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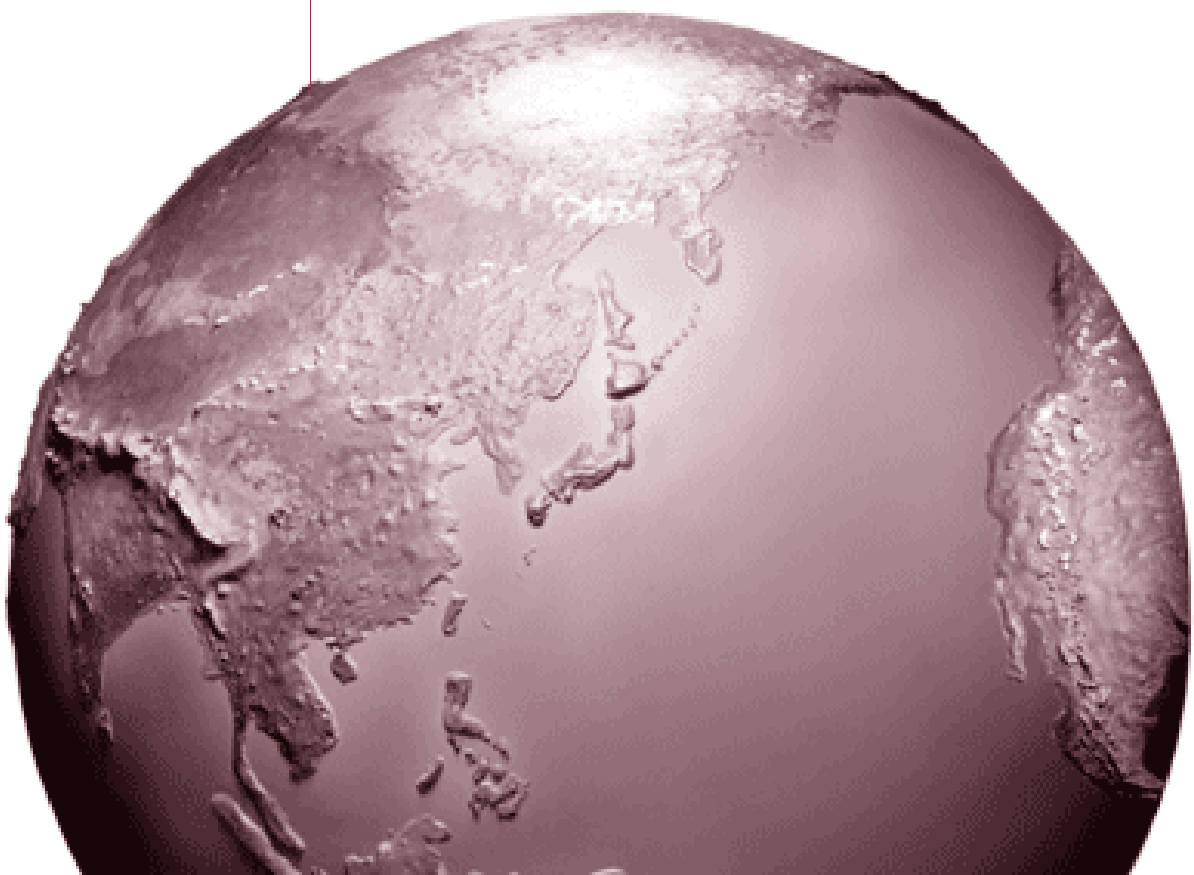
Outdoor Air Pollution and Health in the Developing Countries of Asia: A Comprehensive Review

HEI International Scientific Oversight Committee



Outdoor Air Pollution and Health in the Developing Countries of Asia: A Comprehensive Review

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Special Report 18
Health Effects Institute
Boston, Massachusetts

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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 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 certain research programs. The Public Health and Air Pollution in Asia (PAPA) Program was initiated by the Health Effects Institute in part to support the Clean Air Initiative for Asian Cities (CAI-Asia), a partnership of the Asian Development Bank and the World Bank to inform regional decisions about improving air quality in Asia. Additional funding was obtained from the U.S. Agency for International Development and the William and Flora Hewlett Foundation.

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.

CONTENTS

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This HEI Special Report was prepared on behalf of the International Scientific Oversight Committee (ISOC) of the Public Health and Air Pollution in Asia (PAPA) program, which oversaw its planning and production and reviewed its content and conclusions. The ISOC thanks the contributing authors who drafted the report and the eight peer reviewers who commented on several drafts. The ISOC also thanks Davida Schiff (HEI 2006–2008 Research Assistant, currently a Boston University Medical School student) for her early involvement in searching databases.

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EXECUTIVE SUMMARY

Outdoor Air Pollution and Health in the Developing Countries of Asia: A Comprehensive Review

EXECUTIVE SUMMARY

Asia is undergoing economic development at a rapid rate, resulting in levels of urban air pollution in many cities that rival the levels that existed in Europe and North America in the first decades of the 20th century. This development is also transforming the demographic and epidemiologic characteristics of the population in ways that are likely to affect its vulnerability to air pollution. Nearly two thirds of the estimated 800,000 deaths and 4.6 million lost years of healthy life worldwide caused by exposure to urban air pollution in 2000 occurred in the developing countries of Asia (also referred to as "developing Asia"; World Health Organization [WHO*] 2002). In rural areas and urban slums, indoor air pollution from the burning of solid fuels confers its own large burden of disease and contributes to increased outdoor concentrations of pollutants in some locales. Developing Asia's poorest populations are also susceptible to the unexpected effects of climate change, including possibly substantial health effects.

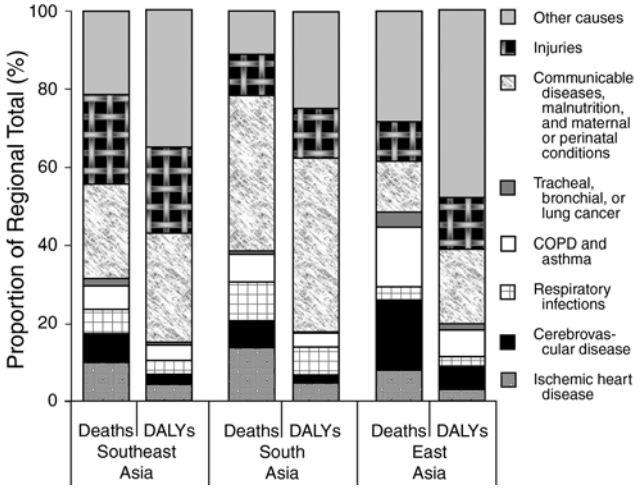
Effective public policy responses to the public health challenges posed by air pollution in developing Asia require high-quality scientific evidence on the health effects of air pollution in the region. Epidemiologic studies are among the most important and critical components of the required evidence; together with toxicologic and clinical studies, they provide estimates of the quantitative relation between exposure and disease. These estimates both demonstrate the existence of a public health hazard and allow its magnitude to be estimated. Owing to limitations of the available epidemiologic studies in Asia, estimates from assessments of the health impact of air pollution in Asian populations (e.g., the estimate from the WHO cited above), rely in large part on extrapolation, with considerable uncertainty, of the results of Western studies to Asian populations (Cohen et al. 2004; HEI International Scientific Oversight Committee [ISOC] 2004).

HEI initiated the Public Health and Air Pollution in Asia (PAPA) program in 2002 to reduce uncertainties about the health effects of exposure to air pollution in the cities of developing Asia. The first major publication of the PAPA program was Special Report 15, *Health Effects of Outdoor Air Pollution in Developing Countries of Asia: A Literature Review* (HEI ISOC 2004). That report was also the first comprehensive review of the peer-reviewed Asian literature on the health effects of air pollution, a literature that at that time (2004) comprised over 100 studies in nine countries. On the basis of Special Report 15, the PAPA program initiated a coordinated set of analyses of air pollution and daily mortality in four selected Asian cities (Hong Kong, Bangkok, Wuhan, and Shanghai). These studies have now been completed (HEI Public Health and Air Pollution in Asia Program 2010), as have additional studies in India and Vietnam (Balakrishnan et al. 2010; Rajarathnam et al. 2010; Collaborative Working Group on Air Pollution, Poverty, and Health in Ho Chi Minh City 2009).

The current report is the second PAPA literature review, *Outdoor Air Pollution and Health in Developing Countries of Asia: A Comprehensive Review* (HEI Special Report 18). It begins with a broad overview of the status of and trends in air pollution sources, emissions, concentrations, and exposures in the developing countries of Asia, as well as factors related to urban development, population health, and public policy that set the context for the health effects of air pollution. Next, the review describes the current scope of the Asian literature on the health effects of outdoor air pollution, enumerating and classifying more than 400 studies identified through 2007 via HEI's Web-based Public Health and Air Pollution in Asia — Science Access on the Net (PAPA–SAN) literature survey (HEI 2006). In addition, a systematic and quantitative assessment (conducted using St, George's Air

This document was made possible, in part, through support provided by the United States Agency for International Development (USAID) and the William and Flora Hewlett Foundation. The opinions expressed herein do not necessarily reflect the views of USAID or any other sponsors of HEI.

* A list of abbreviations and other terms appears at the end of the summary.



Executive Summary Figure 1. Deaths and disability-adjusted life-years (DALYs) in Asia in 2004, by region and cause. "Southeast Asia" corresponds to WHO Southeast Asian Region B; "South Asia" to WHO Southeast Asian Region D; and "East Asia" to WHO Western Pacific Region B. [Data compiled from WHO 2008.]

Pollution Epidemiology Database [APED]) of 82 time-series studies that estimate the effect of short-term exposure to air pollution on daily mortality and hospital admissions for cardiovascular and respiratory disease — four times the number of studies analyzed for Special Report 15. The studies covered in the current review include the coordinated studies of air pollution and daily mortality in four Asian cities conducted as part of HEI's PAPA research program, as well as a first-ever critical and qualitative analysis of Asian studies of long-term exposure to air pollution and chronic respiratory disease, lung cancer, and adverse reproductive outcomes. The review concludes with a discussion that places the Asian health effects studies in the context of the worldwide literature, identifies gaps in knowledge, and recommends approaches by which to address them.

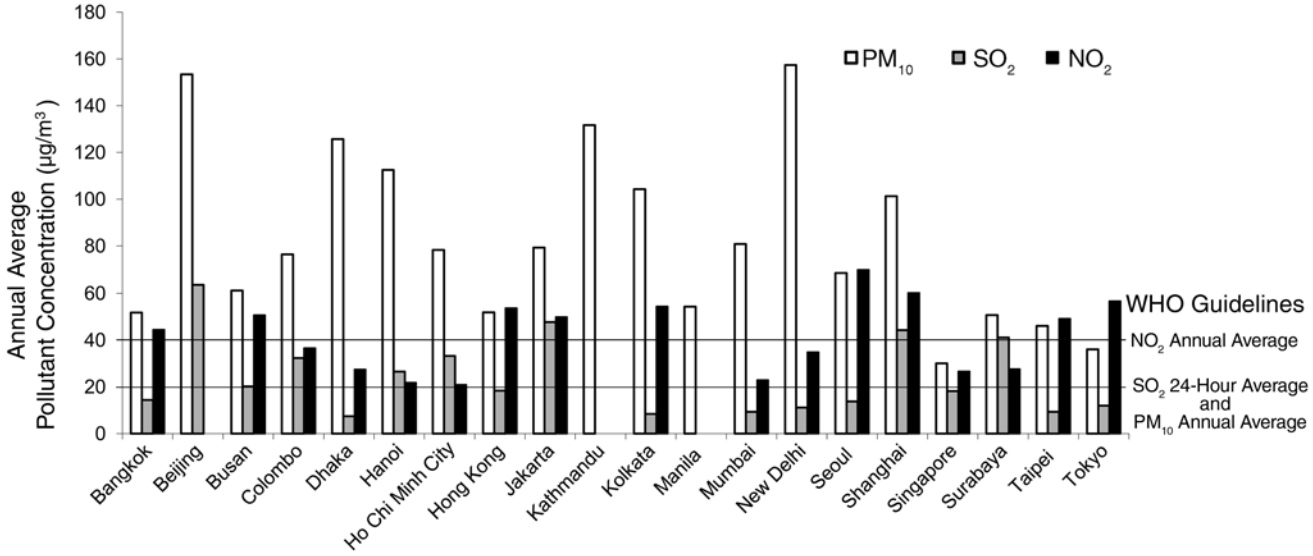
THE ASIAN LITERATURE IN CONTEXT

Development, Air Pollution, and Population Health

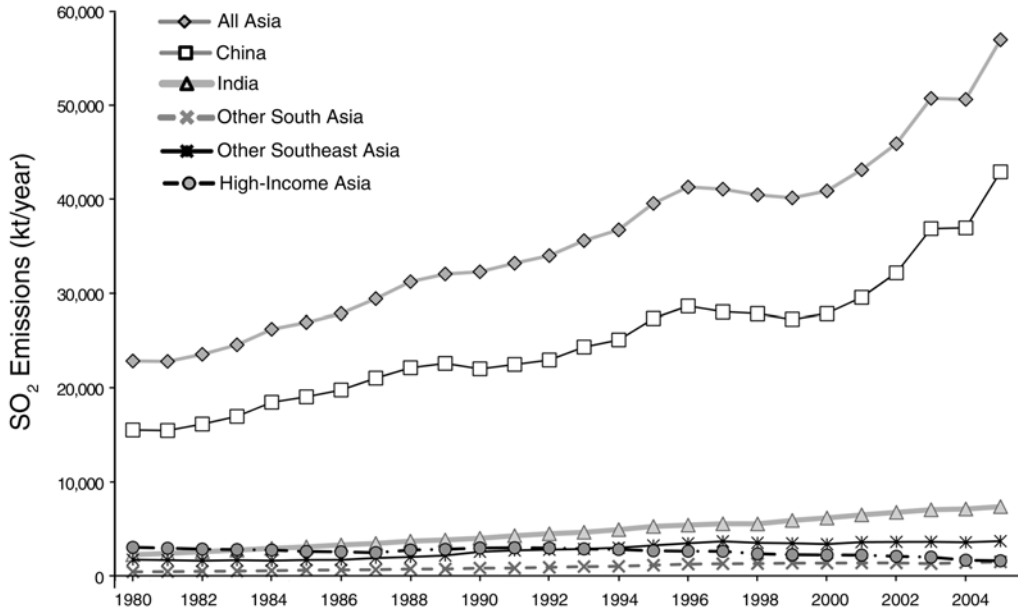
This review evaluates the evidence of health effects of outdoor air pollution in developing Asia in the context of ongoing changes in both air quality and population health. The nature of the health risks associated with the natural and built environments changes as economic development occurs. Economic development and attendant urbanization has been, and continues to be, based in large part on the increased combustion of fossil fuels. This pattern of development has led, in some countries, to impressive reductions in poverty levels and increased life expectancy. Economic development and poverty reduction have also led to gradual decreases in environmental risks at the household level, such as indoor air pollution from the burning of solid fuels and poor water quality, although the burden of disease associated with these exposures in young children and women remains substantial. There have also been commendable improvements in urban air quality in many parts of Asia. At the same time, the size of the exposed population that may be vulnerable to air pollution is increasing, as is evidenced by a large and growing burden of disease from chronic noncommunicable diseases — such as ischemic heart disease (IHD), cerebrovascular disease, chronic obstructive pulmonary disease (COPD), and cancer. The increased population size is, in part, owing to larger numbers of people living to older ages and to the increased prevalence of tobacco smoking, higher rates of obesity, and changes in dietary patterns (Figure 1).

Air pollution has become a major policy issue in many parts of Asia and has prompted actions to improve air quality. As a result, there have been improvements in air quality across the region, even in the face of increasing fossil-fuel consumption. These improvements, however, have not occurred in all highly populated areas, and some areas have in fact been experiencing deteriorating air quality. In general, air pollution concentrations in Asian cities greatly exceed current WHO health-based air quality guidelines and many current national standards (Figure 2). In a large number of studies worldwide, air pollution has been found to adversely affect people with chronic cardiovascular and respiratory diseases, and it may also contribute to the development of those diseases in otherwise healthy people. Thus, even as air quality has improved in some locations, there remains an important adverse impact on public health, which may grow as populations age and rates of chronic disease and urbanization increase.

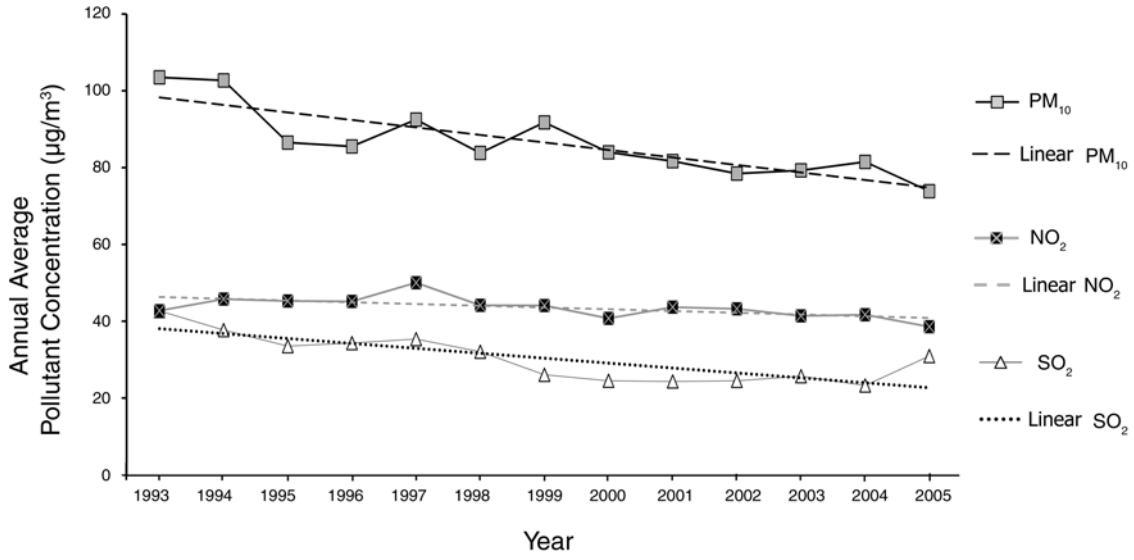
Air quality in Asia reflects complex and evolving relations between increased energy consumption for transport and power generation and measures being taken to improve air quality. Overall, estimates of pollution emissions as well as measured and estimated ambient concentrations indicate that air quality is improving throughout much of urban Asia (Figures 3 and 4). Trends in air quality have largely shown improvement during periods of dramatically increased energy use in Asia, a testament to the impact of effective air



Executive Summary Figure 2. Five-year (2000–2004) average PM_{10}, SO_2, and NO_2 concentrations in selected Asian cities. Standards are from WHO Air Quality Guidelines, 2005 Global Update (WHO 2006a): PM_{10} annual average 20 µg/m^3; SO_2 24-hr average 20 µg/m^3; NO_2 annual average 40 µg/m^3. PM_{10} is particulate matter 10 µm or smaller in aerodynamic diameter; SO_2 is sulfur dioxide; NO_2 is nitrogen dioxide. [Reprinted with permission from Clean Air Initiative for Asian Cities [CAI-Asia] Center (*www.cleanairnet.org/caiasia*; accessed January 2008).]



Executive Summary Figure 3. SO_2 emissions in Asia, 1980–2005. Data are given in kilotons per year. *China* comprises mainland China, Hong Kong, and Taipei,China. *Other South Asia* comprises Afghanistan, Bangladesh, Bhutan, Maldives, Nepal, Pakistan, and Sri Lanka. *Other Southeast Asia* comprises Cambodia, Indonesia, Laos, Malaysia, Myanmar (Burma), Philippines, Thailand, Timor-Leste, and Vietnam. *High-Income Asia* comprises Brunei, Japan, South Korea, and Singapore. Values for All Asia are weighted averages of the regions. [Adapted from the Frontier Research Center for Global Change 2007 with additional material from T. Ohara (personal communication, May 26, 2008).]



Executive Summary Figure 4. Annual averages of PM_{10}, SO_2, and NO_2 aggregated among selected Asian cities, 1993–2005. The cities used for this figure are shown in Executive Summary Figure 2. The straight lines are smoothed estimates of trends in air pollution levels. [Reprinted with permission from CAI-Asia (*www.cleanairnet.org/caiasia*; accessed January 2008).]

quality management as well as improved energy efficiency and reduced intensity of energy use.

From past experience in Western countries, it seems clear that substantial increases in the combustion of fossil fuels for power generation and transportation can improve economic conditions but can also, if not controlled, have important negative consequences for human health and environmental quality in Asia and elsewhere, through transboundary transport of pollutants. It is also clear that effective approaches to controlling and reducing pollution exist. Investment in these approaches need not necessarily impede economic growth and, on the basis of documented experience in developed countries and emerging evidence in Asia, the developing countries of Asia may be able to avert increased environmental degradation and associated adverse health impacts while reducing poverty and providing economic security for their populations (Clean Air Initiative for Asian Cities [CAI-Asia] Center 2008, U.S. Environmental Protection Agency [U.S. EPA] 2008).

Climate change and emissions of climate-forcing pollutants present a considerable challenge for Asia but are offer an additional rationale for continued improvement in air quality, with near-term benefits for public health from reductions of short-lived greenhouse gases (GHGs). Strategies for reducing GHG emissions, though directed toward climate change, may also have direct impacts on local and regional air quality, resulting in faster and larger improvements than would otherwise occur in the region.

Review of Epidemiologic Studies of Air Pollution

As a result of these developments, the need for high-quality research on the health effects of air pollution in Asia has never been greater, and the scientific community is responding with an increasing number of studies of the effects of exposure to air pollution on morbidity and mortality due to cardiovascular or respiratory diseases, adverse reproductive outcomes, and other health effects. Systematic searches of the peer-reviewed literature in HEI's PAPA–SAN database have identified over 400 studies of the health effects of air pollution in Asia published from 1980 through 2007 (Figure 5). The studies have been conducted in 13 countries, and the rate of publication has increased over the past 20 years. The spectrum of adverse health effects associated with air pollution exposure ranges from acute and chronic respiratory symptoms and changes in pulmonary function to increased mortality from cardiovascular or respiratory diseases or lung cancer — the same spectrum of adverse health outcomes associated with air pollution in the West.

Meta-Analyses of Daily Time-Series Studies

Time-series studies of the effects of short-term exposure on morbidity and mortality from cardiovascular or respiratory diseases continue to provide some of the most current and consistent evidence of serious adverse health effects of air pollution in Asia, with over 100 studies published since 1980. This literature now includes the results of the

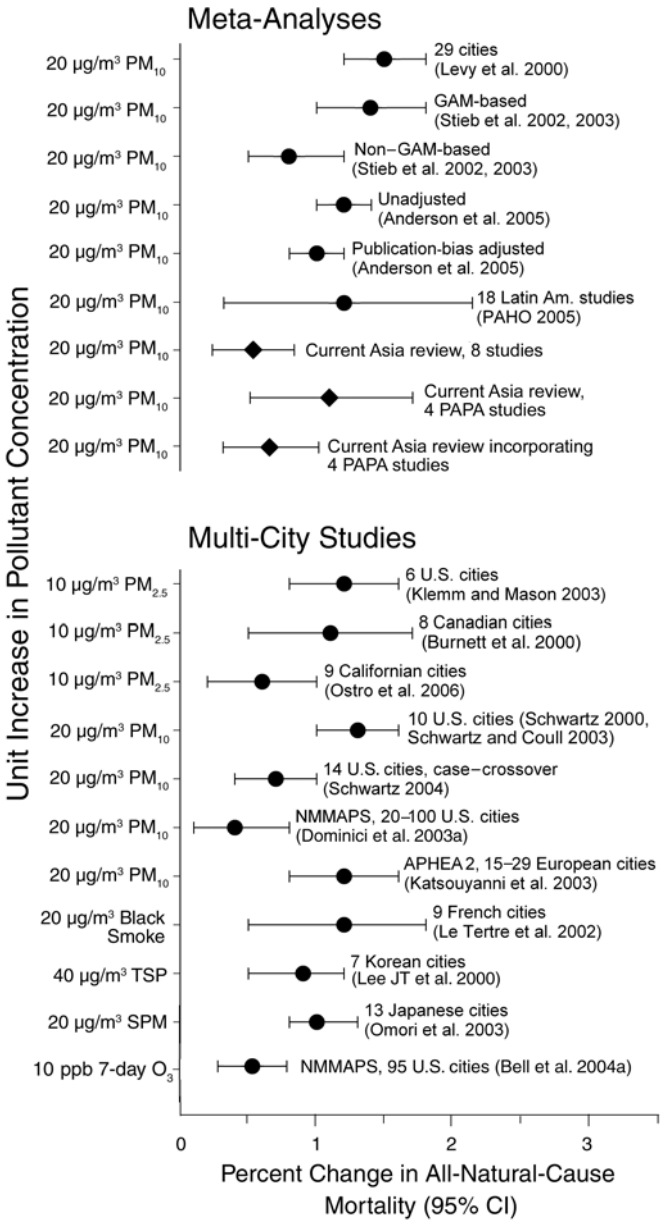
Executive Summary Figure 5. Number of studies identified in the PAPA–SAN Asian literature review, according to country of study.

coordinated studies in four Asian cities funded under the PAPA program (HEI Public Health and Air Pollution in Asia Program 2010), consisting of the type of evidence that has contributed most importantly to international guidelines and science-based regulatory policies in Europe and North America (Samoli et al. 2008; WHO 2005). Meta-analyses of time-series studies and coordinated multi-center studies of the effect on mortality of an increase in the concentration of a given air pollutant (called "effect estimates", shown here as the percent change in daily all-natural-cause mortality), including those from the four PAPA studies, are presented in Figure 6.

The updated meta-analyses of Asian time-series studies presented in this review summarizes results from 82 reports published through August 2007, more than three times the number in the 2004 review (HEI ISOC 2004), providing more reliable and detailed estimates of the magnitude of the effect of exposure on daily mortality and hospital admissions in Asian populations and allowing for more definitive comparisons of Asian evidence with results from other regions. Short-term exposure to particulate matter (PM) with an aerodynamic diameter $\leq$ 10 µm (PM_{10}) is estimated to increase daily mortality from all natural causes by 0.27% (95% confidence interval [CI], 0.12–0.42) per 10-µg/m^3 increase in pollutant concentration, an effect similar to that reported in meta-analyses and multi-city studies in Europe, North America, and Latin America. Underlying this estimate is the increased daily mortality from cardiovascular disease (chiefly IHD and stroke), the major current and future cause of death of adults in the region (Figure 7). Consistently larger exposure-related increases in all-natural-cause, cardiovascular, and respiratory mortality were also observed among older people, who represent an increasingly large proportion of Asian populations.

The PAPA Studies

The PAPA studies constitute the first designed and coordinated multi-city set of studies of the health effects of air pollution in Asia. As such, they provide a unique, if limited, picture of the short-term impact of current ambient concentrations of particulate air pollution on mortality in four large metropolitan areas in East and Southeast Asia: Bangkok, Hong Kong, Shanghai, and Wuhan. In the combined analysis of the city-specific results (Wong CM et al. 2008; Wong CM and the PAPA Teams 2010a), a 10-µg/m^3 increase in PM_{10} concentration was associated with an increase of 0.6% (95% CI, 0.3–0.9) in the daily rate of death from all natural causes, estimates similar to or greater than those reported in multi-city studies in the United States and Europe. This proportional increase in daily mortality is seen at levels of exposure (mean PM_{10} concentration, 51.6–141.8 µg/m^3) several times higher than those in most large Western cities, and in each of the four Asian cities, daily mortality continues to increase over a fairly large range of daily average ambient PM_{10} concentrations, up to several hundred micrograms per cubic meter.



Executive Summary Figure 6. Estimates of percent change in daily mortality from all natural causes for selected meta-analyses and multi-city studies of daily changes in air pollution exposure, by study type and unit change in pollutant concentration. TSP is total suspended particles; SPM is suspended particulate matter; O_3 is ozone; GAM is generalized additive model; PAHO is Pan American Health Organization; NMMAPS is National Morbidity, Mortality, and Air Pollution Study; APHEA2 is Air Pollution and Health: A European Approach. [Adapted from Pope and Dockery 2006 with estimates added from the meta-analysis in this Asian Literature Review (diamond data points; see Table 15 in the main report).]

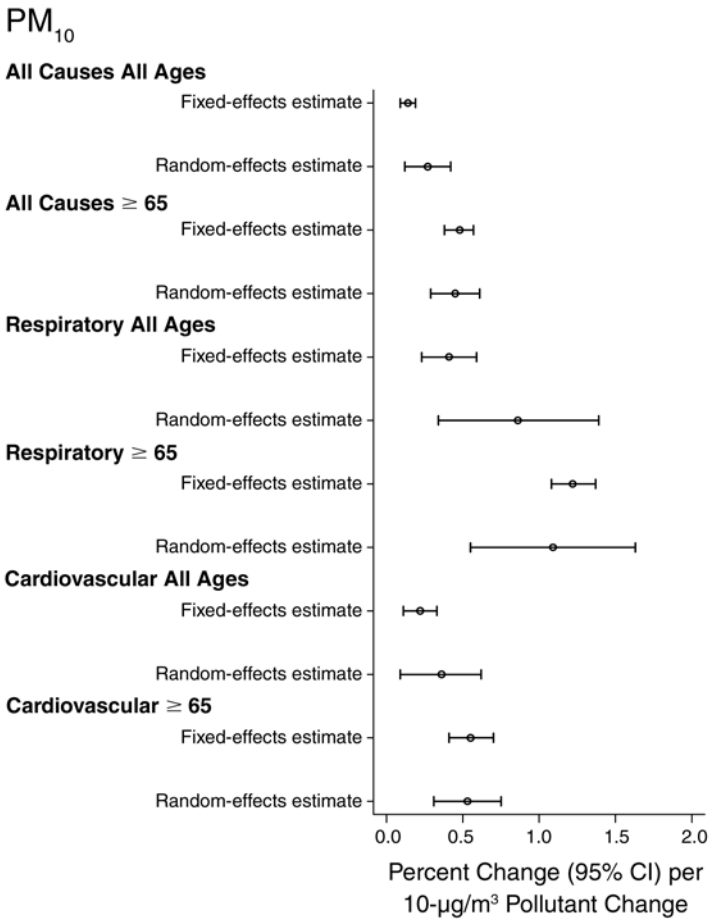
The summary estimate for PM_{10} from the four cities of the PAPA studies exceeds the overall meta-analytic summary estimate for the Asian studies for all pollutants and outcomes (with the exception of respiratory mortality, for which the PAPA estimates are lower) (Figure 6). The reasons for these differences are unclear but may be due to the higher estimates in Bangkok (for unknown reasons) or the systematic selection and quality control of air quality data according to rigorous and explicit protocols in the PAPA studies. Stochastic variation, or the play of chance, is another possible explanation. When the PAPA city-specific estimates are added to the meta-analyses, the summary estimate for all-natural-cause mortality per 10-µg/m^3 increase in PM_{10} becomes 0.33% (95% CI, 0.16–0.51), versus 0.27% (95% CI, 0.12–0.42). Pre-

liminary data from the PAPA-funded studies in two Indian cities, Delhi (Rajarathnam et al. 2010) and Chennai (Balakrishnan et al. 2010), also show increased rates of all-natural-cause mortality in association with short-term exposure to PM_{10}, though the point estimate for Delhi, 0.15% (95% CI, 0.07–0.23) per 10-μg/m^3 increase in PM_{10} concentration, is roughly half the meta-analytic summary estimate.

The studies in Hong Kong, Wuhan, Shanghai and Bangkok were designed to provide a combined picture of the effects of short-term exposure to pollution on daily mortality across the four cities, but each study also explored more detailed aspects of the epidemiology of exposure to air pollution in each location, providing additional insight into how factors such as weather and social class might modify the estimated relative risk of health effects of air pollution. The study in Wuhan, one of China's "oven cities" (cities that experience very high temperatures and humidity), found that the estimated relative risk may increase by a factor of 5 at extremely high temperatures, as compared with temperatures typical of temperate zones (Qian Z et al. 2008, 2010). The studies in Hong Kong (Wong CM et al. 2008, 2010b) and Shanghai (Kan H et al. 2008, 2010) found evidence of higher relative risks among the economically disadvantaged and those with the least education, respectively, corroborating the results of some earlier studies in Western cities (O'Neill et al. 2003).

Critical, Quantitative Review of Chronic-Effects Studies

The Asian literature on the chronic effects of long-term exposure to air pollution is more limited than the literature from Europe and North America, especially with regard to chronic cardiovascular disease. The design and quality of the studies also vary widely. Nonetheless, the results of this review suggest that long-term exposure to air pollution from combustion sources is contributing to



Executive Summary Figure 7. Summary effect estimates for all-natural-cause, respiratory, and cardiovascular mortality per 10-μg/m^3 change in PM_{10} concentration. Y-axis labels in bold specify the cause of death and the age group.

chronic respiratory disease in both children and adults, to lung cancer, and to adverse reproductive outcomes in Asian populations.

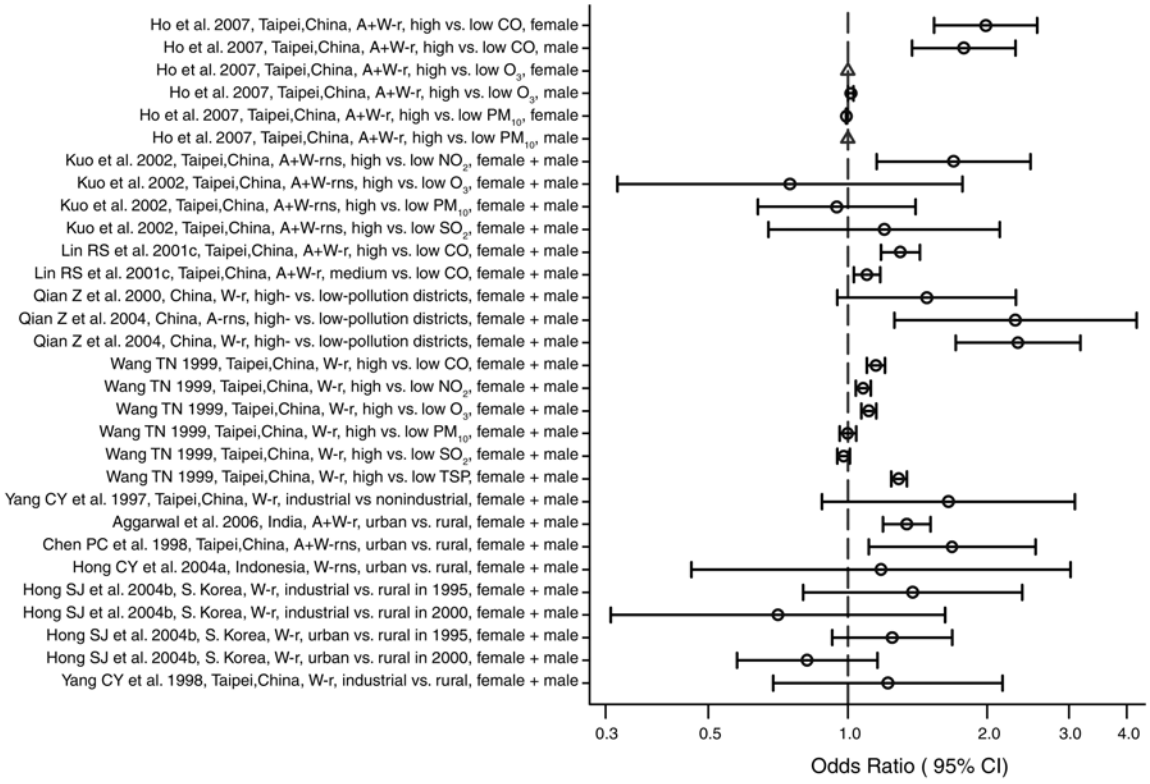
The prevalence of chronic phlegm, a major symptom of chronic respiratory disease indicating long-term exposure to inhaled irritants, was found to be associated with exposure to combustion-source air pollution both in qualitative comparisons among areas with differing concentrations of pollution and in quantitative comparisons of measured concentrations of air pollution. Studies that controlled for major potential confounding factors, including tobacco smoking and indoor air pollution from the burning of solid fuels, reported relative risk estimates generally between 1.1 and 5.0, regardless of how pollution was characterized. Associations were observed with both PM, measured as PM_{10}, and gaseous combustion-source pollutants such as sulfur dioxide (SO_2) and nitrogen dioxide (NO_2). Similar associations with pollution, after controlling for smoking, have been reported in many surveys in North America and Europe, including some in which the sources and concentrations of pollutants were similar to those in some Asian cities in this review. The increased prevalence of chronic phlegm was seen in both adults and children. In children, although it is not an indicator of chronic bronchitis or COPD (which are diseases of later life), chronic phlegm may be correlated with repeated respiratory infections, which in turn may result in reduced pulmonary function. Reduced lung function in childhood and early adulthood is associated with an increased risk of developing COPD later in life (Fletcher and Peto 1977; Rennard and Vestbo 2008).

Evidence from Asia regarding air pollution effects on ventilatory lung function is limited, and few studies of adults have used acceptable methods with regard to testing protocols or control for the effects of tobacco smoking. The studies of children and non-smoker adults that have been conducted suggest adverse effects of ambient exposures, but because these studies are cross-sectional and because the air pollution exposures were estimated for whole geographic areas rather than for individuals, it is difficult to reach firm conclusions as to the impact of ambient pollution on pulmonary function. Such cross-sectional studies suggest detrimental effects of exposure to air pollution on children's lung function, providing snapshots of events that are part of a dynamic process that affects lung growth and development. Longitudinal studies are needed to determine whether the cross-sectional associations with air pollution represent a slower-than-normal growth of lung function that results in permanent deficits (and might subsequently lead to an accelerated decline in lung function in adulthood) or, as some studies in Western countries suggest, a transient worsening of pulmonary function with recovery as pollution concentrations improve. There is also the potential for residual confounding, such as confounding due to differences among factors related to socioeconomic status. These problems are encountered in cross-sectional studies in Western counties as well as in Asia.

A similarly diverse and overlapping collection of studies also provides some evidence for an increased prevalence of asthma and asthma-related symptoms in association with exposure to air pollution from a variety of sources. Most studies show an elevated prevalence in association with air pollution exposure, with estimated relative risks generally greater than 1.0 but less than 2 (Figure 8). Studies of long-term exposure to ambient air pollutants and asthma symptoms and diagnoses vary considerably in design. Differences in exposure have been estimated at between- and within-city levels, with the within-city comparisons including roadside exposures. The studies also vary widely in terms of statistical power, quality of exposure assessment, and degree of control of confounding factors. Overall, however, the design and conduct of the studies are similar to those from Western countries. Although the results of the studies reviewed in this Special Report are not entirely consistent, there is some evidence for a modest effect of air pollution on asthma prevalence — and possibly more evidence than in current Western studies. We still know little about the reasons for this effect or for the observed heterogeneity among the findings. Asthma prevalence ranges widely among children in Asia, but this variation seems unlikely, on the basis of the current evidence, to be explained by the corresponding range of ambient air pollution concentrations (Anderson et al. 2010).

There is limited evidence regarding air pollution and lung cancer in Asian populations. The two studies that have addressed potential confounding by strong risk factors, such as tobacco smoking and indoor air pollution from the burning of solid fuels, report relative risks for lung cancer in the range of 1.5 to 3.0, consistent with those from studies in Europe and North America; but more definitive evidence will require larger studies that directly assess the effects of exposure using measured, rather than estimated, concentrations of air pollutants (at the higher concentrations) and that adequately control for potential confounders.

The Asian literature suggests that the risks of adverse reproductive outcomes, such as low birth weight and preterm delivery, in association with exposure to air pollution are relatively small. Overall, the estimated relative risks are between 1.04 and 2.0. Exposure in early pregnancy appears to be most strongly associated with risk. These results are generally consistent with those from the larger global literature on air pollution and adverse pregnancy outcomes. However, the Asian studies, like those con-



Executive Summary Figure 8. Odds ratios (and 95% CIs) for asthma or wheeze from qualitative comparisons between areas with differing pollutant concentrations in studies of urban and rural locations, or between or within cities. Y-axis labels give study information in the following sequence: reference citation; location, outcome, pollutant levels or areas that were compared; and sex of subjects (female; male; or combined). Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting was unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), W-rns (wheeze symptoms whose time of reporting is unspecified or not recent) or a combination of asthma and wheeze (A+W-r and A+W-rns). CO is carbon monoxide; TSP is total suspended particles. A log scale is used on the x axis to accommodate the range of estimates. Two studies reported "no significant association" with no odds ratio; data points are shown here as triangles without CIs. For study details, see Table 19 of the main report.

ducted elsewhere, are limited by incomplete control for potential confounders, such as maternal smoking, and are subject to exposure measurement error owing to the use of routinely collected health and air quality data. Some studies have reported the risk per quantitative increases in pollutant concentrations, but most have instead characterized exposure in terms of residential proximity to sources of pollution, mainly industrial sources, thus limiting comparisons of these studies with the larger international body of studies that use ambient air pollution measurements to characterize exposure.

IMPLICATIONS FOR ASSESSMENT OF HEALTH IMPACTS

Very large populations are exposed to high concentrations of air pollution in developing Asia. Indeed, 30 million people currently reside in just the first four cities studied in the PAPA research project. Thus, the estimated effects of both short- and longer-term exposures reviewed in this report, though small in relative terms, probably translate to large numbers of illnesses and deaths attributable to air pollution. The WHO estimated that over 500,000 deaths in Asia in 2000 were due to outdoor air pollution exposure, accounting for approximately two thirds of the total global burden of deaths attributed to air pollution (WHO 2002). Indoor air pollution from use of solid fuel contributed an additional 1.1 million deaths. Other impact assessments have reported similar estimates (World Bank and the Chinese State Environmental Protection Administration [SEPA] 2007). That said, air pollution is but one of many factors that affect the health of people in developing Asia (Ezzati et al. 2002). Nonetheless, the substantial health impacts of exposure to air pollution are clearly a factor that should be considered in transportation and energy policy in the region.

In Asia and elsewhere, an increased life expectancy is a major social benefit of economic growth and its attendant, if often variable, reductions in poverty. Exposure to air pollution acts to reduce healthy life expectancy, shortening lives by months and even years, on average, in some populations (Brunekreef et al. 2007). Although the time-series studies reviewed here document the occurrence of excess mortality related to short-term exposure, the study results cannot currently be used directly to estimate reductions in life expectancy due to extended exposure (Rabl 2006; Burnett et al. 2003). Such estimates are provided by cohort studies, in which large numbers of individuals exposed to various concentrations of air pollution are observed for years and the mortality rates in each exposure group are compared. Such studies have been conducted in the United States and Western Europe, but to date, no cohort studies of long-term exposure to air pollution and mortality from chronic cardiovascular or respiratory disease appear to have been reported in Asia. As a result, recent estimates of the health impacts of air pollution in Asia (WHO 2002; World Bank and SEPA 2007) are based on the results of a single U.S. study (Pope et al. 2002).

The broad consistency of the results of Asian time-series studies of mortality with those in Europe and North America, including the evidence of greater rates of cardiovascular morbidity and mortality among older people than among younger people, supports the continued use of data from Western cohort studies to estimate the Asian burden of disease attributable to air pollution. However, developing Asia currently differs from the United States and Europe with regard to energy use, air quality, and population health, which are also dynamically changing. Thus, estimates of the impact of air pollution that are based on even the most carefully executed U.S. studies must be used with caution. One key area of uncertainty is the shape of the concentration–response function relating long-term exposure to air pollution and mortality from chronic disease. The concentrations of PM with an aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$) studied in the American Cancer Society (ACS) study (Pope et al. 2002) were much lower than the concentrations in major cities in China and India, requiring that analysts extrapolating the ACS data to Asia make projections regarding the shape of the concentration–response function at much higher concentrations. The uncertainty in the resulting estimates, when quantified in sensitivity analyses, was substantial (Cohen et al. 2004).

KNOWLEDGE GAPS AND RESEARCH NEEDS

The acute toxicity of short-term exposure to high air pollution concentrations has been appreciated since the early 20th century, and recent multi-city studies in Europe and North America have identified toxic effects at even lower concentrations. Therefore, the results of meta-analyses of Asian time-series studies of daily mortality and hospital admissions are not unexpected and can serve as an important part of the scientific basis and rationale for interventions to improve air quality. Nevertheless, there is much we still need to learn in order to fully understand the substantial air pollution challenges in Asia, and high-quality, credible science from locally relevant studies will be critical to helping decision makers choose which policies are most likely to result in public health benefits.

How Does the Nature of the Air Pollution Mixture Affect Air Quality, Exposure, and Health Effects?

Health impacts in cities in developing countries of Asia result from exposure to a mixture of pollutants, both particles and gases, which are derived in large part from combustion sources (Harrison 2006). This is true in Europe and North America as well, but the specific sources and their proportional contributions in Asia are different. Time–activity patterns, building characteristics, and proximity of susceptible populations to pollution sources in the region also differ from those in Western countries in ways that may affect human exposure and health effects. Our current knowledge of these issues is rudimentary, based largely on studies of individual pollutants, and additional research is needed to inform effective and sustainable control strategies. Without such studies, epidemiologists will have a difficult time assessing the relative effects of various pollution mixtures or specific pollution sources or even interpreting patterns of variation.

What Are the Effects of Long-Term Exposure to Air Pollution?

Although time-series studies will continue to be important potential drivers of environmental and public policy, additional study designs, such as recent U.S. and European cohort studies, are needed in Asia to estimate the effects of long-term exposure on annual average rates of mortality from chronic cardiovascular or respiratory diseases and impacts on life expectancy, the metrics that may be the most meaningful and relevant to policy.

Conducting such studies will be challenging, not least because of rapidly changing air pollution concentrations and exposures in developing Asia, although as some U.S. studies suggest, if chronic effects are due to recent exposure, this problem may not be severe. Recent exposure will also be most relevant in studies of adverse reproductive outcomes and effects on the health of young children. A detailed quantitative review of the larger Asian literature

(including cross-sectional studies of chronic respiratory disease and studies of lung cancer and adverse reproductive outcomes) may better inform extrapolations of the results from the Western studies; the same may be true of the concentration–response functions describing short-term exposure and daily mortality recently reported for three Chinese cities (Wong CM et al. 2008; Wong CM and the PAPA Teams 2010a), but long-term Asian studies will provide the most direct evidence. It may be possible to "retrofit" existing Asian cohort studies, originally designed to address issues other than air pollution, with estimates of air pollution exposure, the approach used in the ACS study. Retrofitting studies requires the building of multidisciplinary teams of investigators, with commitment of adequate, long-term resources, to work in collaboration with government officials, their industrial counterparts, and local stakeholders. In order to assess the current potential for such studies, HEI's PAPA program issued a Request for Information and Qualifications (Health Effects Institute 2009) for teams of investigators to conduct such studies in developing countries of Asia. Based on the responses, they concluded that the potential exists in several locations.

What Do Results of Current Time-Series Studies in a Subset of Asian Cities Tell Us About Health Effects of Air Pollution Exposure in Other, As Yet Unstudied, Asian Locales?

The numbers of time-series studies being reported from across Asia is growing; the first Indian studies of short-term exposure to respirable suspended particles (RSP) and daily mortality, part of the PAPA research program, will soon be published (Balakrishnan et al. 2010; Rajarathnam et al. 2010). Even so, almost all current studies have been conducted in mainland China, Taipei,China, and South Korea. Major population centers in South and Southeast Asia (India, Pakistan, Vietnam, Philippines, Indonesia, and Malaysia) are still largely understudied (with the exception of Bangkok). Differences in the relative prevalence of urban air pollution sources (such as open burning) and urban poverty may modify the effects of exposure. Expanded coordinated multi-city studies, designed and analyzed consistently and conducted across the region, could provide more definitive answers. In some cases, outcomes other than mortality, such as hospital admissions, may also be studied, enabling policy makers to better quantify the health impacts of air pollution.

What Role Does Indoor Air Pollution Play in the Health Effects of Outdoor Air Pollution?

The magnitude and prevalence of exposure to indoor air pollution are high in Asian cities, especially among people living in poverty. We need a better understanding of how air pollution from indoor sources contributes to concentrations of outdoor air pollution and how indoor exposure to air pollution from indoor sources affects risk estimates for outdoor air pollution. Coordinated measurements of exposure and coordinated epidemiologic studies will be needed to address these questions.

What Role Does Poverty Play in the Health Effects of Air Pollution?

Limited evidence, largely from studies in Europe and North America, suggests that economic deprivation increases the rates of morbidity and mortality related to air pollution. One reason may be the higher air pollution exposures that people of lower socioeconomic status experience. The degree of vulnerability can also be affected by factors related to socioeconomic status, such as health, nutritional status, and access to medical services. Studies of these issues are relatively rare in Asia, where extreme poverty is more prevalent than in the West, so that results of the Western studies cannot be simply extrapolated, though the recent results of the PAPA studies in Shanghai and Hong Kong are welcome and much-needed additions (Kan H et al. 2008, 2010; Wong CM et al. 2008, 2010b). Some analyses of U.S. cohort studies suggest that low levels of attained education were associated with larger estimated relative risks of air pollution–related mortality (Krewski et al. 2000), but more recent analyses based on extended follow-up of the largest cohort have not upheld this pattern (Krewski et al. 2009). Studies in Asia that examine the effect of exposure on morbidity and mortality from diseases associated with poverty (such as acute lower respiratory infection in children, and tuberculosis) and studies that estimate effects of exposure across socioeconomic strata are needed. HEI's recently completed study of hospital admissions for acute lower respiratory infection in children in Ho Chi Minh City is, to our knowledge, the only example of such a study (Collaborative Working Group on Air Pollution, Poverty, and Health in Ho Chi Minh City 2009).

What Are the Health Consequences of Changes in Air Pollution Resulting from Climate Change and Efforts to Reduce Emissions of Climate-Forcing Agents?

Changes in air pollution resulting from climate change and from efforts to reduce emissions of climate-forcing agents may have important consequences for health in the

region, especially in low- and middle-income countries. However, major unknowns remain, including (1) the quantitative association between reductions in the concentrations of GHGs (such as carbon dioxide) and toxic air pollutants such as ($PM_{2.5}$), (2) the relative toxicity of short-lived greenhouse pollutants with opposite climate-forcing potentials (such as sulfate and black carbon), and (3) the impact of policy choices. There is also a need to understand more fully how concentration–response functions for air pollution may vary with regard to global and within-region differences in climate, demographics, and pollutant mixes.

Finally, although the ability to conduct research on the health effects of air pollution in developing Asia is improving, it is still constrained by limitations in environmental and public health infrastructures. Air quality monitoring has increased in the region, but there is a need for more extensive monitoring of urban air quality designed to support health effects studies and impact assessments and a corresponding need for more highly trained professionals in air quality monitoring, exposure assessment, and environmental epidemiology. Equally important, there remain considerable deficiencies in registration of vital statistics in Asia, especially regarding accurate and comprehensive assignment of causes of death. There is also a need to encourage cooperation and collaboration in health effects research between health and environmental scientists and public agencies. These deficiencies constitute a major impediment to environmental health research and, more broadly, to the development of appropriate, evidence-based public health policy.

SUMMARY AND CONCLUSIONS: ENHANCED EVIDENCE OF EFFECTS OF AIR POLLUTION IN ASIA

Based on findings from more than 80 Asian time-series studies, including coordinated multi-city time-series studies, the meta-analytic estimates appear consistent in both direction and magnitude with those from other regions. In broad terms, the effects of short-term exposure in Asian cities are on a par with those observed in hundreds of studies worldwide. The same pollutants — RSP and gaseous pollutants such as ozone (O_3), SO_2, and NO_2 — affect older people with chronic cardiovascular or respiratory disease. The adverse effects in some locales, reported in studies published in the 1980s and 1990s, may reflect the effects of air pollution concentrations that have subsequently decreased. However, more recent studies continue to report adverse effects at lower levels in cities in Thailand and Japan, where air quality has improved, as well as in heavily polluted Chinese and Indian cities.

The results of our meta-analyses of time-series studies should serve to reduce concerns regarding the generalizability of the results of the substantial, but largely Western, literature on the effects of short-term exposure to air pollution. They suggest that neither genetic factors nor longer-term exposure to highly polluted air substantially modifies the effect of short-term exposure on daily mortality rates in major cities in developing Asia. This provides support for the notion, implicit in the approach taken in setting the WHO world air quality guidelines (Krzyzanowski and Cohen 2008), that incremental improvements in air quality are expected to improve health, even in areas with relatively high ambient concentrations. The results also suggest that health benefits would result from further reductions in exposure to pollution concentrations below those specified in the WHO guidelines.

The results of the chronic-effects studies reviewed in this report appear to be broadly consistent with those of studies in other regions, suggesting that long-term exposure to air pollution promotes chronic pulmonary disease and other adverse effects that result in reduced life expectancy. Nevertheless, these studies are more susceptible than the time-series studies to uncontrolled confounding by strong risk factors, such as tobacco smoking, indoor air pollution from the burning of solid fuels, and factors related to socioeconomic status, such as diet. These risk factors may also modulate the effect of air pollution, leading to larger effects in some population groups and smaller effects in others. Some of these factors, such as those related to the level of economic development, may be particularly important in developing Asia. If they are ignored or poorly measured, an inaccurate estimate of the effects of air pollution may result, and real differences among study results in various regions may be obscured.

This literature review documents a number of promising improvements in air quality in Asian cities, even in the context of economic growth. However, susceptibility to the effects of air pollution in Asia can be expected to rise because rates of chronic cardiovascular and respiratory disease increase as populations age, exposure to combustion-source air pollution becomes more widespread owing to urbanization, vehicularization, and industrialization, and risk factors increase in prevalence. In future assessments, these changes may yield larger health impacts in the region, although the effects of these changes could be counterbalanced by improved access to medical care and other improvements in the standard of living. Higher estimates of the magnitude of air pollution effects may also contribute to larger impact estimates. For example, the most recent publication from the ACS study (Krewski et al. 2009) reported larger estimates of

the risks of cardiovascular mortality and lung cancer than previously reported.

This review demonstrates that the information on the health effects of air pollution in developing Asia is substantial and continues to grow in both size and quality. As such, it provides an increasingly confident base of scientific evidence to inform critical decisions in the region regarding policies to protect public health while furthering economic development. Important gaps still remain in the range of Asian settings studied and in the types of studies that need to be conducted in order to fully inform public policy decisions. This need will only grow as the attention of policy makers and the public increasingly focuses on issues of regional importance, such as climate change and transboundary air pollution. HEI intends that the publication of this Special Report, and continued funding of a targeted program of research in Asia under the PAPA program, will improve understanding of the problems posed by air pollution in Asia and will further develop the capacity of Asian scientists to conduct additional scientific research toward solutions.

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ABBREVIATIONS AND OTHER TERMS

ACS	American Cancer Society
APHEA	Air Pollution and Health: A European Approach
CAI-Asia	Clean Air Initiative for Asian Cities
CI	confidence interval
CO	carbon monoxide
COPD	chronic obstructive pulmonary disease
DALYs	disability-adjusted life-years
GAM	generalized additive model
GHG	greenhouse gas
IHD	ischemic heart disease
ISOC	International Scientific Oversight Committee
NMMAPS	National Morbidity, Mortality, and Air Pollution Study
NO_2	nitrogen dioxide
O_3	ozone
OR	odds ratio
PAHO	Pan American Health Organization
PAPA	Public Health and Air Pollution in Asia
PAPA–SAN	Public Health and Air Pollution in Asia — Science Access on the Net
PM	particulate matter
$PM_{2.5}$	PM with an aerodynamic diameter $\leq$ 2.5 µm
PM_{10}	PM with an aerodynamic diameter $\leq$ 10 µm
RSP	respirable suspended particles
SEPA	Chinese State Environmental Protection Administration
SO_2	sulfur dioxide
TSP	total suspended particles
U.S. EPA	U.S. Environmental Protection Agency
WHO	World Health Organization

Section I. Introduction

Asia is undergoing economic development at a rapid rate, resulting in levels of urban air pollution in many cities that rival the levels that existed in Europe and North America in the first decades of the 20th century. This development is also transforming the demographic and epidemiologic characteristics of the population in ways that are likely to affect its vulnerability to air pollution. Nearly two thirds of the estimated 800,000 deaths and 4.6 million lost years of healthy life worldwide caused by exposure to urban air pollution in 2000 occurred in the developing countries of Asia (also referred to as "developing Asia"; World Health Organization [WHO] 2002). In rural areas and urban slums, indoor air pollution from the burning of solid fuels for cooking and heating confers its own large burden of disease and contributes to increased outdoor concentrations of pollutants in some locales. Developing Asia's poorest populations are also expected to suffer the effects of global warming, including possibly substantial health effects, due largely to climate change already caused by the emissions of wealthy, developed countries. Asia's contribution to global emissions from climate forcing is great as well and is projected to increase in the future, with consequences for human health in the region that are likely to be considerable.

Developing Asia is not a single entity, however. Underlying the broad trends in air pollution in the region is considerable variation between and within countries with regard to the current pace of economic and social development and the status of and trends in factors that determine air quality, human exposures to air pollution, and the resultant health impacts.

Effective public policy responses to the public health challenges posed by air pollution in developing Asia require high-quality scientific evidence on the health effects of air pollution in the region. Epidemiologic studies are the most important and critical components of the required evidence; together with toxicologic and clinical studies, they provide estimates of the quantitative relation between exposure and disease. These estimates both demonstrate the existence of a public health hazard and allow its magnitude to be estimated. Owing to limitations of the available epidemiologic studies in Asia, estimates from assessments of the health impact of air pollution in Asian populations (e.g., the estimate from the WHO cited above), rely in large part on extrapolation, with considerable uncertainty, of the results of Western studies to Asian populations (Cohen et al. 2004; HEI International Scientific Oversight Committee [ISOC] 2004).

HEI initiated the Public Health and Air Pollution in Asia (PAPA) program in 2002 to reduce uncertainties about the health effects of exposure to air pollution in the cities of developing Asia. The PAPA program is part of the Clean Air Initiative for Asian Cities (CAI-Asia) — a partnership of lenders, governments, industry, environmentalists, and others to improve Asian air quality. The first major publication of the PAPA program was Special Report 15, *Health Effects of Outdoor Air Pollution in Developing Countries of Asia: A Literature Review* (2004). That report was also the first comprehensive review of the peer-reviewed Asian literature on the health effects of air pollution, a literature that at that time comprised over 100 studies in nine countries (HEI ISOC 2004).

Since Special Report 15 was published, that literature has more than doubled in size. It now includes the results of the recently completed coordinated set of analyses of air pollution and daily mortality in four Asian cities (Bangkok, Hong Kong, Shanghai, and Wuhan)† (see Sidebar 1) and in India and Vietnam, studies that were funded by HEI's PAPA program on the basis of Special Report 15. The current Special Report 18 is the second PAPA literature review, which provides an expanded assessment of the Asian literature on the health effects of outdoor air pollution.

This review begins with a broad overview of the status of and trends in air pollution sources, emissions, concentrations, and exposures in the developing countries of Asia, as well as factors related to urban development, population health, and public policy that set the context for the health effects of air pollution. Local examples are highlighted throughout, to illustrate the diversity among countries. Next, the review describes the current scope of the Asian literature on the health effects of air pollution, enumerating and classifying more than 400 studies. These studies were identified through 2007 via HEI's Web-based Public Health and Air Pollution in Asia–Science Access

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* A list of abbreviations and other terms appears at the end of the report.

† A list of cities, provinces, and countries appears at the end of the report.

Sidebar 1: PAPA-FUNDED TIME-SERIES STUDIES OF AIR POLLUTION AND MORTALITY

By bridging the gap between studies conducted locally in Asia and those performed elsewhere, PAPA-funded studies in representative cities provide useful information to policy makers. The PAPA studies are designed and conducted by local investigators in concert with local air pollution and public health officials and international experts. They examine relations between daily changes in air pollution and mortality in cities with varying climates and air pollution concentrations.

The PAPA-funded studies in Asia were conducted using the same types of routinely collected administrative data on mortality and air pollution concentrations used in time-series studies throughout the world and with methodologic rigor that matches or exceeds that of most published studies. Important features of the studies include formal quality control in the form of detailed standard operating procedures for collection and analysis of data and external quality assurance audits of the data overseen by HEI. These studies also benefited from recent efforts to strengthen and refine methods for the analysis of time-series data, and as a result are on a par methodologically with the most recent U.S. and European analyses (Health Effects Institute 2003).

COORDINATED STUDIES IN HONG KONG, SHANGHAI, WUHAN, AND BANGKOK

Coordinated multi-city studies currently provide the most definitive epidemiologic evidence of the effects of short-term exposure to air pollution and as a result play a central role in health impact assessment and environmental policy. Although robust and consistent results have been observed in Europe and North America (Samet et al 2000b; Katsouyanni et al. 2001), few coordinated, multi-city time-series studies have been conducted elsewhere.

Four time-series studies of the health effects of air pollution — in Bangkok, Hong Kong, Shanghai, and Wuhan — initiated by the PAPA program in 2003 represent the first coordinated multi-city analyses of air pollution and daily mortality ever undertaken in local populations in Asia, as well as the first phase of an effort to conduct a series of studies in Asian cities that will deepen our understanding of the effects of air pollution on local populations and inform extrapolation of results from the extensive Western literature to Asian settings.

These studies explore key aspects of the epidemiology of exposure to air pollution in each city. Issues of local as well as global relevance include the effects of exposure at high concentrations and at high temperatures, the potential influence of influenza epidemics on the relation between air pollution and health, and the ways in which social class may modify risks associated with air pollution. Taken together, the four studies and the combined analysis of their findings provide a relatively consistent, if limited, picture of the short-term impact on mortality of current ambient particulate air pollution in several large metropolitan areas in East and Southeast Asia.

In the combined analysis (Wong CM et al. 2008b, 2010a), a 10-μg/m^3 increase in PM_{10} concentration was associated with an increase of 0.6% (95% CI, 0.3–0.9) in daily all-natural-cause mortality, with the city-specific estimates similar to or greater than those reported in U.S. and European multi-city studies. Proportional increases in mortality rates in the four Asian cities were seen at mean PM_{10} concentrations (51.6–141.8 μg/m^3) several times higher than those in most large Western cities. In Hong Kong, Wuhan, and Bangkok (but not Shanghai), the pattern of the exposure–response functions appears to be linear over a fairly large range of ambient concentrations, up to and sometimes exceeding 100 μg/m^3 (Figures 1.1 and 1.2).

Despite the fact that only four cities were studied, these results may begin to allay concerns regarding the generalizability of results of the substantial but largely Western literature on the effects of short-term exposure to air pollution. These PAPA results, which are broadly consistent with those from previous research (HEI ISOC 2004), suggest that neither genetic factors nor longer-term exposure to highly polluted air substantially modifies the effect of short-term exposure on daily mortality in major cities in developing Asia. This evidence provides support for the notion, implicit in the approach taken in the WHO's air quality guidelines (Krzyzanowski and Cohen 2008), that incremental improvements in air quality are expected to improve health, even in areas with relatively high ambient concentrations.

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on the Net (PAPA–SAN) literature survey (Health Effects Institute 2008), part of HEI's PAPA program, created to help researchers studying the effects of air pollution in Asia and to provide policymakers, international lending organizations, and other stakeholders with information to help them make better-informed decisions.

The review then focuses on an analysis of time-series studies that estimate the effect of short-term exposure to air pollution on daily mortality and hospital admissions for cardiovascular and respiratory disease. Time-series studies were chosen because they have been conducted in many regions of the world, including Europe and North America, where coordinated multi-city studies have contributed greatly to public policy decisions. It is therefore possible to compare their results across regions. Such comparisons could inform the extrapolation to Asian populations of results from other types of epidemiologic studies of air pollution (e.g., studies of the effects of long-term exposure on mortality from chronic disease) that have only been conducted in developed Western countries. Time-series

Sidebar 1. PAPA-FUNDED TIME-SERIES STUDIES (*Continued*)

STUDIES IN INDIA

In recognition of the fact that India is a diverse, densely populated country where the burden of disease attributable to ambient air pollution is likely to be substantial, HEI extended the PAPA research program to include three studies of air pollution and all-cause mortality in Chennai, Delhi, and Ludhiana. The Indian studies focused on the association between increased air pollution and all-natural-cause mortality in 2002–2004. Because of key differences in the availability and completeness of data between the first four PAPA studies and the two Indian studies, however, the common protocol developed for the first four studies was neither adequate nor sufficient for use in India. Therefore, the Indian investigators adapted the common protocol by developing city-specific approaches for using available air quality data to calculate estimates of daily exposure.

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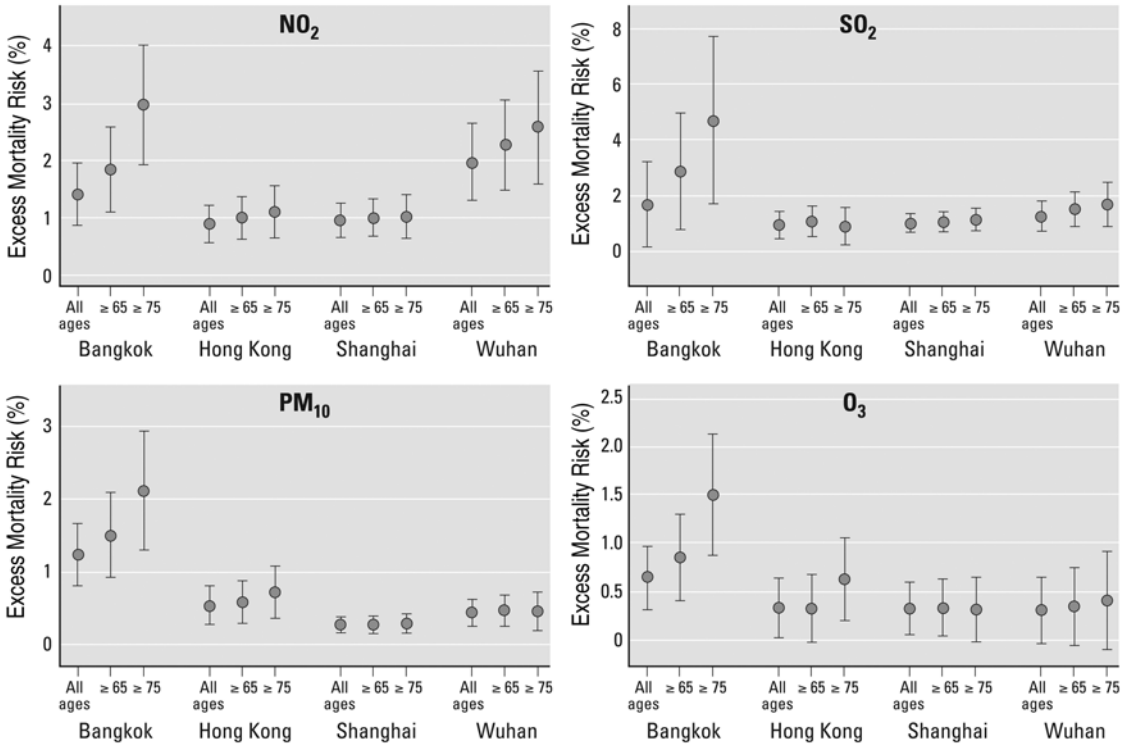

Figure 1.1. Percent excess risk of mortality (and 95% CI) for a 10-μg/m^3 increase in the average pollutant concentration (lag time, 0–1 day) in the PAPA studies, according to age group (in years) and city. Note that each panel has a different scale on the y-axis. [Reprinted from Wong CM et al. 2008b.]

studies that use routinely collected data are time-efficient, cost-efficient, and generally easy to implement compared with long-term follow-up studies of individuals (e.g., cohort studies). For this reason, time-series studies are among the most frequently conducted studies of the health effects of air pollution in Asia and worldwide. A total of 82 time-series studies are included in this analysis, 4 times the number of studies analyzed in Special Report 15 (HEI ISOC 2004). The analysis provides numeric and graphic summaries by country, health outcome, air pollutant, and other study features.

Although time-series studies provide important information about the exacerbation of preexisting disease, studies of long-term exposure to air pollution can provide information about the incidence of disease and the effects of exposure and loss of healthy years of life for use in health-impact assessments. This review includes a first-ever critical and qualitative analysis of Asian studies of long-term exposure to air pollution and chronic respiratory disease, lung cancer, and adverse reproductive outcomes. The review concludes with a discussion that places the Asian health effects studies in the context of the worldwide literature,

Sidebar 1. PAPA-FUNDED TIME-SERIES STUDIES (*Continued*)

Results from Delhi and Chennai suggest a somewhat lower risk of mortality association with air pollution exposure than found in the first four studies. In the Delhi study, there was a 0.15% increase in total all-natural-cause mortality for every 10 μg/m^3 increase in PM_{10} concentration. The Chennai study reported a 0.4% increase for every 10 μg/m^3 increase in PM_{10} concentration. Given substantial limitations and uncertainties in the Ludhiana data, it was not clear that an interpretable result would be possible; the study was terminated early.

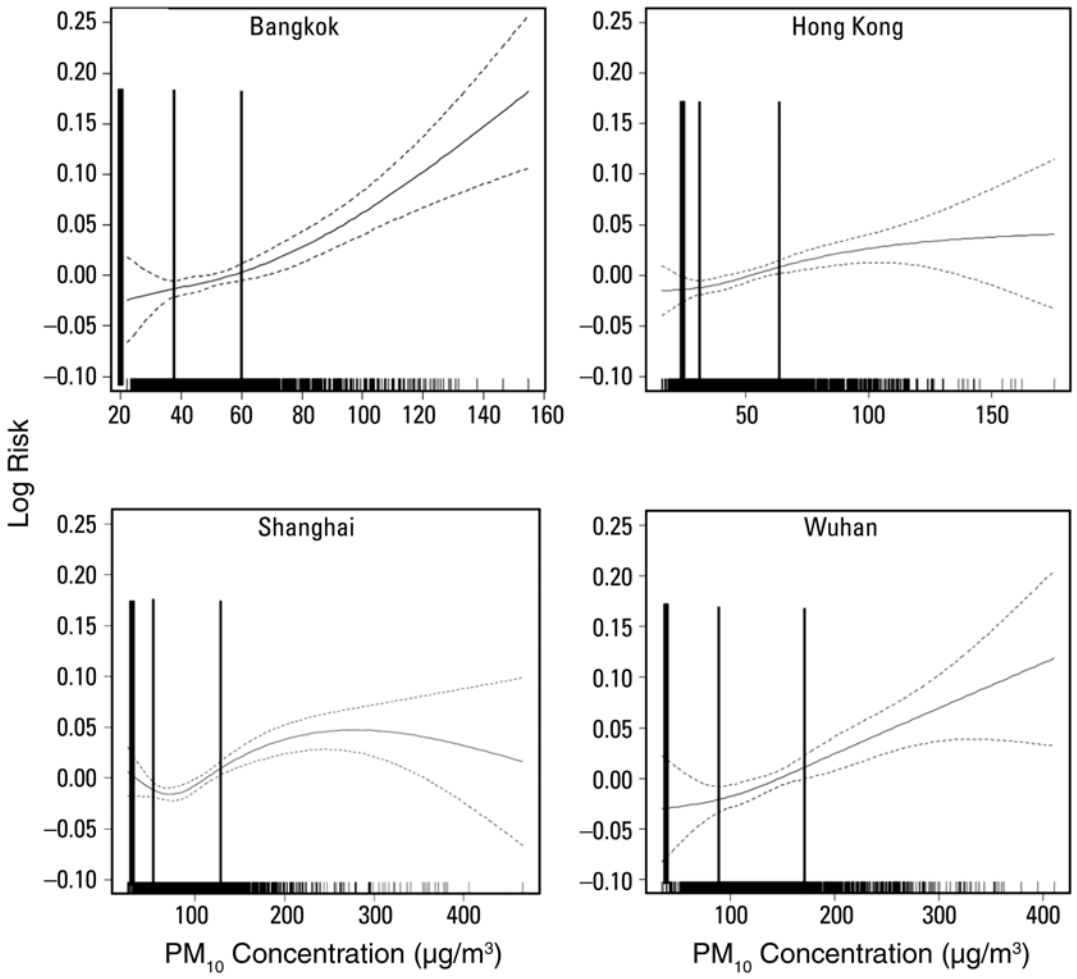

Figure 1.2. Concentration–response curves for PM_{10} and all-natural-cause mortality (lag time, 0–1 day) in the PAPA studies, according to city. The thin vertical lines represent the interquartile range of PM_{10} concentrations. The thick vertical lines represent the WHO guidelines (WHO 2005a) of 20 μg/m^3 PM_{10} for a 1-year averaging time. [Reprinted from Wong CM et al. 2008b.]

identifies gaps in knowledge, and recommends approaches by which to address them.

As this review makes clear, scientific knowledge about the health effects of air pollution in developing Asia is continuing to advance. However, gaps in scientific understanding with important implications for public policy still remain. As PAPA's ISOC noted in the introduction to the first review, future research should continue to be designed to address clear objectives that fill critical gaps in the current evidence. Systematic, quantitative review of the existing literature remains essential to identifying those gaps, informing the design of future research, and providing a critical assessment of currently available evidence to inform present decisions.

Section II. Development, Air Pollution Exposure, and Population Health

TRENDS IN DEVELOPMENT

Developing Asia is in many ways the most dynamic part of the world. Although hundreds of millions of residents still live in poverty, slowing of population growth and steady growth of per capita income are real prospects. In the next decades, if current trends continue, developing Asia may well be nearing the per capita income of middle-income Latin America (United Nations Economic and Social Affairs 2004a).

Increased population growth, economic development, and urbanization are occurring, along with associated increases in industrialization and vehicularization — resulting in shifting patterns of environmental risk. In this section, we discuss some of the major factors in Asia that are expected to most strongly influence the health effects of exposure to air pollution over the next 20 to 30 years: broad demographic trends, urbanization (growth of cities and trends in rural development), patterns of economic and social development vis-à-vis decisions regarding energy use, population health and the infrastructure of health care systems, and the intersection of air pollution, poverty, and health.

In parts of this section, we differentiate among five major Asian regions, roughly on the basis of geography and income level (Table 1). *India* and *China* (i.e., mainland China, Hong Kong, and Taipei,China) are each considered to represent one region, owing to their large geographic areas and population sizes. The other three regions considered are *Other South Asia*, comprising Afghanistan, Bangladesh, Bhutan, Maldives, Nepal, Pakistan, and Sri Lanka; *Other Southeast Asia*, comprising Cambodia, Indonesia, Laos, Malaysia, Myanmar (Burma), Philippines, Thailand, Timor-Leste, and Vietnam; and *High-Income Asia*, comprising Brunei, Japan, South Korea, and Singapore. This review is designed to present an overview of factors influencing the impact of air pollution on health across Asia. Although as units of evaluation the five regions capture some of the heterogeneous nature of Asia, clearly they do not capture all the differences among the regions across the continent or all important within-country differences.

Table 1. Definitions of Asian Regions Referred to in This Section

Region	Country
India	India
China	China, Hong Kong, Taipei,China
High-Income Asia	Brunei, Japan, South Korea, Singapore
Other South Asia	Afghanistan, Bangladesh, Bhutan, Maldives, Nepal, Pakistan, Sri Lanka
Other Southeast Asia	Cambodia, Indonesia, Laos, Malaysia, Myanmar, Philippines, Thailand, Timor-Leste, Vietnam

Broad Trends in Development

Several conceptual frameworks have been developed to describe broad trends in development over time. These include the demographic transition, the epidemiologic transition, and the environmental risk transition (see Figure 1).

The demographic transition is a conceptual framework that describes how changes in the rates of birth and death affect population size. In brief, increased development is expected to lead to decreased death rates, followed eventually by gradual declines in birth rates. The lag time between the two declines initially results in a large population increase. The lag time can be partially explained by the fact that fertility tends to be higher in regions of low development and poverty, where there tends to be a lack of access to education and birth-control methods; therefore, only after development has affected daily life do the measures limiting fertility take effect. Over time, continued decreases in the birth and death rates result in a gradual fall in the rate of population increase; the population may shrink and become increasingly older overall.

Unlike the demographic transition, which focuses only on births and deaths, the epidemiologic transition takes cause-of-death categories into account. Under this framework, increased development is associated with a shift in patterns of disease (and corresponding mortality). As rates of infectious diseases, infant and child mortality, and epidemics (type I diseases) decline, rates of chronic diseases (including cardiovascular and respiratory disease [type II diseases]), especially those affecting older populations,

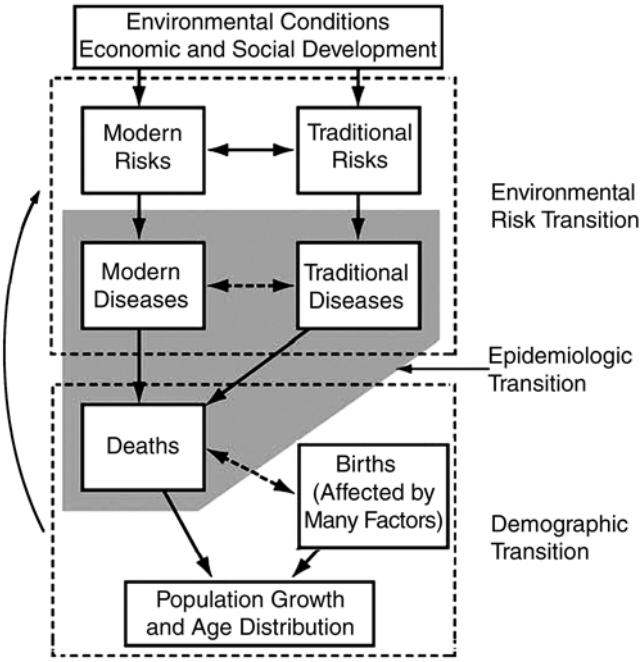

Figure 1. Integrated frameworks describing broad trends in development. The gray area designates the three main factors driving development. Solid arrows represent known relations; dashed arrows represent possible relations. See the text or Glossary for detailed descriptions of the three frameworks. [Adapted from the Annual Review of Environment and the Resources, Smith and Ezzati 2005.]

may take on proportionately greater importance because of changes in both age-specific death rates and population age structure. Thus, the epidemiologic transition can be thought to drive the demographic transition. Sidebar 2 describes the status and trends of respiratory and cardiovascular disease in Asia that underlie the demographic transition.

Similarly, the environmental risk transition focuses on changes in underlying environmental risk factors for disease and therefore may be a major trigger of current and future trends in disease patterns. Development often involves several processes — including urbanization, industrialization, agricultural modernization, and vehicularization. These tend to produce community exposures or potential environmental hazards or risk factors such as outdoor air pollution, solid and hazardous waste, lead exposure, and pesticide use.[a] It is worth noting, however, that exposures may increase but the associated population-level risk may decrease if disease cofactors such as health care reduce the potential effect or the size of the susceptible subpopulations. As

[a] Occupational risks seem to increase as a result of these hazards or risk factors as well, although the occupational hazards of farming in traditional, poor communities in rural areas are not well documented. Occupational risks in the informal unregulated sector of the urban economy are not well described either, although they are probably considerable.

Sidebar 2. CARDIOVASCULAR AND RESPIRATORY DISEASE IN ASIA

Short- and long-term exposure to air pollution is associated with morbidity and mortality among people with preexisting cardiovascular or respiratory disease, and limited evidence suggests that long-term exposure may also increase the risk of these conditions (see Sidebar 13). For that reason, it is important to understand the current levels of, and probable trends in, cardiovascular and respiratory disease and their major causes, such as tobacco smoking and diet, in the developing countries of Asia.

CARDIOVASCULAR DISEASE

Previously considered "diseases of affluence," cardiovascular diseases are now known to pose a serious health burden in developing countries (Ezzati et al. 2005; Yusuf 2001a,b; Goyal and Yusuf 2006; Reddy 2004; Abegunde 2007). Ischemic heart disease (IHD) and cerebrovascular disease were the first and second leading causes of death, respectively, in low- and middle-income countries in 2001, accounting for over 10 million deaths. Ischemic heart disease and cerebrovascular disease are estimated to have caused 23% and 20% of all deaths in 2001 in East Asia and the Western Pacific region and the South Asian region, respectively (Lopez et al. 2006).

Several major risk factors for cardiovascular disease are prevalent and increasing across the region. Developing countries in Asia are experiencing a rapid increase in the prevalence of diabetes mellitus and metabolic syndrome. Hypercholesterolemia and dyslipidemia are particularly prevalent among Indian adults; 37.4% of men aged 15 to 30 years are affected (Nishtar 2002). The prevalence of abdominal obesity and the consumption of a high-glycemic diet are increasing in both India and China, where they are contributing to increased risks of diabetes and cardiovascular disease (Goyal and Yusuf 2006; Ding and Malik 2008). The estimated percentage of men aged 15 to 30 years with hypertension is also high: 36.4% in India, 17% in Pakistan and Sri Lanka, and 9.8% in Bangladesh (Nishtar 2002). The trend among women is similar, although rates within countries may vary widely: Indian studies report a higher prevalence of hypertension in urban areas (20–40%) than in rural areas (12–17%). This pattern has been noted in Pakistan and Sri Lanka as well (Ghaffar et al. 2004).

The frequency of tobacco smoking remains high in the developing countries of Asia, with the prevalence in men far exceeding that in women. The reported prevalence of smoking among men in India in 1998–1999 was 29.4% and in Indonesia in 2001 it was 69%, with a prevalence of 2.5–3% among women in both countries. Economic data on tobacco consumption show declines in India and increases in Indonesia over the past 30 years, but these data are not always reliable indicators of actual tobacco use (Shafey et al. 2003).

Surveys conducted by the Chinese government from 2002 to 2003 reported smoking prevalence of between 49.6% and

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Sidebar 2. CARDIOVASCULAR AND RESPIRATORY DISEASE IN ASIA (*Continued*)

57.4% among males and between 2.6% and 3.0% among women. Since 1990, economic data have indicated declines in tobacco consumption, and national surveys have indicated declines in the reported prevalence of smoking, with rates declining from 60.3% to 49.6% among males and 4.7% to 3.0% among females (Lin HH et al. 2008).

A role of genetic factors in the high prevalence of cardiovascular disease in South Asians is suspected, but definitive evidence is lacking. Numerous studies have reported a high incidence of coronary artery disease (that is not explained by conventional risk factors) among Indian immigrants in Canada, the United Kingdom, the United States, and the Caribbean (Anand et al. 2000). A large case–control study of risk factors for myocardial infarction in 52 countries worldwide, include 5000 cases in East, Southeast, and South Asia, found that the same constellation of risk factors — including abnormal lipid levels, smoking, hypertension, and obesity — accounted for more than 90% of the attributable risk in all regions (Yusuf et al. 2004). Previously, South Asians were reported to have higher rates of myocardial infarction at younger ages than Europeans and Chinese. An analysis of 1700 cases of myocardial infarction in South Asia found that the cases occurring at younger ages were largely attributable to the same constellation of risk factors, just at an earlier age (Joshi et al. 2007).

CHRONIC RESPIRATORY DISEASE

The prevalence of chronic respiratory disease (including chronic obstructive pulmonary disease [COPD] and tuberculosis [TB]) in most Asian countries is quite high, and its current contribution to mortality is substantial. COPD and TB are estimated to have led to 15% of all deaths in the East Asia and Western Pacific region, and to 9% in South Asia, in 2001 (Lopez et al. 2006).

COPD is predicted to increase in importance as a cause of death in Asia in the next two decades because of the high prevalence of smoking, both in the past and present (Murray and Lopez 1997, Lin HH et al. 2008). COPD is estimated to affect 3% of Chinese in 2003; this percentage is expected to increase substantially to reflect an increase in smoking prevalence by a factor of 10 between 1950 and 1990 (Zhang JF and Smith 2003). COPD is estimated to affect 6.7% of the population over 30 years of age in 12 Asian Pacific countries (Regional COPD Working Group 2003). In India, 11 population-based studies suggest that COPD affects 5.0% of men and 2.7% of women over 30 years of age (Jindal et al. 2001). An asthma prevalence of 2.4% was reported in a four-city multi-center study in India (Jindal 2007).

Tobacco smoking increases the risk of TB, and there is increasing evidence that environmental tobacco smoke and indoor air pollution from the burning of solid fuels may do so as well (Bates et al. 2007; Lin HH et al. 2007). There is virtually no information on the effects of exposure to outdoor air pollution on the risk of TB (Cohen and Mehta 2007).

ALRI IN CHILDREN

Acute lower respiratory infection (ALRI) is the chief cause of death among children under 5 years of age worldwide, killing an estimated 3 million in 2001 (Lopez et al. 2006). Although the disease affects both young and old, pneumonia kills so many children younger than 2 years of age that the disease accounts for a large number of lost life-years. ALRI accounted for 6.3% of the global burden of disease in 2000, in terms of disability-adjusted life-years, making it arguably the most important single category of disease in the world (WHO 2001).

All young children worldwide appear to suffer similar rates of acute upper respiratory infection (Rudan et al. 2004). In developed countries, these infections are usually viral, mild, and self-limiting. In contrast, most serious cases of ALRI in developing countries are thought to be bacterial and consequently treatable by antibiotics. If untreated, these bacterial infections may progress rapidly, and a large fraction of cases progress to serious and sometimes fatal ALRI, consisting of bronchiolitis and pneumonia.

There are several known risk factors for ALRI, the most important of which is malnutrition. The WHO Comparative Risk Assessment exercise (WHO 2002) estimated that some 40% of ALRI cases are directly attributable to protein malnutrition in the world and 16% of cases are due to zinc deficiency (Black 2003). Other risk factors include those that promote diarrhea, malaria, and measles, because children with those diseases are more likely to contract severe ALRI. Crowding and chilling have also been associated with ALRI, but with less consistency. Exposure to high concentrations of indoor air pollution from the burning of solid fuels increases the risk of pneumonia by 80% (Dherani et al. 2008). As a result, nearly one third of global ALRI cases in children is attributed to indoor air pollution in developing countries (Smith et al 2000a). About 1% is attributed to urban outdoor air pollution, though the epidemiologic evidence for this association is considerably weaker (WHO 2002; Romieu et al. 2002).

