−0.85 to 1.11)	1.24 (−0.94 to 3.47)
PM_{10}	−0.21 (−1.83 to 1.42)	0.94 (−0.91 to 2.83)
O_3	0.63 (−1.10 to 2.38)	−0.10 (−2.11 to 1.95)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day, except PM_{10} at current day.

Table 16. Excess Risk of Mortality Due to Air Pollution from PM Chemical Species in 5-Year Pre-Intervention Period and Change in Excess Risk After the Intervention[a]

Disease / Age Group / Pollutant	Main Effects (5-Year Pre-Intervention Period) ER (95% CI)	Change in Effects (vs. 5-Year Pre-Intervention Period) ER (95% CI)
All Natural Causes		
All ages		
Al	0.008 (−0.066 to 0.082)	−0.012 (−0.088 to 0.064)
Fe	0.005 (−0.054 to 0.063)	−0.006 (−0.066 to 0.054)
Mn	−0.335 (−1.913 to 1.268)	0.400 (−1.247 to 2.073)
Ni	3.460 (0.654 to 6.344)	−3.008 (−6.094 to 0.180)
V	1.134 (0.125 to 2.153)	−1.068 (−2.299 to 0.179)
Pb	−0.086 (−0.714 to 0.547)	0.087 (−0.574 to 0.752)
Zn	−0.254 (−0.460 to −0.046)	0.296 (0.060 to 0.532)
65+		
Al	−0.018 (−0.099 to 0.063)	0.023 (−0.060 to 0.105)
Fe	−0.017 (−0.079 to 0.046)	0.030 (−0.033 to 0.093)
Mn	−0.839 (−2.540 to 0.891)	1.457 (−0.318 to 3.263)
Ni	5.273 (1.953 to 8.702)	−3.986 (−7.469 to −0.372)
V	1.477 (0.277 to 2.692)	−1.295 (−2.719 to 0.149)
Pb	0.118 (−0.548 to 0.788)	−0.155 (−0.846 to 0.541)
Zn	−0.170 (−0.404 to 0.065)	0.193 (−0.068 to 0.454)
Cardiovascular		
All ages		
Al	−0.023 (−0.167 to 0.121)	−0.011 (−0.157 to 0.136)
Fe	−0.071 (−0.183 to 0.042)	0.060 (−0.053 to 0.174)
Mn	−2.540 (−5.506 to 0.520)	2.576 (−0.596 to 5.850)
Ni	−0.272 (−5.804 to 5.585)	0.600 (−5.831 to 7.470)
V	−0.384 (−2.464 to 1.741)	0.209 (−2.349 to 2.834)
Pb	−0.824 (−1.990 to 0.355)	0.624 (−0.609 to 1.873)
Zn	−0.763 (−1.169 to −0.354)	0.665 (0.204 to 1.128)
65+		
Al	−0.095 (−0.261 to 0.071)	0.068 (−0.101 to 0.237)
Fe	−0.130 (−0.260 to −0.001)	0.123 (−0.008 to 0.254)
Mn	−4.374 (−7.707 to −0.921)	4.737 (1.034 to 8.575)
Ni	0.765 (−5.537 to 7.486)	0.396 (−6.816 to 8.166)
V	−0.218 (−2.605 to 2.228)	0.265 (−2.641 to 3.258)
Pb	−1.516 (−2.832 to −0.182)	1.349 (−0.061 to 2.778)
Zn	−0.839 (−1.299 to −0.378)	0.779 (0.258 to 1.303)

(*Table continues on next page*)

[a] Data are expressed in % per 10-ng/m^3 unit change.

Table 16 (*Continued*). Excess Risk of Mortality Due to Air Pollution from PM Chemical Species in 5-Year Pre-Intervention Period and Change in Excess Risk After the Intervention[a]

Disease / Age Group / Pollutant	Main Effects (5-Year Pre-Intervention Period) ER (95% CI)	Change in Effects (vs. 5-Year Pre-Intervention Period) ER (95% CI)
Respiratory		
All ages		
Al	−0.021 (−0.138 to 0.098)	0.015 (−0.105 to 0.136)
Fe	−0.004 (−0.096 to 0.089)	0.002 (−0.093 to 0.097)
Mn	−0.700 (−3.205 to 1.871)	0.748 (−1.882 to 3.449)
Ni	5.456 (0.710 to 10.426)	−3.040 (−8.113 to 2.313)
V	2.014 (0.319 to 3.738)	−0.886 (−2.917 to 1.188)
Pb	−0.055 (−1.026 to 0.926)	−0.026 (−1.047 to 1.005)
Zn	−0.223 (−0.568 to 0.122)	0.173 (−0.213 to 0.561)
65+		
Al	−0.040 (−0.166 to 0.086)	0.042 (−0.086 to 0.171)
Fe	0.005 (−0.091 to 0.101)	0.008 (−0.089 to 0.105)
Mn	−0.844 (−3.477 to 1.862)	1.126 (−1.612 to 3.940)
Ni	6.056 (0.763 to 11.627)	−3.416 (−8.897 to 2.393)
V	2.186 (0.295 to 4.112)	−0.854 (−3.072 to 1.415)
Pb	0.258 (−0.784 to 1.311)	−0.400 (−1.477 to 0.689)
Zn	−0.140 (−0.512 to 0.234)	0.129 (−0.283 to 0.542)
Accidental		
All ages		
Al	−0.194 (−0.461 to 0.075)	0.229 (−0.040 to 0.499)
Fe	−0.151 (−0.356 to 0.055)	0.177 (−0.029 to 0.383)
Mn	−4.263 (−9.493 to 1.269)	5.286 (−0.517 to 11.428)
Ni	1.044 (−7.909 to 10.868)	0.709 (−9.570 to 12.156)
V	−0.544 (−3.938 to 2.969)	0.850 (−3.370 to 5.254)
Pb	−0.695 (−2.668 to 1.319)	0.759 (−1.341 to 2.903)
Zn	0.037 (−0.659 to 0.737)	−0.085 (−0.869 to 0.704)

[a] Data are expressed in % per 10-ng/m^3 unit change.

DISCUSSION

On July 1, 1990, the Hong Kong government implemented a regulation restricting the sulfur content in fuel as an intervention to improve air quality. The intervention was applied to both conventional liquid fuels and solid fuels in plants but not for vessels, vehicles, or aircraft. Several Hong Kong studies demonstrated that there were declines in SO_2 concentrations and health gains after the intervention. However, it was unclear if the changes in health gains after the intervention were related only to this air pollutant.

Changes in Air Pollutants

Ambient concentrations of SO_2 were shown to have declined after the intervention in the 5 years of the post-intervention period (Hedley et al. 2002). NO_2 showed a slight decrease, while O_3 levels increased slightly. Because of the chemical interaction between NO_2 and O_3, the statistically significant increase in O_3 might have been a result of a reaction with other pollutants such as nitrous oxides (NO_x), leading to the nonsignificant decreasing pattern of NO_2.

As the intervention pertained to fuel, this study focused on fuel-related PM chemical species, including Al, Fe, Mn, Ni, V, Pb, and Zn. After the intervention, there was a decline in Ni and V in industrial areas (see Table B.2 in Appendix B). Also, as Ni and V are indicators of fuel combustion, the decline of these PM chemical species, together with the changes in SO_2 levels, might give support for the hypothesis that the sulfur restriction in fuel might have led to changes in imported fuel from different refinery processes, affecting the composition of the fuel and subsequent emissions of pollutant concentrations from combustion.

Besides being present in crude oil, Ni and V are also found in residual oils, which are also high in sulfur. In order to comply with the sulfur restriction regulation, residual oils must be desulfurized. However, during the hydrodesulfurization process, their existence can seriously affect product yield and catalyst activity, so a number of refineries have adopted a catalyst process to remove metals, including Ni and V. Lippmann and colleagues (2006) reported that 93% of the U.S. emissions of Ni and V were from states that border the Atlantic and Gulf Coasts and from California (Energy Information Administration 2001). Thus Ni and V in the United States are usually emitted from oil-fired power plants and/or ocean-going ship boilers consuming high-sulfur residual oils as fuels. In Hong Kong, both Ni and V were very high in the pre-intervention period, with means of 21.6 and 60.3 ng/m^3, respectively, at station KC, and 9.2 and 18.7 ng/m^3, respectively, at station KT. The correlations between Ni and V were 0.92 and 0.49 in pre- and post-intervention periods, respectively (Table B.5 in Appendix B). Although the correlation between stations KC and KT was less than |0.45|, both are located in industrial areas and near the harbor and are presumably affected by common sources of pollution. After implementation of the fuel sulfur regulation in 1990, annual mean concentrations of SO_2, declined sharply, as did concentrations of Ni and V at both station KC (with reductions ranging from 14.9 to 18.0 for Ni, and 41.2 to 53.1 ng/m^3 for V) and station KT (with reductions ranging from 4.0 to 6.1, and 4.5 to 15.5 ng/m^3, respectively) in the 5 years of the post-intervention period. These declines can be explained by factories switching from residual oils high in sulfur, Ni, and V to other more refined and desulfurized oils after the regulation. However, what remains unexplained is why these pollutants were higher before the intervention at station KC than at station KT and then decreased to such a greater extent after the regulation. A change in fuel emissions from vessels was not a possible explanation for the declines because the regulation did not apply to marine traffic. However, it is now almost certain that an electric company power plant that was located in Tsing Yi Island near station KC (Figure 15) used residual oils as fuel during the 1980s and until it closed in 1995 (CLP Power Hong Kong 2003). It is also highly likely that after the sulfur restriction, the Tsing Yi Island power plant switched to a cleaner fuel or a mix of fuels with low sulfur content (CLP Holdings Annual Report 1998), leading to a much greater decline in SO_2, as well as in Ni and V, at KC than at KT. To what extent this change in type of oil made an impact on the local air quality, as measured at stations KC and KT, is still a matter of interest to be investigated further.

Health Effects of Air Pollutants

During the period from 1988 to 1992, several studies were carried out in the Kwai Tsing district of Hong Kong, which includes the Kwai Chung district and Tsing Yi Island. These studies showed that respiratory problems (Peters et al. 1996) and bronchial hyperresponsiveness (Wong et al. 1998) in schoolchildren and their mothers (Wong et al. 1999) in the polluted Kwai Tsing district were substantially reduced after the intervention compared with the less polluted Southern district. Using the less-polluted Southern district as a control, these studies provided convincing evidence of strong associations between environmental exposures and the health experience of primary school children (8 to 10 years old) and their mothers, as well as health gains after the restriction of sulfur in fuel.

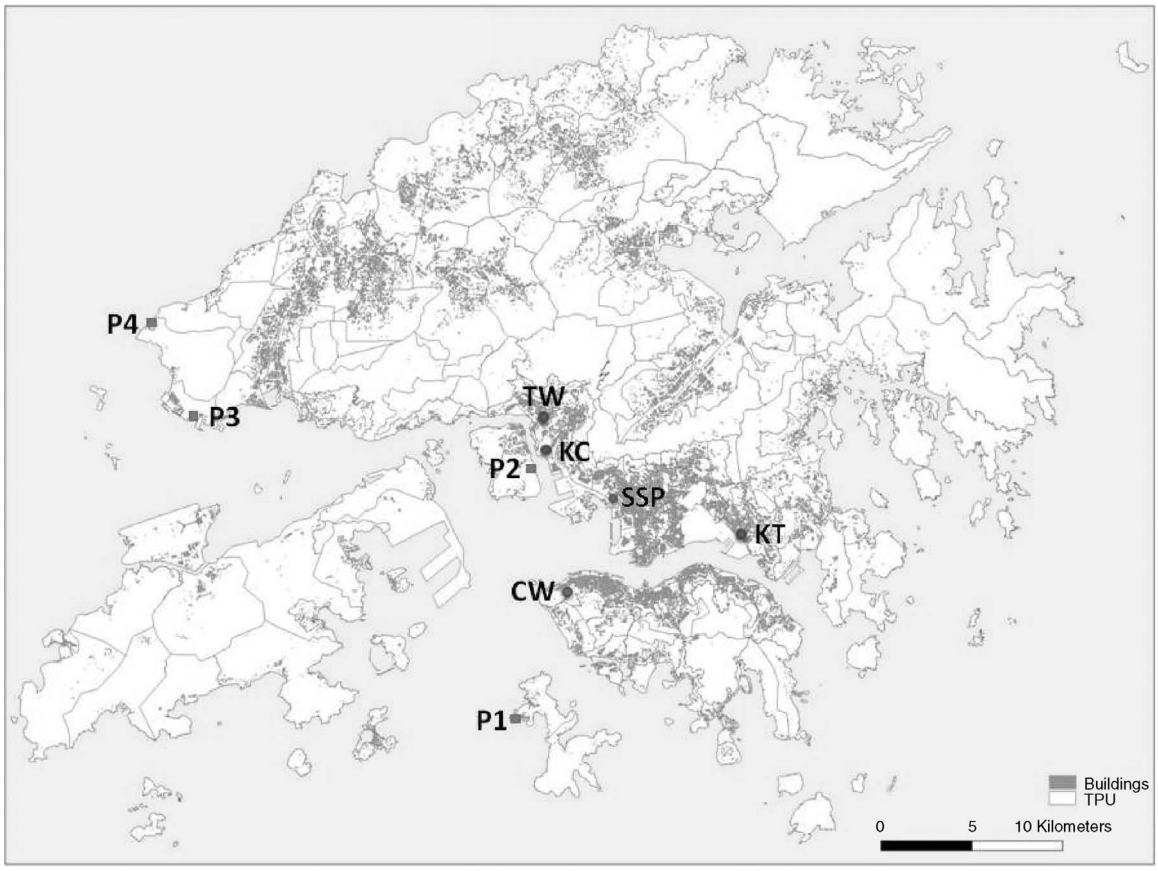

Figure 15. A map of the monitoring stations used in this study and power plants in Hong Kong. P1, P2, P3, and P4 are power plants at Lamma Island, Tsing Yi, Castle Peak, and Black Point, respectively. CW, KC, KT, SSP, and TW represent monitors at Central and West, Kwai Chung, Kwun Tong, Sham Shui Po, and Tsuen Wan, respectively. Gray areas represent building density (based on 2001 data). TPU stands for *tertiary planning unit*, part of the Hong Kong town planning system.

In a later study using a time series of monthly death counts, Hedley and colleagues (2002) showed that there were substantial declines in mortality trends in the 5 years after the intervention. These trends were associated with reductions in cardiopulmonary mortality in the cool season in the first post-intervention year, followed by an increase in deaths in the cool season in the second and third year, with a return to the expected seasonal pattern in the fourth year. The results from this mortality study and other child health studies (Peters et al. 1996; Wong et al. 1998) support a finding that there were health benefits and other changes from the intervention. However, the question remains whether PM chemical species, apart from SO_2, with marked declines in concentration levels after the intervention contributed to some of the observed health benefits — or possibly were completely responsible.

In this study, we found that increased concentrations of both Ni and V were significantly associated with an increased risk of respiratory mortality in both the all-ages and 65+ groups for the 10-year whole assessment period (Figure 16) (Table 12). Sensitivity analyses for a shorter study period of 7 compared with 10 years and for 2 compared with 4 pre-intervention years showed that the excess risks were consistent and increased for the effects of Ni and V in all health outcomes except for accidental mortality and for the effects of V on respiratory mortality (Tables 13 and 14). Variations in the levels of pollutants in industrial areas were higher than those in mixed areas. Since station KC, which is in an industrial area, was operating two years before the intervention, we would expect that the ER effects obtained from the regression models containing data with higher daily variations would be higher.

Furthermore, in the 5-year pre-intervention period, the ER estimates for the associations between Ni and V and mortality in all health outcomes under study were all higher than in the 10-year whole-assessment period (Figure 16).

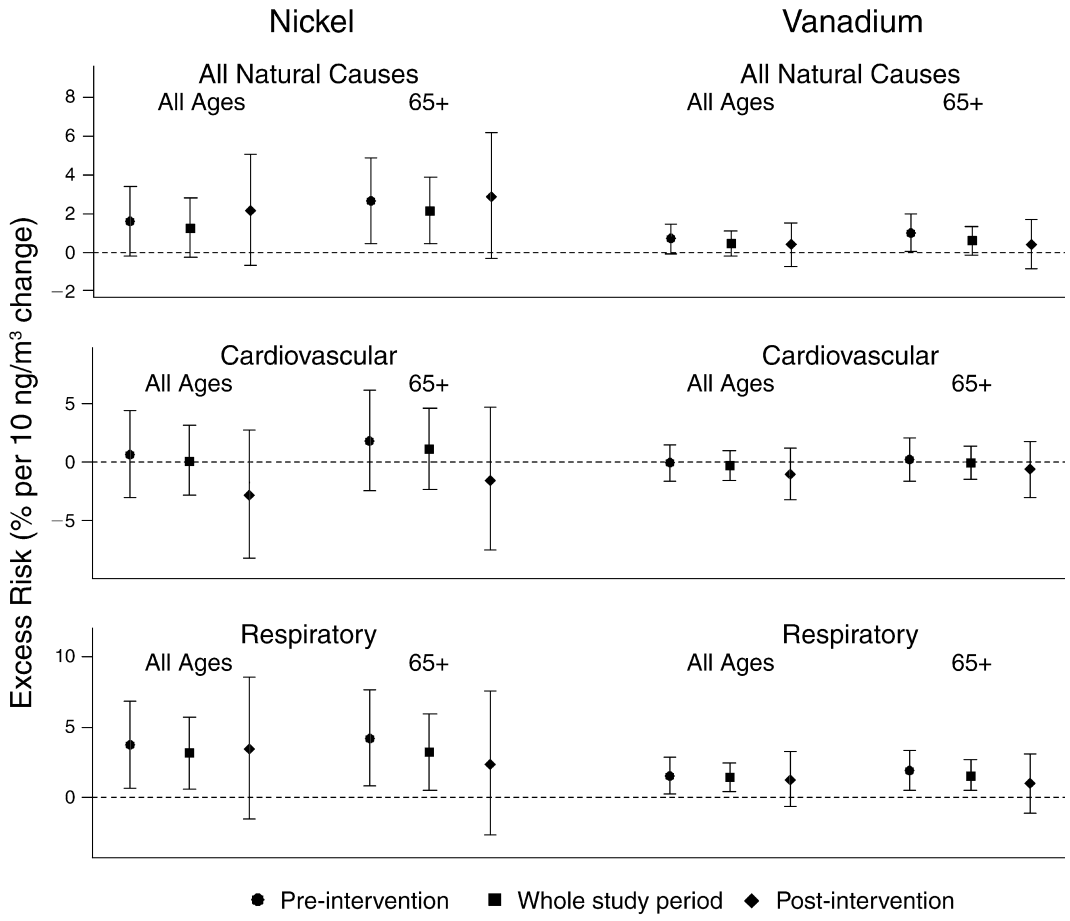

Figure 16. Excess risk of mortality due to Ni and V per 10-ng/m^3 unit change by study period.

Particularly for respiratory diseases, there were significantly increased excess risks for mortality in the 5-year pre-intervention period, which all became lower and statistically nonsignificant for both age groups after the intervention (Figure 16). In cardiovascular diseases, the pattern was similar, although none of the estimates were significant. Although there were no consistent significant results in the analyses stratified by periods because of an insufficient number of PM chemical species measurements, the results may provide some evidence for rejection of the null hypotheses that Ni and V are not related to increased risks of mortality and that the changes in effects are not related to the change in their concentration levels.

Nevertheless, our analyses of Ni showed that it might have the greatest impact on mortality. This was consistent with the findings of a Canadian study (Burnett et al. 2000) that Ni from fine PM was one of the components strongly associated with mortality. Our findings are also supported by other analyses in the United States that this PM chemical species may increase the risk of mortality in humans (Laden et al. 2000; Dominici et al. 2007).

Moreover, Ni may be an agent that could change the mortality risk associated with SO_2. SO_2 effects (from the smaller data set comprising dates when data were available) on mortality due to all natural causes and to the subcategory of respiratory diseases for all ages and those aged 65+ consistently increased after adjustment for Ni in the 5-year pre-intervention period and in the 7-year and 10-year pre- and post-intervention periods combined (Table B.6–B.10 in Appendix B). However, there are no studies investigating the relation between Ni and SO_2 in the existing literature. Caution should also be taken because of the possibility of collinearity due to moderate correlation ($r = 0.65$ in the 10-year period; $r = 0.64$ in both the 5-year pre- and 5-year post-intervention periods) between Ni and SO_2 (Table B.5).

There is evidence in other studies of acute and chronic health effects in animal and human studies, as

well as genotoxicity and carcinogenicity, due to Ni and V (Zakrzewksi 2002; Lippmann et al. 2006; WHO 2006; Department for Environment, Food and Rural Affairs 2007; Zhang et al. 2009). Therefore, although Ni and V may not have a significant association with short-term mortality after the intervention, we could not eliminate their longer-term impacts on mortality.

Limitations

Our health impact assessment was based on the average concentration of pollutants at five monitoring stations. Although our findings on air pollutant concentrations showed that the changes might be in industrial areas only, we took a simple average to represent the population exposure. Since measurements of pollutants were obtained from just five stations, it was hard to perform spatial analyses. The estimates that we obtained were the most conservative ones.

Our analyses used state-of-the-art regression modeling to assess associations between daily mortality and short-term exposure to air pollutants, with a modeling strategy similar to that used in many other air pollution time-series studies. However, we did not find consistent results between different assessment periods for the same PM chemical species. This might be because of large numbers of missing pollutant measurements in some stations in the first 3 years of the study and relatively much higher pollutant concentrations in the fourth and fifth years of the pre-intervention period. Nevertheless, the findings were not incompatible with the limited existing literature on epidemiologic studies of fine PM. In a daily time-series study of 8 Canadian cities on particulate- and gas-phase components, daily nonaccidental deaths were negatively associated with an increase in several PM chemical species at the current day, but the direction of the association was not the same as at lag–1 day (Burnett et al. 2000). In time-series analyses on mortality and fine particulates for the whole study period in California, there were very few significant and positive associations between mortality and PM chemical species; also in those significant associations that were found, the results with cardiovascular or respiratory deaths were not consistent with those of deaths from all causes (Ostro et al. 2007). However, in our Hong Kong study, we found very strong signals with big differences between pollutant concentrations before and after the intervention, and if we had an appropriate method for estimating lag 0–1 day effects with data available every 6 days, we might be in a much better position to take this investigation further.

In this study, we first showed a strong impact of the intervention on mortality due to all natural causes and respiratory diseases. Second, we observed a corresponding reduction in the excess risks associated with two PM chemical species (Ni and V) after the intervention. Furthermore, we did not find significant effects for most pollutants, using the incomplete data in the pre-intervention period. However, it might be difficult to detect changes in ER after an intervention, without sufficient power, even if there are some real changes in the magnitude and nature of exposure.

Using our simulation data on power function, we estimated that with 80% power and a 5% level of significance, the hypothetical changes in ER for mortality due to all natural causes for SO_2 and PM_{10} would be at least 0.5% and 1.1%, respectively, based on the assumption that the ER in the pre-intervention period is 2% per 10 $\mu g/m^3$ (see Appendix D on the HEI Web site). Thus our simulation results, assuming such large changes in ER, suggest that the power of our study might be smaller than 80%.

Another difficulty in testing the changes in ER after the intervention was posed by the implementation of several regulations potentially affecting air pollution levels and population exposure after the 1990 intervention (Table 17). These regulations were related to a wide spectrum of interventions (involving installation of furnaces, ovens, and chimneys; dust and grit emissions; combustion processes; and vehicle designs) that, in addition to sulfur fuel restriction, might affect the sources of pollutants and therefore increase the difficulties we might encounter assessing the short-term effects of PM chemical species potentially related to a specific intervention.

Lastly, we found an increased ER of mortality for cardiovascular diseases due to SO_2 after the intervention. This was counterintuitive; we expected that the effects of SO_2 might decrease following a decrease in concentration after the intervention. However, examining the concentration–response curve from our recent PAPA study (Wong et al. 2008; HEI Public Health and Air Pollution in Asia 2010), we observe that SO_2 levels in Hong Kong followed a concave downward curve. It is therefore not necessarily the case that a decrease in health effects associated with the pollutant would follow from a lower concentration after the intervention. The concentration–response curve for SO_2 from our previous study is different from the one observed in that same study for PM_{10}, which was very linear (Wong et al. 2008; HEI Public Health and Air Pollution in Asia 2010). Higher effect estimates at lower concentrations for SO_2 have also been reported in a German study (Wichmann et al. 2000). In this respect, one might postulate, regarding the biologic mechanism, that the scrubbing effects of the upper airway in response to SO_2 may be stronger at a higher concentration than at a lower concentration of the pollutant (Schlesinger 1999). However, further work on the mechanism of action and the shape of the concentration–response curves is warranted.

Table 17. Regulations Under the Air Pollution Control Ordinance (Chapter 311) in Hong Kong

1. Furnaces, ovens, and chimneys (installation and alteration)	December 15, 1972
2. Dust and grit emission	January 1, 1974
3. Smoke	October 1, 1983
4. Specified process[a]	October 2, 1987
5. Fuel restriction	July 1, 1990
6. Vehicle design standards (emissions)	January 1, 1992
7. Specified processes (specification of required particulars and information) order 1993[b]	August 6, 1993
(removal of exemption) order 1993[c]	November 26, 1993
8. Motor vehicle fuel (supplying or selling motor vehicle diesel)	June 25, 1994; updated April 1, 1995
9. Specified processes (specification of required particulars and information) order 1994[d]	August 26, 1994
(removal of exemption) order 1994[e]	August 26, 1994
10. Open burning	February 26, 1996
11. Asbestos (administration)	May 1, 1996
12. Construction dust	June 16, 1997
13. Petrol filling stations (vapor recovery)	April 1, 1999
14. Dry cleaning machines (vapor recovery)	November 1, 2001
15. Emission reduction devices for vehicles	December 1, 2003
16. Volatile organic compounds	April 1, 2007

Source: Hong Kong Legal Information Institute 1990.