Vaccines show increasing promise of preventing serious cases, especially if access to vaccinations is ensured for people living in poverty, but prevention through better nutrition and cleaner environments will also be essential to substantially reduce the incidence of ALRI and its associated mortality (Mulholland et al. 2008).

countries modernize further and environmental controls are tightened, community-level exposures tend to decline. This decline leads to the third stage of the environmental risk transition, in which the richest countries contribute most to global risks that are due to greenhouse gas (GHG) emissions and other causes of global environmental change.

Overall, exposures leading to potential environmental health risks generally seem to decline with economic development, both in absolute and relative terms. Household exposures are considerable in poor areas and decline with increasing development; community exposures first rise, as modern industrial economic development begins,

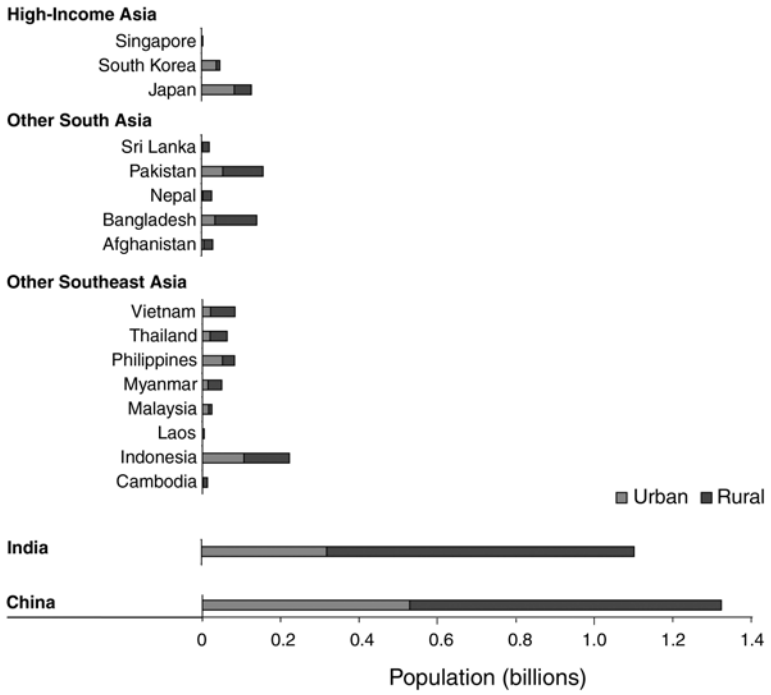

Figure 2. Urban and rural populations in selected Asian countries in 2005. [Data compiled from United Nations Population Fund 2007.]

and then decline (Smith and Ezzati 2005). Residents of urban areas living in poverty, who are particularly numerous in Asia, experience considerable risk from both community and household exposures (Smith 1990). Such populations are likely to have a larger environmental burden of disease, and a larger portion of their entire disease burden due to environmental risk factors, than do populations living in the more developed regions (Smith et al. 1999).

Figure 1 illustrates the integrated nature of the demographic transition, the epidemiologic transition, and the environmental risk transition. Although these frameworks can be used to aid the understanding and management of environmental risks, there can be significant local departures from the overall trends predicted, because the processes driving these trends are dynamic and interrelated.

Changes in Population Age Structures

Population sizes in 2005 in major countries in Asia are provided in Figure 2 by country; other than China and India, countries are grouped into the three major Asian regions: High-Income Asia, Other South Asia, and Other Southeast Asia. About half the world lives in Asia, and approximately 80% of that half lives in India, Indonesia, or China. Evidence of a demographic transition can be seen in population projections for India, Indonesia, and China in Figure 3. At the earlier stages of development, the countries are characterized by a "youth bulge" (greater percentages of the population having younger ages) in their demographic profile. Over time, as the younger population ages and as advances in health care and public health infrastructure promote increased longevity of the older population, the population becomes more evenly distributed across age groups.

Status of and Trends in Disease Burden

In the developing countries of Asia, the demographic and epidemiologic trends discussed above are reflected in the burden of disease in terms of numbers of deaths in a given year and lost years of healthy life (expressed as disability-adjusted life-years [DALYs]) (Table 2). The burden of disease from communicable diseases, malnutrition, and maternal conditions (e.g., those related to childbearing, such as from obstructed labor) or perinatal conditions persists across Asia, but at the same time, the burden of disease from chronic noncommunicable diseases (such as hypertension, diabetes, ischemic heart disease [IHD], and cancer) is both substantial and increasing, owing to a variety of factors but most importantly to the increased prevalence of tobacco smoking, as well as increasing rates of obesity and changes in dietary patterns (Yusuf et al. 2001b; WHO 2002; Ding and Malik 2008). Indoor air pollution resulting from domestic use of solid and fossil fuels is widespread, particularly in rural areas and urban slums, where it accounts for a large fraction of cases of several diseases (including respiratory infections and chronic obstructive

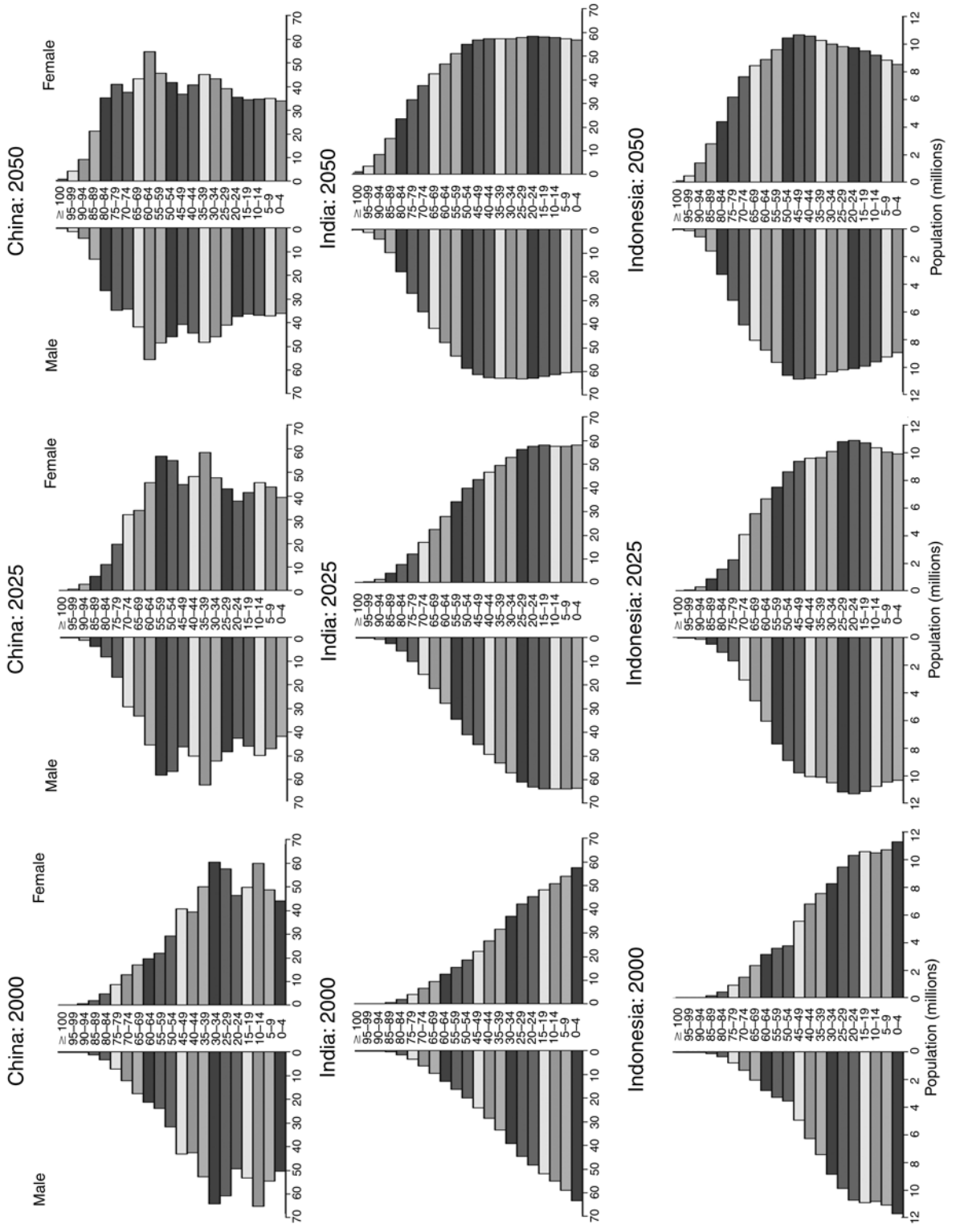

Figure 3. Past and projected population pyramids stratified by age groups for China, India, and Indonesia: 2000, 2025, and 2050. [Adapted from U.S. Census Bureau 2008.]

Table 2. Deaths and DALYs in Asia in 2004, by WHO Region and Cause[a]

	Region[b]			
	Southeast Asia	East Asia	South Asia	World
Population (thousands)	304,830	1,581,888	1,367,074	6,436,826
Total deaths (thousands)	2,741	10,995	12,539	58,772
Total DALYs (thousands)	70,721	248,631	372,258	1,523,259
Cause of Death	Number of Deaths (%) Number of DALYs (%)[c]			
Communicable diseases, malnutrition, and maternal or perinatal conditions	659 (24.0) 19,644 (27.8)	1,432 (13.0) 47,417 (19.1)	4,977 (39.7) 165,233 (44.4)	17,971 (30.6) 603,993 (39.7)
Lower respiratory infections	168 (6.1) 2,405 (3.4)	346 (3.1) 5,282 (2.1)	1,245 (9.9) 25,916 (7.0)	4,177 (7.1) 94,511 (6.2)
Noncommunicable diseases	1,453 (53.0) 35,476 (50.2)	8,452 (76.9) 168,711 (67.9)	6,242 (49.8) 159,809 (42.9)	35,017 (59.6) 731,652 (48.0)
Malignant neoplasm	301 (11.0) 3,533 (5.0)	2,023 (18.4) 22,002 (8.8)	894 (7.1) 10,606 (2.8)	7,424 (12.6) 77,812 (5.1)
Tracheal, broncheal, or lung cancer	51 (1.9) 492 (0.7)	425 (3.9) 3,980 (1.6)	103 (0.8) 1,007 (0.3)	1,323 (2.3) 11,766 (0.8)
Diabetes mellitus	81 (3.0) 1,556 (2.2)	192 (1.7) 4,087 (1.6)	199 (1.6) 3,336 (0.9)	1,141 (1.9) 19,705 (1.3)
Cardiovascular disease	642 (23.4) 6,646 (9.4)	3,708 (33.7) 29,385 (11.8)	3,233 (25.8) 35,415 (9.5)	17,073 (29.0) 151,377 (9.9)
Ischemic heart disease	277 (10.1) 2,940 (4.2)	898 (8.2) 7,076 (2.8)	1,734 (13.8) 18,643 (5.0)	7,198 (12.2) 62,587 (4.1)
COPD and asthma	131 (4.8) 2,111 (3.0)	1531 (13.9) 14,530 (5.8)	791 (6.3) 11,712 (3.1)	3,312 (5.6) 46,513 (3.1)
Injury	629 (22.9) 15,601 (22.1)	1,112 (10.1) 32,503 (13.1)	1,320 (10.5) 47,217 (12.7)	5,784 (9.8) 187,614 (12.3)

[a] Data compiled from WHO 2008.

[b] For the following WHO regions: "Southeast Asia" corresponds to Southeast Asian Region B; "South Asia" to Southeast Asian Region D; and "East Asia" to Western Pacific Region B.

[c] Data are shown as the number of deaths and number of DALYs from a given cause of death, each followed by the corresponding regional percentage, which is calculated as the number of deaths or DALYs divided by the total number of deaths or DALYs for the region, multiplied by 100.

pulmonary disease [COPD]), especially among young children and women. High concentrations of outdoor air pollution from stationary and mobile sources also contribute considerably to the burden of disease from disorders of the cardiovascular and respiratory systems (WHO 2002).

Trends in Environmental Risks

In this section, we describe trends in household- and community-level environmental exposures that illustrate the environmental risk transition in Asia. As described above, household environmental exposures generally decline with increased development, whereas community-level exposures increase initially and then gradually decline. The distinction between household and community exposures may depend on whether the setting is urban or rural. For example, in urban areas, the use of solid fuel may contribute to both household and community exposures.

Household Environmental Risks Household risks, such as use of solid fuel[b], unsafe drinking water, and inadequate sanitation dominate the environmental burden for people living in poverty. Because infrastructure is easier to establish and sustain in urban areas than rural areas, urban areas benefit sooner and often to a greater extent, particularly in

[b] Household use of solid fuel is a common indicator of indoor air pollution in developing countries, where cooking and heating with solid fuels is generally done with inefficient stoves and inadequate ventilation. See *Household Use of Solid Fuels* in Section II.

poorer developing countries, from decreases in household environmental risks associated with increased economic development.

Figure 4 and Figure 5 provide a cross-sectional view of household environmental risks in Asian countries. The patterns shown are not representative of longitudinal patterns in environmental risk, but they do suggest that substantial declines in household use of solid fuel, inadequate sanitation, and unsafe drinking water are associated with increased income. The difference in the environmental risks in rural and urban households is striking; most countries with average annual per capita incomes of more than U.S. $5000 have eliminated a large proportion of the household environmental risk in urban areas but not rural areas. This is consistent with the notion that people living in poverty, in both rural and urban areas, tend to bear a larger burden of environmental risk.

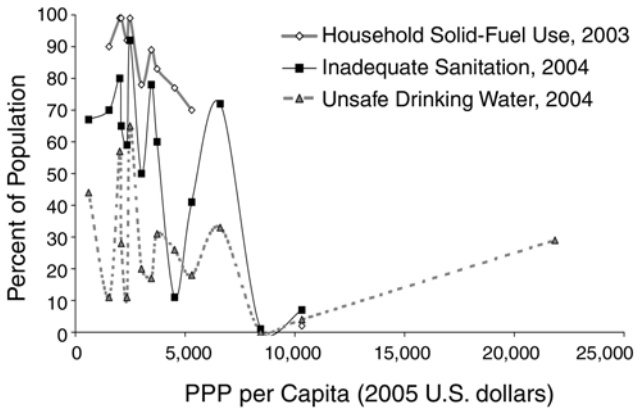

Figure 4. Trend of environmental risks in rural Asia, according to household income. PPP is purchasing power parity, which is the GDP adjusted by local prices and normalized to U.S. conditions. [Data compiled from WHO 2007b.]

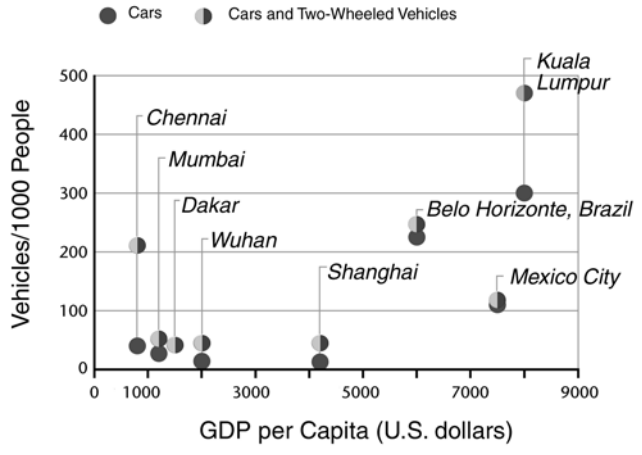

Figure 6. Motorization rates, according to per capita income. [Adapted from World Business Council for Sustainable Development 2004.]

Community-Level Risks Motor-vehicle ownership is one indicator of the community-level risk associated with air pollution. Consistent with the environmental-risk-transition framework, motor-vehicle ownership plotted against income yields a roughly S-shaped curve. Ownership increases very slowly until it approaches a threshold, at which point there is a sudden increase and then gradual attenuation. A cross-sectional comparison of developing countries suggests that numbers of motorized vehicles increase as the gross domestic product (GDP) per capita increases (Figure 6). The data differ by type of motor vehicle; two-wheeled motor vehicles, which may (depending on the type of engine) have higher emissions than automobiles, tend to be more affordable and generally are associated with a lower income threshold at which the rate of ownership rises. One example of this is the relation of income to vehicle ownership in Chennai, India, in 1993 (Figure 7). More detailed information on motor-vehicle

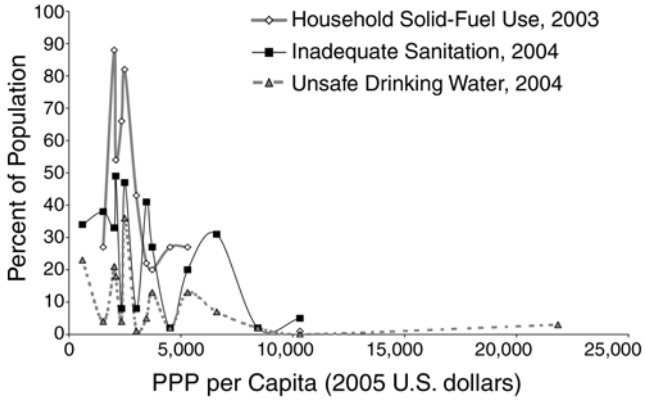

Figure 5. Trend of environmental risks in urban Asia, according to household income. PPP is purchasing power parity, which is the GDP adjusted by local prices and normalized to U.S. conditions. [Data compiled from WHO 2007b.]

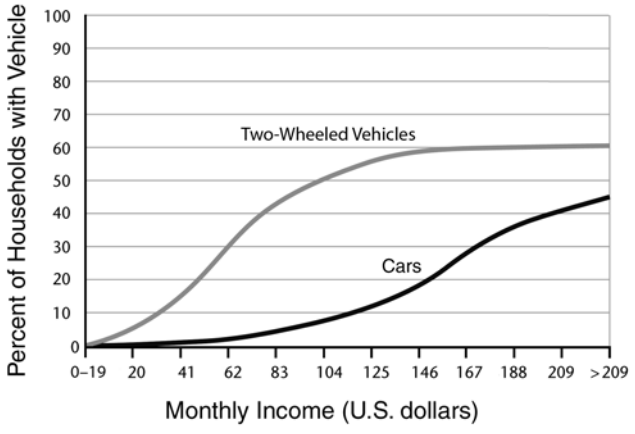

Figure 7. Motor vehicle ownership in Chennai in 1993, according to monthly income. [Adapted from World Business Council for Sustainable Development 2004.]

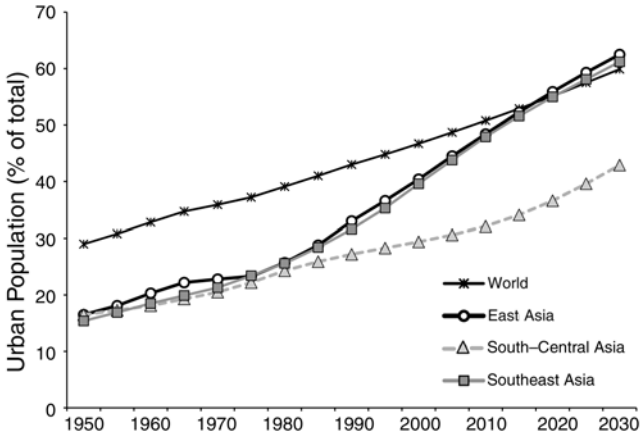

Figure 8. Urban populations in the world and various regions of Asia, including future projections. [Data compiled from United Nations Economic and Social Affairs 2006.]

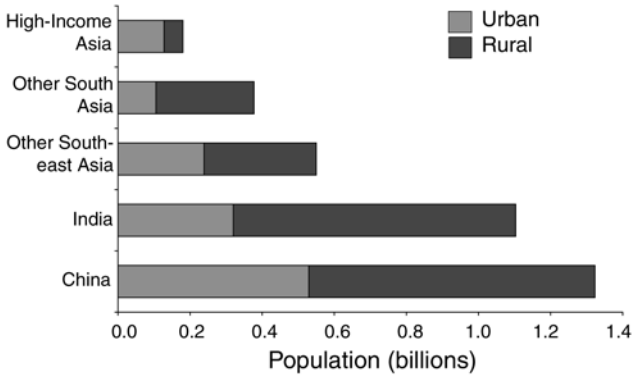

Figure 9. Urban and rural fractions of the populations in Asia. Regions are defined in Table 1. [Data compiled from WHO 2007b.]

ownership in Asia is provided later in Historic Patterns and Trends in Vehicle Production and Use of Motor Vehicles in Section II.

Increased motor-vehicle ownership is also associated with increased road-traffic fatalities. Projections suggest substantial increases in motor vehicle–related fatalities, especially in Asia (Mathers and Loncar 2006).

Urbanization

Urbanization is a major factor driving the environmental risk transition. As countries develop, there is a tendency for increased urbanization. Several years ago it was projected that by 2008, more than half of the world's 3.3 billion people would be living in cities (United Nations Economic and Social Affairs 2006; Figure 8). This population shift is especially pronounced in Asia, where the urban population is expected to double in the next 22 years, from 1.36 billion to 2.64 billion. Figure 9 depicts current levels of urbanization in the five major Asian regions (see Table 1). It is projected that by 2030, Asia's urban population will make up over half the world's urban population.

Patterns of urban growth differ around the world (United Nations Population Fund [UNFPA] 2007). Though urban growth is most often attributed to migration from rural to urban areas, natural increases in the urban population and reclassification of city boundaries also contribute to the increase in city populations. For example, in India, natural increase is the primary factor driving urban growth. In contrast, migration is the main factor driving urbanization in many Chinese cities, where population growth is tightly regulated. Sidebar 3 describes various patterns of urbanization in several Asian cities.

Sidebar 3. CITIES IN TRANSITION

SHANGHAI, CHINA

In 2004, Shanghai was estimated to have a population of 17.8 million people (Asia Pacific Energy Research Centre 2007; National Bureau of Statistics of China 2004). This is about 1.3% of China's total population of 1.3 billion. The city itself covers an area of about 6340 square kilometers, which is approximately 0.06% of China's total area. Consequently, the population density of Shanghai is quite high, with 2657 people per square kilometer (United Nations Economic and Social Commission for Asia and the Pacific 2008).

Shanghai was one of the first cities in China to experience a negative natural growth rate, and this trend continues to the present. However, the total population of Shanghai is still increasing, owing to migration of people into the city. In 1997, migration caused a net increase of about 3.6 people per 1000 population. In addition to migrants who permanently settle in Shanghai, the municipality also has a large floating population (defined as a population without household registry) that includes migrant workers and tourists.

A low birth rate, coupled with an increased life expectancy, has led to an aging population in Shanghai. In 2000, just 12.91% of Shanghai's population was below the age of 14 years, the lowest percentage of any Chinese city. However, Shanghai also had the highest proportion of people over the age of 65 years, 11.53%, which is approximately 1.93 million people.

The increase in life expectancy is due to the decrease in mortality rates. Since 1949, the mortality rate in Shanghai has been steadily decreasing to a level of about 6.2–6.7 deaths per 1000 people in 1990. However, with the aging population

Continued on next page

Sidebar 3. CITIES IN TRANSITION
(Continued)

Shanghai has also been experiencing a trend toward the decentralization of population, employment, and industrial output since 1990 (United Nations Human Settlements Program 2004). Factors said to be contributing to this trend include high taxes, crime, congestion, and low environmental quality within the city, as well as the lower land taxes and development costs in the outskirts of the city. The improvement of transport systems has also allowed for the trend toward peri-urbanization.

Increased decentralization and increased urban growth have fueled the need for more private automobiles. Currently, China is the world's second largest automobile market and the world's third largest automobile manufacturer, home to plants of almost every car manufacturer in the world. In Shanghai alone, the number of private automobiles increased from 10,000 to 250,000 in just 8 years.

HONG KONG, CHINA

Hong Kong's population has been rising steadily for about the last half century, tripling from 1950 to about 2001. Since then, however, the rate of population growth has declined. The current annual growth rate is estimated to be about 0.9%, which from 2005 to 2006 represented the addition of 62,900 people, for a total of 6.9 million. By 2031, the population is expected to equal about 8.72 million.

Underlying much of the decrease in population growth is a decrease in the fertility rate among Hong Kong women. This decline has been attributed to improved socioeconomic conditions, increased numbers of women in the workforce, and a trend toward marrying and raising children outside of Hong Kong. The decreasing fertility rate coupled with a decreasing death rate has led to the aging of the population. It is estimated that by 2031, a quarter of Hong Kong's population will be over the age of 65 (Figure 3.1) and the number of those over the age of 85 will triple (Census and Statistics Department 2010).

Hong Kong's decreasing growth rate, which contrasts strongly with many cities in mainland China, is due not only to the decreasing fertility rate but also to Hong Kong's strict immigration policy. This policy, termed the one-way permit scheme, restricts immigration into Hong Kong to 150 people per day. This limit has remained unchanged since its establishment in 1995. However, immigration still accounts for 93% of the population growth.

BANGKOK, THAILAND

Bangkok's population has been steadily growing for the last half century and is expected to continue to do so. However, its portion of Thailand's total urban population is declining because the growth rate in smaller urban areas is increasing while Bangkok's growth rate is decreasing. From 1950, the population growth rate of Bangkok has halved. Yet Bangkok is still the largest city in Thailand, with a population of about 8.1 million people.

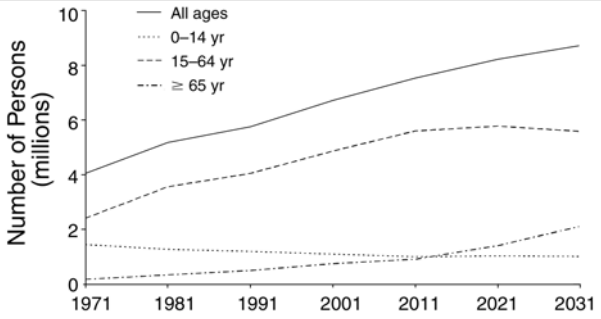

Figure 3.1. Trends in the population of Hong Kong, 1971–2031. [Data from Hong Kong Annual Digest of Statistics, 2002 Edition; Hong Kong Monthly Digest of Statistics, November 2002; and Projected Mid-Year Population by Age Group and Sex for 2002–2031, Census and Statistics Department (Government of Hong Kong Special Administrative Region).]

Bangkok's inner-city population density decreased over 1970–1998, while the fringes of the city increased in density. A study by Murakami and colleagues (2005) using various urbanization models classified Bangkok as being on the brink of suburbanization.

CHENNAI, INDIA

Chennai is the fourth largest city in India, with an estimated 7.5 million people and a population density of about 5847 people per square kilometer in 2006; in the same year, the urban population of Chennai was estimated at 4.3 million. In recent years, Chennai's growth rate has slowed to about less than 1% (World Bank 2005).

The population of the Chennai slums has been particularly well characterized. A 2001 population study of Chennai classified about 0.8 million people as living in slums, making up approximately 22% of the city's population at that time (Census of India 2001). The Chennai residents living in slums accounted for 5% of India's total slum population in 2001. Just 2 years later, in 2003, the slum population in Chennai was estimated at 1.08 million people, accounting for over a quarter of the city's total population (Chandramouli 2003). A survey of Chennai housing in areas classified as slums showed that 64% of the houses are permanent, with approximately 40% of these rented out rather than owner-occupied. Researchers believe this combination of permanent homes being rental property is evidence of the presence of landlords who profit from but do not maintain their properties (Chandramouli 2003). Most of the housing consists of one-room tenements, suggesting a severe lack of space. Of the more than 1 million people living under such conditions in Chennai, only 26% have access to drinking water near their homes.

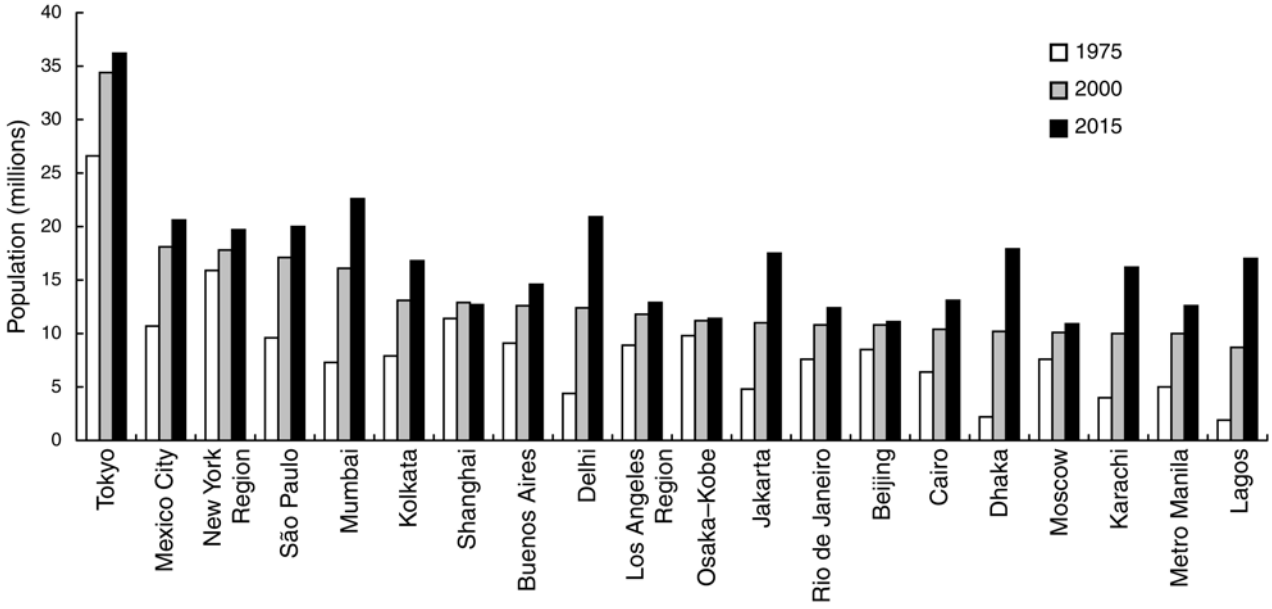

Figure 10. Populations of various megacities (> 10 million inhabitants) worldwide, including future projections. Megacities are ordered according to their population size in 2003. [Adapted from United Nations Economic and Social Affairs 2004b.]

City Size Megacities are cities with a population over 10 million. As of 2000, over half the 20 megacities in the world were in Asia: Tokyo, Mumbai, Delhi, Shanghai, Kolkata, Jakarta, Dhaka, Osaka–Kobe, Karachi, Beijing, and Manila (United Nations Economic and Social Affairs 2004b). Six of these megacities are among the top 10 largest cities in the world. Although Tokyo is currently the only city in Asia with more than 20 million residents (i.e., a *metacity*), it is projected that Asia will have five metacities by 2020 (United Nations Human Settlements Programme [UN-Habitat] 2006a) (see Figure 10).

Despite the large size of megacities, they are home to only about 4% of the world's urbanites (UN-Habitat 2006b). Smaller towns and cities have rapidly increasing populations and account for the bulk of the trend in urban growth. The majority of people undergoing the shift from rural to urban areas come from the smaller cities and towns in the poorer developing world. About 53% of the worldwide urban population lives in cities with < 500,000 residents and another 22% live in cities with 1–5 million residents. Moreover, despite the rapid growth in urban populations, Asia continues to have a substantial rural population. As of 2005, 71% of all rural dwellers lived in Asia.

Urban Land Use Urban sprawl can be categorized as residential suburbanization or peri-urbanization. Residential suburbanization refers to planned growth consisting mostly of housing for single families in the middle to upper classes built at low density away from the city center. This suburbanization can lead to increased commuting and associated increases in energy use and vehicular pollution. Peri-urbanization refers to the unplanned expansion of economic activities into the areas surrounding an urban center; this occurs mostly in the transitional zones between urban and rural boundaries. In Asia, peri-urbanization is often characterized by the incorporation of adjacent rural areas or small towns into larger urban settlements. Peri-urbanization is often associated with disorderly growth and lack of regulation, followed by environmental degradation.

While urbanization in Asia is increasing, factors such as decreases in family size and increased car ownership are contributing to declining urban density and greater urban sprawl and suburbanization. For example, from 1989–2001, while the Kuala Lumpur metropolitan area experienced a near-doubling in its built environment (the man-made structures in an environment) and an increase in its urban population from 2.7 million to 5 million, the city's population density declined from 7130 to 6160 people per square kilometer (Leather 2008). If Asia follows a Western pathway of suburbanization, increased sprawl may result in increased formation of ozone (O_3), emissions of nitrogen oxides (NO_x) and carbon dioxide (CO_2), and more regional air pollution.

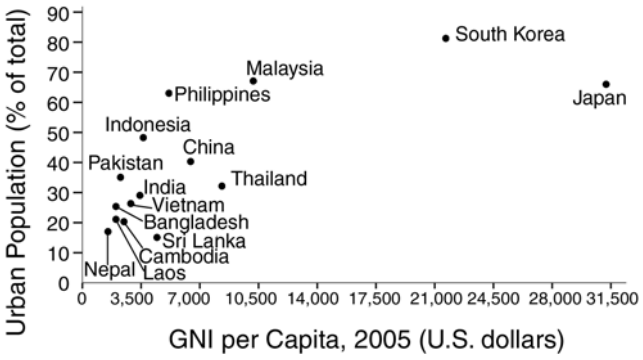

Figure 11. Degree of urbanization according to gross national income (GNI) per capita in various Asian countries in 2005. [Data compiled from WHO 2007b.]

A cross-sectional sampling of Asian countries shows that increases in income are associated with a larger proportion of the population living in urban areas (Figure 11). This is particularly true at the lower end of income and urbanization scales. Once approximately half the population in a given Asian country lives in urban areas, the degree of wealth in that country ranges widely.

While increased economic growth is associated with increased urbanization, the majority of urban population growth in developing countries occurs among people living in poverty and results in increases in the number of informal settlements, called "slums," "shantytowns," or "squatter housing." A "slum household" is defined by the UN-Habitat as "a group of individuals living under the same roof in an urban area who lack one or more of the following: durable housing, sufficient living area, access to improved water, access to sanitation and secure tenure" (UNFPA 2007). Worldwide, there are over 1 billion people living in "slum households," accounting for about one sixth of the total global population. The United Nations estimates that in 2001, 554 million people in Asia lived in such households — more than half the urban population — with 262 million in South Asia alone (UN-Habitat 2003).

Air Pollution, Poverty, and Health

There is emerging evidence, largely from studies in Europe and North America, that economic deprivation increases the magnitude of air pollution–related morbidity and mortality (Krewski et al. 2000). There are two major reasons why this may be true (O'Neill et al. 2003): First, economically disadvantaged communities are exposed to higher concentrations of air pollution; and second, because of poorer nutrition, less access to medical care, and other factors, people in these communities experience more health impact per unit of pollution exposure (i.e., are more vulnerable to health effects). In addition, air pollution could exacerbate the conditions of poverty. The relative impact of air pollution has been observed to be modified by group or neighborhood characteristics, such as living in slums (Gouveia et al. 2004) or living in highly polluted areas (Jerrett et al. 2004). At the same time, because of the challenges of explaining the observed group-level effects, individual estimates of exposure and socioeconomic position continue to be used in analyses.

Previous studies using characteristics of individuals to assess changes in the magnitude of the air pollution effect estimate (i.e., effect modification) by socioeconomic position suggest that a low level of education (Gouveia et al. 2004; Jerrett et al. 2004) and low family income (Gouveia et al. 2004) are associated with increased health effects that are related to air pollution. Evidence also suggests that group-level indicators, such as residence in a poor neighborhood, could be risk factors above and beyond the socioeconomic position of an individual (Malmström et al. 1999). This finding underscores the importance of using analyses that include hierarchical models that integrate individual and group-level indicators of socioeconomic position.

The public health and social policy implications of the relations among health, air pollution, and poverty are likely to be important, especially in areas such as Asia, where air pollution concentrations are high and many live in poverty. However, the interaction between poverty and the health effects of air pollution has been studied little in developing countries in general and in Asia in particular. Therefore, results from Western studies can only be extrapolated with considerable uncertainty (Cohen et al. 2004). The composition and relative contribution of indoor and outdoor sources of exposure are likely to be very different in the West and in Asia, and the impacts of exposure, as well as the influence of economic deprivation on those impacts, may be greater in Asia. To help address this issue, HEI's PAPA program, with support from the Asian Development Bank, conducted a study of air pollution, poverty, and health in Ho Chi Minh City, Vietnam, with results to be published in 2011 (see Sidebar 4).

SOURCES

To place air pollution in Asia in the context of our existing knowledge, it is helpful to describe historical patterns of air pollution associated with development. Air pollution has traditionally been associated with reduced atmospheric visibility and increased mortality and morbidity. For example, during a pollution episode in London in December 1952, the concentration of British smoke (an indicator of particulate matter [PM]) and sulfur dioxide (SO_2) exceeded 1000 μg/m^3 and there were thousands of

Sidebar 4. THE PAPA STUDY IN VIETNAM

With extensive numbers of the world's lowest-income people living in highly polluted areas of Asia's cities, increased effects of air pollution on the health of these populations would have large impacts on both public health and relevant policy. However, few studies of the interaction between poverty and the health effects of air pollution have been conducted in developing countries in general and in Asia in particular. The ability to conduct such studies is currently compromised by the relative lack of reliable and easily accessible data on health outcomes, routinely collected air quality data, and opportunities for collaboration between the health and environment sectors. To date, no studies of this type have been conducted in the poorest Southeast Asian countries: Laos, Cambodia, and Vietnam.

In cooperation with the Asian Development Bank, HEI's PAPA program, and the Ho Chi Minh City People's Committee, an interdisciplinary team of local and international experts launched a unique collaboration to assess the health effects of air pollution among residents of Ho Chi Minh City who live in poverty. The project consists of a hospital-based assessment of the effect of short-term exposure to air pollution on the respiratory health of young children, as well as a household-based characterization of exposures of people living in poverty to multiple sources of pollution and an assessment of the correlation between ambient concentrations and personal exposures.

The hospital component of the study estimated the effects of short-term exposure to air pollution on hospital admissions for ALRI in young children (< 5 years) in Ho Chi Minh City. It also compared the magnitude of the effect of air pollution, particularly PM_{10}, on children living in poverty with that among children who do not live in poverty, using individual-level and group-level indicators of socioeconomic status.

The ambient monitors used to assess exposure in the hospital component of the study would not capture data that could show whether economically disadvantaged children experience higher actual exposures to air pollution than children who are not economically disadvantaged. Therefore, the collaborators acknowledged the need to assess the extent to which localized sources may contribute to error in the measurement of exposure arising from the use of ambient monitoring data to estimate health impacts, particularly in subgroups.

One objective of the household component of the study is to assess determinants of personal exposure for each economic group and to explore whether the use of ambient monitors as a surrogate for personal exposures results in exposure misclassification across socioeconomic strata.

The broader objective of the household study is to characterize the exposures of residents of poor households in Ho Chi Minh City to air pollution from multiple sources. Using monitoring and modeling-based approaches, daily average PM_{10} concentrations from ambient monitoring sites (Figure 4.1) are being compared with repeated measures of personal exposures of residents from various socioeconomic groups residing close to the ambient monitors. Household surveys are being integrated with repeated (longitudinal) measurements of personal exposures to provide a better assessment of how exposure patterns vary by age, sex, and socioeconomic status.

By comparing more precise estimates of individual personal exposure with estimates based on ambient monitoring data, the presence of systematic daily differences in the major

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excess deaths per day (Brimblecombe 1987; Bell and Davis 2001). This combination of pollutants, called sulfurous or London smog, has largely disappeared from London and other cities in developed countries, because of the enactment of clean-air legislation that led to the marked decrease in or outright elimination of the use of "dirty" coal. In many Asian cities, however, uncontrolled combustion of coal and other dirty fuels still occurs on both domestic and industrial scales, resulting in pollutant concentrations similar to those observed during London's sulfurous smog episodes of the 1950s.

For example, the 95th-percentile values of the daily average concentrations of total suspended particles (TSP) ranged from 493 to 1280 $\mu g/m^3$ in the Chinese cities of Chongqing, Guangzhou, Lanzhou, and Wuhan during 1993–1996 (Qian ZM et al. 2001). The air pollution sources contributing to such TSP concentrations represent an opportunity for relatively straightforward actions to improve air quality in Asia, following on the efforts in North America and Western Europe that have been undertaken over the past 50 years. Developing countries in Asia have the potential to benefit from existing air pollution control technologies and previous experience with air quality management programs to improve air quality more rapidly than developed countries did. Of great relevance to Asia is that the North American and Western European experience demonstrates that air quality can be improved during periods of rapid economic development.

Another important pollutant is photochemical smog or Los Angeles smog, as it was hypothesized to be a component of the air pollution problem in Los Angeles in 1954. Photochemical smog is formed by complex chemical reactions of hydrocarbons and NO_x in the presence of solar radiation. Secondary pollutants that arise from these photochemical reactions include O_3 and other oxidants such as peroxyacetyl nitrate, sulfuric acid, nitric acid,

Sidebar 4. THE PAPA STUDY IN VIETNAM (*Continued*)

sources and levels of exposure across the spectrum of socioeconomic status can be assessed. This information will suggest the extent to which ambient monitoring data reflect personal exposures among subpopulations. As such, it will aid in the interpretation of the results from the hospital component, as well as the design of future epidemiologic studies of air pollution, poverty, and health in Ho Chi Minh City.

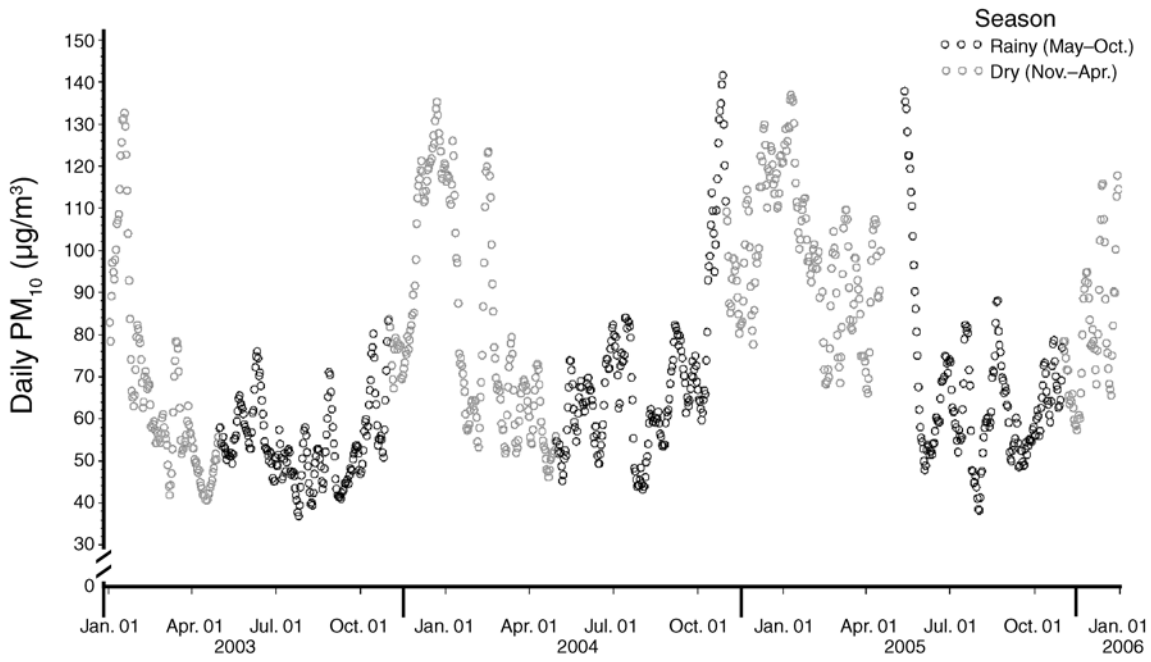

Figure 4.1. Average daily PM_{10} concentrations in Ho Chi Minh City, 2003–2005. The mean lag time was 1 to 7 days. [Source: Collaborative Working Group on Air Pollution, Poverty, and Health in Ho Chi Minh City 2009 (Draft Investigators' Report submitted to HEI).]

sulfate (SO_4^{2-}) and nitrate (NO_3^-) particles, and oxidized organic compounds. Automobile emissions are mainly responsible for the photochemical smog observed in Los Angeles. The number of automobiles has increased around the world, especially in Asian developing countries in recent years. Thus, photochemical smog now is commonplace in many metropolitan areas of Asia (e.g., Beijing, Guangzhou, Delhi, and Bangkok) (Oanh and Zhang 2004; Song Y et al. 2007; Xu J et al. 2008). Photochemical smog is not limited to urban centers, but rather spreads throughout larger areas (Sidebars 5 and 6). Transport processes allow O_3 and PM with an aerodynamic diameter $\leq$ 2.5 µm ($PM_{2.5}$) to travel great distances (i.e., more than thousands of kilometers) away from their sources or the sources of their precursors (U.S. Environmental Protection Agency [U.S. EPA] 1993).

The magnitude and duration of periods of overlap between sulfurous and photochemical smog may be much larger in cities in developing countries today than in Western cities historically, owing to the presence of sources that contribute to both types of pollution. For example, locations in which high-sulfur diesel fuel is in use also have considerable emissions of primary SO_4^{2-}, and SO_2 (Santoso et al. 2008). Thus, motor vehicles may simultaneously contribute to both sulfurous and photochemical smog.

Sidebar 5. THE PEARL RIVER DELTA

The Pearl River Delta, located mainly in China's Guangdong Province, covers an area of approximately 10,000 square kilometers and extends to Hong Kong and Macao (Figure 5.1). The Pearl River Delta has one of the highest population densities in China, accounting for approximately 38% of the total population (i.e., 83 million people) of Guangdong Province. The total population in the delta is estimated to be 39 million people, including about 7 million in Hong Kong and 0.4 million in Macao.

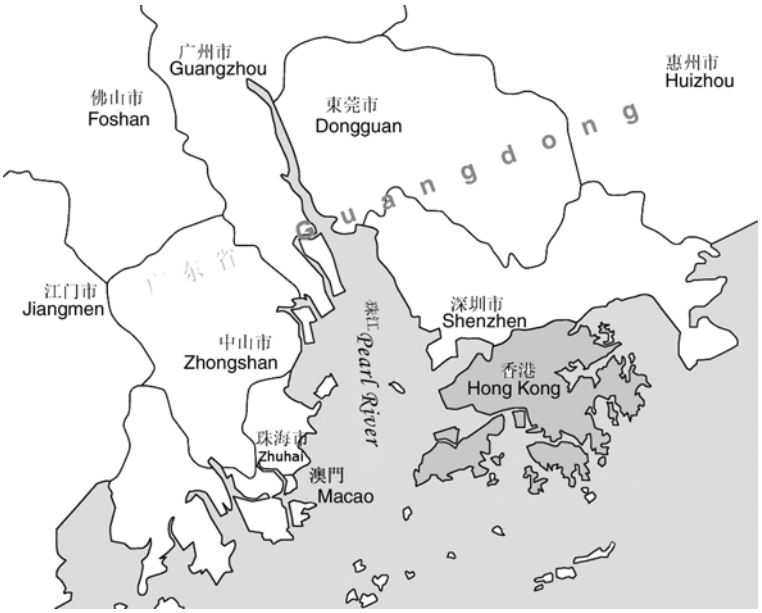

Figure 5.1. Area and cities surrounding the Pearl River Delta. [Reprinted with permission from Croquant 2007 under license from *http://creativecommons.org/licenses/by/3.0/deed.en*.]

Before 1985, the Pearl River Delta area (excluding Hong Kong and Macao) was one of the main agricultural bases in China, with a GDP of U.S. $8 billion in 1980. The Pearl River Delta was the first region to become open to free-market economic reform and thus experienced rapid economic growth. In 2005, the region's GDP was approximately U.S. $232 billion. The Pearl River Delta region is now the largest base of the manufacturing industry in China, employing more than 11 million workers in 2003, while still remaining one of the main agricultural bases for rice production. The growth in the urban and industrial sectors has been dramatic and has led to increased overall pollutant emissions as well as a greater spatial distribution of emissions (Figure 5.2).

The Pearl River Delta has a subtropical climate, because the mountains in the north prevent cold air from coming from that direction. South of the region lie the South China Sea and the Pacific Ocean; thus the climate of Pearl River Delta is affected by seasonal monsoons. In the winter, prevalent winds are from the north; and in the summer, winds are mainly from the south. The region has long daylight hours (up to 14 hours in summer and 11 hours in winter) with 1900–2200 hours of daylight per year on average and an average annual temperature of > 20°C. This climate favors the formation of photochemical smog; O_3 concentrations in Hong Kong tend to be higher in winter owing to cloud cover in the summer (wet) season.

The rapid urbanization and industrialization in the Pearl River Delta have caused heavy air pollution, which is receiving increasing attention from the public and the government. Numerous air pollution studies have been conducted to characterize air pollution in the region. All these studies reported high concentrations of PM and O_3. For example, in Shenzhen in 2004, the 24-hour average summer $PM_{2.5}$ and PM_{10} concentrations were 35 μg/m^3 and 57 μg/m^3, respectively, and winter $PM_{2.5}$ and PM_{10} concentrations were 99 μg/m^3 and 137 μg/m^3, respectively (Niu et al. 2006). In Guangzhou, the summer 24-hour average concentration of $PM_{2.5}$ was 97.5 μg/m^3 (Wang XH 2006). An analysis of air quality in Guangzhou from 1981 to 2005 reported that annual mean SO_2 concentrations fluctuated above and below the limit of the 60 μg/m^3 established as the Chinese secondary air quality standard. The same analysis showed that annual mean NO_x concentrations were higher than the Chinese secondary standard for NO_x (50 μg/m^3) in all years studied and even exceeded the tertiary standard (100 μg/m^3) in some years (Zhou et al. 2007).

Many studies characterizing air pollution conducted in the Pearl River Delta area used various source-apportionment techniques to identify major sources and meteorologic conditions that affect ambient concentrations of PM and PM constituents (e.g., SO_4^{2-}, elemental carbon (EC), organic aerosols, and metals). In Guangzhou, a recent study estimated that SO_4^{2-}, organic carbon (OC), and EC account for about 70–90% of the particulate mass. Two major sources contributing to the PM concentrations measured in Guangzhou in the summer were vehicular emissions (accounting for approximately 38% of the measured concentration) and coal combustion (approximately 26%) (Wang XH et al. 2006).

Measurements of TSP and its metal constituents in urban and suburban areas of Hong Kong and Guangzhou, obtained from December 2003 to January 2005, found elevated concentrations of metals, especially cadmium, lead, vanadium, and zinc, in Guangzhou as compared with Hong Kong; elevated concentrations were found in several other urban areas in Asia as well (Lee CSL et al. 2007a). Distinct seasonal patterns were observed in heavy-metal concentrations in Hong Kong, with higher concentrations

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Sidebar 5. THE PEARL RIVER DELTA (*Continued*)

during the winter monsoon period and lower concentrations during the summer. However, seasonal variations in metal concentrations were less distinct in Guangzhou, suggesting the dominance of local sources of particulate metal pollution around the city. This study also found that the enrichment of heavy metals in Hong Kong and Guangzhou was closely associated with an air mass from the north and northeast that originated in northern China, reflecting the long-range transport of heavy-metal contaminants from the northern inland areas of China to the Pearl River Delta (Lee CSL et al. 2007a).

Because of the complexity of local sources and long-range transport, it is extremely difficult to develop effective source-control strategies for the Pearl River Delta region. For example, research has been conducted to address whether regional photochemical smog formation is dependent on NO_x or volatile organic compound (VOC) concentrations, yet there is still no clear answer. As the population in the Pearl River Delta undergoes change, source characteristics also are changing rapidly. It is clear that the number of motor vehicles is still rapidly increasing, but emission controls on vehicular emissions are increasingly being tightened, and high-sulfur coal is being replaced with lower-sulfur coal or even with liquid or gaseous fuels.

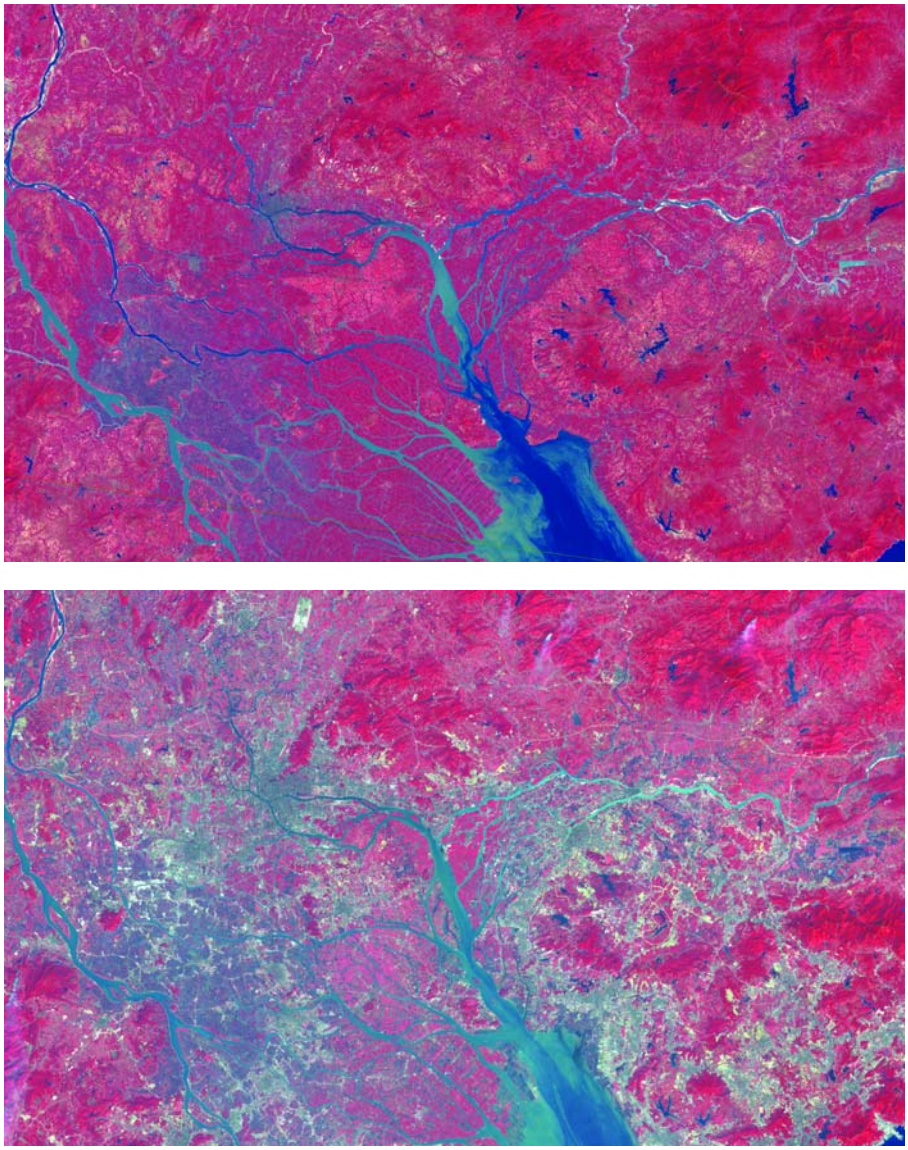

Figure 5.2. Landsat Images of the Pearl River Delta in 1979 and 2003, showing the expansion of urban and industrial areas. Red indicates vegetation, blue indicates water, and gray indicates buildings or paved surfaces. [Images provided by the National Aeronautics and Space Administration Earth Observatory (2007) and created by Jesse Allen. Landsat 3 MSS data provided by the University of Maryland's Global Land Cover Facility. Landsat 7 ETM+ data provided courtesy of the Landsat Project Science Office, NASA/Goddard Space Flight Center.]

The population of the Pearl River Delta has a large percentage of migrants, as do other areas undergoing rapid industrialization and urbanization. People from rural areas and other countries immigrate for purposes of employment, and many will live in the area only for a short time. The prevalence of air-conditioner use and installation of heating, ventilation, and air-conditioning (HVAC) systems is rapidly increasing, which will affect the proportion of time spent indoors as opposed to outdoors.

People in the delta region who do not have air conditioning at home typically spend their evenings (and even entire nights) outside in summer. The use of air conditioning or HVAC decreases the rate of outdoor–indoor air exchange. Therefore, even when people stay indoors, air-conditioning use decreases the penetration of outdoor pollutants into the indoor environment, leading to decreased total exposure to pollutants of outdoor origin. On the other hand, the use of air conditioning and HVAC may increase exposure to pollutants generated indoors.

Sidebar 6. ATMOSPHERIC BROWN CLOUDS AND MINERAL-DUST STORMS

ATMOSPHERIC BROWN CLOUDS

The Indian Ocean Experiment (INDOEX) study (Gupta PK et al. 2001b), conducted in 2001 in the Maldives and the nearby Indian Ocean, found very high concentrations of black carbon (BC), suggesting an influence of anthropogenic sources of pollution over the Indian Ocean. This study led to the concept of the atmospheric brown cloud, which is thought to consist of carbonaceous aerosols emitted from anthropogenic sources that remain in the atmosphere over much of the region in South and Southeast Asia (Ramanathan et al. 2001). As this and related research has evolved, it has become evident from remote sensing that such clouds exist downwind of nearly all densely populated regions of the world (Figure 6.1). In Asia, in addition to the original cloud identified over the Indian Ocean, dust from China and Mongolia is transported over the Pacific Ocean and eventually reaches North America.

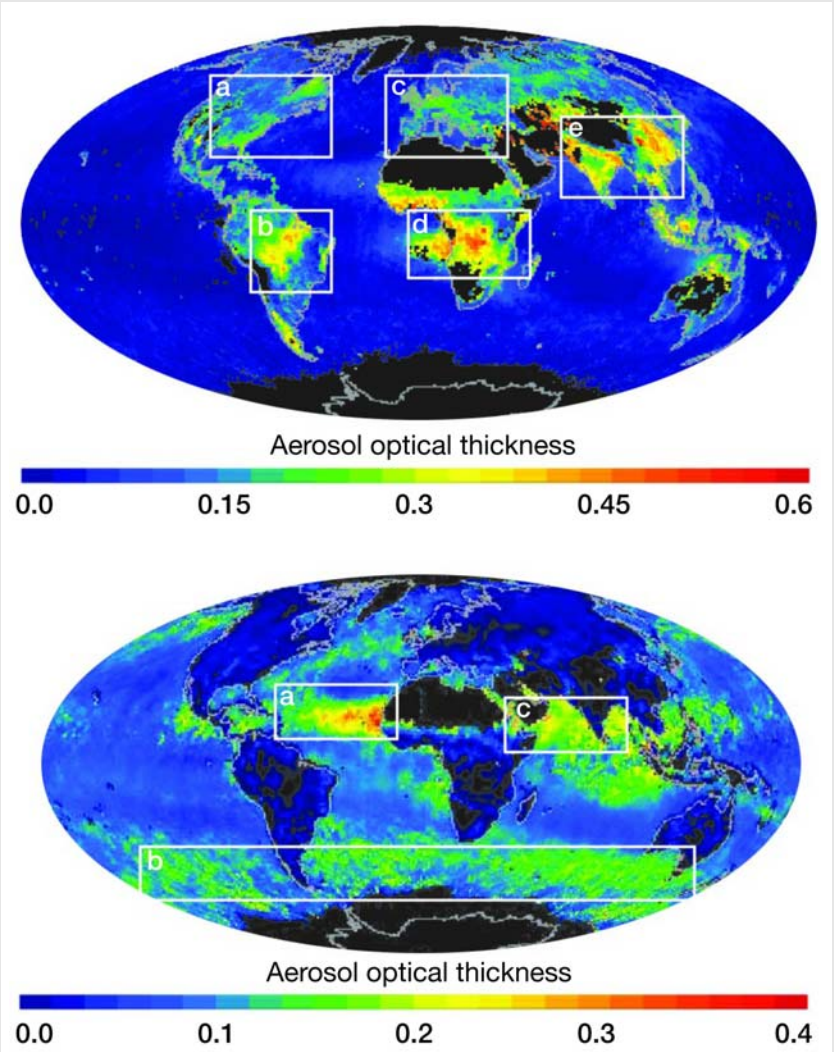

Figure 6.1. Global distribution of atmospheric brown clouds, composed of fine and coarse PM, according to the MODIS satellite measurements of aerosol optical thickness. The boxes labeled with a letter indicate the locations of atmospheric brown clouds. [Reprinted from Kaufman et al. 2002 with permission from Nature Publishing Group, Macmillan Publishers Ltd.]

Today it is recognized that atmospheric brown clouds are, by definition, very large air masses containing contaminants at concentrations similar to those found in polluted air in heavily populated regions (Ramanathan and Crutzen 2003). Such clouds result from the transport of pollutants from source regions. The INDOEX measurement campaign and related studies have shown that the atmospheric brown cloud located in southern Asia is derived from a mixture of pollutants mainly from fossil-fuel combustion (especially in motor vehicles) and residential burning of biomass. Although the relative contribution of these major sources varies depending on the specific location and on meteorologic conditions, studies of chemical composition suggest a greater contribution of fossil-fuel combustion than biomass burning, despite the greater emissions of biomass-combustion products in India and nearby countries. As discussed later in *Trends in Energy Use and Projected Growth in Major Point Sources and Emissions* (in Section II), trends in emissions suggest that pollutants from residential burning of biomass may have begun to stabilize, whereas fossil-fuel emissions, especially in India and China, are increasing.

Although the health impacts of the Asian atmospheric brown cloud have not been directly measured, studies of mixtures thought to contribute to the atmospheric brown cloud have shown adverse health effects similar to those of combustion-based particles observed throughout the world. A recent health-impact analysis suggested that the Asian atmospheric brown cloud has major effects on health (United Nations Environment Programme 2008). For example, assuming a 20-μg/m^3 increase in $PM_{2.5}$ concentration resulting from the atmospheric

Continued on next page

Sidebar 6. ATMOSPHERIC BROWN CLOUDS (*Continued*)

brown cloud and assuming that half the populations of China and India are exposed, this exposure would result in 337,000 excess deaths per year (95% CI, 181,000–492,000).

MINERAL-DUST STORMS

Mineral-dust storms originating in the Gobi Desert, northern and western China, and Mongolia, especially during spring, have been studied extensively (Lim and Chun 2006). They have been shown to impact air quality in South Korea (Lee BK 2006a), Japan (Minoura et al. 2006), Taipei,China (Liu et al. 2006), mainland China (VanCuren 2006), and North America (VanCuren 2006). For example, mineral dust has been estimated to account for up to 20% of $PM_{2.5}$ concentrations in Beijing in summer and 37% in winter (Sun 2004). Figure 6.2 shows the major dust-transport patterns in Northeast Asia. A forecasting system (Uno et al. 2003) to predict episodes of Asian dust and anthropogenic aerosols is operated by the Japanese National Institute for Environmental Studies (*www-cfors.nies.go.jp/~cfors/index.html*).

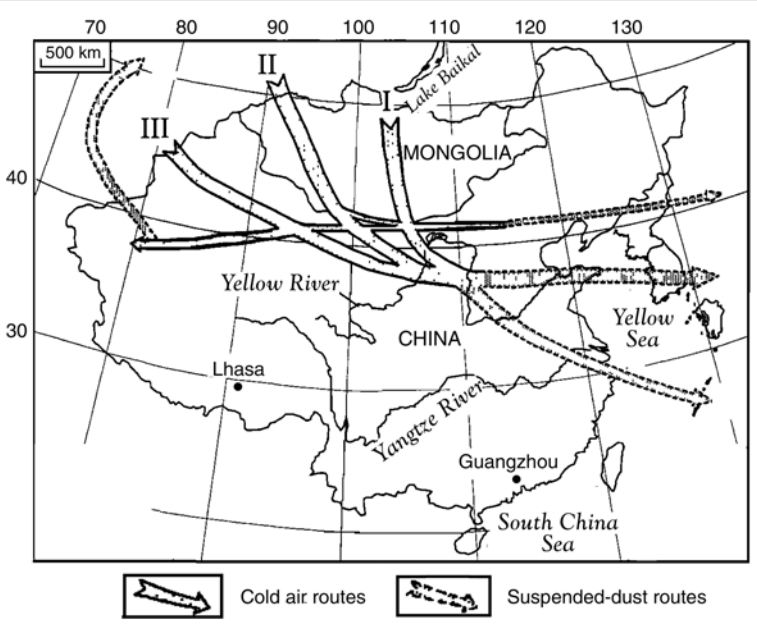

Figure 6.2. Patterns of mineral-dust transport in Northeast Asia. [Adapted from Sun JM et al. 2001 with permission from American Geophysical Union.]

A number of studies have examined the health impacts of mineral-dust episodes within Asia (reviewed in Bell et al. 2007). Although the findings have not been entirely consistent, there appears to be a general association between adverse cardiopulmonary effects and elevated concentrations of coarse particles during dust events.

Common Sources of Air Pollution

Common sources of air pollution are industrial and domestic stationary sources, mobile sources, marine emissions, solid-waste burning, rural area sources, and terrestrial and transport sources.

Stationary Sources

Industrial Industrial sources of air pollution include coal-fired power plants that may or may not be equipped with emission controls. Often, these plants burn high-sulfur coal, leading to high emissions of SO_2 and PM. These sources also include combustion facilities such as large-scale boilers in industrial settings.

Domestic Domestic sources include boilers for heating commercial buildings and stoves for household heating and cooking. Fuels used for this purpose vary by location, depending mainly on the affordability as well as fuel availability and reliability of the fuel supply. A mix of fuel types, including coal, wood, liquefied petroleum gas (LPG), coal gas, and natural gas, can often be found in a single city. Restaurant emissions, consisting of both cooking fumes and fuel-combustion products, appear to be an important contributing source to PM in some cities (Zhao et al. 2007).

Mobile Sources Mobile sources include emissions from motor vehicles and road dust generated from the vehicular traffic. An important characteristic of vehicles in developing countries is the large variation (both within and between cities) in the degree of emission controls on vehicles. For example, three- and two-wheeled motor vehicles are commonly seen on the streets of Bangkok and Delhi but not in Beijing. In some areas of Asia, many of these vehicles are powered by two-stroke engines, which emit a large amount of hydrocarbons (from unburned fuel) that can be a major contributor to O_3 formation. Two-stroke engines may also be a major source of ultrafine and fine metal oxide particles, often resulting in visible tailpipe emissions of white smoke. In Dhaka, the replacement of two-stroke, three-wheeled taxis fueled by compressed natural gas (CNG) resulted in a 25% reduction in $PM_{2.5}$ concentrations in a high-traffic area (Begum et al. 2008). Leaded gasoline has been phased out recently in some, but not all, cities in Asian developing countries. As the number of motor vehicles grows exponentially, contributions from mobile sources to both local and regional air pollution are increasing.

Marine Emissions In port cities, emissions from marine vessels may be important sources of air pollution. Marine emissions, especially those arising from the burning of furnace

fuel oil (the most common bunker fuel), include substantial amounts of PM (in the form of black carbon and primary SO_4^{2-}), hydrocarbons, and SO_2. These emissions have been estimated to make substantial contributions to health impacts of air pollution in coastal cities of Asia (Corbett et al. 2007).

Solid-Waste Burning Open burning of solid waste may be a major source of air pollution in outlying or peri-urban areas of major cities and in smaller cities where waste collection is not efficient. There is limited information about the impact of waste burning on air quality, but source-apportionment studies suggest it can be a large contributor to airborne particle concentrations.

Rural Area Sources In developing countries, the rural population still relies mainly on solid fuels (biomass and coal) for cooking and household heating. Solid fuels are difficult to burn in simple combustion devices, such as household stoves, without substantial emissions of pollutants, because of the difficulty of completely premixing the air and fuel during burning. Solid-fuel combustion in household stoves not only leads to high indoor concentrations of PM, carbon monoxide (CO), and SO_2 (especially with coal burning), but also collectively, from numerous households, contributes to neighborhood concentrations of air pollution and possibly local and regional air pollution. Burning of crop residues, long recognized as a source of air pollution in many parts of Asia, may in fact become more widespread with increasing cultivation and as other solid fuels become available and affordable. For example, in wealthier regions of China (and perhaps other countries), farmers are becoming reluctant to gather biomass residues from the field and store them for use throughout the year, preferring easier-to-handle fuels such as coal briquettes or LPG. This shift to modern fuels creates an excess of crop residues, which are commonly burned in the field, resulting in widespread ambient pollution that affects nearby urban areas in some seasons. Another phenomenon of the developing rural economy is the growth of local industry, typically equipped with outdated, heavily polluting machinery and facilities. Pollutants generated in rural areas may be transported to nearby cities.

Terrestrial and Transport Sources Terrestrial and transport sources of air pollution include natural emissions of precursors of photochemical smog (Mouli et al. 2006) and also episodes of "Asian dust" (also known as sandstorms or dust storms) (Arimoto et al. 2006; Wang KY 2007; see Sidebar 6). These sources are highly seasonal. For example, episodes of Asian dust typically occur in early spring when the weather is dry and the wind blows out of the Gobi Desert (Arimoto et al. 2006). In contrast, hydrocarbons emitted by trees and other vegetation contribute to O_3 and PM concentrations during warmer weather (Mouli et al. 2006; Wang KY 2007). Although Asian dust consists principally of coarse particles in the ground-level atmosphere (below ~2 km above sea level), the dust is usually mixed with anthropogenic pollutants, including fine and ultrafine particles (Arimoto et al. 2006).

Trends in Energy Use and Projected Growth in Major Point Sources and Emissions

To help assess the potential future air pollutant concentrations in Asia, this section focuses on two types of trends associated with degraded air quality: trends in inputs to the econometric Global Model for Ambient Particulates (GMAPs) that is used to predict PM concentrations in Asian cities (described further in *Modeled Estimated Pollutant Concentrations and Projected Trends in Emissions* in Section II), and recent trends in pollutant emissions according to the Regional Emissions Inventory in Asia (REAS; Frontier Research Center for Global Change 2007).

Trends in Energy Use and Fuel Consumption

Although recent evidence (Gustafsson et al. 2009) suggests that biomass combustion is an important contributor to regional air pollution, for example in South Asia, combustion of fossil fuels is the primary source of anthropogenic fine PM in much of Asia, especially in urban areas. Furthermore, fossil-fuel combustion is likely to gain in importance because of increased fuel use and decreased biomass combustion accompanying development. The International Energy Agency annually compiles "energy balances" for over 130 countries, in collaboration with national governments. The energy balance for each country provides an accounting of the total available supply of energy for each fuel type, its transformation into other forms of energy, and the final consumption of energy by various sectors of the economy. The total primary energy supply provides the most comprehensive measure of the total energy consumed in a country. Figure 12 shows the distribution of total primary

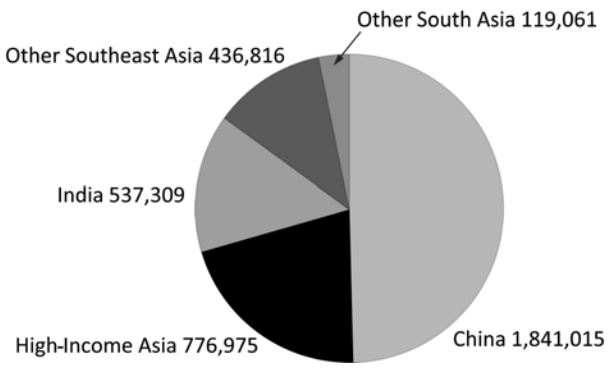

Figure 12. Total primary energy consumption in Asia in 2005. The values for each country or region are thousands of metric tons of oil equivalent. Regions are defined in Table 1. [Data compiled from International Energy Agency 2007.]

energy consumption across Asia in 2005. According to this measure, China accounts for approximately 49% of the total energy consumption in Asia, followed by High-Income Asia with 21% of the total, Other Southeast Asia with 12%, India with 14%, and Other South Asia with the remaining 3%.

From 1990 through 2005, the total primary energy consumption for Asia increased by 75%, at an annual average rate of 3.9% (Figure 13). Overall, the total primary energy supply increased fastest in Other Southeast Asia and China, with a near doubling during the 15-year period. Other Southeast Asia has shown a steady increase throughout the period except for a temporary dip in the late 1990s associated with the Asian economic crisis. In contrast, China's marked economic growth has primarily occurred in the last 5 years, over which time its total primary energy supply has grown at an annual average rate of 11%.

Although economic growth has been the primary driver of increased energy consumption in Asia, the increase has not been in proportion to the increase in level of economic activity. Figure 14 shows the intensity of energy use, measured as the ratio of the total primary energy supply (in millions of metric tons of oil equivalent) to each unit of the GDP (in constant U.S. dollars normalized to 2005 U.S. dollars). While most countries in Asia have shown marginal improvements in the intensity of energy use during the 15-year period, a few countries have become more energy intensive: the intensity of energy use in Other South Asia, Other Southeast Asia, and High-Income Asia changed by < 10%. In contrast, intensity of energy use in India declined by over 40% from 1990 through 2005, largely owing to the growing share in the GDP of the services sector, which is less energy intensive than industrial sectors. Improved efficiency of energy use and a changing fuel mix in the industrial sector also contributed to the improvement. The most dramatic improvements in intensity of energy use have occurred in China, with a reduction by more than 100% in the energy intensity index during the 1990s. Intensity of energy use in China, however, increased between 2002 and 2004 owing to surges in the demand for electricity, in part from manufacturers of metals, building materials, and chemicals.

Table 3 shows the trends in the fuel mix of various regions of Asia during the past 15 years. Although the consumption of all of types of energy increased during this period, throughout the region, there was a notable shift away from combustible renewable fuel (biomass) toward coal, natural gas, and oil. Choices among oil, coal, and natural gas depended on the relative availability of these fuels. For instance, China and India used their abundant supply of coal to meet most of their increased fuel needs during this

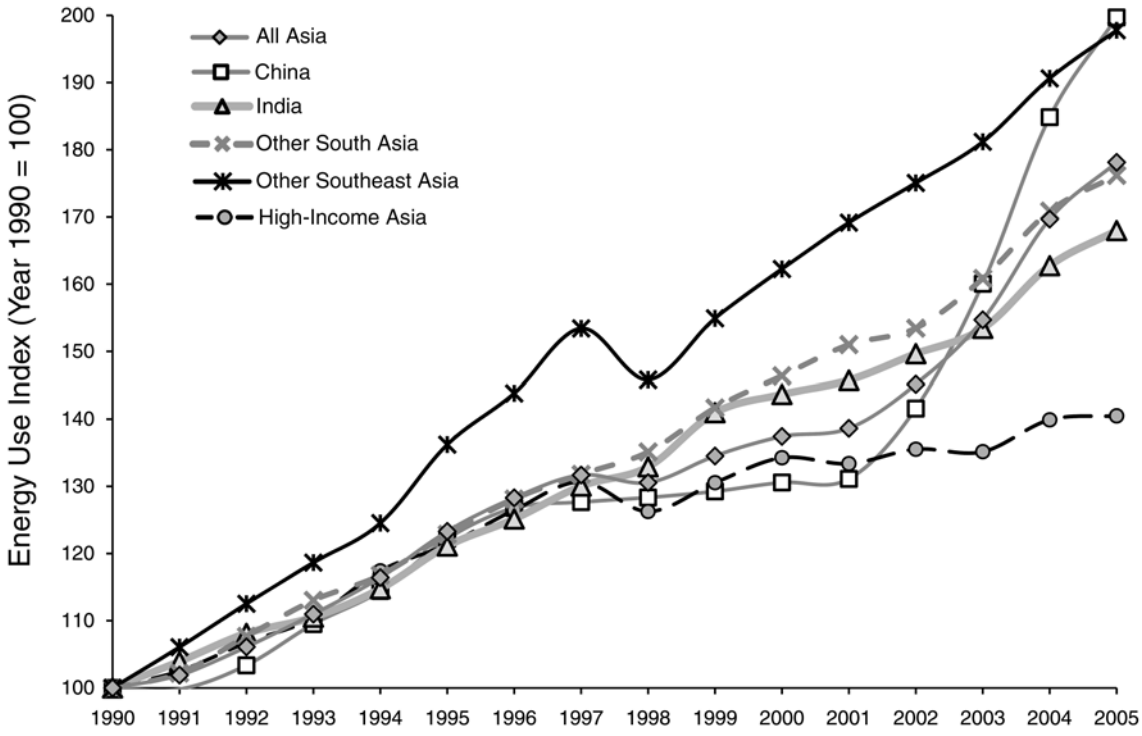

Figure 13. Total primary energy consumption in Asia, 1990–2005. The energy use index is a unitless measure of total primary energy consumption for which the value in 1990 is equal to 100. Regions are defined in Table 1. Values for All Asia are weighted averages of the regions. [Data compiled from International Energy Agency 2007.]

period. Similarly, Pakistan took steps to reduce its oil imports by promoting the use of CNG and LPG in the transport, agriculture, and power sectors and to make natural gas the fuel of choice for power generation.

Vehicular pollution is an important source of pollution in many urban areas. Rapid urbanization and increased motorization has led to increased consumption of gasoline and diesel in the transport sector. Figure 15 shows the

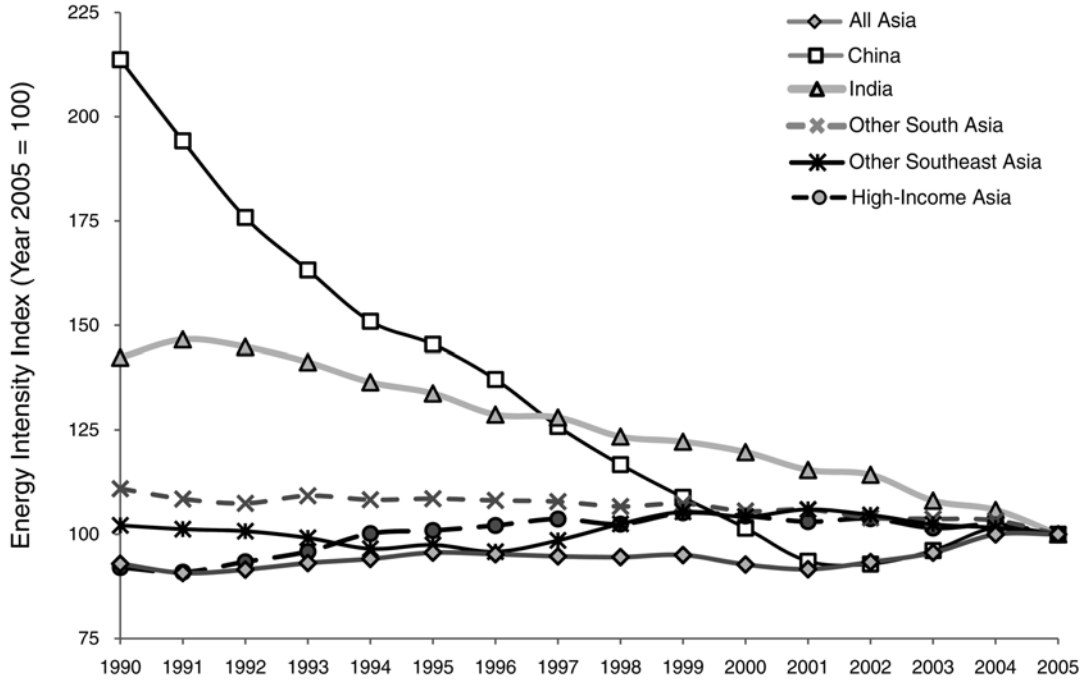

Figure 14. Energy intensity in Asia, 1990–2005. The energy intensity index is the ratio of the total primary energy consumption (in millions of metric tons of oil equivalent) and each unit of the GDP (in constant U.S. dollars normalized to 2005 U.S. dollars; i.e., 2005 values are equal to 100, as shown). Regions are defined in Table 1. Values for All Asia are weighted averages of the regions. [Data compiled from International Energy Agency 2007.]

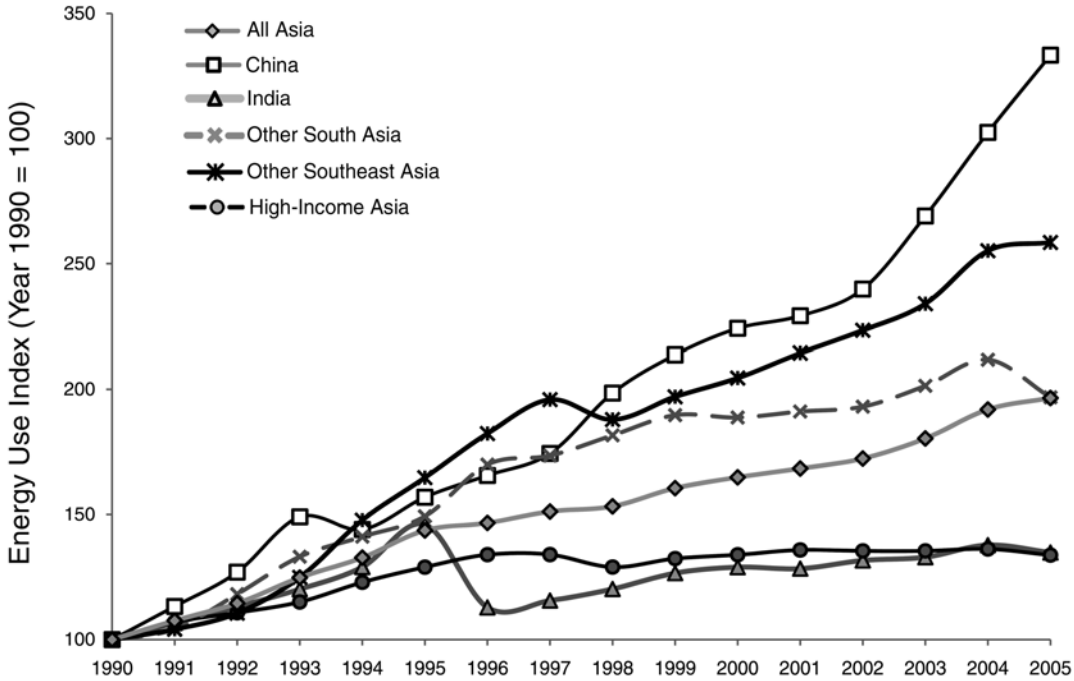

Figure 15. Fuel consumption in the transport sector in Asia, 1990–2005. The energy use index is a unitless measure of total primary energy consumption for which the value in 1990 is equal to 100. Jet fuel is excluded. Regions are defined in Table 1. Values for All Asia are weighted averages of the regions. [Data compiled from International Energy Agency 2007.]

Table 3. Fuel Mix Trends in Asia by Region, 1990–2005[a]

	Metric Tons (thousands) (% of Total)			
Fuel	1990	1995	2000	2005
Total Asia				
Coal	768,908 (37)	945,590 (37)	993,757 (35)	1,564,052 (42)
Oil	619,658 (30)	813,887 (32)	935,374 (33)	1,068,981 (29)
Combustible, renewable fuel	471,958 (23)	494,289 (19)	521,375 (18)	555,013 (15)
Natural gas	117,893 (6)	170,656 (7)	234,636 (8)	313,295 (8)
Other fuel	107,041 (5)	144,737 (6)	171,867 (6)	205,562 (6)
Total	2,085,458 (100)	2,569,159 (100)	2,857,009 (100)	3,706,903 (100)
India				
Coal	106,069 (33)	138,682 (36)	164,346 (36)	207,979 (39)
Oil	62,647 (20)	84,002 (22)	114,365 (25)	128,582 (24)
Combustible, renewable fuel	133,459 (42)	140,003 (36)	148,793 (32)	158,119 (29)
Natural gas	9,833 (3)	16,256 (4)	20,991 (5)	28,841 (5)
Other fuel	7,762 (2)	8,323 (2)	10,809 (2)	13,112 (2)
Total	319,770 (100)	387,266 (100)	459,304 (100)	536,633 (100)
China				
Coal	545,265 (59)	673,067 (60)	658,790 (55)	1,133,078 (62)
Oil	141,533 (15)	199,375 (18)	267,659 (22)	373,247 (20)
Combustible, renewable fuel	200,474 (22)	205,717 (18)	214,387 (18)	224,637 (12)
Natural gas	14,539 (2)	18,661 (2)	28,671 (2)	50,948 (3)
Other fuel	20,167 (2)	29,702 (3)	34,287 (3)	59,075 (3)
Total	921,978 (100)	1,126,522 (100)	1,203,794 (100)	1,840,985 (100)
High-Income Asia				
Coal	102,768 (19)	111,429 (17)	137,402 (19)	161,545 (21)
Oil	318,506 (58)	383,674 (58)	388,036 (53)	372,804 (48)
Combustible, renewable fuel	5,723 (1)	6,166 (1)	7,240 (1)	8,456 (1)
Natural gas	48,559 (9)	65,002 (10)	85,690 (12)	105,678 (14)
Other fuel	74,723 (14)	100,670 (15)	120,174 (16)	124,714 (16)
Total	550,279 (100)	666,941 (100)	738,542 (100)	773,197 (100)
Other South Asia				
Coal	2,424 (4)	2,743 (3)	2,599 (3)	4,652 (4)
Oil	14,427 (21)	20,578 (25)	26,883 (27)	26,198 (22)
Combustible, renewable fuel	34,971 (52)	38,409 (46)	42,881 (43)	48,310 (41)
Natural gas	13,801 (20)	18,648 (22)	24,040 (24)	35,987 (30)
Other fuel	1,953 (3)	2,637 (3)	2,495 (3)	3,916 (3)
Total	67,576 (100)	83,015 (100)	98,898 (100)	119,063 (100)
Other Southeast Asia				
Coal	12,382 (5)	19,669 (6)	30,620 (9)	56,798 (13)
Oil	82,545 (37)	126,258 (41)	138,431 (39)	168,150 (38)
Combustible, renewable fuel	97,331 (43)	103,994 (34)	108,074 (30)	115,491 (26)
Natural gas	31,161 (14)	52,089 (17)	75,244 (21)	91,841 (21)
Other fuel	2,436 (1)	3,405 (1)	4,102 (1)	4,745 (1)
Total	225,855 (100)	305,415 (100)	356,471 (100)	437,025 (100)

[a] Regions are defined in Table 1. Data are given as thousands of metric tons of oil equivalent (with percentage of total in parentheses). Data compiled from International Energy Agency 2007.