[a] Acrylates; aldehydes; amines; ammonia and its compounds; asbestos; bromine and its compounds; carbon monoxide; chlorine and its compounds; cyanogen and its compounds; fluorine and its compounds; fumes and dust containing aluminium, antimony, arsenic, beryllium, cadmium, copper, iron, lead, mercury, nickel, tin, vanadium, zinc, or their compounds; fumes or vapors from petrochemical works; fumes or vapors from gas works; fumes or vapors from tar and bitumen works; hydrogen sulfide; metal or metallic oxide fumes; oxides and oxyacids of nitrogen; hydrocarbons; smoke, soot, grit, and dust; sulfur dioxide and sulfurous acid; sulfur trioxide and sulfuric acid; volatile organic sulfur compounds.

[b] Aluminium works; cement works; ceramic works; copper works; gas works; petrochemical works; sulfuric acid works; tar and bitumen works; lead works; asbestos works; chemical incineration works; hydrochloric acid works; hydrogen cyanide works; sulfide works; pathologic waste incinerators; petroleum works.

[c] Not applied to: aluminium works; petrolchemical works, tar and bitumen works, cement works premises situated less than 100 m from any other premises that are wholly or partly used, where solely and principally for dwelling purpose.

[d] Organic chemical works; zinc galvanizing works; rendering works; nonferrous metallurgic works; glass works.

[e] Not applied to: acrylates works; cement works; ceramic works; copper works; iron and steel works; mineral works; frit works; zinc galvanizing works; nonferrous metallurgic works; glass works.

AIR POLLUTION EFFECTS ON CHANGES IN LIFE EXPECTANCY USING LINEAR REGRESSION (OBJECTIVES 2 AND 4)

In this section, we present an assessment of the long-term effects of air pollution exposure on changes in life expectancy, based on a time series with distributed lags. Using a time series to determine the total (acute + chronic) impact on mortality of air pollution and the loss of life expectancy would be of great interest because time-series data are widely available and time-series studies are far less costly than cohort studies to conduct. We implemented our approach in the context of a regulation restricting sulfur content in fuel, as mentioned earlier, for a much longer period of 21 years (1985–2005).

METHODS AND DESIGN

Direct, Displaced, and Observed Deaths

Our time-series study examined the relation between air pollution and death rates (D, or number of deaths per unit time). D is specified in terms of deaths per short time period (δt), usually taken as $\delta t = 1$ day. To begin, let us consider a population that is stationary, with a constant death rate (D_{ref}) in the absence of fluctuations due to pollution:

$$D_{ref} = \text{constant} \quad \text{and} \quad \Delta D(t) = D(t) - D_{ref} \tag{3}$$

Now consider a hypothetical situation where a pollution pulse (i.e., an episode of a rapid rise in air pollution) increases the death rate by ΔD between t_0 and $t_0 + \delta t$ and decreases life expectancy (LE) by exactly the change in individual life expectancy (ΔL_{ind}) for all affected individuals, as shown in Figure 17. Since everyone dies exactly once, the direct effect of the pulse (the "direct deaths," from t_0 to $t_0 + \delta t$) must be followed by an equal and opposite change (the "displaced deaths," from $t_0 + \Delta L_{ind}$ to $t_0 + \Delta L_{ind} + \delta t$). In such a situation, with a single pollution pulse, it would be easy to determine ΔL_{ind} by simply looking for the dip due to the displaced deaths.

In reality, some of the pollutant-induced deaths will be delayed (as evidenced in studies of mortality due to chronic exposure), and different individuals suffer different losses of survival time. For air pollution, the time distributions of direct deaths, $\Delta D_{dir}(t)$, and displaced deaths, $\Delta D_{displ}(t)$, overlap, and therefore only the net change

$$\Delta D(t) = \Delta D_{dir}(t) + \Delta D_{displ}(t) \tag{4}$$

is observable, as illustrated schematically in Figure 18. The total number of direct deaths — in other words, the

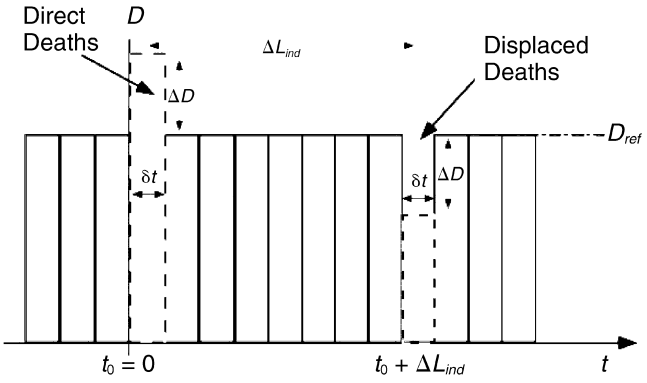

Figure 17. A hypothetical example of a pollution pulse that increases the death rate D by ΔD between t_0 and $t_0 + \delta t$ and decreases the life expectancy of affected individuals by exactly ΔL_{ind} (t = time; δt = short time period; ΔL_{ind} = loss of the affected individuals).

time integral of $\Delta D_{dir}(t)$ — equals the total number of displaced deaths (i.e., equals $-1 \times$ the time integral of $\Delta D_{displ}(t)$). If all the deaths due to a pollution pulse were immediate, one could determine the average, $\Delta L_{ind,\mathrm{av}}$, of the individual losses as the integral of $-t\Delta D_{displ}(t)$, divided by the number of deaths due to the pulse. But whereas that may be a good assumption for heat deaths, it is certainly not for deaths related to air pollution because some of the direct deaths are delayed and overlap the displaced deaths. Because of this overlap, one cannot determine either the number of deaths, as shown by Rabl (2006), or the average of the individual losses.

Time-series studies can, however, yield information on the average loss in life expectancy of an entire population, ΔL_{pop}, averaged over both the number of deaths due to pollution and those that were not. That is the quantity of interest for policy applications, and it has been calculated by the life table methods for the cohort studies mentioned in the "Introduction" to this report.

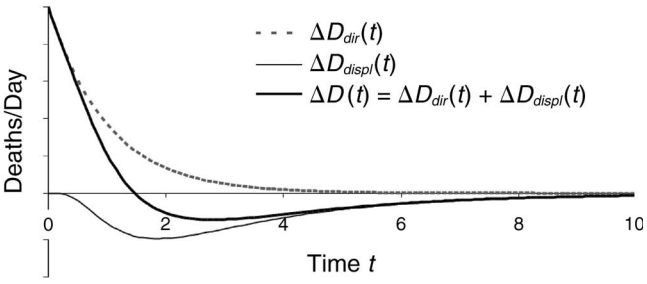

Figure 18. An example of the change in death rate due to a pollution pulse. The change is the net result of an increase due to deaths that have been advanced (or direct deaths), labeled $\Delta D_{dir}(t)$, and a displaced decrease, labeled $\Delta D_{displ}(t)$, happening later when those deaths would have occurred in the absence of the exposure. Only the total, $\Delta D(t) = \Delta D_{dir}(t) + \Delta D_{displ}(t)$, is observable. These curves represent a hypothetical example with an arbitrary scale.

Relation Between Changes in Death Rates and Life Expectancy

To understand how one can obtain information about ΔL_{pop} from the fluctuations of death rates, consider a situation where the death rate D, after being constant at D_{ref}, decreases to $D_{ref} - \Delta D$ during a short interval δt, thereafter resuming its old value. This means that at the end of δt, a fraction $(|\Delta D|/D_{ref})$ of the population has lived δt longer than a population with an unchanged rate. Their deaths have been postponed by δt beyond whenever they would otherwise have occurred; in other words, this part of the population experiences a life expectancy gain of δt. Averaged over the entire population, the gain per person is

$$\delta L_{pop} = -\delta t \frac{\Delta D}{D_{ref}} \tag{5}$$

The minus sign in equation 5 is there because ΔD is negative for a decrease of D. Thus a single dip in the death rate implies a population-averaged life expectancy gain. The instantaneous rate of change in life expectancy, averaged over the entire population, during this interval is*

$$L'_{pop}(t) = \frac{\delta L_{pop}}{\delta t} = \frac{-\Delta D}{D_{ref}} \tag{6}$$

This argument can be applied generally to any sequence of increases and decreases in the death rate. Thus the cumulative change in life expectancy due to a sequence of changes, $\Delta D(t)$, between t_1 and t_2 of the population-averaged relative risk is obtained by integrating equation 6:

$$\Delta L_{pop} = -\int_{t_1}^{t_2} dt \frac{\Delta D(t)}{D_{ref}} \tag{7}$$

This equation for the change in population-averaged life expectancy is generalizable, regardless of what causes the changes in death rate.

Two simple examples may be instructive as illustrations. The first is seen in Figure 17, where a pollution

*A previous paper (Rabl 2006) presented a more complicated derivation of this result. However, the extension to cumulative changes in life expectancy (in equation 9 of that paper) was not quite right because of a slight difference in time scales between the left (time of life expectancy gain) and right (time of risk change) sides of that equation. Furthermore, the model for life expectancy change as a function of exposure (in Section 3.3 of that paper) was not appropriate because it implied an unrealistic relation between the timing of exposure and mortality rates. Instead, in the present paper, the relation between exposure and life expectancy change is based directly on the results of time series without assuming a specific model. The only assumption is linearity (i.e., proportionality between the changes in death rates and the changes in exposure).

pulse decreases L_{pop} by $\delta t \Delta D/D_{ref}$ at t_0 and all the affected individuals lose ΔL_{ind}. At $t_0 + \Delta L_{ind}$, the population returns to its original state, and L_{pop} resumes its old value. As a second example, consider an intervention that permanently decreases the concentration of the pollutant by Δc, starting at $t = 0$, for a homogeneous population of whom a fraction $|\Delta D|/D_{ref}$ instantaneously obtains individual gains of exactly ΔL_{ind}. This is shown in Figure 19 where the death rate drops by $|\Delta D|$ at $t = 0$. After ΔL_{ind}, the death rate returns to the original level because the displaced deaths hide the decrease in direct deaths. During each time step δt between $t = 0$ and $t = \Delta L_{ind}$, the population gains $\delta t\,|\Delta D|/D_{ref}$, and thus the total population-averaged gain is $\Delta L_{pop} = \Delta L_{ind}\,|\Delta D|/D_{ref}$, in agreement with equation 7.

There have been two real-world interventions that come close to the situation described in the example in Figure 19. One is the regulation banning the burning of coal in Dublin after June 1990 (Clancy et al. 2002); the other is the intervention on which the current study is based banning the use of high sulfur fuel in Hong Kong, also after June 1990 (Hedley et al. 2002). In principle, ΔL_{pop} for these interventions could be calculated directly by inserting the $\Delta D(t)/D_{ref}$ data into equation 7. But when we tried this approach with the mortality data for Hong Kong, we encountered several problems. Above all, before inserting the data into equation 7, one has to make sure that $D(t)/D_{ref}$ includes only changes due to the intervention. There are both seasonal variations and long-term trends that must be removed from the data. In particular, there has been a persistent and fairly constant long-term trend of increasing life expectancy (2 to 3 years per decade), comparable to what has been observed in other developed countries and large enough to totally confound the intervention effect (World Bank). Also, the seasonal variation is not exactly periodic from year to year, and therefore the seasonality correction during the intervention is uncertain. Furthermore, the reduction of SO_2 concentrations in 1990 occurred gradually over several months rather than being a sharp step change, while the concentrations of other pollutants also varied. The difficulties in Dublin were similar. Therefore, the direct use of equation 7 is problematic for these interventions. As an alternative, we proceeded in two stages. First, we developed, by regression of mortality and pollution data for a long period, a dynamic model for the effect of air pollution using the ratio $D(t)/D_{ref}$. Then we inserted this model into equation 7, which was evaluated for a step change in concentration, as described later in this section under "Population-Averaged Change in Life Expectancy from Time-Series Data." The results for Hong Kong are presented later in this section under "Results."

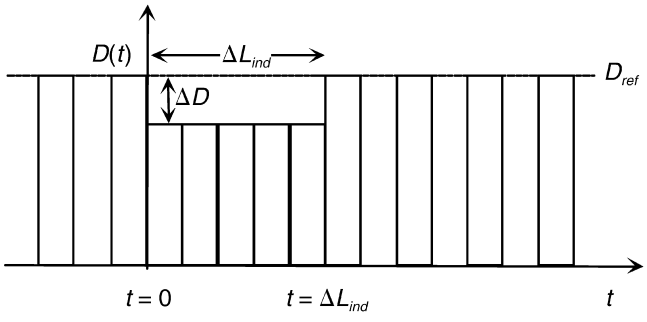

Figure 19. An example of an intervention that permanently decreases the pollutant concentration by Δc for a hypothetical population of whom a fraction ($\Delta D/D_{ref}$) instantaneously gains exactly ΔL_{ind}. ($D(t)$ = number of deaths per unit of time; D_{ref} = constant death rate; L_{ind} = individual life expectancy.)

A Model for Time Series

In choosing a suitable model for time-series studies, a crucial constraint is linearity, in view of numerous studies that have investigated in detail population-level exposure–response functions for air pollution and that provide evidence that such functions are linear and without a threshold (see Schwartz et al. 2008 for cohort studies; see Daniels et al. 2002 and Samoli et al. 2005 for time-series studies). The most general linear model for change in the death rate during day j, $\Delta D(j)$, resulting from an exposure pulse of concentration $c(i)$ during day i can be written in the form

$$\frac{\Delta D(j)}{D_{ref}} = f(j - i)\, c(i) \tag{8}$$

The impact of an entire exposure sequence $\{c(i)\}$ of $i_{\max}$ days up to day j is obtained by summation and can be written (after a simple change of variables) as

$$\frac{\Delta D(j)}{D_{ref}} = \sum_{i=0}^{i_{\max}} f(i)\, c(j - i) \tag{9}$$

Impact coefficients are represented by $f(i)$. To capture the full impact, $i_{\max}$ should be at least as large as the sum of the relevant exposure durations (i.e., the time interval before time t during which exposures have a non-negligible health impact at time t) and the average individual life expectancy gains, so that the corresponding $f(i)$ become negligible. That sum is sometimes called a "cessation lag" because it represents the length of time for the effects of a past exposure to completely disappear from a population. Equation 9 is a straightforward generalization of a conventional time series; it is a distributed-lag model with

i_{max} + 1 lags covering the entire set of exposures that may have an impact. A variety of distributed-lag models have been developed for air pollution studies (Zanobetti et al. 2000) to take into account past exposures (typically only within the first month or two). In the present report, we extended the time horizon to include exposures several years into the past — exposures that have been shown to be very important by several large studies of chronic mortality (Dockery et al. 1993; Pope et al. 2002). By making the time horizon sufficiently long, one can thus measure the population-averaged life expectancy loss, ΔL_{pop}, due to pollution, if the data are not too noisy.

We emphasize that ΔL_{pop} is the average loss *over the entire population*, including those who are affected by pollution and those who are not. Because of the overlap between direct and displaced deaths, we found no way of determining the loss of only the individuals (ΔL_{ind}) who are affected by the air pollution. Even though knowledge of ΔL_{ind} would be valuable for understanding the effects of pollution, for policy purposes only ΔL_{pop} matters.

A Comment on the Assumption of Linearity

The linearity shown in equation 9 assumes that the pollutant concentrations are sufficiently low that there are no saturation effects — in particular that the segment of the population that is frail never goes away entirely and that the model need not take into account correlations between successive exposures. An absence of such correlations is assumed by practically all time-series studies. In reality such correlations, or *sequence effects*, probably do exist. For instance, after a particularly heavy pollution episode, the remaining number of frail individuals may be so small that the exposure during the following days entails almost no acute deaths. Accounting for such sequence effects would render the model nonlinear.

Not only would sequence effects be awkward to include in a model, but for policy applications, they are irrelevant. The ultimate purpose of all this work is to provide guidance for environmental policy. It would not be appropriate to design environmental policies for a particular pollution sequence (which is unpredictable because it depends on the weather); rather policies have to be applicable to any pollution sequence that may occur. Thus, we conclude that the time-series model represented in equation 9 is indeed the appropriate choice; it is based on the average life expectancy over typical pollution sequences. But because of sequence effects, the life expectancy change for impact coefficients determined from any particular data set may be different from the average life expectancy change for the coefficients determined from typical sequences.

Sign Reversals and Constraint for the Time-Series Coefficients

A constraint arises from causality: the displaced deaths must occur after the direct ones. Therefore, the integral of $\Delta D_{dir}(t')$ from $t' = 0$ to $t' = t$ must always be larger than the corresponding one of $-\Delta D_{displ}(t')$, and the integral of the change in observed death rate $\Delta D(t')$ from $t' = 0$ to $t' = t$ must be nonnegative for any t. This implies that the sum $F(j)$ of the coefficients $f(i)$ must satisfy the constraint of being nonnegative for all j:

$$F(0,j) = \sum_{i=0}^{j} f(i) > 0 \qquad (10)$$

Additionally, in the limit $j \to \infty$, it must approach zero because everybody dies exactly once. Therefore, there must be at least one sign reversal in the sequence of the impact coefficients $f(i)$.

Population-Averaged Change in Life Expectancy from Time-Series Data

With discrete time steps of duration T_{day} = 1 day, equation 7, representing the gain in population-averaged life expectancy due to a change in the death rate (ΔD), becomes

$$\Delta L_{pop} = -T_{day} \sum_{j=0}^{\infty} \frac{\Delta D(j)}{D_{ref}} \qquad (11)$$

Inserting a permanent concentration change (Δc) starting at $t = 0$ for $\Delta D(j)/D_{ref}$ into the time-series equation 9, one obtains

$$\Delta L_{pop} = -\Delta c\, T_{day} \sum_{j=0}^{\infty} \sum_{i=0}^{j} f(i) \qquad (12)$$

The life expectancy change attributable to air pollution can be calculated as the change due to a permanent concentration change (Δc). When the time series is truncated — in other words, when the observation window i_{max} (or the time interval of exposures before time t taken into account in a regression analysis for the health impact at time t) in equation 9 is not sufficiently long — the constraint defined in equation 10 results in a lower bound for ΔL_{pop}.

The available evidence suggests that exposure to air pollution can affect mortality over a long period and, therefore, that the lags of the time series should span several years. In principle, if one had sufficiently good data, one could determine all impact coefficients $f(i)$ by linear regression; but in practice, most of the resulting coefficients for large lags turn out to be not statistically significant.

In order to reduce the number of coefficients and their relative standard errors (SEs), one can use polynomial fits (i.e., replace the coefficients $f(i)$ in equation 9 with a polynomial of lag i day). Another approach for reducing the

number of coefficients is to regress ΔD against concentrations that are averaged over longer time intervals, with coefficients that are the sums of the $f(i)$ over the corresponding intervals. With intervals of a duration longer than 1 day, N_k, the coefficients $f(i)\,c(j - i)$ of the time series shown in equation 9 are replaced by the sums $F(i_k - N_k, i_k)$ of the daily coefficients $f(i)$ from $i_k - N_k$ to i_k, multiplied by the corresponding average concentrations. Then the coefficients $f(i)$ for the respective intervals are obtained as

$$f(i) = \frac{F(i_k - N_k, i_k)}{N_k} \qquad \text{for } i_k - N_k \le i \le i_k \tag{13}$$

The optimal choice of intervals is a matter of trial and error. An exponential time scale might be appropriate for daily data, the intervals being short immediately after a pulse and increasing to several years over the long duration. A possible choice might be intervals that increase exponentially into the past — for example, for a length of 3^k days with k from 0 to 6: {day 1, days 2–4, days 5–13, days 14–40, days 41–121, days 122–364, days 365–1093}. The choice could be different for different pollutants since the corresponding time scales of the physiologic processes are different. Another possibility is to employ equal intervals, for instance, month-long or annual.

But ΔL_{pop} also can be determined directly by regression of the concentration data. Let us define

$$G(k) = \sum_{j=0}^{k} F(0, j), \tag{14}$$

where $F(j)$ is the sum of the coefficients $f(i)$ from $i = 0$ to j, as defined in equation 10. Since all the $F(j)$ are positive, $G(k)$ increases monotonically with k, and its limit $G(\infty)$ is equal to the ΔL_{pop} of equation 12. Noting that $f(0) = F(0)$ and $f(i) = F(i) - F(i - 1)$ for $i > 0$, one can write the time series shown in equation 9 in terms of $F(i)$:

$$\Delta RR(j) = F(0, i_{\max})\, c(j - i_{\max}) + \sum_{i=0}^{i_{\max}-1} F(0, i)[c(j - i) - c(j - i - 1)] \tag{15}$$

Replacing analogously $F(i)$ by $G(i) - G(i - 1)$, for $i > 0$, with $F(0) = G(0)$, one obtains the following:

$$\begin{aligned}\Delta RR(j) = {} & G(i_{\max})\, c(j - i_{\max}) \\ & + G(i_{\max} - 1)[c(j - (i_{\max} - 1)) - 2c(j - i_{\max})] \\ & + \sum_{i=0}^{i_{\max}-2} G(i)[c(j - i) - 2c(j - i - 1) + c(j - i - 2)].\end{aligned} \tag{16}$$

The advantage of a regression with equation 16 is that it yields directly the $G(i)$ and their CIs; furthermore, the first coefficient $G(0) = f(0)$ is the time-series coefficient for lag 0 day. We shall refer to the combination of concentrations $[c(j - i) - 2c(j - i - 1) + c(j - i - 2)]$ as "second differences."

Our Calculation Method for Change in Life Expectancy vs. That Used in Cohort Studies

Since the calculation of life expectancy loss due to air pollution is usually based on cohort studies, such as the study by Pope and colleagues (2002), we should comment on how that method of calculation compares with the method developed in the present report. The calculation based on cohort studies uses the measured increase in age-specific mortality as input into a life table calculation, which yields the corresponding decrease in life expectancy. The calculation is static because the cohort studies on which it is based are in effect steady-state comparisons of metropolitan areas, which in reality experience different exposure levels.

By contrast, the method presented here is dynamic, because it is based on changes in total (rather than age-specific) mortality, as measured by time-series studies. It yields only a lower bound for the loss in life expectancy, which approaches the full value only to the extent that the observation window is sufficiently long to cover all relevant exposures. Another difference is that cohort studies analyze the deaths of a group of individuals, whereas time-series studies examine a population that is in effect replenished continually because deaths rates are normalized to a constant reference.

In view of these differences, there can be no direct comparison between these two methods. However, certain advantages to the method presented in this report are apparent. First, time-series studies have far lower costs associated with them than cohort studies. Second, time-series studies can provide direct information on the relevant exposure window: as the observation window becomes longer than the relevant exposures, the resulting estimate by time series of the loss in life expectancy reaches the full value that would be calculated by cohort studies and life tables. All this assumes, of course, that the data do not contain too many confounders. This section of the report will explore the applicability of the time-series method.

THE INTERVENTION

In principle, the gain in population-averaged life expectancy, ΔL_{pop}, for the Hong Kong sulfur intervention could be calculated directly by inserting the $\Delta D(t)/D_{ref}$ data into equation 7. But when we tried this approach with the Hong Kong mortality data, we encountered problems. Before inserting the data into equation 7, it is important to

make sure that $D(t)/D_{ref}$ includes only changes due to the intervention. Both seasonal variations and long-term trends must be removed from the data. Unfortunately, the seasonal variation in our study is not sufficiently periodic: the shape of the variation and the timing of peak and amplitude vary significantly from year to year. There were no systematic long-term trends from 1985 to about 1992, but since then there has been a persistent and fairly constant long-term trend of decreasing mortality and hence increasing life expectancy (two to three years per decade). (This is comparable to what has been observed in other industrialized countries.)

Therefore, the seasonality correction during the intervention is uncertain, and so is the correction for long-term trend after 1992. For these reasons, the direct use of equation 7 is problematic for the Hong Kong intervention, as can be seen in Figure 20, which shows both ΔL_{pop} (calculated according to equation 7) and D/D_{ref}. D is the death rate (deseasonalized, as described in the section "Adjustments Made Before the Regressions Against Pollution" later in this report and shown as a 30-day moving average to reduce the day-to-day variations), and D_{ref} is the average death rate during the four years preceding July 1, 1990. Beginning in 1992, there is a steady increase in life expectancy, reaching almost 3 years in length by 2006, consistent with conventional life expectancy calculations for Hong Kong. But the effect of the intervention is not very clear. During the first year after the intervention, there is indeed a life expectancy gain of about 10 days; however, most of that is lost during the following year. How much of the long-term gain is attributable to the intervention cannot be determined from equation 7 and Figure 20 because the result depends on the assumptions about what the seasonal pattern would have been in the absence of the intervention. A further, although less important, complication is that the reduction of SO_2 concentrations in 1990 occurred gradually over several months rather than suddenly in a sharp step change, and SO_2 concentrations fluctuated significantly both before and after the intervention.

In the Dublin study (Clancy et al. 2002; Goodman et al. 2004), these difficulties were circumvented by using the mortality data of the surrounding counties as a reference. Unfortunately, we have no suitable reference data for Hong Kong because the data from that time are not available.

The drop in life expectancy observed in our calculations after the first year is consistent with the post-intervention mortality data reported in the study by Hedley and colleagues (2002) and reproduced here in Figure 21. These authors fit the data by assuming a sinusoidal variation with a single annual frequency. During the first year after the intervention, the death rate did not show the usual winter peak (corresponding to a reduced amplitude in Figure 21), but during the following year, the change went in the opposite direction. Respiratory and cardiovascular mortality displayed the same pattern.

The patterns in Figures 20 and 21 are entirely plausible if most of the individuals whose deaths were postponed gained approximately one to two years. During the first year, the death rate drops below the reference rate (i.e., the

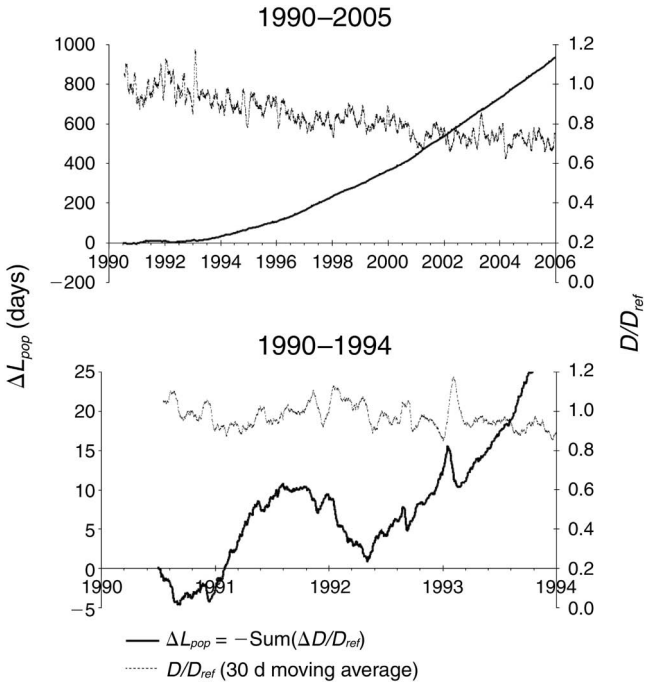

Figure 20. Population-averaged change in life expectancy (ΔL_{pop}) after the intervention. The ΔL_{pop} was calculated according to equation 7, but the integral $\Delta D/D_{ref}$ was replaced with the sum of daily values.

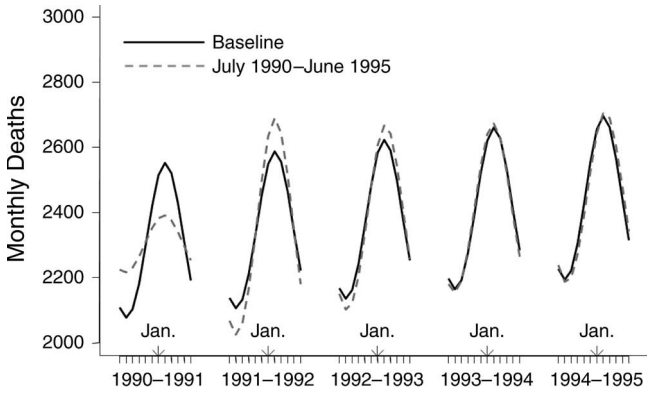

Figure 21. The post-intervention all-cause mortality data in Hong Kong for 1990–1995 reported in a study by Hedley and colleagues (2002). (Figure based on data from Hedley et al. 2002.)

rate without intervention), but during the second year, the deaths of these individuals are added to the reference rate. The details of the observed patterns depend on the distribution of the individual gains in the population, which are not known and cannot be disentangled from the data because of the uncertainties about the seasonal variations and long-term trends.

For another illustration of the difficulties in directly analyzing the intervention period, we show in Table 18 the change in death rates pre- and post-intervention, with two different time cutoffs: 12 months and 18 months (before and after July 1, 1990, respectively). There is a decrease, amounting to about 3.5% for the 12-month period, but it is not statistically significant.

Because of these difficulties, we chose an alternative approach, proceeding in two stages. First, we developed, using a time-series regression of mortality and pollution data for a long period, a dynamic model for the effect of air pollution on the ratio $D(t)/D_{ref}$. Second, we inserted this model into equation 7, which was evaluated for a step change in concentration, as described earlier in the section "Population-Averaged Change in Life Expectancy from Time-Series Data." The results for Hong Kong are presented later in this section under "Results." Even though seasonality can also be a confounder in a time-series regression, the effect on the calculation of life expectancy gain is totally different in the time-series approach from that in a direct examination of the intervention period. The latter is very sensitive to the short-term irregularities during the intervention period, requiring choices that are more or less arbitrary about the time cutoffs before and after the intervention used for calculating the effect. By contrast, the time-series approach uses the entire 21-year period (1985–2005) to obtain the impact coefficients, and no arbitrary time cutoff was imposed other than the length of the observation window. The effect of the observation window is unambiguous: one can determine only a lower bound for the life expectancy gain, and this lower bound increases with the length of the window.

Table 18. Change in Death Rates Pre- and Post-Intervention for Two Different Time Cutoffs

Averaging Period	Average Death Rate	SE
Death rate average 18 months before July 1990	0.01357	0.00201
Death rate average 18 months after July 1991	0.01325	0.00204
Decrease (pre – post)	0.00032	0.00286
Death rate average 12 months before July 1990	0.01362	0.00203
Death rate average 12 months after July 1991	0.01314	0.00210
Decrease (pre − post)	0.00048	0.00292

DATA ANALYSIS

Since the design of this part of the study (supporting Objectives 2 and 4) assumes a stationary population, we adjusted the mortality data so that they corresponded accordingly. We did this in several steps, the first being an adjustment to the age structure. A plot of the age distribution showed that the age structure of Hong Kong changed during the study period and that the time average of the distribution was not very different from the standard age profile of the World Health Organization (WHO 2000). Therefore, we chose this age profile to calculate age-standardized mortality rates.

Additional adjustments were needed for temperature, humidity, seasonal patterns, and long-term trends. For these adjustments, we took two different approaches described in the next two sections. In the first, we made the adjustments before the regressions against pollution. In the second, we included the adjustments within the regressions.

Adjustments for Temperature and Relative Humidity *Before* the Regression Against Pollutants

With this approach, we began with nonparametric smoothing to correct for temperature (T) and relative humidity (RH) to obtain the equivalent death rates at the average T and RH. Then we deseasonalized the mortality rates because they displayed fairly systematic seasonal variations, even after correction for T and RH. To deseasonalize, we calculated the standard seasonal profile by first replacing the data with their 30-day moving averages, then averaging for each day of the year the values for the respective day of each of the 21 years, and finally normalizing the profile to an average of unity. (We have also tried moving averages for periods shorter than 30 days but found that shorter averaging periods yielded too many irregular daily variations stemming from the limited number of data and did not reflect the true seasonal trend.) We then divided the death rates by this profile. We used the same method for deseasonalizing the concentrations.

Finally, we removed the long-term trend in life expectancy, because Hong Kong, like most cities in industrialized countries, has been enjoying a steady increase in life expectancy, averaging about two to three years per decade. We calculated the reference death rate D_{ref} as a moving

average of $D(t)$ over the observation window. For example, with a choice of 7 intervals of duration 3^0 to 3^6 days, the total observation window is the sum of these 7 intervals (1093 days), and $D_{ref}(t)$ is the average of $D(t')$ from $t' = t$ to $t - 1092$. This step removes the long-term mortality trend from the ratio $D(t)/D_{ref}(t)$ since the trend in the numerator and denominator cancel out.

Since D_{ref} includes the average effect of pollution, a ΔD of 0 corresponds to the average pollution level during the observation window. Therefore, we regressed $\Delta D(t)/D_{ref}$ against the deviations of the concentrations from their moving average during the observation window.

Adjustments for Temperature and Relative Humidity *Within* the Regression Against Pollutants

An alternative approach, which functions as a sensitivity analysis, to deseasonalizing death rates before adjustments for T and RH is to break the death rates into three components: seasonality trends, long-term trends, and residuals. We followed an approach similar to that proposed by Schwartz (2000) and used an STL algorithm (Cleveland et al. 1990) to separate the death rates into the three components. We then adjusted the deseasonalized death rates, $D(t)$, for trends, T, and RH by means of regression. We applied locally estimated scatterplot smoothing (LOESS) to each regressor with the smoothing parameter of 0.5. We obtained the reference death rate D_{ref} by calculating the adjusted means of $D(t)$, evaluating T and RH at their mean levels, and regressing $\Delta D(t)/D_{ref}$ against the deviations of the concentrations from their moving averages during the observation window.

RESULTS

Results with Adjustments for Temperature and Relative Humidity *Before* the Regression Against Pollutants

For the regressions against averaged concentrations (using the times series represented by equation 9 but with averages over intervals according to equation 13), we show results for 7 intervals of lengths 3^0 to 3^6 days, for single-pollutant regressions with PM_{10} separately and with SO_2 separately, and for a two-pollutant regression with PM_{10} and SO_2 together. The intervals are day 0, days 1–3, days 4–12, days 13–39, days 40–120, days 121–363, and days 364–1092. The coefficients $F(i_k - N_k,i_k)$ for these intervals (k = 0–6) are shown in Figure 22, with the one-pollutant and two–pollutant fits indicated by type of line (solid line and dashed line, respectively). (See Table B.14 in Appendix B for the coefficient estimates and standard errors.) However, the lines do not directly show the impact

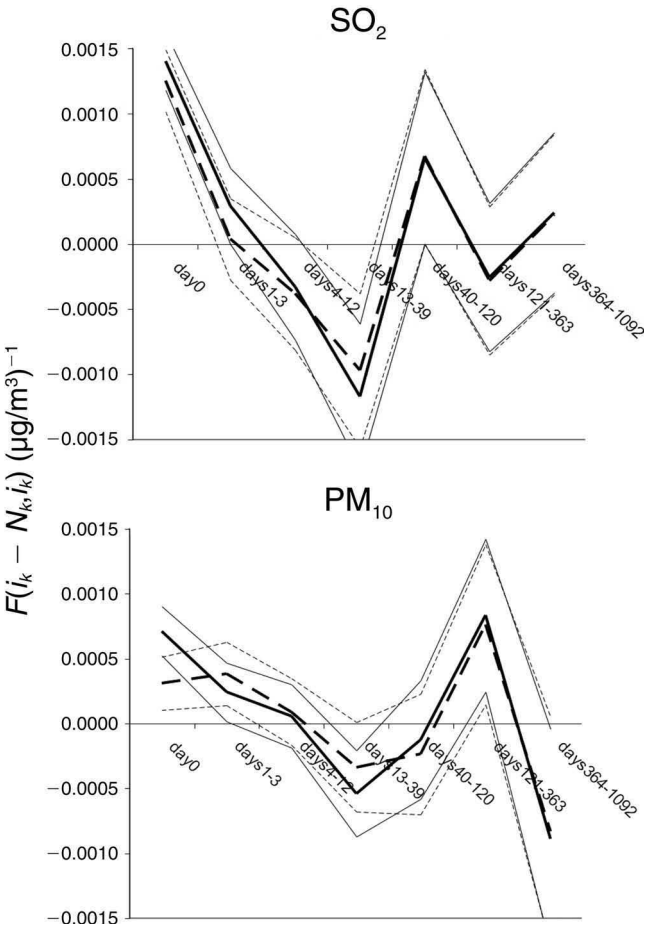

Figure 22. The coefficients $F(i_k - N_k,i_k)$ for fits (with adjustments *before* the regressions against pollutants) with 7 intervals of lengths $N_k = 3^k$ days (k = 0 to 6) for SO_2 and PM_{10} (solid line = one-pollutant fit; dashed line = two-pollutant fit). The pairs of thin lines shown above and below the thick dashed and sold lines indicate the CIs. Note that the units are $(\mu g/m^3)^{-1}$; to convert to the more customary percentage per 10 $\mu g/m^3$, simply multiply by 1000.

coefficients $f(i)$ versus day i because the x-axis represents interval label k, not day i, and the $f(i)$ would be $F(i_k - N_k,i_k)$ divided by the respective interval lengths N_k, as shown in equation 13. We show $f(i)$ versus day i in Figure 23, by using two graphs with different scales to accommodate the large variations. For the graphs of $f(i)$ shown in Figure 23, we placed the values of $F(i_k - N_k,i_k)/N_k$ at the midpoints of the respective intervals and used a smoothed line. The first coefficient, $F(0,1)$, equals the coefficient $f(0)$ of the time series since the first interval is 1 day; it can be compared with the results of conventional time series. For SO_2 we have $f(0) = 0.0014$ from the single-pollutant fit and 0.0013 from the two-pollutant fit (data not shown). For PM_{10} it is 0.00071 from the single-pollutant fit and 0.00031 from the two-pollutant fit.

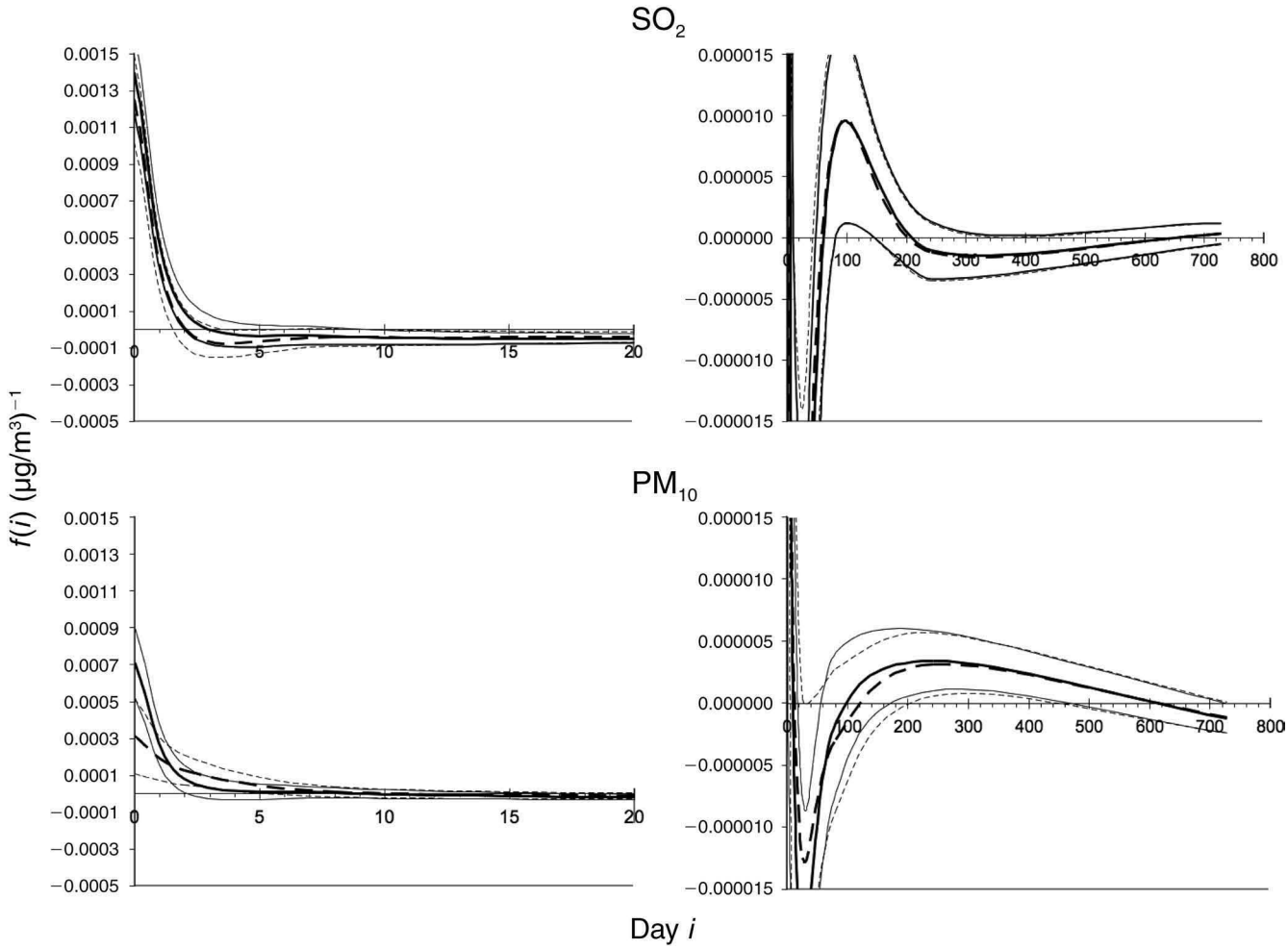

Figure 23. The coefficients $f(i)$ for the fits from Figure 20 for SO_2 and PM_{10} shown in two graphs with different scales to accommodate large variations. Note that the units are $(\mu g/m^3)^{-1}$ (to convert to the more customary percentage per 10 $\mu g/m^3$, simply multiply by 1000).

The change in life expectancy is 0.78 days per 1 $\mu g/m^3$ for SO_2 alone and 0.48 days per $\mu g/m^3$ for SO_2 in the two-pollutant fit with PM_{10}. For PM_{10} the change in life expectancy is 0.79 days per 1 $\mu g/m^3$ for PM_{10} alone and 0.60 days per $\mu g/m^3$ for PM_{10} in the two-pollutant fit with SO_2 (data not shown). These numbers are lower bounds since the observation window is not long enough.

Results for the $G(i)$ coefficients of the regression against the second differences (see equation 16) are shown in Figure 24 for single-pollutant regressions only, with an observation window of 3 years (1096 days). The first coefficient, $G(0)$, equals the coefficient $f(0)$ of the time series. $G(0)$ is 0.67% of the estimated ER per 10 $\mu g/m^3$ for PM_{10} and 1.4% per 10 $\mu g/m^3$ for SO_2. The last coefficient, $G(1096)$ in this case, is a lower-bound estimate of the change in life expectancy; it equals 19.2 days per 10 $\mu g/m^3$ for PM_{10} and 19.7 days per 10 $\mu g/m^3$ for SO_2 (data not shown).

Results with Adjustments for Temperature and Relative Humidity *Within* the Regression Against Pollutants

For the approach with adjustments included within the regression, we have fits only for $G(i)$, as shown in Figure 25. For SO_2 the first coefficient, $G(0)$, is 0.75% of the estimated ER per 10 $\mu g/m^3$ for the single-pollutant fit and 0.73% per 10 $\mu g/m^3$ for the fit with two pollutants; the last coefficient, $G(1096)$ in this case, is 12.8 days per 10 $\mu g/m^3$ for the single-pollutant fit and 3.0 days per 10 $\mu g/m^3$ for the fit with two pollutants (data not shown). For PM_{10} the first coefficient, $G(0)$, is 0.21% per 10 $\mu g/m^3$ for the single-pollutant fit and 0.19% per 10 $\mu g/m^3$ for the fit with two pollutants; the last coefficient, $G(1096)$ in this case, is 31.4 days per 10 $\mu g/m^3$ for the single-pollutant fit and 17.4 days per 10 $\mu g/m^3$ (data not shown) for the two-pollutant fit.

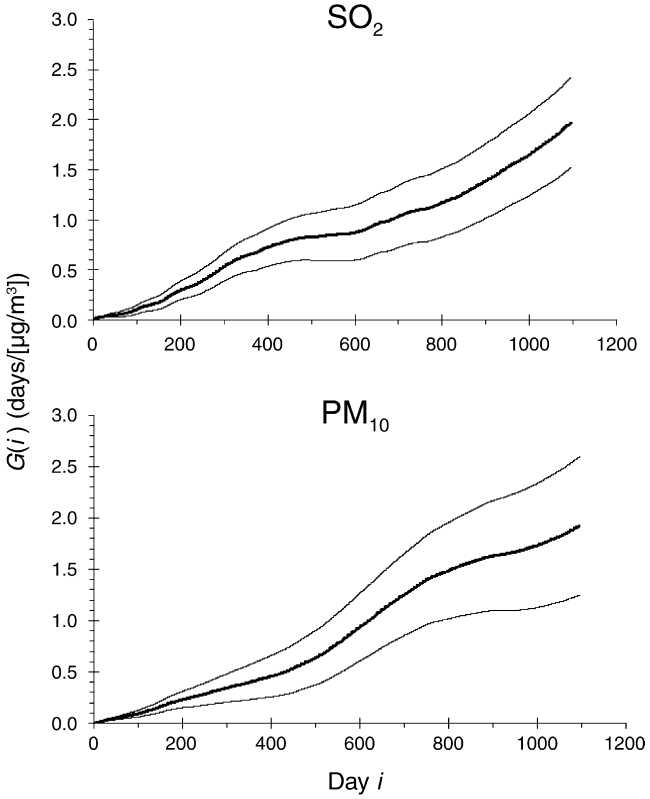

Figure 24. Results for the *G*(*i*) coefficient of the regressions against the second differences (see equation 16), using single-pollutant fits with adjustments *before* the regressions, for SO_2 and PM_{10}. (Thinner lines represent CIs.)

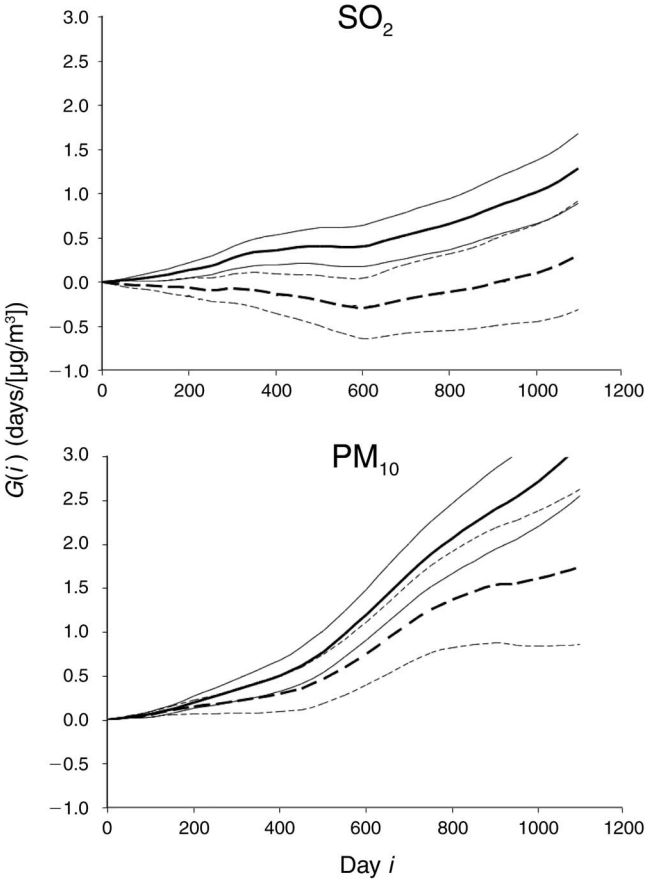

Figure 25. Results for the *G*(*i*) coefficient of the regressions against the second differences, with adjustments *within* the regressions (see equation 16), for SO_2 and PM_{10} (solid line = one-pollutant fit; dashed line = two-pollutant fit; thinner lines represent CIs).

Sensitivity Analyses

To answer possible concerns about adjustments for confounders, we performed several sensitivity studies. The first addressed the adjustment for T and RH. We included in these adjustments for only the current day, for the current day plus the preceding 2 days, and for the current day plus the preceding 30 days. Table 19, as an example, shows a comparison of results using the different adjustment periods for T and RH for the single-pollutant regression against the second differences of SO_2, with an observation window of 1096 days; the adjustments for T and RH and for seasonal and long-term trends were done *within* the regression. The table shows both the coefficient $f(0)$, which equals $G(0)$, and the change in life expectancy obtained with this observation window. Clearly, different adjustment periods for T and RH entail changes in these coefficients that are entirely negligible compared with the SE.

To test the sensitivity to seasonal confounding, we repeated some of the regressions while adding an extra-sinusoidal variation to the death rates, comparable in magnitude to the observed seasonal variation. Specifically, we

Table 19. Sample Sensitivity Study for Adjustment for T and RH: Results for Single-Pollutant Regression Against the Second Differences of SO_2[a]

Days for T and RH adjustment	Coefficient	SE
$f(0)$		
1 day	0.00747	0.0013
3 days	0.00804	0.0013
30 days	0.00883	0.0013
Life expectancy change[b]		
1 day	12.85	2.0
3 days	12.93	2.0
30 days	12.44	2.0

[a] $f(0)$ indicates the coefficient for lag 0 day of conventional time series; RH indicates relative humidity; "second differences" equal the combination of pollutant concentrations; T indicates temperature.

[b] Life expectancy change is for an observation window of 1096 days.

took the death rates (as adjusted for seasonality and trend *before* the regression) and multiplied them by a factor of $1 + 0.05 \times \cos[2\pi(\text{day} + \text{day}_0)/365]$, choosing different values (0, 91, 182, and 273) for day_0. Then we repeated the second difference regressions (as described in the earlier subsection "Adjustments Made Before the Regressions Against Pollution") using these modified rates. The results for the $f(0)$ coefficient changed by less than the SE. For the change in life expectancy, the results varied by ± 28% for SO_2 and by ± 22% for PM_{10}, depending on the choice of day_0. Such variation is a bit larger than the SE but still within the 95% CI. We conclude that seasonal confounding can be appreciable but does not invalidate this approach.

DISCUSSION

Comparison with Other Short-Term Studies

Table 20 summarizes our results for the impact coefficient $f(0)$ ("lag 0"), which has also been measured in most of the conventional time-series studies of the effects of air pollution on mortality. Among the many studies of that type, we cite Stieb and colleagues (2002) and Daniels and colleagues (2004). The study led by Stieb is appropriate because it was a large, worldwide meta-analysis (109 studies) and included SO_2; its estimate of excess risk for mortality due to all natural causes for single-pollutant models, using pooled random effects, is shown in Table 20. Daniels and colleagues (2004) reported the results for PM_{10} of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) for the 20 largest cities in the United States, as found using the log-linear model for the average of the current and previous day. Note that the time-series studies before 2003 suffered from the fact that the default algorithm used for generalized additive models tended to overestimate the effect by perhaps a third; the current study is not affected because it does not use that algorithm. Table 20 compares our results for Hong Kong with these two studies. The results are not out of line, considering that the CIs of meta-analyses and large multicity studies are much smaller than the range of values found in individual studies. The current results are also in line with those from a local study, namely, an ER of 0.87% (95% CI, 0.38–1.36) for SO_2, and 0.53% (95% CI, 0.26–0.81) for PM_{10} (Wong et al. 2008; HEI Public Health and Air Pollution in Asia 2010).

Comparison with Other Long-Term Studies

Table 21 shows a summary of our results for the change in life expectancy and a comparison with other long-term studies. Here the recent study of long-term mortality due to black smoke and SO_2 by Elliott and colleagues (2007) is most relevant. These authors measured the coefficients $F_{0\text{–}4\text{yr}}$, $F_{0\text{–}8\text{yr}}$, $F_{0\text{–}12\text{yr}}$, and $F_{0\text{–}16\text{yr}}$, corresponding to the exposure windows of 0–4, 0–8, 0–12, and 0–16 years. These coefficients represent the sums of the impact coefficients from 0 to 4, 8, 12, and 16 years, respectively. The results of the study by Elliott and colleagues for mortality due to all natural causes are reproduced in Table 22. The

Table 20. Comparison of Results of the Current Study for Impact Coefficient $f(0)$ with Conventional Time-Series Studies[a,b]

Study	PM_{10} ER (95% CI)	SO_2 ER (95% CI)	Comments
Current study: regressions of concentrations, time series of equation 9, but with sums over variable intervals in equation 13	0.71 (0.52 to 0.90)	1.40 (1.18 to 1.62)	Single-pollutant regressions with 7 intervals of length 3^k days, k = 0–6, adjustments *before* regression
Current study: regressions of second differences of concentrations, equation 16	0.67 (0.4 to 0.93)	1.40 (1.1 to 1.7)	Single-pollutant regressions with 1096 coefficients, adjustments *before* regression
Current study: regressions of second differences of concentrations, equation 16	0.21 (−0.03 to 0.46)	0.75 (0.49 to 1.0)	Single-pollutant regressions with 1096 coefficients, adjustments *within* regression
Stieb et al. (2002)	0.64 (0.48 to 0.77)	0.36 (0.28 to 0.48)	Worldwide meta-analysis (109 studies)
Daniels et al. (2004)	0.28 (0.16 to 0.41)		NMMAPS of the 20 largest cities in the U.S.