Table 4. Transport Sector's Market Share of Gasoline Consumption in Asia, 1990–2005[a]

Region	Market Share (%)			
	1990	1995	2000	2005
India	16	15	24	29
China	68	68	57	52
High-income Asia	51	50	54	55
Other South Asia	21	17	16	19
Other Southeast Asia	47	43	45	48
All Asia[b]	48	46	48	49

[a] Regions are defined in Table 1. Data compiled from International Energy Agency 2007.

[b] Each percentage for All Asia is a weighted average of the market shares for regions.

trends in fuel consumption (excluding jet fuel) in the transport sector in Asia, for 1990–2005. The total consumption of gasoline and diesel approximately doubled in Asia overall and more than tripled in China. In contrast, the growth in fuel consumption in the transport sector in India was similar to that in high-income Asia. The proportion of gasoline in the overall fuel mix remained relatively unchanged for most countries in Asia (Table 4). However, China and India are notable exceptions: the market share of diesel fuel rapidly decreased in China since 1997 but increased in India as of 1995.

Emissions The REAS inventory is a comprehensive emissions inventory for Asia, covering past, present, and future emissions. Major anthropogenic emissions sources in the inventory include (1) the combustion of fossil fuels and biofuel as part of power production, transport, and domestic and industrial activities; and (2) noncombustion sources such as industrial processes (e.g., oil extraction and solvent production), agricultural activities (e.g., tilling of soil and practices related to livestock), and natural soil emissions. Emissions from international shipping and aviation within Asia, although not assigned to specific countries or regions, are included in the total for the continent. The inventory does not include emissions from open-air burning of biomass (e.g., agricultural burning or wildfires) that may make substantial regional and local seasonal contributions to air pollution. Emissions are estimated as the product of source activity, emission factors, and the removal efficiency of emission controls (Ohara et al. 2007). In the absence of enhanced emission controls and air quality management programs, future emissions are likely to follow these historical trajectories.

Given the importance of PM and O_3 to the health impacts of air pollution, in this section we focus on emissions of PM as well as major gaseous precursors (SO_2 and NO_x) and O_3 precursors (NO_x and non-methane volatile organic compounds [NMVOCs]). BC emissions are presented as a marker of primary particle production via combustion processes (Figure 16). Increased emissions of crustal PM resulting from increasingly frequent and intense dust storms have also been described (Sidebar 6). Dominant sources of BC emissions are domestic combustion of biomass and coal, with the relative biomass contribution being nearly 80% in India, 40% in China, and 60% across all of Asia (Ohara et al. 2007). Data on particle emissions in India suggest that 80% of carbonaceous aerosols emitted from the country originate from biomass combustion (Parashar et al. 2005). The dominant contribution of biomass to particle emissions in India is based on the importance of biomass to the rural energy supply, as biomass supplies 70–80% of rural energy needs. Gupta PK and colleagues (2001b) used state-level data on food consumption and energy required for cooking in India and estimated the overall contributions to the total biomass emissions from BC (estimated as 220 gigagrams/yr [95% confidence interval [CI], 65–760) as 75% from wood, 16% from dung cakes, and 9% from crop waste. For India, therefore, BC is a good indicator of PM emissions from biomass.

Although the relative contribution of biomass to overall air pollution is larger in India than in China, total BC emissions are higher in China, with the majority originating from coal combustion in the industrial sector (33%) and the residential sector (35%), residential combustion of wood (12%) and agricultural waste (7%), and agricultural burning (7%) (Cao GL et al. 2006). The importance of residential sources of air pollution in China — mostly coal and coal briquettes and, to a lesser degree, biomass — was also identified by Streets and colleagues (2003), who estimated a residential-source contribution (for 2000) of 54.5% of total BC emissions.

An analysis of trends in BC emissions (Figure 16) indicates the dominance of emissions from China and India and some stabilization of emission levels, especially in China, due to decreases in coal use by households and industry (Streets et al. 2003). Emissions from elsewhere in Asia (except High-Income Asia) still appear to be increasing slowly, and Chinese emissions have increased since 2000 due to recent increases in emissions from industrial coal burning and diesel vehicles. Similar trends are evident for emissions of organic carbon (OC), although overall, 90% of OC emissions derive from domestic biomass combustion (Ohara et al. 2007).

SO_2 emissions are used as an indicator of combustion of fossil fuels, especially coal. Although recent data on emissions trends suggest a stabilization of BC emissions in India, there has been a continued increase in fossil-fuel (SO_2) emissions in India and China, most recently characterized by a period of stabilized SO_2 emissions followed by a period of extremely rapid growth in emissions (Figure 17). Overall, these trends suggest that combustion of biomass is

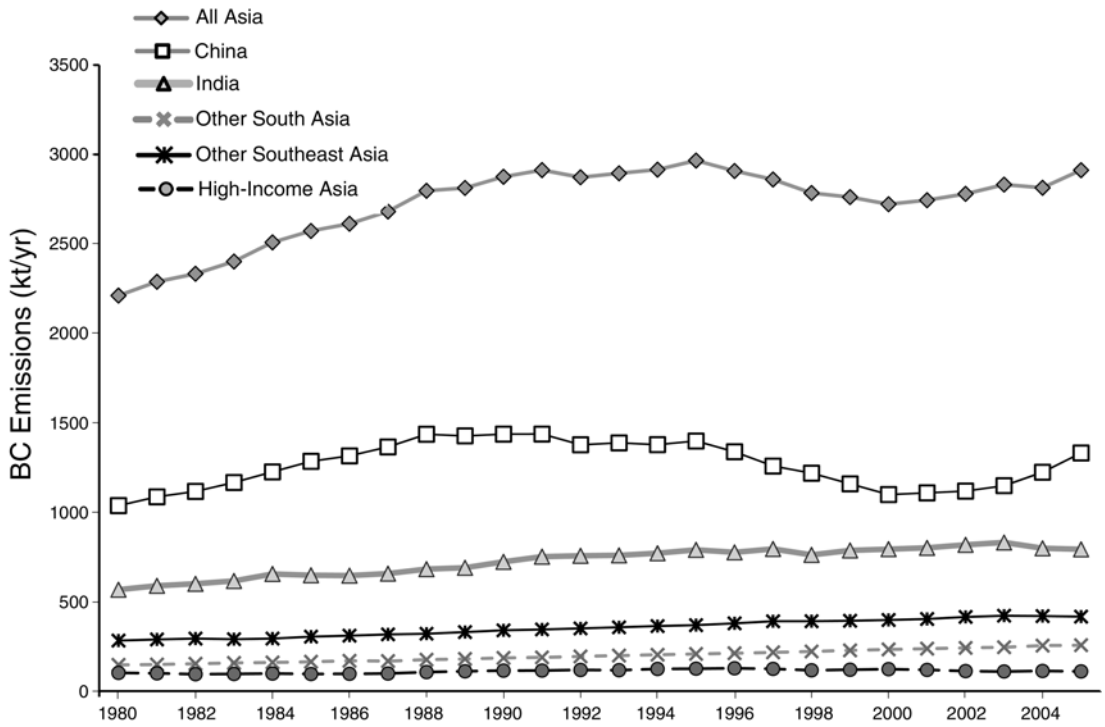

Figure 16. BC emissions in Asia, 1980–2005. Data are given in kilotons per year. Regions are defined in Table 1. Values for All Asia are weighted averages of the regions. [Adapted from the Frontier Research Center for Global Change 2007 with additional material from T. Ohara (personal communication, May 26, 2008).]

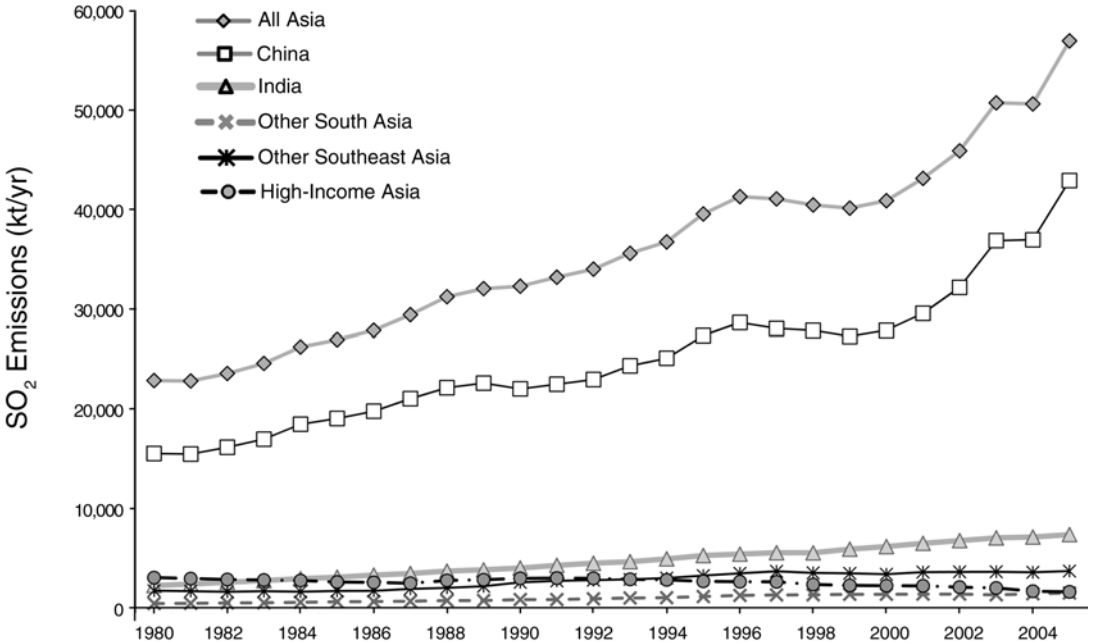

Figure 17. SO_2 emissions in Asia, 1980–2005. Data are given in kilotons per year. Regions are defined in Table 1. Values for All Asia are weighted averages of the regions. [Adapted from the Frontier Research Center for Global Change 2007 with additional material from T. Ohara (personal communication, May 26, 2008).]

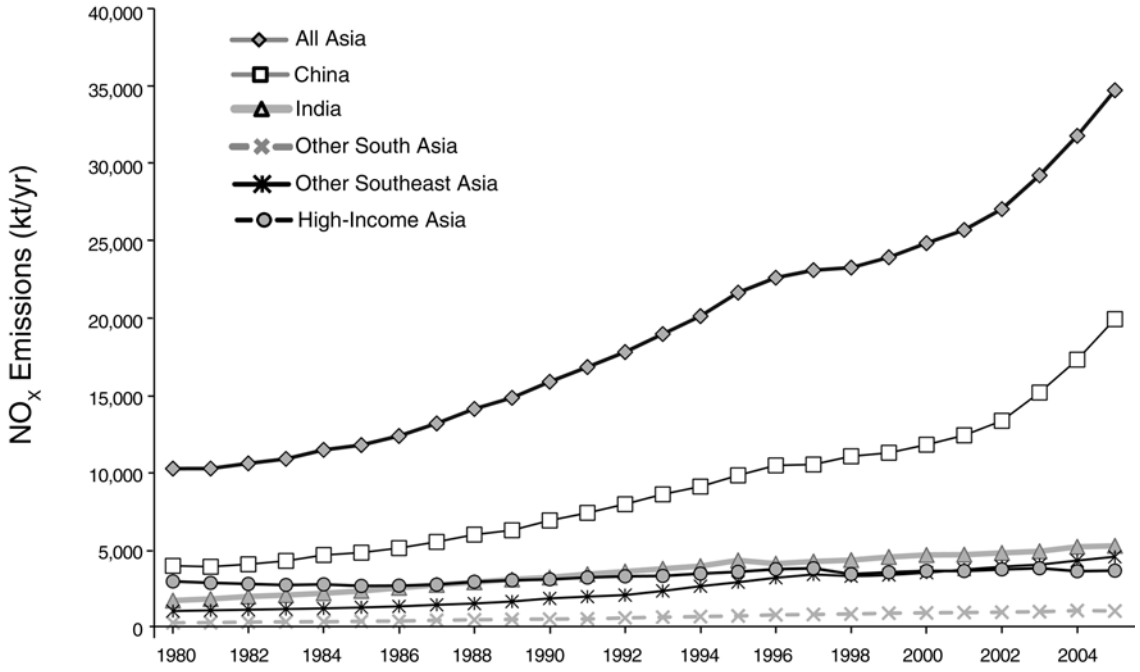

Figure 18. NO_x emissions in Asia, 1980–2005. Data are given in kilotons per year. Regions are defined in Table 1. Values for All Asia are weighted averages of the regions. [Adapted from the Frontier Research Center for Global Change 2007 with additional material from T. Ohara (personal communication, May 26, 2008).]

reaching a plateau across Asia, whereas contributions from fossil-fuel combustion (as evidenced by SO_2 and NO_x emissions, as shown in Figure 17 and Figure 18, respectively) are increasing, especially in China and India.

SO_2 emissions in China, by far the largest component of Asian emissions, stabilized during 1995–2000 owing to decreases in coal use resulting from an economic slowdown, and a switch to lower-sulfur coal (Streets et al. 2003) and desulfurized coal. For example, the average sulfur content of coal in China has decreased from 1.35% before 1985 to 1.12% in 1995 and 1.08% in 2000 (Ohara et al. 2007). Despite these improvements in fuel quality, SO_2 emissions from China have increased dramatically since 2002, because of increased coal combustion. In China, the contribution of power plants to SO_2 emissions increased from 28% to 51% during 1980–2005. As of 2004, roughly 35% of Chinese coal-fired power plants had desulfurization capability, indicating a substantial ability to further reduce SO_2 emissions from this sector (National Academy of Engineering [NAE] and National Research Council [NRC] 2008). SO_2 emissions from India continue to increase gradually. The steady increase (55%) in SO_2 emissions in India in 1990–2000 contrasts with the much lower increase (of 10%) in BC emissions in India over the same period (Figure 17). In China, which accounted for 70% of the SO_2 emissions in Asia in 2003, coal combustion in power plants and industrial sources was responsible for more than 80% of these emissions (Ohara et al. 2007), whereas in India, which accounted for 13% of the 2003 Asian emissions of SO_2, coal burning was responsible for roughly 60%. Emissions from High-Income Asia continue to decrease.

Major sources of NO_x emissions in Asia include transport and coal combustion in power plans and industrial facilities. Since the mid-1990s, NO_x emissions in China, India, and Other Southeast Asia have increased continuously owing to increased motor-vehicle use and power generation, with accelerated growth in China in recent years, including a 15% increase in 2005 alone. In China, emissions increased by 395% between 1980 and 2005. Emissions from High-Income Asia have stabilized but accounted for a relatively low proportion (11%) of the total NO_x emissions in 2005. Although transport-related emissions remain a major source of NO_x emissions (32% of the 2005 total), the contribution of power plants increased from 17% in 1980 to 35% in 2005 and they are now the leading source of NO_x emissions in Asia (Ohara et al. 2007). Measurements of nitrogen dioxide (NO_2) concentrations in the troposphere of have shown even larger increases in NO_2 in China (e.g., a 40% increase between 1996 and 2002) than those estimated from emissions inventories (Richter et al. 2005). The importance of motor-vehicle emissions to NO_2

concentrations in China was demonstrated in a 2-day period when traffic-reduction measures (estimated to have removed 30% of the fleet) were implemented in Beijing during the Sino-Asian Summit. Remote-sensing measurements during this period detected a decrease in NO_2 concentrations by as much as 40% (Wang YX et al. 2007).

Figure 19 presents information about trends in emissions of NMVOCs, one of the major precursors (along with NO_x) of O_3. NMVOC emissions have increased steadily throughout Asia, because of increased automobile use and growth in the production and use of chemicals, solvents, and petroleum products. Unlike the other pollutants discussed above, of which both China and India are clearly the major producers, Other Southeast Asia is an important source of NMVOCs, accounting for 30% of the total emissions.

Historic Patterns and Trends in Vehicle Production and Use of Motor Vehicles The three primary causes of increases in the world's vehicle fleets are population growth, increased urbanization, and economic improvement. As described above, all three are occurring in Asia as well as globally. As a result, the global vehicle population, which was estimated to have exceeded one billion units in 2002, is predicted to continue to grow steadily and substantially, following the historical trends illustrated in Figure 20.

Data on motor-vehicle production are useful as surrogates of vehicle emissions and the overall composition of vehicle fleets within countries. Motor-vehicle production has gradually expanded from North America to Europe since the 1950s and to Asia more recently. Nowhere has the growth been greater than in China, as illustrated in Figure 21. China is now estimated to be the third largest producer of cars, trucks, and buses in the world and is rapidly becoming a major market as well; it is also far and away the largest producer of motorcycles.

Numerous forecasts have indicated that the motor-vehicle population in Asian countries that are not members of the Organisation for Economic Co-operation and Development (OECD; only Japan and Korea are members) will continue to grow rapidly for the foreseeable future, especially in China and India. One of the most comprehensive assessments was carried out by the World Business Council for Sustainable Development (2004). As illustrated in Figure 22, more than a doubling of the vehicle population is forecast to occur between 2000 and 2025, with China driving this growth.

One of the most distinctive characteristics of the region is the dominant role played by two-wheeled vehicles in many Asian cities. Throughout most of non-OECD Asia, there are many more motorcycles and scooters than cars (Figure 23). But this pattern is shifting rapidly, as the rate

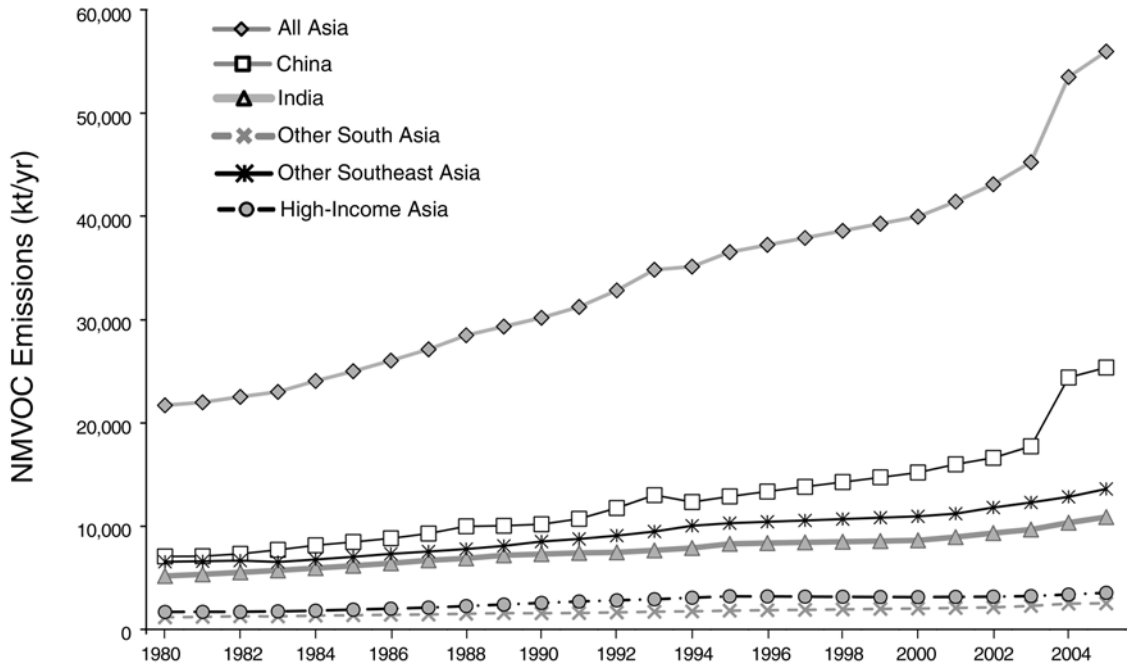

Figure 19. NMVOC emissions in Asia, 1980–2005. Data are given in kilotons per year. Regions are defined in Table 1. Values for All Asia are weighted averages of the regions. [Adapted from the Frontier Research Center for Global Change 2007 with additional material from T. Ohara (personal communication, May 26, 2008).]

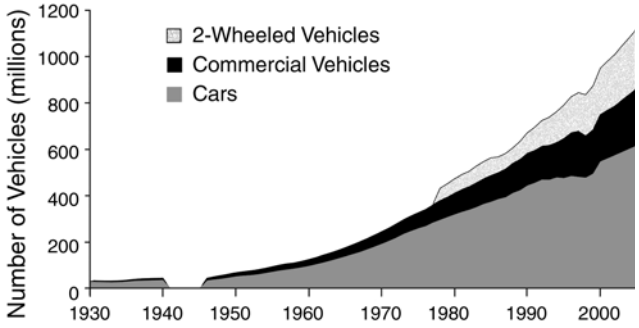

Figure 20. Motor-vehicle production worldwide, 1930–2005. [Data compiled from Ward's Automotive Group 2008.]

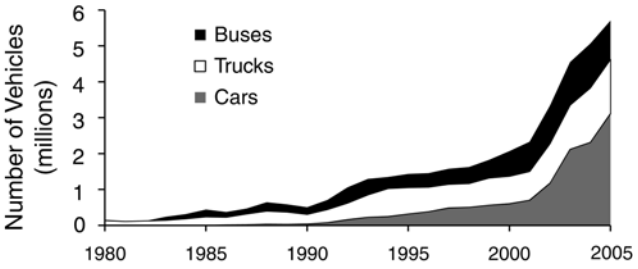

Figure 21. Motor-vehicle production in China, 1980–2005. [Data compiled from Ward's Automotive Group 2008.]

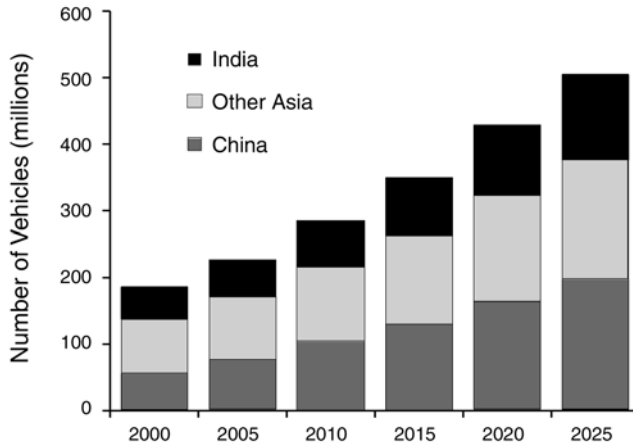

Figure 22. Past and projected distribution of the motor-vehicle population in non-OECD Asia, 2000–2025. (Only Japan and Korea are members of the OECD.) [Data compiled from World Business Council for Sustainable Development 2004.]

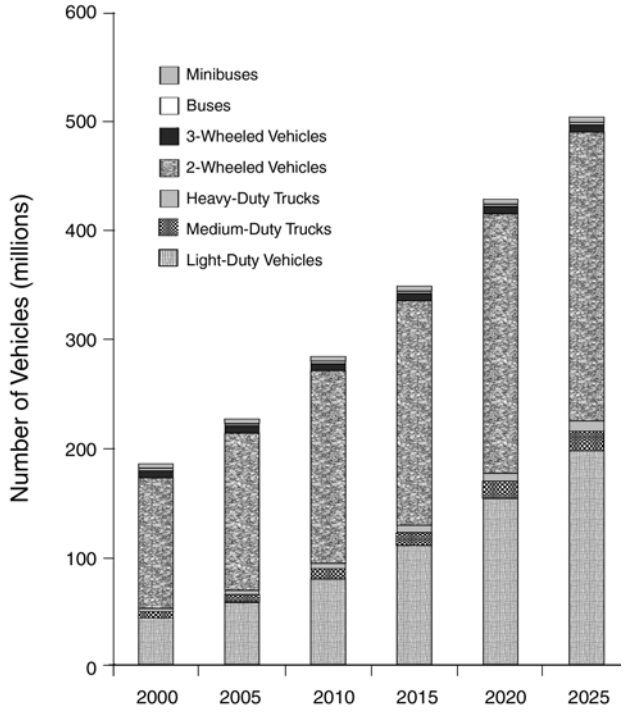

Figure 23. Past and projected trends in the motor-vehicle population in non-OECD Asia, 2000–2025, according to type of vehicle. (Only Japan and Korea are members of the OECD.) [Data compiled from World Business Council for Sustainable Development 2004.]

of growth of the car population eclipses that of motorized two-wheelers. As discussed above, emissions are substantially greater from two-stroke than from four-stroke vehicles, and it is the engine type rather than the number of wheels that is the greatest determinant of emissions (see Sidebar 7).

Stationary Sources: Current Status and Projected Trends Stationary sources are fixed-site producers of pollution. Industrial sources in Asia that emit air pollution include large-scale power plants and manufacturing facilities as well as small, family-owned manufacturing shops — which are numerous and difficult to control, and whose emissions are widespread throughout Asian cities. In some locations (e.g., Hanoi, Dhaka, and Kathmandu), numerous small sources, such as brick kilns, collectively make major contributions to air pollution (CAI-Asia Center 2002; Co et al. 2009). Larger industrial sources typically have ducted emissions, but some also have fugitive dust and nonducted emissions from various processes, which collectively pollute the air. Fuels used by larger stationary sources are mostly coal, furnace oil, LPG, and natural gas. Coal- and oil-fired power plants are usually the largest sources of SO_2 emissions within a city because of the large amount of fuel they consume. Air pollution control technologies for large stationary sources include use of lower-sulfur fuels as well as end-of-the-pipe control equipment such as electrostatic precipitators, scrubbers, baghouses (a type of fabric filter), and flue-gas desulfurization units. Even though large stationary sources such as power plants may

Sidebar 7. MOTOR-VEHICLE PROFILES IN CHINA AND INDIA

Data on emerging motor-vehicle markets in Asia are collected by Segment Y Automotive Intelligence, a Dutch company operating out of India. The following country profiles are based on recent data provided to the Asian Development Bank by Segment Y.

CHINA

China has the fastest growing vehicle population in the world. Figure 7.1 provides a detailed breakdown of the known and expected vehicle population from 2005 to 2025. This forecast shows rapid growth in the car population but also indicates that the two-wheeled vehicle population will likely remain the largest segment of motor vehicles through 2025. However, the growth rate for cars is greater than the growth rates for other vehicles; the projection in Figure 7.2 suggests an increase in the numbers of cars by a factor of nine, and an increase in the numbers of light commercial vehicles by a factor of four, between 2005 and 2025. Vehicle growth is estimated to be roughly evenly divided between gasoline and diesel vehicles (Figure 7.3). However, the absolute number of gasoline vehicles is expected to far outweigh that of diesel vehicles during this period (Figure 7.4). In the future, the pressure to curtail oil

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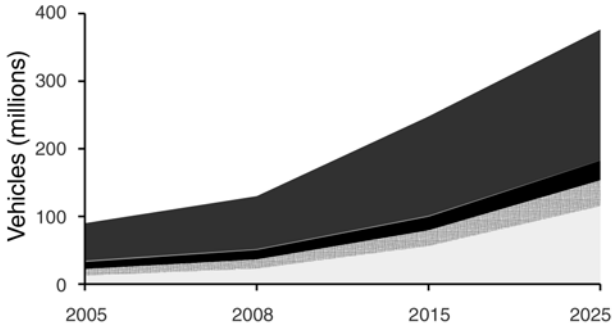


	2005	2008	2015	2025
Automobile	12.90	23.42	56.84	115.84
Light Commercial	9.38	13.07	22.84	37.73
Heavy Commercial	10.41	13.89	19.88	29.28
Three-Wheeled	1.96	1.51	1.71	0.34
Two-Wheeled	55.33	78.15	146.69	193.19
Total	89.98	130.04	247.96	376.38

Figure 7.1. Current and projected increases of number of motor vehicles in China (in millions), according to vehicle type. [Data compiled from Asian Development Bank 2006c.]

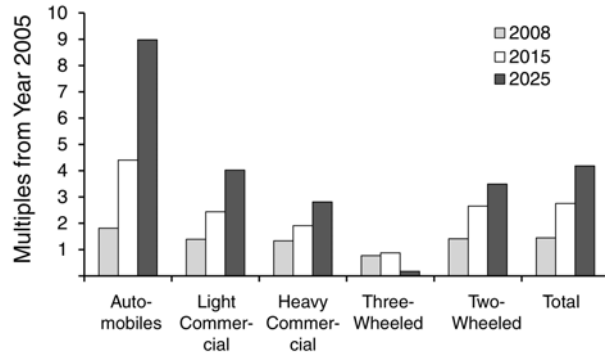


Figure 7.2. Current and projected increases in motor-vehicle populations in China compared with 2005, according to vehicle type. Values are multiples of the number of vehicles present in 2005 (the baseline). (Based on data presented in Figures 20 and 21.)

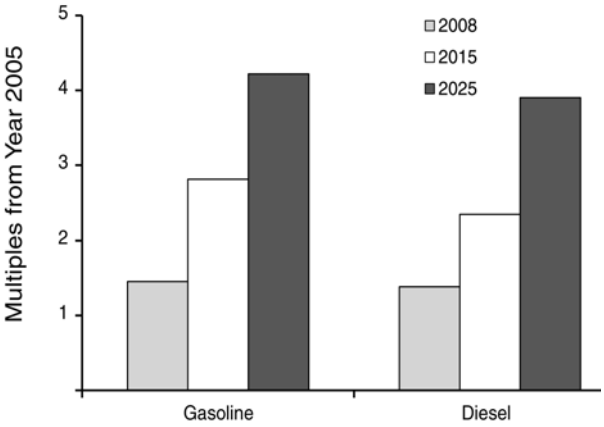
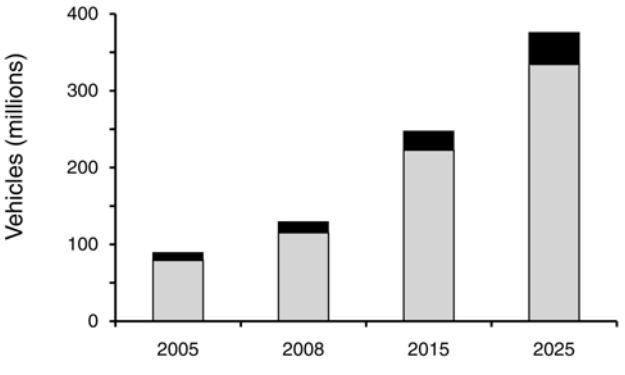


Figure 7.3. Current and projected increases in motor-vehicle populations in China compared with 2005, according to fuel type. Values are multiples of the number of vehicles present in 2005 (the baseline). (Based on data presented in Figures 20 and 21.)



	2005	2008	2015	2025
Gasoline	79.17	115.07	222.63	334.17
Diesel	10.81	14.97	25.33	42.20
Total	89.98	130.04	247.96	376.38

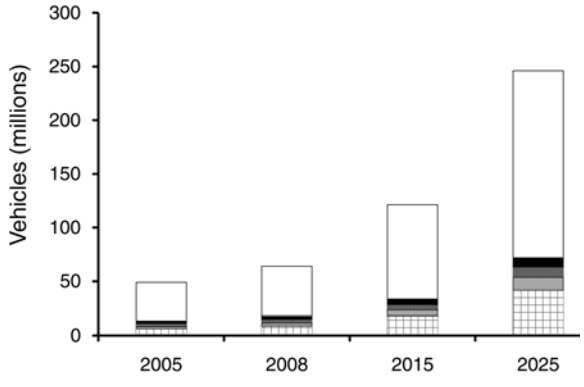
Figure 7.4. Current and projected increases in number of motor vehicle in China, according to fuel type. All values are in millions. [Data compiled from Asian Development Bank 2006c.]

Sidebar 7. MOTOR-VEHICLE PROFILES IN CHINA AND INDIA (*Continued*)

imports through fuel-efficient technologies may come to outweigh the pressure to reduce air pollution; if so, clean diesel vehicles could become available in China, in which case there could be a dramatic increase in the diesel-car population.

INDIA

Figure 7.5 shows that the vehicle population in India is also growing very rapidly, with a predicted increase by nearly a factor of five from 2005 to 2025.



	2005	2008	2015	2025
Automobile	6.18	8.75	17.99	41.58
Light Commercial	2.42	3.16	5.68	12.45
Heavy Commercial	2.42	2.90	4.59	9.09
Three-Wheeled	2.31	3.00	5.33	8.82
Two-Wheeled	35.78	46.10	87.72	174.13
Total	49.11	63.91	121.29	246.07

Figure 7.5. Current and projected increases in number of motor vehicles in India (in millions) compared with 2005, according to vehicle type. [Data compiled from Asian Development Bank 2006c.]

be responsible for a majority of pollutant emissions, compared with mobile sources or smaller-scale point sources, their impact on local concentrations and exposures may be less important if emissions occur above the atmospheric layer in which pollutant mixing occurs.

Asia's rapid urbanization is resulting in increasing air pollution threats in its cities. In the power-generation sector, China and India dominate Asian consumption, using the largest amounts of fuel to produce electricity. India has recently overtaken Japan as the second largest consumer of fuel to generate power, and this trend is projected to continue in the future. In the industrial sector, China, Japan, India, and Southeast Asian countries use the most fuel, whereas in the domestic sector, China and Japan use the most fuel, followed by Southeast Asian countries.

Waste incineration may also be an important stationary source of air pollution in urban areas. Among Asian countries, there is great diversity in the degree of development of municipal waste incineration; these differences are reflective of the varying levels of industrialization, scarcity of land for landfills, consumption habits, and wealth (Hunsicker et al. 1996). Open burning of solid waste is also an important source of air pollution in some locations in Asia that lack modern waste-management practices.

A description of the stationary sources in selected countries, representing the range of industrial development within Asia, is provided below.

China In line with the rapid expansion of the Chinese economy after the late 1970s, there has been an increasing demand for electricity. The situation in Shanghai provides an example of how this energy demand has affected air pollutant emissions and concentrations.

Energy use in Shanghai, China's main industrial city, has traditionally been dominated by coal, which accounted for about 70% of total energy consumption in the early 1990s. In Shanghai, the industrial sector was the single largest energy-consuming sector in 1995, using about 50% of the total energy, whereas power plants used about one third of the total (Li et al. 2004). Li and colleagues projected that power generation will account for 50% of the demand for fuel in Shanghai by 2020. Along with this shift, in the last 10 years, heavy industries have relocated from urban centers to less-populated, remote areas; coal-fired industrial boilers have been replaced by those that use cleaner fuels; and emission controls have been installed at large power plants. These changes have led to reduced emissions of air pollutants from stationary sources. Semi-dry scrubbers and baghouses are the preferred means for meeting emission standards.

India In India, the majority of electricity is generated from coal-based thermal power plants (88%), with small amounts produced by gas plants (9%) and oil-based plants (3%) (Bharati 1997; Reddy and Venkataraman 2002). In addition, India has primary and secondary producers of steel that use coal as their major fuel (with small quantities of oil and gaseous fuels) (Steel Authority of India 1998). Mumbai, the most industrialized Indian city, has 183 air polluting industries; 70 are large scale, 37 are medium scale, and 76 are small scale. In addition, there are 32 stone-crushing plants in the region (Bhanarkar et al. 2005).

Nepal The main stationary sources of airborne pollutants in Nepal are the combustion of fossil fuels for heating and

power and the release of waste gases and dust from industrial processes. Kathmandu Valley is the site of about 50% of the total industries in Nepal (Devkota and Neupane 1994). In the Kathmandu Valley, brick kiln operations are the major source of airborne PM. Industries like carpet and garment producers and printing shops are more centralized in the Kathmandu Valley than in other parts of the country. In the industrial sector in Nepal, the main fuel burned is coal (78%), followed by wood (17%), with brick-making accounting for most of the total fuel use (75%), followed by the cement industry (16%) (Shrestha and Malla 2007).

Japan Japan's economy is mature, with an advanced industrial sector and strictly enforced emission standards. The main stationary sources of air pollution in Japan are the motor-vehicle industry, petrochemical plants, and electronics manufacturing facilities. Japan performs more waste incineration than other Asian countries and has one of the highest percentages of power plants that are waste-to-energy facilities in the world.

Table 5. Major Indoor Sources of Air Pollution[a]

Source	Key Pollutants
Household solid fuel use	$PM_{2.5}$, CO, PAHs, NO_x, VOCs, semi-VOCs
Coal	$PM_{2.5}$, NO_x, sulfur oxides, arsenic, fluorine
Tobacco smoke	$PM_{2.5}$, CO, PAHs, VOCs, semi-VOCs
Cooking	$PM_{2.5}$, PAHs, NO_x, VOCs, semi-VOCs, aldehydes
Cleaning	$PM_{2.5}$
Incense and mosquito coils	$PM_{2.5}$
Consumer products	VOCs, semi-VOCs, pesticides
Construction materials used in remodeling or demolition	VOCs, semi-VOCs, aldehydes, asbestos, lead, radon
Building characteristics related to moisture, ventilation, and furnishings	Biologic pollutants (fungal spores, mites, cockroaches, endotoxins, glucans)
Soil, rock, and water sources under building	Radon
Indoor chemical processes	Free radicals and other short-lived, highly reactive compounds

[a] Adapted from Zhang JF and Smith 2003.

Indoor Air Pollution Major sources of indoor air pollution are summarized in Table 5. Some are also described below.

Household Use of Solid Fuels The use of solid fuels, namely biomass and coal, is the most widespread traditional source of indoor air pollution. Indeed, nearly 3 billion people continue to rely on solid fuels for cooking and heating (Smith et al. 2004; Bruce et al. 2006), with the combustion of fuels in open fires or low-efficiency, unvented stoves often resulting in the release of smoke directly into the home environment. Women and children often experience the greatest exposures, as well as the greatest health impacts, because they spend more time than men indoors in close proximity to the stove or cooking fire. Despite rapid increases in urbanization and shifts to modern lifestyles, household use of solid fuel remains responsible for a substantial burden of disease in developing countries of Asia (Table 6).

Table 6. Household Use of Solid Fuel and Associated Burdens of Disease in Selected Asian Countries in 2002[a]

Region[b] / Country	Solid Fuel Use (%)[c]	Deaths[d]	Disease Burden (%)[e]
India	82	407,100	3.5
China	80	380,700	1.6
High-Income Asia			
Japan	< 5		
South Korea	< 5		
Singapore	< 5		
Other South Asia			
Afghanistan	> 95	23,900	4.9
Bangladesh	89	46,000	3.6
Nepal	81	7,500	2.7
Pakistan	81	70,700	4.6
Sri Lanka	67	3,100	1.3
Other Southeast Asia			
Cambodia	> 95	1,600	1
Indonesia	72	15,300	0.7
Laos	> 95	2,400	3.5
Malaysia	< 5	< 100	0
Myanmar	> 95	14,700	3.2
Philippines	45	6,900	1.6
Thailand	72	4,600	0.8
Vietnam	70	10,600	1.2

[a] Data compiled from WHO 2007a.

[b] Regions are defined in Table 1.

[c] The percentage of the total household fuel used in the country.

[d] The number of deaths attributable to household use of solid fuel.

[e] The percentage of the country's total burden of disease.

Modern Sources With increased economic development, households gradually shift to cleaner gaseous fuels, but this shift is slow and largely confined to high- and middle-income families. In addition, as the housing stock goes from temporary structures to more permanent ones, other indoor sources of air pollution, especially those related to household furnishings, building materials, and cleaning agents, become more prevalent. In general, these result in substantially lower exposures than household solid fuel use, however.

Incense Burning Incense burning also contributes to high indoor air pollution concentrations in both homes and religious centers (Tung et al. 1999). PM with an aerodynamic diameter ≤ 10 μm (PM_{10}), $PM_{2.5}$, volatile organic compounds (VOCs), CO, CO_2, NO_x, SO_2, methane (CH_4), and polycyclic aromatic hydrocarbons (PAHs) have been detected in incense smoke (Jetter et al. 2002; Lung and Hu 2003; Lee SC and Wang B 2004). The generation of PM and associated PAHs from incense burning varies among incense sticks within the same class of incense, among the various classes of incense, and among segments within the same incense stick (Lung and Hu 2003). Since incense ingredients used can vary across cultures, a better understanding is needed of which incense ingredients release harmful compounds when burned (Chang YC et al. 2007).

Environmental Tobacco Smoke Tobacco smoke, a form of biomass combustion, is likely to be the most important source of indoor air pollution in households not using solid fuels for cooking or heating. Although smokers experience the highest exposures, nonsmokers sharing an environment with smokers also inhale environmental tobacco smoke (ETS). As in the West, studies in Asia (e.g., Hong Kong and cities in India) report acute effects of exposure to ETS (Lam et al. 2000; Gupta D et al. 2002; McGhee et al. 2002).

Because household conditions (including ventilation) vary dramatically by income, culture, and climate, generalizing about emissions trends in the absence of field surveys is difficult. Few data are available regarding indoor air pollution concentrations in schools, occupational settings (especially the so-called informal sector that is sizeable in poor countries), public buildings, motor vehicles used for transport, and other non-household indoor locations where people spend much time.

POLLUTANT CONCENTRATIONS

Monitoring Data

Status and Trends in Ambient Concentrations As noted in previous reports (HEI ISOC 2004; WHO 2006a), air quality levels in Asian cities remain well above the maximum levels set by national and international standards and pose a great challenge to Asian megacities as their economies continue to grow at a record pace. Figure 24 shows summary 5-year average concentrations (from 2000–2004) of PM_{10}, SO_2, and NO_2 in 20 Asian cities. PM_{10} is the routinely monitored air pollutant of greatest concern in all these cities, with Beijing, Dhaka,

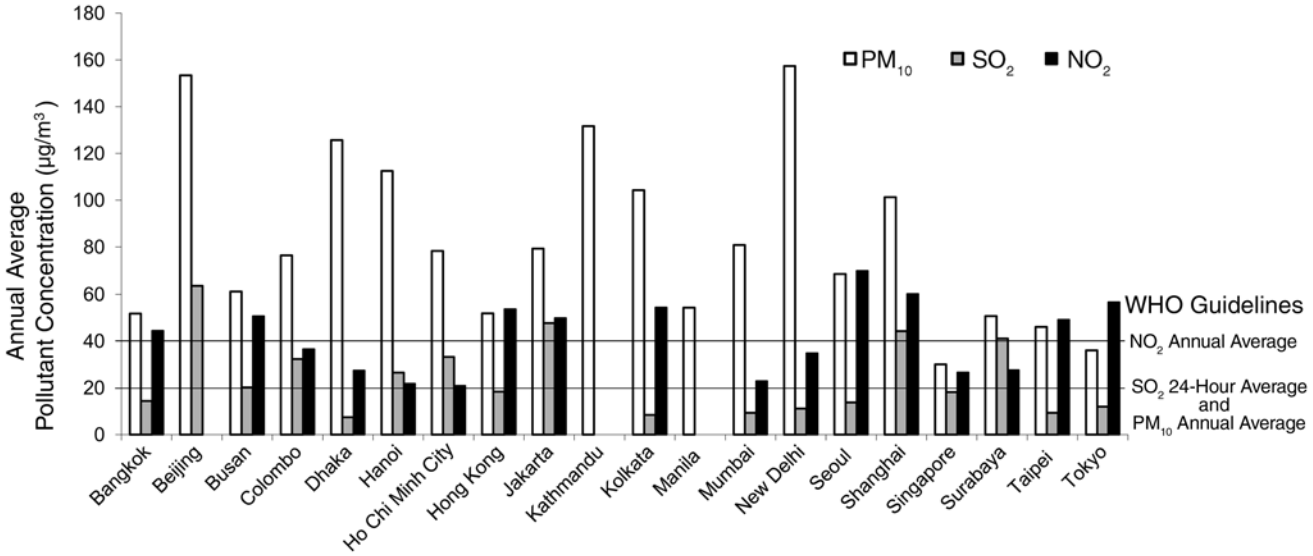


Figure 24. Five-year (2000–2004) average PM_{10}, SO_2, and NO_2 concentrations in selected Asian cities. Standards from WHO air quality guideline, 2005 Global Update (WHO 2006): PM_{10} annual average, 20 μg/m³; SO_2 24-hr average, 20 μg/m³; and NO_2 annual average, 40 μg/m³. [Reprinted with permission from CAI-Asia (*www.cleanairnet.org/caiasia*; accessed January 2008).]

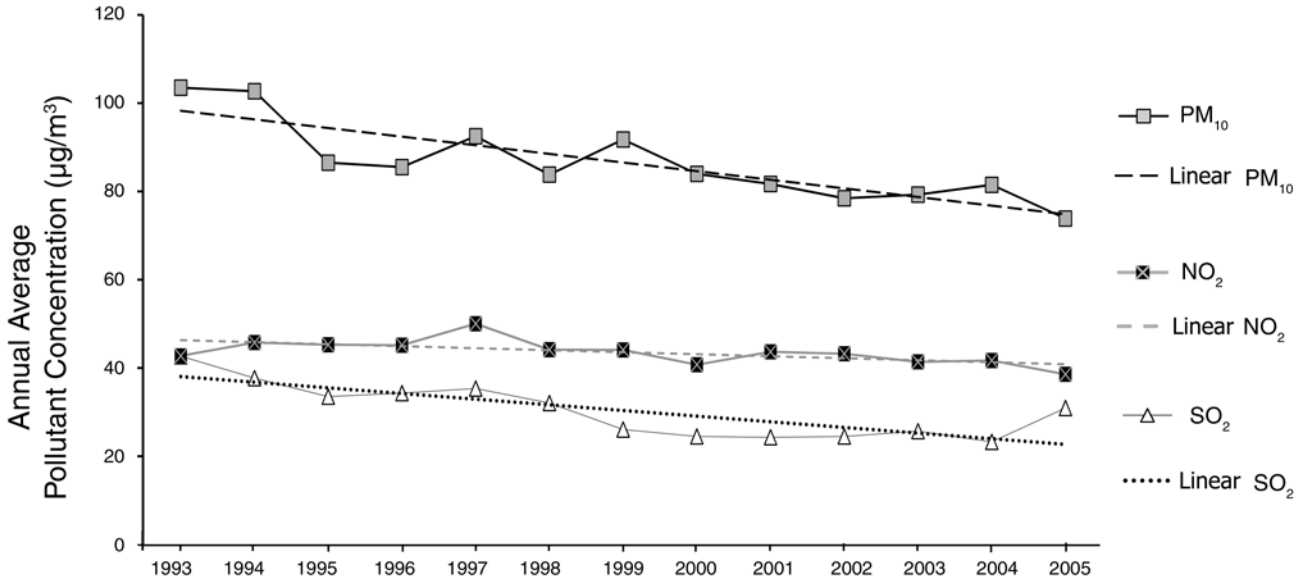


Figure 25. Annual averages of PM_{10}, SO_2, and NO_2 aggregated among selected Asian cities, 1993–2005. Data averaged among the cities listed in Figure 24. The straight lines are smoothed estimates of trends in air pollution levels. [Reprinted with permission from CAI-Asia (*www.cleanairnet.org/caiasia*; accessed January 2008).]

Hanoi, Kathmandu, Kolkata, New Delhi, and Shanghai reporting annual averages > 5 times the WHO guidelines' limit on PM_{10} of 20 μg/m^3. Although $PM_{2.5}$ is not yet part of most regulatory ambient air quality monitoring networks in Asia, several studies have conducted systematic monitoring of $PM_{2.5}$ and PM_{10} in Asian cities (Oanh et al. 2006; Hopke et al. 2008). These reports suggest that annual average $PM_{2.5}$ concentrations are generally above 25 μg/m^3 and as high as 150 μg/m^3, with $PM_{2.5}$:PM_{10} ratios ranging from roughly 0.4 to 0.7 in urban areas of rapidly developing countries in Asia. On the basis of these studies, long-term concentrations well above the WHO guidelines' $PM_{2.5}$ limit of 10 μg/m^3 would appear to be the norm in urban areas throughout much of Asia. Short-term concentrations and concentrations measured at high-impact locations (e.g., in traffic) indicate that concentrations of PM much higher than the estimated annual average are also present intermittently or in specific geographic areas.

The WHO limits for SO_2 and NO_2 are 50 μg/m^3 and 40 μg/m^3, respectively, with only Beijing exceeding the SO_2 limit and 9 cities (Bangkok, Busan, Hong Kong, Jakarta, Kolkata, Seoul, Shanghai, Taipei, and Tokyo) of 20 major cities exceeding the NO_2 limit (Figure 24).

Annual trends in air quality (1993–2005) across major Asian cities suggest a significant downward trend in annual average SO_2 concentrations in urban areas in contrast to the increase in overall country-level emissions (Figure 25), with the exception of an average increase in SO_2 concentration during 2005. These SO_2 reductions occurred in spite of increasing fuel consumption. Regulations requiring the use of low-sulfur fuels and relocation of major coal-fired power plants and industrial facilities to outside of cities were responsible for these decreases. Overall, the trends in urban air quality measurements of SO_2 broadly follow the emission estimates described earlier in this section. However, the emission estimates suggest a substantial increase in SO_2 emissions at the country level in Asia that has been driven by increases in industrial emissions in China since 2002 — a trend confirmed by the Chinese State Environmental Protection Administration (SEPA; NAE and NRC 2008). SO_2 concentrations measured in Chinese cities also reflect the emissions trends, showing large decreases until 2000 but increases in some cities (particularly in north China) after 2000 (Hao and Wang LT 2005). Figure 26 shows trends in SO_2 concentrations in several large Chinese cities for 1980–2005.

PM_{10} concentrations in Asia also have been declining (Figure 25); the aggregated annual average in Asian cities decreased by approximately 25% between 1993 and 2005, as compared with the reduction by about 50% in SO_2 concentrations over the same period. More recent data from the capital cities of several Chinese provinces, over 2003–2006 (Figure 27), do not show consistent decreases, although some local reductions in PM_{10} concentrations have been reported. For example, in Huainan (NAE and NRC 2008), an industrial city of 2.3 million people, increased government regulation and closing of highly polluting sites resulted in decreases in PM_{10} concentrations between 2002 and 2005, although concentrations still exceed the Chinese class II limits of 100 μg/m^3 as the annual average.

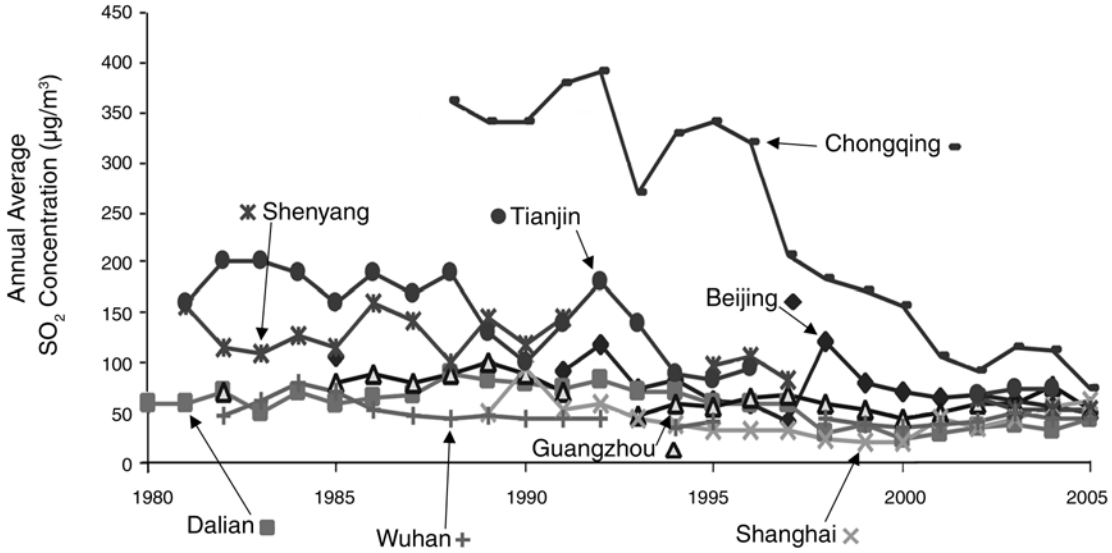


Figure 26. Annual average SO_2 concentrations in several large cities in China. [Adapted from National Academy of Engineering and National Research Council 2008, with permission from the National Academies Press, National Academy of Sciences.]

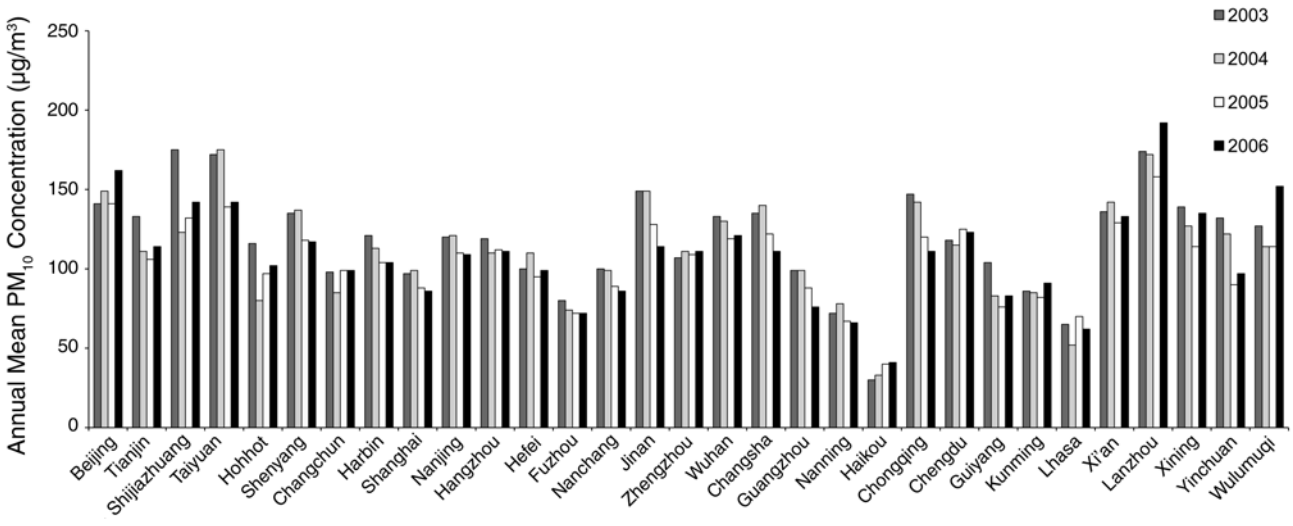


Figure 27. PM_{10} concentrations in capital cities of Chinese provinces, 2003–2006. [Data supplied by National Statistic Communiqué, National Bureau of Statistics of China 2004. Data supplied by National Statistic Communiqué 2008.]

The downward trend in the aggregate annual average NO_2 concentration (Figure 25) is quite modest, < 10% from 1993 through 2005. Overall, estimates of NO_x emissions indicate steady increases, especially in China, over this same period. Remote sensing also indicates even greater increases in NO_x concentrations than predicted by the REAS emissions inventory, especially in China (both urban and rural areas) (He Y et al. 2007).

The apparent discrepancy between the stable or decreasing concentrations measured in urban areas and the sharply increasing emission trends and remote-sensing (satellite-based) measurements is likely related to large increases in NO_x concentrations throughout the Yangtze River Delta (27% per year for 2000–2005) and in rural areas of eastern China (18% per year), beginning in 2000 (He Y et al. 2007). These areas are less likely than urban

Sidebar 8. AIR QUALITY TRENDS IN BANGKOK, THAILAND, A METROPOLITAN AREA OF DEVELOPING ASIA

Trends in annual gasoline and diesel fuel consumption (for 1992–2005) within Bangkok, the Bangkok metropolitan region, and Thailand overall are shown in Figure 8.1. Increases in the consumption of fuel results in increased emissions of SO_2, CO, and NO_x and should subsequently result in increased ambient concentrations of these pollutant species. In the case of the Bangkok's air quality, however, it is apparent that these ambient concentrations have been partially mitigated by the emission-control strategies implemented in the city over the past decade. Ambient concentrations of CO and NO_2 (Figure 8.2) and of SO_2 and PM_{10} (not shown) measured by traffic and roadside monitors in Bangkok suggest a trend of reduced emissions, with distinct downward trends in annual ambient concentrations of CO and SO_2 and smaller reductions in annual NO_2 and PM_{10} concentrations.

The emission controls in use in Bangkok include (1) the installation of catalytic converters on all cars after 1993, resulting from the adoption of vehicle-emissions standards; (2) incentives to shift from two-stroke to four-stroke engines; (3) roadside inspection of vehicles visibly emitting gross amounts of exhaust; (4) establishment of a routine inspection and maintenance programs *Continued on next page*

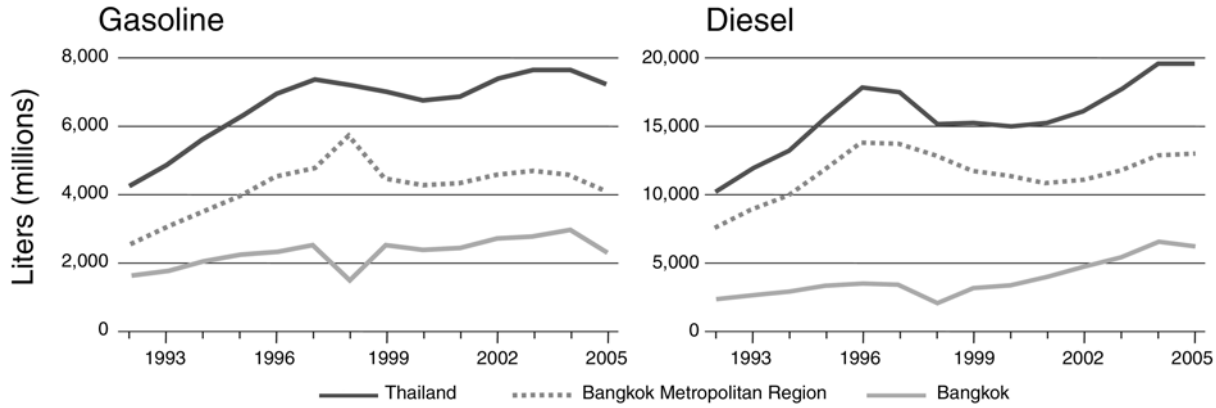


Figure 8.1. Annual gasoline and diesel fuel consumption in Bangkok, the Bangkok metropolitan region, and Thailand, 1992–2005. [Reprinted from Asian Development Bank and CAI-Asia 2006b, with permission from CAI-Asia.]

areas to have data on the impact of these emissions on air quality. Also contributing to this discrepancy may be emission estimates that (1) do not adequately consider recent changes in emission factors associated with urban air quality, regulations and management programs, and improved technology, or (2) are regional or national rather than city-specific and therefore do not capture city-level management efforts, or (3) emissions that occur above the atmospheric layer in which pollutant mixing occurs. Furthermore, Figure 25 presents data aggregated across major Asian cities, whereas the increase in NO_x emissions is dominated by increases in China specifically. Fine PM ($PM_{2.5}$) concentrations in cities, if measured (as they have been only recently in select Asian cities), would also be expected to show a downward trend, reflecting the lower ambient SO_2 concentrations in many urban areas and reduced secondary, fine-particle SO_4^{2-} production.

To better understand these trends and the air quality challenge Asian cities face in light of increasing populations, energy consumption, and vehicle numbers and vehicle-miles traveled, Sidebar 8 presents a case study of air quality trends in Bangkok — one of the Asian cities considered in the aggregate trends in Figure 25. This case contrasts with trends in concentrations of photochemical oxidants and oxidant precursors in Tokyo, which is discussed in Sidebar 9. In Tokyo, Japanese emission controls have resulted in reduced oxidant-precursor concentrations over several decades, with subsequent reductions in oxidant concentrations, but in recent years oxidant concentrations have begun to increase while precursor concentrations continue to fall. This shift may be explained by continually increasing concentrations of oxidants (corresponding to increased emissions) in the region and increasing concentrations of transported oxidants from neighboring countries upwind.

Sidebar 8: AIR QUALITY TRENDS IN BANGKOK (*Continued*)

for all registered vehicles; (5) introduction of natural gas as an alternative fuel for the transport sector in 2003; (6) removal of lead from gasoline and reduction of the sulfur content of diesel fuel from 1.0% to 0.5% by weight in 1996; and (7) a cap on the sulfur content of fuel oil numbers 1 and 2, such that it cannot exceed 2% by weight.

The reduction in CO concentrations measured by roadside monitors after 1993 (Figure 8.2), when catalytic converters were introduced, and the reduction in SO_2 concentrations after 1996, when sulfur content of diesel fuel was reduced, are both apparent in the trend data. The small downward trends in roadside NO_x and PM_{10} concentrations may also be associated with the introduction of catalytic converters and lower-sulfur diesel as well as the shift from two-stroke to four-stroke engines. But these trends are not much more pronounced than the interannual variability in the data, and the trend for PM_{10} is more affected by regional contributions than local contributions and does not vary significantly with the contribution from roadside emissions. Several more years of data will be needed to detect a statistically significant trend. It is also worth noting that the dip in fuel consumption in Bangkok in 1998 (Figure 8.1) has been associated with the Asian economic crisis and also reflects a major reduction in new-vehicle registrations in Bangkok in that year.

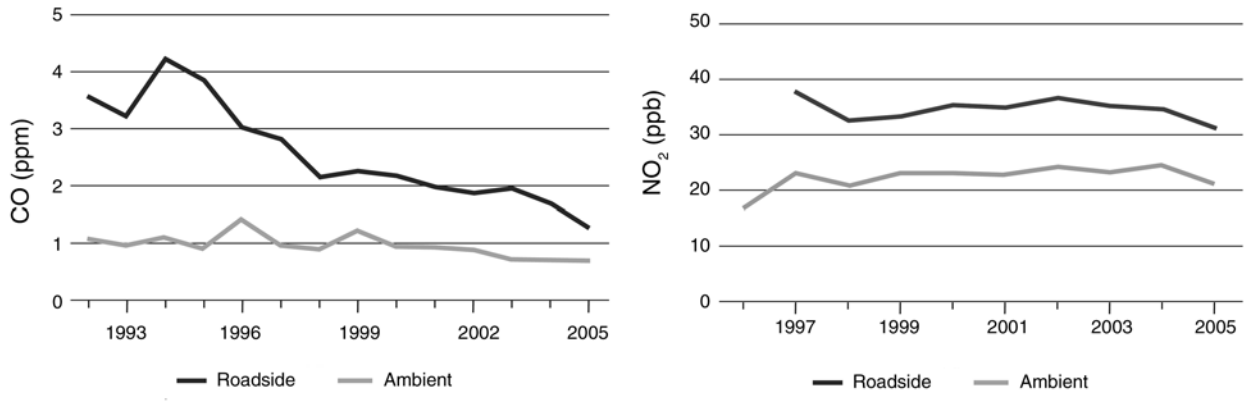


Figure 8.2. Annual CO and NO_2 concentrations measured by roadside and urban ambient monitors in Bangkok. Note the difference in the range of years on the x axes. [Reprinted from Asian Development Bank and CAI-Asia 2006b, with permission from CAI-Asia.]

Sidebar 9. PHOTOCHEMICAL OXIDANTS IN JAPAN

The mitigation of photochemical oxidants, O_3 in particular, remains a challenge to major metropolitan areas around the world. From 1970–1990, Tokyo reduced ambient oxidant concentrations by nearly 50% through the implementation of aggressive emission controls on the oxidant precursors VOCs and NO_x (Figure 9.1). But reductions lessened after 1990, resulting in an increase in the O_3 concentration in 2003 by approximately 40% over the 1989 annual mean low of 0.018 ppm.

Figure 9.2 shows the annual oxidant data for Tokyo in terms of the number of hours in which the concentrations exceeded the air quality standard. These data show the reversal in the downward trend in oxidant concentrations, beginning around 1989. They also show a significant and unprecedented increase since 2000 in the number of hours in which oxidant concentrations exceed 0.12 ppmV, suggesting that, on average, oxidant episodes are more severe than in the past 30 years.

There are at least two possible explanations for the reversal in the oxidant trend observed in metropolitan Tokyo. The first relates to changes in the ambient VOC/NO_x ratio in Tokyo, which has decreased from about 14:1 in the mid-1970s to about 6.5:1 in the past decade. The changing ratios move the oxidant-control environment from a VOC-limited regime (in the 1970s–1980s) to a NO_x-limited regime (from the 1990s–present). The changes in the VOC/NO_x ratio can affect the production of photochemical oxidants and the efficiency of precursor-control strategies, which in turn can alter the spatial distribution of oxidants within the metropolitan area. Lower VOC/NO_x ratios typically delay oxidant formation, resulting in concentrations peaking at farther distances from the urban emission sources. This explanation for increasing oxidant concentrations in Tokyo is conceivable but unlikely, because decreasing concentrations of both precursor components would most likely result in a net decrease in oxidant concentrations when averaged over space and time.

The second possible explanation relates to potential oxidant increases over the past decade in the air that flows into the Tokyo metropolitan area from other regions. The rapidly growing economies in the regions surrounding Tokyo have resulted in significant increases in precursor emissions that have likely increased oxidant concentrations in those regions (see *Trends in Energy Use and Projected Growth in Major Point Sources and Emissions* in Section II for discussion of trends in NO_x and NMVOC emissions). Those higher oxidant concentrations have most likely resulted in higher background concentrations and therefore enhanced interregional transport of oxidants into the Tokyo area. For another example, based on satellite observations, NO_2 concentrations in China have been increasing significantly in the past decade as well (Figure 9.3; van der A et al. 2006).

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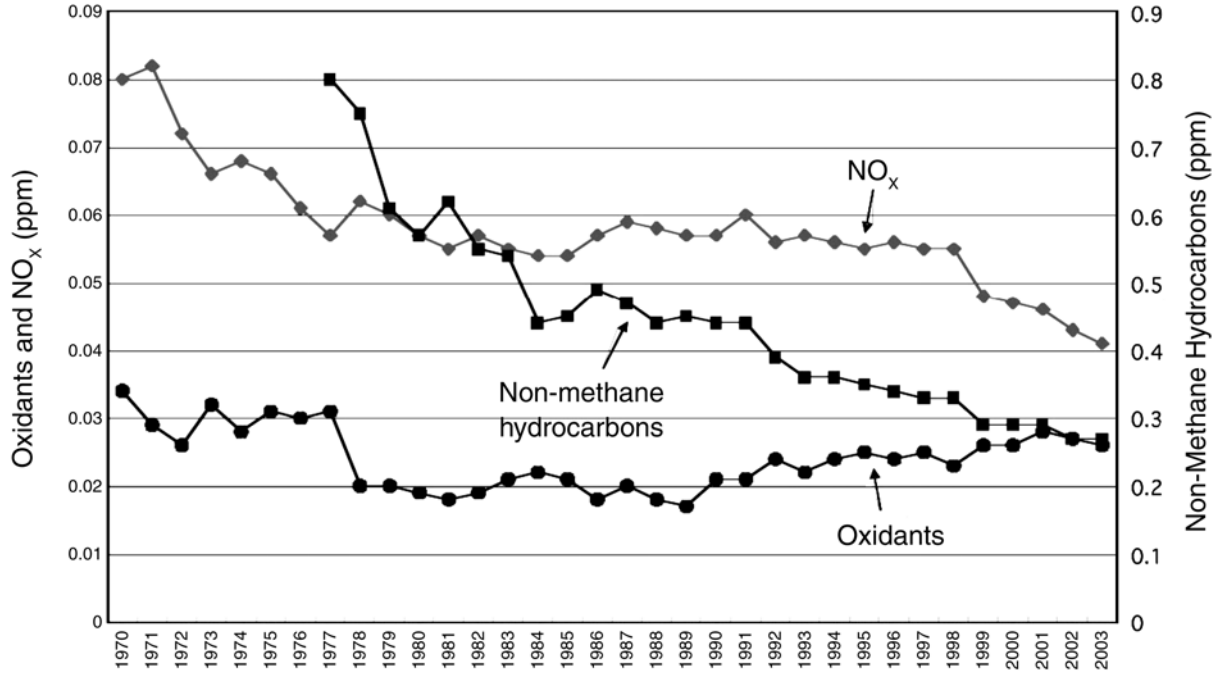

Figure 9.1. Annual average concentrations of oxidants and their precursors in Tokyo, 1970–2003. Data are the averages of measurements at 23 stations. [Adapted from the Report of the Committee on Photochemical Oxidant Control, Tokyo Metropolitan Government 2007.]

Sidebar 9. PHOTOCHEMICAL OXIDANTS IN JAPAN (*Continued*)

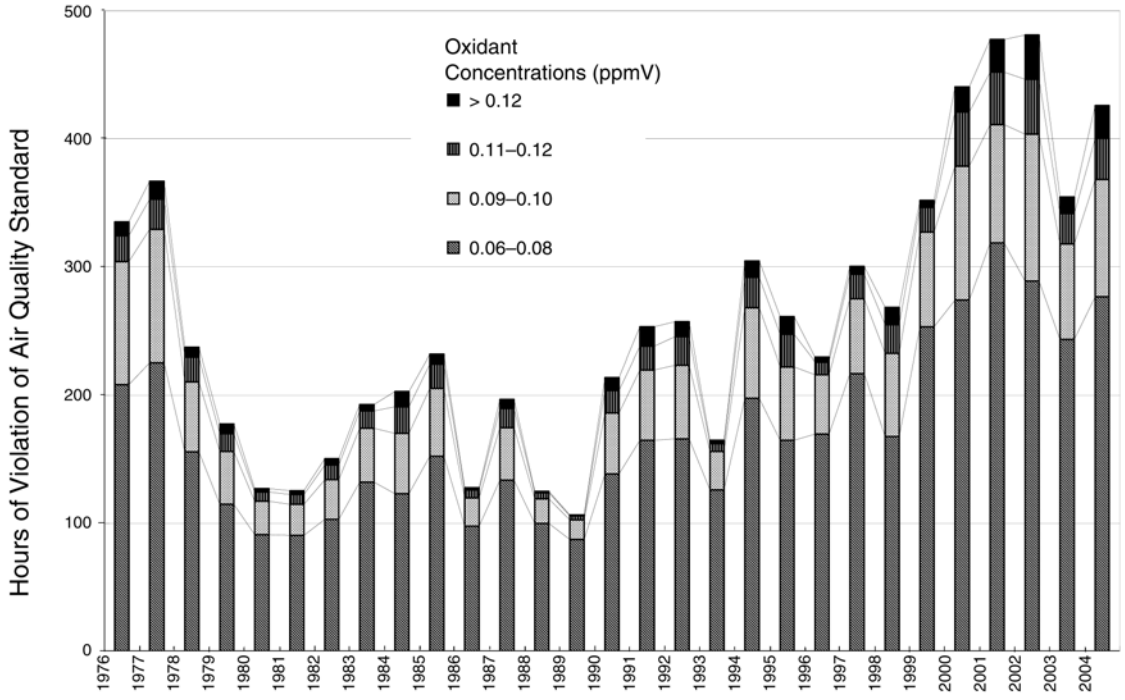

Figure 9.2. Annual numbers of hours in which oxidant concentrations [in parts per million by volume] in Tokyo exceeded the air quality standard. [Adapted from the Report of the Committee on Photochemical Oxidant Control, Tokyo Metropolitan Government 2007.]

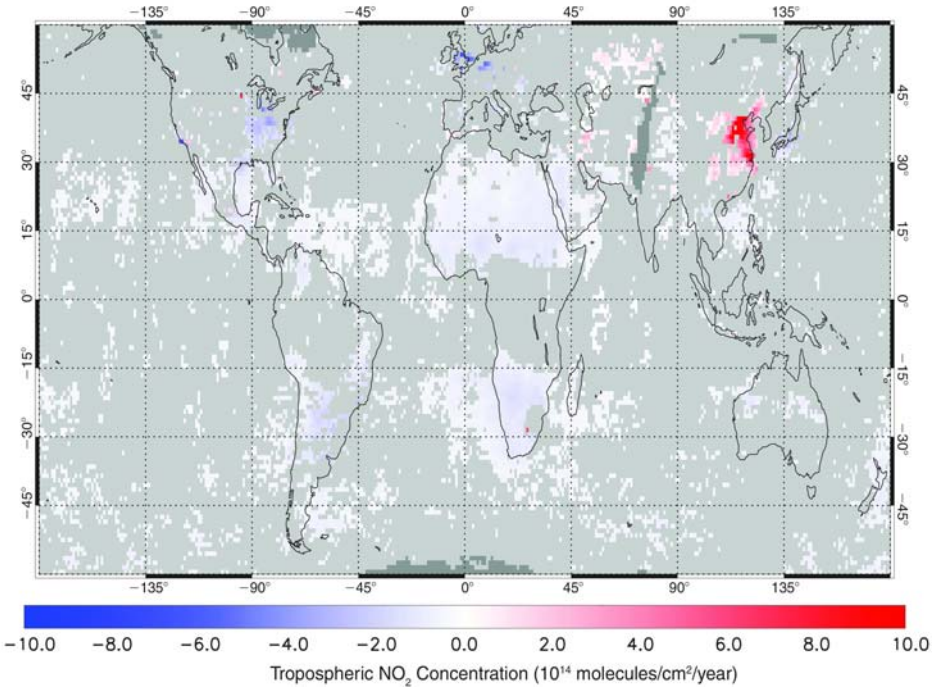

Figure 9.3. Trend in annual tropospheric NO_2 concentrations, 1996–2005. Data are derived from observations recorded by GOME and SCIAMACHY satellites. Light gray areas are those for which no significant trend was found, and dark gray areas are those for which not enough observations were available to construct a time series. [Reprinted from van der A et al. 2006 with permission from American Geophysical Union.]

Current Capabilities and Coverage of Air Quality Monitoring Air quality monitoring capabilities across major Asian urban centers have improved dramatically over the past decade. The deployment of air quality networks has improved both in terms of the instrumentation technologies used and the proportion of the population living in areas with monitoring networks. In addition, many developing countries have introduced sophisticated measurement networks through collaborations and partnering arrangements with developed countries. Table 7 summarizes the key features of the monitoring networks in select Asian cities: the number of monitoring stations within the city centers, the key pollutants measured, the proximity of monitors to local sources (e.g., within traffic, at the roadside, or at industrial sites), an estimate of the overall city population and population coverage by the monitoring site, the sampling frequency and averaging time (the period over which pollution concentrations are averaged in an analysis) used for the measurements, and Web sites that provide access to metadata or measurements.

Modeled Estimated Pollutant Concentrations and Projected Trends in Emissions

As discussed in the previous section, ambient concentrations of pollutants are typically measured through a network of fixed monitoring stations. The cost of maintaining such a network, including expenses related to maintaining the technical capacity to use information generated from such networks, has limited the number of monitors and monitoring networks. The number of monitoring stations has grown rapidly in the past few years, but most urban areas in Asia still lack adequate numbers of stations. Furthermore, the limited monitoring data are often not publicly available or accessible for use in a systematic examination of pollutant concentrations across cities or over time. For instance, the Global Burden of Disease Comparative Risk Assessment study conducted by WHO (Cohen et al. 2004) reported on 304 cities worldwide with monitored data for TSP or PM_{10}, with only 32 of these cities in Asia.

For purposes of examining temporal trends in ambient concentrations in Asia, we used results from the GMAPs. This model was used in 2002 to estimate the global burden of disease from outdoor urban air pollution.

GMAPs is a fixed-effects model designed to predict PM_{10} concentrations in residential areas of world cities with populations over 100,000, by determining the relation between emissions and ambient concentrations. It is specifically based on the relation between monitored PM concentrations at fixed locations and national and local factors affecting ambient pollution during the 1985–1999 period. The national factors represented are per capita income, total energy consumption, the fuel mix in use, the amounts of fuels used for transportation, and a composite factor including fuel types, total population, and population density.[c] The local factors represented are the overall city population, the city population density, and a suite of 18 variables reflecting geographic and long-term climatic characteristics and the location of the monitoring sites.[d]

The GMAPs explains 88% of the observed variation in PM_{10} concentrations in the 304 cities with monitoring data, including the 32 cities in Asia. The correlation coefficient (r) between model predictions and actual monitoring data for PM_{10} in Asian cities was 0.82, based on 95 estimates from 15 cities in South, Southeast, and East Asia and the Western Pacific (excluding Australia and New Zealand). The correlation for TSP was 0.84, based on 213 estimates in 26 cities. The model estimates indicate that higher energy consumption and greater reliance on dirtier fuels such as coal and biomass result in higher PM concentrations. On the other hand, as countries have grown richer, they have successfully put in place polices and regulatory regimens to reduce ambient PM concentrations for a given level of combustion activity — such as stringent standards for diesel in India, the phasing out of vehicles that are old or require dirty fuel in Bangkok (Sidebar 8), and moving industrial sites away from population centers in Katmandu (CAI-Asia Center 2006b). The model results also indicate declines in PM concentrations of between 1% and 3.5% per year over and above those that can be directly attributable to combustion sources or to historical improvements in policies as income levels rose, suggesting that countries are more proactive in managing air pollution than in the past. This unexplained decline in PM concentrations

[c] The national variables in the GMAPs include per capita consumption of coal, oil, natural gas, nuclear power, hydroelectric power, and combustible renewables and wastes to account for total energy consumption and the fuel mix; rates of emissions from various sources; economic activity; and intensity of fuel use. All these national variables are compared across countries. National factors also include per capita gasoline and diesel use in the transport sector, an important source of particle emissions.

[d] Local variables include: the annual averages (and standard deviations, representing seasonal changes) of monthly mean temperatures, diurnal temperatures, precipitation, barometric pressure, wind speed, percent cloud cover, and frequencies of wet days, sunny days and frosty days. The climatic variables were constructed from a global mean monthly climatology map with a resolution of $0.5° \times 0.5°$ latitude and longitude, developed by researchers at the Climate Research Unit of the University of East Anglia (New et al. 1999). For each city, all the climate variables were based on estimated conditions at the city center. In addition, two meteorologic variables related to energy demand — heating and cooling degree-days (in Fahrenheit) — were estimated from the mean monthly temperature for each city. Elevation and distance from the coast were included to consider topographic factors related to atmospheric dispersion and transport of pollutants. The model also included a measure of the local population density, calculated on the basis of population grids from a map of the world population (Center for International Earth Science Information Network 2004).

Table 7. Summary of Monitoring Systems in Selected Asian Cities[a]

City and Country	Stations (*n*)	Pollutants Measured[b]	Site Characteristics	Total Population (millions) and Population Density (persons per km^2)	Sampling Frequency and Averaging Time	Relevant Web Site[c]
Bangkok, Thailand	17	CO, NO, NO_x, NO_2, SO_2, O_3, PM_{10}	13 Urban, 4 in traffic, and 13 with meteorology	5.6 ~ 3700	Continuous 1-hr average gaseous pollutants and PM_{10}	*www.aqnis.pcd.go.th/station/allstation.htm* *www.pcd.go.th*
Beijing, China	27	CO, NO_2, SO_2, O_3, TSP, PM_{10}, lead	14 Urban and 13 other sites	10.9 ~ 650	Continuous 1-hr average gaseous pollutants and PM_{10}, 24-hr average TSP and lead sampled every 6th day	*www.bjepb.gov.cn*
Dhaka, Bangladesh	1	CO, NO, NO_x, SO_2, O_3, PM_{10}, $PM_{2.5}$	1 at city center	12 ~ 40,000	Continuous 1-hr average gaseous pollutants and 24-hr average PM sampled every 3rd and every 6th day	*www.doe-bd.org*
Hanoi, Vietnam	7	CO, NO, NO_x, SO_2, O_3, TSP, PM_{10}	5 Urban, 2 in traffic, and 1 with meteorology	4.0 ~ 4400	Continuous 1-hr average gaseous pollutants and 24-hr average PM sampled every 3rd and every 6th day	*www.cleanairnet.org/caiasia/1412/csr/vietnam.pdf*
Ho Chi Minh City, Vietnam	9	CO, NO, NO_x, SO_2, O_3, TSP, PM_{10}	5 Urban, 4 in traffic, 1 with meteorology	4.85 ~ 2300	Continuous 1-hr average gaseous pollutants and 24-hr average PM sampled every 3rd and every 6th day	*www.hepa.gov.vn*
Hong Kong, China	14	CO, NO, NO_x, NO_2, SO_2, O_3, TSP, PM_{10}, lead	11 Urban, 3 in traffic, and 14 with meteorology	7.0 ~6300	Continuous 1-hr average gaseous pollutants and RSP (PM_{10}), 24-hr average TSP sampled every 3rd and every 6th day	*www.epd-asg.gov.hk*
Mumbai, India	3	NO_2, SO_2, SPM, RSP/PM_{10}, hydrogen sulfide, ammonia	2 Residential and 1 industrial	17.3 ~ 39,800	24-hr average concentrations sampled twice per week (104 samples/year)	*www.cpcb.nic.in/Air/monitoringNetwork.html*
Delhi, India	10	NO_2, SO_2, SPM, RSP/PM_{10}	11 Total, divided into residential, industrial, and traffic-level	14.2 ~ 9600	4-hr average gaseous pollutants over 24 hr, and 8-hr average PM sampled twice per week (104 samples/year)	*www.cpcb.nic.in/Air/monitoringNetwork.html* *www.cpcb.nic.in/Air/air_quality_of_delhi.html*
Seoul, South Korea	27	CO, NO_x, SO_2, O_3, PM_{10}, dust, lead		9.7 ~ 16,000	Continuous 1-hr average gaseous pollutants and PM_{10}	*www.cleanairnet.org/caiasia/1412/article-58984.html*
Shanghai, China	44	CO, NO_x, SO_2, O_3, TSP, PM_{10}, dust, lead	21 Automated stations	12.8 ~ 2000	Continuous 1-hr average gaseous pollutants and PM_{10} and 24-hr average gaseous pollutants and PM_{10} (except O_3, for which 8-hr averaging was performed [10AM–6PM])	*www.sepb.gov.cn/english/main.jsp*
Taipei,China	8	CO, NO_2, SO_2, O_3, PM_{10}	5 Automated stations	2.5 ~ 9200	Continuous 1-hr average gaseous pollutants and PM_{10}	*www.epa.gov.tw/en/*
Wuhan, China	7	NO_2, SO_2, PM_{10}	7 Automated stations (4 urban and 3 regional industrial)	9.7 ~ 947	Continuous 1-hr average gaseous pollutants and PM_{10}	*www.whepb.gov.cn/CMSWEB/default.aspx*

[a] Data compiled from CAI-Asia 2006b, Schwela et al. 2006, and the Web sites listed.

[b] Not all pollutants are measured at all stations in the city. In addition, some sites report total hydrocarbons/VOCs, methane, non-methane hydrocarbons, and selected air toxics. All stations typically report meteorologic parameters: wind speed and direction, temperature, relative humidity, barometric pressure, and solar radiation.

[c] All Web sites were accessed in January 2008.

is of greater magnitude in poorer countries (a 3.5% decline at per capita incomes of U.S. $300, vs. a 1% decline at per capita incomes of U.S. $10,000).

The GMAPs can be used to predict PM concentrations in a city on the basis of economic activity, energy efficiency, emission factors, and city-specific, time-invariant geoclimatic factors. The predictions assume that cities located in similar climatic and geographic environments would have similar PM concentrations for any given level of economic and combustion activity. The model can also be used to predict changes in PM concentrations for cities on the basis of changes in the city population and in national income, energy consumption, fuel mix, and a time trend specific to national income level. Model estimates for a city for a given year are based on the impact of the geoclimatic factors and the observed variations in the time-varying country-level variables. Since the model accounts for differences in economic activity within a country in a limited manner, the estimates for cities with monitoring data are improved by adjusting the PM_{10} estimates by the average residual between monitored data and predicted data. The GMAPs estimates that are provided in this report, however, are based on input data and measurement data that have not been updated beyond 2000. Accordingly, the estimates for the more recent years may include greater levels of uncertainty. There is considerable uncertainty around the estimates in general, especially for countries with no or limited monitoring data; nevertheless, the GMAPs results can be a useful first estimate, especially for countries without monitoring networks.

Figure 28 shows the time trend in the city population–weighted average PM_{10} concentrations for various regions in Asia, based on predictions from the GMAPs. Overall, the average concentrations in China, India, and Other Southeast Asia are more than double those in High-Income Asia. The average concentration in Other South Asia is nearly twice those in China, India, and Other Southeast Asia. Overall, PM_{10} concentrations have been declining in all regions in Asia, despite increased energy consumption. Three primary factors are likely to be driving this downward trend: increased energy efficiency, switching of fuel from biomass and refuse toward cleaner fuels, and declines in emission factors associated with changing technology.

The extent of the decline in PM_{10} concentrations in Asia varies from region to region and is greatest in Other South Asia, which had the highest initial concentrations. A large part of the decline in Other South Asia is a direct result of

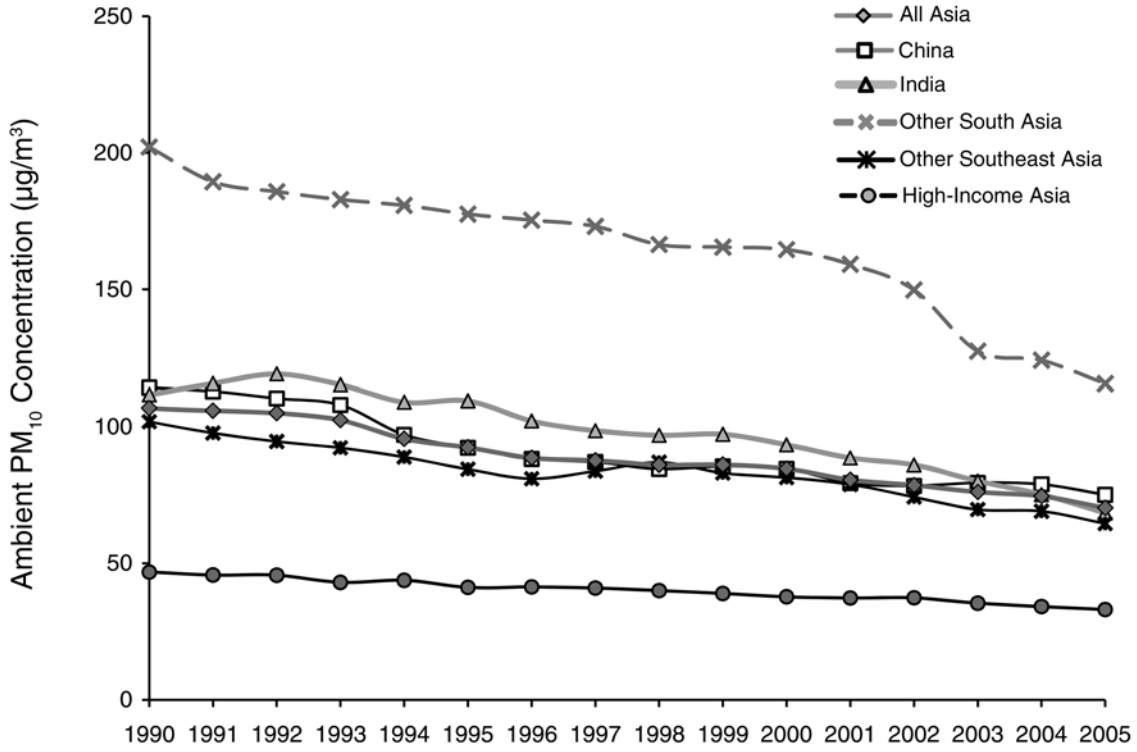

Figure 28. Trends in urban ambient PM_{10} concentrations in urban areas in Asia. Data are estimates from GMAPs. Regions are defined in Table 1. Values for All Asia are weighted averages of the regions. [Data compiled from World Bank 2008.]

steps taken by Pakistan to reduce its oil imports by promoting local CNG and LPG for use in the transport, agriculture, and power sectors and to make CNG the fuel of choice for power generation.

Average PM concentrations in China declined gradually throughout the 1990s, despite the steady rise in total energy consumption, partially as a result of changes in government policy. These changes included relocating pollution sources away from population centers and introducing more stringent vehicle and stationary-source emission standards, such as desulfurization of power plants (NAE and NRC 2008). Existing policies have not been sufficient to mitigate the emissions resulting from the sharply increasing energy demand since 2000. As a result, population-weighted average PM concentrations in 2005 were approximately the same as those in 2000. Furthermore, the strategy of relocating sources outside of populated areas as an approach to reduce exposure may not be sustainable if new urban areas develop adjacent to the relocated facilities. PM concentrations have most likely been rising in many Chinese cities over the same period during which they have been falling in others.

Average PM concentrations in India have declined with the declining share of diesel in the transport sector since the early 1990s as a result of various measures taken by the government, including progressively more stringent emission standards, fuel quality improvements, and incentives to convert transport vehicles from diesel fuel to CNG.

Reduced reliance on biomass fuels has also contributed to the steady decline. Similarly, PM concentrations in Other Southeast Asia declined steadily during the early 1990s, leveled off for a few years around the time of the Asian economic crisis (the late 1990s), and have since continued their downward trajectory.

Overall, estimates of emissions, as well as measurements and estimates of pollutant concentrations, indicate that air quality is improving throughout much of urban Asia. The improvement in air quality during periods of dramatically increased energy use in Asia is a testament to the impact of effective air quality management as well as improved efficiency and reduced intensity of energy use. Climate change and the emission of GHGs present an important challenge for Asia but are also an opportunity for continued improvement in air quality. Specific strategies for GHG-emission reduction, though directed toward climate change, may also have a direct impact on local and regional air quality (Sidebar 10). International funding for reducing GHG emissions may result in faster and larger improvements in air quality than would otherwise occur in the region.

Current Source-Apportionment Studies in Asia

Source apportionment quantitatively relates source emissions to their impact on ambient air pollution. The method is useful for relating observed concentrations and trends in concentrations to categories of emission sources. Source apportionment can be performed with dispersion modeling or receptor modeling. In dispersion modeling, a pollutant emission rate and meteorologic information are used to predict the resulting pollutant concentration at a point in space and time. In receptor modeling, chemically speciated ambient air contaminants measured at a particular location are used to identify and quantify the sources contributing to the measured pollutant concentration at that receptor location. Although both modeling approaches are used in source apportionment, receptor modeling is most common. In Asian cities, source apportionment based on receptor modeling is particularly useful, since it does not require detailed emissions inventories, which are costly and time consuming to conduct.

Source-Apportionment Studies in Asia In this section, we summarize studies in Asian countries, conducted since 1995, that have used receptor modeling to estimate source contributions. Although many of these studies have a limited number of samples and should therefore be interpreted with caution, they provide useful quantitative insight into the source makeup of air pollution in many Asian cities. We identified the studies by searching the peer-reviewed literature through the Web of Science, PubMed, and Google Scholar, with supplementary searches of major air pollution journals (Table 8).

Table 8. Source-Apportionment Studies in Asia, 1995–2007

Country/ City	PM Type	Study Period	Site Type	Method[a]	Citation
Bangladesh					
Dhaka	Fine, coarse	2001–2002	Semi-residential, traffic hot-spot	PMF, CMB	Begum et al. 2004, 2005, 2007
Rajshahi	Fine, coarse	2001–2002	Urban	PMF	Begum et al. 2004
China					
Hong Kong	Fine	2000–2001	Urban, rural	CMB	Zheng M et al. 2006
	PM_{10}			PMF	Lee E et al. 1999
	TSP			PCA	Fung and Wong 1995
Beijing	Fine	1999–2000	Downtown, residential	Chemical	He KB et al. 2001
			Urban, rural		Duan et al. 2004
	Fine	2000	Urban, rural, roadside	CMB	Zheng M et al. 2005
	Fine	2000	Urban, rural, roadside	PMF	Song Y et al. 2006
Kaohsiung County	Fine	Winter 2003	Residential, industrial	Gausian trajectory model	Tsai YI and Chen CL 2006
India					
Delhi	Fine	2001	Residential	CMB	Chowdhury et al. 2007
Mumbai	Fine	2001	Residential	CMB	Chowdhury et al. 2007
Kolkata	Fine	2001	Residential	CMB	Chowdhury et al. 2007
Chandigarh	Fine	2001	Residential	CMB	Chowdhury et al. 2007
Chennai	Fine, coarse	2001–2004	Urban	CMB	Oanh et al. 2006, 2007
Indonesia					
Bandung	Fine, coarse	2001–2004	Urban	CMB	Oanh et al. 2006, 2007
South Korea					
Seoul	TSP		Urban residential, suburban, and industrial	PCA	Kim KH et al. 2006, Baek et al. 1997
Gosan	Fine	Spring 2002	Background	PMF	Han et al. 2006
Sihwa industrial area	Fine	1998–1999	Residential, industrial	CMB	Park SS et al. 2001
Busan	Fine	Winter 2002	Background	Chemical	Kim KH et al. 2006
Maldives and Indian Ocean					
Various background sites	Fine	2001	Background	Chemical	Ramanathan et al. 2001 Chowdhury et al. 2001
Philippines					
Manila	Fine, coarse	2001–2004	Urban	CMB	Oanh et al. 2006, 2007
Thailand					
Bangkok	Fine, coarse	1995–1996	Urban, suburban residential	PMF	Chueinta et al. 2000
	Fine, coarse	2001–2004	Urban	CMB	Oanh et al. 2006, 2007
Vietnam					
Ho Chi Minh City	Fine, coarse	1992–1996	Urban	PCA	Hien et al. 1999, 2001
Hanoi	Fine, coarse	2001–2004	Urban	CMB	Oanh et al. 2006, 2007

[a] CMB indicates chemical mass balance; PCA indicates principal-components analysis; PMF indicates positive matrix factorization. The chemical method quantifies the chemical makeup of the air pollution sources from ambient chemical elements or compounds used as tracers.

Sidebar 10. HEALTH CO-BENEFITS OF POTENTIAL ADAPTATION AND MITIGATION MEASURES FOR CLIMATE CHANGE IN ASIA

BACKGROUND

In their 2007 report, the Intergovernmental Panel on Climate Change concluded that "there is high agreement and much evidence that in all analyzed world regions near-term health co-benefits from reduced air pollution, as a result of actions to reduce GHG emissions, can be substantial and may offset a substantial fraction of mitigation costs." Health co-benefits, or ancillary benefits, of GHG mitigation policies are defined as health improvements other than those caused directly by reductions in GHG emissions in particular that arise as a consequence of mitigation policies (Davis et al. 2000). Potential health co-benefits are not limited to those from air quality mitigation; for example, the promotion of walking, cycling, and other active modes of transport rather than driving will likely prevent some of the 1.9 million annual deaths associated with physical inactivity and the 1.2 million deaths due to road-traffic accidents. Incentives to use biofuels could affect food availability and prices, in turn, could affect the 3.5 million annual deaths from under-nutrition.

The WHO and other agencies are beginning to describe in qualitative terms the range of links between energy policies and health. Better understanding of these links will help inform decision makers about the potential health benefits and harms of various policies to mitigate GHG emissions. However, assessments of these links have not been systematic, have not typically used standard methods developed for outdoor and indoor air pollution, and have not included all sectors that can affect health. The economic benefits (and potential costs) of the links between policy and health are likely to be realized long before the benefits of climate change mitigation efforts. The accurate and comprehensive assessment of co-benefits a therefore a critical component of decision-making about climate policy and lends urgency to the development of methods and research agendas to close knowledge gaps in this area.

ASSOCIATIONS BETWEEN ENERGY USE AND AIR POLLUTION

More than any other sector, energy production and use is associated with environmentally mediated, premature morbidity and mortality, primarily through exposure to harmful indoor and outdoor air pollutants (Smith and Haigler 2008). The WHO's global Comparative Quantification of Health Risks project estimated that approximately 2.4 million premature deaths in 2000 were associated with indoor and outdoor particulates from the combustion of fuels, including both biomass and fossil fuels (Ezzati et al. 2004). A substantial scientific literature has established that the greatest impacts are occurring in populations nearest to emission sources: people living in poverty worldwide who use dirty biomass fuels for indoor cooking and heating, as well as those who reside or work in close proximity to major roadways.

In addition to harm from exposure to air pollution, patterns of energy use and transport may also contribute to morbidity and mortality through accidents (both occupational and non-occupational) and unhealthful changes in physical activity. Figure 10.1 portrays some of the links between energy use and population health.

REVIEW OF THE LITERATURE ASSESSING HEALTH CO-BENEFITS IN ASIA

A number of studies have assessed the health co-benefits accruing from reductions in GHGs in Asia. The most detailed studies have come from China.

Garbaccio and colleagues (2000) assessed the health co-benefits arising from the hypothetical imposition of a tax on carbon in China. They modeled the effects of carbon taxes sufficient to achieve 5% and 10% reductions in CO_2 emissions and the consequent decrease in PM emissions. Separating emission sources on the basis of the height of the smokestack or tailpipe from which they are released, the authors estimated a decline by 3.4% in the average urban concentration of PM_{10} in the first year of a 5% reduction policy, with a resulting drop of approximately 4.5% in the incidence of premature deaths and other adverse health outcomes. The authors note that increasing wealth of the population and differences among the sources of PM emissions most sensitive to the carbon taxes would result in a decline in the relative amount of health co-benefits over time (as modeled for 15-year and 25-year time spans).

In South Korea, a U.S. EPA initiative revealed that modest reductions in GHG emissions can have significant health benefits through PM_{10} reductions. A reduction of GHG emissions by 5–15% in South Korea's energy sector by 2020 could prevent 40–120 premature deaths and 2800–8300 cases of asthma and other respiratory disease per year in the Seoul metropolitan area (Joh et al. 2001).

A 2004 study in Shanxi Province, China (Aunan et al. 2004), calculated the health benefits of CO_2 mitigation relating to coal consumption, weighing the co-benefits of six mitigation options (coal washing, briquetting, improved operations management, boiler replacement, co-generation, and modified boiler design) in terms of effectiveness in improving local air quality. Net co-benefits ranged from U.S. $1.03 billion for briquetting to U.S. $10.3 million for co-generation. The authors ranked these six mitigation options in terms of relative cost–benefit ratios and found that although briquetting had the greatest marginal costs, the net social benefits also were greatest for this option. The authors pointed out the relevance of co-benefit assessments for developing countries and the

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Sidebar 10. CO-BENEFITS OF MITIGATION MEASURES FOR CLIMATE CHANGE IN ASIA (*Continued*)

potential for a greater awareness of co-benefits to motivate developing countries to participate in GHG mitigation programs.

Chen CH and colleagues (2006a) modeled the changes in air pollution and consequent health co-benefits from caps of 50 million tons and 45 million tons on coal use in Shanghai, China. The authors noted that although both caps would result in increased emissions of PM and SO_2 by 2020, the increases would be substantially less than those under the counterfactual "business-as-usual" scenario. The health impacts of the changes in air pollution were not estimated in the study.

In a second study, Chen CH and colleagues (2007) modeled the health co-benefits of four energy scenarios ("business as usual," increased energy efficiency, use of gas instead of coal, and generation of electricity by wind) in 2010 and 2020 for Shanghai, China. They found significant potential economic benefits associated with decreases in PM pollution, ranging from U.S. $2642 million to U.S. $6192 million. Most of the dose–response coefficients and economic values used in the analysis were derived from Chinese studies.

Cao J and colleagues (2008) used an integrated modeling approach to compare the health co-benefits of three environmental tax options for China: a carbon tax, a fuel tax, and an energy-output tax. Combining a top-down macroeconomic model with a bottom-up energy-sector model, the authors estimated that health co-benefits in the year 2020 would be substantial, ranging from 10.3 billion yuan for the energy-output tax to 49.6 billion yuan for the carbon tax. The authors noted that although health co-benefits per ton of carbon reduction were greatest for the output tax, the co-benefits came at the

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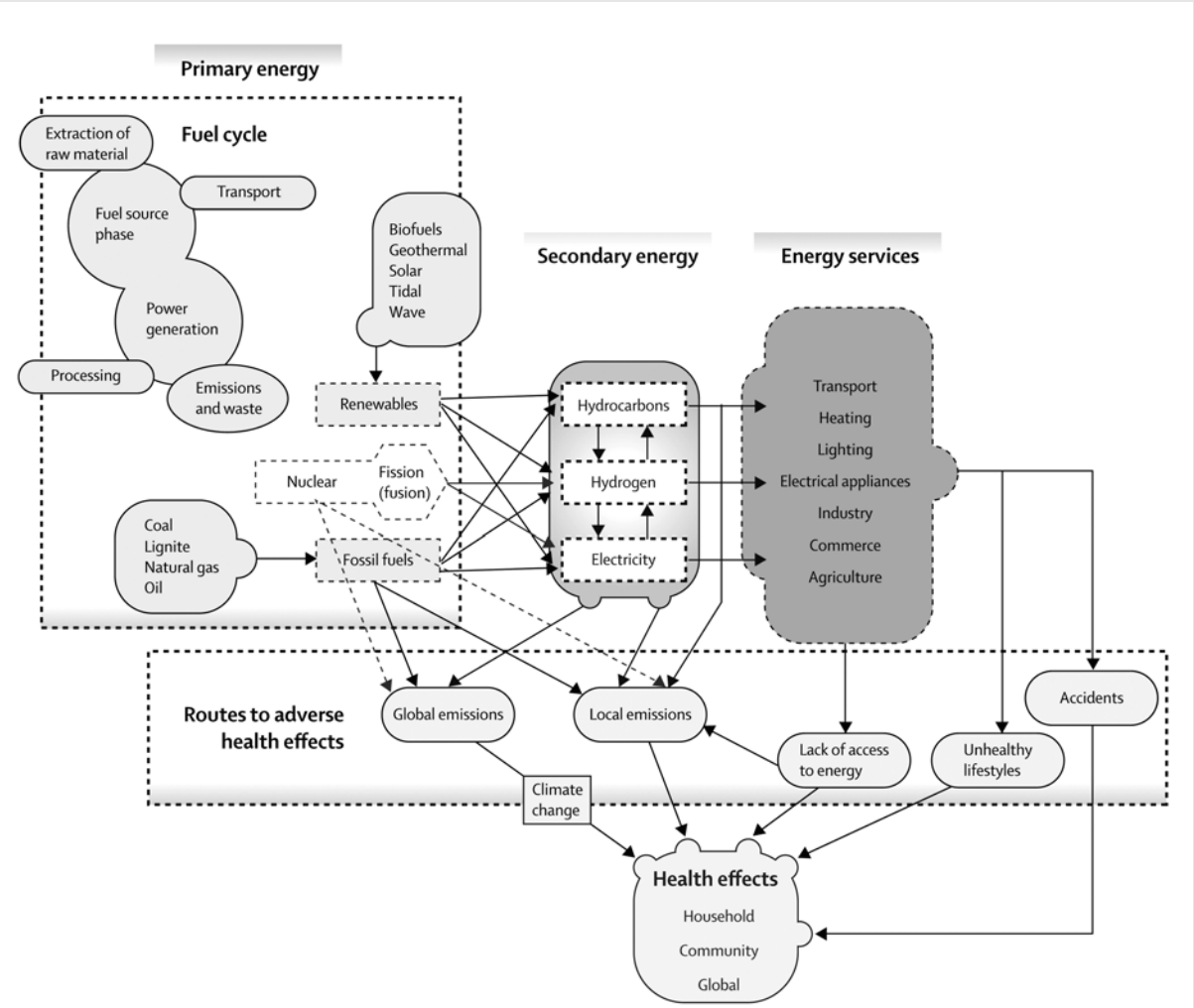

Figure 10.1. Links between energy and health. [Reprinted from Wilkinson et al. 2007 from The Lancet with permission from Elsevier.]

Sidebar 10. CO-BENEFITS OF MITIGATION MEASURES FOR CLIMATE CHANGE IN ASIA (*Continued*)

expense of more modest carbon reductions. They concluded that the optimal policy would be a combination of a national tax with caps on the output of specific energy sectors.

Smith and Haigler (2008) provide an initial, detailed consideration of a framework for assessments of health co-benefits designed to compare cost reductions associated with health co-benefits and those from other economic assessments in cost–benefit analyses of GHG-reduction policies. One example, of providing cook stoves that are biomass gasifiers in China to replace stoves that use direct combustion of fossil fuels, was found to have combined climate and health benefits that were highly cost-effective, with a cost–benefit ratio of 6.

SELECTED STUDIES OF HEALTH CO-BENEFITS IN OTHER REGIONS

Cifuentes and colleagues (2001a,b) found significant near-term public health benefits from reductions in O_3 and ambient PM associated with policies to reduce GHG emissions in Mexico City, Santiago (Chile), São Paolo, and New York. The authors concluded that immediately undertaking policies to achieve modest reductions in GHG emissions in the four cities could prevent a total of 64,000 premature deaths, 65,000 cases of chronic bronchitis, 91,000 hospital admissions, 787,000 emergency room visits, 6.1 million asthma attacks, and 37 million person-days of lost work or restricted activity over the next few decades.

A 2006 study by West and colleagues found that reducing total anthropogenic methane emissions by 20% beginning in 2010 would prevent about 30,000 deaths from all causes, owing to reduced O_3 concentrations, by 2030 and about 370,000 deaths from all natural causes by 2030. Cost-effectiveness calculations suggest that benefits to air quality and health can justify reductions in methane emissions that are undertaken to reduce global warming, irrespective of other benefits of methane and O_3 reductions.

Nishioka and associates (2006) used life-cycle assessment (analysis of the total environmental impact of a given product) and risk assessment to evaluate the net impacts of a change in energy policy and to compare health impacts of reduced PM concentrations, changes in GHG emissions, and changes in income. Theirs was among the first studies to quantify health end points associated with both environmental and economic changes related to energy policy. They quantified the economic savings for homeowners, the PM-related impacts of increased residential insulation and its associated energy savings, and the changes in GHG emissions, health effects associated with climate change, and disposable income of homeowners over the life cycle of the insulation and other energy-efficiency products. The authors converted the morbidity and mortality associated with PM and GHG emissions and income changes into DALYs. Their analysis demonstrated the feasibility of comparing the three pathways — reductions PM emissions, reductions in GHG emissions, and income change — through a combination of risk assessment and life-cycle assessment and found that all three pathways result in positive net benefits over the life of an insulated home. Because the estimated benefits are similar in magnitude among all three pathways, they concluded that no pathway can be ruled out at this time.

A number of themes emerge from the limited literature in this area. First, the health co-benefits of GHG mitigation activities can be substantial, dominating the overall cost–benefit assessment. Second, the health co-benefits occur over a relatively short time, making them potentially powerful drivers and justifiers of specific mitigation policies. Third, although co-benefits related to air quality may be the best characterized, there are multiple other links between GHG mitigation policies and health, including changes in physical activity patterns, changes in rates of physical injury, and changes in nutritional health (related both to biofuels and policies limiting development of cattle operations and those of other ruminant livestock). A fourth theme specific to studies of less-developed countries is the question of transferability of dose–response relations and economic valuations from studies of a wealthy region, such as the United States or Europe, to a less wealthy nation, such as China. This transfer leads to greater uncertainties because of disparities in economic status, underlying demographic and disease-incidence patterns, and cultural attitudes.

CRITICAL RESEARCH GAPS

Critical uncertainties throughout the current literature include the quantitative association between reductions in GHGs and those of criteria air pollutants. In general, policies that lead to relatively simple reductions in energy generation, such as increased efficiency of the generation or use of electric power, allow for a simple proportional analytic approach. Fuel substitutions are often more difficult to assess, owing to insufficient data on emissions related to alternative fuels in many cases. In developed countries, a common source of uncertainty is the interaction between regulatory controls for criteria pollutants and new GHG-reduction policies. For example, automobile emissions standards are set independently of fuel-economy standards. If fuel-economy standards are increased to reduce GHG emissions, it is unclear whether automobile manufacturers will simply adjust controls for criteria-pollutant emissions

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Sidebar 10. CO-BENEFITS OF MITIGATION MEASURES FOR CLIMATE CHANGE IN ASIA (*Continued*)

in order to continue to meet emissions standards only or whether they would take advantage of the lower concentrations of pollutants delivered to catalytic converters and other control technology and produce automobiles with appreciably lower GHG emissions. Similarly, if emissions from power plants were capped, it is not clear whether the reduced demand for electricity due to energy-efficiency measures would lead to reductions below the cap for criteria pollutants.

Research that provides more insight into the variability of dose–response relations in association with differences in climate, population demographics, and pollutant mixes is needed. Region-specific data on willingness-to-pay and cost-of-illness values are also lacking. In addition, there is an immediate need to develop a consensus on methods and assumptions for conducting co-benefits assessments, including elements such as discount rates and time horizons.

The majority of co-benefits research has focused on reductions in concentrations of conventional air pollutants. Additional ancillary benefits from GHG mitigation measures need to be better quantified, potentially including increased physical activity from increased use of active means of transport, changes in patterns of vehicular injury owing to shifts in modes of transport, and changes in patterns of injury occurring during fuel-cycle activity in association with increased development and use of renewable energy sources. Other, unrecognized health co-benefits probably also exist. ExternE, a research project of the European Commission focusing on the external costs of energy (i.e., costs not routinely accounted for in market transactions), has characterized some of the underlying health risks associated with energy production and use (European Commission 2005). These external costs need to be more broadly incorporated in economic models of GHG mitigation policies.

IMPLICATIONS FOR IMPROVED PUBLIC HEALTH POLICY

Decisions made within the energy and transport sectors to reduce GHGs must be made with a clear understanding of the implications of those decisions on human health. In many cases, there are trade-offs, both obvious and subtle, that if unaddressed may result in human suffering and net economic loss and may undermine the effectiveness of envisioned GHG reductions. Examples include the promotion and substitution of diesel cars for gasoline cars without consideration of the consequences for urban air pollution (Walsh 2008) or the substitution of corn ethanol for gasoline without consideration of the consequences for food supplies or O_3 air pollution.

GHGs are complex, have a short life, and depend on local conditions — all of which factors make it difficult, if not impossible, to establish the potential role of GHGs in reducing global warming for purposes of policy making. At present, they are not included in most policy deliberations. This omission is increasingly problematic as the world seeks ways to reduce the risks of global warming in ways that are both cost-effective and compatible with other goals, such as protection from outdoor air pollutants.

There are large health benefits from reducing the emissions of carbon-containing aerosols, and a recent study suggests that black carbon may cause climate forcing that accounts for roughly 30% of the climate forcing from all the major GHGs combined (Ramanathan and Carmichael 2008). Reductions in BC concentrations may have specific co-benefits for Asia, because deposition of BC in the snowcap of the Himalayas is believed to be contributing to the accelerated melting of Himalayan glaciers. Although climate benefits from reducing concentrations of BC may be partially offset by simultaneous reductions in concentrations of OC and SO_4^{2-} aerosols, which cool the atmosphere, the health co-benefits from reducing BC remain substantial.

Health damages are the largest component of the external costs of energy (European Commission 2005). Reductions in health-related costs of co-benefits can cancel out the expense of GHG mitigation policies. For example, Barker and Rosendahl (2000) modeled the ancillary benefits from Western Europe's meeting its Kyoto Protocol targets. On the basis of ExternE estimates of "damage costs" (e.g., health impacts of PM), the authors estimated the ancillary benefits would be worth 9 billion Euros per year during 2008–2012, constituting 15–35% of the change in GDP due to the mitigation policies. These yearly benefits include 104,000 saved life-years and 5.4 million avoided days of restricted activity. Another study, conducted by Burtraw and colleagues (2001), evaluated the co-benefits arising from a carbon tax in the electricity sector in the United States. They found that a tax of U.S. $25 per metric ton of carbon would result in U.S. $12–14 per metric ton of carbon of ancillary benefits, of which U.S. $8 per ton would come from health benefits due to reductions in NO_x concentrations. A cost–benefit analysis of the U.S. Clean Air Act further supports the magnitude of benefits that can result from environmental policies: a benefit–cost ratio of 4:1 in 2000, resulting from the U.S. $71 billion of monetized benefits, most of which would be health related (U.S. EPA 2000).

A fuller accounting of these public health benefits should be included in decisions of climate-change policy. In addition, recognition of the aggregate health savings may justify greater GHG reductions and promote more aggressive strategies, ultimately protecting the public's health against negative external costs of energy, in the short term, and against adverse effects of climate change, in the long term.

Particle-Size Distribution Given that combustion processes largely contribute to the fine ($PM_{2.5}$) fraction of PM, the particle-size distribution can be used as one measure to differentiate the contributions of source categories to air quality. As discussed previously (in *Current Capabilities and Coverage of Air Quality Monitoring* in Section II), routine government monitoring of particle concentrations in Asia is mainly limited to measurement of PM_{10}. Through specific studies, however, the ratios of $PM_{2.5}$ concentrations to PM_{10} concentrations for various Asian cities have been measured and may be used to infer $PM_{2.5}$ concentrations from PM_{10} measurements. Table 9 provides a summary of these ratios for some major cities in Asia, as derived from the cited references. This ratio ranges between 0.23 and 0.35 in the Other South Asian cities of Dhaka, Rajshahi, Chennai, and Islamabad, where road dust, soil dust, and uncontrolled construction dust dominate in the ambient air (Begum et al. 2004, 2005; Oanh et al. 2006, 2007; Shah et al. 2006). With progressive development in recent years, this ratio is probably shifting toward equal portions of both fine and coarse fractions, as seen in many East Asian cities. In Bangkok, Beijing, Taipei,China, and Bandung, the fine fraction has become slightly greater than the coarse fraction as vehicle emissions and industrial activity have progressively increased over the last decade (Oanh et al. 2006, 2007; Tsai YI and Chen CL 2006). In the more developed countries of South Korea and Japan, the ratio is as high as 0.77 (Wang XL et al. 2005; Kim KH et al. 2006a), most likely reflecting the control of coarse-fraction emissions, whereas the even higher ratio observed in

Table 9. Ratios of $PM_{2.5}$ to PM_{10} in Various Asian Cities

Country / City	Ratio of $PM_{2.5}$ to PM_{10}			Citation
	Average	Minimum	Maximum	
Bangladesh				
Dhaka	0.34	0.34	0.34	Begum et al. 2007
Rajshahi	0.35	0.35	0.35	Begum et al. 2004
China				
Beijing	0.63	0.58	0.73	Oanh et al. 2006
Taipei,China	0.62	0.61	0.63	Tsai YI and Chen CL 2006
India				
Chennai	0.31	0.26	0.35	Oanh et al. 2006
Indonesia				
Bandung	0.63	0.52	0.73	Oanh et al. 2006
Japan				
Kanazawa	0.71	0.71	0.71	Wang XL et al. 2005
Pakistan				
Islamabad	0.23	0.23	0.23	Shah et al. 2006
Philippines				
Manila	0.73	0.61	0.81	Oanh et al. 2006
South Korea				
Seoul	0.77	0.77	0.77	Kim KH et al. 2006
Busan	0.56	0.56	0.56	Kim KH et al. 2006
Sihwa	0.75	0.75	0.75	Park SS et al. 2001
Thailand				
Bangkok	0.59	0.47	0.68	Oanh et al. 2006
Vietnam				
Hanoi	0.78	0.62	0.95	Oanh et al. 2006
Ho Chi Minh City	0.34	0.34	0.34	Hien et al. 2001

Hanoi and Manila suggest an important contribution of fossil-fuel combustion in these cities (Oanh et al. 2006, 2007). Typical ratios in populated areas of developed countries in Europe and North America are 0.7 or more (Gehrig and Buchmann 2003; U.S. EPA 2004).

Source Categories More detailed receptor modeling involves the measurement of chemical composition, mostly as related to PM. Using the results of such modeling, we can illustrate and compare the sources of PM in various Asian cities and at some representative background sites (Figure 28) on the basis of available data. Because of differences in the source-apportionment methods and sources identified in each study, five major emissions categories have been designated: mobile, stationary, biomass, fugitive, and other. Mobile-source emissions consist of emissions from gasoline, diesel, and two-stroke engines. Stationary-source emissions consist of all industrial emissions as well as emissions from solid-waste combustion and refuse incineration. Fugitive emissions consist of soil dust, road dust, and construction dust. Other emissions consist mostly of emissions from other unidentified sources, as well as secondary organics, secondary sulfates, secondary nitrates, and secondary ammonium compounds (formed in the atmosphere) that could not be apportioned to specific sources.

Rajshahi, Bangladesh (population 700,000), and Chandigarh, India (population 900,000), are the only background sites where detailed source apportionment was conducted, as identified in the literature review. Even though these locations have fairly large populations by Western standards, they are still relatively small compared with major cities in Asia. The Sihwa industrial area, South Korea, is believed to have a PM makeup heavily influenced by long-range transport of air pollution from China. From Figure 29, it is evident that both source contributions to and concentrations of fine PM differ greatly among Asian cities. Beijing, Delhi, Kolkata, Kaohsiung County (adjacent to Kaohsiung City in Taipei,China), Mumbai, and Hanoi have the highest concentrations of fine PM. For Beijing, the predominant source of $PM_{2.5}$ is mostly secondary PM in nature, with considerable fugitive emissions, whereas Delhi, Kolkata, Taipei, and Mumbai, have no single predominant source, but rather a mixture of sources from each of the five major categories: mobile, stationary, biomass, fugitive, and other emissions. On the other hand, in Hanoi, biomass burning appears to be quite an important source, as is also the case in several other cities, such as Bangkok, Bandung, and Rajshahi. Select location-specific results are described in more detail below.

China In addition to the studies in Hong Kong, described in Sidebar 5, several studies have identified possible sources of PM in Beijing. He KB and colleagues (2001) identified dust storms, vehicle emissions, and biomass burning as the main sources of fine-particle air pollution at residential and downtown sites in Beijing. Duan and colleagues (2004) also noted an important contribution of biomass combustion, as well as traffic and industrial emissions, the major contributors to OC and elemental carbon (EC) concentrations during summer, with coal combustion being the dominant contributor during winter. Song Y and colleagues (2006) identified the contribution to $PM_{2.5}$ mass of eight sources in Beijing in 2000: biomass burning (11%), secondary SO_4^{2-} (17%), secondary NO_3^- (14%), coal combustion (19%), industry (6%), motor vehicles (6%), road dust (9%), and other (18%) — which includes yellow dust (dust from nonroad sources).

India Chowdhury and colleagues (2007) recently completed a comprehensive source-apportionment study in India using a source-apportionment model based on molecular markers to quantify the primary-source contributions to the $PM_{2.5}$ mass concentrations in each of the four seasons in Delhi, Mumbai, and Kolkata and in summer in Chandigarh. Five primary sources were identified and quantified: diesel engine exhaust, gasoline engine exhaust, road dust, coal combustion, and biomass combustion. Important trends in the seasonal and spatial patterns of the impacts of these five sources were observed. On average, primary emissions from combustion of fossil fuels (coal, diesel, and gasoline) were responsible for about 25–33% of the $PM_{2.5}$ mass in Delhi, 21–36% in Mumbai, 37–57% in Kolkata, and 28% in Chandigarh. The corresponding contributions of biomass combustion to ambient $PM_{2.5}$ were 7–20%, 7–20%, 13–18%, and 8%. Although the relative contributions from diesel, gasoline, and coal combustion varied by location and season, diesel was responsible for approximately 20–25% of the fossil-fuel contributions in the larger Indian cities, with increased contributions from coal combustion in winter. Chandigarh is the only smaller Indian city where source apportionment has been conducted and can be viewed as a typical background location in India. Diesel (13%), gasoline (16%), road dust (32%), and biomass (8%) were the major contributors to $PM_{2.5}$, with a secondary-particle contribution of 24%.

Bangladesh Extensive studies in Dhaka (Begum et al. 2004, 2005, 2007), identified major coarse-particle contributions to $PM_{2.5}$ from soil dust and road dust (total contribution, 50%). The motor-vehicle source apportionment was approximately 40%; the remaining 10% consisted of

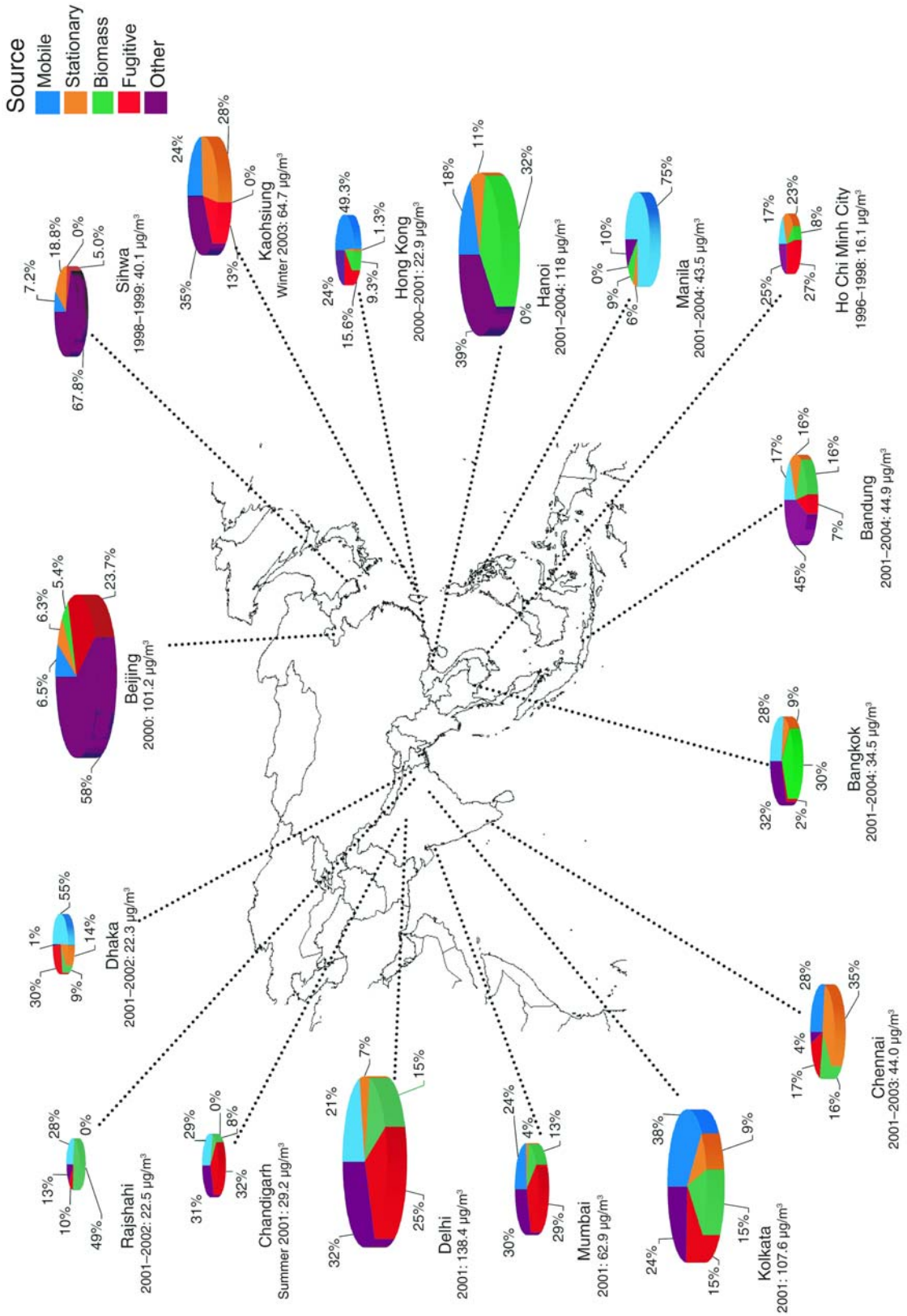


Figure 29. Source apportionment of average annual $PM_{2.5}$ mass in representative urban and rural locations in Asia. The data given for Chandigarh and Kaohsiung are seasonal averages for summer and winter, respectively, rather than the average annual $PM_{2.5}$ mass. [Data compiled by Zohir Chowdhury (*http://chowdhuryonline.net/zohir/BAQ_Source_Apportionment.pdf*).]

emissions from two-stroke engines, sea salt, and metal-smelting emissions. Motor-vehicle emissions in which emissions from two-stroke engines were included accounted for about 48% of the $PM_{2.5}$ mass. In a hot-spot traffic area in central Dhaka, Begum and colleagues (2005) estimated that approximately 50% of the total fine-particle mass could be attributed to motor vehicles, including those with two-stroke engines. In Rajshahi, a smaller city in the western part of Bangladesh, wood burning contributed 50% of the fine-particle mass, with a 23% contribution from motor vehicles.

Thailand Oanh and colleagues (2006, 2007) estimated the source contributions in Bangkok in both the dry and wet seasons. They reported a mobile-source contribution to $PM_{2.5}$ pollution of 35% in the dry season and 21% in the wet season, and a biomass-combustion contribution of 31% in the dry season and 29% in the wet season. Considerable amounts of secondary PM (29–36%) were also identified and quantified in these studies.

OVERVIEW OF EXPOSURE ISSUES

Human exposure to a pollutant occurs when a person comes into contact with the pollutant for a certain period of time. The occurrence of exposure, hence, requires the coexistence of the person and the pollutant in the same space at the same time. However, ambient concentrations may be measured without consideration of this coexistence. For example, although air pollutant concentrations are very high near a large coal-fired power-plant stack, very few people may be actually exposed if the stack is at a remote site. From the standpoint of health effects, exposure is a better predictor or determinant than ambient concentrations, as it is closer to health effects in this conceptual source–to–health effects continuum:

Source emissions → Ambient (outdoor) concentrations → Exposure (intake) → Dose → Health effects

The average population exposure reflects the number of people exposed at different concentrations for different periods of time: the number of people × the pollutant concentration × the exposure duration. The term *exposure* refers to contact with any part of the exterior of the human body — not just via the inhalation route alone. The term *intake* may be more exclusively used to describe inhalation exposure to airborne pollutants. The dose, or the amount of pollutant that actually crosses the exterior boundary of the body or is absorbed by a body tissue or organ, is even more directly related to health effects. However, estimating the dose requires knowledge of many additional parameters (e.g., inhalation rate, absorption rate) at the individual level.

Ambient (outdoor) concentrations have often been used in epidemiologic investigations of the health effects of urban air pollution. An underlying assumption of these studies is that measurements or model estimates of ambient concentrations serve as a proxy of population exposure to air pollution. How accurate this assumption is depends on a large number of factors (see below) concerning the *intake fraction* — a unifying concept that has evolved over the past two decades (Bennett et al. 2002).

Intake Fraction and the Role of Human Activity

The purpose of the intake fraction is to provide a simple description of the relation between emissions and exposure by incorporating major parameters that affect the continuum of source emissions to ambient concentrations to exposure levels (Zhang JJ and Lioy 2002).The intake fraction for a primary air pollutant is defined as the total mass inhaled from a particular emission source divided by the total mass emitted from the emission source. For a given emission source (e.g., industrial stack, cigarette, motor vehicle fleet, or household stove) and pollutant (e.g., CO or $PM_{2.5}$), the intake fraction is the cumulative mass inhaled by the exposed population (e.g., the total population of a city or the children residing in a city) over a defined time period divided by the total mass emitted over the same time period.

One of the most important factors affecting the intake fraction is whether the pollutant is released within a confined space (indoors) or in an open environment (outdoors). Because people in developed countries, on average, spend more than 85% of their time indoors, pollutants released into occupied indoor environments will produce much higher exposures even if indoor concentrations are the same as outdoor concentrations. In most cases, however, indoor releases of pollutants (e.g., from cooking stoves or cigarettes) generate substantially high indoor concentrations. Having evaluated typical intake fractions for nonreactive air pollutants emitted in various scenarios (e.g., into a moving vehicle, into a residence, from a ground-level line source [i.e., vehicular traffic]), Marshall and Nazaroff (2007) concluded that the typical difference in intake fraction between indoor and outdoor releases is about three orders of magnitude, informally referred to as the *rule of 1000*. That is, regarding pollutant intake, 1 gram of emissions into indoor environments is roughly equivalent to 1000 grams of emissions into outdoor environments. This is consistent with the well-known fact that for an air pollutant that has indoor sources, outdoor concentrations generally account for only

a small fraction of the total exposure and thus generally are poor predictors of total personal exposure.

Even if two outdoor sources emit the same mass of pollutant, if one source is in a densely populated urban area and the other is in a remote rural area, the pollutant emitted by the urban source will have a substantially higher intake fraction. Based on several estimates from developed countries, intake fractions are as low as 0.1 per million people for outdoor emissions in remote rural areas, roughly 10 per million for near-ground outdoor emissions in urban areas, and approximately 5000 per million for indoor releases in occupied buildings (Marshall and Nazaroff 2007). Such a large range of intake fractions is the result of the "three Ps": population, proximity, and persistence, or the size of exposed population, the proximity of the source to the exposed population, and the persistence of the pollutant in the atmosphere, respectively. These three parameters can significantly affect the relation of emissions to exposure. More persistent pollutants are more likely to be transported into the atmosphere and reach more people. In contrast, pollutants that are very reactive or readily scavenged via the processes of wet and dry deposition are less likely to affect people who are not in the immediate vicinity of the emission source.

Few studies have invoked the concept of intake fraction to evaluate source–intake relations in developing countries. However, developed and developing Asian countries differ greatly in the density and distribution of their populations, in source characteristics (especially those that affect the proximity of sources to the population), and in time–activity patterns. For example, by evaluating 17 power plants in Hunan province in China, Li and Hao (2003) estimated average intake fractions within 500 km of sources as 9.73, 2.39, and 2.47 per million people for primary fine PM, SO_4^{2-}, and NO_3^-, respectively. The estimated PM intake fraction of 9.73 per million people is for PM emitted by coal-fired power plants. This Chinese estimate is 1 to 2 orders of magnitude higher than the fractions estimated for U.S. coal-fired power plants and is similar to the fractions for U.S. near-ground urban emissions (Smith 1993; Marshall and Nazaroff 2007). The intake fraction for Chinese power plants is higher because more people live closer to power plants there than in the United States.

Another example of the fact that the proximity of the population to PM-emitting sources affects the source–intake relation is provided by an analysis of the major sources of nonoccupational PM_{10} exposures in China (Florig et al. 2002). Models of pollutant emissions, pollutant dispersion, the location of the population, and the time–activity patterns of the population were developed to estimate total PM_{10} exposure for six major types of sources and four population subgroups in both urban and rural settings. The PM_{10} sources evaluated were ETS, cooking stoves, heating stoves, heating boilers serving entire urban districts, on-road vehicles, and coal-fired power plants. The four subgroups considered were based on age: preschool-aged children, schoolchildren, working adults, and older persons. For typical urban areas with cold winters, residential heating systems were the most important source of exposure to PM_{10} for all four subgroups, followed by ETS, cooking stoves, on-road vehicles, and coal-fired power plants. In rural areas, cooking and heating sources produced the largest PM_{10} exposures, with an additional, significant contribution by ETS. Except for cooking, there was little source-to-source variation in contributions across the subgroups. Exposures of preschool children and older persons to PM_{10} from cooking stoves were approximately 45% and 30% higher than those of schoolchildren and working adults, respectively, because preschool children and older persons spend more time in the cooking area. This analysis was designed to reflect the typical 1990s urban and rural lifestyles in China. However, the increased use of natural gas and LPG as cooking fuels will likely have resulted in reduced PM_{10} exposure, and the source ranking for population exposure to PM_{10} today may be quite different, at least in urban areas. The PM_{10} exposures from on-road vehicles may be greater today as well, given the rapid growth in the number of motor vehicles.

Understanding source–intake relations (i.e., intake fractions) can provide useful insights about the relation between ambient concentrations and exposure. Because the three Ps (population, proximity, and persistence) are the major factors that affect the intake fraction, they can be used to evaluate the appropriateness of the selection of ambient monitoring stations. To best capture a population's exposure, monitoring stations should be placed as close as possible to where the exposed population spends most of its time.

Urban ambient monitoring stations are often selected to represent "average" urban background concentrations of air pollution. In some cases, monitors are sited at putative hot spots to characterize worst-case scenarios. From an exposure standpoint, urban background emissions are only one component of a person's total exposure. Other components include localized outdoor emissions (e.g., emissions at bus terminals or roadside emissions), which may be captured by hot-spot monitors, or indoor emissions (e.g., from stoves, ETS, or building materials) and emissions from personal activities (e.g., smoking, cleaning, or cooking), which are not measured by ambient monitoring.

Limitations of Ambient Pollutant Concentrations as a Metric of Exposure

Time-series studies and other studies of the health effects of air pollution often attempt to assess the impact of urban background air pollution by using average pollutant concentrations that are measured at multiple fixed monitoring stations across the city. In densely populated Asian cities, monitoring stations tend to be close to major roadways. This placement results in recording higher "average" ambient concentrations, owing to contributions of roadside emissions, than those obtained from monitors located farther from major roadways. Therefore, a 10-μg/m^3 change in an ambient pollutant concentration may represent quite different magnitudes of overall population exposure in various cities, owing to varying distances between monitoring stations and major roadways. This variation makes it difficult to directly compare the effect estimates (i.e., the changes in relative risk [RR] per 10-μg/m^3 change in pollutant concentration) of Asian cities of varying population densities with the effect estimates of Western cities.

Exposure to pollutants of outdoor origin occurs when people spend time outdoors and also when they are indoors if pollutants enter the indoor environment. High population densities force many people in developing Asian cities to work or spend time outdoors at roadsides, where a mix of air pollution sources can be found: the urban motor-vehicle fleet, charcoal- or coal-burning stoves used by street-food vendors, as well as tuk-tuks (motorized rickshaws) and motorcycles. People who work at roadsides are exposed to the highest concentrations of outdoor air pollution for the longest time on a daily basis. Also, substandard residences along the roadsides are likely to have cracks in the walls and broken or loose windows and doors, resulting in considerable penetration of outdoor air pollution into the indoor environment and subsequent exposure. This implies that the intake fraction from ground-level line sources (i.e., vehicular traffic) is probably markedly higher in cities in developing Asia than in Western cities (Lai et al. 2000).

On the other hand, a growing number of modern buildings that include residences are being erected in Asian cities. Modern buildings are typically air-tight and equipped with air conditioners; thus, they have a low rate of exchange of outdoor and indoor air. People working or living in new buildings will be exposed to a smaller fraction of outdoor air pollution while staying indoors. This heterogeneity makes citywide average pollutant concentrations less accurate representations of "average" population exposure.

Some 28–59% of people in Asian cities are estimated to live in urban slums (UN-Habitat 2003). Many households in these areas rely on solid fuels (e.g., coal or wood) for cooking, which may expose residents to high concentrations of indoor air pollution in addition to their high roadside exposures (HEI ISOC 2004). On the other hand, modern urban residences in Asian cities, in general, use modern fuels (e.g., electricity, natural gas, or LPG) that are substantially cleaner than solid fuels. Therefore, exposures resulting from indoor air pollution may vary substantially across households within a city (Mestl et al. 2007). Given that the gap between people living in poverty and people with wealth seems to be increasing continuously in many cities in developing countries, this inequality in urban air pollution exposure may not diminish for some time. Such inequality also has important implications for using citywide averages of ambient pollutant concentrations in studies of the health effects of air pollution, because indoor emissions from solid fuel, as well as roadside emissions, have substantially higher intake fractions than background emissions from urban outdoor sources. The assumption of a constant relation between source and intake (or ambient concentration and intake) for the entire population within a city may be reasonable in Western cities where high roadside and indoor solid-fuel exposures appear to affect few people, but the situation is clearly different in many developing Asian cities.

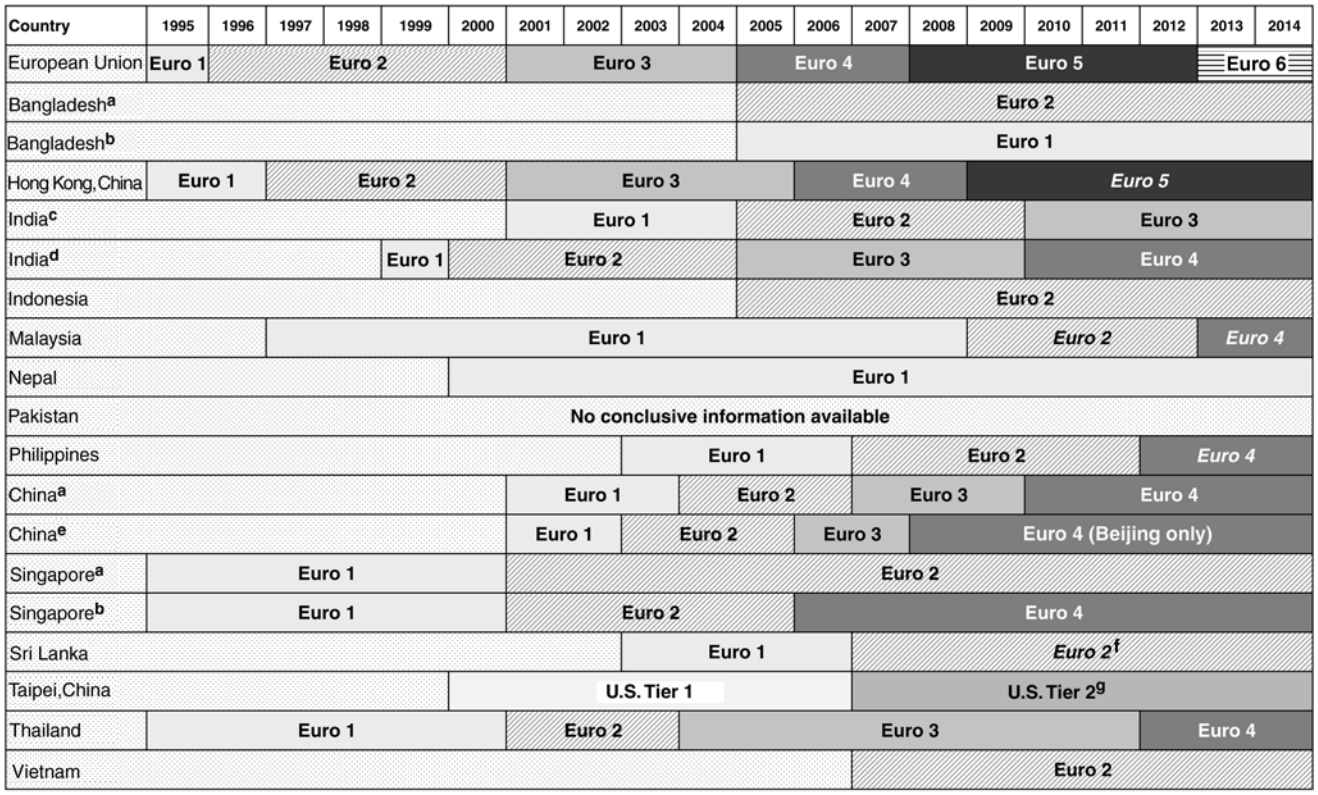
POLICY ISSUES IN AIR QUALITY MANAGEMENT

Actions Taken to Reduce Air Pollution

Countries throughout Asia have taken action to address the problem of air pollution over the past decade. This action was prompted by the development of monitoring systems that document air pollution concentrations (albeit with widely varying comprehensiveness and sophistication) and growing public awareness of the high concentrations of air pollution experienced in everyday life, especially in congested cities.

Many Asian countries have adopted National Ambient Air Quality Standards (NAAQS) for several air pollutants, such as SO_2, PM, and, in some cases, O_3 (Figure 30). Some countries, such as India, have now begun to reconsider their standards in light of current air quality guidelines of the WHO (Sidebar 11).

In addition, some countries have begun to reduce emissions from specific sources, most notably motor vehicles, industries, and electricity-generating facilities (see *www.cleanairnet.org/caiasia/1412/channel.html*). For example, many Asian countries now require that new automobiles sold meet minimum standards equivalent to



[a] Emission standards for gasoline vehicles.
[b] Emission standards for diesel vehicles.
[c] Emission standards for the entire country other than cities named in footnote d.
[d] Emission standards for Delhi, Chennai, Mumbai, Kolkata, Bangalore, Hyderabad, Agra, Surat, Pune, Kanpur, Ahmedabad, Sholapur, and Lucknow.
[e] Beijing and Guangzhou adopted Euro 3 standards in Sept. 2006; Shanghai adopted Euro 3 standards at the end of 2008. Of these 3 cities, only Beijing adopted Euro 4 standards in 2008.
[f] Euro 2 was not mandated but fuel meeting Euro 2 standards was available at pump stations.
[g] Equivalent to Euro 4 emission standards.

Figure 30. Timetable for emissions standards for new light-duty vehicles in Asia (as of April 2009). European Union (top row) is provided for comparison. [Data compiled from CAI-Asia 2007b.]

Sidebar 11. THE WHO AIR QUALITY GUIDELINES

In October 2006, the WHO issued its new air quality guidelines (Table 11.1), which suggest that reducing concentrations of one pollutant — PM_{10} — could reduce deaths in polluted cities by as much as 15% every year (WHO 2006b). The 2006 WHO guidelines were the first to address all regions of the world. They are more restrictive than the national standards currently applied in most of Asia, and their application in some cities would mean reducing current pollution concentrations by more than two thirds. For example, in many cities of developing countries, the average annual PM_{10} concentrations (the main source of which is the burning of fossil and other fuels) exceed 70 μg/m^3, and in Asia, the concentrations can be substantially higher. To prevent adverse health effects, the WHO guideline for PM_{10} is set at 20 μg/m^3.

The guidelines were based on the latest scientific evidence and set targets for air quality that would protect the large majority of individuals from the health effects of air pollution. They were established after consultation with more than 80 expert scientists, and reviews of thousands of recent studies, from all regions of the world. As such, the guidelines present the most widely agreed on and up-to-date assessment of health effects of air pollution, recommending targets for air quality at which health risks are significantly reduced. However, it should be recognized that these guidelines are not levels of absolute safety. The underlying scientific evidence indicates adverse health effects at levels throughout the range of ambient concentrations in the guidelines, suggesting that effects also occur at concentrations below the strictest guidelines.

Recognizing the need for governments to set national standards according to their own particular circumstances, the WHO guidelines provide the basis for all countries to build their own air quality standards and health policies according to solid, scientific evidence. Specifically, the guidelines propose progressively strict interim targets and milestones to achieve better air quality. The guidelines and interim targets aim to allow countries to measure their distance from these objectives and estimate the health impacts of current pollution concentrations and the health gains achieved by reducing them.

Table 11.1. WHO Air Quality Guidelines[a]

Pollutant and Averaging Time	Guideline
$PM_{2.5}$	
1 Year	10 μg/m^3
24 Hours (99th percentile)	20 μg/m^3
PM_{10}	
1 Year	20 μg/m^3
24 Hours (99th percentile)	50 μg/m^3
O_3	
8 Hours, daily maximum	100 μg/m^3
NO_2	
1 Year	40 μg/m^3
1 Hour	200 μg/m^3
SO_2	
24 Hours	20 μg/m^3
10 Minutes	500 μg/m^3

[a] Data compiled from WHO 2006a.

Euro 1 standards; several countries also require or are planning to require compliance with more advanced European standards (Table 10). Since 2001, these efforts have been augmented by the formation of CAI-Asia, which was organized with assistance from the Asian Development Bank and the World Bank to promote sharing of technical advice among regulatory agencies, industries, nongovernmental organizations, and other key stakeholders in Asia. In addition, municipal fuel-switching programs for public-transit fleets have been shown to reduce concentrations of urban air pollutants. In Delhi, for example, switching to CNG has resulted in both local air quality improvements and reductions in emissions related to climate change, an example of a co-benefit (Reynolds and Kandlikar 2008).

Relocation of industries away from populated city centers, and in some cases their replacement with cleaner industrial processes, has also been an effective mechanism of managing urban air quality — for example, in Dalian, China (Bai 2002) — and was a major component, along with traffic restriction, of the actions taken to improve air quality in Beijing in preparation for the 2008 Olympic games (Sidebar 12). Relocation that is not accompanied by other strategies to reduce emissions may result in the transformation of a local air quality problem into a regional concern, and reductions in exposure may not be sustained if urban growth eventually occurs at the relocation site.

Actions to improve air quality have resulted in measurable declines in the concentrations of some pollutants

Table 10. PM Standards in Asia[a]

	PM_{10} (μg/m^3)			$PM_{2.5}$ (μg/m^3)	
Location	1 Hr	24 Hr	1 Yr	24 Hr	1 Yr
Standards for Comparison, 2006					
WHO	—	50	20	25	10
European Union	—	50	40	—	—
U.S. EPA	—	150	—	35	15
Country or City Standards, 2007					
Afghanistan	—	—	—	—	—
Bangladesh	—	150	50	65	15
Bhutan	—	—	—	—	—
Cambodia	—	—	—	—	—
Hong Kong	—	180	55	—	—
India[b]	—	100	60	—	—
Indonesia	—	150	—	—	—
Japan	200	100	—	—	—
Laos	—	—	—	—	—
Malaysia	—	150	50	—	—
Mongolia	—	—	—	—	—
Nepal	—	120	—	—	—
Pakistan	—	—	—	—	—
China[c]	—	150	100	—	—
South Korea	—	150	70	—	—
Singapore	—	150	50	65	15
Sri Lanka	—	—	—	—	—
Thailand	—	120	50	—	—
Vietnam	—	150	50	—	—

[a] Adapted from CAI-Asia 2007c. The PM concentrations are current as of July 10, 2007, unless otherwise stated. WHO, European Union, and U.S. EPA standards (updated on December 17, 2006) are given for comparison.

[b] Indian standards are for residential, rural, and "other" areas.

[c] Chinese standards are based on the Class 2 category.

Sidebar 12. AIR POLLUTION REDUCTION DURING THE 2008 BEIJING OLYMPICS

Beijing, capital of China, is regarded as one of the world's most-polluted cities. From 2000 to 2007, a large proportion of days each year were considered days on which air quality guidelines were not attained (Table 12.1). During this period Beijing, as well as China as a whole, experienced sustained and rapid economic growth. Despite the fact that the number of motor vehicles in Beijing more than doubled from 2000 to 2007, the number of days of attainment of air-quality guidelines has been increasing every year (from 177 days in 2000 to 246 days in 2007). This increase is due to many actions to control air pollution, including the closure or relocation of high-polluting industrial facilities (e.g., oil refineries and steel-making factories); the change from a coal-based energy infrastructure to natural gas pipelines, resulting in the phase-out of numerous coal-fired boilers and domestic stoves; the control of wind-blown dust from construction sites; the implementation of stricter vehicle-emission standards; the replacement of older fleets of buses, passenger cars, and trucks with newer and cleaner fleets; and the installation or upgrade of stack emission-control devices in power plants.

However, before the 2008 Beijing Olympics, ambient concentrations of common pollutants were still far above health-based standards and several times as high as concentrations typically measured in cities in developed countries. This situation caused serious concerns in the international community about the health and performance of Olympic athletes. Extensive media coverage of this topic preceded the commencement of the Beijing Olympics.

To ensure reasonably good air quality during the Olympics (held from August 8–24, 2008) and the Paralympics (from September 6–16, 2008), the Chinese government launched a series of aggressive measures to reduce pollutant emissions in Beijing and the surrounding areas for a period of more than 2 months, including the time of the games. Starting on July 1, 2008, all vehicles that failed to meet the Euro 1 standards for exhaust emissions were banned from traveling on Beijing's roads. Mandatory restrictions for private vehicles were in effect from July 20 through September 20. During this period, private vehicles in Beijing were permitted to be driven every other day (with the day assigned on the basis of whether the license-plate number was odd or even), with a slight relaxation of this control measure during the Paralympics, when private cars could be used without restriction outside the Sixth Ring Road. As a result, traffic on the Sixth Ring Road was reduced by 32% on average in August as compared with June, leading to a decrease of vehicular emissions of various pollutants (such as CO, NO_x, VOCs, and PM_{10}) by about 50%, as estimated from roadside emissions data collected by a research team at Tsinghua University.

The control actions were not limited to traffic emissions. During the same period, other area and point sources in Beijing were placed under strict control. Several heavy-polluting factories were mandated to reduce levels of operation or completely shut down during the games, with approximately 50% of the industrial boilers halted. Construction activities were all paused, gasoline stations were renovated to minimize gasoline evaporation, and painting activities in the furniture-making and car-making industries were restricted. Power plants in Beijing were required to reduce their emissions by 30% from their June levels, even if they had already met the Chinese emission standards. The smokestacks of selected power plants were monitored by the Tsinghua University researchers to verify the effectiveness of the control policies. Emission controls on large industrial sources were also applied in Tianjin and surrounding provinces (e.g., Inner Mongolia, Shanxi, Hebei, and Shandong). The Tsinghua research team estimated that, as a result of all the emission-reduction

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Table 12.1. Attainment of Chinese Air Quality Guidelines and Annual Mean Concentrations of Pollutants in Beijing, 2000–2007[a]

Year	Attainment Days *n* (% of year)	PM_{10} ($\mu g/m^3$)	SO_2 ($\mu g/m^3$)	NO_2 ($\mu g/m^3$)	CO (mg/m^3)
2000	177 (48.5)	162	71	71	2.7
2001	185 (50.7)	165	64	71	2.6
2002	203 (55.6)	166	67	76	2.5
2003	224 (61.4)	141	61	72	2.4
2004	229 (62.7)	149	55	72	2.2
2005	234 (64.1)	142	50	66	2.0
2006	241 (66.0)	161	53	66	2.1
2007	246 (67.4)	148	47	66	2.0

[a] From J Zhang (personal communication, 2009).

Sidebar 12: AIR POLLUTION REDUCTION DURING THE 2008 BEIJING OLYMPICS (*Continued*)

Table 12.2. Means (±SD) of 24-Hour Average Concentrations of Pollutants Measured Before and During the 2008 Beijing Olympics at a Site in Central Beijing[a]

Pollutant	Concentration Before Olympics (June 26–July 6)	Concentration During Olympics (August 8–24)	Percent Change
$PM_{2.5}$ (μg/m^3)	96.9 ± 60.4	44.6 ± 30.4	−54
SO_2 (ppb)	9.6 ± 9.1	5.9 ± 13.3	−39
NO (ppb)	5.0 ± 2.5	1.8 ± 0.8	−64
NO_2 (ppb)	27.4 ± 9.7	12.1 ± 2.1	−56
NO_x (ppb)	32.1 ± 12.8	13.7 ± 2.6	−57
CO (ppm)	1.3 ± 0.64	0.6 ± 0.20	−55
O_3 (ppb)	32.2 ± 27.5	38.3 ± 15.6	+ 19
O_3 (ppb, 1-hr maximum)	57.6 ± 34.9	81.3 ± 28.1	+ 41

[a] From J Zhang (personal communication, 2009).

measures, total emissions of NO_x, SO_2, VOCs, PM_{10} were reduced from their concentrations during the same period in 2007 by 55%, 58%, 59%, and 61%, respectively.

These emission reductions were directly reflected in markedly reduced ambient concentrations of common pollutants. On the basis of data from the Measurement of Pollution in the Troposphere (MOPITT) sensor on the Terra satellite of the U.S. National Aeronautics and Space Administration (NASA), there was a decrease in CO concentrations by approximately 20% and in NO_2 concentrations by approximately 50% in Beijing during August 2008 as compared with the average for August 2005, 2006, and 2007 (NASA 2009). In the Health Effects of Air Pollution Reduction Trial (HEART) — cofunded by HEI and the U.S. National Institute of Environmental Health Sciences Institute, National Institutes of Health — researchers from Peking University and the University of Medicine and Dentistry of New Jersey measured ambient concentrations of air pollutants at a monitoring site located in central Beijing. Preliminary results (Table 12.2) showed that concentrations of $PM_{2.5}$, NO_2, NO_x, SO_2, and CO during the Olympics were 54%, 56%, 57%, 39%, and 55% lower than the respective concentrations before the Olympics.

The only pollutant with increased concentrations during the Olympics was O_3, for which the 24-hour average and 1-hour maximum concentrations increased (Table 12.1). Despite the increase, O_3 concentrations at the HEART site were below both the Chinese air quality standard and the U.S. air quality standard during the Olympics. The increase in O_3 was due to the so-called NO-titration phenomenon in urban areas. Nitric oxide (NO) reacts rapidly with O_3, serving as an effective O_3 sink. During the Olympics, the major source of NO, motor-vehicle

Continued on next page

Sidebar 12: AIR POLLUTION REDUCTION DURING THE 2008 BEIJING OLYMPICS (*Continued*)

emissions, was reduced substantially at the study site, resulting in a 64% reduction in the NO concentration. This finding should be interpreted with caution, however. O_3, a secondary pollutant resulting from photochemical reactions in the atmosphere, typically has large temporal and spatial variability. For example, measurements made at a suburban Beijing site by the Tsinghua University research team showed that O_3 concentrations were reduced during the Olympics as compared with O_3 concentrations measured in June and July 2008 (data not shown) and concentrations measured in June and July in both 2006 and 2007 (see Table 12.3). More careful spatial and temporal analyses of O_3 are needed to better understand this complex pollutant and other secondary pollutants.

In general, the data from the suburban monitoring site collected by the Tsinghua University research team showed similar results for pollutants other than O_3 (Table 12.3). In addition to concentrations of $PM_{2.5}$, CO, and NO_x, those of black carbon (BC) and total carbon in $PM_{2.5}$ were significantly lower during the Olympics than their average concentrations before the games. The degree of the observed reductions exceed the reductions that would be expected on the basis of meteorologic variation alone, confirming the effectiveness of the emission-reduction measures implemented in Beijing during the Olympics and Paralympics.

Whether the drastic reduction in air pollution during the Beijing Olympics resulted in improved health is an important question regarding the accountability of the control policies used. In HEART, physiologic and molecular changes are being measured in Beijing residents in relation to air pollution changes. It is hypothesized that cleaner air during the Beijing Olympics directly translated into reduced oxidative stress, reduced levels of pulmonary and systemic inflammation, and improved cardiovascular function (represented by various biomarkers). Confirmation of this hypothesis will provide objective and direct evidence of the improvement of public health by means of air pollution interventions. HEART will also provide evidence to support or refute several prominent hypotheses concerning the biological mechanism of health effects of air pollution.

12.3. Changes in Ambient Concentrations of Pollutants Measured at a Site in Suburban Beijing During the Olympics and Paralympics Compared with Before the Olympics (Baseline)[a]

	Percent Change from Baseline Concentration[b]	
Pollutant	Olympics August 8–24, 2008	Paralympics September 6–17, 2008
$PM_{2.5}$	−40	−28
BC	−37	−21
O_3	−34	−23
Total carbon component of $PM_{2.5}$[c]	−45	
CO[c]	−50	
NO_x[c]	−53	

[a] From J Zhang (personal communication, 2009).

[b] Baseline concentration is the average of the concentrations from June 20 through July 20, 2008, except for O_3 (a secondary pollutant with natural month-to-month variations), for which the baseline concentration is the average of the concentrations from June 20 through July 20 in both 2006 and 2007.

[c] The periods for total carbon, CO, and NO_x are from baseline to either August or September 2008.

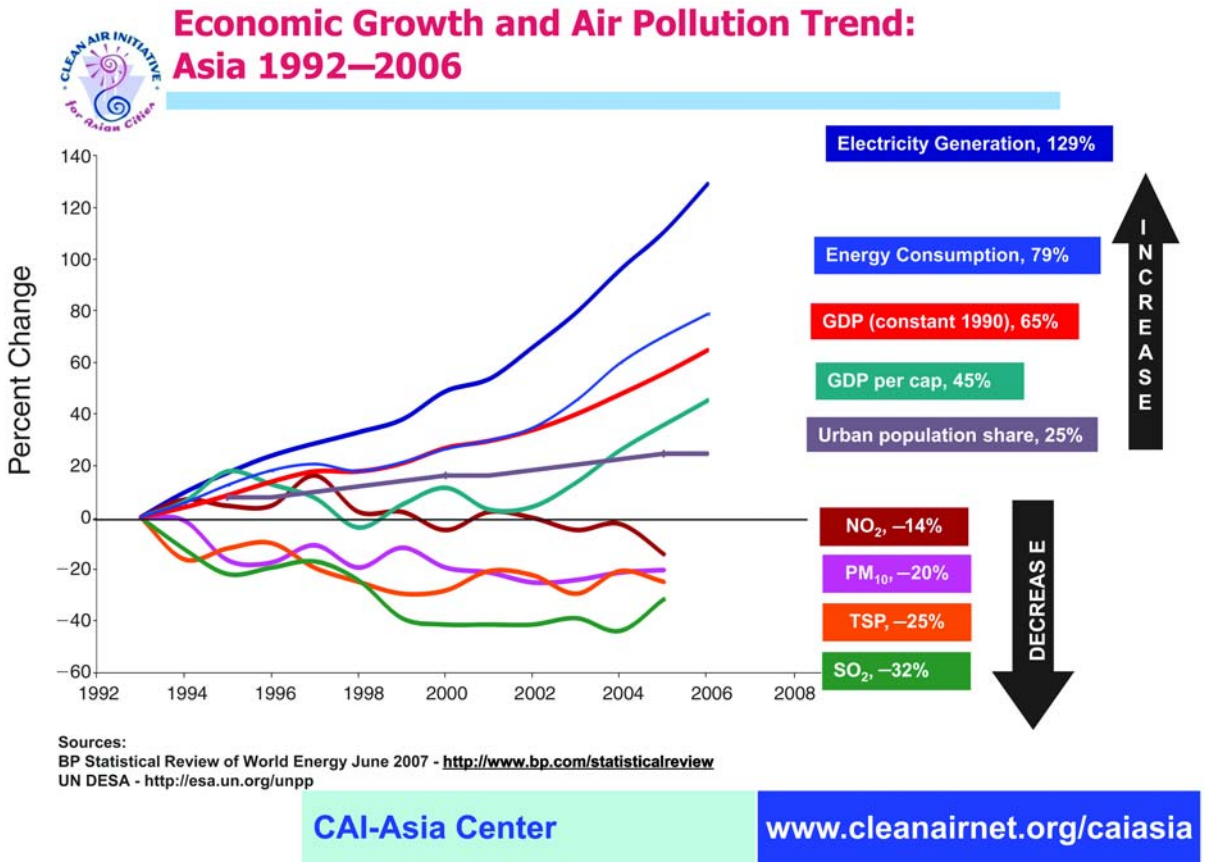

Figure 31. Declines in air pollution levels in Asia, 1992–2006, due to air quality management and substantial economic growth. [Adapted with permission from CAI-Asia 2008b.]

even during a period of substantial economic growth in Asia (Figure 31). In countries that have removed lead from gasoline, both exposures to lead and blood lead levels in children and other exposed groups have declined markedly (e.g., Vichit-Vadakan and Sasivimolkul 2002). In many areas, efforts to control sulfur concentrations in fuel and to reduce emissions from coal-burning industries and electricity-generating facilities have resulted in even longer-term reductions in ambient SO_2 concentrations (Hedley et al. 2002). And in some locations (e.g., Bangkok), comprehensive programs targeting motor vehicles and other sources have resulted in substantial reductions in concentrations of several pollutants that were historically very high.

Continuing Challenges

Despite this progress, however, considerable challenges to improving air quality in Asia remain. Few Asian countries have systematic air quality management programs to set standards, identify actions to meet those standards, implement those actions, and then monitor the air quality to determine whether the actions have been successful (CAI-Asia Center 2004).

Although standards for new models of motor vehicles have been tightened, a substantial number of older, high-emitting motor vehicles are still operating in most of Asia. In addition, the quality of automotive fuel continues to be a barrier to retrofitting air pollution controls onto existing vehicles or applying the best available controls to new designs. Reducing emissions from existing sources will require extensive interventions: maintenance and inspection programs, enhanced fuels to reduce emissions, retrofitting with control technologies, and use of alternative fuels and technologies. In addition, the rapid growth of motor-vehicle fleets in many Asian countries, and the advent of even cheaper vehicle alternatives (e.g., the

1-lakh [approximately U.S. $2500] car in India), will offset at least some of the gains made, unless the growth is accompanied by continuing enhancement and upgrading of standards for fuels and vehicles. A recent, extensive policy analysis by CAI-Asia — *A Roadmap for Cleaner Fuels and Vehicles in Asia* — lays out the many opportunities for continuing to improve emissions as fleets expand (CAI-Asia Center 2007a).

A second important and growing source of emissions is the rapid expansion of electricity-generating facilities, often fueled by coal, to meet the growth in electricity demand from expanding economies. Recently, some countries, especially China, have set ambitious targets for reducing overall emissions from these facilities, even when allowing for industrial growth (e.g., the current Chinese Five-Year Plan calls for a 10% reduction in total SO_2 emissions). However, implementing these plans and enforcing their completion continues to be a challenge.

In addition to more common mobile and stationary sources, Asian cities face a wide array of diffuse, difficult-to-control sources (e.g., open burning, emissions from combustion of low-quality indoor fuels, and large numbers of unregulated small businesses and industries). There are also a number of noncombustion sources (e.g., construction dust and desert sand blown from great distances) that also are associated with considerable health impacts. Although dust from natural events may not be easily controlled, dust from human activities such as construction, garbage from construction sites deposited on streets, and poor agricultural practices can and have been successfully controlled in many countries.

A further challenge for Asia is the transport of air pollution from one region to the other and the challenges that poses for local jurisdictions that want to control emissions but do not have authority over the sources. This can be a problem within one country (e.g., the effects of emissions from the Pearl River Delta on air quality in Hong Kong; Sidebar 5), between countries (e.g., the transport of pollutant-laden dusts from China to Japan), and globally (e.g., the transport of Asian pollutants all the way to the West Coast of the United States).

Beyond setting standards for the control of individual sources, there are two other major challenges: ensuring sufficient institutional capacity and obtaining adequate air quality and health data.

Institutional Capacity Key elements of success for air quality management include having the staff, resources, and legal tools necessary to enforce the standards and ensure a level playing field for all sources. This is a major challenge in Asia where, although some countries have enacted legislative and regulatory frameworks, other countries (e.g., Vietnam) have not yet done so, and in all Asian countries, environmental agencies are severely understaffed.

Adequate Air Quality and Health Data Although there has been major progress in implementing more modern air quality monitoring systems, there are still many areas of Asia where the quality and reliability of monitoring is seriously deficient. Even more so, the availability of regularly collected data on health outcomes (e.g., hospitalizations or mortality) is very limited. These deficiencies hinder the ability to both set and track success in achieving air quality policies and reduce the ability of the scientific community to conduct regular and enhanced studies of the health effects of air pollution to support future analyses of health impacts and air quality decisions.

New and Emerging Challenges

In addition to these continuing challenges for air quality management in Asia, there are two major, rapidly emerging challenges that will require approaches that are substantially more comprehensive: traffic and land use and climate change.

Traffic and Land Use The rapid motorization of many Asian cities has substantial implications for air quality and, as noted above, will require more systematic efforts to improve fuels and vehicle emissions. These trends, however, have greater significance for urban development, traffic congestion, safety and noise, and the future quality of life in Asian cities. Addressing these issues will require more comprehensive approaches, engaging not only environmental agencies but also transport, economic development, and planning authorities.

Climate Change The strengthening world consensus about the need to address climate change has substantial implications for air quality management in Asia, as discussed above and in Sidebar 10. First, it is becoming increasingly apparent that climate change is likely to exacerbate exposures to certain air pollutants (e.g., O_3) (Royal Society 2008) and will make reducing those air pollutants more difficult. Second, there is the potential for significant co-benefits from integrated air quality and climate actions (e.g., potential health benefits from reducing emissions of the BC component of PM, which, at the same time, could reduce climate forcing). Finally, addressing these combined challenges will require more comprehensive and coordinated development of energy and environmental policy.

Future Directions

To date, the decision to take action to improve air quality in Asia has been driven by a number of factors: (1) growing public awareness of (and negative reaction to) very high, visible concentrations of pollution; (2) understanding of the health effects of air pollution derived from European and North American studies (and most notably from the WHO air quality guidelines [WHO 2005b] based on that literature); and (3) a small but growing number of local health studies conducted in Asian cities that have documented health effects of air pollution. In a few instances, action has also been spurred by concern over the effect of air pollution on historical monuments (e.g., the Taj Mahal in Agra, India) and overall visibility.

Actions have, for the most part, been taken by legislatures and regulatory agencies in Asia on the basis of reviews of existing information and technologies. The most notable progress in this regard has been (in some countries) the removal of lead from gasoline and the adoption of increasingly stringent emissions standards for new automobiles.

The speed and strength of such actions has, however, been lessened by several factors: the need of Asian governments to focus on a wide variety of other challenges (including other environmental public health challenges, such as waterborne diseases); the perceived conflict between objectives for economic growth and potential costs of environmental actions; and a reluctance to extrapolate the results of studies conducted in Western countries to assess probable health effects of air pollution in Asia. In some cases (especially recently in India), frustration with the slow pace of action has led to intervention by the courts, which have given direct orders to take action (e.g., ban the driving of older motor vehicles and require conversions to cleaner, alternative fuels).

Despite these challenges, several Asian cities have made substantial progress in implementing new measures, most often when those activities were informed by targeted efforts to document the local health consequences of air pollution and to estimate the economic impact. For example, air quality agencies in both Thailand and Hong Kong have sponsored health studies and economic analyses by local scientists; these studies have provided some rationale for substantial interventions to improve industrial fuel quality, to improve vehicle emission standards, and to take other actions. As a result, both Bangkok and Hong Kong have been among the leaders in Asia in implementing comprehensive air quality management programs.

Over the past several years, other efforts have been undertaken to better quantify the impact of air pollution on health and the economy, both in Asia and around the world. As part of its Integrated Environmental Strategies program, the U.S. EPA has worked with investigators in Shanghai, China; Hyderabad, India; Manila, Philippines; and others to conduct systematic analyses of the health and economic consequences of alternative energy use and air pollution control policies (National Renewable Energy Laboratory 2008).

These U.S. EPA analyses have attempted to use local health studies to estimate the health impact of air pollution. In many cases, however, the absence of local data has necessitated the extrapolation to Asian populations of effect estimates from North American and European studies. In 2002, the WHO also published the results of its Global Burden of Disease project (WHO 2002). WHO estimates that in 2000, Asian urban air pollution was responsible for approximately 520,000 premature deaths (WHO 2002). Although the analysis was conducted carefully, it also relied on U.S. data and modeled, rather than measured, air pollution concentrations to achieve a consistent set of analyses across all countries. Substantial improvement of local studies of the health effects of air pollution will be an important contribution to all future analyses of the health and economic consequences of actions to improve air quality.

CAI-Asia has organized extensive networks of local and national officials, environmentalists, industry representatives, scientists, and engineers to identify the most effective actions for cleaner air and, through regular workshops and the biannual Better Air Quality conference, have provided ample opportunity for the transfer of knowledge of air quality management to the wide range of stakeholders necessary for action to be taken.

SUMMARY

The Size of the Vulnerable and Potentially Exposed Population Is Increasing.

Demographic and disease trends occurring in rapidly developing countries of Asia suggest that the size of the population vulnerable to the adverse health impacts of air pollution is increasing. Specifically, larger numbers of people living in cities and surviving to older ages, an increased prevalence of tobacco smoking, increasing rates of obesity, and changes in dietary patterns are leading to increases in the burden of disease from cardiovascular disease, COPD, and cancer. Because air pollution can contribute to the incidence of these diseases and accelerate their progression, it is likely that the burden of disease related to air pollution in Asia will increase, even if air quality improves.

Asia Is Highly Dynamic and Heterogeneous, and Exposure to Air Pollution Is Becoming Increasingly Varied and Complex As Development Progresses.

Although this report provides an overview of air pollution exposure within Asia, the situation is highly complex and variable, both spatially and temporally, making it difficult to accurately predict future trends in air pollution concentrations in the region. For example, global and regional economic downturns may slow the pace of industrial development and temporarily reduce the rate of increase in emissions or provide opportunities for the adoption of more efficient and lower-emission technologies. The opposite may also occur, depending on national or local priorities. In addition, depending on specific local stages of development, environmental management, and regional influences, evidence suggests that ambient air quality is improving in some areas but may be worsening in others. Air pollution exposure is even more complex and is not well understood. For example, the impact of urbanization and poverty on air pollution exposures in Asia requires further research to make generalizations regarding specific groups that have elevated exposures and might therefore be at increased risk of health impacts.

Dramatic Increases in the Activities of Sources Are Occurring.

Despite this fact, however, pollutant concentrations in most large urban areas have generally stabilized or are decreasing (albeit with some notable exceptions). General trends in air quality, reflecting its heterogeneous nature within Asia, suggest that air pollutant concentrations are decreasing or have stabilized in most large urban areas in Asia. These reductions in air pollution have occurred in spite of dramatic increases in source activity (e.g., power generation, vehicle production, and vehicle ownership), demonstrating the effectiveness of air quality management, especially within large urban areas, and the adoption of more efficient energy use. Along with these improvements in air quality is recent evidence of increases in air pollutant concentrations at some locations and of the persistent and increasing importance of degradation of regional air quality.

Regional Air Quality and Climate Change Are Key Emerging Issues.

Some strategies of air quality management, however, may lead to new air quality issues for Asia. For example, relocation of power generation and industrial point sources outside of urban areas may improve urban air quality while leading to the overall degradation of regional air quality. As total air pollution sources and overall emissions increase, yet become more dispersed, regional, and transboundary air quality issues are likely to become increasingly important in Asia.

Climate change and the emission of GHG pollutants present not only an important challenge for Asia but also an opportunity for continued improvement in air quality. As discussed above, trends in air quality have largely shown improvement during periods of dramatically increased energy use in Asia, a testament to the impact of effective air quality management as well as improved efficiency and reduced intensity of energy use. Strategies for reduction in the emissions of GHG pollutants, although directed toward climate change, may also have direct impacts on local and regional air quality, resulting in faster and larger improvements than would otherwise occur in the region, with the potential for improved public health. The actual benefits to climate and public health that might accrue, however, will depend on which GHG pollutants are reduced, their relative potential for climate forcing, and their relative toxicities — matters that are still incompletely understood.

Section III. Summary of Current Evidence on Health Effects of Air Pollution: Implications for Asia

The past 10 to 15 years have seen a remarkable increase in research on the effects of air pollution on health. It is now widely accepted that exposure to outdoor air pollution is associated with a broad range of acute and chronic health effects, ranging from minor physiologic perturbations to death from respiratory or cardiovascular disease (Bascom et al. 1996; American Thoracic Society [ATS] 2000). This knowledge is based on observational epidemiologic studies of disease occurrence in human populations, in vivo experimental studies with animals and humans, and in vitro studies with human and animal tissues and cells. Epidemiologic research on the health effects of air pollution provides estimates of the health effects of both short- and long-term exposure to a variety of air pollutants in human populations in many parts of the world. Because these estimates apply to humans living in real-world conditions, they have been the scientific basis of increasingly stringent air quality regulations for some pollutants.

Epidemiologic studies of air pollution harness various dimensions of exposure variability that are observable in real-world settings (Pope and Burnett 2007). Individual epidemiologic studies often focus on only one dimension of exposure variability at a time within a population. Well over 100 published studies have used short-term temporal variation (over one or a few days) in air pollution concentrations to estimate effects on daily morbidity and mortality. Spatial variation in long-term mean concentrations of air pollutants has been the basis for cross-sectional and cohort studies of long-term exposure. A much smaller number of studies have used longer-term temporal variation (over weeks or months) resulting from natural experiments (naturally occurring events) or interventions (deliberate manipulation of conditions through direct or indirect actions) or have used relatively long lag times in time-series studies to explore time scales of exposure longer than just a few days but shorter than years. A more extensive summary and analysis of the results from these studies has been published (Pope and Dockery 2006).

STUDIES OF VARIABILITY IN EXPOSURE

Short-Term Temporal Variability

In air pollution epidemiology, the most studied dimension of exposure variability is short-term temporal variability. Episodes in which concentrations of air pollution increased rapidly and remained markedly elevated for a period of days occurred in Europe and the United States in the mid–20th century. These episodes provided some of the earliest epidemiologic evidence of the health effects of short-term exposure to air pollution (Anderson 1999). Today, such episodes are much less frequent in developed Western countries (Wichmann et al. 1989; Anderson et al. 1995), but in Asia, very high concentrations of air pollution due to burning of biomass can still result in high daily pollutant concentrations in some cities.

In the early 1990s, the results of several daily time-series studies were reported (Fairley 1990; Schwartz and Marcus 1990; Schwartz 1991; Dockery et al. 1992; Pope et al. 1992; Schwartz and Dockery 1992a,b; Schwartz 1993). These studies used time-series data and Poisson regression models to estimate the association between changes in daily mortality and daily changes in pollution while controlling for other time-dependent covariables that were potential confounders. These studies found small but statistically significant effects of air pollution on mortality, even at relatively low concentrations. The original research was reanalyzed and largely replicated (Samet et al. 1995), with similar associations observed in other cities with different climates, different modeling approaches used to describe weather conditions, different pollution mixes, and different demographic characteristics (Pope and Kalkstein 1996; Samet et al. 1998; Pope and Dockery 1999; Bell et al. 2004a).