[a] Data are expressed in % excess risk for all-cause mortality per 10-μg/m^3 increase in pollutant.

[b] $f(0)$ indicates the coefficient for lag 0 of conventional time series; NMMAPS stands for National Morbidity, Mortality, and Air Pollution Study.

Table 21. Comparison of Life Expectancy Losses from Different Studies[a]

Study	PM_{10} (days)	SO_2 (days)	Comments
Current study: regressions of concentrations, time series of equation 9, but with sums over variable intervals in equation 13	7.9[b]	7.8[b]	Single-pollutant regressions with 7 intervals of length 3^k days, k = 0–6, adjustments *before* regression
Current study: regressions of second differences of concentrations, equation 16	19.2 (12.5–25.9)[b]	19.7 (15.2–24.2)[b]	Single-pollutant regressions with 1096 coefficients $G(i)$, adjustments *before* regression
Current study: regressions of second differences of concentrations, equation 16	31.4 (25.6–37.2)[b]	12.8 (8.9–16.8)[b]	Single-pollutant regressions with 1096 coefficients $G(i)$, adjustments *within* regression
Elliott et al. (2007)	39 × concentration conversion factor of black smoke/PM_{10}	48	Concentration conversion factor is location dependent
Cohort studies, in particular Pope et al. (2002), with calculation of life expectancy loss by Rabl (2003)	92[c]	110[d]	

[a] Data are expressed in days per 10 μg/m^3.
[b] Note that the estimates from the current study are only a lower bound because the $G(i)$ had not yet leveled off.
[c] Taking relative risk = 1.06 for 10 μg/m^3 $PM_{2.5}$ from Table 2 of Pope et al. (2002) and assuming a factor of 0.6 for the conversion from $PM_{2.5}$ to PM_{10}.
[d] Rough estimate, using relative risk from Figure 5 of Pope et al. (2002).

fact that the coefficient F decreased with an increase in the length of the window indicates that the impact coefficients are negative after 4 years (due to displaced deaths) and are still different from zero between 12 and 16 years, for both black smoke and SO_2.

Using equation 12 (but employing the time interval of 4 years instead of 1 day) and the data in the Elliott study, we calculated a loss in life expectancy of 39 days per 10-μg/m^3 increase in black smoke and of 48 days per 10-μg/m^3 increase in SO_2.

Table 22. Results of Elliott et al. (2007) for All-Cause Mortality by Exposure Window (Adjusted for Deprivation and Urban/Rural Classification)[a]

Exposure Window (years)	ER % (95% CI)
Black smoke (per 10 μg/m^3)	
0–4	1.3 (1.0 to 1.6)
0–8	0.7 (0.6 to 0.9)
0–12	0.5 (0.5 to 0.6)
0–16	0.4 (0.4 to 0.4)
SO_2 (per 10 ppb)	
0–4	4.2 (3.6 to 4.8)
0–8	2.5 (2.2 to 2.7)
0–12	1.6 (1.4 to 1.7)
0–16	1.0 (0.9 to 1.1)

[a] Based on data from Elliott et al. 2007.

A calculation for loss of life expectancy in cohort studies has been devised by Rabl (2003) and Miller and Hurley (2003), based on the study by Pope and colleagues (2002). The results depend, of course, on the value of the relative risk chosen, and the results vary somewhat with the population to which the relative risk is applied. Typical results for loss of life expectancy are around 92 days per 10 μg/m^3 of $PM_{2.5}$ (PM with an aerodynamic diameter ≤ 2.5 μm). Pope and colleagues also found a significant association of mortality due to all natural causes with SO_2 as shown in Figure 5 of their report (Pope et al. 2002), and Krewski and colleagues concurred in the reanalysis (Krewski et al. 2000). From the report by Pope and colleagues, we obtained a rough estimate of 110 days per 10 μg/m^3 of SO_2.

Parallels between our study and a cohort study run by Schwartz and colleagues (2008) should also be noted. These authors carried out an extended follow-up of the original Harvard Six Cities cohort study (Dockery et al. 1993). One of the features of that work was an analysis by exposure window ("lag"), with exposure intervals of 1 to 5 years preceding death. Only associations with $PM_{2.5}$ were reported.

However, whereas Schwartz and colleagues found that the relative risk decreased rapidly from one year to the next and was negligible beyond the second year, we found that past exposure was significant for at least 3 years. This result of the Schwartz study is very different from that of another recent cohort study by Puett and colleagues (2009), which found that the coefficients for mortality due to all natural causes attributable to $PM_{2.5}$ increased for exposures in previous years up to 3 years and begin to decline only slightly in the fourth year (the longest exposure considered in this study). We conclude that the issue of the relevant exposure window does not yet seem to be settled.

Our results for change in life expectancy represent the lower bounds and are consistent with what is implied in the long-term studies of Pope and colleagues (2002) and Elliott and colleagues (2007) (see Table 21). Like Elliott and colleagues, we found a very significant association of mortality with SO_2 in Hong Kong, even though many epidemiologists doubt that SO_2 could have such an effect. Conceivably, this effect could be due to transition metals, in particular Ni and V, that are emitted by the dominant SO_2 sources, namely, the combustion of oil or coal. Such metals have been identified in some studies as possible agents in the increase in toxicity of ambient PM (Lippmann et al. 2006). In Hong Kong, the SO_2 comes mainly from the combustion of heavy fuel oil, which contains significant amounts of such metals. The ratios Ni/SO_2 and V/SO_2 are extremely variable from site to site because the trace metal content of coal and oil may vary by an order of magnitude between different sources; additional differences can arise from the pollution control technologies used. Such variability could explain the lack of consistency between different studies of the health impacts of SO_2.

As is so often the case with time-series studies, the coefficients may change more than one would expect when another pollutant is added to a single-pollutant model. This is due to the unfortunate fact that the concentrations of different pollutants tend to be correlated. Even for a single pollutant, the concentrations during different time intervals are correlated, and so it is not surprising that the impact coefficients may turn out to be fairly different between regressions with different interval choices.

The losses in life expectancy shown in the current study were expected to be and in fact were smaller than the ones implied by Elliott and colleagues (2007) and Pope and colleagues (2002) since our observation window was only 3 years, whereas these other studies had windows of 16 years (see Table 21). In particular, the results of Elliott and colleagues in Table 22 imply that the impact coefficients are still nonzero between 12 and 16 years. Thus, with a window of only 3 years, we could not capture the full life expectancy loss.

Relation Between Short- and Long-Term Effects

Our model, in particular equation 9, is a straightforward generalization of the conventional time-series model, which includes the effect of long-term exposures. The observation window (i.e., the number of lags included in equation 9) determines how far back in time the exposures are taken into account. The same consideration applies to cohort studies. However, whereas most cohort studies have exposure windows longer than four years, it is difficult to find data for a time-series study with such a long observation window. For the present study, we had access to an exceptionally long data series — 21 years' worth of air pollution data — and we could have tried longer windows, up to a maximum length of about 10 years. However, we also wanted to include the period of the sulfur intervention of July 1990, and since the mortality data started only in January 1985, we could not extend the window much beyond four years (recall that if the time series starts at time t_0, then the pollution data must be available for the entire length of the observation window before t_0).

Finally, we point out that the unique characteristic of displaced deaths — specifically, that they always occur once and only once — in a time-series study does not apply to endpoints such as hospital visits, which an individual can experience several times or not at all. Such endpoints show up very differently in a time-series study of interventions. After a permanent reduction in pollution, mortality will decrease only temporarily and will then return to the initial rate when the postponed deaths finally occur, whereas the incidence of other endpoints will be permanently reduced.

ESTIMATION OF CHANGES IN LIFE EXPECTANCY USING POISSON REGRESSION (OBJECTIVES 3 AND 4)

In developing the method discussed in the last section, "Air Pollution Effects on Changes in Life Expectancy Using Linear Regression (Objectives 2 and 4)," we assert that for any small increase in mortality rate after a pulse of air pollution, the small period of time taken for that increase is equal to the loss in life expectancy for the deaths effected. While this model is simple, it is difficult to observe in reality. In this section, we discuss another approach we developed to assess the long-term effects of air pollution. This approach is based on a state-of-the-art method using Poisson regression modeling with long distributed-lag effect patterns and a mathematically and empirically proven relationship between changes in life expectancy and changes in standardized mortality ratios.

The data used to illustrate the methods in this section are from daily time series of mortality, observations of

meteorologic conditions, and air pollution measurements in Hong Kong for 1985–2005. Since we had the age-group–specific number of deaths and population statistics from 1981–2007, we were able to calculate the life expectancy and standard mortality ratio for each year during this period. Daily concentrations for the whole territory of Hong Kong were estimated as the arithmetic means over the five stations. This approach to obtaining the population-averaged exposure is justified, as the correlations among the stations are high. The Spearman correlations between the five stations ranged from 0.90 to 0.93 for PM_{10}; 0.41 to 0.66 for SO_2; 0.56 to 0.82 for NO_2; and 0.67 to 0.90 for O_3. Daily mean temperature (°C) and relative humidity (%) were obtained from the Hong Kong Observatory.

METHODS AND DESIGN

Survival Curves for Dynamic Cohorts

To illustrate our model, we constructed a dynamic population with cohorts of subjects at consecutive age groups entering the population year by year. This population is subject to the same set of ASDRs as the reference (unexposed) population.

Suppose at time i and for age group x, we obtained the death rate $\mu_i(x)$ in the reference population and $\mu_i^*(x)$ in the exposed population. In the case in which air pollution affects all age groups by the same magnitude, there would be a fixed relative risk (RR) that applies across all age groups, so that $\mu_i^*(x) = RR \times \mu_i(x)$, and the change in life expectancy could be estimated by means of the life table approach (Brunekreef 1997). With the assumption of homogeneity, the ratio $\mu_i^*(x)/\mu_i(x)$ equals SMR_i (Tsai et al. 1992). These calculations suggest that SMR can be regarded as an RR if the index population is the exposed group and the reference population is the unexposed group. However, the assumption in mortality ratios of homogeneity among age groups is in general not true. Lai and colleagues (1996) suggests the following formulation:

$$\frac{\mu_i^*(x)}{\mu_i(x)} = SMR_i + \delta(x) \qquad (17)$$

This is subject to $\Sigma w_i(x) \times \delta(x) = 0$, where $w_i(x) = \mu_i(x)\, P_i^*(x) / \Sigma \mu_i(x)\, P_i^*(x)$ are weights that are set in proportion to the expected number of deaths; $\delta(x)$ is the residual fluctuation of the age-specific mortality ratio; and $P_i^*(x)$ is the proportion of the exposed population of age group x. Note that with the introduction of $\delta(x)$, we allow variations in the relation between changes in life expectancy and SMR, which we discuss in the next section.

Burnett and colleagues (2003) showed that the short-term time-series method can be used to assess air-pollution–associated changes in mortality for dynamic cohorts, provided that the following conditions hold: (a) the environmental covariates vary in time and not between individuals; (b) on any given day, the probability of death is small; (c) each subject of the at-risk population has the same probability of death after adjusting for known risk factors; (d) all members of the at-risk population share a common effect of environmental covariates on mortality; and (e) the population-averaged baseline hazard function and association between risk factors and death can be approximated adequately by smooth functions of time. Conditions (a) and (b) should be applicable to both time-series and dynamic cohort settings. Condition (c), since the estimation is for population average and the assessment is made for subjects between time points, is usually not a problem to meet in time-series studies and for time-dependent factors of a dynamic cohort model. Condition (d) is a basic assumption in all time-series studies and could also be applicable to dynamic cohorts. The model we developed, known as the "Core Model," can be assessed for how adequately it handles the adjustment of time varying confounding using a state-of-the-art methodology for air pollution and mortality time-series studies, and therefore can be used as the baseline hazard function for dynamic cohorts over time, usually by means of smooth functions of time under condition (e). With conditions (a) through (e), dynamic cohorts are in homogeneous survival models under which the adjusted mortality risks are the same for all the age groups under study.

From Figure 26, we can see that for any change in mortality, $\mu_i^*(x) - \mu_i(x)$, there is a corresponding change in

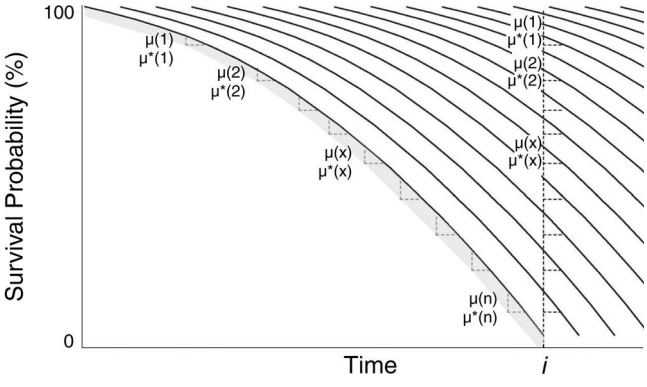

Figure 26. Hypothetical survival curves for various birth cohorts, assuming each birth cohort has the same set of ASDRs (at time i, observed deaths, $D_i = \Sigma \mu_i^*(x)\, P^*(x)$, and expected deaths, $E_i = \Sigma \mu_i(x)\, P_i^*(x)$, where $\mu_i^*(x)$ and $\mu_i(x)$ are death rates in the exposed and reference population, and $P^*(x)$ is the exposed population of age group x). The left-most curves represent the survivals for a reference cohort, and the shaded area represents the decline in survival after the exposure. The vertical dotted line forming a triangle shows the mortality rate in the reference population, $\mu_i(x)$, and exposed population, $\mu_i^*(x)$, in time i, corresponding to the left-most survival curves.

life expectancy, and that at any time point, the SMR can be derived from observed (exposed) mortality counts and expected mortality counts from a dynamic cohort, under the assumption of homogeneous survival, as the reference. Burnett and colleagues (2003) also point out that under the assumption of a homogeneous survival model, the survival for a fixed cohort is equivalent to that of a dynamic cohort, and under this circumstance, an assessment of the changes in life expectancy can be made. In the following subsection, we describe how we developed a method to assess the relation between changes in death rates and changes in life expectancy; and in the subsequent subsection, we discuss a method to assess changes in life expectancy from a time-series analysis.

Relation Between Changes in Mortality Rates and Life Expectancy

We defined LE_i^* as life expectancy at age a for the exposed subjects, and LE_i as the reference population at time i. For ease of illustration here, we dropped the index i. Thus,

$$\begin{aligned}
LE^* &= \int_a^\infty \exp\{-\int_a^x \mu^*(s)\,ds\}dx \\
&= \int_a^\infty \exp\{-\int_a^x [\mu^*(s)/\mu(s)]\,\mu(s)\,ds\}dx \\
&= \int_a^\infty \exp\{-\int_a^x [SMR + \delta(s)]\,\mu(s)\,ds\}dx \ldots \\
&\qquad\qquad \text{(from equation 17 above)} \\
&= \int_a^\infty \exp\{-SMR\int_a^x \mu(s)\,ds - \int_a^x \delta(s)\,\mu(s)\,ds\}dx \\
&= \int_a^\infty \exp\{-(SMR-1)\int_a^x \mu(s)\,ds - \int_a^x \delta(s)\,\mu(s)\,ds \\
&\quad - \int_a^x \mu(s)\,ds\}dx
\end{aligned}$$

Using the Mean Value Theorem for integration (Lai et al. 1996), we see there exists an a′ in (a, ∞), such that

$$\begin{aligned}
LE^* = \exp\{-(SMR-1)\int_a^{a'} \mu(s)\,ds - \int_a^{a'} \delta(s)\,\mu(s)\,ds\} \\
\int_a^\infty \exp\{-\int_a^x \mu(s)\,ds\}dx
\end{aligned}$$

In general a′ should be specific to the individual SMR. Here, for simplicity, we assume it is the same for all SMR; but we will take another possibility into account in the Discussion later in this section of the report.

Now, we define LE as follows:

$$LE = \int_a^\infty \exp\{-\int_a^x \mu(s)\,ds\}dx$$

Therefore,

$$LE^* = \exp\{-(SMR-1)\int_a^{a'} \mu(s)\,ds - \int_a^{a'} \delta(s)\,\mu(s)\,ds\} \times LE$$

$$\begin{aligned}
\log\frac{LE^*}{LE} &= -(SMR-1)\int_a^{a'} \mu(s)\,ds - \int_a^{a'} \delta(s)\,\mu(s)\,ds \\
&= \gamma(SMR-1) + \alpha, \qquad (18)
\end{aligned}$$

where $\gamma = -\int_a^{a'} \mu(s)\,ds$; and $\alpha = -\int_a^{a'} \delta(s)\,\mu(s)\,ds$

We obtain an estimate for the slope, γ, and the intercept, α, by fitting equation 18 with data points for each of the years 1981 through 2007, using year 1996 as the reference.

We compare two exposures at concentration level c and $c + 1$ with reference to the same LE at the same time point, and obtain the following:

$$\log\frac{LE^*_{c+1}}{LE^*_c} = \gamma(SMR_{c+1} - SMR_c) = \gamma\Delta SMR, \qquad (19)$$

where $\Delta SMR = SMR_{c+1} - SMR_c$.

We estimated γ from equation 18 using the available data sets, which were conveniently based on yearly data (provided that there was no seasonality or other time-varying confounding).

As mentioned earlier, γ and α should be specific to each SMR. In order to take this into account, we could have used random effects instead of the fixed effects found in equation 18 by employing the following equation:

$$\log\frac{LE_j^*}{LE} = \gamma_j(SMR-1) + \alpha_j, \qquad (20)$$

where γ_j and α_j follow a normal distribution with a certain mean and variance. In this study, we did not implement equation 20, since in a linear model, the parameter estimates are expected to be similar in fixed- and random-effects models. Under a random-effects model, the SE might be larger, but in our example, this variation in the SE size should be relatively minor compared with the other expected variations.

In the earlier subsection "Survival Curves for Dynamic Cohorts," we showed that the SMR at any time point can be treated as a ratio of mortality counts from dynamic cohorts to those expected from a fixed cohort. In this subsection, we have shown how change in the SMR after change in an exposure level may result in a change in life expectancy. In the next section, we show that a change

in SMR and the corresponding change in life expectancy can be estimated from a time series of death counts, using state-of-the-art daily air pollution time-series analysis methods.

Estimation of Changes in Life Expectancy from Time-Series Analyses

In time-series studies, we define D_i as the number of deaths observed at time i; and the expected number, $E_i = \Sigma\mu_i(x)\, P_i^*(x)$, where $P_i^*(x)$, is the proportion of the exposed population of age group x (Figure 26).

We modeled a time series for $i = 1, 2, \ldots, n$ days:

$$\log[\mathrm{E}(D_i)] = \mathrm{offset}(\log(E_i)) + i + \sin(2\pi i/365.25) + \cos(2\pi i/365.25) + \mathrm{temperature}_i + \mathrm{humidity}_i + \beta_0 + \sum\beta_r c_{i-r} + \varepsilon_i \quad (21)$$

where E( ) is an expectation of the outcome variable. For simplicity in the above model equation, we ignored the coefficient associated with each variable except that for the exposure variable c_{i-r}, which stands for pollutant concentrations of up to r lag days.

We defined the Core Model as follows:

$$\text{Core Model} = \mathrm{offset}(\log(E_i)) + i + \sin(2\pi i/365.25) + \cos(2\pi i/365.25) + \mathrm{temperature}_i + \mathrm{humidity}_i + \beta_0 \quad (22)$$

This equation filters out long-term trend, seasonality, and other time-dependent potential confounding factors, as mentioned in the earlier subsection "Survival Curves for Dynamic Cohorts," and when we discussed developing equation 19. Note that "offset" is a function that defines the coefficient of $\log(E_i)$ as equal to 1, so that in modeling $\log(D_i)$, we are in fact modeling $\log[E(D_i/E_i)]$ — in other words, $\log[E(\mathrm{SMR}_i)]$.

We obtained the estimate

$$\log(\hat{D}c_i) = \text{Core Model} + \sum\hat{\beta}_r c_{i-r}, \quad (23)$$

where $\hat{D}c_i$ is model-fitted deaths when the exposure is set at c_{i-r}, where $r = 0,1,2, \ldots$

Note that, in practice, the total expected number of deaths, E, is derived for each year, and then the expected E_i for each day is obtained by dividing it by the total number of days in a year. The lack of seasonal variation in this definition should be taken into account by the seasonality terms in the Core Model (as shown by the seasonality pattern in Figure 27). The following equation illustrates this:

$$\log(\hat{D}_{c_i+1}) = \text{Core Model} + \sum\hat{\beta}_r c_{i-r} + \sum\hat{\beta}_r \quad (24)$$

For convenience, we dropped index i in the following formulation subtracting equation 23 from equation 24:

$$\log\frac{\hat{D}_{c+1}}{\hat{D}_c} = \hat{\beta} \ldots \text{ where } \hat{\beta} = \sum\hat{\beta}_r$$

This can be restated as

$$\log\frac{\hat{\mathrm{SMR}}_{c+1}}{\hat{\mathrm{SMR}}_c} = \hat{\beta} \ldots$$

(with the expected number of deaths obtained from the same reference), so that

$$\Delta\hat{\mathrm{SMR}} = \hat{\mathrm{SMR}}_c[\exp(\hat{\beta}) - 1] \cong \hat{\mathrm{SMR}}_c\hat{\beta}, \quad (25)$$

where $[\exp(\hat{\beta}) - 1]$ is the excess risk, which can be approximated by $\hat{\beta}$ when $\hat{\beta}$ is small.

Now, building on equation 19 in order to generalize to a change in Δc, we have the following:

$$\mathrm{LE}^*_{c+\Delta c} = \mathrm{LE}^*_c[\exp(\hat{\gamma}\Delta c\Delta\hat{\mathrm{SMR}})] = \mathrm{LE}^*_c[\exp(\hat{\gamma}\Delta c\hat{\mathrm{SMR}}_c\hat{\beta})] \ldots$$

using equation 25 and estimating ΔSMR from $\Delta\hat{\mathrm{SMR}}$.

Therefore, we can let $L = \mathrm{LE}^*_c$, representing the life expectancy for a reference with an exposure level c, so that $\mathrm{SMR}_c = 1$, and the life expectancy after a change of Δc units of pollutant concentration is L*:

$$\mathrm{L}^* = L[\exp(\hat{\gamma}\Delta c\hat{\beta})] \quad (26)$$

The change in life expectancy can be estimated by the following:

$$\Delta L = \mathrm{L}^* - L = L[\exp(\hat{\gamma}\Delta c\hat{\beta}) - 1] \quad (27)$$

In this equation, we use all ages as an illustration; however, $\hat{\gamma}$ and $\hat{\beta}$ can be estimated for specific age groups (Lai et al. 1996). This is an important advantage over a standard life table approach in which uniform RR is assumed for all age groups.

Note that $\hat{\gamma}$ is negative; therefore $\exp(\hat{\gamma}\Delta c\hat{\beta})$ is a fraction less than 1 for positive $\hat{\beta}$, and a negative ΔL signifies a loss in life expectancy after the exposure.

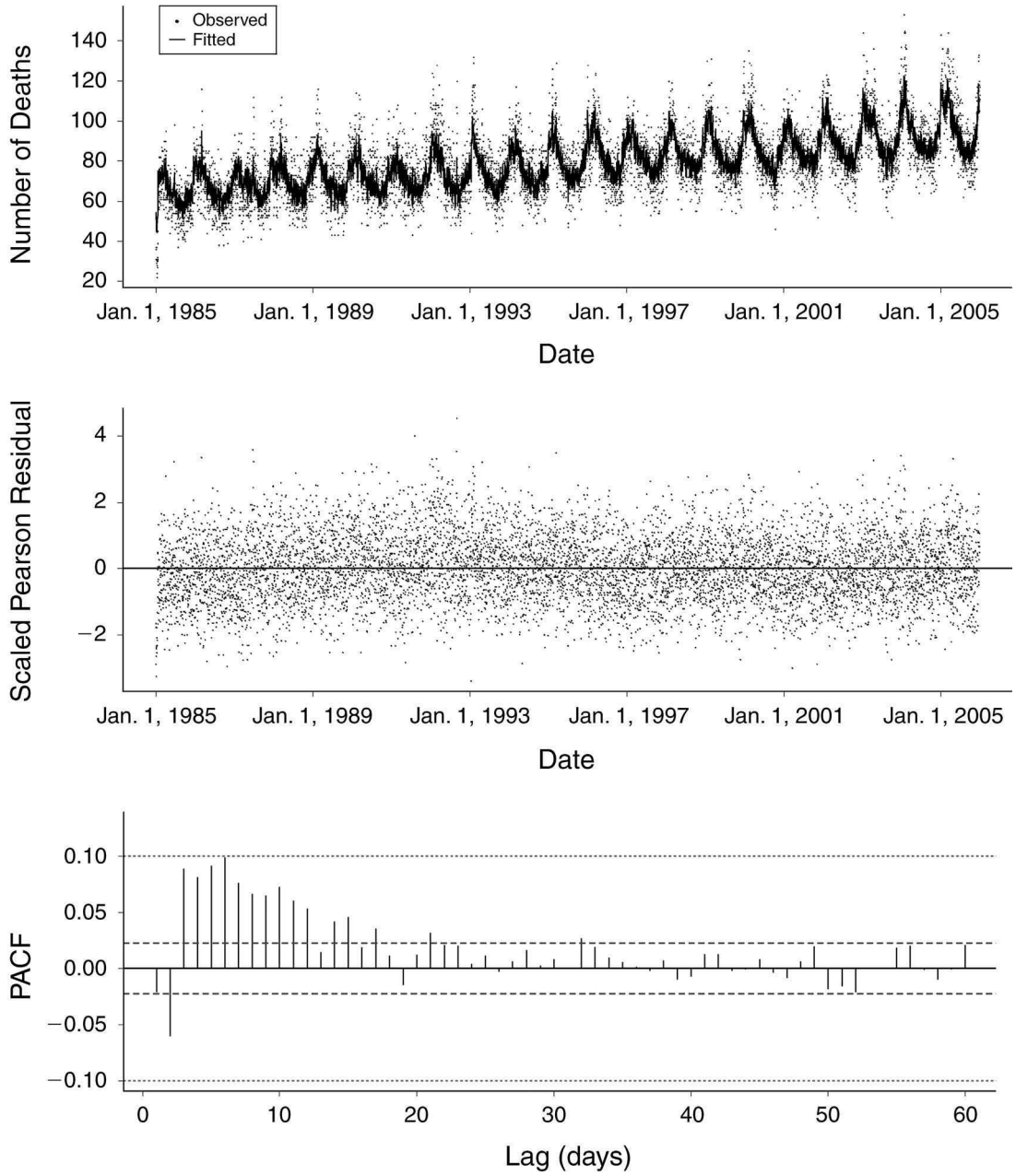

Figure 27. Plots of fitted and observed mortality counts, scaled Pearson residuals, and PACFs of residuals resulting from fitting the Core Model with adjustments for trend, seasonality, temperature, and relative humidity with a second-order autoregressive term, for 1985–2005.