There have been concerns regarding the specifics of the various modeling approaches used to assess short-term variability in exposure. However, over time, increasingly rigorous modeling techniques have been used in attempts to better estimate pollution–mortality associations while controlling for seasonality, other long-term time trends,

and weather variables such as temperature and relative humidity (Dominici et al. 2003c; Dominici 2004). Another methodologic approach, the case–crossover study design (Maclure 1991), has also been applied to studying effects on morbidity and mortality of daily changes in particulate air pollution (Lee JT and Schwartz 1999; Neas et al. 1999; Pope and Dockery 2006). The case–crossover design is an adaptation of the common retrospective case–control design. Exposures of study subjects at the time of the event (the case period) are matched with one or more periods when the event did not occur (control periods), and the potential excess risk associated with the event are estimated using conditional logistic regression. Individuals essentially serve as their own controls. By strategically choosing control periods, this approach restructures the analysis such that day of week, seasonality, and long-term time trends are controlled for by the study design rather than by statistical modeling (Janes et al. 2005). Useful reviews and discussions of the statistical techniques that have been used in these time-series and related studies have been published (Dominici et al. 2003c; Dominici 2004).

The most recent studies of short-term exposure have found associations between concentrations of airborne PM and a large range of outcomes, which have been reviewed extensively (Pope and Dockery 1999; Pope et al. 2006). PM pollution has been associated with daily rate of mortality (from all natural causes, respiratory causes, or cardiovascular causes), hospital admission for respiratory disease (from all natural causes, COPD, asthma, or pneumonia), and hospital admission for cardiovascular diseases (acute myocardial infarction or congestive heart failure). The magnitude of the estimated RRs from time-series studies of daily mortality depends on the approach used to model both the temporal pattern of exposure (Braga et al. 2001) and potential confounders that vary with time (such as season and weather) (Health Effects Institute 2003).

The majority of the daily time-series mortality studies have been conducted in North American or Western European cities, where air pollution concentrations are low and still falling and Western lifestyles and patterns of disease predominate (Stieb et al. 2002; Cohen et al. 2004). Fewer studies have been conducted in Central and South American countries and Asian countries. In virtually all locales that have been studied, however, daily mortality is positively associated with short-term (several days') exposure to approximately the same degree.

Since the publication of numerous single-city daily time-series mortality studies, many quantitative reviews and meta-analyses of data from these studies have been performed, to estimate the effect on mortality or other health measures of an increase in the concentration of a given air pollutant (called the effect estimate). The pooled effect estimates for mortality (in this report, usually shown as the percent change in daily mortality) from several of these meta-analyses and reviews are presented in Figure 32. For example, a meta-analysis was applied to 29 estimates of the effect of PM_{10} on mortality from 21 published studies to yield pooled estimates and to evaluate whether various study-specific factors explained some of the variability in the effect estimates across the studies (Levy et al. 2000). Elevated concentrations of PM_{10} were associated

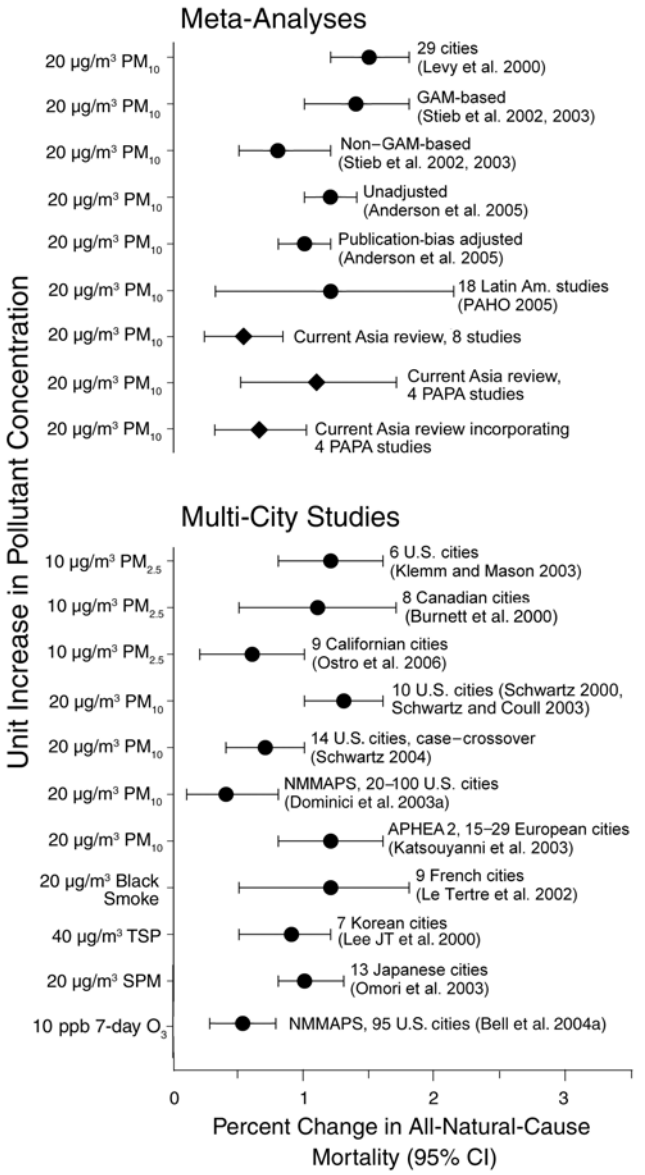

Figure 32. Estimates of percent change in daily mortality from all natural causes for selected meta-analyses and multi-city studies of daily changes in air pollution exposure, by study type and unit change in pollutant concentration. [Adapted from Pope and Dockery 2006 with estimates added from this Asian Literature Review meta-analysis (Table 15).]

with increased numbers of deaths (Figure 32) and PM effect estimates for mortality were greater in cities in which $PM_{2.5}$ makes up a higher proportion of the PM_{10}. Stieb and colleagues (2002, 2003) extracted effect estimates from reports of 109 time-series studies and calculated the random-effects pooled estimates of excess mortality. Statistically significant positive associations were observed between daily mortality and various measures of air pollution, including PM_{10}, but differences in effect estimates were observed, according to the modeling approach used. One concern regarding air pollution effect estimates for mortality from published single-city studies is the potential for city-selection and publication bias. Anderson and colleagues (2005) explored the existence of city-selection and publication bias in a meta-analysis of 74 time-series studies of daily mortality in single cities. Summaries of effect estimates were obtained, summarized, and examined for publication bias using an analysis of asymmetry in funnel plots (in which estimates are plotted against their standard errors). Although there was some evidence of publication bias, the effect estimates were not substantially altered after using a trim-and-fill method to correct for this bias (Figure 32).

Recently, large studies have been conducted using uniform methods for assembling and analyzing data from multiple cities. Pollution effect estimates for mortality from these multi-city studies are also presented in Figure 32. The studies represented include analyses of 6 U.S. cities (Klemm and Mason 2003), 10 U.S. cities (Schwartz 2000, Schwartz and Coull 2003), a study of 8 of Canada's largest cities (Burnett et al. 2000), and a study of 9 California cities (Ostro et al. 2006). A multi-city study that used the case–crossover study design was conducted using data from 14 U.S. cities (Schwartz 2004). As can be seen in Figure 32, statistically significant PM_{10}–mortality associations were found, similar to those estimated using the time-series approach.

One of the largest multi-city daily time-series studies is the National Morbidity, Mortality, and Air Pollution Study (NMMAPS). This study began with efforts to replicate several early single-city time-series studies (Samet et al. 1995) and was designed to address concerns about city-selection bias, publication bias, and confounding by copollutants. Results of various analyses of these data have been reported (Samet et al. 2000a; Dominici et al. 2000, 2003b,c; Peng et al. 2005). The PM–mortality effect estimates were somewhat sensitive to various modeling and city-selection choices. However, relatively small but statistically significant PM–mortality associations of a size comparable to those reported in Dominici et al. 2003a were consistently observed (Figure 32). The NMMAPS analysis also directly evaluated the influence of copollutants and found little evidence that the PM–mortality effect was attributable to any of the copollutants studied (NO_2, CO, SO_2, or O_3). Nevertheless, as indicated in Figure 32, an additional NMMAPS analysis detected associations of mortality with O_3 (Bell et al. 2004a). Multi-city time-series studies were also conducted in Europe. As part of the Air Pollution and Health: A European Approach (APHEA) and APHEA2 projects, various analyses were used to examine the short-term PM–mortality effects in 15–29 European cities (Katsouyanni et al. 1996, 1997, 2001, 2003; Zmirou et al. 1998; Samoli et al. 2001, 2003; Aga et al. 2003; Analitis et al. 2006) and PM was found to be significantly associated with daily mortality (see Katsouyanni et al. 2003 and Figure 32). Similar results were observed in a study of nine French cities (Le Tertre et al. 2002).

Three Asian multi-city studies — one of 7 major South Korean cities (Lee JT et al. 2000), one of 13 Japanese cities (Omori et al. 2003), and the PAPA studies in four cities in East and Southeast Asia (Wong CM et al. 2008b, 2010a, and Sidebar 1) — have also reported associations of daily mortality with measures of PM. As shown in Figure 32, the PM–mortality effect estimates from these multi-city studies in Asia do not appear to be substantially different from effect estimates from comparable studies in other parts of the world. In *Section V. Quantitative Assessment of Daily Time-Series Studies*, the Asian multi-city studies are discussed further in the context of the time-series meta-analysis.

Other approaches have also been used to study the acute health effects of short-term exposure to air pollution. For example, in panel studies, small groups (or panels) of subjects are followed over short intervals, and health outcomes, exposure to air pollution, and potential confounders are ascertained for each subject on one or more occasions. Panel studies have generally reported that exposure to outdoor air pollution is associated with increased upper and lower respiratory symptoms, small transient declines in lung function, and increased rates of asthma attacks and asthma medication use (Pope and Dockery 1999). In addition, several case–crossover studies from the United States and Europe have observed that short-term exposure to $PM_{2.5}$ is associated with an increased risk of acute IHD events (Peters A et al. 2001; D'Ippoliti et al. 2003; Zanobetti and Schwartz 2005; von Klot et al. 2005; Pope et al. 2006).

Long-Term Spatial Variability

Another important source of exposure variability is spatial (or cross-sectional) variability that includes differences in the long-term (i.e., 1 year or more) average

ambient concentrations across metropolitan areas as well as differences in average concentrations across smaller communities or neighborhoods within metropolitan areas. In the 1970s and 1980s, various population-based cross-sectional studies found that mortality rates in U.S. cities were associated with $PM_{2.5}$ (Lave and Seskin 1970; Evans et al. 1984; Özkaynak and Thurston 1987).

Several prospective cohort studies (in which large numbers of individuals exposed to various concentrations of air pollution are followed-up for years and their mortality rates are compared) have also investigated spatial variability in long-term average concentrations of air pollution. PM–mortality effect estimates from these studies are summarized in Figure 33. Original reports of the Harvard Six Cities (Dockery et al. 1993) and the American Cancer Society (ACS) (Pope et al. 1995) prospective cohort studies indicated that mortality was strongly associated with spatial differences in long-term average concentrations of $PM_{2.5}$, even after controlling for cigarette smoking and numerous other individual risk factors. Extensive reanalyses (Krewski et al. 2000) and extended analyses (Pope et al. 2002; Jerrett et al. 2005; Laden et al. 2006) of findings from these two early prospective cohort studies demonstrated that the $PM_{2.5}$–mortality effect estimates were remarkably robust to various modeling specifications and to controlling for individual risk factors including age, sex, race, smoking status, alcohol use, marital status, education, body-mass index, occupational exposures, and diet. Even analyses that included spatial smoothing (Pope et al. 2002) or ecologic (including socioeconomic) variables (Krewski et al. 2000, 2009; Jerrett et al. 2005) found little evidence of spatial confounding or confounding by "contextual conditions" at the neighborhood level. The two studies that also included within-city variability in exposure, by Jerrett and colleagues (2005) and Miller and associates (2007) (Figure 34), observed that the estimated pollution effects were relatively large — suggesting that studies that evaluate differences in exposure only across metropolitan areas may be underestimating the effects of pollution.

Other prospective cohort studies of adult mortality have generally observed positive, but less consistent or less statistically significant, PM–mortality associations (Abbey et al. 1999; McDonnell et al. 2000; Enstrom 2005). $PM_{2.5}$ was more closely associated with mortality than PM_{10} or TSP (sometimes referred to as suspended particulate matter [SPM]). Results of a study conducted in the Netherlands (Beelen et al. 2008), an extended analysis of a cohort of U.S. Veterans Administration patients in the United States (Lipfert et al. 2006), and a Norwegian cohort study (Nafstad et al. 2004) suggest effects of long-term exposure to air pollution, particularly air pollution related to road traffic.

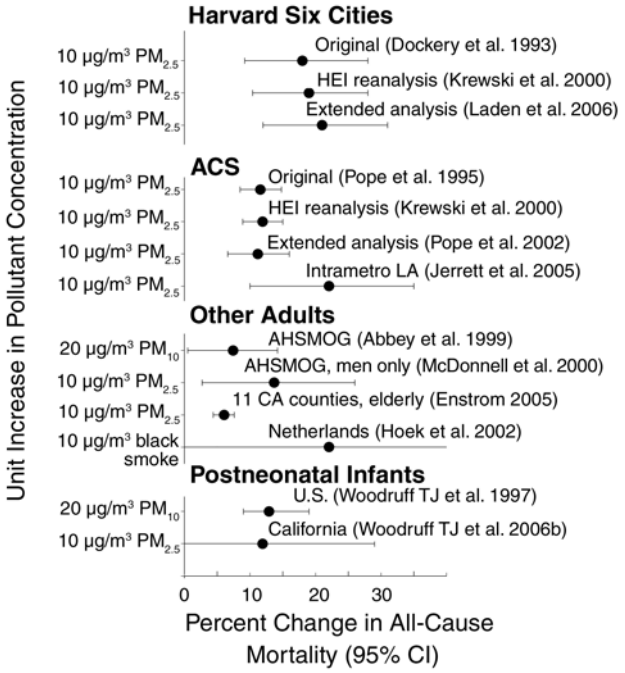

Figure 33. Estimates of percent change in daily all-natural-cause mortality from selected studies of long-term air pollution exposure, by study type and unit change in pollutant concentration. [Adapted from Pope and Dockery 2006.]

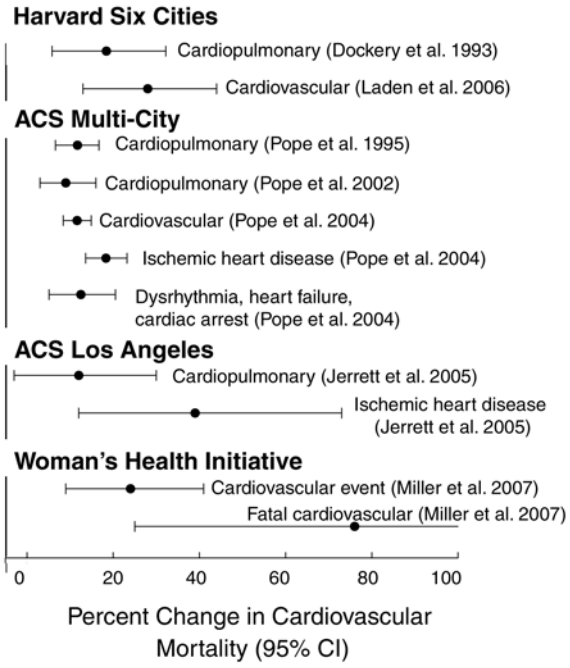

Figure 34. Estimates of percent change in risk of cardiovascular mortality per 10-µg/m³ change in annual average $PM_{2.5}$ concentration from selected cohort studies of long-term exposure. [Adapted from Pope and Dockery 2006.]

Interestingly, two studies have also observed similar pollution-related increases in post-neonatal infant mortality (Woodruff TJ et al. 1997, 2006b).

Analyses of the prospective cohort studies according to specific causes of death indicate that the long-term PM exposure is most consistently associated with cardiopulmonary or cardiovascular mortality, or both (Pope et al. 2004). Figure 34 presents adjusted RRs for cardiovascular mortality associated with $PM_{2.5}$ for various analyses of the Harvard Six Cities data (Dockery et al. 1993; Laden et al. 2006) and the ACS data (Pope et al. 1995, 2002, 2004; Jerrett et al. 2005). Also shown are adjusted RRs for nonfatal and fatal cardiovascular events estimated from a large cohort study of postmenopausal women without previous cardiovascular disease (Miller et al. 2007). A limitation of all of these cohort studies of mortality, however, is that no similar studies have been conducted in developing countries, and thus global estimates made from these studies carry large uncertainties (Cohen et al. 2004). Recently, HEI funded a pilot study in which the investigators attempted to retrospectively estimate long-term exposure to particulate air pollution in an existing cohort of older residents of Guangzhou, China. Despite careful execution, the investigators were unable to identify a population with an informative contrast in exposure using existing data from Guangzhou's air monitoring network — thus highlighting the difficulties in conducting studies of the chronic effects of long-term exposure in many Asian settings (Sidebar 13).

Long-Term Temporal Variability

The PM–mortality effect estimates from long-term prospective cohort studies (Figure 33) are substantially larger than those from the daily time-series and case–crossover studies (Figure 32). Much of the difference in the PM–mortality associations observed between the daily time-series and the prospective cohort studies may be due to the dramatically different time scales of exposure (a few days vs. years). Several epidemiologic studies of air pollution have attempted to evaluate intermediate scales of exposure by using medium-term temporal variability in exposure (over weeks to months). Examples include several studies of natural experiments or of interventions.

In the Utah Valley region of the United States, abrupt reductions in air pollution that accompanied a 13-month period of closure of a steel mill were associated with reductions in hospitalizations and mortality (Pope et al. 1992, Pope 1996). Substantial reductions in air pollution resulting from a ban on the use of coal burning in Dublin, Ireland (Clancy et al. 2002), resulted in reductions in respiratory and cardiovascular deaths. Reductions in seasonal mortality were observed in the first 12 months after restrictions were imposed on the sulfur content of fuel in Hong Kong (Hedley et al. 2002). Also, a recent analysis indicated that small but statistically significant drops in mortality were associated with one historical event: an 8.5-month strike by employees at a copper smelter that resulted in sharp reductions in SO_4^{2-} and PM concentrations and concentrations of related air pollutants across four states in the southwestern United States (Pope et al. 2007). This result was quite robust to the statistical control of time trends, mortality in bordering states, and nationwide numbers of deaths from influenza or pneumonia, cardiovascular causes, and other respiratory causes.

Another approach to analyzing daily time-series data for time scales of exposure substantially longer than just a few days is the use of extended distributed-lag times in time-series analyses. Schwartz (2000) used distributed-lag models to evaluate associations over periods of 1 to 5 days using data from 10 U.S. cities. Distributed-lag models were used to estimate associations for up to 40 days using data from 10 European cities (Zanobetti et al. 2002, 2003) and Dublin, Ireland (Goodman et al. 2004). In all of these analyses, the cumulative PM–mortality effect was larger when time scales longer than a few days were used.

Figure 35 compares the percent changes in risk of mortality associated with PM, estimated for various time scales of exposure, integrating the evidence from the studies discussed above. The estimated percent change in risk of mortality associated with a change of 10 µg/m^3

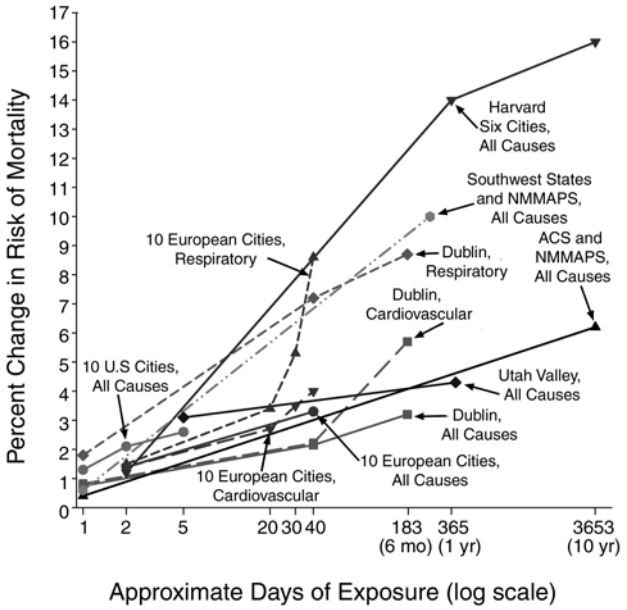

Figure 35. Comparison of percent changes in risk of associated with a change of 10 µg/m^3 $PM_{2.5}$, 20 µg/m^3 PM_{10}, or 20 µg/m^3 black smoke concentrations across various time scales of exposure. [Adapted from Pope 2007.]

Sidebar 13. A PILOT STUDY IN GHANGZHOU

Although long-term cohort studies provide information about the effects of long-term exposure to air pollution, currently available data from Western countries, such as from the U.S.-based ACS cohort study, inform the air pollution concentration–response function only up to around 30 μg/m^3 $PM_{2.5}$. Because of the dearth of data from regions exposed to the relatively higher concentrations of air pollution found in Asia, however, data from the ACS cohort are routinely used in health impact assessments regarding ambient air pollution in regions around the world (Cohen et al. 2004).

To assess the effects of long-term exposure to the relatively high pollution concentrations found in Asia, the PAPA program funded a pilot study to assess the feasibility of conducting a prospective cohort study of the effect of long- and short-term exposure to air pollution on morbidity and mortality from respiratory and cardiovascular diseases among older people in Guangzhou, China.

Dr. Tai Hing Lam and colleagues at the University of Hong Kong explored whether the available data were adequate to characterize long-term, cumulative exposure to air pollution in the study population of approximately 10,000 older Guangzhou residents (Figure 13.1). Unfortunately, the investigators were unable to demonstrate a sufficient gradient of exposures in Guangzhou, as even the relatively clean area in the study had PM_{10} concentrations of 135 μg/m^3 during the study period, and because even in the winter, when the most extreme gradient of pollution was observed (Figure 13.2), there was very little variation among the exposures of the study population. Assessing the effects of long-term exposure to air pollution in Asia remains a key research priority.

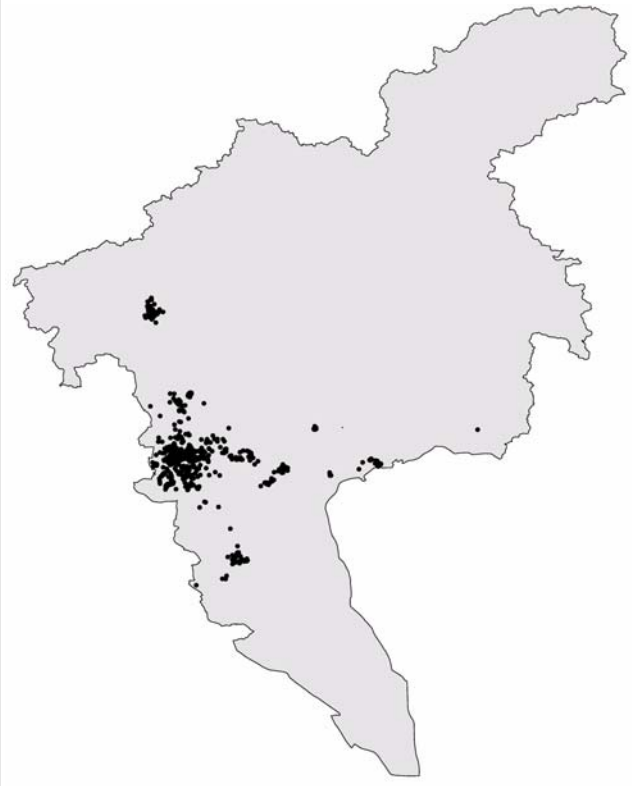

Figure 13.1. Geographic distribution of patients in Guangzhou. [Reprinted from Lam et al. 2009.]

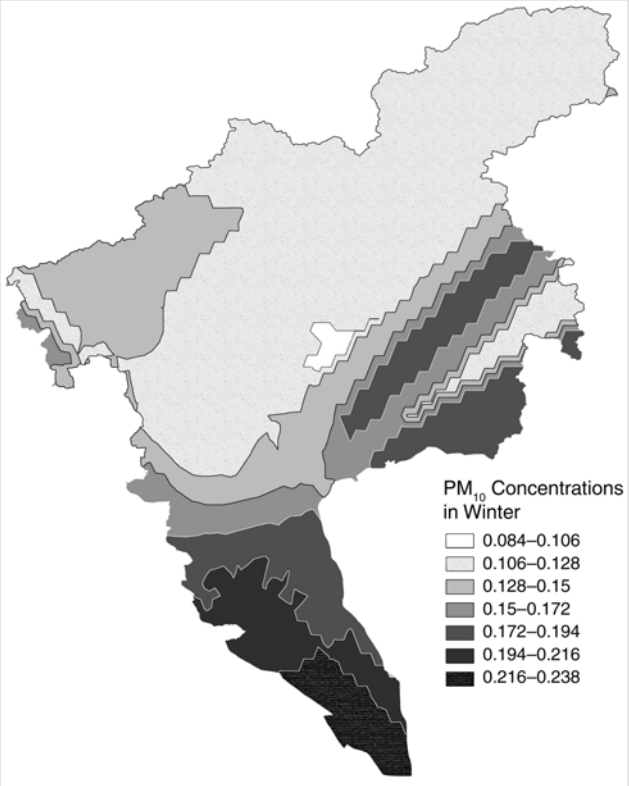

Figure 13.2. Predicted winter concentrations of PM_{10} (mg/m^3) in Guangzhou. [Adapted from Lam et al. 2009.]

$PM_{2.5}$, 20 μg/m^3 PM_{10}, or 20 μg/m^3 black smoke is plotted according to the exposure period (approximate number of days, on a log scale). The PM effects increase with increasing duration of exposure, and the most recent exposures have the largest effects (thus the need for a log scale for time). With regard to the natural experiments and intervention studies in the Utah Valley, in Dublin, and at the Southwestern U.S. copper smelter, the expected drop in the risk of mortality due to a reduction in exposure may not be completely realized over the relatively short period — just 6 to 13 months. However, extended analyses of the Harvard Six Cities Study suggest that the reduction in mortality risks

may be largely realized within as short a time as 1 to 2 years. Regardless, the evidence suggests that the short-term exposure studies capture only a small amount of the overall health effects of long-term, repeated exposure to PM. Adverse health effects are dependent on both exposure concentrations and length of exposure, and long-term exposures have larger, more persistent cumulative effects than short-term exposures. (For a more detailed discussion, see Pope and Dockery 2006 and Pope 2007.)

AIR POLLUTION, ADVERSE REPRODUCTIVE OUTCOMES, AND CHILD HEALTH

As noted above and indicated in Figure 33, air pollution has been associated with postneonatal infant mortality (Woodruff TJ et al. 1997; 2006b). Recent reviews have generally concluded that PM air pollution exposure is fairly consistently associated with postneonatal respiratory mortality but that there is less compelling evidence of a link between PM and sudden infant death syndrome (Glinianaia et al. 2004a; Tong and Colditz 2004; Lacasana et al. 2005; American Academy of Pediatrics Committee on Environmental Health 2004; Dales et al. 2004; Maisonet et al. 2004; Šrám et al. 2005). Time-series studies of daily mortality in Mexico, Thailand, and Brazil have reported associations between short-term exposure to particulate pollution and daily mortality from all natural causes or acute respiratory infections in children less than 5 years of age (Romieu et al. 2002; Cohen et al. 2004). A rapidly growing literature about the effects of air pollution on adverse reproductive outcomes such as low birth weight, premature birth, fetal growth retardation, intrauterine mortality, and birth defects (American Academy of Pediatrics Committee on Environmental Health 2004; Maisonet et al. 2004; Šrám et al. 2005) suggest that air pollution exposure impacts fetal growth, premature birth, and related birth outcomes, though important gaps in our knowledge remain regarding these associations.

With regard to air pollution and the health of growing children, several studies provide evidence that children with long-term exposure to air pollution experience slower growth in lung function. For example, a study of U.S. and Canadian children was conducted in the 1990s (Raizenne et al. 1996) and a more recent long-term prospective study was performed with children 10 to 18 years of age living in 12 southern California communities (Gauderman et al. 2004, 2007). Gauderman and colleagues (2004) reported that children living in communities with relatively high concentrations of air pollution have relatively low levels of growth in lung function and an increased risk of having a forced expiratory volume in 1 second [FEV_1] of < 80% of the predicted value (Figure 36). Further analysis of these children (Gauderman et al. 2007) showed deficits in their growth of lung function that increased as residential distance to major roadways decreased (Figure 36). The extent to which these deficits in growth of lung function will affect the long-term health of these children is not fully understood, but given the importance of lung function as a determinant of morbidity and mortality throughout adulthood, these pollution-related deficits in growth between 10 and 18 years of age are a substantial concern.

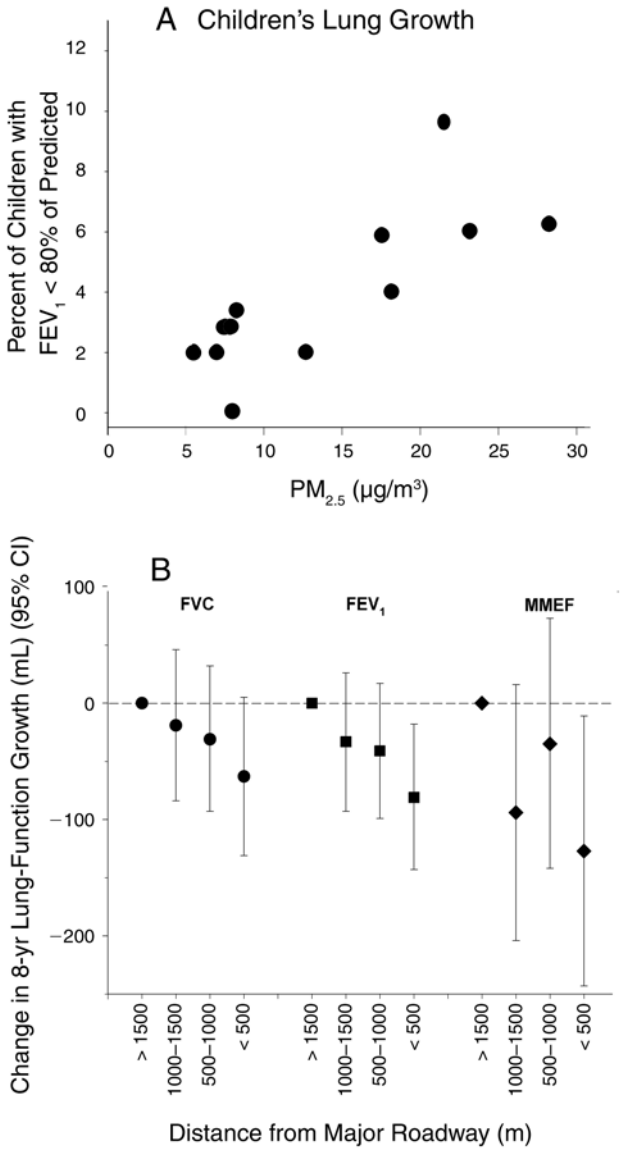

Figure 36. Data from the Children's Health Study regarding the effect of air pollution exposure on lung development. A: Percent of children with growth in lung function (measured as FEV_1) of < 80% of the predicted value, according to community mean $PM_{2.5}$ concentrations. **B:** Mean (95% CI) change in growth of lung function (measured as FVC, FEV_1, and MMEF) at the end of an 8-year period, as compared with the start of the period, according to distance of residence from major roadways. [Adapted from Gauderman et al. 2004 (A), 2007 (B).]

CONTRIBUTION OF COPOLLUTANTS

As noted above, fairly consistent associations between $PM_{2.5}$ and various cardiopulmonary health endpoints have been observed, yet the roles of specific constituents of $PM_{2.5}$ and the role of various gaseous copollutants have yet to be fully resolved. Measured concentrations of $PM_{2.5}$ can in many contexts be thought of as an index of combustion-related air pollutants that include copollutants such as O_3, CO, SO_2, and NO_2.

There is substantial evidence, however, that combustion-related copollutants also contribute independently or synergistically to the observed health effects. To date, in addition to PM, the evidence has led to a focus on three additional, related types or sources of pollution: tropospheric O_3, traffic-related pollutants, and sulfur oxides.

As noted earlier and as indicated in Figure 32, analysis of the NMMAPS data suggested daily changes in mortality associated with daily changes in O_3 concentrations (Bell et al. 2004a). A recent extended analysis of data from the ACS cohort attempted to evaluate the contribution of long-term O_3 exposure to air pollution–related mortality (Krewski et al. 2009). Although cardiovascular mortality appeared to be more strongly associated with $PM_{2.5}$ pollution than O_3, O_3 exposure appeared to be associated with an increased risk of respiratory mortality, even after controlling for $PM_{2.5}$ exposure.

Exposure to PM from traffic sources includes exposure to substantial secondary NO_3^- and coexposure to NO_2 and CO. There is growing evidence of a role of traffic-related exposures more generally. The analyses by Lipfert and colleagues (2006) of U.S. Veterans Administration data for a cohort of male patients with hypertension suggested that traffic density was a more "significant and robust predictor of survival in this cohort" than $PM_{2.5}$. A recent Dutch study (Beelen et al. 2008) and others (Adar et al. 2007; Samet 2007) have found that indicators of traffic-related exposure such as NO_2, NO_x, and traffic density and living near major roads are associated with an increased risk of mortality.

Likewise, exposure to pollution from coal-burning sources (or the burning of any sulfur-containing fuels) typically includes substantial exposure to secondary SO_4^{2-} and exposure to SO_2, in addition to PM exposure, and there is ample evidence that sulfur oxide–related pollution contributes to health effects. As examples, in the Harvard Six Cities Study (Dockery et al. 1993) and the ACS cohort study (Krewski et al. 2000; Pope et al. 2002), mortality was associated with both $PM_{2.5}$ and various sulfur oxides (including SO_4 and SO_2). The mortality reductions associated with the 8.5-month strike at a copper smelter (mentioned above) appeared to be due to reductions in the concentrations of SO_4^{2-} and related air pollutants across four Southwestern states (Pope et al. 2007). Also, at a workshop on source apportionment, several independent research teams that evaluated effects of short-term exposure found that the SO_4^{2-}-related components of $PM_{2.5}$ were most consistently associated with daily mortality (Thurston et al. 2005).

Depending on the source of pollution, concentrations of O_3, SO_2, NO_2, or CO may be reasonable indexes of exposure to pollution from various sources and are often associated with adverse health effects similar to those from PM. However, the literature has yet to provide evidence that a single constituent or source of PM is responsible for the health effects (WHO 2006a, 2007c). Though there is ample evidence that various related copollutants contribute to observed health effects, the independent, additive, or synergistic effects of gaseous copollutants are not fully understood and remain a matter of debate. A substantial challenge regarding air pollution studies in many parts of Asia has to do with limited air pollution monitoring of $PM_{2.5}$, O_3, and various other pollutants.

GAPS AND LIMITATIONS OF CURRENT KNOWLEDGE

Much has been learned about the adverse health effects of outdoor air pollution in the past 25 years, with epidemiologic research playing a critical role in the growth of this knowledge. Nonetheless, important gaps remain — especially the following questions that are critical to public policy. Which constituents of the pollutant mix, and which sources contributing to it, are the most toxic? Which diseases, social conditions, or genetic factors place people at increased risk when they are exposed to air pollution? To what extent does long-term exposure, especially during childhood, affect the development of chronic, life-threatening disease? The answers to these questions may well be different in Asia than in the developed West, where to date the vast majority of studies have been conducted.

Of particular relevance to Asia is the current uncertainty regarding the shape of the air pollution concentration–response function for long-term exposure and mortality from chronic cardiovascular and respiratory disease at levels in major Asian cities that exceed, by several orders of magnitude, the levels that are observed in contemporary U.S. and European studies.

Section IV. Updated Review of the Asian Literature on Air Pollution and Health

LITERATURE REVIEW

The objective of this section is to describe the epidemiologic studies of the health effects of air pollution in Asia that were reported in peer-reviewed literature from 1980 through 2007. For this Special Report, we chose three regions of Asia: East Asia (including China [mainland China, Hong Kong, and Taipei,China], Japan, and South Korea), South Asia (including India), and Southeast Asia (including Indonesia and Thailand).

Since the first comprehensive literature review was completed and reported on in Special Report 15 (HEI ISOC 2004), HEI has regularly updated and maintained a database of the relevant epidemiologic studies through its PAPA–SAN program. At the time of this publication, the PAPA–SAN Web site (*www.healtheffects.org/Asia/papasan-home.htm*) contained summaries of over 400 publications of air pollution and health conducted across Asia that were systematically compiled from the peer-reviewed scientific literature. In order to provide policy makers, international lending organizations, and other key stakeholders with easy access to the latest air pollution research from across Asia, the PAPA–SAN results are summarized on the HEI Web site (Health Effects Institute 2008). For each report, PAPA–SAN provides the citation, a summary of findings, other key data and, when available, a link to the abstract. Summaries are presented in table form, sorted by country or region, study design, pollutant, and health outcome; they also are provided in written form by country.

SEARCH METHODS

The first step in the literature review was to update the PAPA–SAN database by identifying all studies of populations in the Asian region. In January and August 2007, searches of the peer-reviewed literature were performed to identify any new, relevant studies on the health effects of outdoor air pollution in Asia published from 1980 through 2007. Structured literature searches of the PubMed (*www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=PubMed*), Web of Science (*http://isiwebofknowledge.com/*), and EMBASE (*www.embase.com/*) databases were conducted. Table 11 shows the medical subject headings (MeSH; the

Table 11. Search Strings Used in the PAPA-SAN Asian Literature Search for 1983–2007, by Search Engine

Search Engine	Search String
PubMed	(Asia or China or Hong Kong or India or Indonesia or Japan or Korea or Malaysia or Philippines or Singapore or Taiwan or Thailand or Vietnam or Bangladesh or Bhutan or Nepal or Sri Lanka or Cambodia or Burma or Laos) and (air pollution or air pollutants or air quality or dust or smog or smoke or vehicle emissions) and (health or mortality or morbidity or adverse effects or prevalence or epidemiology)
EMBASE	(Asia or China or Hong Kong or India or Indonesia or Japan or Korea or Malaysia or Philippines or Singapore or Taiwan or Thailand or Vietnam or Bangladesh or Bhutan or Nepal or Sri Lanka or Cambodia or Burma or Laos) and (air pollution or air pollutants or air quality) and (health or mortality or morbidity or adverse effects or prevalence or epidemiology)
Web of Science	(Asia or China or Hong Kong or India or Indonesia or Japan or Korea or Malaysia or Philippines or Singapore or Taiwan or Thailand or Vietnam or Bangladesh or Bhutan or Nepal or Sri Lanka or Cambodia or Burma or Laos) and (air pollution or air pollutants or air quality) and (health or mortality or morbidity or adverse effects or prevalence or epidemiology)

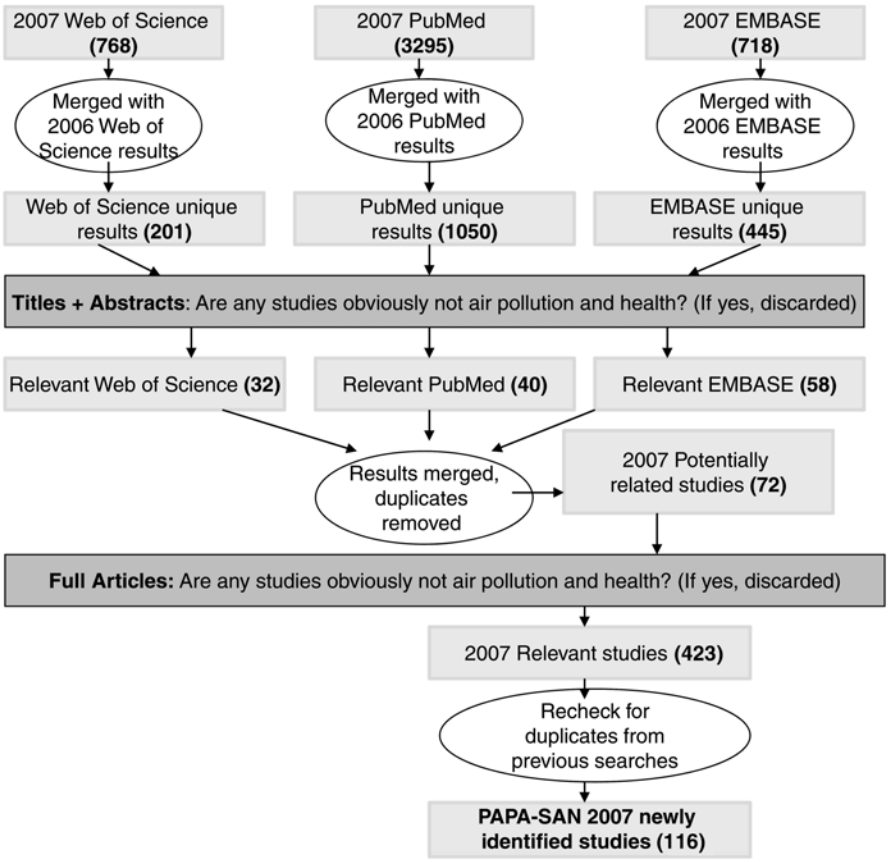

Figure 37. Steps of the PAPA–SAN Asian literature review.

National Library of Medicine's controlled vocabulary used for indexing articles) and other terms that were used to search each of the three databases.

Searches of leading preventive medicine and epidemiology journals in Chinese and South Korean languages were also conducted using the China National Knowledge Infrastructure (*www.cnki.net*) and KoreaMed (*www.koreamed.org*). A manual search of the International Society for Environmental Epidemiology (ISEE) and other institutional Web sites was conducted to identify conference proceedings and Web publications pertinent to PAPA–SAN. These include the following: WHO regional and national offices, the United Nations Environment Programme (UNEP), CAI-Asia, the World Bank, and the Asian Development Bank.

The results of the searches from each database, including the full reference and abstract (if available), were combined into a master database using Procite software.

Figure 37 shows the flow chart used for evaluating relevant studies and removing duplicate results. We excluded studies if (1) they were conducted in countries that were not relevant to PAPA–SAN, (2) they pertained to exposures other than ambient outdoor air pollution (i.e., indoor or occupational exposures), (3) they conducted animal toxicology or clinical studies with humans, or (4) they assessed or measured only concentrations of air pollutants and not health effects. In addition, articles that did not report original research or reported only estimates of either health effects or air pollution effects but not both were also excluded. Finally, specific key words that were used to exclude studies from the review were as follows: Turkey, Israel, Lebanon, Saudi Arabia, Kuwait, Iraq, Iran, Uzbekistan, Kazakhstan; indoor, tobacco, cigarette, smoking, cooking; radiation, radioactive, radon, arsenic, dioxin, cadmium, formaldehyde, mercury, mite, mold, fungi, pollen, asbestos; rat, mice, insect, in vitro; occupational, workplace, workers, industry, laboratory; clinic, sick building, social, tree, plant, diet, war, x-ray, acid rain; and policy, education, review, case report, accident, cost–benefit, economic, and meta-analysis.

After all of the searches were complete and the relevant papers were identified, results were compared with the St. George's Air Pollution Epidemiology Database (APED;

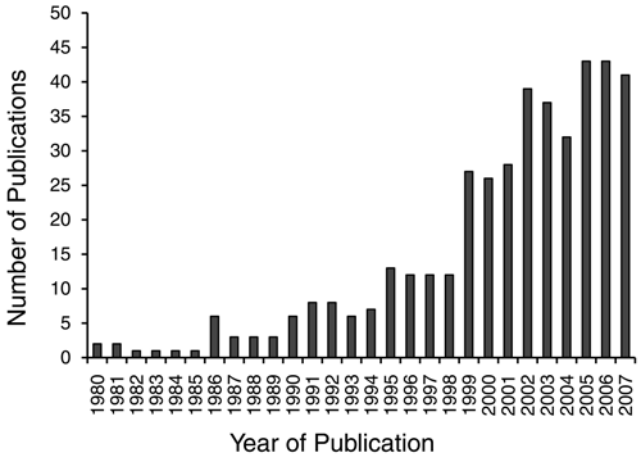

Figure 38. Studies identified in the PAPA–SAN Asian literature review, according to year of publication, 1980–2007.

described below in more detail in *Section V. Quantitative Assessment of Daily Time-Series Studies*), which contains a body of time-series and panel studies that is periodically updated using systematic search methods. References identified in either PAPA–SAN or APED, but not both, were shared, and the reasons for the omission in one database were examined. Discrepancies between the listings of a given study in the two databases were also reconciled.

A form was developed for extracting key information from each pertinent study. The key information included the author or authors, publication journal and year; study location, design, study period, population, exposure, and outcomes; and major findings as reported and summarized by the authors. For quality control, data extraction was conducted independently by two reviewers trained in epidemiology. Any discrepancies were resolved by consensus of the reviewers. All relevant publications were added to an Access (Microsoft Corp., Redmond, WA) database and summary statistics were tallied using Statistical Package for the Social Sciences software, version 15.0.

Table 12. Studies Identified in the PAPA-SAN Asian Literature Review, by Location Studied[a]

Location	Publications (*n*)
Bangladesh	2
China	110
Hong Kong	25
India	43
Indonesia	10
Japan	60
South Korea	51
Malaysia	3
Pakistan	3
Singapore	10
Sri Lanka	3
Taiwan[b]	80
Thailand	26

[a] Three of the 423 studies spanned multiple countries and are counted with each country, hence 426 in this table.

[b] Taiwan was used as a search term for Taipei,China.

SEARCH RESULTS

The current database includes results of a search conducted in January 2007 and another in August 2007 that identified 144 publications, bringing the total number of publications in the PAPA–SAN database to over 400 that were published between 1980 and 2007 (Figure 38). These publications included studies from 11 Asian countries: Bangladesh, China (mainland China, Hong Kong, and Taipei,China), India, Indonesia, Japan, South Korea, Malaysia, Pakistan, Singapore, Sri Lanka, and Thailand. Table 12 and Figure 39 show the number of publications according to the country in which the study was conducted. For the first time, the search identified studies from Bangladesh, Pakistan, and Sri Lanka. Most of the newly identified publications focused on respiratory and cardiovascular outcomes, which continue to illustrate the adverse effects of ambient pollution in the area. In addition, 27 publications summarized the public health impacts of air pollution, with some studies including estimates of the public health costs in economic terms. Studies examining subclinical markers of lead exposure (21) and benzene exposure (11) were also identified.

Figure 39. Number of studies identified in the PAPA–SAN Asian literature review, according to country of study.

Over half of the studies (216) estimated the health effects of exposure to both PM and gaseous pollutants, and others estimated the effects of exposure to PM only (50) or gaseous pollutants only (30). Collectively, the studies examined health outcomes that span the range of health effects recently identified as "adverse" by the ATS (2000) (including death, respiratory symptoms, lung function, and adverse pregnancy outcomes) (Table 13). Several also examined biomarkers of exposure and intermediate effects, including hospital admissions. The studies used a variety of designs (Table 13). Cross-sectional studies (chiefly of respiratory symptoms and pulmonary function) and time-series studies (including episode studies) made up approximately 63% of the total.

A detailed quantitative review was conducted for a subset of the 423 studies identified, focusing on the time-series studies of daily mortality and hospital admissions. On the basis of the results as reported by the authors and summarized in the PAPA–SAN database, we found that most studies conducted in Asia report that air pollution is associated with an increased risk or prevalence of a wide variety of adverse health outcomes in adults and children (ATS 2000). Given the wide diversity of study designs and data sources, the quality of each of the 423 studies was not assessed, with the exception of the daily time-series studies (which were the focus of APED). In this initial review, it was also not possible to assess the likelihood that only, or predominantly, positive studies had been published (i.e., the likelihood of publication bias) for the literature as a whole.

Table 13. Characteristics of the 423 Studies Identified in the PAPA-SAN Asian Literature Review, 1980–2007

Characteristic	Studies (*n*)
Health Outcome[a]	
Death	99
Hospital admission, visit, or discharge	46
Respiratory symptoms, lung function, or asthma	174
Biomarker	47
Adverse pregnancy outcomes	23
Lung cancer	18
Economic assessment index	22
Other	44
Study Design	
Cross-sectional	168
Time series[b]	98
Cohort	28
Panel	28
Case–control	10
Case crossover	20
Health impact	44
Ecologic	24
Descriptive	2
Unspecified	1
Exposure[a]	
TSP, RSP, SPM	118
PM_{10}	147
$PM_{2.5}$	52
NO_x	73
SO_2	207
CO	86
O_3	104
Lead	21
Unspecified	34
Other	44

[a] Studies could have addressed more than one health outcome or exposure.

[b] Time-series studies included episode studies.

Section V. Quantitative Assessment of Daily Time-Series Studies

INTRODUCTION

Time-series studies of daily mortality have been conducted in a wide range of cities and countries around the world using broadly similar methods of analysis and reporting of results. The evidence they provide contributes to public policy decisions at national and international levels. The contribution of the Asian time-series literature to this international body of evidence was critically evaluated in 2004 (HEI ISOC 2004). This section presents updated results for the quantitative analysis of the time-series studies of daily mortality and hospital admissions in Asia. Publications of time-series studies in English and Asian languages were identified in the PAPA–SAN literature search, as described above, and in APED, which is detailed below. We first describe the general characteristics of these studies (their locations, population characteristics, and details of exposures) and then discuss in greater detail studies of selected combinations of pollutants and health outcomes. Where appropriate (and possible), we assessed variation (heterogeneity) in the direction and sizes of the effect estimates extracted from these studies before calculating summary estimates. We also evaluated the possibility that publication bias may have influenced the results of the meta-analysis.

Two search strategies were used to identify appropriate studies for inclusion in this review. The first, used to construct the PAPA–SAN database, focused on the identification of air pollution studies on health in Asia. The second search strategy, used to construct the APED at St. George's, University of London, focused on the identification of daily time-series studies of the short-term health effects of air pollution worldwide. Results from the two databases were then combined to arrive at a definitive list of Asian time-series studies, as described above.

SYSTEMATIC ASCERTAINMENT OF RELEVANT TIME-SERIES STUDIES FOR APED

Literature Search

The search strategy used in APED aimed to identify published time-series studies indexed in PubMed, EMBASE, and Web of Science. Search strings were developed to identify time-series studies on the basis of study design, pollutants, and health outcomes. An example of these search strings is as follows (the syntax varies slightly among the three search engines; see Figure 37): "(air pollution or pollution or smog or particle or particulate or ozone or black smoke or sulfate or sulfur dioxide or nitric oxide or nitrogen dioxide or carbon monoxide) and (timeseries or time-series or time series or daily) and (mortality or death or dying or hospital admission or admission or emergency room or visit or attendance or 'a&e' or 'a and e' or accident and emergency or general pract or physician or consultation or emergency department)". The studies identified in the three search engines were downloaded into Reference Manager software, version 11 (Thompson Reuters, Carlsbad CA), where they were combined and duplicates removed (see Figure 37).

The results of the APED searches were then combined with the results of searches of time-series studies in particular for PAPA–SAN. The resulting list was supplemented by studies identified by local investigators and from searches of local Asian Web-based literature databases, as described above.

A two-stage selection process was then used to remove the studies that were not obviously time-series studies and then to identify, from study abstracts, those that were definitely time-series studies. Printed copies were then obtained and their suitability for inclusion in APED was assessed. The result of this sifting process was a list of time-series studies that may or may not provide numerical estimates of the short-term effects of air pollution on health. The utility of these studies depended on the details of the study design, statistical methods, and presentation of results. The studies were then checked to see whether they met the selection criteria, all of which had to be met for inclusion:

- The study was based on at least 1 year of daily data.
- The analysis included some attempt to control for important confounding factors (such as season and long-term time trends in the data).
- The study reported sufficient information for the calculation of a standardized effect estimate for comparison in the quantitative analysis (i.e., effect estimates

for a standard increment in pollution). For this to be possible, regression coefficients must have been reported. (A study that reported only correlation coefficients would not meet this criterion.)

- The study focused on a general population. Studies of any kind of subgroup of the general population, such as smokers, women, or people with heart disease did not meet the criterion.

Data Abstraction

For each time-series study that provided sufficient numerical information to enable their effect estimates to be standardized, a data extraction form was completed. Each form was divided into two parts, the first part containing study information and the second part, estimate information. Study information included the study identifier (Reference Manager identification number), title, authors, and full reference listing. Estimate information included details about the health outcome and pollutant being studied plus many more data necessary for quantification and standardization, such as the units of measurement, the duration of each daily measurement (e.g., 24 hours or 1 hour), and the range used to scale the effect estimates.

The information on each data extraction form was then entered into a Microsoft Access (version 2002) database. Data related to the study as a whole included study title, authors, journal name, volume and page numbers, length of the study period, year of the study, and the continent and Asian region in which the study occurred. Data specific to each regression coefficient that quantified the change in a given health outcome per unit change in a given pollutant were also recorded. These data were used to calculate standardized effect estimates and their 95% CIs.

The studies reported RRs, regression coefficients, or percent changes in the mean number of events per day as measures of the association between pollutant concentrations and health outcomes. In order to make results comparable across the studies, we used Microsoft Access queries to convert estimates into a standard form: the percent change in the mean number of daily events associated with a 10-μg/m^3 increase in pollutant concentration (or a 1-mg/m^3 increase for CO), which, for simplicity, we refer to as estimates. When the logarithm of pollutant concentration was used as the measure of exposure in the model, the estimates were calculated for a 10-μg/m^3 increase above the mean or median pollutant concentration (or a 1-mg/m^3 increase for CO).

Selection of Lag Times

The short-term relations between air pollution and health effects are complex and not wholly captured by the regression techniques generally used. The number of hospital admissions or deaths on any one day is likely to have been affected by exposure to air pollution on the same day and on several previous days. Also, air pollution on a particular day is likely to affect health on a number of subsequent days. In time-series analyses, the cumulative effects of several days of previous exposure are sometimes investigated by means of a cumulative lag time, in which exposures are averaged over 2 or more previous days. More recently, distributed-lag models have been used to estimate the independent contribution to health outcomes on a given day of each of several previous days. Both analytic approaches tend to estimate larger effects than analyses based on single-day lag times (Samet et al. 2000b; Zanobetti et al. 2002), although single-day lag times are used far more commonly.

Investigators vary in which lag time (single, cumulative, or distributed) they choose to analyze and present in publications. They may systematically choose results that indicate larger or smaller effects. Particularly in studies with low statistical power, the play of chance (random error) will lead to a wider scatter of estimates from which to choose. This fact has led some analysts to test or report a relatively restricted range of lagged estimates. Many studies report on one or more single-day models, but the results are not generally chosen or presented in a consistent way. For this reason, we could not select a particular lag time (e.g., 1 day before the health event of interest) a priori without having to remove studies from the analysis. We therefore adopted the following approach for selecting a lag time for analysis. First, we focused our selection on single-pollutant rather than multi-pollutant models. Single-pollutant analyses are reported in virtually all studies. The pollutants included in multi-pollutant models tend to vary among studies; therefore, their results are more difficult to compare. However, when only results from multi-pollutant models were given, the results from the model with the most pollutants were selected and recorded. This act simplified the abstraction of data from papers in which many combinations of pollutants were examined.

Using this approach, we identified selected lag times from the results presented in the papers. If an estimate of only one lagged time was presented (because only one was either analyzed or reported), this estimate was recorded in the Access database for the outcome–pollutant pair. If more than one lag measure was presented, we selected one for meta-analysis:

- The lag time that the author focused on or stated a priori,
- The lag time that was the most statistically significant (whether positive or negative), or
- The lag time with the largest effect estimate (whether positive or negative).

Results for a lag time of 0 days (lag 0) and 1 day (lag 1) were also recorded (if different from results for the selected lag time). When available, results for a cumulative lag time (i.e., the average of pollution measures over 2 or more days) chosen according to the same selection criteria were also recorded.

Multi-City Studies

Several studies presented the results of meta-analyses, including effect estimates for individual locations, multiple locations, and summary estimates (i.e., effect estimates for all the locations together). If such studies used previously published data, we recorded only the summary estimates. If previously unpublished city-specific results were presented, we recorded those as well as the summary estimates. The city-specific results were then processed and assessed as if they came from a single-city study.

META-ANALYTIC METHODS

Multiple Studies of a Single City

A number of studies have been conducted in the same city, with varying degrees of overlap in the years studied. Some studies are by the same investigators; others are not. We recorded all results from all studies. However, when calculating summary estimates across cities, we used the most recent estimate for each city, on the basis that it would be the most likely to reflect current analytic techniques and recent pollution concentrations. By not including several estimates from one city in the meta-analysis, we avoided weighting that city disproportionately, at the cost of losing informative estimates.

Computation of Summary Estimates

For pollutant–outcome pairs for which there were four or more estimates, we computed summary estimates of the percent changes and their 95% CIs using a fixed-effects model (with inverse variance weighting), as well as a random-effects model using the method of DerSimonian and Laird (1986). The *metan* procedure in the software package STATA was used to calculate the summary estimates from the specific regression coefficients and their associated SEs. These statistics were determined from the RRs presented in the studies. Summary estimates from both the fixed-effects and random-effects models are presented in forest plots. We tested the study-specific estimates for evidence of differences in result size or direction (heterogeneity) using the χ^2 test for heterogeneity. Because of the relatively low power of this test to detect such differences, P values less than 0.2 were considered to indicate evidence of heterogeneity.

Investigation of Publication Bias

The meta-analysis used results published in peer-reviewed journals. These may not represent all available evidence, because some relevant reports may be published in other formats or may not be published at all. The exclusion of representation of these reports could affect our findings: Publication bias refers to the tendency for findings that support a particular hypothesis (in this case, that air pollution has an adverse effect) to be published preferentially in peer-reviewed journals (Sterling 1959; Mahoney 1977; Simes 1986; Begg and Berlin 1989; Dickersin 1997). Publication bias could have two consequences for our results. First, it could lead to a false conclusion regarding the degree of support in the literature for an association between air pollution and a health outcome. Second, it could cause the size of a true association to be estimated inaccurately.

Time-series studies of routine health data may be particularly subject to publication bias. One reason is that the data are relatively cheap to obtain and analyze, so that authors (having invested relatively little effort to conduct the analysis) may be less inclined to publish findings thought to be uninteresting or to contradict the prevailing scientific consensus. Another reason is that the analytic techniques used in time-series studies affect the results to some extent; analysts may choose the techniques that give the results that they think are most plausible. Finally, as discussed above, time-series studies have the potential to generate a large number of results for various outcomes, pollutants, and lag times; the author's preexisting beliefs may affect the choice of which results to present. Publication bias may be mitigated in planned multi-city studies (such as APHEA and NMMAPS) that commit to publishing results from all locations and that adopt an a priori lag-time specification.

Until recently, little attention has been paid to this possible source of bias in the air pollution literature (Anderson et al. 2005). One method for investigating the extent of publication bias is the funnel plot, in which estimates are plotted against their SEs. If the data lack publication bias, they will be scattered symmetrically in a funnel shape (Light and Pillemer 1984). Apparent asymmetry in the funnel plot, initial evidence of publication bias, can be verified by performing one of two types of regression analysis on the data against the inverse of the SE: an adjusted-rank-correlation method, called the Begg test for asymmetry (with continuity correction) (Begg 1997) and a regression approach called the Egger test for publication bias (Egger et al. 1997). Both approaches were used for each meta-analysis; results are reported as P values indicating the strength of the evidence for asymmetry in the funnel plot.

RESULTS

Study Characteristics

A total of 115 time-series studies indexed in medical databases (up to August 2007) were identified by the PAPA–SAN and APED search strategies. The 115 studies were divided into those that yielded usable numerical estimates of the effects of air pollution on health and those whose content was closely related to this topic but not appropriate for a quantitative systematic review. The published article for one study could not be found. Of the 114 studies for which a copy of the published article was found, 82 were deemed to be suitable for quantitative meta-analysis. The details of these studies are given in Appendix Table A.1. Of the 32 studies not included, 17 did not meet the inclusion criteria, six did not provide numerical estimates, and nine focused on the health effects of dust storms. (Studies of the health effects of dust storms were not considered, because they are not investigations of day-to-day effects of routine exposure to air pollution but rather focus on the health effects of unusual pollution episodes.) The details of the 32 excluded studies are given in Appendix Table A.2.

Two time-series study designs were applied to the data obtained from the 82 time-series studies identified, for purposes of quantitative analysis: conventional ecologic studies based on numbers of health events, and case–crossover studies. Both study designs provide regression coefficients suitable for further quantitative analysis and both use data from all days within the study period — that is, they do not focus on isolated pollution episodes.

Geographic Distribution of the Studies

The majority of the 82 time-series studies selected for quantitative analysis were from China (46 in all: 16 in Taipei,China, 10 in Hong Kong, 8 in Shanghai, 7 in Beijing, 2 in Wuhan, and 1 each in Benxi, Chongqing, and Shenyang). The vast majority of the 28 studies of South Korean cities were of Seoul. The other studies were conducted in Japan (3), Thailand (2), India (1), Singapore (1), and Malaysia (1).

Health Outcomes

A range of health outcomes were studied. We categorized them as cause-specific mortality and cause-specific hospital admissions (Appendix Table A.3), as well as cause-specific community care. Among the studies of mortality, the most common outcome was mortality (38 studies) from all natural causes of death, followed by mortality from cardiovascular disease (24) and all respiratory diseases (20). A smaller group of mortality studies examined more specific causes of cardiovascular and respiratory mortality, such as cerebrovascular events or COPD. Studies of hospital admissions (including emergency room admissions but not visits) covered a range of respiratory and cardiovascular diagnoses, the most common single diagnosis being asthma (13 studies), followed by cerebrovascular events (6), and IHD (5). Studies of community care were fewer; outcomes in those studies included emergency transport, emergency room visit, primary care consultation, and absence from school.

Mortality studies tended to include either people of all ages or people 65 years of age or older, the group in which most deaths occur. Studies also reported results for other age groups (Appendix Table A.4). Furthermore, in some Asian countries and cities, infant mortality is substantial. In some of these locations, researchers have been able to study the association between air pollution and mortality in infants and children. In many studies, hospital admissions tend to be subdivided by age to reflect the relative importance of admissions in younger age groups and the fact that some conditions (such as asthma) differ in their clinical presentation between young and older patients.

Pollutants

Most pollutants studied in North America and Europe were represented in the 82 selected studies, with the exception of black smoke (which is measured using reflectance and is measured almost exclusively in Europe). Other pollutants studied were SO_2 (63 studies), O_3 (52), NO_2 (48), PM_{10} (56), $PM_{2.5}$ (9), TSP (23), and CO (32). For O_3, 24-hour, maximum 8-hour, and maximum 1-hour periods were used as averaging times and running means were calculated.

Effect Estimates

Effect estimates for the main pollutant–outcome pairs for subjects of all ages from the time-series studies are summarized in "forest plots" (Figures 40 to 60 and Appendix Figures A.1 to A.30). The estimates are presented as the percent change in the mean number of daily deaths or hospital admissions per 10-μg/m^3 increase in mean pollutant concentration (except for CO, for which the unit increase studied was 1 mg/m^3) and the associated 95% CI, which indicates the precision of the estimate. For each pollutant, estimates were plotted separately for daily numbers of deaths from all natural causes, deaths from respiratory causes, deaths from cardiovascular causes, hospital admissions for respiratory causes, and hospital admissions for cardiovascular causes. Within these broad groups, all diagnoses for which estimates were available are plotted. Each estimate is labeled with the publication citation, and study location, and where applicable, cause of death or admission, age group, and averaging time (for O_3).

PM_{10} Fifteen estimates for the association between PM_{10} and all-natural-cause mortality at all ages were reported from eight cities (Figure 40). The majority of estimates were positive, indicating increases (of < 1%) in mean numbers of deaths per day per 10-μg/m^3 increase in PM_{10}. Twelve of the fifteen estimates had lower confidence limits that were greater than zero. Only one estimate was negative (i.e., suggesting that an increase in the pollution concentration results in a decrease in the mortality rate). All-natural-cause mortality was also studied in a range of age groups (Appendix Figure A.1a), including four studies of mortality in children and infants (Appendix Figure A.1b).

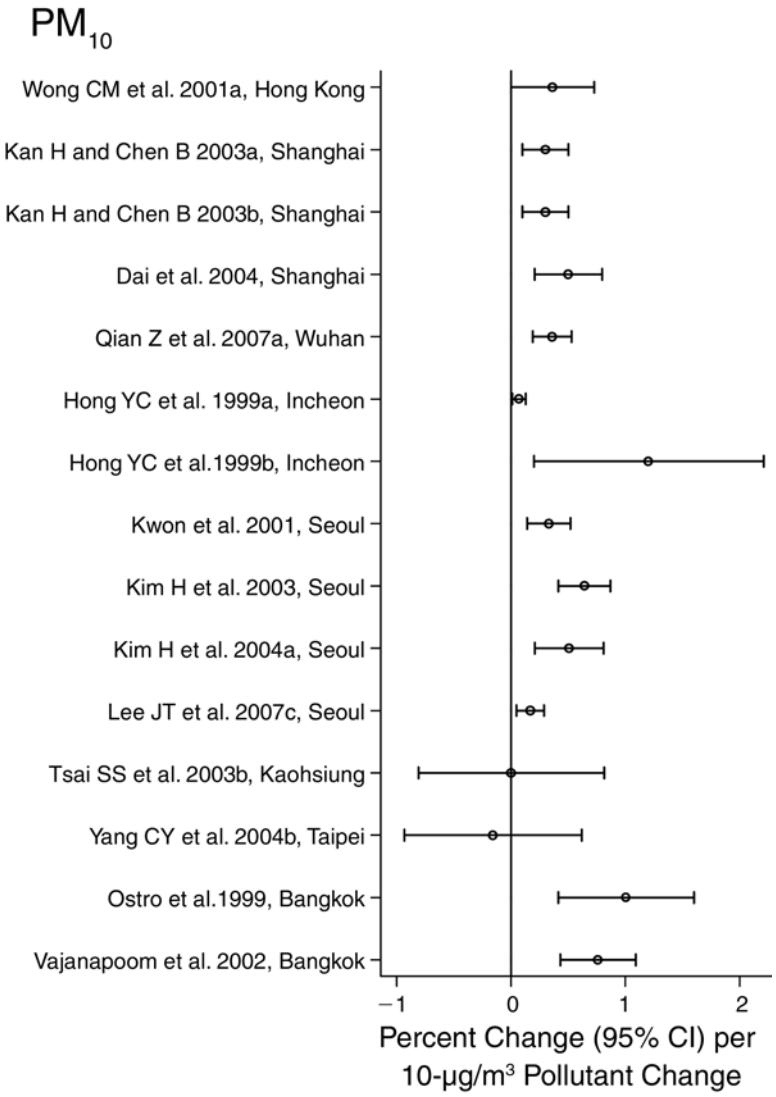

Figure 40. Percent change in the mean number of daily deaths from all natural causes per 10-μg/m^3 change in 24-hr mean PM_{10} concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation and study location.

Sixteen estimates of the association between PM_{10} and deaths from respiratory diseases were available from eight cities (Figure 41). The majority of studies examined deaths from all respiratory diseases, with only a few assessing associations for specific respiratory conditions such as asthma or COPD. The effect estimates tended to be larger than those observed for all-natural-cause mortality (ranging from increases of 0% to 3% per 10-μg/m^3 increases in PM_{10}) and all but one were positive. Regarding age-specific respiratory mortality, estimates from 25 reports were found (Appendix Figure A.2).

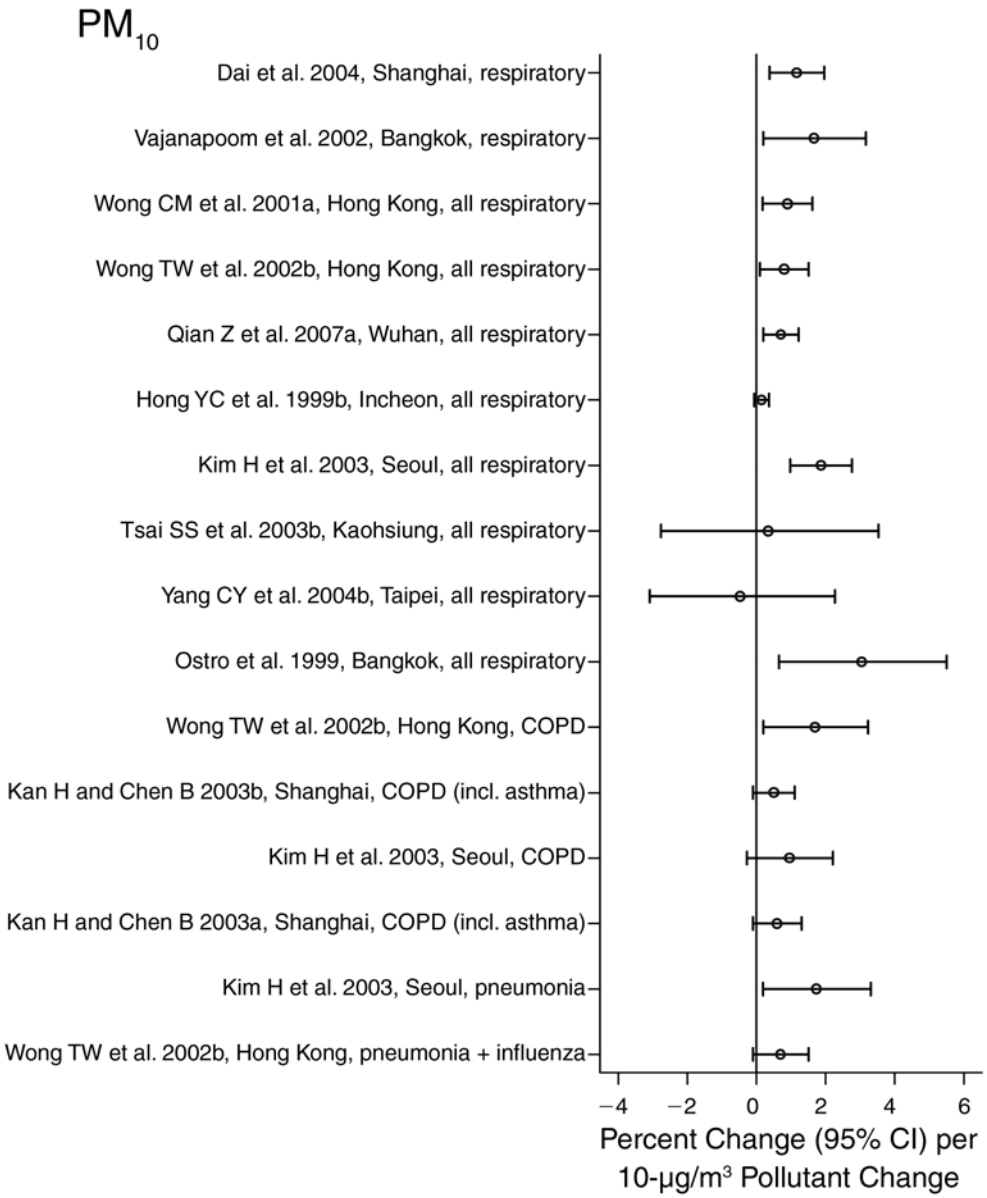

Figure 41. Percent change in the mean number of daily deaths from respiratory causes per 10-μg/m^3 change in 24-hr mean PM_{10} concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of death.

Analyses of all mortality from cardiovascular disease yielded 23 PM_{10} effect estimates from eight cities (Figure 42) for a range of cardiovascular endpoints. A further 30 PM_{10} effect estimates from 16 cities were reported for specific age groups for a range of cardiovascular diseases including cardiac disease, stroke, and pulmonary heart disease (Appendix Figure A.3). For mortality from cardiovascular disease at any age, 13 reports were found; all but two of these estimates were positive.

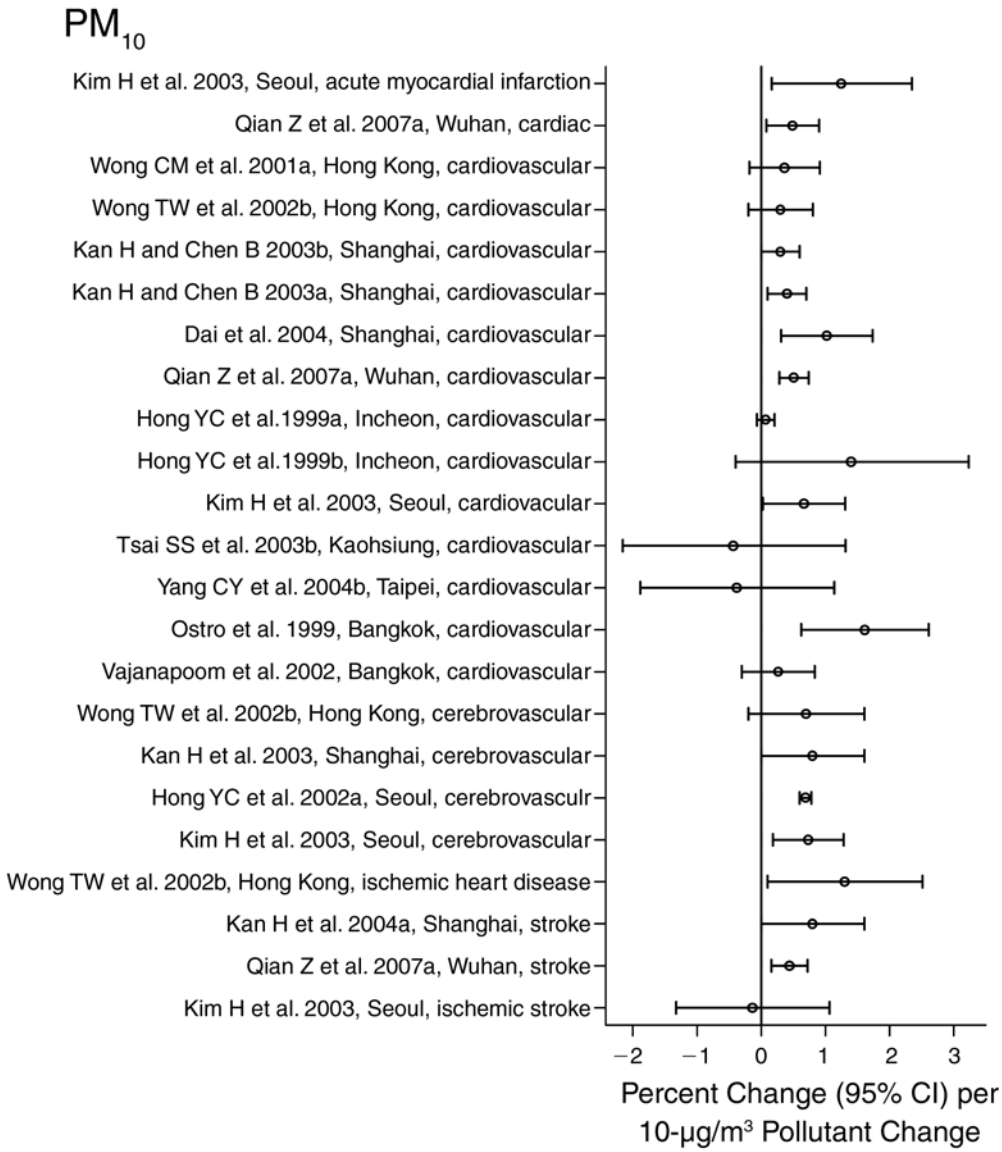

Figure 42. Percent change in the mean number of daily deaths from cardiovascular causes per 10-µg/m^3 change in 24-hr mean PM_{10} concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of death.

Also shown is the substantial body of evidence for positive associations between PM_{10} and hospital admissions for respiratory disease (Figure 43 and Appendix Figure A.4) and from cardiovascular disease (Figure 44 and Appendix Figure A.5). These results are presented for all ages and also for specific age groups.

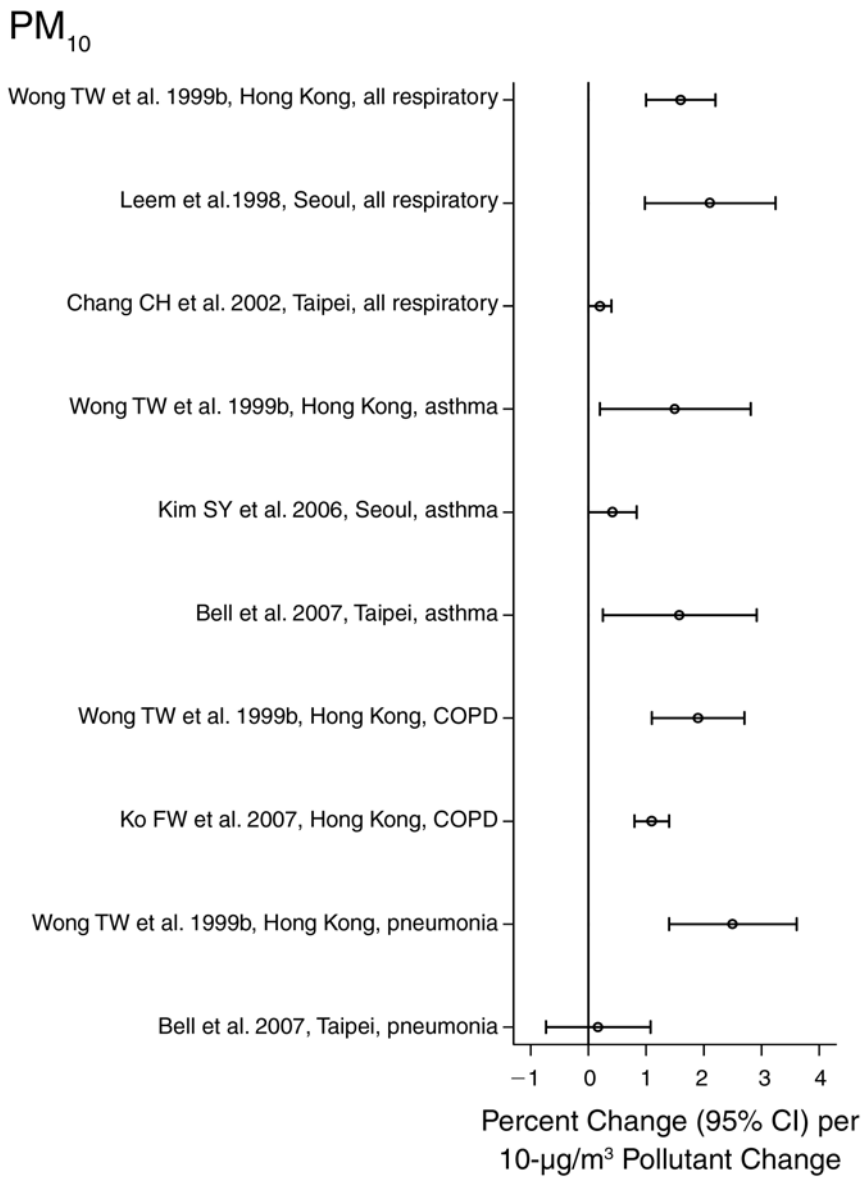

Figure 43. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-μg/m³ change in 24-hr mean PM_{10} concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of admission.

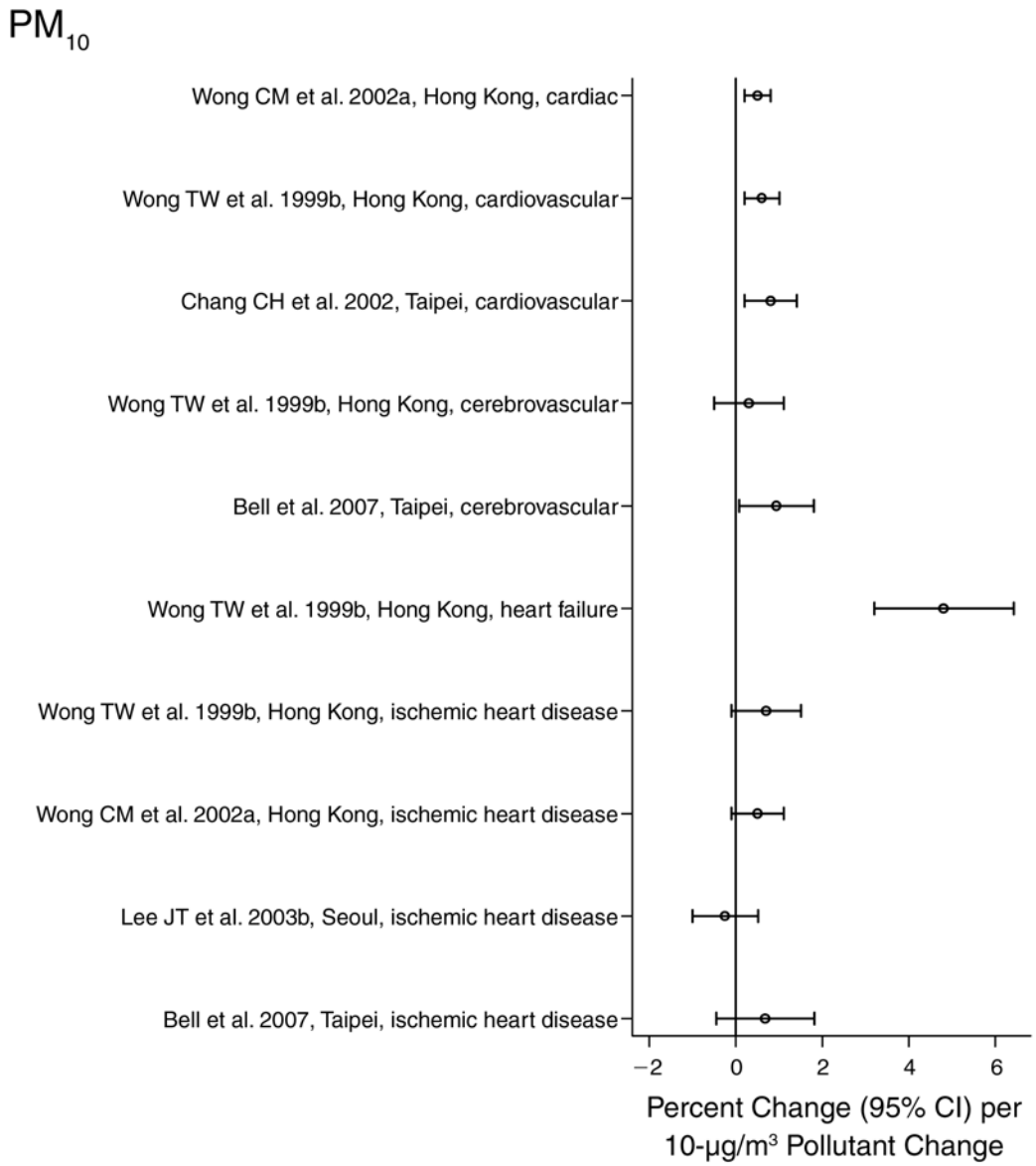

Figure 44. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 10-μg/m^3 change in 24-hr mean PM_{10} concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of admission.

$PM_{2.5}$ Studies of $PM_{2.5}$ analyzed all-natural-cause and cause-specific mortality as well as hospital admissions for cerebrovascular, ischemic, and hemorrhagic stroke, IHD, asthma, pneumonia, and diabetes. All estimates are shown in Figure 45. The majority of estimates were positive; however, negative associations between hospital admissions and $PM_{2.5}$ were observed by Bell and colleagues (2007) and Chan and associates (2006).

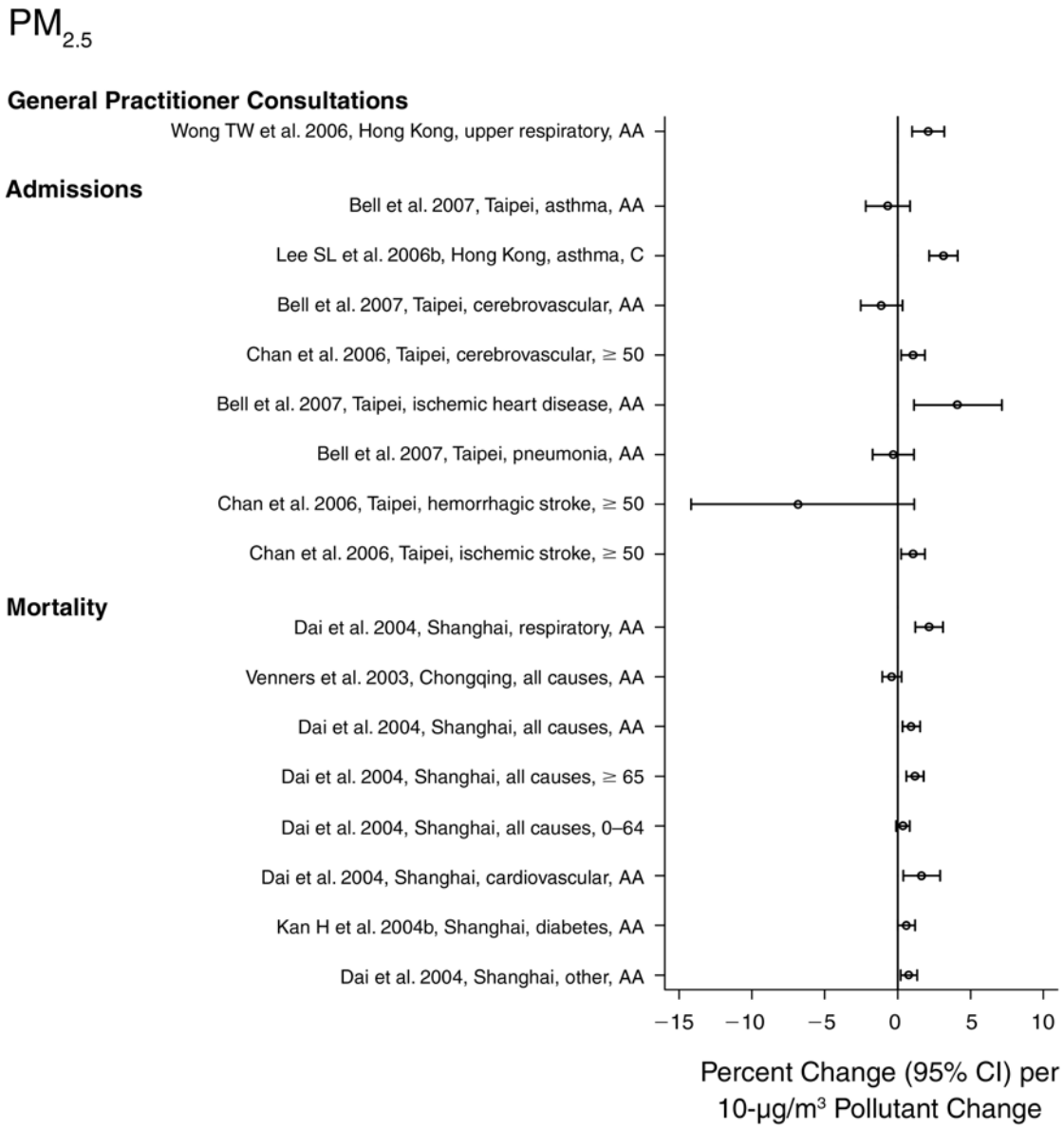

Figure 45. Percent change in the mean number of daily general practitioner consultations or emergency hospital admissions for, or deaths from, respiratory or cardiovascular causes per 10-μg/m^3 change in 24-hour mean $PM_{2.5}$ concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death or admission, and age group: All Ages (AA), Children (C), or years of age. "Other" cause of death denotes death from other respiratory or cardiovascular causes.

TSP We identified 30 estimates for the association of TSP and all-natural-cause mortality, 17 for all ages from 10 cities (Appendix Figure A.6), and 13 for specific age groups (Appendix Figure A.7). For all-age, all-natural-cause mortality, sixteen of these estimates were positive, with the majority reflecting increases of less than 0.5% per 10-μg/m^3 increase in TSP. Regarding cause-specific mortality, a smaller number of studies (eight in total) reported results. These covered both cardiovascular and respiratory mortality across all age groups (Appendix Figures A.8 and A.9).

Only 2 studies (Cho et al. 2000; Im et al. 2000) analyzed TSP and hospital admissions (Appendix Figure A.10). No estimates of the effect of TSP on cardiovascular admissions were found.

NO_2 All studies of NO_2 and mortality used a 24-hour daily averaging time. We found 11 estimates of the effect on all-natural-cause mortality, from seven different cities for all ages. These results are shown in Figure 46: all were positive and represented increases in the number of deaths of less

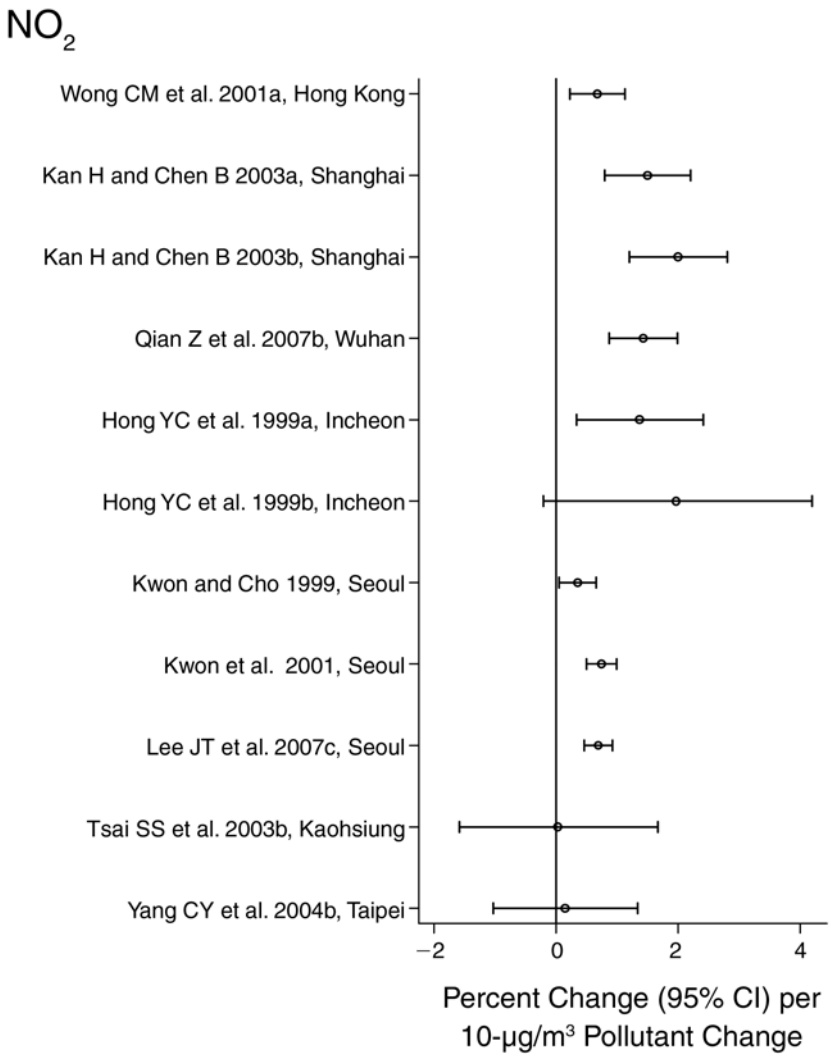

Figure 46. Percent change in the mean number of daily deaths from all natural causes per 10-μg/m^3 change in 24-hr mean NO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation and study location.

than 2% per 10-µg/m^3 increase in NO_2. A number of different age ranges were also studied (Appendix Figure A.11).

For death from any respiratory disease, 7 studies examined associations with NO_2 in 6 different locations (Figure 47) and 2 studies examined age- and disease-specific associations yielding 5 estimates (Appendix Figure A.12). Most of these associations were positive and typically indicate an increase in the number of deaths of less than 5% per 10-µg/m^3 increase in NO_2.

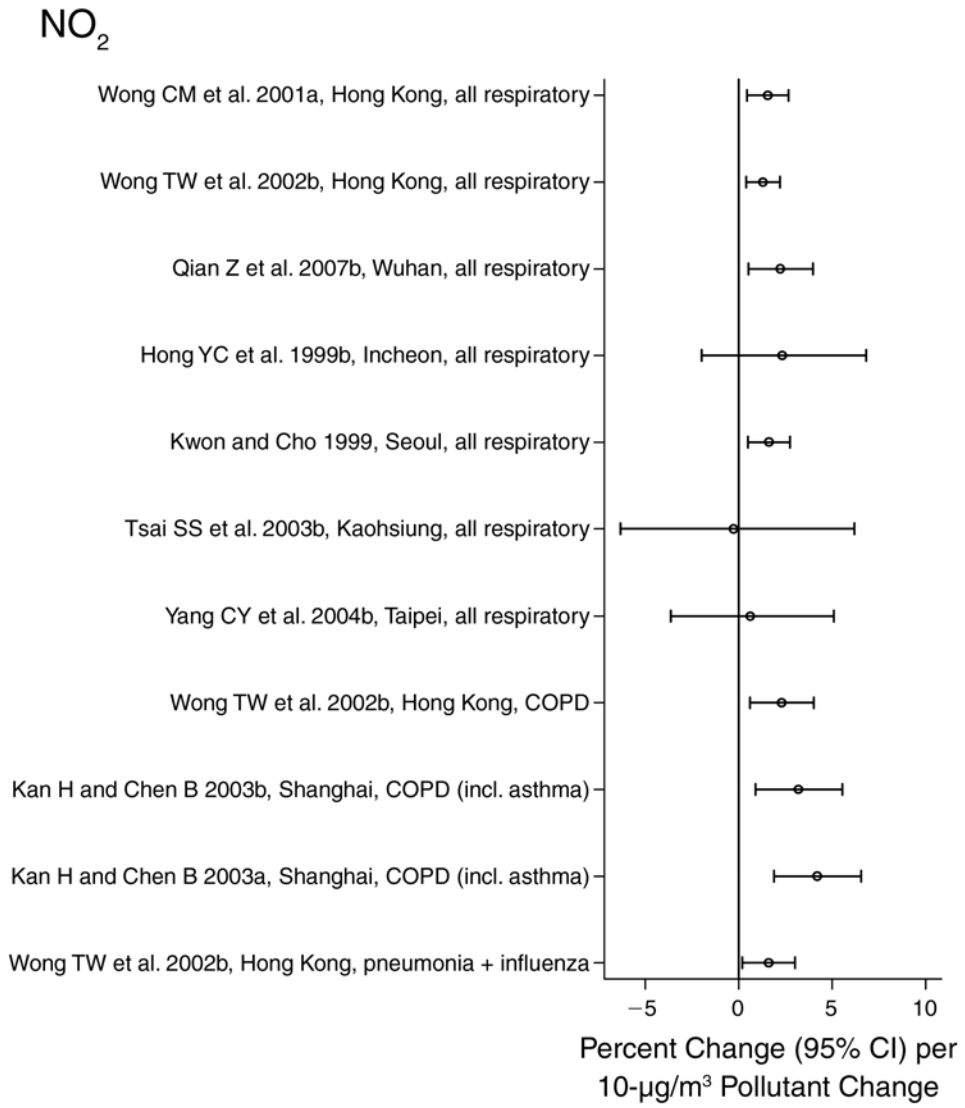

Figure 47. Percent change in the mean number of daily deaths from respiratory causes per 10-µg/m^3 change in 24-hr mean NO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of death.

Studies of the associations between NO_2 and cardiovascular deaths were more common: 18 estimates were found for all age groups (Figure 48), as were 6 age-specific estimates (Appendix Figure A.13).

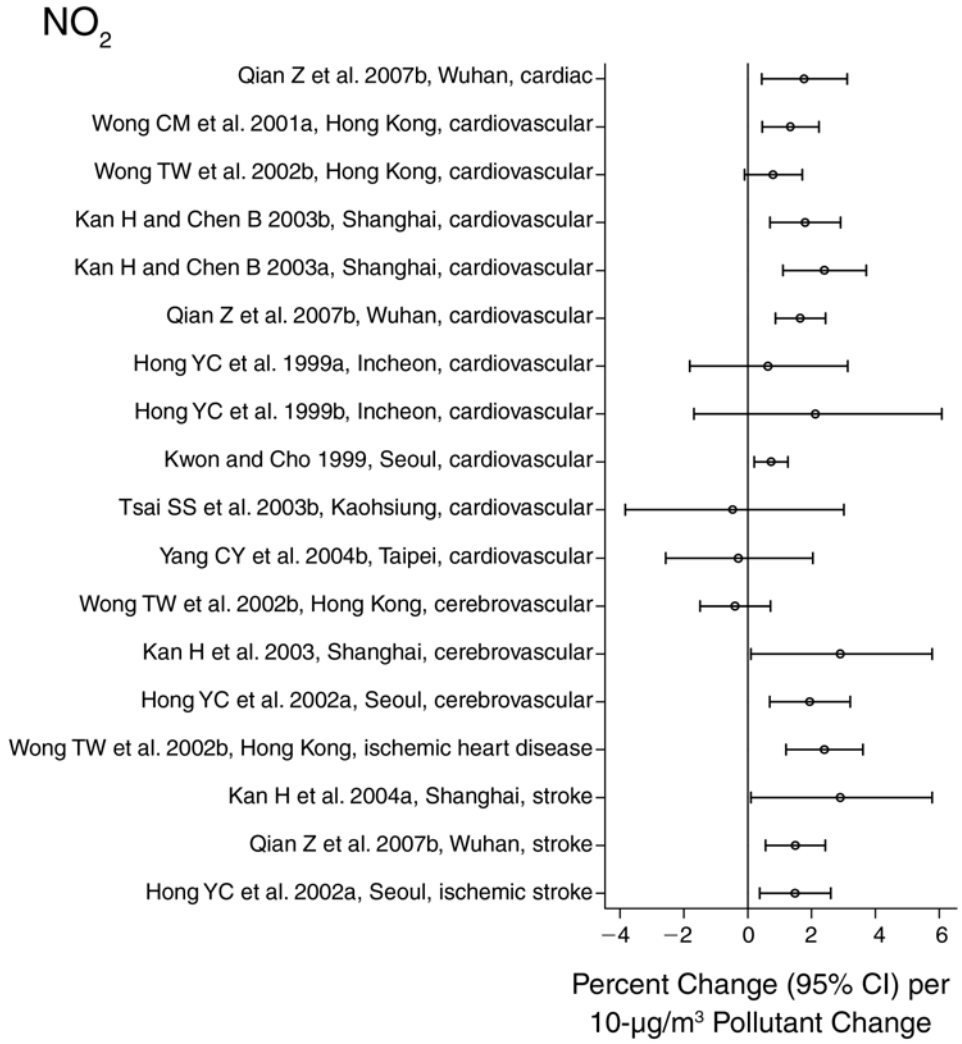

Figure 48. Percent change in the mean number of daily deaths from cardiovascular causes per 10-µg/m^3 change in 24-hr mean NO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of death.

Only 1 study (Chang CH et al. 2002) reported associations for the maximum1-hour-average NO_2 concentration and hospital admissions, all others choosing to focus on 24-hour averages. Since Chang and colleagues also reported estimates for 24-hour average NO_2 concentrations, we focused our meta-analyses on the 24-hour averages. We found 12 estimates of the association between NO_2 and admission to a hospital for respiratory disease, across all ages (Figure 49) — 5 for any respiratory disease, 3 for asthma, 2 for COPD, and 2 for pneumonia. Most of these associations were positive and represented an increase in the number of admissions of up to 3% per 10-µg/m^3 increase in NO_2. A number of combinations of diseases and age groups were also analyzed (Appendix Figure A.14). Two estimates were negative, the rest positive. For cardiovascular admissions related to NO_2, a number of studies reported positive associations with cardiovascular diseases (Figure 50), including IHD (4 estimates), heart failure (1), and cardiac disease (1). Associations for admissions in adults are shown in Appendix Figure A.15.

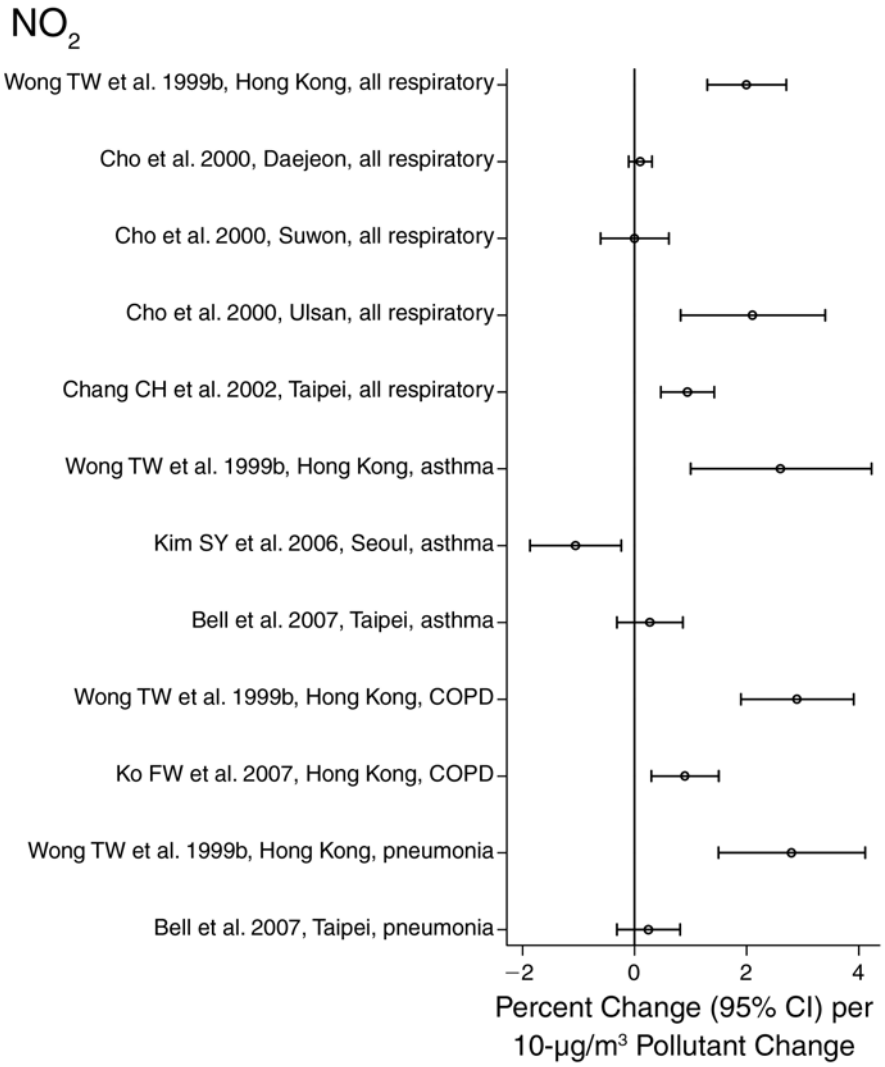

Figure 49. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-µg/m^3 change in 24-hr mean NO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of admission.

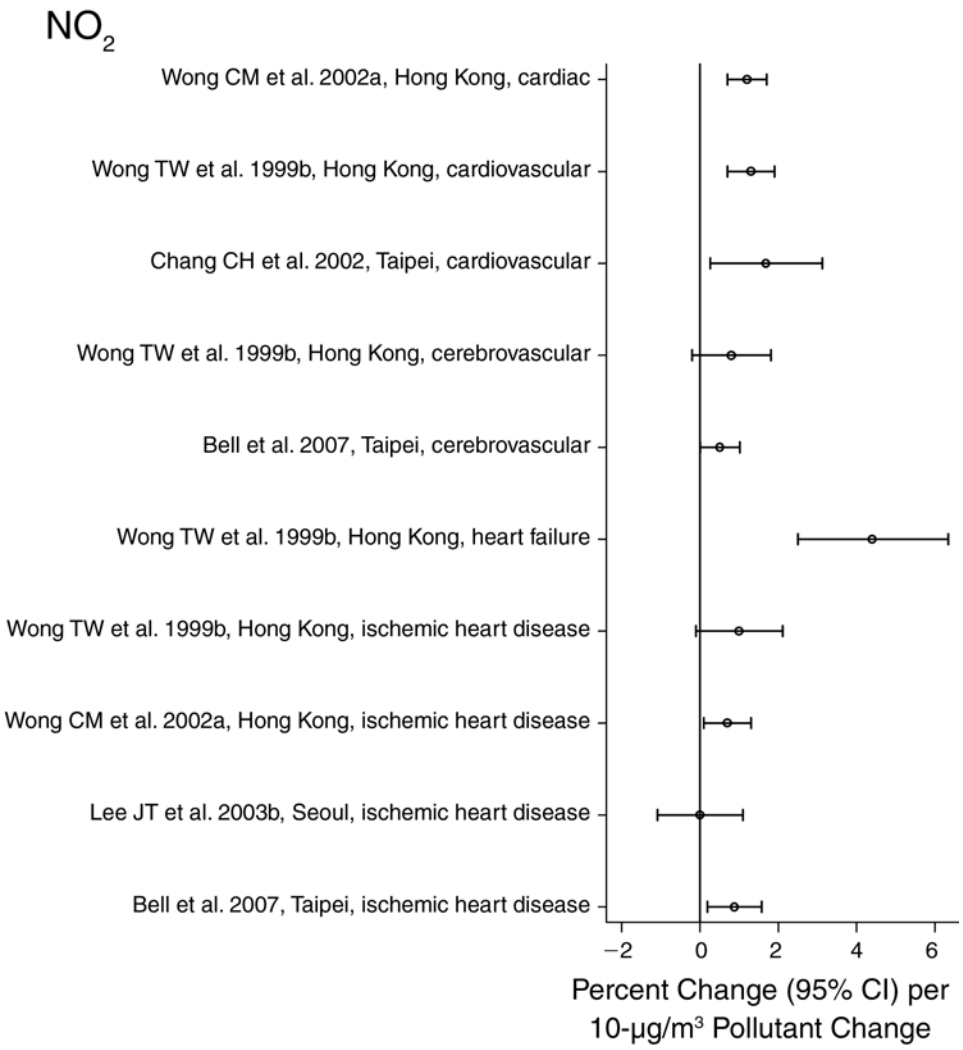

Figure 50. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 10-μg/m^3 change in 24-hr mean NO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of admission.

O_3 A total of 52 studies reported results for O_3, with 12 reporting results for the maximum 1-hour averages, 28 for 24-hour averages, and 14 for the 8-hour averages. These metrics are usually correlated temporally, but we have not attempted to make adjustments in order to combine results for the different metrics.

Figure 51 shows the results for all-natural-cause mortality: five used maximum 1-hour estimates, five 24-hour estimates, and four 8-hour estimates. Four estimates were negatively associated with daily numbers of deaths, with the rest positive, and all but one estimated increase was less than 1% for a 10-μg/m^3 increase in O_3. Age-specific associations were dominated by estimates based on maximum 8-hour estimates (Appendix Figure A.16).

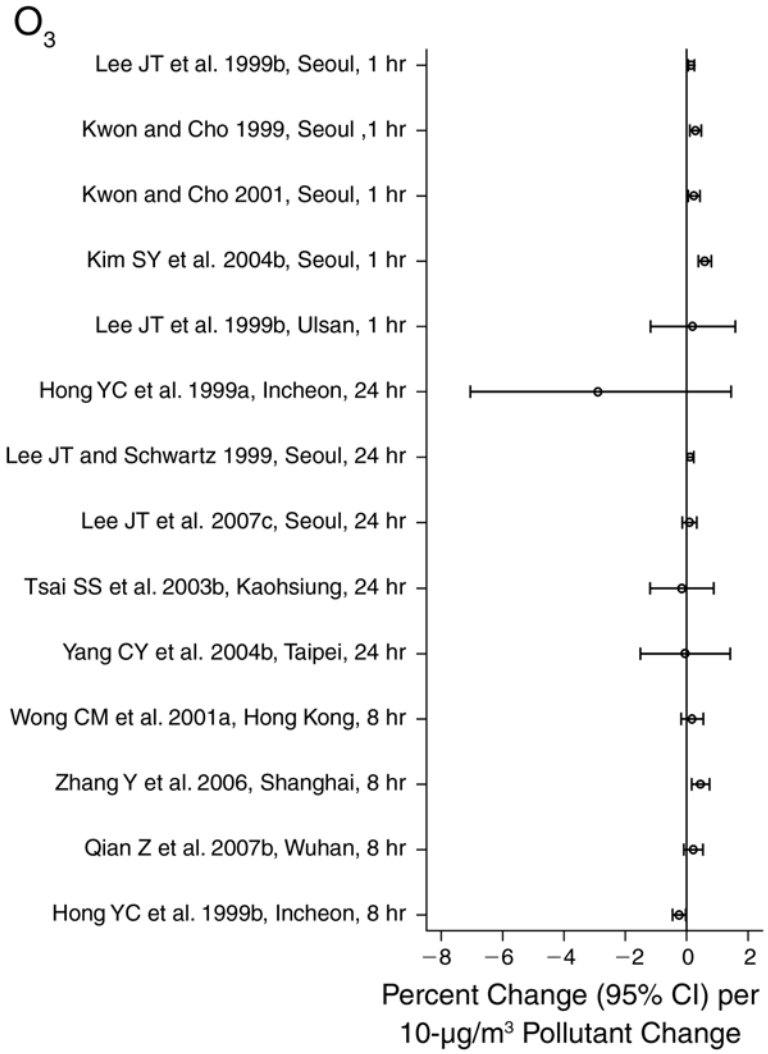

Figure 51. Percent change in the mean number of daily deaths from all natural causes per 10-μg/m^3 change in mean O_3 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and O_3 averaging time.

For cause-specific mortality, the majority of results reported were for respiratory or cardiovascular mortality at all ages, and these were dominated by 8-hour results (Figures 52 and 53 and Appendix Figures A.17 and A.18). In both disease groups, the majority of estimates were positive. Despite the variation in averaging times, there were sufficient estimates (i.e., four or more) for summary estimates to be calculated for respiratory and cardiovascular mortality.

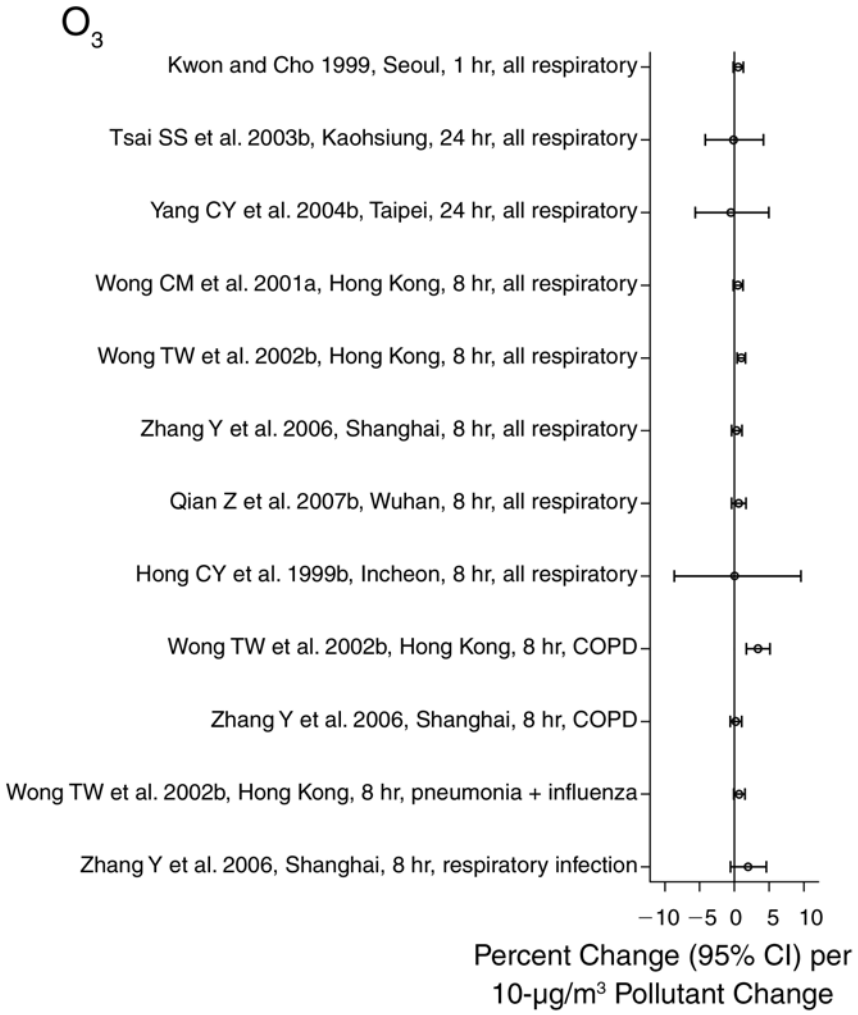

Figure 52. Percent change in the mean number of daily deaths from respiratory causes per 10-µg/m^3 change in mean O_3 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, and cause of death.

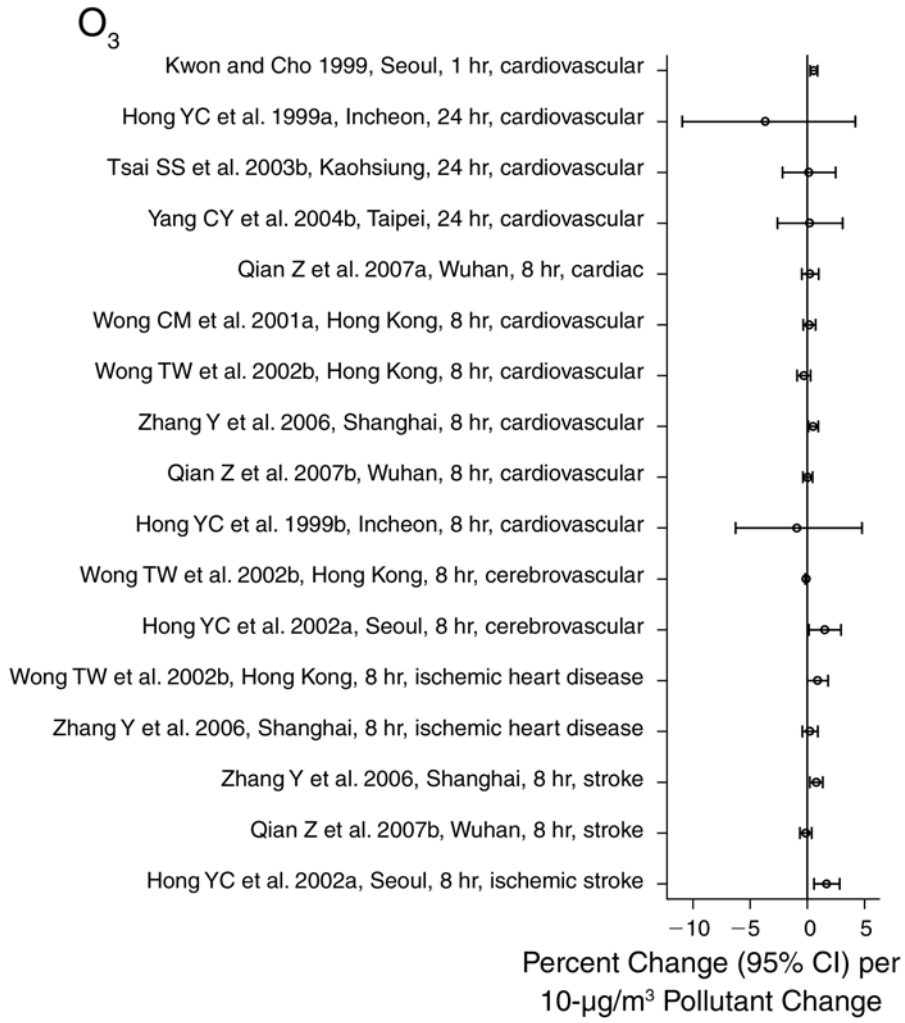

Figure 53. Percent change in the mean number of daily deaths from cardiovascular causes per 10-µg/m^3 change in mean O_3 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, and cause of death.

A large number of studies have reported associations between measures of O_3 and respiratory and cardiovascular admissions, according to specific diseases and ages (Figures 54 and 55, respectively, and Appendix Figures A.19 and A.20, respectively). We found 13 estimates of the association between respiratory admissions and O_3 and 17 estimates for specific age groups. All associations — irrespective of averaging time, age, or respiratory disease group — were positive. A similar number of reports were found for cardiovascular admissions related to O_3, with similarly positive associations overall, albeit with a small number of suggested negative associations.

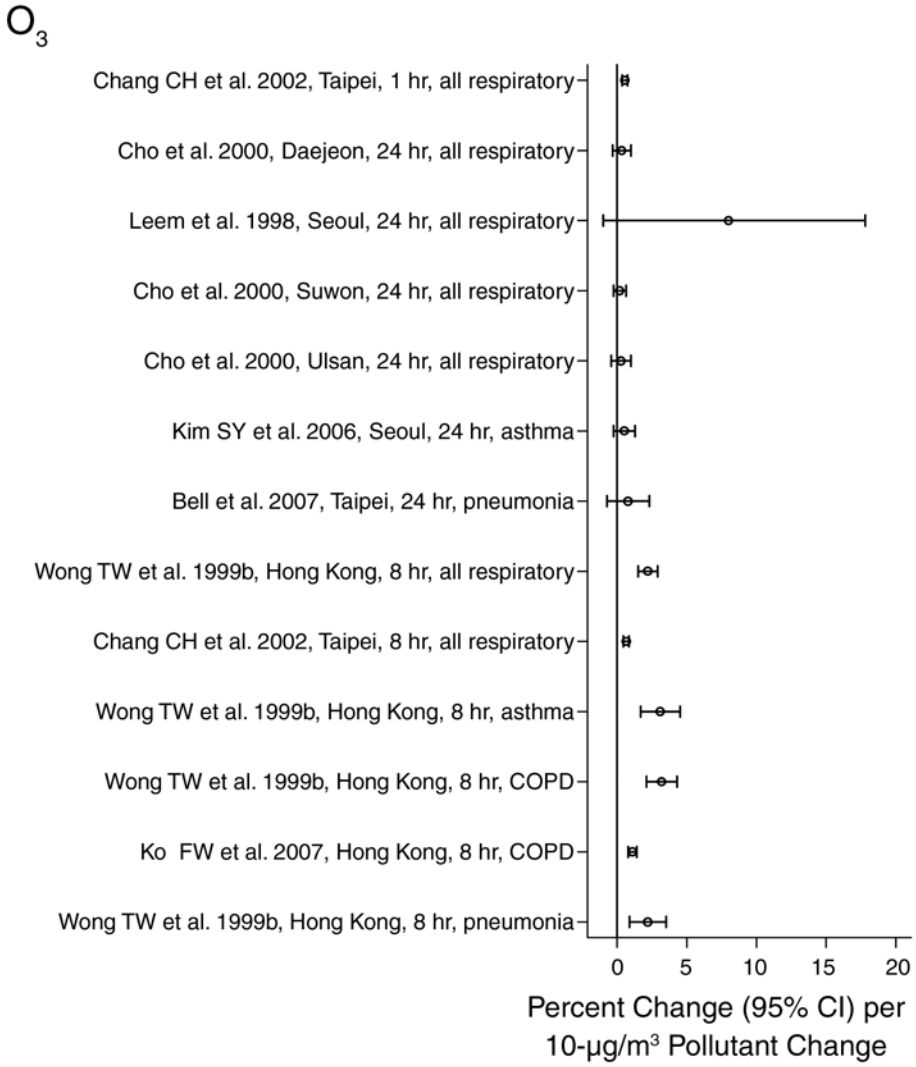

Figure 54. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-µg/m^3 change in mean O_3 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, and cause of admission.

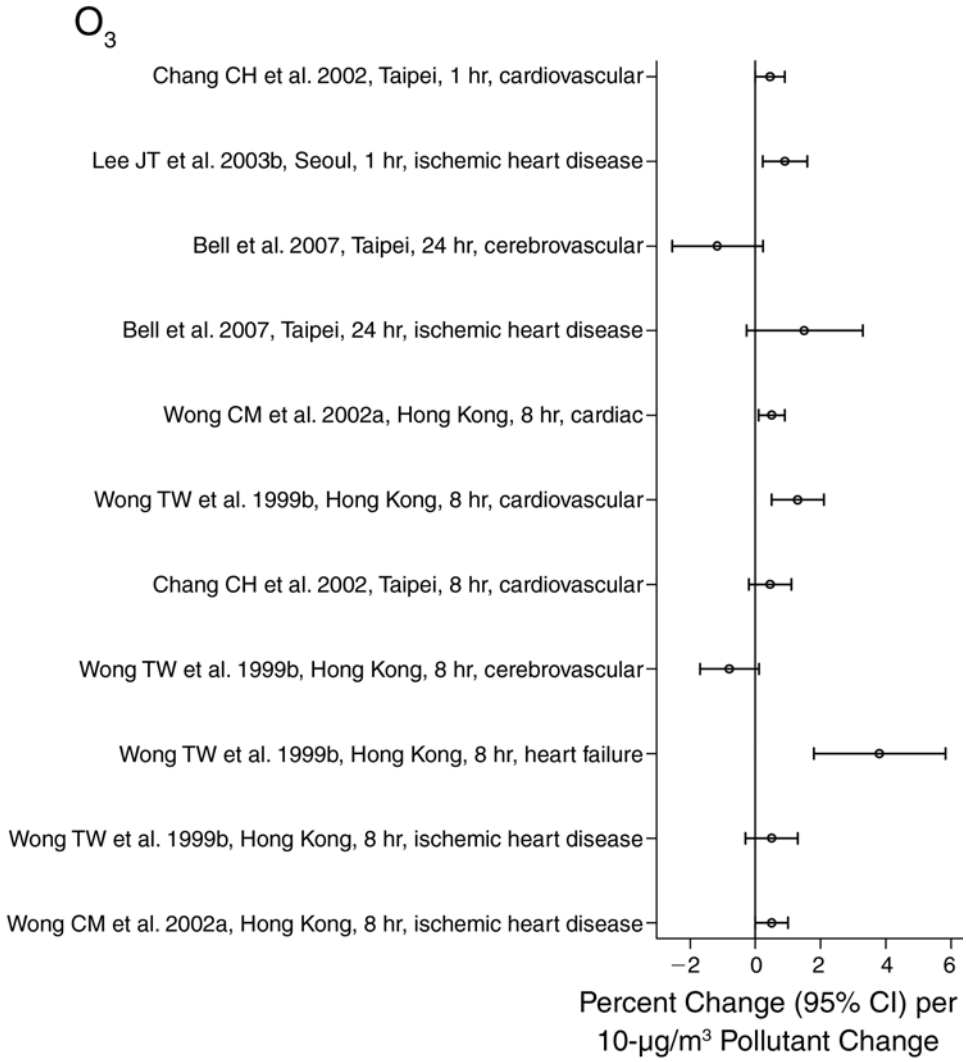

Figure 55. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 10-µg/m^3 change in mean O_3 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, and cause of admission.

SO_2 Numerous studies have reported associations between SO_2 and daily mortality. Twenty-five estimates for all-natural-cause mortality at all ages were found (Figure 56). Nearly all of these data, collected from studies in 15 different cities, show positive associations between increases in SO_2 and increases in daily numbers of deaths from all natural causes. Associations were also reported for age-specific mortality (Appendix Figure A.21).

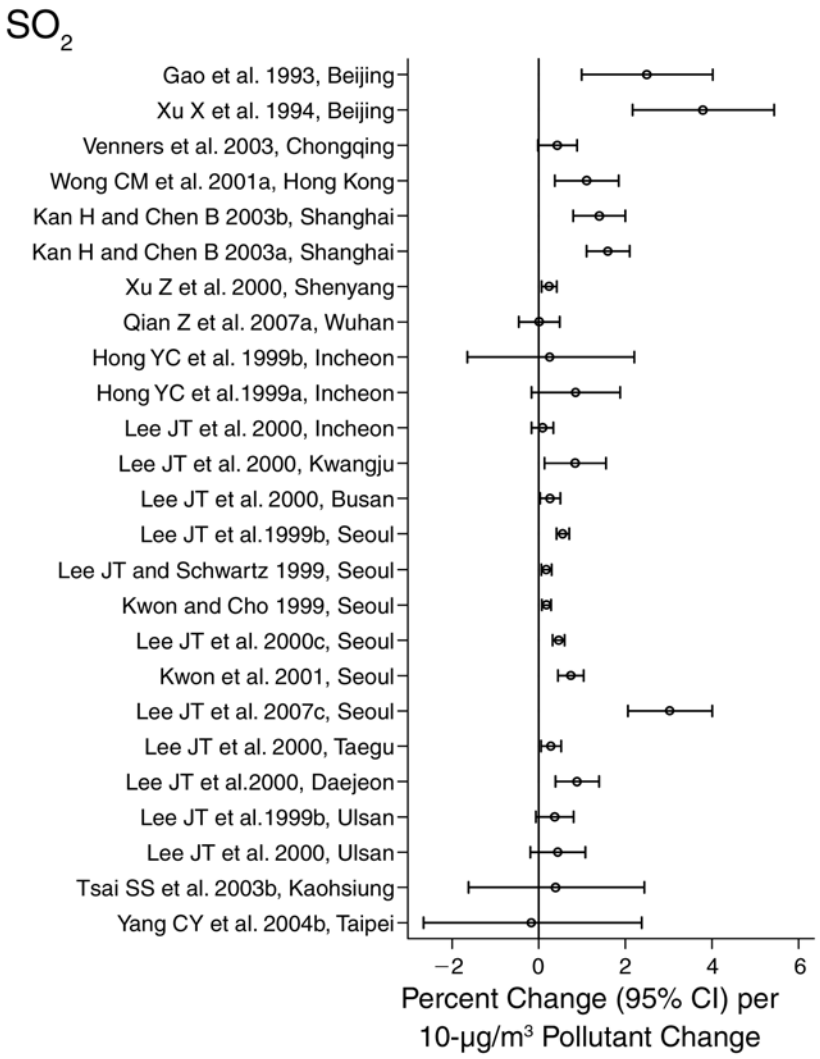

Figure 56. Percent change in the mean number of daily deaths from all natural causes per 10-µg/m^3 change in 24-hr mean SO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation and study location.

Numerous studies reported associations between SO_2 and respiratory mortality for all ages (Figure 57) and also specific age groups (Appendix Figure A.22). The estimates for all ages were positive, with one exception. The estimates for mortality from all respiratory diseases tended to be smaller than the estimates for specific conditions.

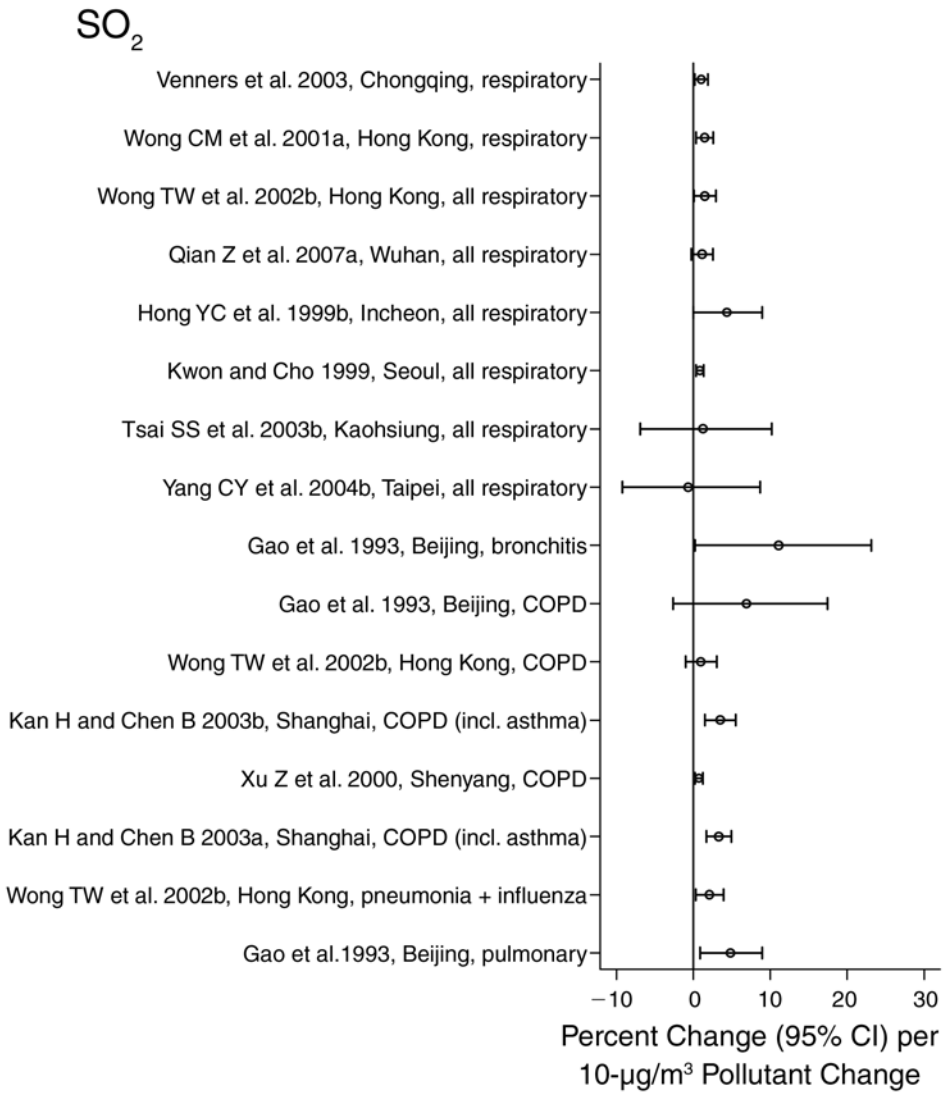

Figure 57. Percent change in the mean number of daily deaths from respiratory causes per 10-µg/m^3 change in 24-hr mean SO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of death.

A range of cardiovascular outcomes was studied in relation to SO_2. These included mortality from all cardiovascular diseases or from specific disease subgroups such as cardiac or cerebrovascular disease, IHD, or stroke. The majority of reports were for all ages (Figure 58). Most of these associations were positive, with 10-μg/m^3 increments in SO_2 associated with increases in mean daily numbers of deaths of between 0 and 3%. Fewer reports were found for specific age groups (Appendix Figure A.23).

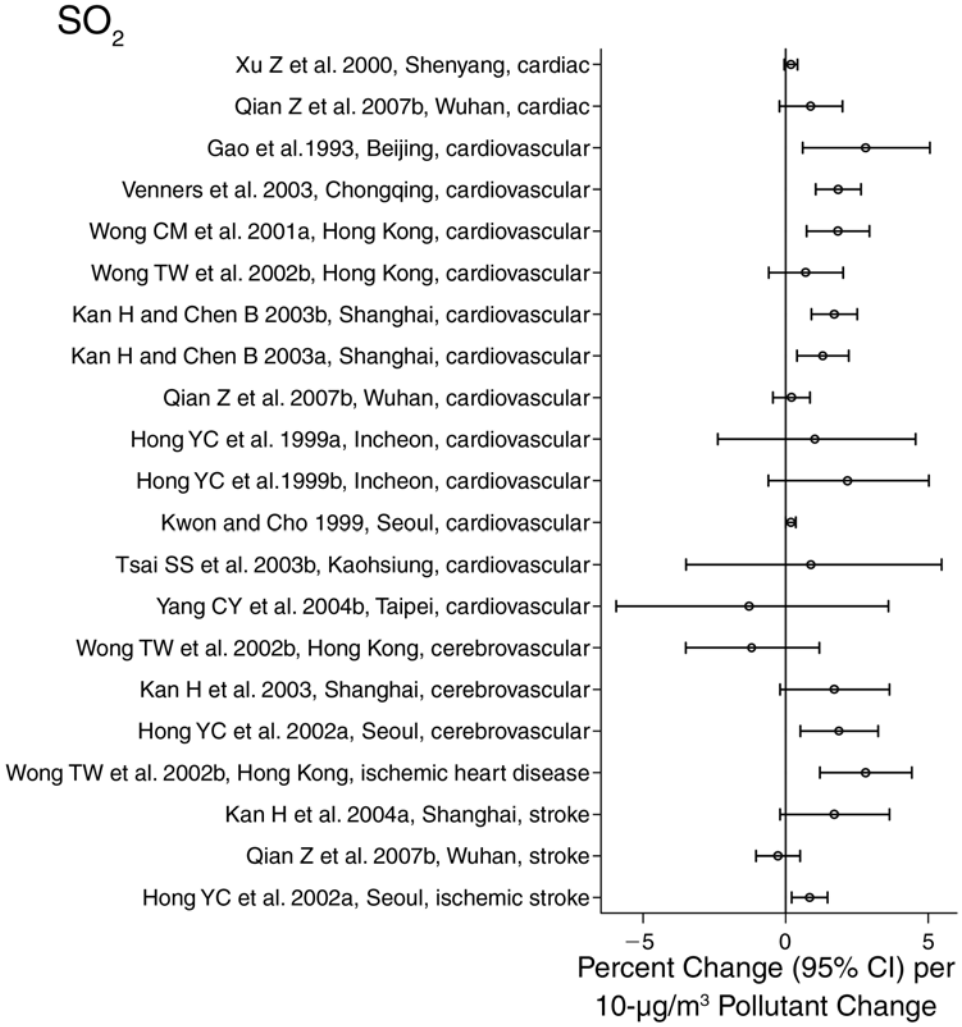

Figure 58. Percent change in the mean number of daily deaths from cardiovascular causes per 10-μg/m^3 change in 24-hr mean SO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of death.

Admissions to the hospital for respiratory conditions have been studied by a number of investigators. We found seven articles reporting results for a range of respiratory outcomes, including all respiratory conditions, COPD, pneumonia, and asthma. Effect estimates from these studies are given in Figure 59; most indicate positive associations with SO_2. Results for hospital admissions in specific age categories tended to be positive and more variable in size than the estimates for all age groups combined, as illustrated in Appendix Figure A.24.

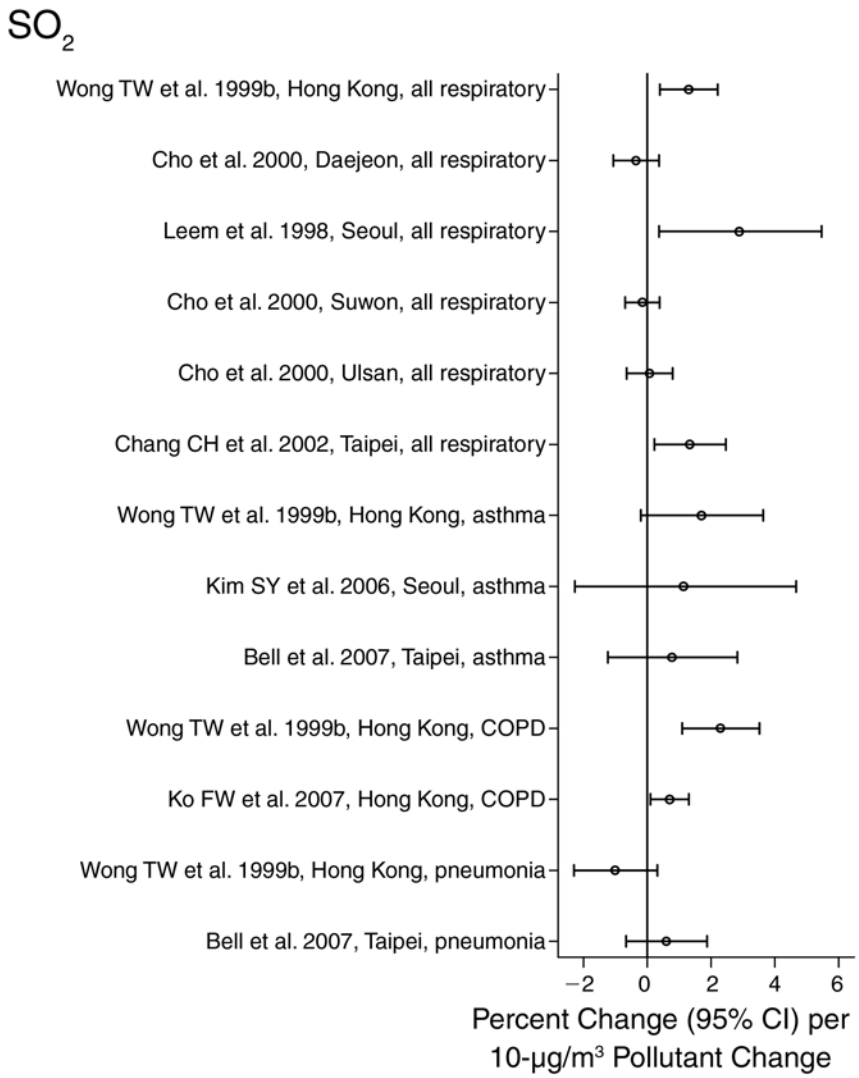

Figure 59. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-µg/m^3 change in 24-hr mean SO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of death.

A smaller number of studies examined associations between SO_2 and admissions for cardiovascular diseases: 10 estimates for admissions in all age groups are shown in Figure 60, as are 9 estimates for specific age groups in Appendix Figure A.25. A small number of these associations were negative, with upper confidence limits just greater than 0. Most estimates were positive, however, representing increases in the number of admissions of between 0 and 5%.

CO Estimates from studies of CO are illustrated in Appendix A. We found eight studies reporting associations between CO and all-natural-cause mortality, either across all age groups or for specific age groups (Appendix Figure A.26). One of the 11 estimates was negative and two, for children under 1 year of age, were imprecisely estimated; the remaining 8 estimates were positive, indicating increases in the number of deaths of between 0% and 5% per 1-mg/m^3 increase in CO. Fewer estimates were available for cause-specific mortality associated with CO, from five reports of respiratory mortality (Appendix Figure A.27) and five reports of cardiovascular mortality (Appendix Figure A.28). These effect estimates suggest generally

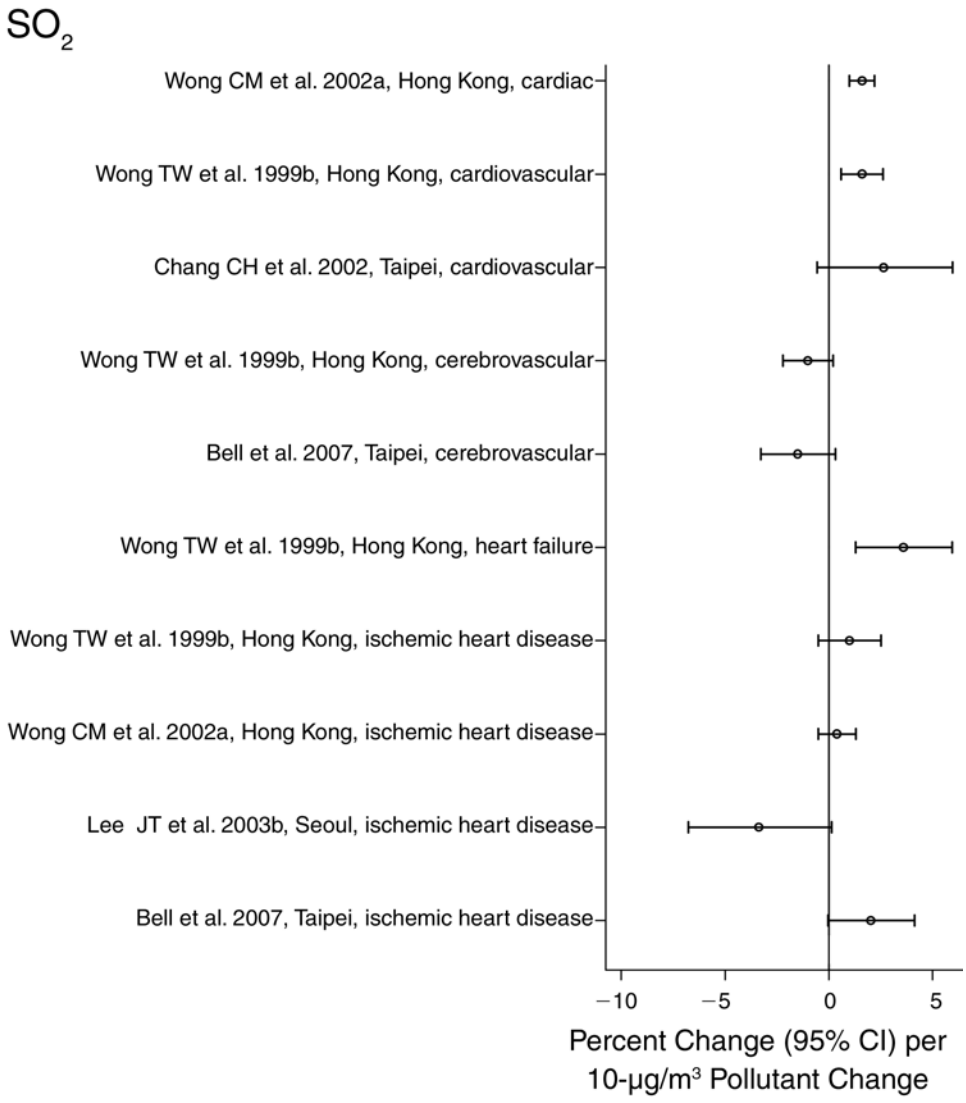

Figure 60. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 10-µg/m^3 change in 24-hr mean SO_2 concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, and cause of admission.

positive associations between increases in CO concentrations and respiratory mortality and more equivocal results for cardiovascular mortality.

Hospital admissions have been studied in relation to CO, with 5 studies reporting effect estimates for respiratory admissions (Appendix Figure A.29) and three for cardiovascular admissions (Appendix Figure A.30).

Table 14. Summary Effect Estimates of the Percent Change in RR of Various Mortality and Admissions Outcomes in Various Age Groups Due to a Change in Pollutant Concentration, by Pollutant and Outcome[a]

Pollutant and Outcome	Percent Change (95% CI) Fixed Effect / Random Effect	Estimates (*n*)	χ^2 Value	*P* Value: χ^2 Test for Heterogeneity	*P* Value: Egger Test for Publication Bias	*P* Value: Begg Test for Asymmetry (with Continuity Correction)
PM_{10}						
Mortality						
All causes						
All ages	0.14 (0.09 to 0.19) 0.27 (0.12 to 0.42)	8	30.4	< 0.001	0.127	1.000
≥ 65 yr	0.48 (0.38 to 0.57) 0.45 (0.29 to 0.61)	15	26.3	0.016	0.371	0.913
Respiratory						
All ages	0.41 (0.23 to 0.59) 0.86 (0.34 to 1.39)	8	25.3	0.001	0.085	0.902
≥ 65 yr	1.22 (1.08 to 1.37) 1.09 (0.55 to 1.63)	15	78.1	< 0.001	0.621	0.322
Cardiovascular						
All ages	0.22 (0.11 to 0.33) 0.36 (0.09 to 0.62)	8	18.7	0.009	0.405	0.666
≥ 65 yr	0.55 (0.41 to 0.70) 0.53 (0.31 to 0.75)	14	19.8	0.101	0.440	0.443
TSP						
Mortality						
All causes, all ages	0.21 (0.14 to 0.27) 0.21 (0.14 to 0.27)	9	4.3	0.833	0.971	0.917
NO_2						
Mortality						
All causes, all ages	0.83 (0.64 to 1.01) 0.98 (0.54 to 1.42)	7	17.7	0.007	0.484	0.548
Respiratory, all ages	1.74 (0.85 to 2.63) 1.74 (0.85 to 2.63)	6	1.1	0.901	0.524	0.221
Cardiovascular, all ages	1.03 (0.67 to 1.40) 1.08 (0.59 to 1.56)	6	8.0	0.236	0.946	1.000
Admissions						
Respiratory, all ages	0.37 (0.19 to 0.54) 0.92 (0.17 to 1.68)	5	41.4	< 0.001	0.129	0.221

Table continues next page

[a] The change in pollutant concentration was 10 μg/m^3 for all pollutants except CO, for which the change was 1 mg/m^3. Two estimates are given for each pollutant–outcome pair — the fixed-effects estimate followed by the random-effects estimate.

Summary Estimates

Summary effect estimates were calculated when four or more studies provided estimates for individual pollutant–outcome pairs. If more than one estimate was available for a city, the most recent result published was used. The fixed-effects and random-effects summary estimates for the pollutant–outcome pairs are shown in Table 14.

Table 14 (*Continued*). Summary Effect Estimates of the Percent Change in RR of Various Mortality and Admissions Outcomes in Various Age Groups Due to a Change in Pollutant Concentration, by Pollutant and Outcome[a]

Pollutant and Outcome	Percent Change (95% CI) Fixed Effect / Random Effect	Estimates (*n*)	χ^2 Value	*P* Value: χ^2 Test for Heterogeneity	*P* Value: Egger Test for Publication Bias	*P* Value: Begg Test for Asymmetry (with Continuity Correction)
O_3, 8-hr Averaging Time						
Mortality						
All causes, all ages	0.07 (−0.16 to 0.30) 0.07 (−0.16 to 0.30)	4	16.0	0.000	0.240	1.000
Respiratory, all ages	0.73 (0.30 to 1.16) 0.73 (0.30 to 1.16)	4	1.8	0.615	0.693	1.000
Cardiovascular, all ages	0.16 (−0.11 to 0.44) 0.12 (−0.29 to 0.54)	4	5.5	0.138	0.648	0.734
O_3, 24-hr Averaging Time						
Mortality						
All causes, all ages	0.07 (−0.07 to 0.21) 0.14 (−0.19 to 0.47)	4	2.1	0.559	0.156	0.308
Admissions						
Respiratory, all ages	0.26 (−0.06 to 0.59) 0.26 (−0.06 to 0.59)	4	2.9	0.400	0.016	0.308
SO_2						
Mortality						
All causes, all ages	0.35 (0.26 to 0.45) 0.68 (0.40 to 0.95)	15	78.2	< 0.001	0.010	0.048
Respiratory, all ages	1.00 (0.60 to 1.40) 1.00 (0.60 to 1.40)	7	3.2	0.777	0.224	1.000
COPD, all ages	0.93 (0.46 to 1.40) 1.72 (0.10 to 3.36)	4	8.4	0.038	0.218	0.734
Cardiovascular, all ages	0.32 (0.16 to 0.49) 0.95 (0.30 to 1.60)	9	28.7	< 0.001	0.078	0.917
Admissions						
Respiratory, all ages	0.22 (−0.10 to 0.54) 0.51 (−0.17 to 1.19)	6	18.1	0.003	0.044	0.133
CO						
Mortality						
All causes, all ages	0.34 (0.11 to 0.57) 1.89 (−1.59 to 5.50)	4	60.4	< 0.001	0.474	1.000

[a] The change in pollutant concentration was 10 μg/m^3 for all pollutants except CO, for which the change was 1 mg/m^3. Two estimates are given for each pollutant–outcome pair — the fixed-effects estimate followed by the random-effects estimate.

PM_{10} For each 10-μg/m^3 increase in PM_{10} concentration, according to random-effects estimates, the mean daily all-natural-cause mortality in all age groups increased by 0.27% (95% CI, 0.12–0.42); respiratory mortality, by 0.86% (95% CI, 0.34–1.39); and cardiovascular mortality, by 0.36% (95% CI, 0.09–0.62) (Figure 61 and Table 14). These random-effects estimates were each based on eight estimates; within each set of estimates, there was evidence of heterogeneity.

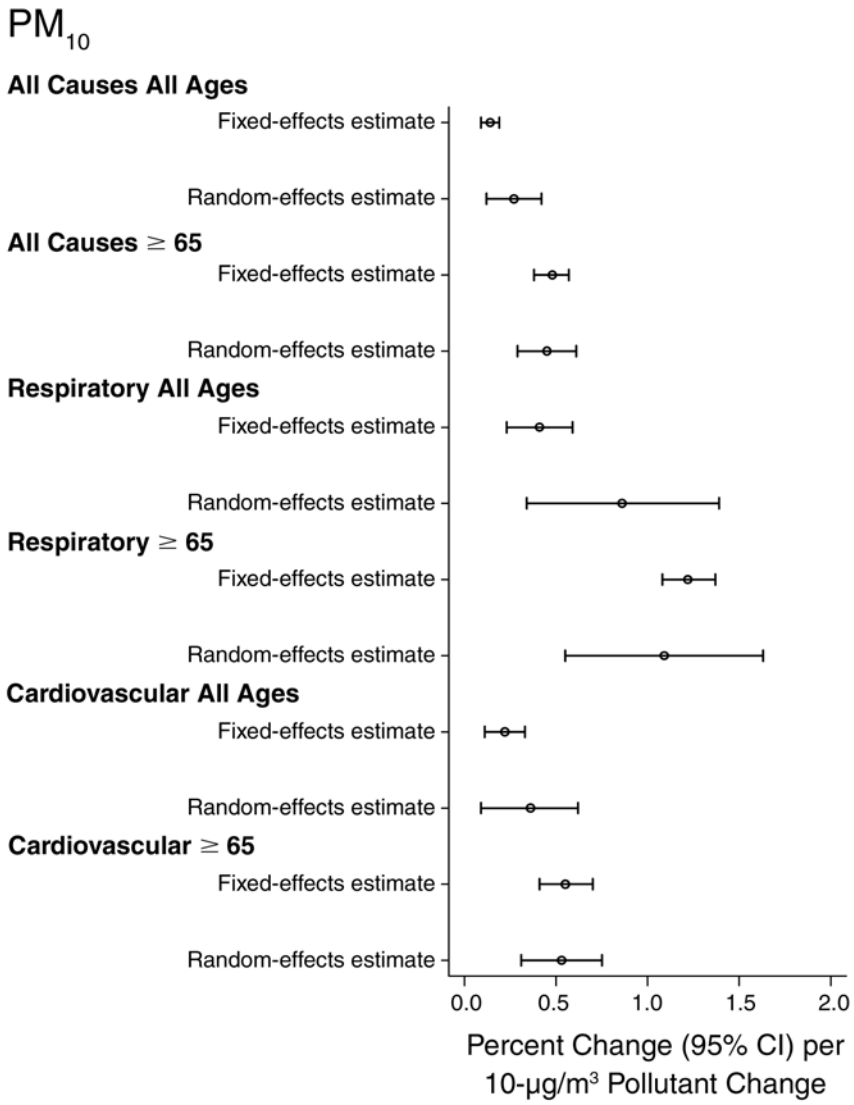

Figure 61. Summary effect estimates for all-natural-cause, respiratory, and cardiovascular mortality per 10-μg/m^3 change in PM_{10} concentration. Y-axis labels in bold type specify the cause of death and the age group.

There were also sufficient numbers of estimates to calculate summary effect estimates for the age group of 65 years or older (Figure 61). Associations in this age group tended to be larger than those for all ages combined and were also heterogeneous. For all pollutant–outcome pairs analyzed, the lower CIs were above zero.

TSP For TSP, sufficient estimates for inclusion in the meta-analysis were available for only a single health outcome. For a 10-μg/m^3 increase in TSP concentration, the daily mean all-natural-cause mortality in all age groups increased by 0.21% (95% CI, 0.14–0.27), on the basis of nine estimates.

$PM_{2.5}$ For $PM_{2.5}$, none of the pollutant–outcome pairs provided sufficient data for meta-analysis.

NO_2 For NO_2, there were 7, 6, and 7 estimates available for meta-analysis across all age groups for all-natural-cause, respiratory, and cardiovascular mortality, respectively. The random-effects summary estimates were 0.98% (95% CI, 0.54–1.42), 1.74% (95% CI, 0.85–2.63), and 1.08% (95% CI, 0.59–1.56) per 10-μg/m^3 increase in NO_2. A meta-analysis of the results for all-natural-cause mortality suggested significant heterogeneity among the estimates. However, incorporating this uncertainty in a random-effects model did not yield results that were substantially different from those derived from the fixed-effects model. No evidence of heterogeneity was found for cause-specific mortality. A single summary estimate for hospital admissions was also positive: a 0.92% (95% CI, 0.17–1.68) increase in respiratory admissions associated with a 10-μg/m^3 increase in NO_2. This random-effects estimate was considerably larger than the corresponding fixed-effects estimate (of 0.37% [95% CI, 0.19–0.54]). All the fixed-effects and random-effects summary estimates calculated had lower CI limits above 0.

O_3 Summary estimates for O_3 were available for a number of pollutant–outcome pairs and for both 24-hour and maximum 8-hour averaging times. The summary estimates, each based on just four results, were all positive, and the lower confidence limits were below 0 in all but one case. Evidence of heterogeneity was found for all-natural-cause mortality only; the random-effects estimate was 0.07% (95% CI, −0.16 to 0.30) per 10-μg/m^3 increase in the maximum 8-hour O_3 concentration.

SO_2 For each 10-μg/m^3 increase in SO_2 concentration, daily mean numbers of deaths from all natural causes, respiratory causes, and cardiovascular causes increased by 0.68% (95% CI, 0.40–0.95), 1.00% (95% CI, 0.60–1.40), and 0.95% (95% CI, 0.30–1.60), respectively. These random-effect estimates were based on 15, 7, and 9 results, respectively, and evidence for heterogeneity among the estimates was found for both all-natural-cause mortality and cardiovascular mortality. Four reports for deaths from COPD resulted in a summary estimate very similar to that for all respiratory diseases. Meta-analysis of results from six studies of hospital admissions for respiratory disease produced a summary estimate of 0.51% (95% CI, −0.17 to 1.19), a result based on a random-effects model.

CO For CO, sufficient estimates for inclusion in the meta-analysis were available for only a single health outcome. For each 1-mg/m^3 increase in CO concentration, daily mean numbers of deaths from all natural causes increased by 1.89% (95% CI, −1.59 to 5.50) — a result with a high degree of heterogeneity, as indicated by the wide CI, based on nine reports.

Investigation of Sources of Heterogeneity

Several summary estimates highlighted above were derived from a random-effects model. This model takes into account variation in effect estimates among cities when calculating the summary estimate and its precision. Evidence of heterogeneity in a collection of city-specific effect estimates suggests that the variation among cities is not simply random variation. Potential reasons for the variation may be the composition, physical characteristics, and sources of the pollution to which the population is exposed, the health status and health care systems of the study population, and variation in meteorologic conditions or the analytic strategy adopted by the investigators. Some relevant data are available from the articles themselves. For example, the year of publication could be an indicator of the statistical protocols used (because their sophistication has increased over time), the time period studied (because recently published studies tend to use more recent data), or both. Other relevant data include average pollution concentrations (because the effects of pollution may be greater in more-polluted cities) and geographic region (as indicated by the region classifications assigned by the WHO).

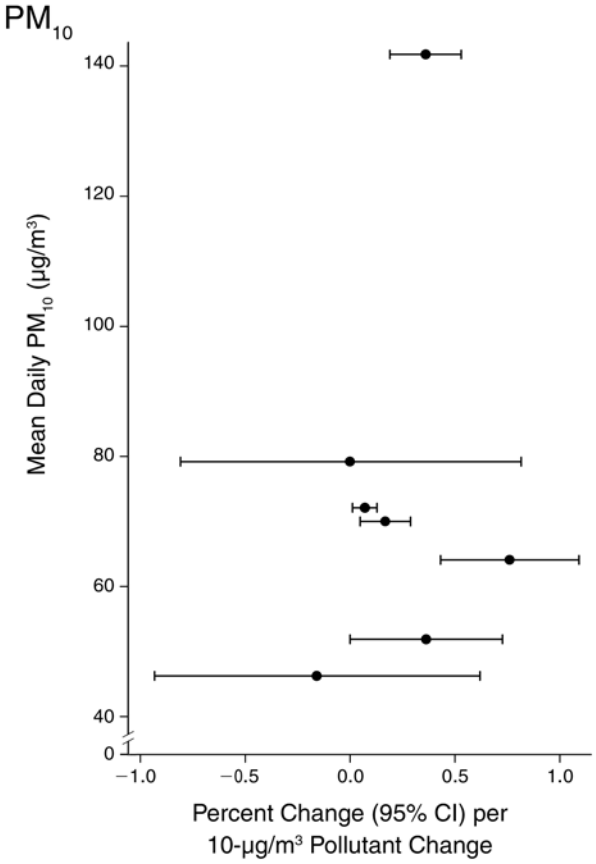

Figure 62. Effect estimates for all-natural-cause mortality per 10-µg/m³ change in 24-hr mean PM_{10} concentration, among persons of all ages, according to daily mean PM_{10} concentration. The data are also shown in Figure 63 with reference citations.

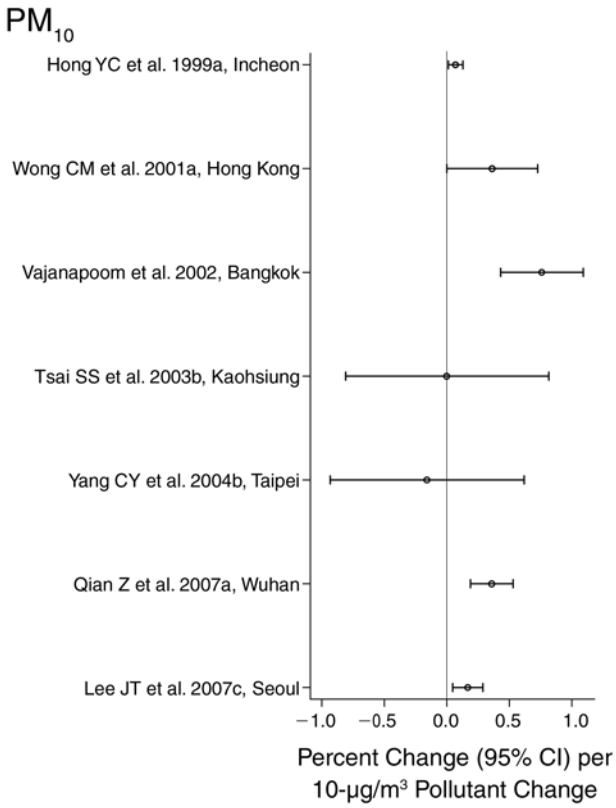

Figure 63. Effect estimates for all-natural-cause mortality per 10-µg/m³ change in 24-hr mean PM_{10} concentration, among persons of all ages, according to publication. The data are also shown in Figure 62. Y-axis labels give study information in the following sequence: reference citation and study location.

The city-specific estimates for PM_{10} used in the calculation of the summary estimate are shown according to pollutant concentration (Figure 62) and year of publication (Figure 63). Similar plots for the other pollutant–outcome pairs for which it was possible to calculate summary estimates are shown in Appendix A (Figures A.31 to A.40). None of these figures suggests that either pollutant concentration or year of publication was associated with the magnitude of the pollutant effect estimates observed. There were insufficient numbers of estimates from the different WHO regions to investigate whether effect estimates varied according to WHO region.

Another possible source of heterogeneity among study estimates that we cannot rule out in our review may be the lag times selected by the authors for presentation in their papers (Anderson et al. 2005).

DISCUSSION

Review of Findings

In this review, we identified over 100 daily time-series studies of the health effects of short-term exposure to air pollutants in Asia. This represents a substantial increase in the body of evidence from this kind of study within Asia since the last review, when 45 studies were identified. The additional studies have allowed us to calculate summary effect estimates for more outcomes and to base these summary measures on more data. Our review protocol dictated that we select the most recently published estimates from each city for inclusion in the meta-analysis. Hence, although the numbers of studies have increased substantially, the evidence may still be concentrated in relatively few cities within Asia.

A comparison of Table 20 in the 2004 literature review (Special Report 15; HEI ISOC 2004) and Table 14 in this

report shows that the number of individual estimates (for all-natural-cause mortality in all age groups) included in the summary estimates increased from 4 to 8 for PM_{10} and from 11 to 15 for SO_2. These additional data enabled us to calculate summary estimates for more pollutants and for more outcomes. Notably, all pollutants were positively associated with adverse health effects, and the majority of the summary estimates have lower confidence limits that are above zero (showing a percent increase in the RR of the outcome) and upper limits that indicate estimates of an increase in the RR of between 1% and 4% per unit increase in pollutant concentration. A new finding from this review is the suggestion that associations between PM_{10} and death are greater for persons 65 years of age or older than for those of all ages — a pattern observed for all-natural-cause mortality and mortality from respiratory causes or cardiovascular diseases. In each case, the point estimate for the ≥ 65-year age group is greater than the point estimates for the corresponding all-ages group, although the CIs overlap (Figure 61). This is an important finding, given the aging population in Asia. Recent evidence from the Air Pollution and Health: A European and North American Approach (APHENA) study (Katsouyanni and Samet et al. 2009) also suggests a higher degree of risk in the older population than among younger adults. Some findings suggest the contrary, however, at least for hospital admissions. In a study by Anderson and colleagues (2003), no evidence was found to suggest that short-term exposure to air pollutants increased the risk of admission in older people — though the attributable risk was higher in that group than in younger groups because of the greater numbers of people at risk.

Comparison with Results from the PAPA Studies in Four Asian Cities

The HEI PAPA program initially funded a set of time-series studies of daily mortality in four large Asian cities. The project followed a common design and data management and statistical protocols to coordinate investigation of the associations between daily all-natural-cause mortality or daily cause-specific mortality, and PM_{10}, NO_2, O_3, and SO_2 in Bangkok (from 1999 to 2003), Hong Kong (from 1996 to 2002), Shanghai (from 2001 to 2004), and Wuhan (from 2001 to 2004). (The results from these important studies were in press at the time this analysis was being conducted, but they are discussed here because they bring important new evidence to this area [HEI Public Health and Air Pollution in Asia Program 2010].)

Table 15 compares the summary measures calculated for all Asian time-series studies of all-natural-cause mortality, with the meta-analytic estimates of the four cities in the PAPA studies reported by Wong CM and colleagues (2008b, 2010a). A clear and consistent pattern is evident: summary estimates from the PAPA studies for all-natural-cause and cardiovascular mortality are higher than those calculated from the systematic review of the published literature for each of the four pollutants investigated (PM_{10}, NO_2, O_3, and SO_2). Conversely, except for SO_2, the PAPA summary estimates are lower than the review estimates for respiratory mortality (although the CIs overlap substantially). For PM_{10}, NO_2, and SO_2, these differences do not alter the conclusions one might draw regarding the evidence for a potential hazard; in each case, the associations were positive, with lower confidence limits above zero. However, the differences in the magnitude of the associations are relevant to the calculations of the public health impact of air pollution and therefore are important. The one exception is O_3: the summary estimate for all-natural-cause mortality and the daily maximum 8-hour averages obtained from our meta-analysis was 0.07% (95% CI, −0.16 to 0.30); the corresponding estimate derived from the four PAPA cities was 0.38% (95% CI, 0.23–0.53).

Each of the four cities in the PAPA program has been studied previously, and estimates for all or some of these cities are included in the summary estimates presented in the current report. To update the currently reported estimates, we replaced the previous estimates for Bangkok, Hong Kong, Shanghai, and Wuhan with those from the PAPA studies (or added them, if none was previously included), as appropriate (Table 15). These new data from the PAPA studies were especially selected because of their importance to the study of the health effects of outdoor air pollution in Asia. There may be other time-series studies of Asian cities in progress with imminent publication that have not been included in this update of the evidence.

It is unclear why estimates from the PAPA studies for all-natural-cause and cause-specific mortality were higher than those obtained from the existing Asian literature for each of the pollutants studied. We expected some degree of publication bias in the existing literature, leading to an upward bias in summary effect estimates (Anderson et al. 2006). However, there was little systematic evidence of publication bias (as indicated by the Begg and Egger statistical tests) in mortality effect estimates for any of the pollutants. The PAPA studies used a common protocol, incorporating current methodologic approaches and knowledge, both for data collection and statistical analysis in each of the four participating cities. The PAPA protocol shared one important feature with the APHEA study protocol (Katsouyanni et al. 1996) — the appropriate control for seasonality (using natural splines for data smoothing) was determined using the partial autocorrelation function (PACF). However, it is unlikely that this was a significant factor in the difference between the PAPA results and the current results. The APHENA study (Samoli et al. 2008) assessed a range of statistical models and reported little change in the overall summary estimates for PM_{10} and

Table 15. Summary Effect Estimates of the Percent Change in RR of Mortality in All Age Groups Due to Change in Pollutant Concentration, by Meta-Analysis[a]

	Current Review of Asian Literature		PAPA Studies[b]		Recalculated Estimates After Incorporation of PAPA Study Results		
Pollutant and Cause of Death	RR Percent Change (95% CI)	Studies with Data (*n*)	RR Percent Change (95% CI)	Studies with Data (*n*)	RR Percent Change (95% CI)	Studies with Data (*n*)	Estimates Replaced or Added
PM_{10}							
All causes	0.27 (0.12 to 0.42)	8	0.55 (0.26 to 0.85)	4	0.33 (0.16 to 0.51)	8	Replaced Bangkok, Hong Kong, Shanghai, and Wuhan estimates
Respiratory	0.86 (0.34 to 1.39)	8	0.62 (0.22 to 1.02)	4	0.64 (0.26 to 1.02)	8	Replaced Bangkok, Hong Kong, Shanghai, and Wuhan estimates
Cardiovascular	0.36 (0.09 to 0.62)	8	0.58 (0.22 to 0.93)	4	0.41 (0.15 to 0.67)	8	Replaced Bangkok, Hong Kong, Shanghai, and Wuhan estimates
NO_2							
All causes	0.98 (0.54 to 1.42)	7	1.23 (0.84 to 1.62)	4	1.02 (0.71 to 1.34)	8	Replaced Hong Kong, Shanghai, and Wuhan estimates; added Bangkok estimate
Respiratory	1.74 (0.85 to 2.63)	6	1.48 (0.68 to 2.28)	4	1.38 (0.93 to 1.83)	9	Replaced Wuhan estimate; added Bangkok, Hong Kong, and Shanghai estimates
Cardiovascular	1.08 (0.59 to 1.56)	7	1.36 (0.89 to 1.82)	4	1.14 (0.76 to 1.53)	8	Replaced Hong Kong, Shanghai, and Wuhan estimates; added Bangkok estimate
O_3, 8-hr Averaging Time							
All causes	0.07 (−0.16 to 0.30)	4	0.38 (0.23 to 0.53)	4	0.25 (−0.07 to 0.56)	5	Replaced Hong Kong, Shanghai, and Wuhan estimates; added Bangkok estimate
Respiratory	0.73 (0.30 to1.16)	4	0.34 (−0.07 to 0.75)	4	0.34 (−0.07 to 0.75)	5	Replaced Hong Kong, Shanghai, and Wuhan estimates; added Bangkok estimate
Cardiovascular	0.12 (−0.29 to 0.54)	4	0.37 (0.01 to 0.73)	4	0.36 (0.03 to 0.69)	5	Replaced Hong Kong, Shanghai, and Wuhan estimates; added Bangkok estimate
SO_2							
All causes	0.68 (0.40 to 0.95)	15	1.00 (0.75 to 1.24)	4	0.75 (0.48 to 1.02)	16	Replaced Hong Kong, Shanghai, and Wuhan estimates; added Bangkok estimate
Respiratory	1.00 (0.60 to1.40)	7	1.47 (0.85 to 2.08)	4	1.12 (0.76 to 1.47)	9	Replaced Hong Kong and Wuhan estimates; added Bangkok and Shanghai estimates
Cardiovascular	0.95 (0.30 to1.60)	9	1.09 (0.71 to 1.47)	4	1.16 (0.53 to 1.80)	10	Replaced Hong Kong, Shanghai, and Wuhan estimates; added Bangkok estimate

[a] The change in pollutant concentration was 10 μg/m^3 for all pollutants except CO, for which the change was 1 mg/m^3.

[b] The PAPA study included results from Bangkok, Hong Kong, Shanghai, and Wuhan (Wong CM et al. 2008b, 2010a).

all-natural-cause mortality on the basis of various methods of selecting the degree of adjustment for season in the models. For example, similar effect estimates resulted from the use of an a priori 8 degrees of freedom per year to control for season, compared with the number of degrees of freedom determined using the PACF criterion (by minimizing the sum of the sample's PACFs): 0.9% (95% CI, 0.3–1.4) and 1.1% (95% CI, 0.6–1.6) per 10-μg/m^3 increment in PM_{10} concentration, respectively (Katsouyanni and Samet et al. 2009).

Another possible reason why the estimates from the PAPA studies for all-natural-cause and cause-specific mortality were higher than those calculated on the basis of the existing Asian literature may be that the PAPA studies used the most recent data available and that the adverse health effects of air pollution are increasing over time. However, as Figure 63 and Appendix Figure A.36 and A.37 show, there is little evidence of such a temporal trend in the existing Asian literature.

In their report of the PAPA studies in four cities, Wong CM and colleagues (2008b, 2010a) noted that estimates for Bangkok were generally higher than those for the three Chinese cities (Hong Kong, Shanghai, and Wuhan) and therefore they also calculated summary effect estimates for the three Chinese cities only. These summary estimates were generally lower than those based on all four cities and were more in line with the meta-analytic estimates calculated in this review. The large effect estimates for Bangkok in the PAPA studies (Vichit-Vadakan et al. 2008, 2010) therefore seem to account for most of the differences between the two sets of summary effect estimates.

Comparison with Results from India

Two additional PAPA reports were in press at the time of this writing and therefore were not included in this meta-analysis. The first, by Rajarathnam and colleagues (2010), reported results from Delhi from 2002 through 2004, recording a 0.15% increase in the RR of all-natural-cause mortality associated with a 10-μg/m^3 increase in PM_{10}. The second study, in Chennai (Balakrishnan et al. 2010) and also conducted from 2002 through 2004, reported an association that was estimated to be 0.4% per 10-μg/m^3 increase in PM_{10}. These results fall below and above the summary estimate from the current review, respectively, and below the meta-analytic estimate from the PAPA studies in the four cities.

Comparison with Results from Other Parts of the World

An authoritative and comprehensive review published in 2006 by Pope and Dockery (2006) summarized time-series results from key studies of PM_{10} and mortality, and reported results for not only PM_{10} but also $PM_{2.5}$, black smoke, TSP, and SPM. Figure 32 shows the resulting range of effect estimates, with the findings from the current literature review falling into the lower end of the range. Thus, although the summary estimate for PM_{10} from the current systematic review of the Asian literature is on the low side, it is consistent with the range of observed effects found in other parts of the world.

Limitations of This Review

Since the first review of the Asian literature published in 2004 (HEI ISOC 2004), new search procedures, used in our updated review, have been developed and refined. A key improvement has been contact with local investigators who are able to identify and translate studies published locally within Asia, usually not in English. Every effort has been made to identify all relevant studies, but some may have been missed, so we cannot claim to have fully represented the actual body of evidence in this field. We know of one article that was identified in the literature search, but the full article could not be obtained.

Although the number of time-series studies published has increased substantially since the previous review (115 vs. 45), there remains a disappointing lack of publications on $PM_{2.5}$. We were able to identify only eight such studies (compared with one in the 2004 review) reporting effect estimates that could be included in a quantitative meta-analysis. After stratification by outcome, disease, and age, the results did not yield sufficient estimates to calculate a summary effect estimate for $PM_{2.5}$. The relative paucity of studies of $PM_{2.5}$ represents a substantial shortcoming in the accumulating body of evidence from Asia, particularly since $PM_{2.5}$ is a better measure of particle pollution from combustion sources than either TSP or PM_{10}. In addition, little new evidence regarding TSP has been published since 2004 — probably owing to increased monitoring of PM_{10}, rather than of TSP, in the region.

Heterogeneity and Effect Modification

Many pollutant–outcome relations reported here showed considerable heterogeneity. Heterogeneity can result from differences in the quality of the data or the methods of analysis. For example, errors in measurement of exposure among cities can produce the appearance of differences in effects where none truly exist (Samet et al. 2000a). Heterogeneity can also reflect effect modification: real differences in the magnitude of the effect estimate. Effect modification can be related to factors such as differences in the underlying vulnerability of the population to air pollution or differences in the toxicity of the air pollution mixture among cities. The small number of studies in the Asian literature and the lack of data on possible effect-modifying factors precluded a detailed evaluation of effect modification in this review, but the results of large multi-city studies in the U.S. and Europe (Samet et al. 2000b; Katsouyanni et al. 2001) and recent meta-analyses of the broader literature (Levy et al. 2000) suggest that such factors exist. We did examine, graphically, the possibility that average pollution concentrations in the cities represented in the Asian literature may explain variation in effect estimates among the

cities, but we found no evidence to support this hypothesis. Appendix Figure A.34 suggested a possible trend toward increasing effect size with increasing O_3 concentrations. However, because only four estimates were plotted, interpretation should be cautious. We also investigated whether year of publication, a surrogate for a number of potential effect modifiers (e.g., statistical method or trends in pollution concentrations), was associated with effect size; we found no evidence to support this possibility. As the Asian literature grows, the investigation of sources of heterogeneity and potential effect modifiers will become an informative and valuable area for further research.