Estimation of Changes in Life Expectancy After an Intervention

Suppose the effect of an air quality intervention lasts for n days, and we estimate the effect of air pollution per unit concentration with β_i for $i = 1, 2, \ldots, n$ days. Then, from equation 26, the life expectancy after n days of the intervention with a constant change in concentration (Δc) would be

$$L^* = L\{\exp[\hat{\gamma}\Delta c(\sum\hat{\beta}_i)]\} \quad (28)$$

for $i = 1$ to n days of intervention.

Therefore, change in life expectancy after the intervention would be calculated as follows:

$$\Delta L = \mathrm{LE}\{\exp[\hat{\gamma}\Delta c(\sum\hat{\beta}_i)] - 1\} \quad (29)$$

for $i = 1$ to n.

Note that since $\hat{\gamma}$ is negative and since, in the case where the intervention leads to a reduction in air pollution, $\Delta c(\Sigma\hat{\beta}_i)$ will have a negative value, we have $\exp[\hat{\gamma}\Delta c(\Sigma\hat{\beta}_i)] > 1$. This yields a positive ΔL, indicating a gain in life expectancy. Also note that this $(\Sigma\hat{\beta}_i)$ is similar to the effect estimate of (Σf_i), which we developed in the section "Air Pollution Effects on Changes in Life Expectancy Using Linear Regression (Objectives 2 and 4)," and that an average effect for each day can be estimated by $(\Sigma\hat{\beta}_i)$ divided by the total number of days in the model. Therefore, the cumulative effect of all the lag days is $(\Sigma\hat{\beta}_i)$.

DATA ANALYSIS

Definition of a Core Model for Short- and Long-Term Exposure

We assessed the effects of air pollution on acute daily deaths for populations with short- and long-term exposure. We defined an a priori Core Model to be used in equation 23, with a simple linear trend for the time variable i ($i = 1, 2, \ldots, n$ days), a pair of trigonometric terms for seasonality, and T and RH. We checked the plots for residuals and for partial auto-correlation functions (PACFs) for model adequacy. We used the same criteria that we adopted in the Public Health and Air Pollution in Asia project (Wong et al. 2008; HEI Public Health and Air Pollution in Asia 2010) to adequately adjust for time-varying confounding when PACF is between −0.1 and 0.1 for some lag days and if there were no discernible patterns in the residuals. We were able to add observable time-variation variables and/or auto-regressive terms for a maximum of 5 lag days, if these criteria were not met.

In our main analysis, we assessed the effects of air pollution on mortality with lags of up to 1461 days (4 years) for all ages. We believe this captures the major effects of any pulse of air pollution, including the changes in mortality advancement.

Sensitivity Analysis

We performed a sensitivity analysis with the following changes:

- Used one auto-regressive term more or one less than the main analysis in the Core Model
- Adjusted the duration of the observation window to 1096 lag days (3 years)
- Adjusted the distributed lag with a constraint using a fourth-degree polynomial function to define the shape of the distributed lag for the β coefficients (Goodman et al. 2004)
- Changed the age group to 40 years or older
- Adjusted for smoking prevalence
- Adjusted for gross domestic product to account for the positive correlation between high gross domestic product and increased longevity (Swift 2011)

RESULTS

With an "offset" in expected deaths, a linear term for time, and a pair of trigonometric terms for seasonality, we readily accounted for the trend and seasonal variations in mortality counts over a period of 21 years. The plots of fitted values were close to the observed mortality counts, scaled Pearson residuals appeared random, and the PACF plots of residuals were between −0.1 and 0.1 (Figure 27), showing that the Core Model was adequate in controlling for time-varying confounding. The estimated coefficients, SEs, and t values of the variables in the Core Model were statistically significant (Table B.10 in Appendix B). The pattern of estimated excess risk for a cumulated lag effect was positive for PM_{10} and SO_2, null for NO_2, and negative for O_3 (Figure 28). The estimate risk for PM_{10} was 1.65% (95% CI, −0.04 to 3.34) and for SO_2 was 3.18% (95% CI, 2.26 to 4.10) (Table 23).

The relation between change in life expectancy and SMR is shown in Figure 29; parameter estimates of the slope are −0.143 with an SE of 0.002. For each unit change in SMR, the change in LE was about 14% from the reference year 1996 (data not shown).

The change in life expectancy for a 10-μg/m^3 increase in pollutant concentrations in the past 4 years was −69.1 (95% CI, −133.1 to 1.4) for PM_{10} and was −133.1 (95% CI, −171.8 to −94.4) for SO_2 (Table 23).

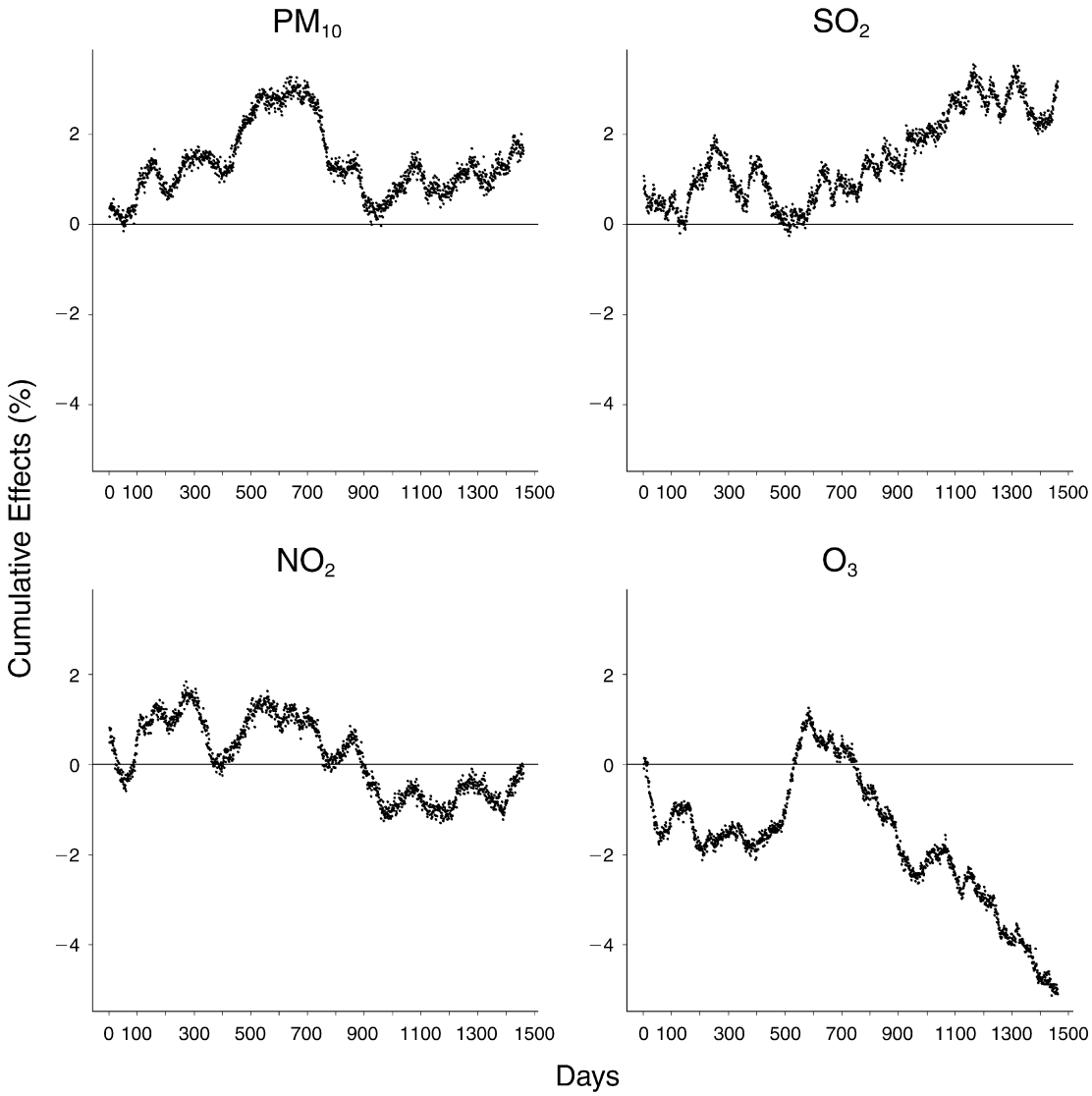

Figure 28. The sum of the cumulative effects (%) for PM_{10}, SO_2, NO_2, and O_3 per 10 µg/m^3 increase in pollutant for an unconstrained distributed-lag model using 1461 lag days, 1985–2005.

Table 23. Estimated Cumulative Effects and Change in Life Expectancy for Gaseous Pollutants and PM_{10} Using Poisson Regression[a]

Pollutant	Estimated Cumulative Effects % (95% CI)	Change in Life Expectancy Days (95% CI)
NO_2	−0.19 (−1.86 to 1.48)	7.97 (−61.93 to 77.87)
SO_2	3.18 (2.26 to 4.10)	−133.08 (−171.75 to −94.41)
PM_{10}	1.65 (−0.04 to 3.34)	−69.13 (−139.69 to 1.44)
O_3	−5.09 (−6.34 to −3.84)	214.27 (160.88 to 267.67)

[a] Data are expressed per 10 µg/m^3 for unconstrained distributed-lag model with 1461 lag days.

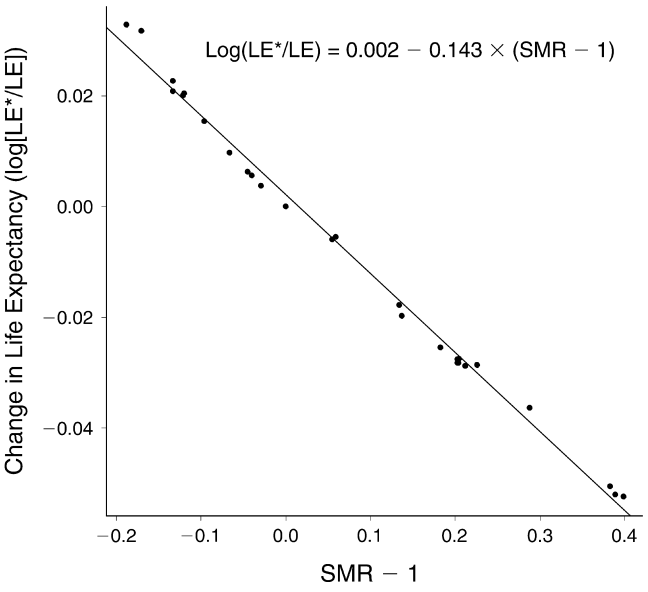

Figure 29. Plot of observed and fitted regression line for standardized mortality ratio (SMR), with change in life expectancy between the exposed (LE*) and the reference (LE) populations using the ASDRs in 1996 for expected deaths in Hong Kong, 1981–2007.

With a more stringent criterion (using one more autoregressive term than in the main analysis) and a more lax criterion (using one less autoregressive term), the respective excess risk estimates varied from 1.7% to 1.4% and 2.0%, respectively, for PM_{10}, and from 3.2% to 3.0% and 3.5%, respectively, for SO_2 (see Tables 23 and 24). The variations are in the expected direction. With the exposure variable, using a time scale of 1096 days (3 years), the estimate increased to 2.4% for PM_{10} and decreased to 2.8% for SO_2; with the constraint variable, the PM_{10} estimate decreased to 1.0% and the SO_2 estimate decreased to 3.0%; and with the use of the ASDR for the year 1990 as the standard, the PM_{10} estimate increased to 1.7% and the SO_2 estimate to 3.3%. With the control for smoking prevalence, the excess risk estimate decreased to 1.2% (from 1.7%) for PM_{10}, but increased to 3.8% (from 3.2%) for SO_2. But with control for the gross domestic product, the PM_{10} estimate decreased to 0.9% and the SO_2 estimate to 2.8% (Table 24).

Based on a 12-μg/m^3 reduction in concentration for SO_2 (the interquarter range of the annual average for the whole study period) after the intervention, the gain in life expectancy was 161 days (95% CI, 114 to 207) as a demonstration of how this method can be applied in assessing the effects of the Hong Kong intervention (data not shown).

DISCUSSION

We have developed a new approach for the estimation of life expectancy that is compatible with that developed in the previous section "Air Pollution Effects on Changes in Life Expectancy Using Linear Regression (Objectives 2 and 4)." Both approaches are based on estimates derived from the excess risks with long lags in a time-series analysis. Ideally, these excess risks are generic and transferable between populations of similar susceptibility. However, the approach discussed in this section generates estimates that, together with the relationship established in the literature (Lai et al. 1996) between changes in mortality and life expectancy, would allow an estimation of changes in life expectancy due to air pollution applicable to other populations. This is different from the approach used in the last section, which assesses the absolute changes in effects, and consequently, the results of which may not be applicable to other populations with different life expectancies as baselines.

Assumptions Used for the Reference Population

In the approach developed in this section, we made an assumption of homogeneous survival in deriving a reference for the daily death counts and in obtaining a change in SMR interpretation of the effect estimates from time-series models. After we derived the effect estimate with the use of an "offset" and control for seasonality and time-dependent covariates, it no longer was reliant on the choice of the reference. As we have shown in the sensitivity analysis, with the use of different references (with different ASDRs), the effect estimation is rather robust. However, in the actual implementation, a reference based on the whole period of the study rather than on a particular year may be the better choice. On the other hand, other sources contributing to a lack of homogeneity are not accounted for.

Poisson Regression Modeling

Using an "offset" on the expected number of deaths according to a standard set of ASDRs, we came to an interpretation of the effect estimate as a change in SMR. The "offset" also facilitated fitting a much simpler Core Model for daily mortality counts. This was achieved by incorporating the information from the standard set of ASDRs and the population distribution for each year into the Core Model. Thus, confounding due to changes in the population structure was adjusted for directly in a simple and explicit way. This approach is recommended in fitting the regression model to the data from a time series covering a long period.

Table 24. Sensitivity Analysis: Estimated Cumulative Effects and Change in Life Expectancy for PM_{10} and SO_2 Using Poisson Regression[a]

Model / Pollutant	Estimated Cumulative Effects % (95% CI)	Change in Life Expectancy Days (95% CI)
Core model with AR(1)[b]		
PM_{10}	2.02 (0.31 to 3.73)	−84.61 (−155.97 to −13.24)
SO_2	3.49 (2.57 to 4.41)	−146.02 (−184.72 to −107.32)
Core model with AR(3)[c]		
PM_{10}	1.41 (−0.26 to 3.08)	−59.08 (−128.84 to 10.68)
SO_2	3.02 (2.12 to 3.92)	−126.40 (−164.25 to −88.55)
Window of 1096 lag days		
PM_{10}	2.43 (1.16 to 3.70)	−101.75 (−155.09 to −48.41)
SO_2	2.80 (2.04 to 3.56)	−117.21 (−149.34 to −85.08)
Constraint with 4th degree polynomial		
PM_{10}	1.16 (−0.50 to 2.83)	−48.70 (−118.46 to 21.06)
SO_2	3.11 (2.20 to 4.03)	−130.32 (−168.64 to −92.00)
40 years or older age group		
PM_{10}	1.12 (−0.59 to 2.83)	−41.53 (−104.70 to 21.63)
SO_2	2.86 (1.94 to 3.78)	−105.83 (−139.98 to −71.69)
Adjustment for smoking prevalence		
PM_{10}	1.19 (−0.63 to 3.01)	−49.87 (−126.21 to 26.47)
SO_2	3.77 (2.77 to 4.77)	−157.71 (−199.68 to −115.73)
Adjustment for gross domestic product		
PM_{10}	0.88 (−2.30 to 4.06)	−36.89 (−170.07 to 96.30)
SO_2	2.84 (1.76 to 3.92)	−118.88 (−164.11 to −73.65)
Using 1990 as reference year		
PM_{10}	1.74 (0.05 to 3.43)	−89.86 (−176.80 to −2.91)
SO_2	3.34 (2.42 to 4.26)	−172.23 (−219.86 to −124.61)

[a] Data are expressed in days per 10 μg/m^3.

[b] Core model with AR(1): $\log[E(D_i)] = \text{offset}(\log(E_i)) + i + \sin(2\pi i/365.25) + \cos(2\pi i/365.25) + \text{temperature} + \text{humidity} + \log(D_{-1})$.

[c] Core model with AR(3): $\log[E(D_i)] = \text{offset}(\log(E_i)) + i + \sin(2\pi i/365.25) + \cos(2\pi i/365.25) + \text{temperature} + \text{humidity} + \log(D_{-1}) + \log(D_{-2}) + \log(D_{-3})$, where D_{-1} D_{-2}, and D_{-3} are the single lags of previous 1 day, previous 2 days, and previous 3 days of mortality, respectively.

Effect Estimates for Excess Risk for Exposure to PM_{10}

In this study, our most conservative ER estimate was 1.65% (CI: −0.04, 3.34) for mortality due to all natural causes per 10 μg/m^3 of PM_{10} exposure over 4 years. This is equivalent to 2.36% per 10 μg/m^3 of $PM_{2.5}$ exposure (assuming a $PM_{2.5}/PM_{10}$ ratio of 0.7) (Ho et al. 2003), which is comparable to a range of 2.8% to 4.4% in excess deaths per 10 μg/m^3 of $PM_{2.5}$ exposure for 5 years published in an analysis of the American Cancer Society (ACS) data (Krewski et al. 2009). The estimates from the ACS study are, in general, smaller than those from the Harvard Six Cities Study (Dockery et al. 1993) but are close to the estimates in our study. A recent reanalysis of the Harvard Six Cities Study (Laden et al. 2006) estimated an excess risk of 16% per 10-μg/m^3 increase in $PM_{2.5}$ exposure for 1 to 8 years, which is much higher than our estimate and those of other cohort studies.

However, there were some important differences between the Harvard Six Cities Study and the ACS study: in the Harvard Six Cities Study, the samples were obtained from a smaller number of cities in the eastern region of the United States, while those of the ACS study were more geographically representative, with a large number of participants from cities distributed across the whole United States. The discrepancy in results from these studies highlights the importance of having a representative population sample in cohort studies.

Effect Estimates for Excess Risk for Exposure to Gaseous Pollutants

The long-term effects of SO_2 seen in this section are consistent with estimates from the previous section, "Air Pollution Effects on Changes in Life Expectancy Using Linear Regression" (in the subsection "Results with Adjustments for Temperature and Relative Humidity Before the Regression Against Pollutants [Objectives 2 and 4]"), and were found to be larger than for PM_{10}. These results are also consistent with the short-term effects of air pollution in Hong Kong found in a previous study by Wong and colleagues (Wong 2008; HEI Public Health and Air Pollution in Asia 2010), as mentioned in the Discussion in the previous section. In an extended ACS cohort study with a follow-up of more than 16 years, elevated mortality risks were found to be strongly associated with $PM_{2.5}$ and SO_2, but not with the other gaseous pollutants (Jerrett et al. 2009). In our study, we did not find any positive effects of NO_2 and O_3 in the long term. Conversely, we found significant negative effects of O_3 (Table 23). Further research on the long-term effects of gaseous pollutants, particularly for O_3, is needed.

Effect Estimates on Changes in Life Expectancy

In the previous section, "Air Pollution Effects on Changes in Life Expectancy Using Linear Regression (Objectives 2 and 4)," we presented the dependent variable as a ratio of the observed mortality counts over a reference rate (using the WHO population profile as the standard) and as a continuous variable. We, therefore, analyzed it using a linear regression model. Ordinarily, a regression coefficient is interpreted as a change in the ratio of mortality rates per unit change in the pollutant concentration and is considered to be related to instantaneous changes in the average life expectancy of the population. However, in this section of the report (covering Objectives 3 and 4), we considered the dependent variable to be a count variable, and therefore, we used the Poisson regression model. But, since we included an "offset" of a log transformation of the expected counts derived from a standard set of ASDRs, the dependent variable was interpreted as log SMR and the coefficient as the change in log SMR, which can also be related to the change in life expectancy. In contrast, in the previous section, we treated change in life expectancy as a direct identity using coefficients from the linear regression.

The effect estimates for changes in life expectancy calculated using Poisson regression are the same level of magnitude as those calculated using linear regression. The reason that estimates from the Poisson regression calculation were higher than those from the linear regression (69 and 133 days vs. 19.2 and 19.7 days, respectively, for PM_{10} and SO_2) is that the observation window used in the Poisson regression was longer than that in the linear regression calculation (4 years vs. 3 years, respectively). The difference may also be due to the use of a value of life expectancy as the reference in the Poisson calculation. Basing the estimate of changes in life expectancy on a certain population reference may be an advantage for public health interpretation, as a population with a shorter life expectancy would be expected to have a smaller change in mortality per unit of pollutant concentration than one with a longer life expectancy.

Using linear regression, we found clear effects of SO_2 and PM_{10} on life expectancy, both in single- and two-pollutant models. Since the curves of the coefficients $G(i)$ and i do not level off within the largest observation window under consideration, we have only lower bounds for the change in life expectancy due to PM_{10} and SO_2. These are consistent with other estimates of change in life expectancy, in particular those by Elliott and colleagues (2007) and Pope and colleagues (2002).

Using Poisson regression, in this section we assessed the effects of SO_2, NO_2, PM_{10}, and O_3 on life expectancy. We did not find effects for NO_2, a finding that is consistent with the U.S. cohort studies mentioned earlier but inconsistent with a European study (Brunekreef et al. 2009). We did find a negative excess risk with an increase in O_3, but as O_3 is a secondary pollutant that may be reduced by increasing levels of NO_2, we therefore will not attempt to interpret the results.

Using Poisson regression, for PM_{10} and SO_2, we found an effect estimate consistent with other long-term effect studies. In our estimation, we captured all the temporal variations in more than 20 years for the whole population and obtained the best available estimates. In addition to these long-term effect estimates, we assessed the effect due to a government intervention, which led to a reduction of SO_2, in terms of a change in life expectancy. But the life expectancy estimate was based on a uniform reduction in SO_2 after the intervention, and should be interpreted with caution in view of our discussion in the previous section of the difficulties encountered in assessing the changes after the intervention.

Sensitivity Analyses

We performed a number of sensitivity analyses with a view to assessing the robustness of the assumptions and presumed conditions in our model. We used a method to control for serial correlations that involved introducing autoregressive terms so that the PACF was between -0.1 and 0.1 using the criteria for acceptance of a core model in a recently established common protocol used in several

Asian studies (Wong et al. 2008; HEI Public Health and Air Pollution in Asia 2010). We added or subtracted one autoregressive term in the core model, used a window of 1096 lag days and a constraint of a fourth-degree polynomial for the lag effect patterns, included subjects 40 years or older, adjusted for smoking prevalence or gross domestic product, and used 1990 as reference year. The results showed that, in general, the estimates of the sum of excess risks and changes in life expectancy were fairly robust and were consistent in their magnitude and direction as expected.

According to the time-series methodology, further adjustments may not be necessary in the assessment stage, as time-varying confounding should have been dealt with during Core Model development. However, in our sensitivity analysis, with further adjustments we saw some variations in effect estimates; but such variations are in the direction we expected, and the magnitude of the changes are reasonably small.

Relation Between Short- and Long-Term Effects

Researchers have shown that the use of distributed-lag time-series Poisson regression models is a valuable instrument in the estimation of short- to middle-term effects of air pollution (Zanobetti et al. 2002; Goodman et al. 2004). For lags longer than a few months, it is assumed that it is not possible to disentangle the estimates from uncontrolled long-term effects. However, we propose that this may be possible using long distributed-lag models, for both short- and long-term effects.

The results of our study provide support for the hypothesis that time-series Poisson regression modeling can be used for assessing two categories of death associated with air pollution suggested by Künzli and colleagues (2001): specifically, that "air pollution increases both the risk of underlying diseases leading to frailty and the short-term risk of death among the frail"; and that "air pollution is unrelated to risk of chronic diseases but short-term exposure increases mortality among persons who are frail." With an established state-of-the-art method to control for time-varying confounding, the time-series analysis method can be used to assess these two categories of death associated with air pollution. However, as compared with a cohort design, our design for the assessment of long-term effects is less complete, and the effect estimates we calculated are population-averaged rather than subject-specific.

With the control of the change in population structure in the Core Model, we can interpret excess risks as change in SMR due to air pollution and transform the estimate into gain in life expectancy. Indeed, with all of the information about the time variations of air pollution and mortality available in a homogeneous population over a long period and with an assumption of homogeneous survival in models, a time series may be regarded as comprising dynamic cohorts, and the resulting analysis may be an appropriate approach to the assessment of change in life expectancy due to short- and long-term air pollution exposure.

Limitations

The most important limitation of our approach involves the assumption of homogeneous survival. The assumption of homogeneous survival is unrealistic and hard to verify in data with complicated social, environmental, and climatic conditions. However, this assumption has very little significance, as has been shown in our sensitivity analysis, which demonstrates that by varying the ASDR, the change in effect estimates can be kept small and in the direction consistent with expectation. Even though there may be uncontrolled heterogeneity in data, this condition would not mitigate the usefulness in environmental public health policymaking of effect estimates that use population-averaged estimates.

A second limitation is that we have not taken copollutants into account, which may lead to a failure of the estimates to reflect the unique effects of a specific pollutant. However, controlling for copollutants with more than a thousand lag effect variables in a single model is computationally intensive. Besides, the estimates from copollutant models may be hard to interpret even in simple short-term time-series studies.

Another limitation is that we did not consider the possibility of a distribution of risk relative to exposure within the population. As in all daily time-series analyses, the whole population for a defined geographic location was compared between time points. Time-dependent variables may be a source of confounding; however, time-independent variables do not cause the same problem, and, therefore, the estimates in the current study are not subject to confounding. However, we are aware that populations residing in different geographic areas may have different susceptibilities because of differences in socioeconomic and environmental factors, and the effects of these factors might be heterogeneous.

Furthermore, the impact of air pollution at different ages has been accounted for in our study only implicitly; however, in order to allow variations in effects on different ages, we should have assessed the relations between life expectancy and SMR in many different specific age groups. In theory, we can extend similar computations to other age groups, such as those comprising people 25 years or older, in order to obtain ASDRs and life expectancy rates, as well as the relation between SMR and life expectancy. Also, the length of the observation period used in this study

is meant to serve only as an example. In theory, this should be determined according to biologic and toxicologic effects of any pulse of air pollution to which people are exposed. Further work is warranted in assessing the validity of the method and the appropriate length of observation.

Finally, similar to other long-term effect studies, our study design assumed that the mobility of the population was low so that we could use monitored air pollution concentrations as proxy measures for personal exposure. This may introduce errors into the exposure measurements, leading to lower estimates from this analysis. Also, similar to other time-series studies, our study only estimates the population average and did not consider subject-specific effect modifiers.

CONCLUSIONS

Particulate matter chemical species are health hazards and increase the risk of mortality. In particular, concentrations of both Ni and V (chemical species in PM_{10}) were associated with an increased risk of mortality before the 1990 sulfur restriction intervention in Hong Kong. Their levels were markedly reduced after the intervention. We cannot exclude the possibility that decreases in their concentrations were responsible for some of the observed health benefits due to the intervention. However, with the PM chemical species data available only once every 6 days, and with data not available at all at some monitoring stations, the observed differences in the health effects between the pre- and post-intervention periods cannot be confirmed.

With complete daily air pollution and mortality data over a long period of time, time-series analysis methods can be applied to assess the short- and long-term effects of air pollution, in terms of changes in life expectancy. Two approaches to the assessment of the health effects of air pollution are available: one based on an additive model using linear regression, and the other based on a multiplicative model using Poisson regression. In the former, absolute changes in life expectancy with given changes in air pollutant concentrations over past exposure can be obtained by direct summation of the impacts for individual daily exposures. In the latter, proportional changes in life expectancy can be obtained by summation of the effects from past exposures, with adjustment by a multiplicative factor, in order to reflect the relation between changes in life expectancy and changes in SMR, and to account for the life expectancy of a reference population. Both approaches can be applied to the assessment of air quality improvement after an intervention, and together they form the basis of a methodology for the assessment of life expectancy due to air pollution from daily time-series data.

This methodology may prove to be an important development in the formulation of environmental public health policies regarding the effects of air pollution in terms of life expectancy and may produce results relevant to local populations and understandable to the general public. This will be particularly useful for locations outside of North America and Western Europe, where there have not been cohort studies to provide estimations of the long-term effects of air pollution.

Further work is warranted to assess the duration and the pattern of effects of an air pollution pulse. In particular, there is a need to determine an appropriate length in time relating to the biologic and toxicologic effects of a pulse of air pollution, and the pattern of effects over time that can be assumed.

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APPENDIX A. HEI Quality Assurance Statement

The Final Report of this study was subjected to independent audit by Linda Morris Brown, M.P.H., Dr.P.H.; Breda Munoz, M.S., Ph.D.; and Constance V. Wall, B.S. (RTI International). They are experts in quality assurance for air quality monitoring studies, epidemiology, statistics, and data management. The date of the audit is listed below with the audit findings.

April 2011: Review of Draft Final Report

The audit included a review of the procedures for data collection, processing, and analysis, and of the data set utilized in the final report relative to original data set for the project. A written report of the audit was provided to the HEI project manager, who transmitted the findings to the Principal Investigator. No issues related to data quality were noted, though some inconsistencies between the data

in the report and some tables and figures were identified. Additional information on the QA/QC of the collected data was also requested.

September 2011: Review of QA Audit Report Queries

The Principal Investigator submitted detailed responses to the QA audit report queries. The quality assurance team noted that their queries were addressed adequately. The audit demonstrated that the study was conducted by an experienced team with a concern for data quality. The final report appears to be an accurate representation of the study.

Linda Morris Brown, M.P.H., Dr.P.H.
Senior Research Epidemiologist, RTI International
September 21, 2011

APPENDIX B. Additional Data and Results

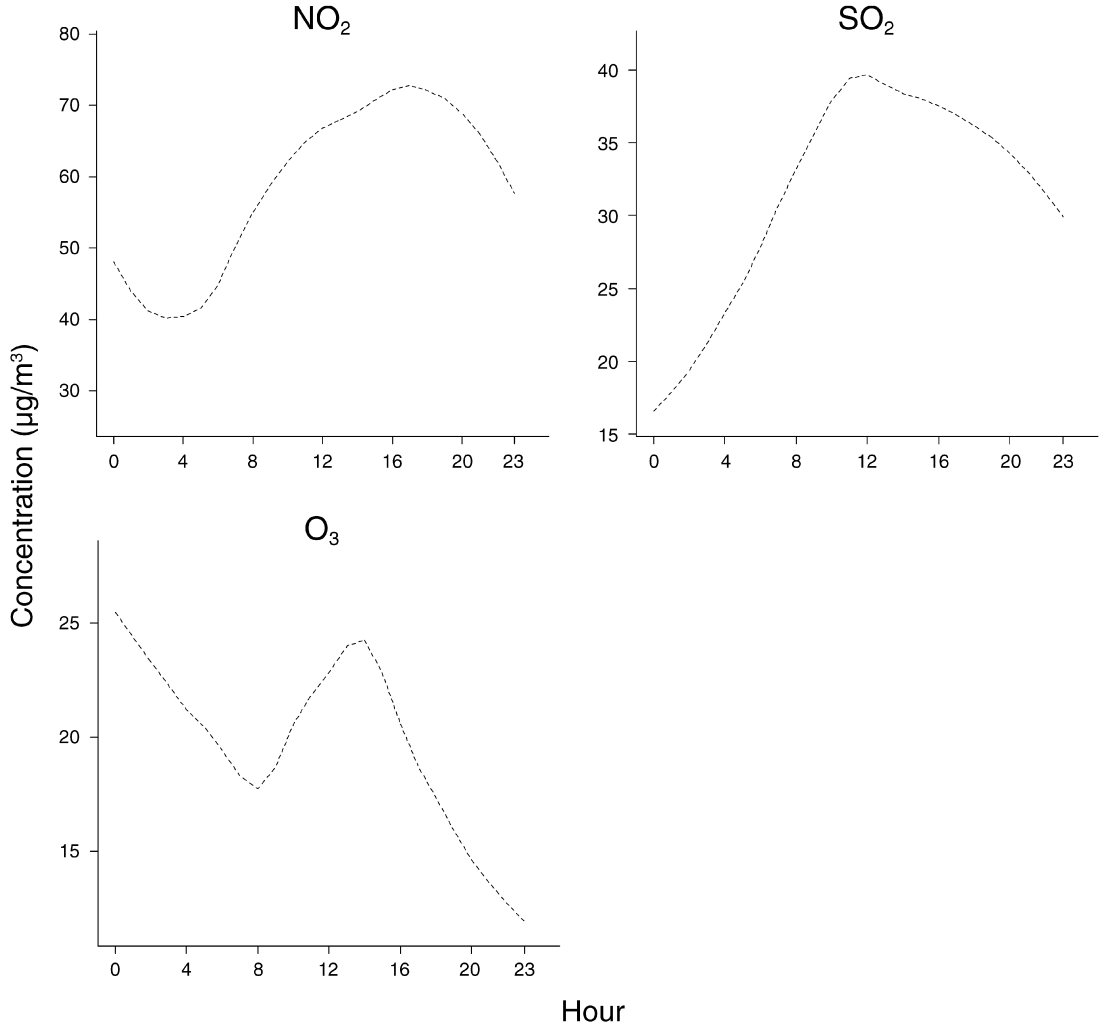

Figure B.1. Diurnal plot of NO_2, SO_2, and O_3 concentrations, averaged over 5 stations, from 1985–1995.

Table B.1. Time of Operation for Monitoring Stations in Hong Kong

Analyses / Abbreviation	Monitoring Station	Operation Period
Main		
CW	Central/Western	1983–present
KT	Kwun Tong	1983–present
SSP	Sham Shui Po	1984–present
KC	Kwai Chung	1988–present
TW	Tsuen Wan	1988–present
Excluded		
HKS	Hong Kong South	1989–1993
JB	Junk Bay	1981–1993
TST	Tsim Sha Tsui	1982–1993
CB	Causeway Bay	1982–1990
MK	Mong Kok	1991–present[a]
ST	Sha Tin	1991–present
TP	Tai Po	1990–present
YL	Yuen Long	1995–present

[a] MK became a roadside monitoring station in year 2001.

Table B.2. Summary Statistics of PM Chemical Species by Type of Area in Different Time Periods[a]

	Whole				Pre-Intervention				Post-Intervention			
Pollutant	*n*	Min	Max	Mean ± SD	*n*	Min	Max	Mean ± SD	*n*	Min	Max	Mean ± SD
Al	561	20.33	6173.00	358.60 ± 421.99	274	20.33	6173.00	407.80 ± 461.69	287	25.40	3650.00	311.50 ± 375.06
Industrial	388	0.00	3300.00	293.60 ± 336.65	106	0.00	1374.00	311.40 ± 243.55	282	23.50	3300.00	286.80 ± 365.73
Mixed	557	20.33	6173.00	370.90 ± 435.14	272	20.33	6173.00	418.10 ± 466.25	285	25.33	4000.00	325.90 ± 398.87
Fe	569	13.00	7099.00	576.00 ± 521.05	282	94.34	7099.00	598.50 ± 519.45	287	13.00	5200.00	553.90 ± 522.57
Industrial	388	0.00	4850.00	505.40 ± 505.08	106	51.24	2062.00	517.90 ± 329.90	282	0.00	4850.00	500.80 ± 557.31
Mixed	565	5.00	7099.00	597.00 ± 534.59	280	115.90	7099.00	608.70 ± 523.30	285	5.00	5850.00	585.50 ± 546.13
Mn	569	0.00	173.50	19.62 ± 16.81	282	0.00	173.50	21.43 ± 16.81	287	0.00	155.00	17.84 ± 16.65
Industrial	388	0.00	130.00	15.34 ± 14.74	106	0.00	62.00	15.72 ± 12.25	282	0.00	130.00	15.20 ± 15.60
Mixed	565	0.00	180.00	20.84 ± 17.69	280	0.00	173.50	22.09 ± 16.86	285	0.00	180.00	19.61 ± 18.42
Ni	569	0.00	46.00	6.40 ± 5.91	282	1.20	46.00	8.57 ± 6.67	287	0.00	45.00	4.27 ± 4.07
Industrial	388	0.00	45.00	7.90 ± 7.58	106	0.00	40.00	15.84 ± 7.35	282	0.00	45.00	4.92 ± 5.12
Mixed	565	0.00	46.00	5.36 ± 5.34	280	0.00	46.00	7.03 ± 6.50	285	0.00	18.50	3.72 ± 3.10
V	569	0.00	120.00	14.55 ± 14.18	282	1.00	110.00	17.87 ± 16.51	287	0.00	120.00	11.29 ± 10.49
Industrial	388	0.00	120.00	21.17 ± 20.53	106	0.91	99.00	42.95 ± 19.70	282	0.00	120.00	12.98 ± 13.75
Mixed	565	0.00	110.00	11.47 ± 12.02	280	0.00	110.00	13.18 ± 15.08	285	0.00	53.50	9.79 ± 7.61
Pb	569	6.00	358.00	70.64 ± 43.43	282	18.75	248.70	74.43 ± 32.91	287	6.00	358.00	66.92 ± 51.52
Industrial	388	4.85	275.00	64.31 ± 46.57	106	12.50	144.00	64.41 ± 31.63	282	4.85	275.00	64.27 ± 51.12
Mixed	565	6.00	486.70	71.13 ± 44.43	280	19.00	248.70	74.79 ± 33.17	285	6.00	486.70	67.55 ± 53.03
Zn	563	7.00	607.00	127.20 ± 80.74	276	14.50	607.00	146.00 ± 88.61	287	7.00	542.00	109.00 ± 67.72
Industrial	388	0.00	521.00	114.90 ± 75.12	106	19.00	521.00	143.60 ± 84.98	282	0.00	500.00	104.20 ± 68.17
Mixed	559	7.33	740.00	126.30 ± 84.70	274	14.50	607.00	143.20 ± 92.07	285	7.33	740.00	110.10 ± 73.56

[a] Data are expressed in ng/m^3; *n* indicates number of days measured.

Table B.3. Sensitivity Analysis of Assessment Periods of Different Lengths: Excess Risk of Mortality Due to Air Pollution in 2-Year Pre-Intervention Period and Change in Excess Risk After Intervention[a]

Disease / Age Group / Pollutant	Main Effects (2-Year Pre-Intervention Period) ER (95% CI)	Change in Effects (vs. 2-Year Pre-Intervention Period) ER (95% CI)
All Natural Causes		
All ages		
NO_2	0.34 (−0.41 to 1.09)	0.87 (0.02 to 1.73)
SO_2	0.32 (−0.20 to 0.84)	0.31 (−0.41 to 1.04)
PM_{10}	0.10 (−0.36 to 0.57)	0.06 (−0.52 to 0.64)
O_3	0.59 (−0.15 to 1.33)	0.07 (−0.73 to 0.86)
Cardiovascular		
All ages		
NO_2	0.01 (−1.30 to 1.35)	1.04 (−0.44 to 2.55)
SO_2	0.94 (0.03 to 1.85)	0.44 (−0.82 to 1.72)
PM_{10}	−0.26 (−1.23 to 0.72)	0.17 (−1.02 to 1.38)
O_3	0.30 (−1.01 to 1.64)	−0.33 (−1.73 to 1.09)
65+		
NO_2	−0.18 (−1.68 to 1.35)	1.28 (−0.41 to 3.01)
SO_2	1.20 (0.17 to 2.25)	0.48 (−0.96 to 1.93)
PM_{10}	0.13 (−0.90 to 1.17)	−0.32 (−1.58 to 0.96)
O_3	0.14 (−1.37 to 1.67)	−0.24 (−1.85 to 1.39)
Respiratory		
All ages		
NO_2	0.15 (−1.52 to 1.86)	1.55 (−0.34 to 3.48)
SO_2	−0.39 (−1.55 to 0.77)	1.16 (−0.45 to 2.80)
PM_{10}	0.43 (−0.36 to 1.23)	−0.08 (−1.06 to 0.91)
O_3	1.18 (−0.49 to 2.87)	−0.17 (−1.94 to 1.62)
65+		
NO_2	0.67 (−1.17 to 2.54)	1.05 (−1.01 to 3.14)
SO_2	0.02 (−1.24 to 1.30)	1.06 (−0.68 to 2.83)
PM_{10}	0.34 (−0.53 to 1.22)	0.04 (−1.03 to 1.12)
O_3	1.63 (−0.20 to 3.49)	−0.76 (−2.68 to 1.19)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day, except PM_{10} at current day.

Table B.4. Sensitivity Analysis of Assessment Periods of Different Lengths: Excess Risk of Mortality Due to Air Pollution from PM Chemical Species (CS) in 2-Year Pre-Intervention Period and Change in Excess Risk After Intervention[a]

Disease / Age Group / PM CS	Main Effects (2-Year Pre-Intervention Period) ER (95% CI)	Change in Effects (vs. 2-Year Pre-Intervention Period) ER (95% CI)
All Natural Causes		
All ages		
Al	0.041 (−0.032 to 0.115)	−0.036 (−0.112 to 0.040)
Fe	0.030 (−0.028 to 0.089)	−0.026 (−0.087 to 0.034)
Mn	0.598 (−1.014 to 2.236)	−0.277 (−1.943 to 1.417)
Ni	3.981 (1.028 to 7.020)	−1.668 (−5.467 to 2.283)
V	1.241 (0.172 to 2.321)	−0.776 (−2.276 to 0.748)
Pb	0.151 (−0.463 to 0.768)	−0.083 (−0.737 to 0.575)
Zn	−0.135 (−0.345 to 0.076)	0.098 (−0.174 to 0.371)
65+		
Al	0.028 (−0.063 to 0.120)	−0.011 (−0.105 to 0.083)
Fe	0.030 (−0.043 to 0.102)	−0.016 (−0.091 to 0.058)
Mn	0.538 (−1.432 to 2.547)	0.091 (−1.956 to 2.182)
Ni	5.266 (1.690 to 8.967)	−2.342 (−6.808 to 2.339)
V	1.743 (0.449 to 3.054)	−1.545 (−3.341 to 0.284)
Pb	0.185 (−0.574 to 0.951)	−0.148 (−0.954 to 0.666)
Zn	−0.092 (−0.350 to 0.167)	0.011 (−0.325 to 0.348)
Cardiovascular		
All ages		
Al	0.040 (−0.117 to 0.198)	−0.089 (−0.249 to 0.072)
Fe	0.020 (−0.106 to 0.146)	−0.058 (−0.187 to 0.071)
Mn	−0.095 (−3.500 to 3.430)	−0.862 (−4.351 to 2.754)
Ni	2.417 (−3.652 to 8.868)	−0.604 (−8.634 to 8.132)
V	0.665 (−1.582 to 2.964)	−0.360 (−3.542 to 2.927)
Pb	−0.332 (−1.600 to 0.951)	0.120 (−1.230 to 1.489)
Zn	−0.459 (−0.903 to −0.013)	0.288 (−0.289 to 0.868)
65+		
Al	−0.010 (−0.185 to 0.166)	−0.034 (−0.211 to 0.144)
Fe	−0.027 (−0.168 to 0.115)	−0.009 (−0.153 to 0.135)
Mn	−1.904 (−5.638 to 1.977)	0.860 (−3.069 to 4.949)
Ni	2.372 (−4.379 to 9.599)	−0.832 (−9.372 to 8.513)
V	0.473 (−2.005 to 3.014)	−0.045 (−3.466 to 3.498)
Pb	−1.041 (−2.398 to 0.336)	0.878 (−0.564 to 2.342)
Zn	−0.533 (−1.015 to −0.049)	0.294 (−0.305 to 0.896)

(*Table continues on next page*)

[a] Data are expressed in % per 10-ng/m^3 unit change.

Table B.4 (*Continued*). Sensitivity Analysis of Assessment Periods of Different Lengths: Excess Risk of Mortality Due to Air Pollution from PM Chemical Species (CS) in 2-Year Pre-Intervention Period and Change in Excess Risk After Intervention[a]

Disease / Age Group / PM CS	Main Effects (2-Year Pre-Intervention Period) ER (95% CI)	Change in Effects (vs. 2-Year Pre-Intervention Period) ER (95% CI)
Respiratory		
All ages		
Al	0.035 (−0.096 to 0.166)	−0.030 (−0.165 to 0.105)
Fe	0.043 (−0.060 to 0.147)	−0.038 (−0.145 to 0.069)
Mn	0.617 (−2.227 to 3.545)	−0.553 (−3.495 to 2.479)
Ni	6.990 (1.555 to 12.716)	−3.462 (−10.134 to 3.705)
V	2.106 (0.197 to 4.051)	−1.008 (−3.717 to 1.776)
Pb	0.546 (−0.545 to 1.649)	−0.556 (−1.716 to 0.617)
Zn	−0.076 (−0.448 to 0.298)	−0.034 (−0.518 to 0.452)
65+		
Al	0.003 (−0.137 to 0.143)	0.017 (−0.126 to 0.160)
Fe	0.035 (−0.077 to 0.147)	−0.015 (−0.129 to 0.099)
Mn	−0.530 (−3.576 to 2.613)	0.928 (−2.266 to 4.227)
Ni	4.768 (−1.092 to 10.974)	−1.659 (−8.839 to 6.086)
V	1.753 (−0.304 to 3.852)	−0.411 (−3.259 to 2.521)
Pb	0.301 (−0.849 to 1.463)	−0.343 (−1.539 to 0.868)
Zn	−0.067 (−0.473 to 0.341)	−0.049 (−0.543 to 0.448)
Accidental		
All ages		
Al	−0.111 (−0.380 to 0.158)	0.143 (−0.129 to 0.416)
Fe	−0.076 (−0.288 to 0.137)	0.105 (−0.109 to 0.320)
Mn	−2.314 (−7.906 to 3.618)	3.357 (−2.680 to 9.768)
Ni	−0.087 (−9.316 to 10.081)	3.225 (−9.822 to 18.159)
V	−0.556 (−4.041 to 3.057)	0.667 (−4.591 to 6.215)
Pb	−0.293 (−2.375 to 1.834)	0.346 (−1.862 to 2.603)
Zn	−0.069 (−0.794 to 0.662)	−0.006 (−0.925 to 0.921)

[a] Data are expressed in % per 10-ng/m^3 unit change.

Table B.5. Spearman Correlations Among Gaseous Pollutants and PM Chemical Species in Assessment Periods of Different Lengths[a]

	NO_2	SO_2	PM_{10}	O_3	Al	Fe	Mn	Ni	V	Pb	Zn
10-Year Period											
NO_2	1.00	0.17	0.40	0.38	0.49	0.53	0.59	0.22	0.05	0.60	0.55
SO_2	—	1.00	0.13	−0.05	0.14	0.17	0.04	0.65	0.64	0.19	0.13
PM_{10}	—	—	1.00	0.27	0.53	0.56	0.54	0.09	0.04	0.48	0.41
O_3	—	—	—	1.00	0.51	0.45	0.43	−0.10	−0.04	0.29	0.22
Al	—	—	—	—	1.00	0.90	0.83	0.18	0.11	0.53	0.45
Fe	—	—	—	—	—	1.00	0.89	0.21	0.13	0.59	0.57
Mn	—	—	—	—	—	—	1.00	0.15	0.07	0.64	0.67
Ni	—	—	—	—	—	—	—	1.00	0.73	0.23	0.26
V	—	—	—	—	—	—	—	—	1.00	0.09	0.11
Pb	—	—	—	—	—	—	—	—	—	1.00	0.71
Zn	—	—	—	—	—	—	—	—	—	—	1.00
5-Year Pre-Intervention Period											
NO_2	1.00	0.07	0.37	0.23	0.41	0.44	0.51	0.09	0.01	0.49	0.40
SO_2	—	1.00	0.06	−0.08	−0.01	0.05	−0.09	0.64	0.68	0.00	−0.02
PM_{10}	—	—	1.00	0.24	0.45	0.44	0.42	0.01	−0.03	0.29	0.22
O_3	—	—	—	1.00	0.44	0.41	0.37	−0.04	−0.10	0.24	0.13
Al	—	—	—	—	1.00	0.91	0.83	0.07	0.05	0.41	0.33
Fe	—	—	—	—	—	1.00	0.90	0.14	0.10	0.53	0.48
Mn	—	—	—	—	—	—	1.00	0.09	0.04	0.59	0.60
Ni	—	—	—	—	—	—	—	1.00	0.92	0.15	0.19
V	—	—	—	—	—	—	—	—	1.00	0.09	0.14
Pb	—	—	—	—	—	—	—	—	—	1.00	0.59
Zn	—	—	—	—	—	—	—	—	—	—	1.00
5-Year Post-Intervention Period											
NO_2	1.00	0.35	0.76	0.50	0.64	0.70	0.71	0.26	0.19	0.72	0.74
SO_2	—	1.00	0.32	0.00	0.24	0.27	0.19	0.64	0.58	0.26	0.27
PM_{10}	—	—	1.00	0.44	0.73	0.74	0.76	0.23	0.07	0.77	0.71
O_3	—	—	—	1.00	0.66	0.55	0.55	−0.11	0.04	0.41	0.37
Al	—	—	—	—	1.00	0.88	0.84	0.09	0.14	0.63	0.58
Fe	—	—	—	—	—	1.00	0.90	0.17	0.13	0.66	0.68
Mn	—	—	—	—	—	—	1.00	0.13	0.11	0.71	0.75
Ni	—	—	—	—	—	—	—	1.00	0.49	0.11	0.20
V	—	—	—	—	—	—	—	—	1.00	0.04	0.09
Pb	—	—	—	—	—	—	—	—	—	1.00	0.83
Zn	—	—	—	—	—	—	—	—	—	—	1.00

Table B.6. Excess Risk of Mortality Due to Air Pollution with Adjustment for PM Chemical Species in Assessment Periods of Different Lengths[a]

Disease / Age Group / Pollutant	Adjustment for Nickel in Particulates		Adjustment for Vanadium in Particulates	
	7 Years ER (95% CI)	10 Years ER (95% CI)	7 Years ER (95% CI)	10 Years ER (95% CI)
All Natural Causes				
All ages				
NO_2	−0.02 (−3.54 to 3.62)	−0.13 (−3.62 to 3.48)	−0.08 (−1.35 to 1.21)	−0.20 (−1.47 to 1.08)
SO_2	1.21 (−3.09 to 5.69)	0.59 (−3.66 to 5.02)	0.09 (−1.45 to 1.65)	−0.20 (−1.73 to 1.35)
O_3	1.76 (−1.39 to 5.00)	1.14 (−1.98 to 4.36)	0.53 (−0.66 to 1.73)	0.31 (−0.87 to 1.50)
Cardiovascular				
All ages				
NO_2	1.58 (−4.92 to 8.53)	3.22 (−3.91 to 10.87)	0.85 (−1.50 to 3.25)	0.60 (−1.96 to 3.22)
SO_2	1.69 (−6.09 to 10.11)	−0.19 (−8.42 to 8.79)	1.05 (−1.76 to 3.94)	−0.39 (−3.41 to 2.72)
O_3	1.15 (−4.57 to 7.21)	0.74 (−5.44 to 7.33)	0.63 (−1.54 to 2.86)	−0.05 (−2.44 to 2.39)
65+				
NO_2	2.79 (−4.33 to 10.45)	−0.58 (−9.30 to 8.98)	0.67 (−1.89 to 3.29)	0.26 (−3.03 to 3.65)
SO_2	0.83 (−7.56 to 9.99)	2.76 (−8.02 to 14.79)	0.05 (−3.01 to 3.19)	1.25 (−2.65 to 5.32)
O_3	1.60 (−4.58 to 8.18)	0.90 (−7.05 to 9.53)	0.31 (−2.05 to 2.73)	0.70 (−2.41 to 3.92)
Respiratory				
All ages				
NO_2	1.81 (−7.25 to 11.76)	−0.47 (−9.79 to 9.81)	1.21 (−2.14 to 4.68)	0.04 (−3.46 to 3.67)
SO_2	5.36 (−5.82 to 17.86)	3.09 (−8.49 to 16.13)	2.14 (−1.85 to 6.30)	0.94 (−3.24 to 5.30)
O_3	5.22 (−3.07 to 14.23)	1.51 (−7.00 to 10.81)	2.22 (−0.95 to 5.50)	0.63 (−2.71 to 4.08)
65+				
NO_2	3.34 (−6.39 to 14.08)	0.49 (−5.34 to 6.68)	1.53 (−2.04 to 5.22)	−0.90 (−3.02 to 1.27)
SO_2	8.20 (−4.01 to 21.97)	0.21 (−6.78 to 7.73)	2.65 (−1.62 to 7.10)	−1.62 (−4.15 to 0.98)
O_3	7.50 (−1.44 to 17.24)	1.20 (−3.94 to 6.62)	2.75 (−0.63 to 6.25)	−0.33 (−2.31 to 1.69)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day.