Multi-Pollutant Models

A substantial number (44) of the studies included in this review have reported results from analyses of multi-pollutant models. The numbers of studies reporting results (irrespective of outcome) for each pollutant are as follows: 26 for PM_{10}, 4 for $PM_{2.5}$, 27 for O_3, 24 for NO_2, 32 for SO_2, and 12 for TSP. No multi-pollutant analyses involved data for CO.

In many of these studies, various combinations of pollutants were evaluated. However, the numbers of estimates available for comparison tend to fall substantially when the studies target specific outcomes, ages of subjects, diseases, and pollutant combinations. In some papers, results from multi-pollutant models were presented graphically, in which case it was not always possible to obtain the numerical values of the estimates and associated measures of precision for inclusion and standardization in APED. In other cases, many combinations were reported, but according to the APED data-extraction protocol, only some of the results were entered into APED. Hence, the complete articles should be consulted to obtain a complete picture of the robustness of the pollutant–outcome associations.

The most relevant publication that reports results for a comprehensive set of multi-pollutant models is the PAPA studies of daily mortality in Bangkok, Hong Kong, Shanghai, and Wuhan (Wong CM et al. 2008b, 2010a; HEI Public Health and Air Pollution in Asia 2010). The authors investigated the robustness of the associations between all-natural-cause mortality, or cause-specific mortality, and PM_{10} or NO_2 to the inclusion of the other pollutants in the model (i.e., PM_{10} or NO_2, SO_2, or O_3). These results, reported in supplementary material published on the Web, suggest that the associations between NO_2 and mortality were robust to the inclusion of SO_2 and O_3 individually, whereas the associations between PM_{10} and mortality were reduced to almost zero by the inclusion of NO_2 (but not SO_2 or O_3). These findings are only partially in line with those from studies in North America and Europe. Peng and colleagues (2005) reported results from analyses of data from 45 cities in the NMMAPS study in North America and concluded that associations between PM_{10} and mortality were unaffected by the inclusion in the model of data for SO_2, O_3, and NO_2. A similar finding was reported by the APHEA investigators in a study of 29 European cities (Katsouyanni et al. 2001). A detailed analysis of this complex issue is warranted.

Studies of Pollution Episodes

In this review of the time-series literature, we have focused on studies of the health effects of short-term (daily) exposure to outdoor air pollutants. As part of the process of identifying appropriate studies, we found 9 studies of the health effects of pollution during periods of dust storms or forest fires. Our search procedures were not designed to identify these studies; thus, this number may be an underestimate. Nonetheless, it is worth noting the growing literature on this subject and, in particular, the impact these increasingly frequent events may have on the health of the Asian population.

Section VI. Studies of Selected Effects of Long-Term Exposure to Ambient Air Pollution

The health effects of long-term exposure to ambient air pollution range from reduced life expectancy (through increased incidences of various diseases or conditions) to asymptomatic physiologic abnormalities (such as reduced lung function) (ATS 2000; WHO 2006a). Much of the evidence comes from epidemiologic studies of long-term exposure that most often involve comparisons of the incidence or prevalence of health outcomes among areas that vary in their current or historical ambient concentrations of air pollution. The PAPA–SAN database was used to identify such studies of Asian populations published in the peer-reviewed literature from January 1980 through August 2007. Search results were verified by independent searches conducted by APED (described earlier) and a consultant, Dr. Robert Chapman, of Chulalongkorn University, Bangkok. We focused on studies of selected respiratory symptoms, pulmonary function, and illness prevalence (described in more detail below), lung cancer, and adverse pregnancy outcomes (i.e., low birth weight [< 2500 g] and preterm delivery [at < 37 weeks' gestational age]). These endpoints have been studied previously in other regions and are considered likely to contribute to the burden of disease from air pollution in Asia.

In contrast to the Asian time-series studies (reviewed above in *Section V. Quantitative Assessment of Daily Time-Series Studies*), the studies of the chronic effects of long-term exposure are fewer and vary widely with respect to study design, analytic methods, and overall scientific rigor. For these reasons, no summary estimates were calculated. Rather, quantitative estimates for selected outcomes are described and plotted to evaluate the evidence from studies in Asia about the effects of long-term exposure to ambient air pollution on selected chronic health effects and to assess whether it can be compared with the evidence from the broader literature.

Most studies contrasted the prevalence or, in the case of lung cancer, the occurrence of outcomes among populations that lived or attended schools in areas that differed with regard to the presence of major sources of air pollution, the concentrations of ambient air pollution, or both (case–control design). The sources in many studies were stationary — industrial facilities or power plants — but several studies focused on residential proximity to busy roads. In some studies, various cities or areas (e.g., urban vs. suburban or rural) were compared, whereas in others the comparison was among various parts of the same city; still others combined both designs. The studies also differed in the extent to which potential confounding factors were controlled by the design or analysis.

METHODS

This section describes general features of the methods used in this review for the studies of long-term exposure. Further methodologic details regarding studies of specific health outcomes are discussed in the relevant sections below.

Study Selection

Studies were identified on the basis of the most recent update of the PAPA–SAN database, described above. Additional PubMed searches were also conducted, using the search terms specified in the *PAPA–SAN Literature Review Manual* (HEI Public Health and Air Pollution in Asia 2006). Studies were included in this review if they:

1. Presented either new data or a reanalysis of a research data set;
2. (a) Presented effect estimates in relation to increments in the concentrations of specific air pollutants or contained data from which such effect estimates could be estimated, or (b) provided estimates of relative rankings of exposure in qualitative terms (e.g., for urban vs. rural areas);
3. Presented the methods of data collection and analysis with sufficient clarity to allow them to be understood with reasonable confidence; and
4. Reported, or gave sufficient information to derive, CIs for the effect estimates (e.g., SEs of logistic regression coefficients, CIs around these coefficients, or unadjusted counts of symptoms or illnesses at different air pollution concentrations).

Quantification of Results

Most quantitative results are presented in terms of odds ratios (ORs) that estimate the RR of the health outcome in

association with a given increment in exposure, in cross-sectional studies or in studies of incidence or mortality. Effects on pulmonary function are usually quantified in terms of average changes in the level of lung function. The statistical precision of the estimates presented is indicated by the width of the 95% CI or can be inferred from the *P* value from statistical tests, if presented. The ORs (and 95% CIs) for the one or more estimates from each study are displayed in forest plots.

This review presents two types of OR estimates that differ with regard to how exposure to air pollution is characterized.

- The first type is estimates based on *exposure contrasts* (differences in exposures to air pollution) among populations in areas classified qualitatively as more polluted or less polluted. Examples include comparisons of the prevalence of respiratory symptoms in residents of industrial versus nonindustrial areas or areas that vary in proximity to major roads; the OR then estimates the proportional increase in the outcome associated with residence in the more-polluted areas as compared with the less-polluted areas.
- The second type is estimates based on differences in ambient air pollution concentrations. In studies where such estimates were reported, they were recorded for analysis here. Some studies, however, instead reported simply the average concentrations of pollutants in various geographic areas, with most being community-average concentrations calculated over a period of months or years. From these averages, when possible, we calculated an OR that estimated the proportional increase in risk of the health outcome associated with an incremental difference in the ambient concentration. To do so, the reported concentrations were standardized to 10 μg/m^3 for particles and most gases (except CO, which was standardized to 100 μg/m^3).[e]

[e] For studies reporting modeled ORs and sufficient variance-related information and for studies in which unadjusted counts were used, the natural logs of modeled ORs and CIs (or SEs) were derived. Intercommunity differences in the reported average ambient air pollutant concentrations were also calculated in the units in which they were reported (e.g., ppb, ppm, μg/m^3). The number of designated increments (100 μg/m^3 for CO and 10 μg/m^3 for all other pollutants) in each of the intercommunity differences in concentrations was then calculated for purposes of standardization. Thus, for example, if an intercommunity difference in reported NO_2 concentrations was 15.7 ppb, the conversion from ppb to μg/m^3 and the rescaling to a 10-unit increment ([15.7 ppb × 1.88 μg/m^3 per ppb] ÷ 10) would yield 2.95 designated increments between the communities. The natural logs of reported ORs and their CIs (or SEs) were then divided by the intercommunity difference in average pollutant concentration, expressed as the number of designated increments, to yield natural logs for the standardized pollutant increments (standardized logs). (For instance, the standardized logs for NO_2 in the example above would be the result of dividing the natural logs of the reported OR and its CI by 2.95.) The resulting estimates were exponentiated to yield standardized ORs and CIs. This procedure was applied to results from prevalence studies, to case–control studies of lung cancer, and for each pollutant reported. Information was taken from single-pollutant models only; multiple-pollutant models were not used for this purpose.

CHRONIC RESPIRATORY DISEASE

This section focuses on four respiratory-disease outcomes: (1) chronic phlegm (for ≥ 3 months/year, unless otherwise noted) as an indicator of the presence of chronic respiratory disease, (2) pulmonary function, (3) physician-diagnosed asthma, and (4) wheeze. These specific outcomes were selected because they are indicators of the prevalence of COPD and asthma, the two chronic, nonmalignant respiratory diseases of greatest public health importance and of greatest interest with regard to air pollution.

In the studies that assessed specific respiratory symptoms and disease prevalence, questionnaires were used for data collection, with parents reporting on behalf of young children. Most questionnaires covered the same basic symptoms: cough, phlegm, breathlessness, wheeze, and diagnoses of acute and chronic conditions such as asthma and COPD. In the vast majority of the studies, the questionnaires were based on existing standardized and validated questionnaires, with or without modification for local conditions. Details of the questionnaires often varied from study to study. For example, different studies often used different questions to identify asthma, with further qualification in some studies of the severity or period of disease (e.g., lifetime, or 12 months).

Chronic Phlegm

A total of 27 studies estimated the effects of exposure to air pollution from mobile and stationary sources on the prevalence of chronic phlegm, a major symptom of chronic respiratory diseases characterized by chronic, productive cough (e.g., chronic bronchitis, or tuberculosis [TB]) (Table 16). A study was considered to have reported credible estimates for chronic phlegm if the questionnaire requested information about phlegm production of ≥ 3 months/year, as was the case in the large majority of studies, or about another indicator of phlegm production over an extended period. Of such studies, 17 met the four criteria for inclusion as a study of selected effects of long-term exposure (specified in *Study Selection* earlier in Section VI). Ten of these studies attempted to control for potential confounders, including age, cigarette smoking, ETS, and indoor burning of solid fuels. Four studies were conducted in mainland China, one in Hong Kong, one in Taipei,China, three in Japan, and one in India. Of the 10 studies, 7 focused on adults (Nitta et al. 1993; Xu X and Wang L 1993**;** Yang CY et al. 1997; Nakai et al. 1999; Zhang J et al. 1999; Kumar R et al. 2004; Sekine et al. 2004) and 3 on children (Yu TS et al. 2001; Zhang JF et al. 2002; Qian Z et al. 2004). The studies are discussed in detail below, grouped according to which broad category of air pollution sources was studied.

Table 16. Studies of Chronic Phlegm

Citation	Study Location	Study Years	Study Sample	Pollution Variables	Questionnaires Used to Assess Chronic Phlegm	Results
Chhabra et al. 2001	Delhi, India	1988–1998	4171 M and F residents (> 17 years)	TSP, SO_2, and NO_2 concentrations in high pollution and low pollution zones within Delhi	ATS, BMRC, and NHLBI	The prevalence of chronic phlegm did not differ significantly between the two areas in a multi-variable regression analysis.
Duki et al. 2003	Jakarta, Bandar, and Lampung, Indonesia	1996–1997	16,663 M and F children–mother pairs	NO_2 concentrations and distance of residence from road (< 20 and 20 m)	ATS	The prevalence of "phlegm without cold" respiratory symptoms was associated with both NO_2 concentration and residence < 20 m from busy road.
Kagamimori et al. 1986	Awara-machi, Japan	1970–1979	M and F children (6–14 years)	NO_2 and SO_2 trends (following anti-pollution measures) in two areas near power plants (Aware and Honjyo districts) compared to a third district (Kitakata)	BMRC	The prevalence of "subacute phlegm" was associated with exposure to air pollution, especially in children with a positive response to a skin test for dust mites.
Kagamimori et al. 1990	Awara-machi, Japan	1971–1979	M and F children (6–14 years)	SPM, NO_2, and SO_2 concentrations (following anti-pollution measures) in two areas near power plants (Aware and Honjyo districts) compared to a third district (Kitakata)	BMRC	The prevalence of "subacute phlegm" increased after air quality deteriorated in association with the construction of an oil-fired power plant.
Karita et al. 2001	Bangkok and Ayutthaya, Thailand	1998–1999	78 M traffic policemen and 128 M non-traffic policemen	Traffic policemen in an urban area (Bangkok) compared to general policemen within the urban area and a rural area (Ayutthaya)	ATS	Chronic phlegm was more prevalent among policemen assigned to traffic duty (17%) than among non-traffic policemen (2.6%). ~ 45% of subjects in each group were smokers.
Karita et al. 2004	Bangkok, Thailand	1998–1999	530 M police-man-and-wife couples	Traffic policemen and their wives residing within 40 m of the center of Bangkok and grouped by distance from the city center and PM_{10} concentrations	ATS	Policemen working in heavy-traffic areas (vs. light-traffic areas) had an increased prevalence of chronic phlegm (OR, 2.19; 95% CI, 1.47–3.26). Chronic phlegm in their wives was associated with air pollution concentrations at their place of residence (OR for high vs. low concentrations, 1.53; 95% CI, 1.10–2.13).

Table continues next page

Table 16 (*Continued*). Studies of Chronic Phlegm

Citation	Study Location	Study Years	Study Sample	Pollution Variables	Questionnaires Used to Assess Chronic Phlegm	Results
Kumar R et al. 2004	Mandi Gobindgarh and Morinda, India	1999–2001	3603 M and F residents (> 15 years)	TSP, PM_{10}, NO_x, SO_2, CO, and O_3 concentrations in a high-pollution area (Mandi Gobindgarh) and a low-pollution area (Morinda)	BMRC	Prevalence of chronic phlegm was increased in an industrial vs. a non-industrial town (OR, 1.37; 95% CI, 0.96–1.97); chronic phlegm was associated with a 10-$\mu g/m^3$ increase in PM_{10} concentration (OR, 1.09; 95% CI, 0.99–1.20). Potential residual confounding by smoking status.
Langkulsen et al. 2006	Bangkok, Thailand	2004	878 M and F children (10–15 years)	PM_{10} concentrations in three high-pollution areas and a low-pollution area within Bangkok	ATS	Prevalence of "persistent phlegm" was increased among elementary-school children living in highly polluted roadside areas vs. less-polluted areas (2.5% vs. 0.9%; $P > 0.05$) and vs. highly polluted areas in general (9% vs. 0.9%, $P < 0.05$).
Maeda et al. 1991–1992	Tokyo, Japan	1987–1990	2600 F residents (30–60 years) and 360 M and F children (3–6 years)	NO_2 gradient based on distance of residence from road in Sumida Ward, Tokyo (< 20 and 20–150 m) and a residential suburb of Tokyo (Higashiyamato)	ATS	Prevalence of chronic phlegm was elevated among those living < 20 m from busy roads vs. in a residential district with no busy roads: 7.2% vs. 3.6% in analysis controlled for age, years at residence, occupation, smoking status, and type of heat. No variance estimate was provided. Results not adjusted for smoking status.
Nakai et al. 1999	Tokyo, Japan	1987–1990	1986 F residents (30–59 years)	NO_2 gradient based on distance of residence from road in Sumida Ward, Tokyo (< 20 and 20–150 m) and a residential suburb of Tokyo (Higashiyamato)	ATS	Prevalence of chronic phlegm was increased in women living < 20 m from a busy road vs. in a suburban area (OR, 1.79; 95% CI, 1.07–3.01), and was associated with an increment of 10 $\mu g/m^3$ in NO_2 concentration (OR, 1.12; 95% CI, 1.01–1.25). Extension of Maeda et al. 1991–1992. Results not adjusted for smoking status.
Nitta et al. 1993	Tokyo, Japan	1979-1983	4822 F residents (40–59 years)	Distance from road (< 20, 20–50, and 50–100 m)	ATS	Prevalence of chronic phlegm was increased in women living < 20 m vs. > 20–50 m from busy road: OR, 1.26 (95% CI, 0.94–1.70).

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Table 16 (*Continued*). Studies of Chronic Phlegm

Citation	Study Location	Study Years	Study Sample	Pollution Variables	Questionnaires Used to Assess Chronic Phlegm	Results
Ong et al. 1991	Hong Kong, China	1989	3846 M and F primary-school children	TSP, RSP, SO_2, and NO_2 concentrations in a high-pollution area (Kwa Tsing district) and a low-pollution area (southern district)	WHO-recommended, unspecified	In a district with high levels of emissions from factories, primary-school children had significantly higher rates of chronic phlegm than did those in a control group who lived in a less-polluted district (10% vs. 7.8%, $P = 0.05$).
Peters J et al. 1996	Hong Kong, China	1989–1991	3521 M and F children (8–12 years)	TSP, RSP, SO_2, and NO_2 trends (following anti-pollution measures) in a high-pollution area (Kwa Tsing district) and a low-pollution area (southern district)	WHO and BMRC	Differences in the prevalence of chronic phlegm among children in two districts decreased after improvements in air quality in the most-polluted district. The OR before sulfur reduction was 1.11 (95% CI, 0.96–1.30) and after reduction was 0.88 (95% CI, 0.68–1.13).
Qian Z et al. 2000	Chongqing, Guangzhou, Lanzhou, and Wuhan, China	1985–1988	2789 M and F children (5–14 years)	TSP, SO_2, and NO_2 concentrations in high-pollution areas (Langzhou and urban Wuhan) and low-pollution areas (Guanzhou and suburban Wuhan)	ATS	TSP levels were significantly associated with chronic phlegm in three Chinese cities.
Qian Z et al. 2004	Guangzhou, Lanzhou, and Wuhan, China	1993–1996	7058 M and F school-children (5–16 years)	TSP, $PM_{10-2.5}$, $PM_{2.5}$, SO_2, and NO_x concentrations in high- (urban) and low- (suburban) pollution district clusters	ATS	Chronic phlegm was more prevalent among children in the high-pollution area vs. the low-pollution area (OR, 5.44; 95% CI, 3.38–8.78).
Sekine et al. 2004.	Tokyo, Japan	1987–1994	5682 F residents (30–59 years)	Proximity to traffic: roadside areas grouped by NO_2 and SPM concentrations	ATS	Chronic phlegm was more prevalent among women living < 20 m from a busy road vs. in areas of low traffic (OR, 1.78; 95% CI, 1.26–2.53).
Shima et al. 2003	Chiba Prefecture, Japan	1992	2506 M and F children (6–9 years)	SPM and NO_2 concentrations and distance from road (0–49 m and > 49 m) in urban areas (Chiba, Funabashi, Kashiwa, Ichikawa) compared with rural areas (Ichikawa, Tateyawa, Mobara, Kisarazu)	ATS	The prevalence of chronic phlegm was lower among male children living 0–49 m from busy road (0.0%) than among those living > 49 m away (1.3%) or in rural areas (1.3%) ($P_{trend} = 0.555$); the same pattern was found among female children (1.15% vs. 1.7% and 1.9%; $P_{trend} = 0.848$).

Table continues next page

Table 16 (*Continued*). Studies of Chronic Phlegm

Citation	Study Location	Study Years	Study Sample	Pollution Variables	Questionnaires Used to Assess Chronic Phlegm	Results
Tam et al. 1994	Hong Kong, China	1989–1990	423 M and F children (10–12 years)	SO_2, NO_2, and RSP concentrations in a high-pollution area (Kwa Tsing district) and a low-pollution area (southern district)	No standard questionnaire	Little evidence of increased prevalence of chronic phlegm found in more-polluted vs. less-polluted district: 7% vs. 5%, P = 0.50.
Wongsurakiat et al. 1999	Bangkok, Thailand	1996–1997	620 M traffic policemen (20–60 years) and 303 M controls (students and other workers)	Traffic policemen (with and without protective masks) and controls within Bangkok	BMRC	Nonsmoker traffic policemen had a higher prevalence of "cough or phlegm" than nonsmoker controls (OR, 2.4; 95% CI, 1.3–4.6). Use vs. nonuse of a mask showed little evidence of a protective effect among nonsmoker traffic policemen (OR, 0.79; 95% CI, 0.3–2.3).
Xu X and Wang L 1993	Beijing, China	1986	1576 M and F adult never-smokers (40–69 years)	Particulates in industrial, residential and suburban areas	No standard questionnaire	Residence in industrial area vs. suburban area was associated with increased prevalence of chronic phlegm among never-smokers (OR, 1.60; 95% CI, 1.00–2.80). An increment of 10 μg/m^3 in SO_2 concentration was associated with increased prevalence of chronic phlegm (OR, 1.12; 95% CI, 1.01–1.25).
Yang CY et al. 1997	Lin-yuan and Taihsi, Taipei, China	1995	2036 M and F adult residents of Lin-yuan and 1976 controls (30–64 years)	PM_{10}, NO_2, and SO_2 concentrations in a high-pollution area (Lin-yuan) and a low-pollution area (Taihsi)	ATS	The prevalence of chronic phlegm was higher in association with residence in a petrochemical industrial area vs. control area (OR, 2.01; 95% CI, 1.19–3.42); an increase of 10 μg/m^3 in PM_{10} concentration was associated with increased prevalence of chronic phlegm (OR, 1.25; 95% CI, 1.06–1.49).
Yano et al. 1986	Kushira, Sakura-jima, and Oura, Japan	1980	2006 F residents (30–59 years)	TSP and SO_2 concentrations in areas affected by volcanic ash (Kushira and Sakurajima) compared to a control area (Oura)	ATS	Exposure to volcanic ash was not significantly associated with increased prevalence of chronic phlegm (or other chronic respiratory symptoms) after control for age, occupational dust exposure, or smoking (0.6% vs. 0.9% in control area; $P > 0.05$).

Table continues next page

Table 16 (*Continued*). Studies of Chronic Phlegm

Citation	Study Location	Study Years	Study Sample	Pollution Variables	Questionnaires Used to Assess Chronic Phlegm	Results
Yu TS et al. 2001	Hong Kong, China	1994–1995	1660 M and F children (8–12 years)	SO_2, NO_2, and RSP concentrations in a high-pollution area (Kwun Tong district) and a low-pollution area (Shatin district)	ATS	Chronic phlegm was more prevalent among children in the high-pollution vs. low-pollution area (OR, 1.84; 95% CI, 1.06–3.19).
Yu IT et al. 2004	Hong Kong, China	Not specified	821 M and F children (8–13 years)	SO_2, NO_2, and RSP concentrations in a high-pollution area (Kwun Tong district) and a low-pollution area (Shatin district)	ATS	Weak evidence of increased prevalence of chronic phlegm was found in high-pollution vs. low-pollution districts: 9% vs. 6%, $P = 0.13$.
Zhang J et al. 1999	Guangzhou, Lanzhou, and Wuhan, China	1985–1988	4108 M and F adults	TSP, NO_x, and SO_2 concentrations in urban (Lanzhou and urban Wuhan) and suburban (Guangzhou and suburban Wuhan) areas	ATS	The prevalence of chronic phlegm was elevated in the most-polluted city (Lanzhou) vs. the least-polluted city (Guangzhou) among men (OR, 12.62; 95% CI, 5.23–30.44) and women (OR, 7.41; 95% CI, 2.24–24.56).
Zhang JF et al. 2002	Chongqing, Guangzhou, Lanzhou, and Wuhan, China	1993–1996	7621 M and F children (5–12 years)	$PM_{2.5}$, $PM_{10–2.5}$, PM_{10}, TSP, SO_2, and NO_x concentrations in urban and suburban areas within each of the cities	ATS	Increased prevalence of chronic phlegm was found among children per 39-μg/m^3 increase in $PM_{2.5}$ concentration (OR, 2.41; 95% CI, 0.85–6.86) and per 42-μg/m^3 increase in $PM_{10–2.5}$ (OR, 2.83; 95% CI, 1.93–4.16).
Zhou W et al. 2001	Shanghai, China	1998	745 M and F taxi and bus drivers or conductors (19–55 years) and 532 M and F controls (unexposed residents)	Traffic-exposed workers and controls within Shanghai	ATS	The prevalence of chronic phlegm was higher among Shanghai taxi and bus drivers than among controls in analyses adjusted for age, smoking status, and indoor air pollution level (OR, 3.9; 95% CI, 2.6–5.8). Prevalence was directly related to duration of employment as a driver.

Studies of Traffic-Related Exposure Three studies evaluated the prevalence of chronic phlegm with respect to residential proximity to major roads in Japan among women of various ages (ranging from 30–59 years, depending on the study) (Nitta et al. 1993; Nakai et al. 1999; Sekine et al 2004). In two studies, the highest level of exposure was residence < 20 m from a major road and the lowest level was residence either 20–150 m or 50–150 m from a major road (Nitta et al. 1993) or in a suburban location (Nakai et al. 1999). Measured ambient concentrations of pollutants were used to corroborate the residential exposure categories. Sekine and colleagues (2004) compared populations living within 20 m of roads that varied in traffic density and measured roadside concentrations of PM_{10} and NO_2. In all three studies, estimated ORs were adjusted to account for major potential confounders such as age, tobacco smoking, housing characteristics, and sociodemographic factors.

The prevalence of chronic phlegm was associated with residential proximity to major roads in all three studies (Figure 64). The largest effects on the prevalence of symptoms were consistently seen in those living closest to busy roads (Nitta et al. 1993; Nakai et al. 1999**)** or near the busiest roads (Sekine et al. 2004). Analyses based on measured pollutant data from Nakai and colleagues (1999) estimated that an increase in the average roadside NO_2 concentration of 10 μg/m^3 was, over the period of the study, associated with a 12% increase in prevalence (OR, 1.12; 95% CI, 1.01–1.25). Analyses based on measured pollutant data from Sekine and colleagues (2004) estimated that increases in the roadside concentrations of 8-year average PM_{10} and 5-year average NO_2 of 10 μg/m^3 were associated with an increase in prevalence of 60% (OR, 1.59; 95% CI, 1.20–2.11) and 16% (OR, 1.16; 95% CI, 1.06–1.26), respectively. However, the prevalence data were collected as long as 7 years before the averaging periods for PM_{10} and NO_2. A fourth study by Shima and colleagues (2003) did not observe higher reported rates of chronic phlegm to be associated with residential proximity to busy roads in either boys or girls 6–9 years of age, but their analyses did not control for risk factors other than sex, such as parental smoking.

Studies of Proximity to Stationary or Mixed Sources

Studies that evaluated the prevalence of chronic phlegm in adults and children with respect to residential proximity to industrial sources were conducted in China (Xu X and Wang L 1993), Taipei,China (Yang CY et al. 1997), and India (Kumar R et al. 2004) (Table 16).

Xu X and Wang L (1993) studied 1576 adult never-smokers aged 40–69 years in 1986. The never-smokers were a subpopulation of a larger sample surveyed, with a 96% response rate. Exposure was classified according to residence in three areas of Beijing: an industrial area, an urban residential area, and a suburban control area. The 5-year annual average SO_2 concentration (1981–1985) was higher in the urban residential area than the industrial area (128 μg/m^3 vs. 57 μg/m^3) and lowest in the suburban area (18 μg/m^3). PM, measured as TSP, had the highest concentration in the industrial area (449 μg/m^3) and the lowest in the suburban area (261 μg/m^3), but there was considerable seasonal variation. The prevalence of chronic phlegm was 60%; the prevalence was 20% higher in the urban residential area than in the control area (Figure 65) (Xu X and Wang L 1993) in analyses that controlled for age, sex, exposure to indoor air pollution and occupational dust, and socioeconomic status. Analyses based on measured pollutant data estimated that increases of 10 μg/m^3 in the average SO_2 and TSP concentrations over the study period were associated with increases in prevalence of 12% (OR, 1.12; 95% CI, 1.01–1.25) and 3% (OR, 1.03; 95% CI, 1.00–1.06), respectively.

Yang CY and colleagues (1997) and Kumar R and associates (2004) also observed increases in the prevalence of chronic phlegm among adults in Taipei,China, and northern India, respectively, that compared an industrialized area and a non-industrialized area. Yang CY and colleagues (1997) observed a 200% increase (OR, 2.01; 95% CI, 1.19–3.42) in the prevalence of chronic phlegm and a 63% increase in that of chronic bronchitis (OR, 1.63; 95% CI, 0.67–2.59) among adult residents of neighborhoods near a petrochemical facility as compared with those in a non-industrialized (control) area (Figure 65) in analyses adjusted for age, tobacco smoking, and socioeconomic factors. Analyses based on measured pollutant data estimated that annual average PM_{10}, SO_2, and NO_2 concentrations were each associated with increased prevalence of chronic bronchitis and chronic phlegm. The ORs for a 10-μg/m^3 increase in pollutant concentration were most pronounced for chronic phlegm: 1.25, 1.39, and 1.50 for PM_{10}, SO_2, and NO_2, respectively.

Kumar R and colleagues (2004) studied residents of an industrial town and those of a non-industrial town. Concentrations of PM and gaseous pollutants measured at various times over 1 year were consistently higher in the industrial town. PM_{10} concentrations were 112.8 μg/m^3, versus 75.8 μg/m^3 in the non-industrial town, but measurements were obtained on only 37 days in the industrial town and 15 days in the reference town. The authors reported an increased prevalence of chronic phlegm in men, but not women, who resided in the industrial town (4.8%, vs. 2.9% in the non-industrial town; $P < 0.05$), in an

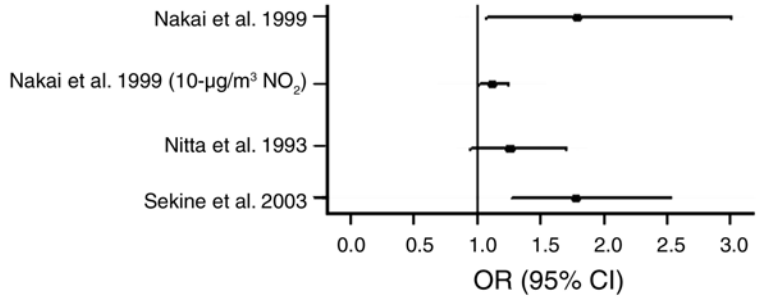

Figure 64. ORs (and 95% CIs) for chronic phlegm among women living within 20 m of a busy road. Y-axis labels give the reference citations. The ORs were estimated relative to women living farther from a busy road (Nitta et al. 1993), within 20 m of less-busy roads (Nakai et al. 1999; Sekine et al. 2004), or within 20 m of less-busy roads per an NO_2 concentration increment of 10 μg/m^3 (Nakai et al. 1999). For study details, see Table 16.

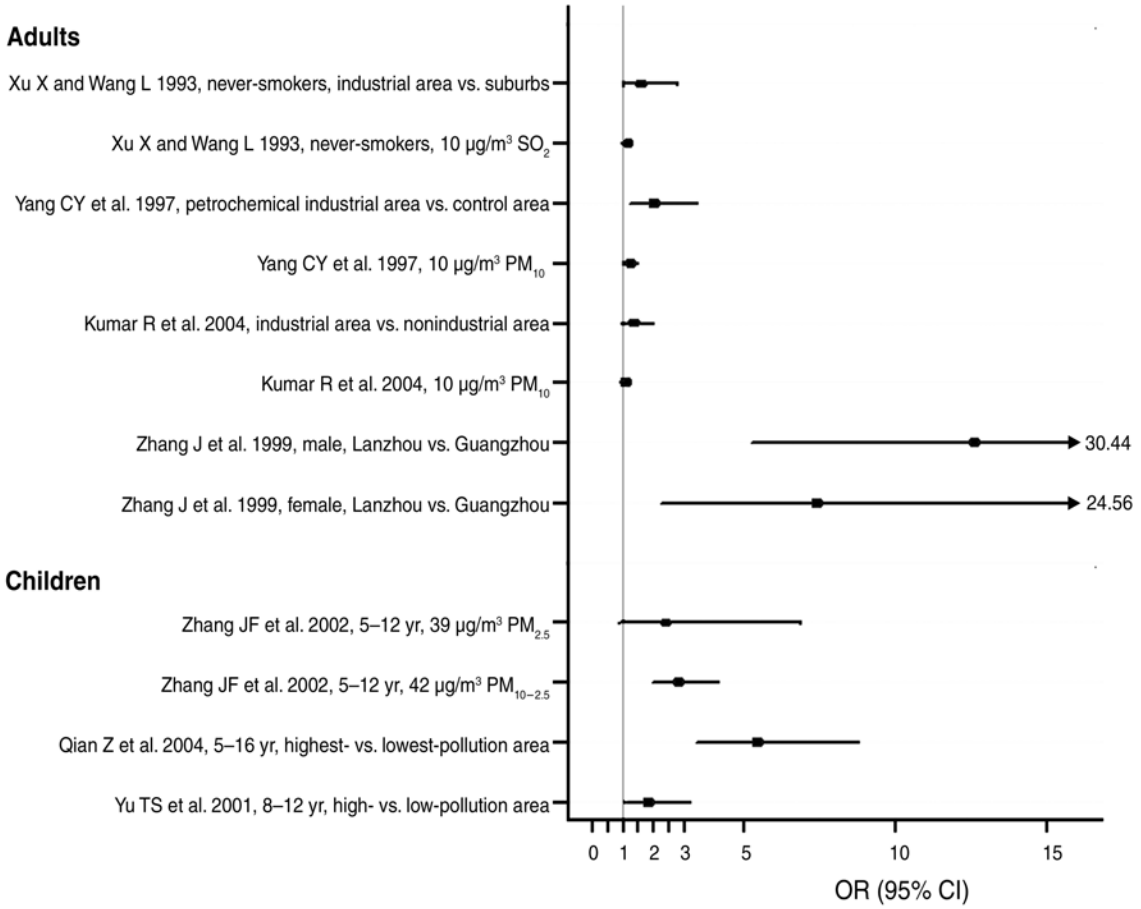

Figure 65. ORs (and 95% CIs) for chronic phlegm in association with stationary or mixed sources of pollutants. Y-axis labels give study information in the following sequence: reference citation; population (in some cases), and areas or locations compared or pollutant increment studied. Y-axis labels in bold type specify the age group. For study details, see Table 16.

analysis without adjustment for potential confounders (e.g., the use of biomass fuels for cooking [more common in the non-industrial town], vs. the cleaner fuel liquid petroleum [more common in the industrial town]). In a logistic regression analysis that did control for potential confounders, the combined prevalence of chronic respiratory symptoms (phlegm, cough, wheeze, and shortness of breath) was increased in the industrial town (OR, 1.5; 95% CI, 1.2–1.8).

Four studies — three in China (Zhang J et al. 1999, 2002; Qian Z et al. 2004) and one in Hong Kong (Yu TS et al. 2001) — estimated the effect of residence in polluted urban areas on the prevalence of chronic phlegm. Pollution was presumed to be the result of emissions from a variety of sources, both stationary and mobile.

Zhang J and colleagues (1999) studied 4108 adults in 1988 in four districts of three Chinese cities: Guangzhou, Wuhan, and Lanzhou. Four-year mean (1985–1988) concentrations of TSP, NO_x, and SO_2 calculated from ambient monitoring data in the least- and most-polluted cities (Guangzhou and Lanzhou, respectively) ranged from 296–1067 μg/m^3 TSP, 89–92 μg/m^3 NO_2, and 110–121 μg/m^3 SO_2. Prevalence of persistent cough and phlegm and self-reported bronchitis was elevated in both men and women in Lanzhou relative to Guangzhou in analyses that controlled for multiple potential confounders including tobacco smoking and indoor air pollution from coal burning (Figure 65).

Respiratory symptoms in children were investigated in three studies. In the mid-1990s, Zhang JF and colleagues (2002) studied 7621 schoolchildren, 5–12 years of age, in urban and suburban districts of Guangzhou, Wuhan, and Lanzhou, as well as Chongqing. Each district had 2-year (1995–1996) average PM_{10}, $PM_{2.5}$, and $PM_{10–2.5}$ concentrations and 4-year average TSP, NO_x, and SO_2 concentrations. Across all 8 districts studied, an interquartile range increment in both $PM_{2.5}$ and $PM_{10–2.5}$, but not in the gaseous pollutants, was associated with an increased prevalence of chronic phlegm (defined as phlegm for at least 1 month/year) in analyses adjusted for indoor burning of solid fuel, parental smoking, and socioeconomic status (Figure 65). Chronic phlegm was also associated with interquartile range increments in long-term average concentrations of PM, NO_x, and SO_2 in analyses that compared urban and suburban districts within each city. The ORs for developing chronic phlegm ranged from 1.8 (for the increase in $PM_{10–2.5}$ concentration) to 1.3 (for the increase in NO_x concentration), but none were significant at the $P = 0.05$ level, and neither SEs nor CIs were presented.

Qian Z and colleagues (2004) published additional analyses of the data collected by Zhang JF and associates (2002), in which they grouped the eight districts into four clusters based on the concentrations of both PM and gaseous pollutants. The prevalence of phlegm in the most-polluted cluster was 5 times that in the least-polluted cluster (Figure 65).

Yu TS and colleagues (2001) studied 1660 Hong Kong children, aged 8–12 years, who resided in two urban districts in 1995. Annual average concentrations of SO_2, NO_2, and respirable suspended particles (RSP) were 22.8, 58.5, and 57.6 μg/m^3, respectively, in the three districts. The prevalence of chronic phlegm was increased in the more-polluted district compared with the less-polluted district in analyses that controlled for parental tobacco smoking and indoor air pollution from cooking or other sources (OR, 1.84; 95% CI, 1.06–3.19). This study corroborates observations in three earlier investigations of the prevalence of chronic respiratory disease in schoolchildren in two other Hong Kong districts that differed in ambient concentrations of air pollution (Ong et al. 1991; Tam et al. 1994; Peters J et al. 1996). Peters J and colleagues (1996) estimated the effect on the prevalence of respiratory disease in the two Hong Kong districts that was associated with a reduction in sulfur concentration in fuels, implemented in Hong Kong in 1990, which resulted in decreases in ambient SO_2 concentrations, changes in the elemental composition of PM, and reductions in long-term average mortality rates (Hedley et al. 2002; Wong CM et al. 2009). After adjustment for potential confounders, residence in the high-pollution district versus the low-pollution district in 1989–1990, prior to the reduction of sulfur concentration in fuels, was associated with an 11% increase in the prevalence of chronic phlegm (OR, 1.11; 95% CI, 0.96–1.30). By 1991, air quality had improved in both districts, with greater improvements in the most-polluted district and no significant difference in the reported prevalence of chronic phlegm (OR, 0.88; 95% CI, 0.68–1.13).

Discussion COPD in adults is among the most important of the outcomes considered in this review, because it is associated with shortened healthy life expectancy. In studies that controlled for major potential confounding factors (including tobacco smoking and indoor air pollution from burning solid fuels), the prevalence of chronic phlegm was associated with exposure to combustion-source air pollution both in qualitative comparisons among areas with differing levels of pollution and in comparisons of measured levels of air pollution. Associations were observed with both PM, measured as PM_{10}, and gaseous combustion-source pollutants, such as SO_2 and NO_2. Similar associations with pollution, after controlling for smoking, have been reported in many surveys in North America and Europe, including some in which the sources

and concentrations of pollutants were similar to those in some Asian cities in this review (Pope and Dockery 1999; ATS 1996a,b).

The increased prevalence of chronic phlegm in children, although not an indicator of chronic bronchitis or COPD (both of which take longer to develop), may be correlated with repeated respiratory infections, which in turn may result in reduced pulmonary function (Johnston et al. 1998). It has also been hypothesized that repeated respiratory infections in childhood and adolescence increase the risk of developing COPD in adulthood, but this question remains unresolved (Reid and Fairbairn 1958; Reid 1964; Vestbo and Hogg 2006). However, reduced lung function in childhood and early adulthood is associated with an increased risk of developing COPD later in life (Fletcher and Peto 1977; Rennard and Vestbo 2008).

Pulmonary Function

Twenty-nine studies evaluated effects of long-term exposure to air pollution on pulmonary function. Both children and adults were studied, and the results for each are described separately below, with particular emphasis on results regarding adults who were nonsmokers.

Studies of Children Pulmonary function in children 15 years of age or younger was assessed in 12 studies (Table 17). Most included relatively large numbers of children, with study populations ranging from approximately 180 to approximately 1300. The studies come largely from East and Southeast Asia, with studies from Hong Kong (4 studies), Taipei,China (2), and China (1) and additional single studies from Singapore, South Korea, and Bangkok. Within each study, region-specific pollution gradients (either measured or estimated as urban–rural gradients) were considered the pollution source. Pulmonary function measurements usually included FEV_1, forced vital capacity (FVC) and forced expiratory flow during the middle half of FVC ($FEF_{25–75}$), as well as FEV/FVC in some cases. The results were typically adjusted for age, sex, and height, as well as exposure to ETS if those data were available. In addition, bronchial hyperresponsiveness (BHR) was assessed in three studies, and changes in maximal oxygen uptake (VO_2max) in one study.

In all, 11 of the 12 studies were judged to be adequate to reach a conclusion as to the association between air pollution and pulmonary function. The one excluded study was by Kamat and colleagues (reported in 1980 and 1992). In the 1980 article, the adjustment of the pulmonary function data by height and age appeared to be applied inconsistently, and in the 1992 article, fewer than 15 children were studied in each exposure region, too few to yield interpretable results. Few studies explicitly addressed potential confounding due to passive smoking, although in studies in which these data were available, passive or household smoking did not appear to influence the results substantially. In studies that reported concentrations of ambient air pollution, PM concentrations were generally 1.5 to 2 times as high in the more-polluted areas than as in less-polluted areas. Concentrations of gaseous pollutants such as SO_2 and NO_2 were 5 to 10 times as high in the more-polluted regions.

Most studies of pulmonary function in children observed adverse effects of air pollution exposure on pulmonary function, defined variously in the different studies. These estimates did not appear to change with control for passive smoking exposure. Goh and colleagues (1986) reported on 684 children from Singapore with an average age of 10.7 years. The authors found a modest but significant reduction in FEV_1 and FVC values among boys living in the industrial area compared with the urban or rural areas. No significant differences were seen in girls, and data for neither boys nor girls were adjusted for passive smoking exposure. The greatest differences in pollution concentrations were found for SO_2; there was little difference in TSP concentrations across the areas surveyed.

Hsiue and colleagues (1991) studied 382 children, aged 9–11 years, in four regions of Taipei,China, one of which was downwind from wire-reclamation and incineration facilities. They reported pollution gradients for NO_2 and SO_2 for which the highest exposure was approximately 9 to 10 times the lowest exposure, as well as evidence for both obstructive and restrictive lung-function deficits after adjustment for passive smoke exposure in the home.

Tam and colleagues (1994) measured BHR in 423 children, aged 10–12 years, in two Hong Kong districts. Average concentrations of SO_2 and NO_2 varied by a factor of 13 between the two districts, and RSP concentrations were about twice as high in one district as in the other. Although no significant differences in standard spirometric measures were found, mild-to-moderate increases in airway reactivity were 4 times as common among nonasthmatic children living in the more-polluted district than the less-polluted one, after controlling for passive smoking.

Studies of Adults Fifteen studies reported pulmonary function data obtained using a standardized protocol and some estimate of pollutant exposure (Table 18). Seven of the studies are from India, 1 from South Korea, three from Thailand, two from Japan, and two from China. In studies of adults, the potential for confounding by smoking is an important limitation when trying to estimate the effects of long-term pollution on the level of pulmonary function.

Table 17. Studies of Pulmonary Function in Children

Citation	Study Location	Study Years	Study Sample of Children	Pollution Variables	PFT Outcomes	Results
Goh et al. 1986	Singapore	1982–1983	684 M and F (6–14 years)	TSP, SO_2, and NO_2 concentrations in industrial (Jurong), urban (Queenstown), and rural (Sembawang) areas	FEV_1 and FVC	Residence in industrial district (vs. urban or rural) associated with reduction in FEV_1 and FVC in boys. No control for ETS.
He QC et al. 1993	Wuhan, China	1988	602 M and F (7–13 years)	TSP and NO_x concentrations in high-pollution urban and low-pollution suburban areas within Wuhan	FEV_1 and FVC	Decrements in FVC of 3–8.5% in polluted region vs. less-polluted region; decrements in FEV_1 of 0.8–4.5% over range of age and height in polluted region. The measures from 1988 represent chronic long-term lifetime exposure. No control for ETS.
Hsiue et al. 1991	Taipei, China	Not specified	382 M and F (9–11 years)	SO_2 and NO_2 concentrations in three polluted areas (Wanli, Chiading, Hunei) and a control area (Annan)	FEV_1 and FVC	Pulmonary obstruction varied across the four areas. Prevalence of abnormal FVC ranged from 4.1–16.5%; abnormal FEV_1, 7.2–11.9%. Range of prevalence of pattern: 4.1–11.9%.
Jang et al. 2003	Yeocheon, Yeosu, and Namwon, South Korea	Not specified	670 M and F (10–13 years)	SO_2, NO_2, and O_3 concentrations in two areas with varying proximity to chemical-factory emissions (Yeochun and Yeosu) and a rural area (Namwon)	FEV_1, FVC, and BHR (defined as a 20% decrease in FEV_1)	No significant difference in PFTs but prevalence of BHR ranged from 32–45% vs. 32% over distance from source. No control for ETS.
Kamat et al. 1980	Mumbai, India	1977–1979	1395 M and F (7–19 years)	SO_2, SPM, and NO_2 concentrations in high-to-low pollution urban areas (Lalbaug, Chembur, and Kaur) and a rural area	FEV_1 and FVC	In all of the urban districts, low pollution was associated with higher lung function in children of all ages studied. In the rural district, however, lung function was significantly lower despite lower pollution. FVC measurements appear too low, making the reliability of PFT measurements questionable.

Table continues next page

Table 17 (*Continued*). Studies of Pulmonary Function in Children

Citation	Study Location	Study Years	Study Sample	Pollution Variables	PFT Outcomes	Results
Kamat et al. 1992	Mumbai, India	1988–1989	585 M and F children (7–19 years)	SO_2, SPM, and NO_2 concentrations in three urban areas (Parel, Dadar, and Maravali) and a rural area (Deonar)	FEV_1 and FVC	Adjusted FEV_1 and FVC values inversely associated. Measures of expiratory flow were lower in the two districts with higher exposures vs. the other two districts ($P > 0.05$).
						PFT protocol poorly described. Too few subjects in age group of interest. No control for ETS.
Langkulsen et al. 2006	Bangkok, Thailand	2004	844 M and F children (10–15 years)	PM_{10} concentrations in three high-pollution areas and a low-pollution area within Bangkok	FEV_1 and FVC	OR for abnormal PFT in areas with polluted roadsides or general residential areas vs. less-polluted areas: 1.4 (95% CI, 0.99–1.1). Significant among junior-high students only.
						Modest effect limited to one group, after control for ETS.
Tam et al. 1994.	Hong Kong, China	1989–1990	423 M and F children (10–12 years)	SO_2, NO_2, and RSP concentrations in a high-pollution area (Kwa Tsing district) and a low-pollution area (southern district)	FEV_1 and FVC	No significant difference between districts in baseline PFTs. Mild-to-moderate BHR among nonasthmatic children in the high-pollution district vs. the low-pollution district: 11% vs. 4%.
Wang JY et al. 1992	Taipei, China	Not specified	178 M and F children (12 years)	SO_2 and NO_2 concentrations in a high-pollution area (Wanli) and a low-pollution area (Tainan)	FEV_1 and FVC	For the high- and low-pollution districts, prevalence of abnormal FVC was 15.6% and 6.5% and of abnormal FEV_1 was 17.5% and 3.2%. BHR reported in 9 of 26 vs. 1 of 28 responders.
Wong CM et al. 1998	Hong Kong, China	1990–1991	320 M and F children (9–12 years)	SO_2, NO_2, and RSP concentrations (following sulfur-reduction measures) in a high-pollution area (Kwa Tsing district) and a low-pollution area (southern district)	BHR (defined as a 20% decrease in FEV_1)	Repeated measures of BHR prevalence among 4th graders in 1990, 28.9% and 21.1%; 4th graders in 1991, 27.9% and 14.1%; and 5th graders in 1991, 15.7%, and 9.7%; and again in 1991, 17.8% and 11.1%. Little evidence of differences in BHR prevalence in 1992.
						Findings suggest reversibility of BHR with pollution decline. No control for ETS.
Yu IT et al. 2004	Hong Kong, China	Not specified	821 M and F children (8–13 years)	SO_2, NO_2, and RSP concentrations in a high-pollution area (Kwun Tong district) and a low-pollution area (Shatin district)	FEV_1 and FVC	Difference in adjusted VO_2max between districts: 1.9 mL/kg of body-surface area/min.
Yu TS et al. 2001	Hong Kong, China	1994–1995	1294 M and F children (8–12 years)	SO_2, NO_2, and RSP concentrations in a high-pollution area (Kwun Tong district) and a low-pollution area (Shatin district)	FEV_1, FVC and $FEF_{25–75}$	Difference in PFTs between districts greater among girls than boys. In polluted vs. less-polluted district, mean FVC was 50 mL vs. 29 mL and mean FEV_1 was 83 mL vs. 54 mL. Similar effects were reported for $FEF_{25–75}$.

Table 18. Studies of Pulmonary Function in Adults

Citation	Study Location	Study Years	Study Sample	Pollution Variable	PFT Outcomes	Results
Chhabra et al. 2001	Delhi, India	1988–1998	4171 M and F residents (> 17 years)	TSP, SO_2, and NO_2 concentrations in high-pollution and low-pollution zones within Delhi	FEV_1, FVC, FEF, PEFR, and $MMEF_{25–75}$	Difference in average predicted PFT between the high- and low-pollution areas, among men and women (respectively): FVC, 3.7% and 2.9%; FEV_1, 5.1% and 6.6%; FEF, 8.3% and 4.8%; PEFR, 7.5% and 2.0%; and $MMEF_{25–75}$, 8.32% and 4.8%.
Hong YC et al. 2005	Incheon, South Korea	2002	293 M and F college students	Distance of residence from road (< 50, 50–100, 100–500, and > 500 m)	FEV_1, FVC, and $FEF_{25–75}$	Personal exposure to NO_2 was associated with decreased FEV_1 ($P = 0.07$) and $FEF_{25–75}$ ($P = 0.05$).
Ingle et al. 2005	Jalgaon, India	2003–2004	60 M traffic policemen and 60 M controls (other workers)	Traffic policemen compared to occupational controls within Jalgoan	FEV_1 and FVC	Spirometric analysis showed significant respiratory impairment in traffic policemen vs. controls. Results difficult to interpret because subjects were grouped by height category; those 3 cm taller had a predicted FVC 500 mL lower.
Kamat et al. 1980	Mumbai, India	1977–1979	2008 M and F residents (> 19 years)	SO_2, SPM, and NO_2 concentrations in high-to-low pollution urban areas (Lalbaug, Chembur and Kaur) and a rural area	FEV_1 and FVC	Air pollution was associated with reduced lung function in subjects of all ages. In the rural area, however, lung function was significantly lower, despite lower pollution concentrations. Results not adjusted for smoking status.
Kamat et al. 1992	Mumbai, India	1988–1989	821 M and F residents (> 19 years)	SO_2, SPM, and NO_2 concentrations in three urban areas (Parel, Dadar, and Maravali) and a rural area (Deonar)	FEV_1 and FVC	No significant differences in lung function associated with residence in polluted areas. Prevalence of $FEV_1/FVC < 70\%$ of the predicted value was associated with residence in polluted areas in an analysis based on a small number of subjects. Differences in socioeconomic status and ethnicity among the four areas not controlled.
Karita et al. 2001	Bangkok and Ayutthaya, Thailand	1998–1999	78 M traffic policemen and 128 M non-traffic policemen (unspecified age range)	Traffic policemen in an urban area (Bangkok) compared to general policemen within the urban area and a rural area (Ayutthaya)	FEV_1 and FVC	After adjustment for age, height, and smoking status, a difference in FEV_1 of ~5% was found between traffic and general police. ~ 45% of subjects in each group were smokers.

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Table 18 (*Continued*). Studies of Pulmonary Function in Adults

Citation	Study Location	Study Years	Study Sample	Pollution Variable	PFT Outcomes	Results
Kumar KS et al. 2000	Hyderabad, India	1991	216 M residents (18–60 years)	SPM, SO_2, and NO_2 concentrations in industrial (Balanagar), commercial (Uppal), and residential (Tarnaka) districts	FEV_1 and FVC	No significant differences in level of pulmonary function among districts or in association with average air pollution concentrations No adjustment for smoking.
Kumar R et al. 2004	Mandi Gobindgarh and, Morinda, India	1999–2001	3603 M and F residents (> 15 years)	TSP, PM_{10}, NO_x, SO_2, CO, and O_3 concentrations in a high-pollution area (Mandi Gobindgarh) and a low-pollution area (Morinda)	FEV_1 and FVC	Prevalence of ventilatory defects ~2× higher in industrial town than non-industrial town after adjustment for age, sex, education, occupation, income, and smoking status. "Ventilatory defect" defined as 1.645× SE of estimated predictive value. Potential residual confounding by smoking status.
Maeda et al. 1991	Tokyo, Japan	1987–1990	2600 F residents (30–60 years)	NO_2 gradient based on distance of residence from road in Sumida Ward, Tokyo (< 20 m and 20–150 m) and a residential suburb of Tokyo (Higashiyamato)	FEV_1 and FVC	No consistent differences in FEV_1 and FVC. Results not adjusted for smoking status.
Nakai et al. 1999	Tokyo, Japan	1987–1990	444 F residents (39–50 years)	NO_2 gradient based on distance of residence from road in Sumida Ward, Tokyo (< 20 m and 20–150 m) and a residential suburb of Tokyo (Higashiyamato)	FEV_1 and FVC	Repeated pulmonary function measurements showed small reductions in FEV_1 and FVC associated with residence near busy roads. Extension of Maeda et al. 1991. Results not adjusted for smoking status.
Pothikamjorn et al. 2002	Bangkok, Thailand	1999	290 F students (15–18 years)	PM_{10} concentrations outside schools located in the center of Bangkok	FEV_1, FVC, and $FEF_{25–75}$	Small differences in $FEF_{25–75}$ in association with pollution levels ($P < 0.05$); no other effects on PFT. No details on PFT methods or smoking status (except that 30% of households had a resident smoker).

Table continues next page

Table 18 (*Continued*). Studies of Pulmonary Function in Adults

Citation	Study Location	Study Years	Study Sample	Pollution Variable	PFT Outcomes	Results
Rao et al. 1992	Ahmedabad, India	Not specified	203 Shopkeepers	NO_2 concentrations at six traffic junctions grouped into three levels of exposure	FEV_1, FVC, and FEF	In high-exposure and low-exposure groups (respectively), FVC was 89.2% and 92.2% (nonsignificant); FEV_1 was 81.1% and 85% ($P < 0.05$); and FEF was 85.2% and 95.9% ($P < 0.05$). ~15% of the highly exposed shopkeepers had clinically significant airway obstruction.
Wongsurakiat et al. 1999	Bangkok, Thailand	1996–1997	620 M traffic policemen (20–60 years) and 303 M controls (students and other workers)	Traffic policemen (with and without protective masks) and controls within Bangkok	FEV_1 and FVC	In traffic policemen (without protective masks) and controls (respectively), FEV_1 was 3.29% and 3.43% ($P < 0.01$); and FVC was 3.86 % and 3.98% ($P = 0.047$).
Xu XP et al. 1991	Beijing, China	1986	1440 M and F residents (40–69 years)	TSP and SO_2 concentrations in residential, industrial, and suburban areas within Beijing	FEV_1 and FVC	Living in the residential and industrial areas was associated with a reduced FEV_1 (69-mL and 62-mL decline) and FVC (257-mL and 177-mL decline).
Zhou W et al. 2001	Shanghai, China	1998	377 M and F taxi and bus drivers or conductors (19–55 years) and 65 M and F controls (unexposed residents)	Traffic-exposed workers and controls within Shanghai	FEV_1 and FVC	Pollution reported to have no significant effect on FEV_1 and FVC, but no specific data reported and cannot separate out non-smokers. No useful PFT data.

For each study, we assessed the degree to which smoking was taken into account, either by separately reporting the results according to smoking status or by statistical adjustment. Most of the studies did not report the methods or results in sufficient detail to determine whether the potential for confounding due to smoking could be excluded as an explanation of the reported results.

Some studies suggest that there are adverse effects on lung function in adults, independent of the effects of tobacco smoking. Three of the eight studies from India provided data on changes in pulmonary function associated with relatively high concentrations of TSP and NO_2 or SO_2. Chhabra and colleagues (2001) studied approximately 4171 asymptomatic nonsmokers who lived in two districts in Delhi. They reported a 3% to 6% difference in levels of FVC, FEV_1, and maximum midexpiratory flow during the middle half of FVC ($MMEF_{25–75}$) among both male and female residents of the more-polluted district compared with values for residents of the less-polluted district, after adjustment for socioeconomic status. The difference in ambient concentrations between high- and low-pollution areas was greater for the gaseous pollutants (for which the high concentrations were twice the low concentrations) than for TSP (for which the high concentrations were 1.3 times the low concentrations).

Rao and colleagues (1992) studied 203 shopkeepers who were nonsmokers, comparing those with high exposures to traffic and those with lower exposures. Measured NO_2 concentrations in the high-exposure areas were twice those in the lower-exposure areas. Adjusted FEV_1 values were 4% lower in the highly exposed group, and roughly 15% of the shopkeepers in the highly exposed group had levels of pulmonary function consistent with clinically significant obstruction of the airways. Finally, Kumar R and colleagues (2004) studied 3603 residents, aged > 15 years, in two towns: one industrial and one non-industrial. Concentrations of PM (measured as TSP and PM_{10}) and gaseous pollutants (NO_x, sulfur oxides, and CO) were measured over a year; the concentrations in the industrial town were 1.5 to 8 times the concentrations in the non-industrial town. The authors reported that the frequency of ventilatory defects in the industrial town was approximately twice that in the non-industrial town, a difference equivalent to a 1-SD unit below the estimated predicted value, after adjustment for age, sex, education level, occupation, income, method of home heating, and smoking. The authors noted the potential for residual confounding by factors related to socioeconomic status.

Effects of exposure to traffic-related air pollution were also reported in a Thai study of traffic policemen who were nonsmokers (Wongsurakiat et al. 1999). Policemen with more than 10 years of heavy exposure to vehicular traffic were 1.7 times as likely to have an FEV_1 level below 80% of the predicted value than less-exposed policemen and other controls. An apparent protective effect of regular use of masks was noted but not confirmed; the number of nonusers was small.

Xu XP and colleagues (1991) compared 1400 adult nonsmokers, aged 40–69 years, living in one of three districts in Beijing. The TSP concentrations among the three districts varied by a factor of 1.7, and the SO_2 concentrations varied by a factor of 7.1; in the most industrial district, the annual average TSP and SO_2 concentrations were 449 $\mu g/m^3$ and 261 $\mu g/m^3$, respectively. In a multivariate analysis with adjustment for a variety of known factors, the authors found significantly reduced FEV_1 and FVC values in residents of the most-polluted district, with a decrease in FVC of 131 mL per log unit of SO_2 and of 478 mL per log unit of TSP. Smaller changes were noted for FEV_1. Issues of confounding by home heating and potential residual smoking effects were not completely resolved in a companion study reported on later (Xu X 1998).

Discussion In general, because these studies are cross-sectional and because the air pollution exposures were estimated for whole geographic areas rather than for individuals, it is difficult to reach firm conclusions as to the impact of ambient pollution on pulmonary function. Cross-sectional studies suggest detrimental effects of exposure to air pollution on children's lung function, providing snapshots of events that are part of a dynamic process of how lung growth and development may be affected. Longitudinal studies are needed to determine whether the cross-sectional associations with air pollution represent a slower-than-normal growth of lung function that results in permanent deficits (and might subsequently lead to an accelerated decline in lung function in adulthood) or, as some studies in Western countries suggest, a transient worsening of pulmonary function with recovery as pollution levels improve (Avol et al. 2001; Sugiri et al. 2006).

Few studies of adults have used acceptable methods with regard to testing protocols or controlling for the effects of tobacco smoking. In a small subset of the studies that controlled for confounding by tobacco smoke, either through restricting the study population to people who did not smoke or by adjustment, there were decrements in pulmonary function; it was difficult to determine, however, whether these decrements were the result of long-term exposure in adulthood or reduced growth of lung function in childhood. There is also the potential for residual confounding, such as confounding due to differences among factors related to socioeconomic status in studies that compare

rates in an industrialized or urban area with those in a more rural area. These problems are encountered in cross-sectional studies in Western countries as well as in Asia.

Götschi and colleagues (2008) recently reviewed the literature on pulmonary function and long-term exposure to air pollution published over the past 20 years. They identified 58 studies of adults or children, but only 9 in Asian countries, all of which were included in this review. They focused on the 35 studies that included four or more locales; only two of these studies were in Asia (Nakai et al. 1999; Sekine et al. 2004). They noted the methodologic diversity of the studies and the challenges this posed for quantitatively summarizing the results, but concluded that the available evidence was consistent with air pollution being associated with adverse effects on lung growth in children and reduced lung function in adults; the latter conclusion was based largely on the same types of cross-sectional studies reviewed above. They also emphasized the need to identify the timing of exposure with regard to effects on lung function and the need to address a range of potential modifiers of the effect of air pollution on lung function, including differences among individuals in susceptibility and characteristics of the air pollution mix.

Longitudinal cohort studies or panel studies that use longitudinal follow-up designs with adequate measures of ambient and personal pollutant exposures are needed to assess the growth and development of pulmonary function in children, as well as to assess the rate of decline of pulmonary function in adults, who will continue to be exposed to ambient pollutants generated both by stationary and mobile sources. Furthermore, it would be useful to identify places in which improvements are scheduled that will reduce ambient exposures, to aid in characterizing the reversibility of lung function deficits as air pollution exposures decrease.

Asthma and Wheeze

Literature searches within the PAPA–SAN and APED databases yielded 48 candidate papers addressing asthma or wheeze published through August 2007 with the full text available in English. The eligibility criteria were that (1) the study contained information about the statistical association between indicators of air pollution and measures of the prevalence or incidence of wheeze (the cardinal symptom of asthma) or a diagnosis of asthma, (2) the study was population based, and (3) estimates were adjusted for age and sex, at least. After a more detailed inspection of the 48 articles, 6 were excluded: 4 because there was inadequate information on exposure or outcome (Hsieh and Shen 1988; Kagamimori et al. 1990; Lahiri et al. 2000; Tsai HJ et al. 2006a), and 2 because they were based on a worker population with occupational exposures (Tamura et al. 2003; Karita et al. 2004). The degree of adjustment for confounders varied; we elected to include studies without statistical adjustment, provided that the comparison groups were balanced with respect to age and sex. All selected articles were sufficiently clearly written to understand what the investigators had done.

The 42 studies are summarized in Table 19, with several characteristics and quantitative results listed. If ORs were given, they were tabulated and presented as forest plots. The largest number of studies was from Taipei,China (13), followed by Japan (10), China (6), Hong Kong (4), India (4), Indonesia (2), and one each from South Korea, Singapore, and Thailand. The publication date was prior to 1990 for 4 articles, between 1990 and 1999 for 14, and between 2000 and 2007 for 24. There were 30 studies of children and 14 of adults (with 2 studies addressing both). A total of 19 studies reported results for females, 13 for males, and 28 for both sexes combined.

To ascertain the presence of asthma, the studies used conventional and widely accepted methods that are based on questionnaires about wheeze, the principal symptom of asthma, or a diagnosis of asthma. Asthma is a relapsing–remitting disease with a large range of individual patterns of frequency, duration, and intensity of wheezy dyspnea. Questions about wheeze generally focus on a limited time period for possible symptoms, generally within the recent past (e.g., the past 12 months), to ensure that recollection is feasible. It is also common to ask whether the respondent has "ever" had a given symptom, in which case the timing is unclear unless defined by the young age of the subject (e.g., < 5 years). Most studies we identified also asked about receipt of a diagnosis of "asthma," sometimes qualifying it as being "doctor-diagnosed." Although this question clearly specifies asthma, the particulars of diagnosing the disease vary considerably. Therefore, the prevalence of asthma symptoms will tend to be underestimated, because not all people with symptoms consult a doctor and because the diagnosis of asthma may not be given until wheezing has been shown to be recurrent or persistent.

Standardized questionnaires have been developed by the ATS, the British Medical Research Council (BMRC), the European Respiratory Health Survey, the International Study of Asthma and Allergies in Childhood (ISAAC), and the WHO. The majority of the studies reviewed used one of these questionnaires outright or as a basis of a study-specific questionnaire. It was not always clear how faithfully the study had followed the standardized questionnaire. All studies used a systematic approach that permitted unbiased comparisons of the groups of interest. To simplify the

Table 19. Studies of Asthma and Wheeze

Citation	Study Location	Study Year[a]	Study Sample[b]	Designs[c]	Pollution Variables or Comparisons	Parameters, Outcomes, and Questionnaires[d]	Summary Findings and Adjustments for Confounders[e]
Aggarwal et al. 2006	Four urban or rural centers in Chandigarh, Delhi, and Kanpur, Bangalore	NR	73,605 M+F adults (≥ 15 years)	Between cities (n = 4)	Urban vs. rural	Prevalence, A-r, IUATLD	Significant association Adjustment for D, S, Sm, and O
Chen PC et al. 1998	Six communities in Taipei,China	1994–1997	4860 M+F primary-school children	Between cities (n = 6)	Urban vs. rural	Prevalence, W-rns and A-rns, WHO	Significant association Adjustment for D, S, I, Sm, and O
Chhabra et al. 2001	Delhi, India	1988–1998	4171 M, F adults living in two areas with different pollution concentrations	Within city	High vs. low	Prevalence, W-rns and A-rns	Investigator reported no significant association (data not shown) Adjustment for D, S, I, Sm, and O
Chhabra et al. 1999	Delhi, India	NR	21,367 M+F children (5–17 years)	Within city	High vs. low	Prevalence, W-r, ATS and BMRC	Investigator reported no significant association (data not shown) Adjustment for D, S, I, Sm, and O
Duki et al. 2003	Jakarta, Bandar, and Lampung, Indonesia	1996–1997	16,663 Pairs of junior-high students and their mothers; children M+F, adults F	Between cities (n = 8) and proximity to traffic	NO_2 concentration; < 20 m vs. ≥ 20 m from road	Prevalence, W-r, ATS	Significant associations with NO_2, no significant associations with living < 20 m from road Adjustment for D and Sm. ORs could not be extracted

Table continues next page

[a] NR indicates that the study year was not reported.

[b] M+F indicates that the data for males and females were combined; M, F indicates that the data for males and females were reported separately.

[c] If several designs were used, the design associated with the reported adjusted effect estimates is listed.

[d] Possible parameters were incidence, prevalence, exacerbation, and trends. Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), or A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting is unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), or W-rns (wheeze symptoms whose time of reporting is unspecified or not recent). The questionnaire was the standardized questionnaire used to assess asthma and wheeze in the study, if any.

[e] Summary findings listed include any positive statistically significant association (noted as "significant association"). Any significant negative association is specifically listed as such. Adjustments for confounders included adjustments for demographic characteristics (D), socioeconomic characteristics (S), indoor air pollution (I), smoking status (Sm), and other variables (O). Comments are also listed, if pertinent.

Table 19 (*Continued*). Studies of Asthma and Wheeze

Citation	Study Location	Study Year[a]	Study Sample[b]	Designs[c]	Pollution Variables or Comparisons	Parameters, Outcomes, and Questionnaires[d]	Summary Findings and Adjustments for Confounders[e]
Goh et al. 1986	Singapore	1983	3216 M+F primary-school children	Between cities	Urban vs. rural vs. industrial	Prevalence, W-r ,W-rns, A-r, and A-rns	Investigator reported no significant association (data not shown) Adjustment for D, by design
Guo et al. 1999	Taipei,China	1995	331,686 M, F nonsmoker children	Between cities (nationwide)	Traffic-related pollution, stationary-source pollution related to fossil-fuel combustion, and CO, NO_x, O_3, PM_{10}, and SO_2	Prevalence, W-r and A-rns	Significant associations with traffic-related pollution, and significant negative associations with stationary sources and fossil-fuel combustion, and significant association with NO_x and CO but not O_3, PM_{10}, or SO_2 Adjustment for D, S, Sm, and O. ORs could not be extracted
Ho et al. 2007	Taipei,China	1995–1996	64,660 M, F junior-high students	Between cities	High vs. low concentrations of CO, NO_2, NO, O_3 and PM_{10}	Prevalence, W-r and A-r, ISAAC, and other	Significant associations Adjustment for D, S, I, Sm, and O
Hong CY et al. 2004a	Kerinci, SP7, and Pelalawan, Indonesia	2001	382 M+F children	Between cities (n = 2)	Urban vs. rural	Prevalence, W-rns, ISAAC	No significant association Adjustment for D, S, I, Sm
Hong SJ et al. 2004b	Seoul and 8 provincial centers (Suwon, Chungju, Chunju, Changwon, Chunchon, Cheju, Ulsan, and Ansan), South Korea	1995–2000	15,214 M, F, and M+F adolescents	Between cities	Urban vs. rural and industrial vs. rural	Prevalence, W-r, ISAAC	No significant associations in 1995 or 2000 Adjustment for D, by design

Table continues next page

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[c] If several designs were used, the design associated with the reported adjusted effect estimates is listed.

[d] Possible parameters were incidence, prevalence, exacerbation, and trends. Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), or A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting is unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), or W-rns (wheeze symptoms whose time of reporting is unspecified or not recent). The questionnaire was the standardized questionnaire used to assess asthma and wheeze in the study, if any.

[e] Summary findings listed include any positive statistically significant association (noted as "significant association"). Any significant negative association is specifically listed as such. Adjustments for confounders included adjustments for demographic characteristics (D), socioeconomic characteristics (S), indoor air pollution (I), smoking status (Sm), and other variables (O). Comments are also listed, if pertinent.

Table 19 (*Continued*). Studies of Asthma and Wheeze

Citation	Study Location	Study Year[a]	Study Sample[b]	Designs[c]	Pollution Variables or Comparisons	Parameters, Outcomes, and Questionnaires[d]	Summary Findings and Adjustments for Confounders[e]
Hwang BF et al. 2005a	22 Municipalities in Taipei,China	2001	32,672 M+F schoolchildren (6–15 years)	Between cities 1 km from monitoring stations (n = 22)	CO, NO_x, O_3, PM_{10}, and SO_2	Prevalence, A-rns, ISAAC	Significant associations Adjustment for D, S, I, Sm, and O. Study includes multi-pollutant models
Imai et al. 1980	Yokkaichi, Mihama, and Kusu, Japan	1973–1974 and 1978	M+F residents (> 40 years)	Within city (23 areas)	Time trends in NO_x and sulfur oxides	Prevalence and trends, A-r, BMRC	Significant association with sulfur oxides but not NO_x Adjustment for D and Sm, NO_x Multiple correlation coefficients calculated using adjusted prevalence estimates
Kagamimori et al. 1986	Awara-machi, Japan	1970–1979	M+F schoolchildren (6–14 years)	Point source (power station)	Time trends in NO_2 and SO_2	Prevalence and trends, W-r, BMRC	Significant correlations over time with NO_2 Adjustment for D
Kumar R et al. 2004	Mandi Gobindgarh and Morinda, India	1999–2001	3603 M, F, and M+F adult residents (> 15 years)	Between cities (n = 2)	High vs. low	Prevalence, W-r, BMRC	Significant association Adjustment for D, by design
Kuo et al. 2002	Central Taipei,China	NR	12,926 M+F junior-high students (13–16 years) in eight schools	Between cities (n = 8)	High vs. low concentrations NO_2, O_3, PM_{10}, and SO_2	Prevalence, A-rns and W-rns, ISAAC	Significant associations Adjustment for D, S, I, Sm, and O
Langkulsen et al. 2006	Bangkok, Thailand	2004	878 M+F schoolchildren	Within city	High (three grades) vs. low	Prevalence, A-r, ATS	Significant associations Adjustment for D, I, Sm, and O

Table continues next page

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[c] If several designs were used, the design associated with the reported adjusted effect estimates is listed.

[d] Possible parameters were incidence, prevalence, exacerbation, and trends. Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), or A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting is unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), or W-rns (wheeze symptoms whose time of reporting is unspecified or not recent). The questionnaire was the standardized questionnaire used to assess asthma and wheeze in the study, if any.

[e] Summary findings listed include any positive statistically significant association (noted as "significant association"). Any significant negative association is specifically listed as such. Adjustments for confounders included adjustments for demographic characteristics (D), socioeconomic characteristics (S), indoor air pollution (I), smoking status (Sm), and other variables (O). Comments are also listed, if pertinent.

Table 19 (*Continued*). Studies of Asthma and Wheeze

Citation	Study Location	Study Year[a]	Study Sample[b]	Designs[c]	Pollution Variables or Comparisons	Parameters, Outcomes, and Questionnaires[d]	Summary Findings and Adjustments for Confounders[e]
Lee YL et al. 2003c	Taipei,China	2001	35,036 M+F schoolchildren (6–15 years)	Between cities (n = 22) 1 km from monitoring stations	Subject-perceived pollution, and CO, NO_x, PM_{10}, SO_2, and O_3	Prevalence, A-rns, ISAAC	CO, NO_x, PM_{10}, and SO_2 concentrations associated with subject-perceived pollution. Subject-perceived pollution associated with asthma. Adjustment for D, S, and Sm
Lee YL et al. 2005	Taipei,China	1995–1996 and 2001	M+F children (12–15 years): 44,104 in 1995–1996 and 11,048 in 2001	Repeated surveys	Time trends in CO, NO_x, O_3, and PM_{10}	Prevalence and trends, W-rns and A-rns, ISAAC	No significant correlation over time between adjusted prevalence and pollutant levels Adjustment for D, S, Sm, and O
Lin RS et al. 2001c	Taipei,China	1995–1996	1,018,031 M+F middle-school students	National	CO concentrations: high vs. low and medium vs. low	Prevalence, A-r and W-r, ISAAC	Significant associations Adjustment for D, S, I, and Sm
Maeda et al. 1991	Tokyo, Japan	1987–1990	2600 Adult women (30–60 years) and 360 M+F children (3–6 years)	Between individuals and proximity to traffic	Proximity to traffic: < 20 m, 20–150 m, and residential away from roads with heavy traffic	Prevalence, W-r and A-r, ATS	Significant association with chronic wheezing; no significant association with asthma Adjustment for D, I, and Sm
Mi et al. 2006	Shanghai, China	2000	1414 M+F schoolchildren	Within city	NO_2 and O_3	Prevalence, W-r and A-r, ECRHS	Significant associations Adjustment for D, I, and Sm

Table continues next page

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[c] If several designs were used, the design associated with the reported adjusted effect estimates is listed.

[d] Possible parameters were incidence, prevalence, exacerbation, and trends. Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), or A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting is unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), or W-rns (wheeze symptoms whose time of reporting is unspecified or not recent). The questionnaire was the standardized questionnaire used to assess asthma and wheeze in the study, if any.

[e] Summary findings listed include any positive statistically significant association (noted as "significant association"). Any significant negative association is specifically listed as such. Adjustments for confounders included adjustments for demographic characteristics (D), socioeconomic characteristics (S), indoor air pollution (I), smoking status (Sm), and other variables (O). Comments are also listed, if pertinent.

Table 19 (*Continued*). Studies of Asthma and Wheeze

Citation	Study Location	Study Year[a]	Study Sample[b]	Designs[c]	Pollution Variables or Comparisons	Parameters, Outcomes, and Questionnaires[d]	Summary Findings and Adjustments for Confounders[e]
Nakai et al. 1999	Tokyo, Japan	1987–1990	1986 Adult women (30–59 years)	Proximity to traffic	Distance from road	Prevalence, W-r, and ATS	No significant associations Adjustment for D, I, and Sm
Nitta et al. 1993	Tokyo, Japan	1979, 1982, and 1983	Adult women (40–59 years): 1148 in 1979, 1758 in 1982, and 1916 in 1983	Proximity to traffic	Distance from road: < 20 m, 20–150 m, and residential away from roads with heavy traffic	Prevalence, W-r	Significant associations Adjustment for D, S, I, and Sm, and O
Ong et al. 1991	Hong Kong, China	1989	3846 M, F, and M+F schoolchildren	Within city	High vs. low	Prevalence, W-rns	Significant associations Adjusted for D, by design
Peters J et al. 1996	Hong Kong, China	1989–1991	3521 M+F children	Within city	Trends in low vs. high	Prevalence and trends, W-r, WHO and BMRC	Reduction in difference between areas after low-sulfur fuel regulation Adjustment for D, by design
Qian Z et al. 2000	Lanzhou, Wuhan, and Guangzhou, China	1985–1988	2789 M+F schoolchildren (5–14 years)	Between and within cities (n = 4)	High vs. low (i.e., Lanzhou vs. Guangzhou)	Prevalence, W-r and A-rns, ATS	No significant association Adjustment for D, S, I, and Sm
Qian Z et al. 2004	Guangzhou, Wuhan, Lanzhou, and Chongqing, China	1993–1996	7058 M+F schoolchildren (5–16 years)	Between cities (n = 4)	High vs. low	Prevalence, W-r and A-rns, ATS	Significant associations Adjustment for D, S, I, and Sm

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[b] M+F indicates that the data for males and females were combined; M, F indicates that the data for males and females were reported separately.

[c] If several designs were used, the design associated with the reported adjusted effect estimates is listed.

[d] Possible parameters were incidence, prevalence, exacerbation, and trends. Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), or A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting is unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), or W-rns (wheeze symptoms whose time of reporting is unspecified or not recent). The questionnaire was the standardized questionnaire used to assess asthma and wheeze in the study, if any.

[e] Summary findings listed include any positive statistically significant association (noted as "significant association"). Any significant negative association is specifically listed as such. Adjustments for confounders included adjustments for demographic characteristics (D), socioeconomic characteristics (S), indoor air pollution (I), smoking status (Sm), and other variables (O). Comments are also listed, if pertinent.

Table 19 (*Continued*). Studies of Asthma and Wheeze

Citation	Study Location	Study Year[a]	Study Sample[b]	Designs[c]	Pollution Variables or Comparisons	Parameters, Outcomes, and Questionnaires[d]	Summary Findings and Adjustments for Confounders[e]
Sekine et al. 2004	Tokyo, Japan	1987–1994	5682 Adult women	Proximity to traffic	Traffic density: high or average vs. medium vs. low	Prevalence,W-r and A-r, ATS	No significant associations Adjustment for D, S, I, Sm, and O
Shima and Adachi 2000	7 Communities in Chiba Prefecture, Japan	1991–1993	842 M, F children (9–10 years)	Between cities (n = 7)	NO_2	Incidence and prevalence, W-r and A-r, ATS	Significant associations with incidence and prevalence Adjustment for D, I, Sm, and O
Shima et al. 2002	8 Communities in Chiba Prefecture, Japan	1989–1992	3049 M, F, and M+F children (6–13 years)	Between cities (n = 8)	NO_2 and PM_{10}	Incidence and prevalence, W-r and A-r, ATS	No significant associations with prevalence, significant associations with incidence Adjustment for D, I, Sm, and O
Shima et al. 2003	Chiba Prefecture, Japan	1991–1995	2506 M, F schoolchildren (6–13 years)	Proximity to traffic, in 8 communities	Proximity to road: 0–49 m vs. ≥ 50 m vs. rural	Incidence and prevalence, W-r and A-r, ATS	Significant association with living < 50 m from road Adjustment for D, I, Sm, and O
Tam et al. 1994	Hong Kong, China	1989–1990	423 M+F children	Within city	High vs. low	Prevalence, W-r, and A-rns	Significant association for W-r, nonsignificant association for A-rns Adjustment for D, by design

Table continues next page

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[c] If several designs were used, the design associated with the reported adjusted effect estimates is listed.

[d] Possible parameters were incidence, prevalence, exacerbation, and trends. Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), or A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting is unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), or W-rns (wheeze symptoms whose time of reporting is unspecified or not recent). The questionnaire was the standardized questionnaire used to assess asthma and wheeze in the study, if any.

[e] Summary findings listed include any positive statistically significant association (noted as "significant association"). Any significant negative association is specifically listed as such. Adjustments for confounders included adjustments for demographic characteristics (D), socioeconomic characteristics (S), indoor air pollution (I), smoking status (Sm), and other variables (O). Comments are also listed, if pertinent.

Table 19 (*Continued*). Studies of Asthma and Wheeze

Citation	Study Location	Study Year[a]	Study Sample[b]	Designs[c]	Pollution Variables or Comparisons	Parameters, Outcomes, and Questionnaires[d]	Summary Findings and Adjustments for Confounders[e]
Wang TN et al. 1999	Kaohsiung and Pintong, Taipei,China	1995–1996	165,173 M+F high-school students (11–16 years)	Between cities (n = 3) or within cities (n = 24)	Above or below median for TSP, PM_{10}, SO_2, NO_2, CO, and O_3	Prevalence, W-r, ISAAC	Significant associations Adjustment for D, S, I, Sm, and O
Xu X and Wang L 1993	Beijing, China	1982	1576 M+F adult never-smokers (40–69 years)	Within city	High vs. moderate vs. low	Prevalence, W-r	No significant associations Adjustment for D, S, I, Sm, and O
Yang CY et al. 1997	Lin-yuan (exposed area) and Taihsi (control area), Taipei,China	1995	2036 M+F adult residents of Lin-yuan (30–64 years) and 1976 controls	Between industrial and non-industrial areas (n = 2)	Industrial vs. non-industrial	Prevalence, W-r, ATS and WHO	No significant association Adjustment for D, S, I, Sm, and O
Yang CY et al. 1998	Lin-yuan (exposed area) and Taihsi (control area), Taipei,China	1994–1995	1081 M+F primary-school children: 470 in a polluted area and 611 in a control area	Between an industrial and a rural area	Industrial vs. rural	Prevalence, W-r, ATS	No significant association Adjustment for D, S, I, Sm, and O
Yang CY et al. 2002c	Yang-Ming and Zhuang-Jing, Taipei,China	1999	6190 M+F primary-school students	Proximity to traffic	Heavy vs. light traffic	Prevalence, W-r and A-r, ATS and WHO	No significant association Adjustment for D, S, I, Sm, and O

Table continues next page

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[d] Possible parameters were incidence, prevalence, exacerbation, and trends. Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), or A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting is unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), or W-rns (wheeze symptoms whose time of reporting is unspecified or not recent). The questionnaire was the standardized questionnaire used to assess asthma and wheeze in the study, if any.

[e] Summary findings listed include any positive statistically significant association (noted as "significant association"). Any significant negative association is specifically listed as such. Adjustments for confounders included adjustments for demographic characteristics (D), socioeconomic characteristics (S), indoor air pollution (I), smoking status (Sm), and other variables (O). Comments are also listed, if pertinent.

Table 19 (*Continued*). Studies of Asthma and Wheeze

Citation	Study Location	Study Year[a]	Study Sample[b]	Designs[c]	Pollution Variables or Comparisons	Parameters, Outcomes, and Questionnaires[d]	Summary Findings and Adjustments for Confounders[e]
Yano et al. 1986	Kushira, Sakurajima, and Oura, Japan	1980	2006 Adult women (30–59 years)	Between areas (n = 3)	Volcanic ash–affected areas vs. control area	Prevalence, W-r	No significant association Adjustment for D
Yu JH et al. 2005	Taichung and Chu-Shan, Taipei,China	2001	14,201 M+F schoolchildren	Within city	Urban vs. rural	Prevalence, W-r and A-r, ISAAC	Significant association Adjustment for D, by design
Yu TS et al. 2001	Hong Kong, China	1994–1995	1294 M+F children (8–12 years)	Between cities (n = 2)	High vs. low	Prevalence, W-r and A-rns, ATS	Significant associations Adjustment for D, S, I, Sm, and O
Zhang JF et al. 2002	Guangzhou, Wuhan, Lanzhou, and Chongqing, China	1993–1996	7557 M+F elementary-school students	Within city	$PM_{2.5}$, $PM_{10–2.5}$, PM_{10}, TSP, SO_2, and NO_x	Prevalence, W-r and A-rns, ATS	No significant association Adjustment for D, S, I, Sm, and O
Zhang J et al. 1999	Guangzhou, Wuhan, and Lanzhou, China	1985–1988	4108 M, F adults	Between cities (n = 4) and within city (urban vs. suburban)	Urban vs. suburban within Wuhan	Prevalence, W-r and A-rns, ATS	Significant associations Adjustment for D, S, I, Sm, and O

[a] NR indicates that the study year was not reported.

[b] M+F indicates that the data for males and females were combined; M, F indicates that the data for males and females were reported separately.

[c] If which several designs were used, the design associated with the reported adjusted effect estimates is listed.

[d] Possible parameters were incidence, prevalence, exacerbation, and trends. Possible outcomes were A-r (doctor-diagnosed or self-reported asthma within the last 3 years), or A-rns (doctor-diagnosed or self-reported asthma, whose time of diagnosis or self-reporting is unspecified or not recent), W-r (wheeze symptoms reported within the last 3 years), or W-rns (wheeze symptoms whose time of reporting is unspecified or not recent). The questionnaire was the standardized questionnaire used to assess asthma and wheeze in the study, if any.

[e] Summary findings listed include any positive statistically significant association (noted as "significant association"). Any significant negative association is specifically listed as such. Adjustments for confounders included adjustments for demographic characteristics (D), socioeconomic characteristics (S), indoor air pollution (I), smoking status (Sm), and other variables (O). Comments are also listed, if pertinent.

questionnaire outcomes for purposes of our review, we classified the reported outcome data into "recent" (symptoms occurring within 3 years before the question was asked, persistent symptoms, or symptoms whose degree of recency was implied in the question) and "degree of recency not specified" (containing responses to the " have you ever had" questions). This classification yielded four outcome categories: recent wheezing (W-r); recent asthma (A-r); wheezing, degree of recency not specified (W-rns); and asthma, degree of recency not specified (A-rns). Some studies reported grades of severity or exacerbation, but these data were not included in the meta-analysis.