Table B.7. Excess Risk of Mortality Due to Air Pollution from Gaseous Pollutants in Assessment Periods of Different Lengths Using a Subset of Data for Which Gaseous Pollutants, Nickel, and Vanadium in PM Were Measured on the Same Day[a]

Disease / Age Group / Pollutant	7 Years ER (95% CI)	10 Years ER (95% CI)
All Natural Causes		
All ages		
NO_2	0.98 (0.35 to 1.62)	0.26 (−0.11 to 0.65)
SO_2	0.73 (0.17 to 1.29)	0.21 (−0.20 to 0.63)
O_3	0.13 (−0.35 to 0.60)	0.10 (−0.30 to 0.49)
Cardiovascular		
All ages		
NO_2	0.33 (−0.80 to 1.47)	0.47 (−0.33 to 1.28)
SO_2	0.87 (−0.13 to 1.88)	0.83 (0.12 to 1.54)
O_3	−0.61 (−1.45 to 0.24)	0.44 (−0.09 to 0.98)
65+		
NO_2	−0.05 (−1.29 to 1.21)	−0.59 (−1.82 to 0.65)
SO_2	1.12 (0.02 to 2.23)	0.56 (−0.52 to 1.65)
O_3	−0.95 (−1.87 to −0.02)	−0.92 (−1.78 to −0.06)
Respiratory		
All ages		
NO_2	1.54 (−0.04 to 3.14)	−0.58 (−1.96 to 0.81)
SO_2	1.01 (−0.40 to 2.44)	0.99 (−0.21 to 2.21)
O_3	0.32 (−0.85 to 1.49)	−1.00 (−1.96 to −0.04)
65+		
NO_2	1.87 (0.19 to 3.58)	0.30 (−1.28 to 1.90)
SO_2	1.34 (−0.17 to 2.86)	0.25 (−1.12 to 1.64)
O_3	0.24 (−0.99 to 1.48)	0.33 (−0.73 to 1.39)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day.

Table B.8. Short-Term Effects on Mortality with Adjustment for Nickel in PM Due to Air Pollution for Pre- and Post-Intervention Periods[a]

Disease / Age Group / Pollutant	Pre-Intervention (5-Year) ER (95% CI)	Pre-Intervention (2-Year) ER (95% CI)	Post-Intervention ER (95% CI)
All Natural Causes			
All ages			
NO_2	2.57 (−0.98 to 6.25)	1.77 (−4.92 to 8.93)	−0.52 (−6.12 to 5.42)
SO_2	3.74 (−0.32 to 7.96)	−0.96 (−8.66 to 7.40)	3.28 (−4.58 to 11.78)
O_3	1.08 (−2.85 to 5.18)	2.14 (−4.09 to 8.78)	2.55 (−2.54 to 7.90)
65+			
NO_2	3.74 (−0.40 to 8.06)	−3.86 (−13.72 to 7.13)	0.24 (−6.16 to 7.08)
SO_2	5.42 (0.57 to 10.49)	−13.08 (−22.72 to −2.24)	5.20 (−3.79 to 15.04)
O_3	2.51 (−2.06 to 7.30)	−4.23 (−13.35 to 5.85)	2.75 (−3.06 to 8.91)
Cardiovascular			
All ages			
NO_2	2.16 (−3.34 to 7.96)	3.63 (−9.71 to 18.94)	1.22 (−9.14 to 12.76)
SO_2	4.49 (−1.84 to 11.24)	−11.63 (−24.54 to 3.48)	1.16 (−12.31 to 16.69)
O_3	1.77 (−4.26 to 8.17)	0.05 (−12.17 to 13.97)	1.37 (−7.76 to 11.40)
65+			
NO_2	3.39 (−2.94 to 10.14)	5.53 (−9.13 to 22.56)	4.33 (−7.10 to 17.17)
SO_2	5.88 (−1.45 to 13.75)	7.63 (−11.31 to 30.61)	6.35 (−8.77 to 23.96)
O_3	3.57 (−3.42 to 11.07)	7.32 (−7.11 to 24.01)	3.85 (−6.14 to 14.90)
Respiratory			
All ages			
NO_2	5.87 (−1.26 to 13.52)	1.78 (−12.44 to 18.30)	−2.25 (−14.86 to 12.24)
SO_2	9.12 (0.75 to 18.19)	0.62 (−17.75 to 23.09)	1.40 (−15.84 to 22.19)
O_3	6.32 (−1.61 to 14.89)	5.15 (−9.28 to 21.87)	1.49 (−10.14 to 14.61)
65+			
NO_2	7.80 (−0.16 to 16.41)	−0.81 (−9.28 to 8.46)	−2.42 (−15.27 to 12.39)
SO_2	12.62 (3.18 to 22.93)	−4.13 (−13.04 to 5.70)	6.17 (−12.26 to 28.47)
O_3	8.53 (−0.28 to 18.11)	1.46 (−6.64 to 10.27)	3.99 (−8.09 to 17.65)
Accidental			
All ages			
NO_2	4.24 (−5.67 to 15.19)	10.26 (−12.87 to 39.53)	18.40 (−3.67 to 45.54)
SO_2	4.00 (−7.31 to 16.68)	15.47 (−11.42 to 50.51)	43.00 (6.99 to 91.14)
O_3	7.22 (−4.09 to 19.87)	5.43 (−14.58 to 30.12)	9.22 (−8.45 to 30.29)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day.

Table B.9. Short-Term Effects on Mortality with Adjustment for Vanadium in PM Due to Air Pollution for Pre- and Post-Intervention Periods[a]

Disease / Age Group / Pollutant	Pre-Intervention (5-Year) ER (95% CI)	Pre-Intervention (2-Year) ER (95% CI)	Post-Intervention ER (95% CI)
All Natural Causes			
All ages			
NO_2	1.02 (−0.45 to 2.52)	0.96 (−1.55 to 3.53)	−0.59 (−2.74 to 1.61)
SO_2	1.52 (−0.19 to 3.25)	0.17 (−2.70 to 3.13)	−0.45 (−3.27 to 2.45)
O_3	0.66 (−0.99 to 2.34)	1.07 (−1.28 to 3.48)	0.58 (−1.52 to 2.72)
65+			
NO_2	1.24 (−0.46 to 2.97)	−1.13 (−4.99 to 2.88)	−1.07 (−3.50 to 1.43)
SO_2	1.89 (−0.14 to 3.95)	−4.10 (−8.10 to 0.08)	−1.07 (−4.24 to 2.20)
O_3	0.86 (−1.03 to 2.79)	−1.28 (−4.88 to 2.46)	−0.03 (−2.42 to 2.43)
Cardiovascular			
All ages			
NO_2	1.10 (−1.24 to 3.49)	1.24 (−3.78 to 6.53)	0.31 (−3.56 to 4.33)
SO_2	2.19 (−0.53 to 4.98)	−3.79 (−9.09 to 1.82)	0.07 (−4.73 to 5.13)
O_3	0.79 (−1.81 to 3.45)	0.01 (−4.72 to 4.97)	0.43 (−3.26 to 4.25)
65+			
NO_2	1.54 (−1.14 to 4.29)	2.46 (−3.15 to 8.40)	0.07 (−4.12 to 4.45)
SO_2	2.68 (−0.44 to 5.89)	3.11 (−3.77 to 10.49)	−0.41 (−5.61 to 5.08)
O_3	1.43 (−1.56 to 4.50)	3.04 (−2.47 to 8.86)	0.13 (−3.86 to 4.29)
Respiratory			
All ages			
NO_2	1.72 (−1.25 to 4.78)	0.19 (−5.36 to 6.07)	0.35 (−4.63 to 5.59)
SO_2	3.11 (−0.39 to 6.74)	−0.53 (−7.45 to 6.90)	1.79 (−4.81 to 8.84)
O_3	2.06 (−1.29 to 5.54)	1.33 (−4.25 to 7.24)	1.73 (−3.06 to 6.76)
65+			
NO_2	1.91 (−1.37 to 5.29)	−0.73 (−3.88 to 2.54)	0.29 (−4.77 to 5.62)
SO_2	3.69 (−0.18 to 7.72)	−1.85 (−5.18 to 1.60)	2.49 (−4.29 to 9.75)
O_3	2.08 (−1.59 to 5.89)	0.05 (−2.99 to 3.17)	2.58 (−2.35 to 7.76)
Accidental			
All ages			
NO_2	1.15 (−3.11 to 5.60)	2.83 (−5.53 to 11.93)	1.30 (−6.67 to 9.96)
SO_2	0.80 (−4.23 to 6.10)	4.01 (−5.19 to 14.12)	3.30 (−6.79 to 14.48)
O_3	2.07 (−2.70 to 7.07)	1.41 (−6.15 to 9.59)	0.19 (−7.19 to 8.16)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day.

Table B.10. Short-term Effects on Mortality Due to Air Pollution for Pre- and Post-Intervention Periods Using a Subset of Data in Which Gaseous Pollutants, Nickel, and Vanadium in PM Were Measured on the Same Day[a]

Disease / Age Group / Pollutant	Pre-Intervention (5-Year) ER (95% CI)	Pre-Intervention (2-Year) ER (95% CI)	Post-Intervention ER (95% CI)
All Natural Causes			
All ages			
NO_2	−0.24 (−0.85 to 0.37)	0.06 (−1.56 to 1.70)	1.31 (0.42 to 2.21)
SO_2	0.11 (−0.47 to 0.69)	1.20 (0.01 to 2.40)	0.47 (−0.38 to 1.33)
O_3	−0.95 (−1.88 to −0.01)	0.17 (−1.38 to 1.76)	0.52 (−0.02 to 1.06)
65+			
NO_2	−0.24 (−1.01 to 0.53)	−0.44 (−3.30 to 2.50)	1.18 (0.16 to 2.22)
SO_2	0.06 (−0.68 to 0.80)	1.78 (−0.39 to 4.00)	0.48 (−0.51 to 1.47)
O_3	−1.35 (−2.56 to −0.13)	1.04 (−1.91 to 4.07)	0.41 (−0.21 to 1.03)
Cardiovascular			
All ages			
NO_2	−0.80 (−1.70 to 0.10)	−2.29 (−5.58 to 1.11)	−0.02 (−1.56 to 1.55)
SO_2	0.30 (−0.65 to 1.26)	2.38 (−0.20 to 5.03)	0.32 (−1.18 to 1.84)
O_3	−0.48 (−1.92 to 0.99)	−0.28 (−3.69 to 3.25)	−0.62 (−1.53 to 0.30)
65+			
NO_2	−0.76 (−1.78 to 0.27)	0.98 (−2.58 to 4.67)	0.20 (−1.48 to 1.91)
SO_2	0.24 (−0.84 to 1.34)	2.28 (−0.30 to 4.92)	0.38 (−1.26 to 2.05)
O_3	−1.15 (−2.79 to 0.53)	0.22 (−3.27 to 3.84)	−0.91 (−1.99 to 0.18)
Respiratory			
All ages			
NO_2	0.18 (−0.97 to 1.34)	1.30 (−2.77 to 5.54)	1.29 (−0.78 to 3.39)
SO_2	−0.38 (−1.56 to 0.81)	3.12 (0.20 to 6.13)	0.19 (−1.80 to 2.21)
O_3	−1.01 (−2.76 to 0.78)	−0.27 (−4.23 to 3.86)	0.61 (−0.59 to 1.83)
65+			
NO_2	0.24 (−1.04 to 1.54)	0.78 (−1.31 to 2.90)	2.96 (0.77 to 5.18)
SO_2	−0.58 (−1.90 to 0.76)	1.12 (−0.41 to 2.68)	1.34 (−0.77 to 3.50)
O_3	−1.38 (−3.36 to 0.65)	0.86 (−1.16 to 2.92)	1.02 (−0.28 to 2.33)
Accidental			
All ages			
NO_2	0.07 (−1.73 to 1.90)	0.83 (−3.90 to 5.80)	0.37 (−2.73 to 3.58)
SO_2	0.58 (−1.08 to 2.26)	2.77 (−0.86 to 6.54)	1.45 (−1.57 to 4.57)
O_3	−1.04 (−3.77 to 1.77)	0.58 (−4.56 to 5.98)	−0.71 (−2.69 to 1.32)

[a] Data are expressed in % per 10-μg/m^3 unit change at lag 0–1 day.

Table B.11. Distribution of Daily Air Pollutant Concentrations, Meteorologic Measurements, Mortality Counts, and Socioeconomic Variables in Hong Kong, 1985–2005 (N = 4018 Days)

		Percentile				
	Mean ± SD	Min	25th	50th	75th	Max
Pollutant (μg/m^3)						
SO_2	24.8 ± 21.4	0.4	12.0	18.3	30.2	259.8
PM_{10}	56.7 ± 25.5	9.0	37.1	52.5	72.1	244.3
NO_2	61.0 ± 27.3	9.3	44.0	56.5	71.4	381.6
O_3	36.6 ± 24.0	0.0	18.7	30.7	50.0	222.0
Temperatures (°C)	23.3 ± 5.1	6.1	19.2	24.4	27.7	32.0
Humidity (%)	78.2 ± 10.3	27.0	74.0	79.0	85.0	98.0
Nonaccidental death (number)						
all ages	79.3 ± 16.1	22.0	68.0	78.0	89.0	153
Nonaccidental death rates (per 10^3)						
all ages	0.011 ± 0.002	0.004	0.010	0.011	0.013	0.025
Socioeconomic variables						
Smoking (%)	15.1 ± 1.4	12.4	14.3	14.9	15.7	18.1
Gross domestic product ($ U.S.)	20,083 ± 3,364	12,784	17,734	20,831	22,237	26,700
Unemployment (%)	3.6 ± 2.1	1.1	1.9	2.8	5.1	7.9

Table B.12. Estimated Coefficients, SEs, and t Values of the Core Model Adjusted for Trend, Seasonality, T, and RH with the Second-Order Autoregressive Term[a,b]

Variable	Coefficients	SE	t Value
Intercept	−1.655000	0.057990	−28.54
AR1 term	0.252400	0.011300	22.34
AR2 term	0.216500	0.011100	19.50
Trend	−0.000079	0.000001	−79.00
Cosine	0.027790	0.004613	6.02
Sine	0.041890	0.003251	12.89
Temperature	−0.001183	0.000687	−1.72
Humidity	−0.001246	0.000163	−7.64

[a] Dispersion parameter: 1.41; residual deviance: 10781 on 7660 degrees of freedom.

[b] RH indicates relative humidity; SE indicates standard error; T indicates temperature.

Table B.13. Percentage of Measurements of Chemical Species in PM_{10} Under the Detection Limit, for the Whole Study Period, by Station[a,b]

	Monitoring Station				
	CW	KC	KT	SSP	TW
Al	—	0.29	0.20	0.41	0.85
Br	—	3.22	0.82	0.41	0.85
C	—	—	—	—	—
Ca	0.22	—	0.41	—	0.28
Cl	—	—	—	—	—
Fe	—	0.58	0.80	0.41	0.85
Mg	—	—	0.20	—	—
Mn	4.07	7.49	4.61	3.26	8.45
Na	—	0.29	0.20	0.20	0.56
NH_4	—	—	—	—	—
Ni	8.99	7.20	6.21	7.33	9.01
NO_3	—	—	—	—	—
Pb	—	—	—	—	0.28
SO_4	—	—	—	—	—
V	9.21	8.93	8.23	9.18	10.42
Zn	1.30	0.29	0.61	0.21	1.41

[a] There were no measurements below the detection limit for gaseous pollutants and PM_{10}.

[b] Dash indicates no measurements below the detection limit.

Table B.14. Coefficient Estimates, $F(i_k - N_k, i_k)$, for Fits (with Adjustments *Before* the Regressions Against Pollutants) with 7 Intervals of Length $N_k = 3^k$ Days (k = 0–6) for SO_2 and PM_{10}

	$F(i_k - N_k, i_k)$	SE
SO_2 with Single Pollutant		
Day 0	0.00140	0.00011
Days 1–3	0.00029	0.00015
Days 4–12	−0.00033	0.00021
Days 13–39	−0.00117	0.00028
Days 40–120	0.00066	0.00034
Days 121–363	−0.00026	0.00029
Days 364–1092	0.00024	0.00031
SO_2 with Two Pollutants		
Day 0	0.00125	0.00012
Days 1–3	0.00004	0.00016
Days 4–12	−0.00038	0.00022
Days 13–39	−0.00097	0.00030
Days 40–120	0.00067	0.00034
Days 121–363	−0.00028	0.00029
Days 364–1092	0.00023	0.00032
PM_{10} with Single Pollutant		
Day 0	0.00071	0.00010
Days 1–3	0.00024	0.00012
Days 4–12	0.00006	0.00013
Days 13–39	−0.00054	0.00017
Days 40–120	−0.00012	0.00023
Days 121–363	0.00083	0.00030
Days 364–1092	−0.00089	0.00043
PM_{10} with Two Pollutants		
Day 0	0.00031	0.00010
Days 1–3	0.00038	0.00012
Days 4–12	0.00009	0.00013
Days 13–39	−0.00034	0.00018
Days 40–120	−0.00024	0.00024
Days 121–363	0.00076	0.00031
Days 364–1092	−0.00083	0.00045

APPENDICES AVAILABLE ON THE WEB

Appendix C. Facsimile of Hong Kong Immigration Department Death Record and Additional Data from the Current Study

Appendix D. Power of the Test for the Interaction of Air Pollution Effects on Mortality Between Pre- and Post-Intervention Periods

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Tai Hing Lam is the chair professor and head of the Department of Community Medicine at The University of Hong Kong. Lam graduated from the Faculty of Medicine of The University of Hong Kong in 1975. He obtained an M.Sc. in medical sociology and an M.Sc. in occupational medicine in 1980 and 1981, respectively, from the University of London. He obtained his M.D. by research from The University of Hong Kong in 1988. Lam is the director of the Public Health Research Centre. His research interests include occupational and environmental health, family planning and youth sexuality, adolescent health, epidemiology of cancer, infectious diseases, cardiovascular and respiratory diseases and their risk factors, molecular epidemiology, health services research with a major focus on tobacco, and other lifestyle-related diseases.

Sarah M. McGhee is a professor in the Department of Community Medicine, School of Public Health at The University of Hong Kong. After moving there from the Department of Public Health at the University of Glasgow in 1994, she formed the Health Services Research Group (HSRG), which brought together the department's activities in health services research, economics, and informatics. Her principal research interests are the application of epidemiologic and economic techniques to questions related to health care. She is also interested in the cost of such risk factors as smoking, passive smoke, and air pollution; the cost-effectiveness of screening for cervical cancer; and child and adult vaccination programs.

H. Ross Anderson qualified in medicine at the University of Melbourne. From 1966 to 1972, he worked in Papua New Guinea, where he investigated chronic lung disease and asthma and their relation to indoor air pollution and other factors. This was followed by two years at the United Kingdom Medical Research Council's Pneumoconiosis Unit in South Wales. He then obtained an M.Sc. in social medicine at the London School of Hygiene & Tropical Medicine. In 1976, he was appointed to St George's, University of London, where he became professor of epidemiology and public health in 1985. He now works part time at the MRC-HPA Centre for Environment and Health at King's College London. His main research is into the epidemiology of asthma and the health effects of air pollution. He is a member of the steering group of the International Study of Asthma and Allergies in Childhood, and a member of the U.K. Committee on the Medical Effects of Air Pollution.

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OTHER PUBLICATION RESULTING FROM THIS RESEARCH

Rable A, Thach TQ, Chau PYK, Wong C-M. 2011. How to determine life expectancy change of air pollution mortality: A time series study. Environ Health 10:25.

ABBREVIATIONS AND OTHER TERMS

ACS	American Cancer Society
Al	aluminum
ANOVA	analysis of variance
ASDR	age-specific death rate
Br^-	bromide
C	carbon
C&SD	Census & Statistics Department
Ca	calcium
Cl	chloride
CW	Central & West
df	degree of freedom
EPD	Environmental Protection Department (Hong Kong)
ER	excess risk
Fe	iron
HKU	The University of Hong Kong School of Public Health, Air Pollution and Health Research unit
HSD	Honestly Significant Difference (Tukey's)
ICD-9	*International Classification of Diseases,* 9th revision
IMMD	Immigration Department, Hong Kong Special Administrative Region
KC	Kwai Chung
KT	Kwun Tong
LE	life expectancy
LOESS	locally estimated scatterplot smoothing
Mg	magnesium
Mn	manganese
Na	sodium
NH_4^+	ammonium
Ni	nickel
NMMAPS	National Morbidity, Mortality, and Air Pollution Study
NO_2	nitrogen dioxide
NO_3	nitrate
NO_x	nitrous oxide
O_3	ozone
PACF	partial auto-correlation function
Pb	lead
PM	particulate matter
$PM_{2.5}$	particulate matter with an aerodynamic diameter $\leq$ 2.5 μm
PM_{10}	particulate matter with an aerodynamic diameter $\leq$ 10 μm
RR	relative risk
SMR	standardized mortality ratio
SO_2	sulfur dioxide
SO_4	sulfate
SSP	Shum Shui Po
TW	Tsuen Wan
V	vanadium
Zn	zinc

Research Report 170, *Impact of the 1990 Hong Kong Legislation for Restriction on Sulfur Content in Fuel*, C.-M. Wong et al.

INTRODUCTION

On July 1, 1990, the government in Hong Kong implemented a new restriction on sulfur in fuel, mandating a limit of 0.5% sulfur by weight. After the full impact of this regulation was realized, airborne sulfur dioxide (SO_2*) concentrations were reduced by 45% on average and by as much as 80% in some districts. However, other components of the pollutant mixture, including particle mass, measured as particulate matter ≤ 10 µm in aerodynamic diameter (PM_{10}), did not decline. The reductions in the SO_2 concentrations were estimated to have resulted in health improvements, including decreases in mortality rates and improvements in life expectancy (Hedley et al. 2002). The current study aimed to extend this earlier work in two ways: (1) by exploring the effects of the sulfur restriction on concentrations of specific components of the pollutant mixture and estimating the effect on daily mortality of changes in specific components; and (2) by developing and applying new statistical methods to estimate the effects of the sulfur restriction on life expectancy.

Dr. Wong's study was funded in 2006 under RFA 04-4, *Measuring the Health Impact of Actions Taken to Improve Air Quality*. The two objectives of this RFA were to fund studies to assess the health impact of regulatory and incentive-based actions at the local up to the national level in order to improve air quality, and to develop methods required for, and specifically suited to, conducting such research. The RFA was primarily intended to estimate the impact of actions taken in the United States. Proposals for studies of actions taken in other countries were to be considered only if the studies were relevant to the current U.S. conditions (i.e., if they were studies of interventions to reduce emissions from sources commonly found in North America at locations with comparable ambient air pollution levels). The RFA primarily sought studies of actions taken with the express purpose of improving air quality, such as the Hong Kong restriction on sulfur in fuel.

This Commentary is intended to aid the sponsors of HEI and the public by highlighting both the strengths and limitations of the study and by placing the Investigators' Report into scientific and regulatory perspective.

SCIENTIFIC BACKGROUND

The Hong Kong Special Administrative Region (HKSAR) is confronting air quality issues that are now, or soon will be, faced by other rapidly expanding urban centers in Asia. These issues include marked increases in emissions from local and regional air pollution sources and deteriorating air quality. Ambient air pollution levels have been monitored in Hong Kong for more than three decades, enabling researchers to assess trends in air quality and estimated health effects. Implementation in 1990 of a regulation reducing the sulfur content of fuel in power plants and for vehicle use provided a unique opportunity to assess the health impact of improvements in air quality, and several studies (Peters et al. 1996; Wong et al. 1998, 1999) examined the effects of the reduction on adverse health outcomes in children and adults. (For more information, see "Sidebar 1. Air Pollution in Hong Kong" in HEI Special Report 15 [HEI International Scientific Oversight Committee 2004], available at *www.healtheffects.org*.) In one well-known study, the team now led by Dr. Wong conducted analyses of the mortality effects associated with the sulfur reduction (Hedley et al. 2002). They reported marked reductions in SO_2 concentrations in the first year after the intervention and during the 5 years after the intervention, but no such changes were seen for PM_{10} or nitrogen dioxide (NO_2). Ozone (O_3) concentrations, on the other hand, increased significantly during the 5 years after the intervention. They reported substantial reductions in mortality from all natural causes and from cardiovascular and respiratory diseases over the 5 years after the

Dr. Chit-Ming Wong's 2-year study, "Impact of the 1990 Hong Kong Legislation for Restriction on Sulfur Content in Fuel," began in November 2006. Total expenditures were $196,077. The draft Investigators' Report from Dr. Wong and colleagues was received for review in July 2009. A revised report, received in September 2010, was accepted for publication in October 2010. 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.

intervention, including marked diminutions in the expected peak in mortality rates during the cool season in the first year after the intervention. The authors reported that a greater decline in mortality was noted in areas with a higher reduction in SO_2 concentrations during the first 2.5 years after the intervention than in areas with smaller reductions in SO_2. They calculated that the intervention had resulted in increases in life expectancy that averaged 20 days in women and 41 days in men (Hedley et al. 2002).

Although few in the scientific community questioned the general conclusion that the sulfur reduction had had beneficial effects on mortality in Hong Kong, there was some skepticism that the reductions in SO_2 concentrations per se were responsible for the reductions in mortality, and there was a continuing interest in identifying other constituents of the air pollution mixture, especially those that might be associated with, but perhaps not well represented by, PM_{10}. In fact, Hedley and colleagues in their 2002 publication already acknowledged that "the apparent benefits of the reduction in SO_2 could have been attributable to other combustion products that are not generated by low-sulphur fuels. Changes in concentration of SO_2 after the fuel regulation was introduced could simply be an indicator of other qualitative changes in fuel and products of combustion, with reduction in another unidentified agent that causes the health effects." Lippmann and colleagues (2006) had suggested that PM-associated transition metals, including nickel (Ni) and vanadium (V), emitted in the combustion of sulfur-rich residual fuel oil (bunker fuel) were associated with adverse cardiovascular effects in animal experiments and with daily mortality in humans in a New York state subset of the U.S. National Morbidity and Mortality Air Pollution Study (NMMAPS).

The results of Hedley and colleagues (2002) appeared to suggest that sufficiently long time-series studies might be able to provide estimates of the effects of air pollution exposure on life expectancy. Time-series studies estimate the effects of short-term exposure on daily rates of adverse health outcomes, such as mortality and admissions to hospital. Hundreds of time-series studies have now been reported from almost all regions of the world, and systematic reviews conducted by the World Health Organization and national governments have concluded that the evidence from these studies of short-term exposure to particulate air pollution and gaseous pollutants is consistent with a causal effect of exposure on daily mortality from cardiovascular and respiratory causes (HEI 2010a). However, time-series studies of mortality provide an estimate of the daily number of deaths-brought-forward but not of the number of years of life lost. In contrast, cohort studies include not only people whose deaths were advanced by recent exposure to air pollution, but also those who died from chronic disease caused by long-term exposure (McMichael et al. 1998; Künzli et al. 2001; COMEAP 2009), and therefore they provide a more comprehensive estimate of the effects on mortality. For this reason, their relative risks can be applied to population life tables to estimate the effects of air pollution on life span (Brunekreef 1997, 2007; COMEAP 2009).

Nonetheless, some have suggested the possibility of estimating effects on survival time from time-series data. They posit that the two designs (time-series and cohort) may be viewed, under some conditions, as estimating the same underlying exposure–disease relationships (Burnett et al. 2003; Rabl 2006).

The regulation reducing sulfur in fuels carried out in Hong Kong in 1990 represents a specific type of action that can be taken to improve air quality and to reduce the adverse health effects of air pollution exposure. The health effects of large, multifaceted, national-scale programs implemented over a period of years, such as the U.S. Clean Air Act, are difficult to study — in part because so many other factors that influence health (e.g., unemployment and health care practices) can change over the same interval (HEI Accountability Working Group 2003; HEI 2010b). However, some interventions may produce relatively rapid changes in air quality, the impact of which may be measurable soon after: for example, a ban on coal sales implemented over a short time frame in Dublin, Ireland, and the Hong Kong regulation reducing sulfur in fuels examined in the current study (Clancy et al. 2002; Hedley et al. 2002; van Erp and Cohen 2009). The relatively discrete step changes in air pollution levels, such as occurred in Hong Kong after the sulfur reduction, may reduce, but by no means eliminate, the possibility of confounding by other risk factors that change more slowly over time since the health response to the intervention can usually be assessed only over somewhat longer periods of time.

STUDY SUMMARY

STUDY OBJECTIVES

Dr. Wong and colleagues set out to explore the role that specific chemical constituents of particulate air pollution may have played in the effects on mortality of the 1990 Hong Kong restriction of sulfur in fuels, hereafter referred to as "the intervention." They also proposed to develop methods for estimating the impact on life expectancy of

an improvement in air quality after the imposition over a brief interval of a change in fuel quality, and to apply these methods in the context of the intervention.

They identified four specific objectives:

1. to evaluate the short-term effects on mortality due to changes in air quality after the intervention, specifically changes in levels of particle and gaseous pollutants and in particular chemical components or "species" of particulate air pollution;
2. to improve the current methods for assessing the health impact, in terms of the change in life expectancy, resulting from interventions taken to improve air quality, using linear regression models;
3. to develop a methodology using Poisson regression specifically suited to quantify the benefits of the intervention in terms of years of life gained; and
4. to determine any relation between short-term and long-term health benefits due to an improvement in air quality.

METHODS

Sources and Compilation of Data

Health Data The investigators obtained counts of daily deaths for the Hong Kong population between January 1, 1985, and June 30, 1995, from the Immigration Department in Hong Kong, a period that extends from 5 years before to 5 years after the imposition of the sulfur reduction. Specific causes of mortality were coded according to the *International Classification of Diseases,* 9th revision (ICD-9), and comprised deaths from all natural causes (ICD-9 001–799), and the subcategories cardiovascular diseases (ICD-9 390–459), respiratory diseases (ICD-9 460–519), and accidental causes (ICD-9 800–999). For each cause, the investigators extracted population-based daily counts for all ages; for all natural causes, cardiovascular diseases, and respiratory diseases, daily death counts for those 65 years or older (65+) were also extracted.

Air Pollution and Meteorology The investigators obtained measured ambient concentrations of air pollutants from the Hong Kong Environmental Protection Department from a network of 13 stations that operated in Hong Kong over the 10-year period of the study. However, they included data from only the 5 monitoring stations that reported estimates for the entire period relevant to the evaluation of the intervention, including the 5 years after the intervention. Two stations were located in residential and commercial areas, in which vehicles were the major source of emissions, and three stations were located in industrial areas in which commercial vehicles, manufacturing plants, and service industries were the major sources of emissions.

Daily average concentrations of the gaseous pollutants NO_2, SO_2, and O_3 were derived from hourly measurements (throughout the 24-hour day for NO_2 and SO_2 and between 10:00–18:00 for O_3) for all days with at least 75% complete hourly data. The Hong Kong average pollutant-specific concentration was estimated as the arithmetic mean of the daily concentrations from the 5 monitors. Cumulated concentrations for multiple days (e.g., the 2 days before death) were calculated as the simple averages of concentrations over the period of accumulation.

Data on PM_{10} and associated chemical species were measured on every third and sixth day, respectively. Concentrations of specific chemical species were estimated by chemical analysis of the PM_{10} collected in filters using the gravimetric method at monitors with at least 75% complete data. Of 28 chemical species measured at any time, 16 (about 57%) met the inclusion criteria. These comprised estimates of the following chemical species: aluminum (Al), bromide (Br^-), carbon (C), calcium (Ca), chloride (Cl), iron (Fe), magnesium (Mg), manganese (Mn), sodium (Na), ammonium (NH_4), Ni, nitrate (NO_3), lead (Pb), sulfate (SO_4), V, and zinc (Zn). Values below the detection limit were coded as 0, and the rate of nondetectable values ranged from 0% to 10%. Concentrations of PM chemical species were aggregated over the 5 stations to represent the exposure for the whole territory of Hong Kong.

The investigators obtained daily mean temperature (°C) and relative humidity (%) data from the Hong Kong Observatory.

Statistical Analyses

Estimates of Changes in Air Pollution Levels The investigators compared mean levels of pollutants between pre- and post-intervention periods using independent *t* tests to assess mean differences in concentrations and using analysis of variance to estimate the mean differences between the average concentration in the pre-intervention period and each of the 5 years in the post-intervention period, with adjustments for multiple comparisons. These methods were also applied to assess the mean differences among sequentially ordered years in the post-intervention period.

Estimates of Pollutant-Specific Effects The investigators used Poisson regression methods to estimate the effects of short-term exposure to air pollution on daily mortality rates for all natural causes and for cardiovascular and

respiratory causes, at all ages and in the 65-years-or-older age group. They used natural spline smoothers to control for potential confounding of the estimated pollution effects due to long-term trends and seasonal variation in daily mortality rates, and specific covariates to control for potential confounding due to temperature and relative humidity. Alternative models were evaluated using the Akaike Information Criterion, and by visually examining the regression residuals from alternative model plots for any evidence of nonrandom patterns.

These models were used to estimate the effects of gaseous pollutants, PM_{10}, and the chemical species Al, Fe, Mn, Ni, V, Pb, and Zn for the periods 5 years before and 5 years after the intervention, as well as in the 10-year period pre- and post-intervention. They also conducted sensitivity analyses in which the pre-intervention time periods were shortened and ran analyses in which indicator variables were used to represent the intervention.

All effects were reported as excess risks (ERs), defined as the percentage change in mortality per 10-μg/m^3 and 10-ng/m^3 increase in the concentrations of four criteria pollutants and PM chemical species, respectively. ERs were estimated for exposure cumulated over the day of death and the preceding day (lag 0–1) and for exposure to chemical species on the day of death (lag 0).

The investigators estimated the change in the magnitude of the ER due to short-term exposure to air pollution before and after the intervention using an indicator variable, p (I_p), to separate pre- and post-intervention periods. They then added the main effect of each air pollutant (AP_i), the time indicator, and an interaction (product) term expressing the interaction between the air pollutant and the indicator into the Poisson regression model:

$$\log(Y_i) = C_i + I_p + AP_i + I_p \times AP_i \qquad \text{for } i = 1, 2, 3 \ldots$$

The coefficient for AP_i estimates the ER of mortality due to the air pollutant before the intervention, and the coefficient estimate of $I_p \times AP_i$ in the interaction model estimates the change in ER after the implementation of the intervention.

Estimates of Effects on Life Expectancy The investigators developed two methods to estimate the effect of the intervention on life expectancy, which differ with regard to the outcome variable: in the first method, this was the daily age-standardized mortality rate, and in the second, the daily mortality count.

- The first method used linear regression models to estimate the age-standardized mortality rate on a given day, $D(j)$ at day j, divided by a reference D_{ref} with adjustments for temperature and relative humidity. The investigators also adjusted for potential confounding due to seasonal variation using a standardized seasonal mortality rate profile (called "deseasonalization"). They adjusted for potential confounding of the estimated pollution effects due to long-term trends by calculating a reference mortality rate $D_{ref}(j)$ as a moving average of the corrected and deseasonalized $D(j)$ over the observation window. They then regressed the outcome variable $D(j)/D_{ref}$ on an entire exposure sequence $\{c(i)\}$ with lags up to 4 years in order to obtain an impact coefficient $f(i)$ from the regression model, as shown here:

$$\frac{\Delta D(j)}{D_{ref}} = \sum_{i=0}^{i_{max}} f(i)\, c(j - i)$$

The investigators calculated the change in life expectancy (LE) for a change of units (Δc) in the concentration of pollutants on T_{day} — representing the short interval (i.e., a day) — using the following equation (ΔL_{pop} = average loss in life expectancy of an entire population):

$$\Delta L_{pop} = -\Delta c\, T_{day} \sum_{j=0}^{\infty} \sum_{i=0}^{j} f(i)$$

- The second approach used Poisson regression models with daily mortality counts as the outcome variables. In this approach, the investigators fit a distributed-lag model for exposure to previous days of up to 4 years in order to obtain the cumulative lag effect $\Sigma\hat{\beta}_i$. They then fit a linear regression model of log (LE*/LE) = γ(SMR − 1) + α to estimate the parameter γ by $\hat{\gamma}$, where LE* and LE are life expectancy for an exposed and an unexposed population, respectively, and SMR represents the standardized mortality ratio. The life expectancy change per increase in concentration (Δc) is

$$\text{LE}\{\exp[\hat{\gamma}\Delta c(\textstyle\sum\hat{\beta}_i)] - 1\} \qquad \text{for } i = 1 \text{ to } n.$$

Both models were used to evaluate the relation between outcome variables and daily air pollution concentrations in the current day back to all previous days in the past 3 to 4 years.

KEY FINDINGS

MORTALITY

The investigators included 275,254 deaths between 1985 and 1995 in the study, after excluding records that did not meet quality control specifications. They reported

a very high level of agreement between the deaths included in their analytic file and the source databases (see Tables 1 and 2 in the Investigators' Report).

The investigators reported that the average daily number of deaths from all natural causes for all ages was 69 (SD, 13) in the 5-year pre-intervention period and 76 (SD, 14) in the 5-year post-intervention period. Cardiovascular mortality, respiratory mortality, and deaths due to accidents comprised approximately 30%, 18%, and 6% of total mortality, respectively, in both periods (see Table 3 in the Investigators' Report).

AIR POLLUTION

The investigators reported decreases in NO_2 and SO_2 concentrations between pre-intervention and post-intervention periods, which were particularly pronounced in the more heavily polluted industrial areas (see Table 4 and Figures 5 and 6, respectively, of the Investigators' Report). They reported no consistent changes in PM_{10} concentrations after the intervention (Figure 7 of the Investigators' Report). Concentrations of O_3 appeared to increase after the intervention, though this pattern was not consistent across all monitoring sites (see Table 6 of the Investigators' Report).

The investigators reported decreases in each of the seven PM_{10} chemical species: Al, Fe, Mn, Ni, V, Pb, and Zn between pre- and post-intervention periods (see Tables 7–10 and Figures 8–14 of the Investigators' Report). Of these, the reductions in Ni and V were the most consistent and were statistically significant.

EFFECTS OF SHORT-TERM EXPOSURE AND THE REDUCTION IN THE SULFUR CONTENT OF FUEL ON DAILY MORTALITY

The investigators present analyses of the relation between short-term exposure to air pollution and daily mortality using data for the 10-year period from 1985 to 1995. Results were reported for the entire 10-year risk period and for intervals defined in relation to the sulfur reduction. Sensitivity analyses were reported based on a 7-year period from 1988 to 1995, which comprised 2 years before and 5 years after the sulfur reduction.

Over the Entire Risk Period

In the time-series analysis of short-term mortality and pollution in Hong Kong over the entire 10-year period starting 5 years before the ban and ending 5 years after the ban, the investigators reported increased ERs for mortality due to all natural causes for both SO_2 and NO_2, and for mortality from cardiovascular causes for SO_2 and respiratory causes for NO_2, based on cumulative exposure on the day of and the day before death (lag 0–1). O_3 was also associated with an increase in deaths due to all natural causes and respiratory disease (see Table 11 in the Investigators' Report). Neither PM_{10} nor most chemical species were consistently or statistically associated with increased ER. However, the investigators reported that both Ni and V were associated with increased ER from respiratory causes, especially in those older than 65 years.

Pre- and Post-Intervention

The investigators reported that only NO_2 was associated with increased daily mortality in the pre-intervention period. This association was particularly pronounced for respiratory causes of death in those older than 65 years. Post-intervention, they reported that the gaseous pollutants were, for the most part, associated with an ER of mortality from all natural causes and from cardiovascular and respiratory diseases (see Table 13 in the Investigators' Report).

The investigators reported no statistically significant associations between PM_{10} and mortality in either the pre- or post-intervention periods (Table 13). They reported that there was little evidence of an association in the pre-intervention period between mortality and exposure to most of the chemical species. Ni and V, however, were associated with mortality from both all natural causes and respiratory disease, especially in those older than 65 years.

In the post-intervention period, the investigators reported that most ERs for the individual chemical species were lower than in the pre-intervention period, though the ER of Ni for mortality due to all natural causes was higher in the 5-year pre-intervention period for both age groups (see Table 14 in the Investigators' Report).

After the Intervention

The investigators reported that based on analyses using data for the entire 10-year risk period, most gaseous pollutants were associated with increases in the ER of mortality after the intervention (see Table 15 in the Investigators' Report). The most pronounced increases were associated with NO_2 and SO_2 on cardiovascular mortality and for NO_2 and O_3 on respiratory mortality (Table 15).

The investigators reported that neither PM_{10} nor most chemical species were consistently associated with changes in ER, although Zn and Mn showed some evidence of associations with cardiovascular mortality (see Table 16 in the Investigators' Report). However, both Ni and V were associated with a decline in ER of mortality (Table 16), a finding that was replicated in a sensitivity analysis using a 7-year assessment period (see Table B.3 in Appendix B in the Investigators' Report).

EFFECTS ON LIFE EXPECTANCY

The investigators reported estimates of effects on life expectancy due to air pollution exposure using the two methods described earlier over a 21-year period from 1985 through 2005, a period which included the sulfur reduction intervention.

Linear Regression Approach

Using the linear regression approach with a window of 1095 days (3 years), the investigators reported reductions in life expectancy associated with a 10-μg/m^3 increase in pollutant concentrations that ranged from approximately 19 to 31 days for PM_{10} and from 13 to 20 days for SO_2, depending on the method used to control for the effects of temperature and relative humidity. They noted that these estimates were smaller than had been reported by other investigators (Elliott et al. 2007; Pope et al. 2009) based on evidence from cohort studies in Western populations, perhaps due to the investigators' use of a shorter time window.

Poisson Regression Approach

Using the Poisson regression approach with a window of 1461 days (4 years), the investigators reported average reductions in life expectancy associated with a 10-μg/m^3 increase in pollutant concentrations of 69 and 133 days for PM_{10} and SO_2, respectively. They reported that the estimates were quite sensitive to variations in the analysis model, specifically in terms of the model definition, exposure windows, constraint of the lag effect pattern, and adjustment for smoking prevalence or socioeconomic status.

Effect of the Intervention on Life Expectancy

The investigators reported that although some analyses suggested a relatively rapid but unsustained increase in life expectancy associated with the intervention, they were unable to reliably estimate the effect of the intervention on life expectancy using either approach, because they could not be confident of adequately adjusting for the effects of season on mortality or for the effects of long-term trends in mortality rates that had occurred in the population of Hong Kong.

HEI EVALUATION AND INTERPRETATION OF THE RESULTS

The reduction in the sulfur content of fuel in Hong Kong, implemented over a relatively brief period in 1990, and the health effects associated with it have been the subject of several epidemiologic studies, including a report by the current authors (Hedley et al. 2002), which reported reductions in mortality rates associated with reductions in ambient concentrations of SO_2, but not PM_{10}, and offered estimates of increases in life expectancy associated with the intervention. The current study by Wong and colleagues explored the effects of the reduction in the sulfur content of fuel on mortality in more detail, taking advantage again of the marked step change in a major source of air pollution. They also had the added advantages of an extended 10-year period of observation of both air pollution and mortality in Hong Kong and of additional, detailed measurements of specific chemical constituents of PM_{10} and gaseous pollutants, whose effects might underlie changes in mortality associated with the intervention. The extended period of observation also afforded Wong and colleagues the opportunity to develop and apply innovative methods to estimate the effects of reduction in the sulfur content of fuel on life expectancy using daily mortality rates, a controversial approach with potential applications to similar interventions. Dr. Wong's team was particularly well suited to carry out this ambitious research agenda, building on their extensive prior work on air quality and health in Hong Kong.

EFFECTS OF SHORT-TERM EXPOSURE AND THE REDUCTION IN THE SULFUR CONTENT OF FUEL ON DAILY MORTALITY

The investigators applied conventional analytic methods for the analysis of daily time-series data and reported associations over a 10-year period from 1985 to 1995 with all-natural-cause and cause-specific daily mortality for the gaseous pollutants (SO_2, NO_2, and O_3), PM_{10}, and various PM-associated chemical species, all derived from the combustion of fossil fuels. They report estimates for the effects of the gaseous pollutants and PM_{10} that are broadly consistent, both in direction and magnitude if not statistical precision, with many previous studies (HEI 2010a; HEI Public Health and Air Pollution in Asia 2010). The analyses of PM-associated chemical species indicated particularly strong associations between mortality and exposure to the specific chemical components Ni and V, constituents derived mainly from the combustion of bunker fuels with high sulfur content used in marine shipping (see Table 12 of in the Investigators' Report). However, the current study has several important weaknesses that limit its contribution to knowledge about the effects of this much-studied intervention.

Although the investigators observed reductions in PM-associated Ni and V concentrations associated with the intervention and reported adverse effects of short-term exposure to both of these components, they were unable to reliably link changes in their concentrations associated

with the intervention to changes in the effects of short-term exposure in the pre- and post-intervention periods. The investigators acknowledge this and attribute their inability to demonstrate any effects of the intervention per se to the fact that Ni and V concentrations were measured only on every sixth day. But other weaknesses may also be responsible.

The comparison of estimated pollution effects between models (in particular between pre- and post-intervention models) was complicated by different specifications of model components for time trends and temperature (see Table C.1 in Appendix C, available only on the Web at *www.healtheffects.org*). It is not clear how sensitive the results were to these alternative specifications, but the situation suggests the possibility of bias in comparisons between effect estimates for these two periods. Moreover, the monitoring data reveal considerable spatial variation in concentrations of the various pollutants across Hong Kong and also considerable spatial differences in the temporal variation of air pollution levels among them. These uncertainties suggest that only tentative conclusions can be drawn from these time-series analyses.

In addition, the investigators were unable to disentangle the effects of individual pollutants, both gases and components in the particle fraction, on mortality over the 10-year period of the study. After adjustment for levels of Ni and V, the associations with SO_2 that had provided the strongest evidence for an effect of the intervention became statistically unstable. These analyses make clear that the effects of SO_2, Ni, and V could not be separated in the current study and likely cannot be separated using the current set of data and analytic methods given the correlations among these pollutants (see Appendix Tables B.5 and B.6 in Appendix B in this volume). Given the common combustion sources of these pollutants (e.g., sulfur-rich bunker fuel used in marine shipping), this is perhaps not surprising. The investigators conclude that they "cannot exclude the possibility that decreases in their concentrations were responsible for some of the observed health benefits due to the intervention." However, based on these results, we cannot confidently attribute such beneficial effects on mortality as may have occurred due to the intervention to any specific component of the air pollution mixture, be it SO_2 or PM-associated metals.

EFFECTS OF LONG-TERM EXPOSURE AND THE REDUCTION IN THE SULFUR CONTENT OF FUEL ON LIFE EXPECTANCY

The investigators reported effects on life expectancy of long-term exposure in the Hong Kong population using new approaches based on long daily time series of air pollution and mortality. They have shown, as suggested earlier by Burnett and colleagues (2003) and by Rabl (2006), that, in principle at least, if the coefficients of an arbitrarily long distributed-lag model are known, then the impact of exposure on life expectancy can be deduced. The problem is in the estimation of the parameters of the longer lag, and the critical question is whether longer lag effects of pollution can be reliably disentangled from confounders in studies with only temporal variation in exposure. Many factors contribute to longer-term (e.g., inter-annual) variation in mortality, and some may be associated with pollution and hence confound the estimate of the pollution effect. Indeed, the investigators concede that they were unable to effectively control for these factors, and they attribute their inability to identify a marginal effect of the intervention to this limitation. The Review Committee agrees but would add that this also calls into question the estimates they reported of associations of air pollution with reduced life expectancy over the entire 10-year period. These results also seem to qualify the strength of the previous conclusions of Hedley and colleagues (2002) with regard to the beneficial effects of the intervention on life expectancy, in as much as the Hedley conclusion depends on parameters similar to those considered inestimable in the current study.

The investigators conclude that "[w]ith complete daily air pollution and mortality data over a long period of time, time-series analysis methods can be applied to assess the short- and long-term effects of air pollution, in terms of changes in life expectancy" and that "[t]his methodology may prove to be an important development in the formulation of environmental public health policies regarding the effects of air pollution in terms of life expectancy." These conclusions are, at best, premature. The results of Wong and colleagues appear to offer little hope for estimating the effects of pollution at very long lags from single time-series studies with acceptable precision. Perhaps with several time series — for example, a "control" series from an otherwise comparable, pollution-stable population — the prospects would improve, but even then it is unlikely to be easy or straightforward. At this point, cohort studies still provide the most reliable evidence for estimating effects on life expectancy, underscoring the importance of conducting cohort studies in regions other than the United States and Western Europe.

SUMMARY AND CONCLUSIONS

The Hong Kong sulfur fuel reduction is one of the best known examples of an action taken to improve air quality in the interest of public health and is widely viewed as

providing evidence for the potential of such actions to produce health benefits (HEI International Scientific Oversight Committee 2004; WHO 2006; van Erp and Cohen 2009). In this study, Wong and colleagues revisited this important intervention and attempted to extend scientific knowledge about its effects in two important ways: by exploring the effects of the sulfur restriction on concentrations of specific components of the pollutant mixture and estimating the effect on daily mortality of changes in specific components, and by developing and applying new statistical methods to estimate the effects of the sulfur restriction on life expectancy. They met with, at best, limited success.

Wong and colleagues documented that the intervention was associated with reductions in a number of potentially health-damaging pollutants, including, as they hypothesized, PM-associated metals, particularly Ni and V. They also observed an increased risk of mortality associated with short-term exposure to these PM-associated metals, corroborating earlier results by Lippmann and colleagues and adding incrementally to current knowledge on the health effects of PM-associated chemical species. Dominici and colleagues (2007) also reported an increased risk of daily mortality associated with short-term exposure to Ni and V in the large U.S. NMMAPS study, though the strength of the estimated effects varied considerably across the United States. The strongest effects were in New York, as also reported by Lippmann and colleagues (2006), where concentrations of Ni and V are the greatest. That said, Ni and V are but two of a number of air pollution constituents hypothesized to contribute to increased mortality. Others, such as black carbon, were not measured in Hong Kong (Janssen et al. 2011). In any case, Wong and colleagues did not succeed in linking the intervention-related reductions in pollution to changes in mortality, and we cannot confidently attribute any beneficial effects of the intervention on mortality to any specific component of the air pollution mixture measured in this study.

Wong and colleagues did conduct the first rigorous effort to estimate from daily time-series data the effects of long-term exposure on life expectancy. The theoretical basis for such estimates had been explored by Burnett and colleagues (2003) and by Rabl (2006), Dr. Wong's collaborator, and the long-term time-series data associated with the Hong Kong intervention seemed to present an ideal opportunity for practical application. Unfortunately, because of the inability to control for the effects of potential confounding factors correlated over the long term with air pollution, the results appear to offer little hope for estimating the effects on life expectancy from daily time-series data for use in scientific or policy applications.

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