To distinguish comparisons of populations exposed to distinct airsheds from comparisons of study subjects within populations (such as intra-city studies) with shared regional and urban background exposures, we categorized[f] the comparisons into two broad classes: between-city comparisons (with "cities" loosely defined as distinct urban communities with separate airsheds), reported in 25 studies, and within-city comparisons (describing those for large conurbations or other areas with unclear boundaries), reported in 17 studies.

The within-city studies included five that investigated the associations between asthma and proximity to traffic. In addition, one study assessed exposure at the individual level, two studies focused on a point power-generation source, and another study investigated the effects of long-term exposure to volcanic air pollution. Three studies correlated the prevalence of asthma with air pollution over time. Some studies without actual measures of community exposure to air pollution made assumptions about the ranking of exposure contrasts, such as in some urban–rural or urban–suburban comparisons and comparisons of communities according to proximity to traffic. Several studies described measures of long-term exposure to pollutants such as PM_{10} but used these data to categorize populations instead of using the pollution data quantitatively in the statistical analysis.

Many studies, particularly the more recent ones, used logistic regression analysis to estimate ORs for the effect of air pollution on asthma symptoms, adjusting for various confounding factors. A total of 33 of the studies analyzed pollution as a qualitative variable; the remainder analyzed the effects per increment of pollution. Sometimes, the analysis dichotomized the annual exposure measurements (e.g., as being above or below the U.S. EPA NAAQS or the median of the measured concentrations). The absence of logistic regression analysis did not necessarily reduce the quality of the study. For example, one very large, multicenter study in Taipei,China, appropriately used multiple linear regression to analyze standardized prevalence estimates (Guo et al. 1999).

Confounding is a major potential problem in studies of long-term exposure, especially when the exposure is assessed for the pollutant mix rather than individual pollutants. The studies varied greatly in the extent of their control for confounding. Some studies controlled only for age or sex, by design (e.g., by selecting the same school grades in all study areas), whereas others controlled not only for demographic factors but also for socioeconomic indicators, a range of indoor environmental factors (e.g., cooking, incense burning), smoking (active and passive), and other factors (e.g., temperature).

The majority of studies did not report a quantitative relation between measured or modeled pollution concentrations and the health outcome, making it difficult to estimate a summary effect in terms of a defined pollution increment. Only a minority of studies estimated summary effects in terms of increments in concentrations of measured pollutants, and the designs, outcomes, pollution exposure contrasts, and specific pollutants analyzed were quite heterogeneous, making it impossible to assemble a sufficiently large number ($n > 4$) of homogeneous studies for a credible quantitative meta-analysis. However, we constructed forest plots of estimated ORs for either wheeze or a diagnosis of asthma from the 26 of 42 studies that reported them (Figures 66, 67, and 68); the corresponding data are also listed in Appendix Tables B.1, B.2, and B.3. Quantitative estimates were standardized to an increase of 10 $\mu g/m^3$ in pollutant concentration. For the studies whose data are not shown in the figures, Table 19 briefly summarizes whether or not significant associations were found.

Qualitative Comparisons Among Geographic Areas

Figure 66 shows ORs for qualitative comparisons between urban and rural communities or between or within cities. There were five comparisons between urban and rural communities, and although ORs were elevated in all these studies, most were estimated with poor precision and only one was statistically significant. There were 11 between-city studies in which ORs were based on a qualitative comparison, 7 of which are plotted. The majority reported an increased prevalence of asthma or wheeze in association with exposure to air pollution. This group of studies was dominated by five studies in Taipei,China, which had used a variety of designs to study asthma symptoms in children. With the exception of one of the five studies, which compared industrial and non-industrial areas (finding no significant association), these studies used an arbitrary cutoff

[f] Although many articles included considerable information on outcomes and pollution exposures, we concentrated on the results presented together with the statistical associations to categorize the outcomes and exposure comparisons.

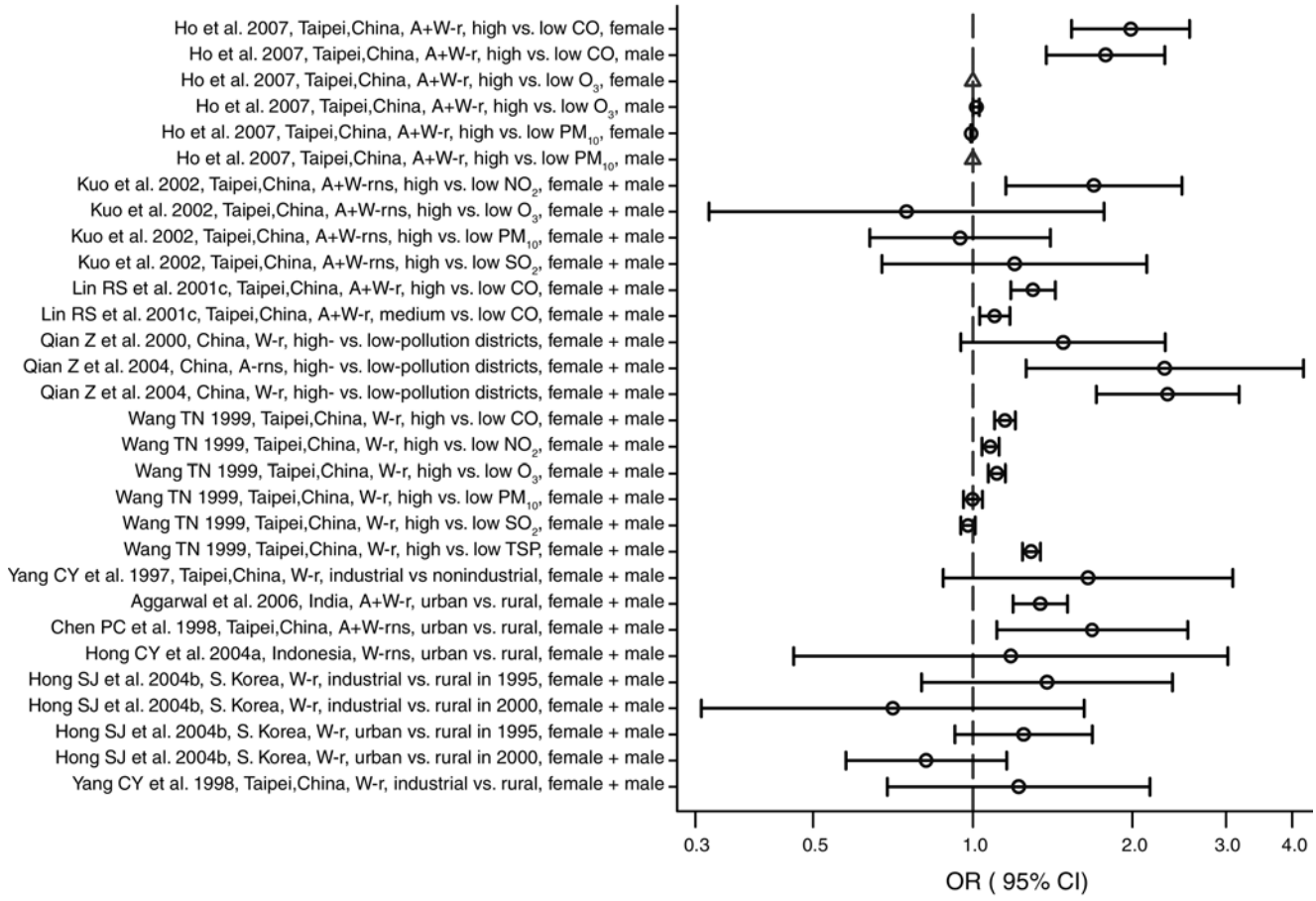

Figure 66. ORs (and 95% CIs) for asthma or wheeze from qualitative comparisons between areas with differing pollutant concentrations in studies of urban and rural locations, or between or within cities. Y-axis labels give study information in the following sequence: reference citation; location, outcome, pollutant levels or areas compared; and sex of subjects (female, male, or combined). A log scale is used on the x axis to accommodate the range of estimates. Two studies reported "no significant association" with no odds ratio; data points are shown here as triangles without a CIs. For study details, see Table 19. The four outcome categories are: recent wheezing (W-r); recent asthma (A-r); wheezing, degree of recency not specified (W-rns); and asthma, degree of recency not specified (A-rns), or a combination of asthma and wheeze (A+W-r and A+W-rns).

for pollutant concentrations (e.g., above or below the U.S. EPA NAAQS or the median of the measured concentrations) based on data from community monitors. In one study (Ho et al. 2007), a significant, positive association of disease with high concentrations of CO was observed, but PM_{10} and O_3 appeared to have no significant effect. In the study of Kuo and colleagues (2002), there was a significant, positive association with high NO_2 concentrations, but no significant association with PM_{10}, O_3, or SO_2 concentrations. Lin MC and colleagues (2001b) found asthma and wheeze to be positively associated with high CO concentrations, and Wang TN et al. (1999) reported significant associations with high NO_2, O_3, CO, and TSP but not PM_{10} or SO_2. In a national study, Guo and colleagues (1999) reported a significant, positive association of wheeze and asthma with traffic-related sources, but not stationary sources or those related to fossil fuels (according to principal-components analysis), and significant associations with NO_x and CO but not PM_{10}, O_3, or SO_2 (data not shown). Though there is a lack of consistency between these studies of essentially the same populations and exposures, it appears that indicators of traffic emissions, such as NO_2 and CO, are more consistently associated with the prevalence of asthma or wheeze than are other pollutants. None of the four studies that investigated PM_{10} found any significant associations with asthma symptoms.

There were 10 studies of qualitative within-city comparisons, conducted in Thailand, India, Japan, China, Taipei,China, and Hong Kong. The four for which ORs

could be extracted are shown in Figure 67. Most reported at least one significant positive association, but one study in Taipei,China and another in Japan found no significant associations, and many studies also reported association that could be explained by the play of chance. One study (by Imai et al. 1980) analyzed a large number of areas, but most comparisons involved only two or three strata of analysis (e.g., urban vs. suburban or high vs. moderate vs. low pollutant level).

The other main qualitative within-city comparisons were for proximity to traffic. There were eight such studies, of which four (all in Japan) found positive associations. The four for which ORs could be extracted are shown in Figure 67. Nakai and coworkers (1999) found no evidence of an association with road proximity. In the study by Nitta and colleagues (1993), the risk of asthma was increased by living within 20 m of traffic, versus 20–150 m from traffic, in 1979 but not 1982 or 1983. Sekine and coworkers (2004) used a complicated combination of traffic proximity, traffic intensity, and NO_2 concentrations to classify the population into three categories of exposure; they found increased, though not statistically significant, associations of disease prevalence in the high- and medium-exposure categories as compared with the low-exposure category. The study by Shima and colleagues (2003) analyzed the risks of both asthma prevalence and incidence over 4 years between populations living 0–49 m or ≥ 50 m from main roads and a population in a rural

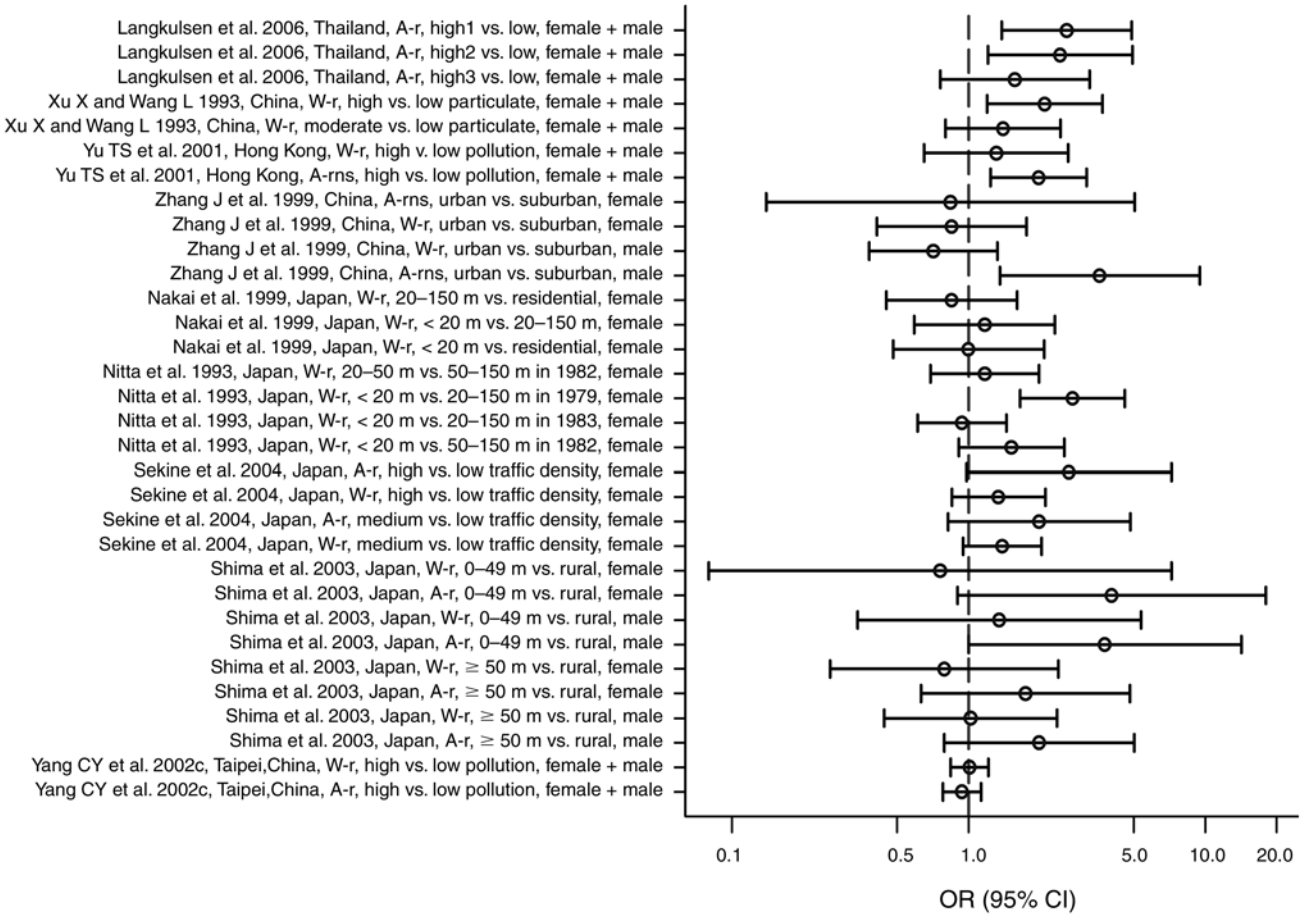

Figure 67. ORs (and 95% CIs) for asthma or wheeze from qualitative comparisons between areas with differing pollutant levels or proximities to traffic within cities. Y-axis labels give study information in the following sequence: reference citation; location; outcome; pollutant levels, areas, or distances from traffic compared; year of residence (if applicable); and sex of subjects (female, male, or combined). A log scale is used on the x axis to accommodate the range of estimates. For A-r outcomes in Shima et al. 2003, the ORs are estimates of the relative risk of new occurrences of asthma. For Langkulsen et al. 2006, high1 indicates highly polluted general area; high2 indicates highly polluted roadside area; high3 indicates moderately polluted roadside area; low indicates control area. For study details, see Table 19. The four outcome categories are: recent wheezing (W-r); recent asthma (A-r); wheezing, degree of recency not specified (W-rns); and asthma, degree of recency not specified (A-rns), or a combination of asthma and wheeze (A+W-r and A+W-rns).

area. There was a significant trend toward higher prevalence among girls, but not boys, living 0–49 m from main roads versus in the rural area. Follow-up of the same populations revealed a significant increase in the number of new cases of asthma, but not wheeze, among boys but no significant increase in the incidence of either outcome among girls in populations living 0–49 m from a main road versus in rural areas. The study did not report on a comparison of children living in a roadside area versus a nonroadside area.

Quantitative Comparisons Based on Ambient Measurements The results of quantitative comparisons based on ambient measurements are summarized in Figure 68; the estimates plotted in the figure are shown in Appendix Table B.3 Although the pattern is heterogeneous, the overall finding is that there is little evidence of an increased prevalence of asthma in association with an increase of 10 μg/m^3 in specific measured pollutants, apart from NO_2 in some studies. The lack of associations with PM, measured as PM_{10}, is especially striking. The following paragraphs give greater detail about the individual studies.

Shima and colleagues (2002) performed a study of elementary schools in eight communities in Japan's Chiba Prefecture, four of which were in rural areas. The prevalence of asthma was not significantly associated with increases in PM_{10} or NO_2 concentrations in boys or girls studied separately. Increased NO_2 concentrations were associated with an increase by a factor of nearly 4 in the incidence of asthma among 6-year-old children followed

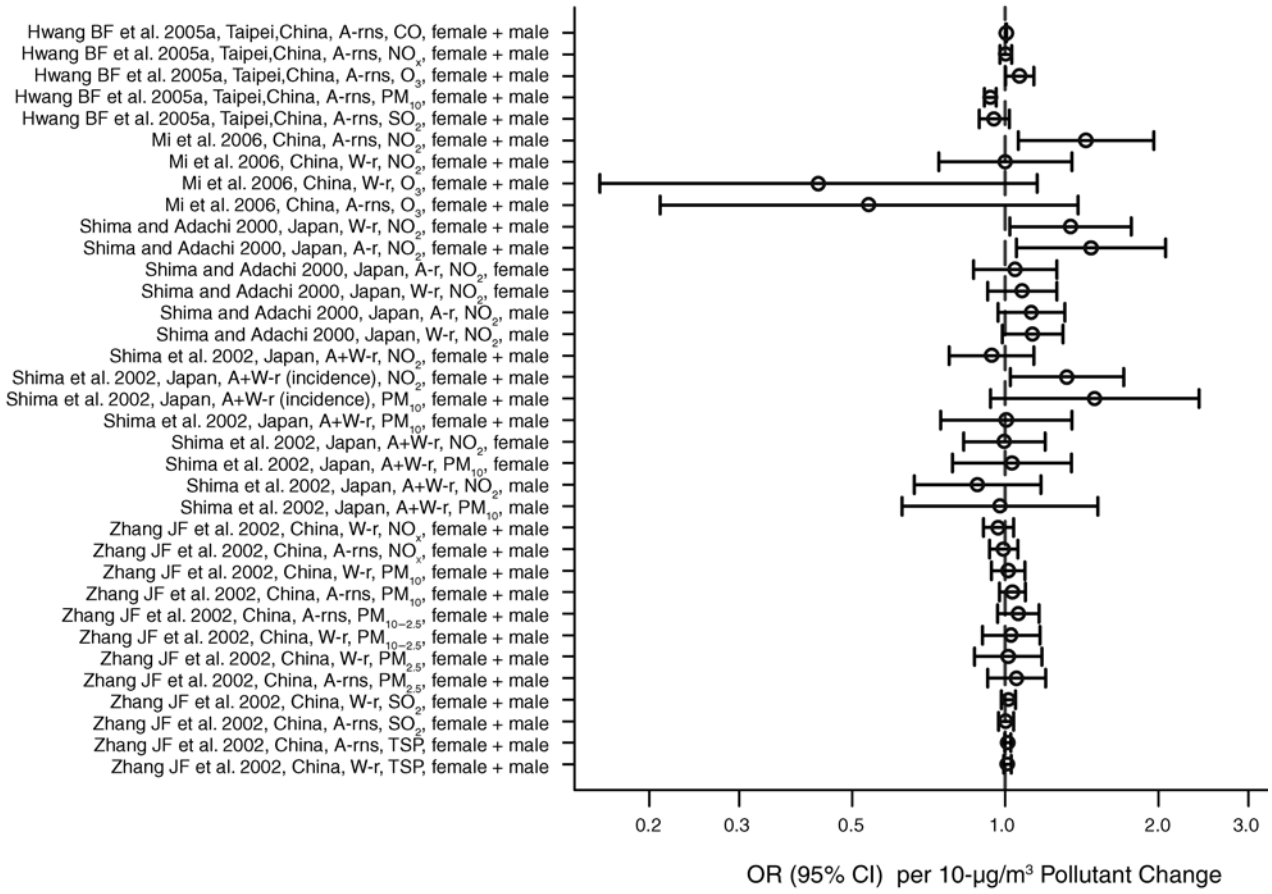

Figure 68. ORs (and 95% CIs) for asthma or wheeze per 10 μg/m^3 change in pollutant concentration from quantitative comparisons. Y-axis labels give study information in the following sequence: reference citation, location, outcome, pollutant, and sex of subjects (female, male, or combined). A log scale is used on the x axis, to accommodate the range of estimates. Two of the ORs for both females and males from the Shima et al. 2002 study were calculated from incidence data. The Mi et al. 2006 study was conducted within a single city. The ORs from the Shima and Adachi 2000 study are for 4th, 5th, and 6th grades combined. For study details, see Table 19. The four outcome categories are: recent wheezing (W-r); recent asthma (A-r); wheezing, degree of recency not specified (W-rns); and asthma, degree of recency not specified (A-rns), or a combination of asthma and wheeze (A+W-r and A+W-rns).

over 4 years (data not shown), though the estimate was extremely imprecise, with CIs that included increases as small as 10% and as large as 1000% (OR, 3.62; 95% CI, 1.11–11.87). An increase in PM_{10} concentration was associated with an increase, by a factor of almost 3, in asthma incidence (OR, 2.84; 95% CI, 0.84–9.58), but this estimate too was highly imprecise (data not shown). The 2002 study was an update of one by Shima and Adachi from 2000, which showed an association between outdoor concentrations of NO_2 and the incidence of wheeze and asthma over 3 years. In the 2002 update, no significant association was found between asthma incidence and indoor concentrations of NO_2, nor were there significant differences in the prevalence of asthma among urban, suburban, and rural districts. Taken together with the results of the study of distance from main roads (Shima et al. 2003) discussed above, it seems that outdoor concentrations of NO_2 and pollutant concentrations reflecting proximity to roads were more related to incidence than to prevalence. Since prevalence is a function of incidence and duration, this suggests that the duration of wheezing illness may be shorter in areas near roads than in areas not near roads.

Hwang BF and colleagues (2005a) carried out an investigation among children in schools in 22 municipalities within 1 km of monitoring stations collecting measurements of background pollution at regular intervals. In comparisons of populations living closer to traffic and those living farther away, adjusted ORs for doctor-diagnosed asthma showed significant associations between the prevalence of asthma and increases in CO concentration (by 100 ppb) (OR, 1.05; 95% CI, 1.02–1.07) and increases in O_3 concentration (by 10 ppb) (OR, 1.14; 95% CI,1.00–1.29), nonsignificant associations with NO_x (OR, 1.01; 95% CI, 0.95–1.06), and no association with PM_{10} or SO_2. The positive results for CO and O_3 were robust in multi-pollutant models.

The associations between asthma and air pollution were investigated among children from eight districts of four Chinese cities (Zhang JF et al. 2002). The OR for developing asthma was increased in relation to an increase in the concentrations of $PM_{2.5}$, $PM_{10–2.5}$, PM_{10}, TSP, and SO_2, but none of the associations were statistically significant. The association with NO_x was negative but also not significant.

Mi and colleagues (2006) investigated the association between asthma and indoor and outdoor NO_2 and O_3 in 10 naturally ventilated schools in Shanghai. Neither pollutant was significantly associated with the prevalence of wheeze, but NO_2 concentration was significantly associated with the diagnosis of asthma (OR, 1.44; 95% CI, 1.06–1.95).

We also identified four relevant studies that do not fit easily into the within- and between-city categories (Table 19). Two reported associations between trends in prevalence and trends in air pollution over time. First, the study by Kagamimori and colleagues (1986) in Japan found correlations of asthma prevalence over time with NO_2, but not SO_2, in a population exposed to stationary-source pollution from fossil-fuel combustion. Second, for Taipei,China, Lee YL and colleagues (2005) reported neither significant associations between trends in childhood asthma between 1995–1996 and 2001 nor trends in CO, NO_x, O_3, or PM_{10} concentrations over the same period. Because of the likelihood of other relevant trends over time, any causal interpretation of these ecologic trends must be extremely cautious.

A more powerful way of studying the effects of air pollution over time using ecologic data is to investigate situations in which there is an important change in air pollution. Such an opportunity arose in Hong Kong when, in June 1990, there was a restriction on the sulfur content of fuel put into place over a single weekend. This resulted in a marked and sudden drop in ambient concentrations of SO_2. Peters J and colleagues (1996) found that differences in the prevalence of wheezing symptoms between a polluted district and a less-polluted district in Hong Kong were attenuated between 1989 and 1991, consistent with a reduction in the effects of air pollution.

A last study is by Yano and colleagues (1986), who compared chronic respiratory symptoms in populations exposed to a range of concentrations of particles from a volcano. No significant associations with asthma were found.

Discussion The studies of long-term exposure to ambient air pollutants and asthma symptoms and diagnoses reviewed in this section vary considerably in design. Differences in exposure have been estimated at between- and within-city levels, with the within-city comparisons including roadside exposures. The studies also vary widely in terms of statistical power, quality of exposure assessment, and degree of control of confounding factors. At one end of the spectrum are studies of only two communities, with small sample sizes, no adjustment for confounders (apart from age and sex, by design, as in studies of schoolchildren in various grades), and a purely qualitative comparison of exposures (e.g., urban vs. rural or urban vs. suburban). At the other end are large multi-center studies of large samples with high-quality exposure assessment, extensive control of confounding factors, and sophisticated multi-level statistical analysis. Some studies confined the outcome to physician diagnosis of asthma only, increasing the potential for diagnostic bias. All studies relied on questionnaire data to ascertain outcomes, rather than using more objective methods, such as measures of BHR, thus increasing the potential for reporting bias by respondents. Overall, however, the design and conduct of

the studies are similar to those from Western countries, and some, such as the very large and well-conducted prevalence studies from Taipei,China, the incidence study from Japan, and the evaluation of sulfur regulation in Hong Kong, make important, new contributions to the international literature.

Most of the reported estimates showed an elevated prevalence of asthma in association with air pollution exposure, and none showed a significantly reduced prevalence, which would have suggested uncontrolled sources of confounding or other error. The estimated RR was generally less than 1.5 for more-polluted locales versus less-polluted locales. The larger estimates were generally less precise, as indicated by wider CIs. Conversely, the most precise estimates, those from Taipei,China, tended to be the lowest. This heterogeneity of results was seen in all of the subgroups compared in urban–rural, between-city, within-city, and traffic-proximity studies. Unfortunately, methodologic differences make it impossible to combine studies in order to compute a more precise summary estimate.

Publication bias could not be formally investigated, but the most extreme ORs tended to have the widest CIs, irrespective of direction of the effect. This observation is evidence against any non-negligible degree of publication bias regarding asthma. Most of the studies were equally, if not more, concerned with other respiratory symptoms, such as chronic cough, making it less likely that negative findings for just one outcome, such as asthma, would affect the decision to publish.

The results of the Asian studies of asthma are very similar to those obtained from research in Western countries. Western studies have tended not to find significant differences between urban and rural areas, and the results of multi-city or multi-area comparisons of prevalence have, if anything, tended toward null results (Dockery et al. 1989, 1996; Braun-Fahrlander et al. 1997; Baldi et al. 1999; Peters JM et al. 1999; Zemp et al. 1999; Pattenden et al. 2006).

Excluding studies of birth cohorts, there are few Western studies of the incidence of asthma to compare with Asian study results. One of the few is the Californian Seventh-Day Adventist Health Study of Smog (AHSMOG), which reported an association between O_3 and the incidence of asthma in adult males (McDonnell et al. 1999). On the other hand, the Californian Children's Health Study of 12 areas did not find a significant association between asthma incidence and community concentrations of acid particles, NO_2, O_3, PM_{10}, or $PM_{2.5}$ (McConnell et al. 2002).

There is now a large body of evidence from Western studies associating proximity to traffic, especially heavy-duty diesel vehicles, with increased prevalence of respiratory symptoms (Heinrich and Wichmann 2004; Salam et al. 2008). The Asian evidence is of great interest. Results from Japan have not supported an association between roadside exposure and asthma prevalence, but there is evidence of an association with asthma incidence over a 4-year period in a cohort of first-grade students (Shima et al. 2003). Such an association of incidence, but not prevalence, with greater exposure to vehicle pollution has been found in Southern California (Gauderman et al. 2005; McConnell et al. 2006). Overall, there remains considerable uncertainty surrounding the effect of traffic proximity on asthma (Gauderman et al. 2005). We did not find any reports of asthma in Asian birth cohorts. A considerable number of birth-cohort studies are now under way, but the results reported so far have been inconsistent (e.g. Gehring et al. 2002; Pierse et al. 2006; Brauer et al. 2007; Oftedal et al. 2007; Morgenstern et al. 2008; Nordling et al. 2008).

A range of interpretations could be made regarding the lack of consistency among associations observed in the Asian studies. On the positive side, it could be argued that the associations are in fact causal but that their heterogeneity reflects variations in toxicity, exposure contrasts, or exposure error, or other limitations of the methods used. On the negative side, it could be argued that the negative associations are true and that the positive associations reflect the play of chance, reporting bias, or uncontrolled confounding at the group or individual level. Taken as a whole, we conclude that there is some evidence for a modest effect of air pollution on asthma prevalence, but we still know little about the reasons for this effect or the observed heterogeneity among the findings. Our impression is that if there are any differences between Asian and Western studies, it is that Asian studies tend to find more positive results. Asthma prevalence ranges widely among children in Asia (Asher et al. 2006), but this variation seems unlikely, on the basis of the current evidence, to be explained by the corresponding range of ambient air pollution concentrations (Anderson et al. 2010). The variability in the results of the asthma studies reviewed in this section should not be taken to imply that air pollution is not harmful to the respiratory system in ways other than causing asthma; there is convincing evidence that air pollution is associated with respiratory symptoms such as chronic cough and impairment of lung function (WHO 2005b).

LUNG CANCER

Studies

Five studies of lung cancer met the criteria for inclusion (Table 20). Three of the five studies were in East Asia (China and Taipei,China), one in Thailand, and one in India. All were case–control studies, which provide estimates of the

Table 20. Case–Control Studies of Lung Cancer

Citation	Study Location	Study Years	Study Sample	Pollutants	Health Outcome	Results
Gupta D et al. 2001a	Chandigarh, India	1995–1997	265 Case subjects and 525 controls matched by sex and age	Urban air pollution, indoor pollution, tobacco smoking, and occupational exposure	Incidence	RR for women with lifetime residence in urban area vs. rural area, 0.29 (95% CI, 0.07–1.17).
Ko YC et al. 1997	Kaohsiung, Taipei, China	1992–1993	117 Women with lung cancer and 117 matched controls, including 106 nonsmokers	Industrial air pollution and cooking fumes	Incidence	RR for nonsmoker women with residence ≥ 21 years within 3 km of industrial area vs. those with residence for < 21 years, 2.7 (95% CI, 0.9–8.9).
Pisani et al. 2006	Lampang Province, Thailand	1993–1995	211 Case subjects and 202 controls (set 1) and 211 controls (set 2)	Power-plant emissions	Incidence	RR for high vs. low SO_2/NO_2 index, 1.2 (95% CI, 0.7–2.0); RR for high vs. low PM_{10} index, 1.1 (95% CI, 0.7–1.8).
Xu ZY et al. 1989	Shenyang, China	1985–1987	1349 Patients and 1345 controls	Industrial air pollution	Incidence	RRs for residence for > 20 years within 200 m of industrial area vs. those with residence for ≤ 20 years: men, 2.7 (95% CI, 1.1–6.5); women, 3.2 (95% CI, 1.0–10.5).
Yang CY et al. 1999	Taipei, China	1990–1994	399 Women with lung cancer and 399 controls matched for sex, year of birth, and year of death	Petrochemical air pollution	Mortality	RR for high vs. low petrochemical air pollution index, 1.66 (95% CI, 1.05–2.61); RR for high vs. low non-petrochemical air pollution index, 1.24 (95% CI, 0.79–1.95); RR for urban vs. rural, 0.92 (95% CI, 0.56–1.49).

RR of lung cancer incidence or mortality associated with exposure to air pollution. Most of the studies were small: the largest, by Xu ZY and colleagues (1989), included 1249 cases of lung cancer, the next largest, 399 (Yang CY et al. 1999), and the three others, 265 or fewer. Four studies characterized air pollution exposure of cases and controls in terms of residential proximity to one or more stationary sources using various approaches, and the Indian study (Gupta D et al. 2001a) compared residents in urban and rural locations. None reported estimated RRs for exposure to measured ambient concentrations of specific pollutants, though one study (Pisani et al. 2006) reported estimated RRs according to an exposure index based on emissions of SO_2 and NO_2 from a power plant. Four studies included smokers, and, of these, three reported results adjusted to control for the effects of tobacco smoking.

Estimates from the five studies are plotted in Figure 69. The largest study, by Xu ZY and colleagues (1989), asked subjects to classify their outdoor residential environment according to its level of "smokiness": not smoky, somewhat or slightly smoky, or smoky. They reported estimated RRs of 1.5 and 2.3 among men (for the comparison between slightly smoky and not smoky and between smoky and not smoky, respectively). These estimates were adjusted for effects of age, education level, smoking status, and indoor air pollution concentration. They estimated that the risk of lung cancer among men and women residing < 1 km from a smelter was approximately 3 times that among subjects > 3 km from it. There was no evidence of a smoothly increasing gradient of air pollution with decreasing distance from the smelter among women in analyses that adjusted for age, educational level, smoking status, indoor air pollution concentrations, and employment at the smelter. However, duration of residence within 200 m of industrial factories for more than 20 years was associated with 70% and 60% increases in the risk of lung cancer among men and women, respectively.

Yang CY and colleagues (1999) studied 399 housewives in Taipei,China, with lung cancer and 399 controls matched for age and year of birth (and death) in municipalities that

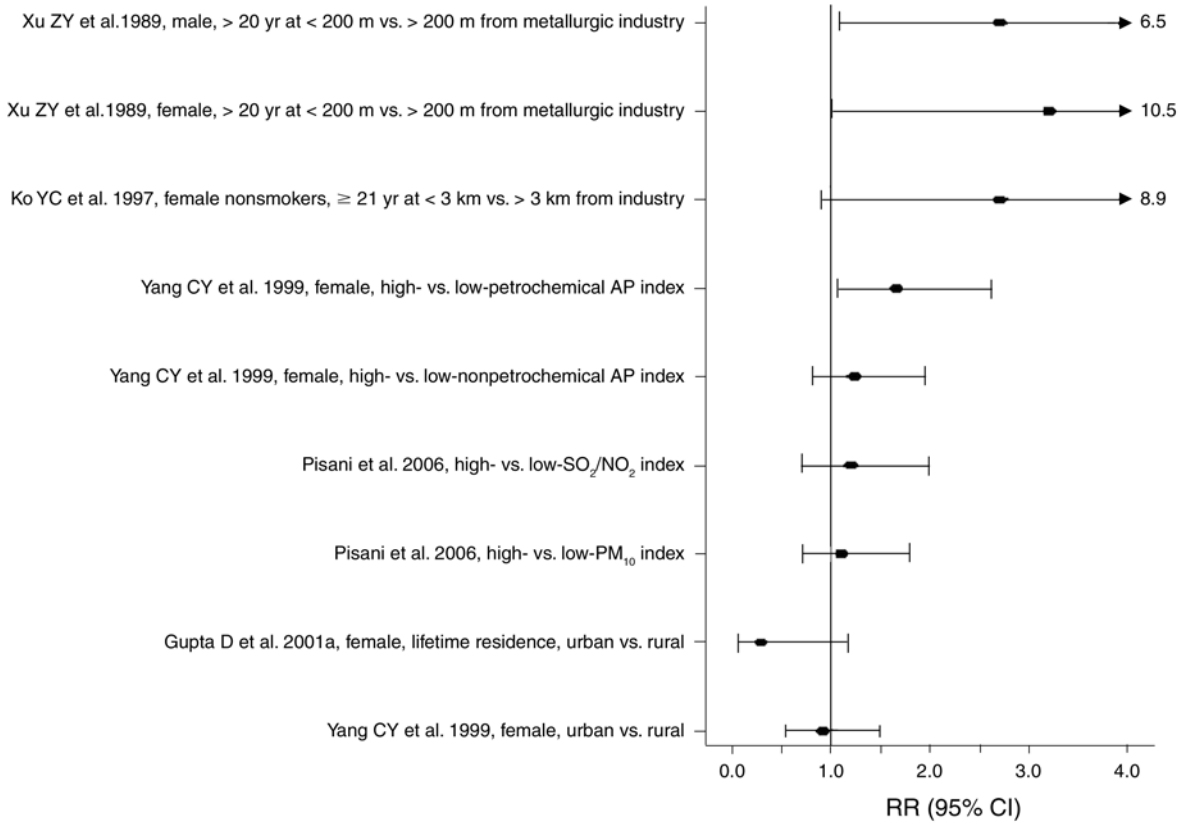

Figure 69. RRs (and 95% CIs) for lung cancer from quantitative comparisons. Y-axis labels give study information in the following sequence: reference citation, sex of subjects (if applicable), smoking status (if applicable), and comparison of interest, including air pollution (AP) index levels. For the Xu ZY et al. 1989 study, > 20 years' residence < 200 m from an industrial area was compared with ≤ 20 years' residence. For the Ko YC et al. 1997 study, ≥ 21 years' residence < 3 km from an industrial area was compared with < 21 years' residence. For study details, see Table 20.

differed with regard to the proportion of residents employed in the petrochemical industry and the level of urbanization. No information was available on tobacco smoking. Rates of lung cancer were elevated among residents in municipalities with the highest proportion of residents employed in the petrochemical industry but not among residents in the most urbanized municipalities.

The one study we identified that was restricted to nonsmokers (106 cases of lung cancer in women), and thus was less prone to confounding by tobacco smoking, reported no significant increase in lung cancer risk among women who had lived near an industrial district for up to 20 years (Ko YC et al. 1997). However, the authors did report an estimated RR of 2.7 (95% CI, 0.9 to 7.8) among those with more than 21 years' residence, after statistical adjustment for age, exposure to indoor air pollution from cooking, and socioeconomic status.

Pisani and colleagues (2006) conducted a small, hospital-based case–control study of 211 lung cancer cases, in which they attempted to quantify cumulative exposure to SO_2 and NO_2 as well as PM, using models based on emissions from power plants. They reported small increases in the risk of lung cancer that did not vary monotonically with the estimated level of cumulative exposure to either SO_2 and NO_2 or PM, in analyses that adjusted for sex, age, and tobacco-smoking status but not exposure to indoor air pollution (Figure 69). However, the controls included individuals hospitalized for causes related to air pollution exposure, such as cardiovascular disease, further complicating the interpretation of the reported results.

The only Indian study (Gupta D et al. 2001a) found no significant association between the incidence of lung cancer among men and lifetime residence in areas classified by the investigators as either mixed urban and rural or > 75% urban, as compared with areas that were > 75% rural (Figure 69). They also reported a statistically imprecise protective effect of lifetime urban residence among women, though exposure to indoor air pollution from burning of solid fuels, which is associated with a risk of lung cancer among women, was apparently not adjusted for in the analysis.

Discussion

Given the small number of studies of lung cancer in our review, and the even smaller number with control for major potential confounders, the evidence regarding air pollution and lung cancer is quite limited. The two studies that have addressed potential confounding by strong risk factors such as tobacco smoking and indoor air pollution from the burning of solid fuels suggest that exposure to outdoor air pollution is associated with an increased risk of lung cancer, with estimated RRs in the range of 1.5 to 3.0. This is generally consistent with results reported in studies from Europe and North America (Vineis et al. 2004; Samet and Cohen 2006), but we identified no Asian studies that quantify excess risk in relation to measured concentrations of air pollution and also address the major potential confounders, tobacco smoking and indoor combustion of solid fuels. The current studies do not allow for confident assessment of the relative effects of individual pollutants and are largely focused on air pollution from large stationary sources. To date, there have been no studies in Asia like those conducted in Europe that specifically address the effects of mobile-source air pollution (Nyberg et al. 2000; Nafstad et al. 2003). Further research is also needed to quantify the relative effects of outdoor and indoor air pollution on lung cancer in various Asian locales and to characterize the relative impact of these two types of air pollution over time.

ADVERSE PREGNANCY OUTCOMES

Studies in our review reported effects of air pollution exposure on either low birth weight or preterm delivery or both. The majority of studies were conducted in Taipei,China (10), with a few studies also conducted in China (3) and South Korea (3). Of the five studies that focused on ambient air concentrations in general, all but one addressed exposure in the first and third trimesters. Studies that reported pollutant-specific risk estimates focused mainly on PM and SO_2, though a handful of studies also explored associations with NO_2 and CO.

Because most studies used data obtained from administrative birth records, control for potential confounders and assessment of effect modification was generally limited to routinely collected information. For example, to avoid effect modification by birth order or multiple births, nearly all studies focused on first, singleton, live births. Maternal characteristics, including maternal age, education level, marital status, and occupation were often addressed. Most studies also adjusted for infant sex, season, and birthplace. None of the studies addressed parental smoking, however.

Preterm Delivery

Ten studies focused on preterm delivery, consistently defined as less than 37 weeks of gestation, with gestational age, where clearly specified, based on last date of menstruation. Seven of the studies were conducted in Taipei,China, two in China, and one in South Korea (Table 21 and Figure 70).

All but two studies focused on specific sources of exposure, with exposures defined according to proximity to

Table 21. Studies of Preterm Delivery

Citation	Study Location	Study Years	Study Sample	Primary Source of Exposure	Exposure Classification	Pollutants	Exposure Windows	Estimated Relative Risk (95% CI)	Adjustments
Chen Z et al. 1995	Guangzhou, China	1985–1992	7695 Newborns: 325 with adverse outcomes and 390 controls	Petrochemical industry	Proximity to petrochemical plant: < 2 km vs. ≥ 5 km	Petrochemical air pollution	Full term	2.13 (0.60–7.64)	Occupation and ethnicity, possibly others
Leem et al. 2006	Incheon, South Korea	2001–2002	52,113 Singleton births	Ambient air pollution	Exposure quartiles based on neighborhood-level air pollution	CO, PM_{10}, NO_2, and SO_2	1st, 2nd, and 3rd trimesters (only 1st shown here)	For CO, PM_{10}, NO_2, and SO_2, respectively: 1.26 (1.11–1.44), 1.27 (1.04–1.56), 1.24 (1.09–1.42), and 1.21 (1.01–1.46)	Maternal age, parity, sex, season, parental education
Lin MC et al. 2001a	Kaohsiung, Taipei, China	1993–1996	498,755 First, singleton, live births	Petrochemical industry	Residence in petroleum refinery area	Petrochemical air pollution	Full term	1.41 (1.08–1.82)	Maternal age, season, marital status, maternal education, infant sex
Tsai SS et al. 2003c	Taipei, China	1994–1997	14,545 Births in industrial areas and 49,670 births in other areas (controls)	Industrial areas	Residence within 2 km of industrial source	Industrial air pollution	Full term	1.11 (1.02–1.21)	Maternal age, season, marital status, maternal education, infant sex, birthplace
Tsai SS et al. 2004	Taipei, China	1993–1996	23,072 First, singleton, births	Thermal power plants	Proximity to thermal power plant: 0–3 km vs. 3–4 km	Industrial air pollution	Full term	1.14 (1.01–1.30)	Maternal age, season, marital status, maternal education, infant sex, birthplace

Table continues next page

Table 21 (*Continued*). Studies of Preterm Delivery

Citation	Study Location	Study Years	Study Sample	Primary Source of Exposure	Exposure Classification	Pollutants	Exposure Windows	Estimated Relative Risk (95% CI)	Adjustments
Xu X et al. 1995a	Beijing, China	1988	25,370 Births	Ambient air pollution (with coal-stove use in 93% of study households)	Continuous exposure	SO_2 and TSP	Full term	For SO_2, 1.21 (1.01–1.46); for TSP, 1.10 (1.01–1.20)	Outdoor temperature and humidity, day of week, season, maternal age, infant sex, residential area
Yang CY et al. 2002b	Kaohsiung, Taipei, China	1993–1996	57,127 Singleton births	Petrochemical industry	Residence in petrochemical area	Petrochemical air pollution	Full term	1.18 (1.04–1.34)	Maternal age, season, marital status, maternal education, infant sex
Yang CY et al. 2002a	Taipei, China	1993–1996	39,750 Singleton births	Petrochemical industry	Residence in petrochemical industrial area	Petrochemical air pollution	Full term	1.03 (0.94–1.13)	Maternal age, marital status, maternal education, infant sex
Yang CY et al. 2003a	Sanming District, Taipei, China	1992–1997	6251 First, singleton, live births	Freeway	Residence within 500 m of freeway vs. 500–1500 m	Traffic emissions	Full term	1.30 (1.03–1.65)	Maternal age, season, marital status, maternal education, infant sex
Yang CY et al. 2004a	51 Boroughs around 3 refineries in Taipei, China	1994–1997	57,483 First, singleton, live births	Petroleum refineries	Residence within 3 km of petroleum refinery	Petrochemical air pollution	Full term	1.14 (1.01–1.28)	Maternal age, season, marital status, maternal education, infant sex

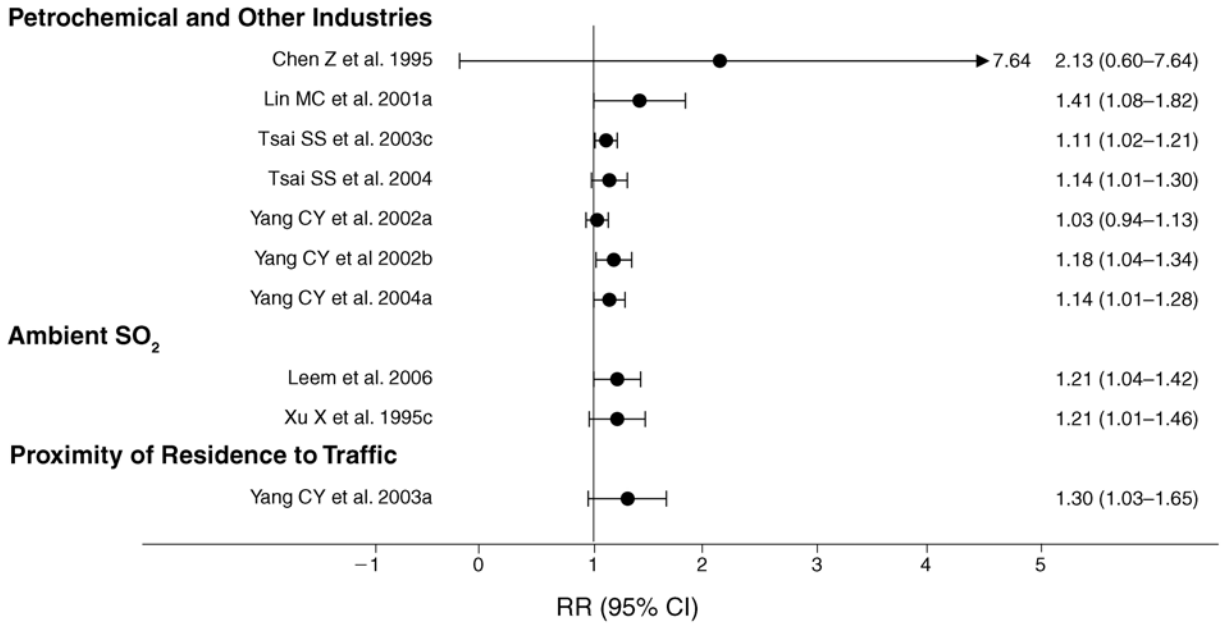

Figure 70. RRs (and 95% CIs) for preterm delivery from qualitative sources of exposure, according to study citation. Y-axis labels in bold type specify the source of exposure. For study details, see Table 21.

petrochemical industries, thermal power plants, industrial sources, or freeways. With a few exceptions, the studies compared differences in exposure between different areas of residence. As a result, the studies estimated risks associated with exposure over the full term of pregnancy, rather than within specific trimesters. The studies consistently demonstrated an increased risk of preterm delivery in association with living in close proximity to pollution sources.

Two studies focused on measures of ambient air pollution. One (Leem et al. 2006) assessed trimester-specific risks of preterm delivery associated with neighborhood levels of air pollution. In analyses comparing residents in neighborhoods with the highest and lowest quartiles of exposure, the authors found an increase in risk by 26%, 27%, 24%, and 21% to be associated with increased exposure to CO, PM_{10}, NO_2, and SO_2, respectively, during the first trimester. Trends toward increased risk with increased exposure were observed for CO, NO_2, and SO_2 during the second trimester. The estimated risks of preterm delivery in relation to increased exposure during the third trimester were somewhat lower, with only those related to increased CO and NO_2 concentrations remaining statistically significant.

The second study that included quantitative ambient measures of pollution was conducted in Beijing at a time when over 93% of households were using coal stoves (Xu X et al. 1995c). Thus, measured ambient pollutant concentrations were extremely high; mean and maximum SO_2 concentrations during the study period were 102 µg/m^3 and 630 µg/m^3, respectively, and mean and maximum TSP concentrations were 10 µg/m^3 and 630 µg/m^3. The study assessed the effects of exposures from the time during the first trimester when women were enrolled through the time of delivery. Interquartile increases in SO_2 and TSP concentrations were associated with 21% and 10% increases in the risk of preterm delivery, respectively.

Low Birth Weight

Eight studies explored the relation between air pollution exposure and low birth weight, defined as a weight < 2500 g (Table 22). Like studies of preterm delivery, these studies focused primarily on specific sources of exposure, mainly proximity to petrochemical industries and household coal use. Unlike the studies of preterm delivery, however, six of these eight studies included quantitative measures of ambient pollution associated with these sources. Four studies explored associations with PM (measured as either TSP or PM_{10}) and SO_2 (Wang X et al. 1997; Ha et al. 2001; Lee BE et al. 2003a; Lin CM et al. 2004b), two of which also assessed associations with CO (Ha et al. 2001; Lee BE et al. 2003a).

The studies that assessed exposures during the first trimester found small but consistent associations between

Table 22. Studies of Low Birth Weight

Citation	Study Location	Study Years	Study Sample	Primary Source of Exposure	Exposure Classification	Pollutants	Exposure Windows	Estimated Relative Risk (95% CI)	Adjustments
Chen Z et al. 1995	Guangzhou, China	1985–1992	7695 Newborns: 325 with adverse outcomes and 390 controls	Petrochemical industry	Proximity to petrochemical plant: < 2 km vs. ≥ 5 km	Petrochemical air pollution (pollutant not specified)	Full term	3.10 (1.00–9.54)	Occupation and ethnicity, possibly others
Ha et al. 2001	Seoul, South Korea	1996–1997	276,763 Births	Ambient air pollution	Interquartile increase in pollutant concentration	CO, SO_2, and TSP	1st and 3rd trimesters	CO: 1st trimester, 1.08 (1.04–1.12); 3rd trimester, 0.91 (0.87–0.96) SO_2: 1st trimester, 1.06 (1.02–1.10); 3rd trimester, 0.93 (0.88–0.98) TSP: 1st trimester, 1.04 (1.00–1.08); 3rd trimester, 0.95 (0.90–0.99)	Unclear
Lee BE et al. 2003a	Seoul, South Korea	1996–1998	388,105 Singleton births	Ambient air pollution	Interquartile increase in pollutant concentration	CO, SO_2, and PM_{10}	1st and 3rd trimesters	CO: 1st trimester, 1.04 (1.01–1.07); 3rd trimester, 0.96 (0.93–0.99) SO_2: 1st trimester, 1.02 (0.99–1.06); 3rd trimester, 0.96 (0.91–1.00) PM_{10}: 1st trimester, 1.03 (1.00–1.07); 3rd trimester, 1.00 (0.95–1.04)	Time trend, gestational age, infant sex, birth order, maternal age, parental education

Table continues next page

Table 22 (*Continued*). Studies of Low Birth Weight

Citation	Study Location	Study Years	Study Sample	Primary Source of Exposure	Exposure Classification	Pollutants	Exposure Windows	Estimated Relative Risk (95% CI)	Adjustments
Lin MC et al. 2001b	Lin-yuan (exposed area) and Taihsi (control area), Taipei, China	1993–1996	2545 Births	Petrochemical industry	Exposed vs. control	Petrochemical air pollution (pollutant not specified)	Full term	1.77 (1.00–3.12)	Maternal age and education, season, marital status, infant sex
Lin CM et al. 2004b	Kaohsiung, and Taipei, China	1995–1997	92,288 Full-term, singleton, live births	Petrochemical industry	Low vs. high	SO_2 and PM_{10}	3rd Trimester and full term	SO_2: 3rd trimester, 1.20 (1.01–1.41); full term, 1.26 (1.04–1.53) PM_{10}: 3rd trimester, 0.97 (0.81–1.17); full term, 0.87 (0.71–1.05)	Gestational age, infant sex, birth order, season of birth, maternal age; for PM_{10}, additional adjustments for maternal education and for other pollutants
Lin CM et al. 2004a	Kaohsiung, and Taipei, China	1995–1997	92,288 Residents living within 3 km of an air quality monitoring station	Petrochemical industry	Exposed vs. control	Industrial air pollution (pollutant not specified)	Full term	1.13 (1.03–1.24)	Gestational age, infant sex, birth order, season of birth, maternal age and education
Wang X et al. 1997	Beijing, China	1988–1991	74,671 First, live births	Household coal use	100-μg/m^3 Increase	SO_2 and TSP	3rd Trimester	SO_2: 1.11 (1.06–1.16) TSP: 1.01 (1.01–1.01)	Gestational age, residential area, maternal age, year of birth, infant sex
Yang CY et al. 2002a	Taipei, China	1993–1996	39,750 Singleton births	Petrochemical industry	Exposed vs. control	Petrochemical air pollution (pollutant not specified)	Full term	1.07 (0.95–1.22)	Maternal age and education, marital status, infant sex

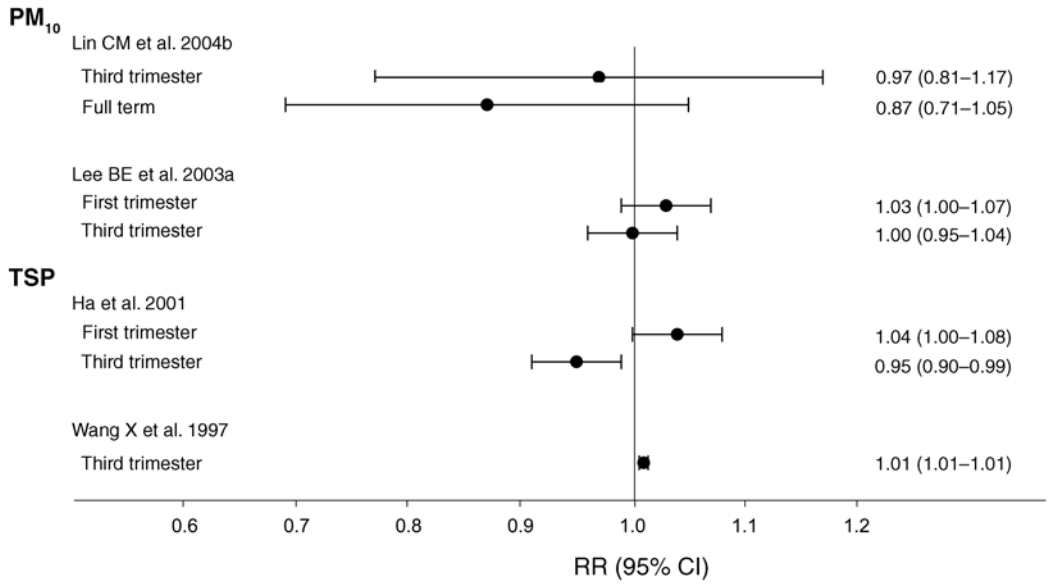

Figure 71. RRs (and 95% CIs) for low birth weight from PM_{10} or TSP exposure, according to study citation and period of exposure. Y-axis labels in bold type specify the type of PM. For study details, see Table 22.

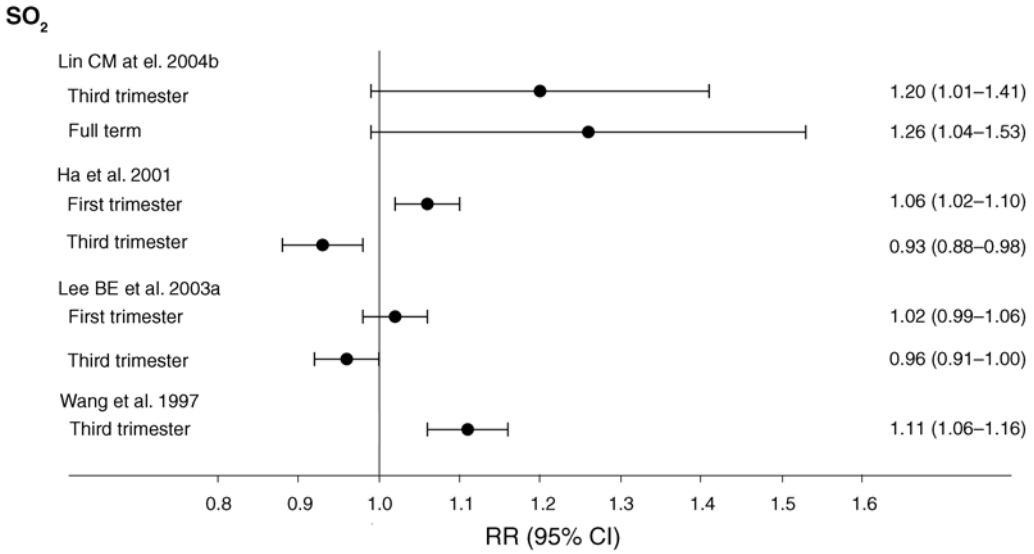

Figure 72. RRs (and 95% CIs) for low birth weight from SO_2 exposure, according to study citation and period of exposure. For study details, see Table 22.

low birth weight and PM exposure (Ha et al. 2001; Lee BE et al. 2003a) (Figure 71). Increased exposure to PM in the first trimester was associated with a 3%–4% increased risk of low birth weight, with estimated declines in birth weight of 5.2 g (95% CI, 1.9–8.5) for every 10-μg/m^3 increase in PM_{10} (Yang CY et al. 2003a) and of 6.06 g (95% CI, 3.85–8.27) for every interquartile change in TSP (Ha et al. 2001). However, full-term or third-trimester exposures to PM had smaller associations, or no significant association, with low birth weight (Wang X et al. 1997; Ha et al. 2001; Lee BE et al. 2003a; Lin CM et al. 2004b).

The evidence for SO_2 and CO was similar to that for PM (Figures 72 and 73, respectively). Associations with low birth weight of magnitude similar to those seen with PM were observed for first-trimester exposures, with lesser effects observed for third-trimester or full-term exposures.

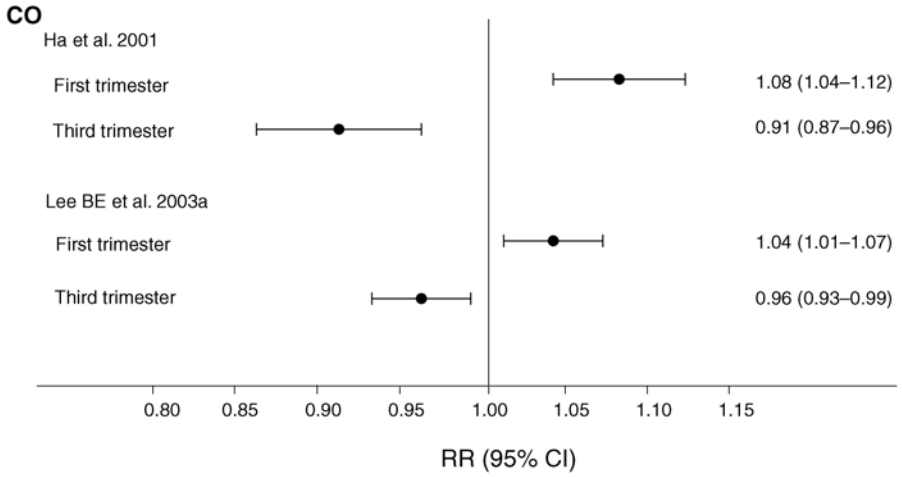

Figure 73. RRs (and 95% CIs) for low birth weight from CO exposure, according to study citation and period of exposure. For study details, see Table 22.

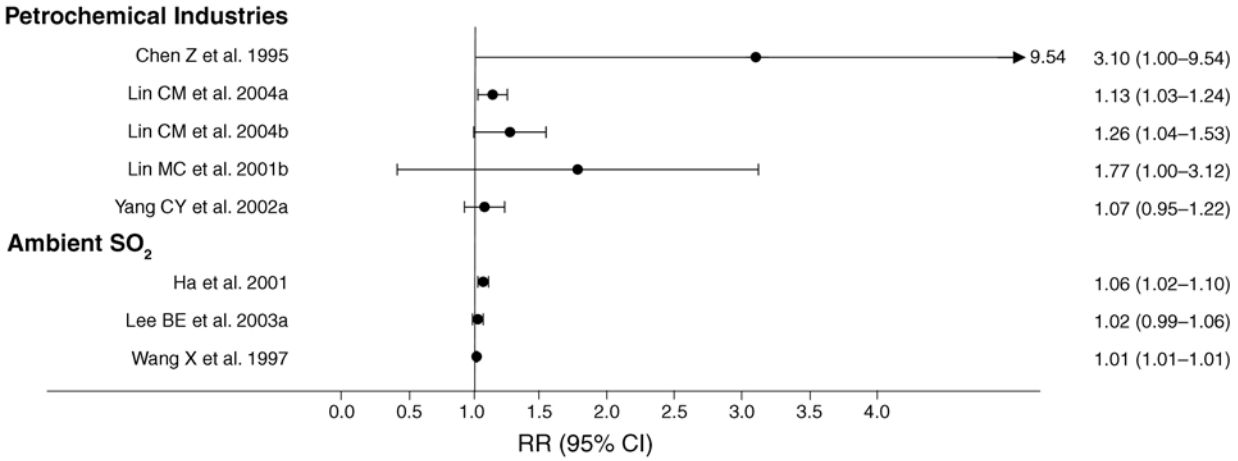

Figure 74. RRs (and 95% CIs) for low birth weight from qualitative sources of exposure, according to study citation. Y-axis labels in bold type specify the source of exposure. For study details, see Table 22.

Each 10-μg/m^3 change in SO_2 during the first trimester was associated with a 5.2 g (95% CI, 0.9–26.3) decrease in birth weight (Yang CY et al. 2003a); each interquartile change in SO_2 was associated with an 8.06 g (95% CI, 5.59–10.53) decrease in birth weight (Ha et al. 2001).

The four studies with qualitative measures of pollution (Chen Z et al. 1995; Lin MC et al. 2001a; Yang CY et al. 2002a; Lin CM et al. 2004a) all focused on proximity to the petrochemical industry (Figure 74). The study conducted in Guangzhou, China (Chen Z et al. 1995) found an extremely high risk of low birth weight (RR, 3.10; 95% CI, 1.00–9.54) among women living < 2 km from a petrochemical plant as compared with women living ≥ 5 m away. The other three studies, conducted in Taipei,China, suggested lower risks, with women living in a petrochemical–industrial municipality having an elevated risk of low birth weight, around 1.07 to 1.77 times the risk among those living in cleaner municipalities.

Discussion

Consistent with the larger global literature on air pollution and adverse pregnancy outcomes, the Asian literature suggests relatively small risks associated with exposure to air pollution, with exposure early in pregnancy appearing to be most strongly associated with an increased risk of low birth weight and preterm delivery (Glinianaia et al. 2004b; Ritz and Wilhelm 2008). Although the evidence suggests that early pregnancy is the critical window during which air pollutants can affect fetal growth, a better understanding of the biological mechanisms of such an effect is needed before causality can be assessed (Ritz and Wilhelm 2008).

Similar to studies conducted elsewhere, the Asian studies took advantage of existing data from birth records; this reliance on routinely collected data provides for a larger sample size but also limits the ability to fully address potential effect modifiers, especially those that have not been documented on the birth records (Slama et al. 2008). For example, because none of the Asian studies addressed parental smoking, potential confounding by smoking remains an issue (Šrám et al. 2005). The use of routinely collected data also increases the potential for exposure misclassification (Slama et al. 2008).

Most of the selected Asian studies focused on specific sources of exposure, usually industrial sources. Even though some studies did include quantitative measures of pollutants, the paucity of studies of ambient air pollution exposures limits the ability to compare this body of evidence with the larger literature.

SUMMARY AND CONCLUSIONS

We assessed the evidence that long-term exposure to ambient air pollution has adverse effects on chronic respiratory disease, lung cancer, or adverse reproductive outcomes in Asia. A rigorous search was conducted to ascertain all important peer-reviewed articles published within the relevant time frame. The literature is diverse, and the studies vary widely in quality of design, presentation, and analysis. For this reason, no summary effect estimates were calculated. However, wherever possible, we calculated effect estimates for standardized increments in measured pollutants. This approach provided additional opportunities for quantitative summarization and comparison of results. Despite these challenges and constraints, some limited conclusions can be drawn. Overall, the available studies provide evidence that long-term exposure to ambient air pollution in Asia is associated with chronic respiratory illness, reduced lung function, lung cancer, and adverse reproductive outcomes. This evidence is broadly consistent with the evidence from other regions. Although publication bias cannot be completely ruled out, several studies reported no significant association with exposure, and it seems unlikely that the finding across regions would be as generally consistent if the results had been significantly influenced by publication bias.

We found no cohort studies that reported mortality estimates for Asian populations exposed over the long term to high concentrations of air pollution. The lack of studies of cardiovascular morbidity and mortality from IHD and stroke is particularly noteworthy, given the burden of disease attributable to these conditions, as discussed in *Section II. Development, Air Pollution Exposure, and Population Health*. However, the current evidence from Asia suggests that these effects are likely. Both COPD and reduced pulmonary function are associated with increased mortality and reduced life expectancy, both in smokers and in nonsmokers (Sin and Man 2005; Sin et al. 2005).

There is also evidence from cross-sectional studies conducted in China of mortality from chronic disease associated with long-term exposure to PM (characterized as TSP) and other combustion-source pollutants (Lave and Seskin 1977). (These studies were similar in design to some of the early U.S. studies that first identified effects air pollution exposure.)

In the 1990s, the WHO, UNEP, and Chinese collaborators conducted cross-sectional studies of mortality in two large Chinese cities that are very heavily polluted: Benxi (Jin et al. 1999) and Shenyang (Xu Z et al. 2000), centers of the iron and steel industries in the country. Annual average concentrations of TSP ranged from 290–620 $\mu g/m^3$ among nine areas of Benxi and 353–560 $\mu g/m^3$ among three areas of in Shenyang; the corresponding annual average SO_2 concentrations were 160–240 $\mu g/m^3$ and 75–212 $\mu g/m^3$. Effects of TSP and SO_2 on mortality from all natural causes and mortality from cardiovascular causes (coronary heart disease [CHD] and cerebrovascular disease) and respiratory diseases were estimated for 1993–1994 in Benxi and 1992 in Shenyang, on the basis of a linear regression of mortality rates adjusted for current age on current average pollution concentrations among the study areas in the two cities. In Benxi, a 100-$\mu g/m^3$ increase in TSP concentration was associated with estimated RRs for mortality from CHD, COPD, and cerebrovascular disease of 1.24 (95% CI, 1.08–1.41), 1.24 (95% CI, 1.04–1.44), and 1.08 (95% CI, 1.00–1.15), respectively. In Shenyang, the air pollution measurements were used to construct a three-level index of air pollution. The estimated RRs for mortality from CHD, cerebrovascular disease, and COPD in association with the highest index level versus the lowest index level were 1.11 ($P < 0.05$), 1.21 ($P < 0.01$), and 1.22 ($P < 0.05$), respectively. The authors

of the Shenyang study also attempted to estimate the effects of exposure on mortality from selected infectious diseases. The rate of mortality from pneumonia in children < 10 years old was considerably lower among residents of the least-polluted of the three areas (0.0 cases/100,000 population) than among residents of the most-polluted area (4.2 cases/100,000 population). The estimated RR for mortality from TB was increased in the most-polluted area (1.91, $P < 0.05$). There was no control for smoking or indoor air pollution from solid fuel use, even at the level of area. This lack of controls for these combustion products adds considerable uncertainty to the reported results. The potential for major confounding may have been limited, however, because estimated RRs were calculated among residents of each city, a procedure that might have resulted in a relatively homogenous distribution of smoking and other potential confounders. In any case, the reported results for cardiovascular and respiratory disease seem broadly consistent with those from the current body of U.S. and European cohort studies, discussed earlier in *Summary of Current Evidence on Health Effects of Air Pollution: Implications for Asia* in Section III.

Section VII. Summary and Conclusions

This review has evaluated the evidence of health effects of outdoor air pollution in developing Asia in the context of ongoing changes in both air quality and population health. The nature of the health risks associated with the natural and built environments changes as economic development occurs. Economic development and its attendant urbanization have been, and continue to be, based in large part on the increased combustion of fossil fuels. Such development has led, in some countries, to impressive reductions in poverty and increased life expectancy but also to a substantial and increasing burden of disease from chronic noncommunicable diseases, such as IHD, cerebrovascular disease, COPD, and cancer. This increasing burden is due to a variety of factors, most importantly to larger numbers of people living to older ages and to the increased prevalence of tobacco smoking, increasing rates of obesity, and changes in dietary patterns. Economic development and poverty reduction have also led to gradual decreases in environmental risks at the household level, such as indoor air pollution from the burning of dirty fuels and poor water quality, although the burden of disease associated with these exposures in young children and women remains substantial.

Despite improvements in air quality across the region, air pollution concentrations in Asian cities often greatly exceed current WHO health-based air quality guidelines and many current national standards. In a large number of studies worldwide, air pollution has been found to adversely affect people with chronic cardiovascular and respiratory diseases, and it may also contribute to the development of those diseases in otherwise healthy people. Thus, even as air quality has improved in some ways, there remains an important impact on public health, which may grow as populations age and rates of chronic disease and urbanization increase.

Air quality in Asia reflects complex and evolving relations between increased energy consumption for transportation and power generation and measures being taken to improve air quality. From past experience in Western countries, it seems clear that substantial increases in combustion of fossil fuels for power generation and transportation can improve economic conditions in developing Asia and elsewhere, but can also, if not controlled, have important consequences for human health and environmental quality. It is also clear that effective approaches to controlling and reducing pollution exist. Investment in these approaches need not necessarily impede economic growth and, on the basis of documented experience in developed countries and emerging evidence in Asia, the developing countries of Asia may be able to avert increased environmental degradation and associated health impacts while reducing poverty and providing economic security for their populations (CAI-Asia Center 2008a; U.S. EPA 2008). Overall, estimates of pollution emissions as well as measured and estimated ambient concentrations indicate that air quality is improving throughout much of urban Asia. Trends in air quality have largely shown improvement during periods of dramatically increased energy use in Asia, a testament to the impact of effective air quality management as well as improved energy efficiency and reduced intensity of energy use. Climate change and emissions of GHGs present a considerable challenge for Asia but also an opportunity for continued improvement in air quality. Strategies for reducing GHG emissions, though directed toward climate change, may also have direct impacts on local and regional air quality, resulting in faster and larger improvements than would otherwise occur in the region.

As a result of these developments, the need for high-quality research on the health effects of air pollution in Asia has never been greater, and the scientific community is responding with an increasing number of studies of the effects of exposure to air pollution on morbidity and mortality due to cardiovascular or respiratory diseases, adverse reproductive outcomes, and other health outcomes. The over 400 studies identified and summarized in the PAPA–SAN database provide substantial evidence of serious health effects of exposure to outdoor air pollution in the developing countries of Asia. The spectrum of adverse health effects associated with air pollution exposure ranges from acute and chronic respiratory symptoms and changes in pulmonary function to increased mortality from cardiovascular or respiratory diseases or lung cancer — the same spectrum of adverse health outcomes associated with air pollution in the West.

In particular, time-series studies of the effects of short-term exposure on morbidity and mortality from cardiovascular or respiratory disease continue to provide some of the most current and consistent evidence of serious adverse

health effects in Asia. Asian scientists have used this study design extensively in recent years, resulting in at least 115 studies published as of 2007, an increase by more than 100% in the number published as of 2004 (HEI ISOC 2004). This literature now includes the results of a coordinated multi-center study (HEI Health and Air Pollution in Asia Program 2010), funded under the PAPA program, which provides the type of evidence that has contributed most importantly to international guidelines and science-based regulatory policies in Europe and North America (Samet et al. 2000b; WHO 2005b; HEI ISOC 2010 [this report]).

The updated meta-analysis of Asian time-series studies presented in this review is now based on results from 82 publications, more than 3 times the number in the 2004 review (HEI ISOC 2004). As a result, it provides more reliable and detailed estimates of the magnitude of the effect of exposure on daily mortality and hospital admissions in Asian populations and allows for more definitive comparisons of Asian evidence with results from other regions. In our meta-analysis, short-term exposure to PM_{10} was estimated to increase daily mortality from all natural causes by 0.27% (95% CI, 0.12–0.42) per 10-μg/m^3 increase in concentration, an effect similar to that reported in meta-analyses and multi-city studies in Europe, North America, and Latin America. Increased daily mortality from cardiovascular disease, chiefly IHD and stroke, the major current and future causes of death of adults in the region, underlies this estimate. Consistently larger exposure-related increases in all-natural-cause, cardiovascular, and respiratory mortality were also observed among older people, who represent an increasingly large proportion of Asian populations.

In our review, there was little evidence of publication bias — specifically, inaccuracy in summary estimates due to selective reporting of results by investigators or selective publication of studies reporting increased risk. As noted earlier in *Section V. Quantitative Assessment of Daily Time-Series Studies*, publication bias may be less of an issue in coordinated multi-city studies, such as the PAPA studies, that apply consistent methods of analysis to data from multiple locations according to a prespecified protocol and report results in a comprehensive, uniform, and rigorous fashion.

THE PAPA STUDIES

The four PAPA studies conducted in China and Thailand constitute the first designed and coordinated multi-city set of studies of the health effects of air pollution in Asia. As such, they provide a unique, if limited, picture of the short-term impact of current ambient concentrations of particulate air pollution on mortality in four large metropolitan areas in East and Southeast Asia (Bangkok, Hong Kong, Shanghai, and Wuhan). In the combined analysis of the city-specific results (Wong CM et al. 2008b, 2010a), a 10-μg/m^3 increase in PM_{10} concentration was associated with an increase of 0.6% (95% CI, 0.3–0.9) in the daily rate of death from all natural causes; this estimate is similar to or greater than those reported in multi-city studies in the United States and Europe. This proportional increase in daily mortality is seen at levels of exposure (mean PM_{10} concentration, 51.6 to 141.8 μg/m^3) that were several times higher than those in most large Western cities, and in each of the four Asian cities, daily mortality continues to increase over a fairly large range of daily average ambient PM_{10} concentrations, up to several hundred micrograms per cubic meter.

Wong CM and colleagues (2008b, 2010a) report a summary estimate for the initial four cities of the PAPA program that exceeds the overall meta-analytic summary estimate for the Asian studies for all pollutants and outcomes (with the exception of respiratory mortality, for which the PAPA estimate is lower) (see Table 15). The reasons are unclear. To some extent, the larger combined estimate may be due to the markedly higher estimates from Bangkok (Vichit-Vadakan et al. 2008, 2010), though excluding Bangkok from the combined analysis of the PAPA studies still resulted in larger estimates (Wong CM et al. 2008b, 2010a) than those in the meta-analysis in this review. The systematic selection of air quality data, and quality control of those data, according to an explicit protocol may have resulted in reduced measurement error in the PAPA studies, though this factor is difficult to evaluate. Stochastic variation, or the play of chance, is another possible explanation. Whatever the reason, addition of the PAPA city-specific estimates to those derived from the other time-series studies identified in this review caused relatively minor changes in the summary estimates: for instance, the increase in all-natural-cause mortality per 10-μg/m^3 increase in PM_{10} concentration was estimated at 0.33% (95% CI, 0.16–0.51) after the PAPA estimates were added, versus 0.27% (95% CI, 0.12–0.42) without these four studies. Preliminary results from PAPA-funded studies in two Indian cities, Delhi (Rajarathnam et al. 2010) and Chennai (Balakrishnan et al. 2010), also show increased rates of all-natural-cause mortality in association with short-term exposure to PM_{10} that are broadly consistent with the results of the current meta-analysis.

The studies in Bangkok, Hong Kong, Shanghai, and Wuhan, were designed to provide a combined picture of the effects of short-term exposure to pollution on daily mortality across the four cities, but each study also explored more detailed aspects of the epidemiology of

exposure to air pollution in each location, providing additional insight into how factors such as weather and social class might modify the estimated RR of health effects of air pollution. The study in Wuhan, one of China's "oven cities" (cities that experience very high temperatures and humidity), found that the estimated RR may increase by a factor of 5 at extremely high temperatures, as compared with temperatures typical of temperate zones (Qian Z et al. 2008, 2010). The studies in Hong Kong (Wong CM et al. 2008a, 2010b) and Shanghai (Kan H et al. 2008, 2010) found evidence of higher estimated RRs among the economically disadvantaged and those with the least education, respectively, corroborating the results of earlier studies in Western cities (O'Neill et al. 2003).

SYSTEMATIC AND CRITICAL REVIEW OF STUDIES OF CHRONIC EFFECTS OF LONG-TERM EXPOSURE TO AIR POLLUTION

The Asian literature on the chronic effects of long-term exposure to air pollution is more limited than the literature from Europe and North America, especially with regard to chronic cardiovascular disease. The design and quality of the studies also vary widely. Nonetheless, the results of this review suggest that long-term exposure to air pollution from a variety of combustion sources is contributing to chronic respiratory disease in both children and adults, to lung cancer, and to adverse reproductive outcomes in Asian populations.

The prevalence of chronic phlegm, a symptom of chronic respiratory disease indicating long-term exposure to inhaled irritants, was found to be associated with exposure to combustion-source air pollution both in qualitative comparisons among areas with differing levels of pollution and in quantitative comparisons of measured levels of air pollution. Studies that controlled for major potential confounding factors, including tobacco smoking and indoor air pollution from the burning of solid fuels, reported ORs generally between 1.1 and 5.0, regardless of how pollution was characterized.

A similarly diverse and overlapping collection of studies also provides some evidence for an increased prevalence of asthma and asthma-related symptoms in association with exposure to air pollution from a variety of sources. Most studies showed an elevated prevalence in association with air pollution exposure, though the estimated increase was small, with ORs greater than 1.0 but less than 1.5.

There is limited evidence regarding a connection between air pollution and lung cancer in Asian populations. The two studies that have addressed potential confounding by strong risk factors, such as tobacco smoking and indoor air pollution from the burning of solid fuels, report estimated RRs for lung cancer in the range of 1.5 to 3.0, consistent with ranges from studies in Europe and North America. But more definitive evidence will require larger studies to estimate effects of exposure at current and past high concentrations using actual measured concentrations, of air pollution and adequate control for potential confounding by those strong risk factors.

The Asian literature suggests that the risks of adverse reproductive outcomes, low birth weight, and preterm delivery in association with exposure to air pollution are relatively small. Overall, the estimated RRs are between 1.04 and 2.0. Exposure in early pregnancy appears to be most strongly associated with risk. These results are generally consistent with those from the larger global literature on air pollution and adverse pregnancy outcomes.

IMPLICATIONS FOR ASSESSMENT OF HEALTH IMPACTS

Very large populations are exposed to high concentrations of air pollution in developing Asia. Indeed, 30 million people currently reside in just the first four cities studied in the PAPA project. Thus, the estimated effects of both short- and long-term exposures reviewed in this report, though small in relative terms, probably translate to large numbers of illnesses and deaths attributable to air pollution. The WHO estimated that in 2000 over 500,000 deaths in Asia were due to outdoor air pollution exposure, accounting for approximately two thirds of the global total attributed to air pollution. Indoor air pollution from use of solid fuel contributed an additional 1.1 million deaths (WHO 2002). Other impact assessments have reported similar estimates (World Bank and SEPA 2007). That said, air pollution is only one of many factors that affect the health of people in developing Asia (Ezzati et al. 2002). Nonetheless, the substantial health impacts of exposure to air pollution should be of concern to policy makers faced with difficult decisions regarding transport and energy policy.

In Asia and elsewhere, an increased life expectancy is a major social benefit of economic growth and its attendant, if often variable, reductions in poverty. Brunekreef and colleagues (2007) argue that exposure to air pollution acts to reduce healthy life expectancy, shortening lives by months and even years, on average, in some populations. Although the time-series studies reviewed here document the occurrence of excess mortality related to short-term exposure, the study results cannot currently be used directly to estimate reductions in life expectancy due to extended exposure (Burnett et al. 2003; Rabl 2006). Such estimates are provided by cohort studies, in which large numbers of individuals exposed to various concentrations

of air pollution are observed for years and the mortality rates in each exposure group are compared. To date, no cohort studies of long-term exposure to air pollution and mortality from chronic cardiovascular or respiratory disease appear to have been reported in Asia. Virtually all recent estimates of the health impacts of air pollution in Asia, including those made by the WHO (WHO 2002) and the World Bank and SEPA (World Bank and SEPA 2007), are based on the results of the ACS study (Pope et al. 2002), the largest and most extensively reviewed cohort study.

The broad consistency of the results of Asian time-series studies of mortality with those in Europe and North America, including the evidence of greater rates of cardiovascular morbidity and mortality among older people than among younger people, supports the continued use of data from Western cohort studies to estimate the burden of disease attributable to air pollution in Asia. However, developing Asia currently differs from the United States and Europe with regard to energy use, air quality, and population health, which are also dynamically changing. Thus, estimates of the impact of air pollution that are based on even the most carefully executed U.S. studies must be used with caution. One key area of uncertainty is the shape of the concentration–response function relating long-term exposure to air pollution and mortality from chronic disease. The concentrations of $PM_{2.5}$ studied in the ACS study were much lower than the concentrations in major cities in China and India, requiring that analysts extrapolating the ACS data to Asia make projections regarding the shape of the concentration–response function at much higher concentrations. The uncertainty in the resulting estimates, when quantified in sensitivity analyses, was substantial (Cohen et al. 2004). The concentration–response functions describing short-term exposure to air pollution and daily mortality recently reported for Hong Kong, Shanghai, and Wuhan (Wong CM et al. 2008b, 2010a) may help to inform future extrapolation of results from U.S. cohort studies to Asian populations in highly polluted cities, but Asian cohort studies are needed to provide more definitive estimates.

ENHANCED EVIDENCE OF EFFECTS OF AIR POLLUTION IN ASIA

Based on findings from more than 80 Asian time-series studies, including coordinated multi-city time-series studies, the meta-analytic estimates appear consistent in both direction and magnitude with those from other regions. In broad terms, the effects of short-term exposure in Asian cities are on a par with those observed in hundreds of studies worldwide. The same pollutants — RSP and gaseous pollutants such as O_3, SO_2, and NO_2 — affect older people with chronic cardiovascular or respiratory disease. The adverse effects in some locales, reported in studies published in the 1980s and 1990s, may reflect the effects of air pollution concentrations that have subsequently improved. However, more recent studies continue to report adverse effects at lower levels in cities in Thailand and Japan, where air quality has improved, as well as in heavily polluted Chinese and Indian cities.

The results of our meta-analysis of time-series studies should serve to reduce concerns regarding the generalizability of the results of the substantial, but largely Western, literature on the effects of short-term exposure to air pollution. They suggest that neither genetic factors nor longer-term exposure to highly polluted air substantially modifies the effect of short-term exposure on daily mortality in major cities in developing Asia. This evidence provides support for the notion, implicit in the approach taken in setting the WHO world air quality guidelines (Krzyzanowski and Cohen 2008), that incremental improvements in air quality are expected to improve health, even in areas with relatively high ambient concentrations. The results also suggest that health benefits would result from further reductions in exposure to pollution concentrations below those specified in the WHO guidelines.

The results of the studies of chronic effects reviewed in this report appear to be broadly consistent with those of studies in other regions, suggesting that long-term exposure to air pollution causes chronic pulmonary disease and other adverse effects that result in reduced life expectancy. That said, these studies are more susceptible than the time-series studies to uncontrolled confounding by strong risk factors, such as tobacco smoking and indoor air pollution from the burning of solid fuels, and factors related to socioeconomic status, such as diet. These risk factors may also modulate the effect of air pollution, leading to larger effects in some population groups and smaller effects in others. Some of these factors, such as those related to the level of economic development, may be particularly important in developing Asia. If they are ignored or poorly measured, an inaccurate estimate of the effects of air pollution may result, and real differences among study results in various regions may be obscured.

This literature review documents a number of promising improvements in air quality in Asian cities, even in the context of economic growth. However, susceptibility to the effects of air pollution in Asia can be expected to rise because rates of chronic cardiovascular and respiratory disease increase as populations age, exposure to combustion-source air pollution becomes more widespread owing to urbanization, vehicularization, and industrialization, and risk factors increase in prevalence. In future assessments,

these changes may yield larger health impacts in the region, although the effects of these changes could be counterbalanced by improved access to medical care and other improvements in the standard of living. Higher estimates of the magnitude of air pollution effects may also contribute to larger impact estimates. For example, the most recent publication from the ACS study (Krewski et al. 2009) reported larger estimates of the risks of cardiovascular mortality and lung cancer than previously reported.

KNOWLEDGE GAPS AND RESEARCH NEEDS

The acute toxicity of short-term exposure to high air pollution concentrations has been appreciated since the early 20th century, and recent multi-city studies in Europe and North America have identified such toxic effects at even lower concentrations. Therefore, the results of meta-analyses of Asian time-series studies of daily mortality and hospital admissions are not unexpected and can serve as an important part of the scientific basis and rationale for interventions to improve air quality. Nevertheless, there is much we still need to learn in order to fully understand the substantial air pollution challenges in Asia. High-quality, credible science from locally relevant studies will be critical to helping decision makers choose which policies are most likely to result in public health benefits.

How Does the Nature of the Air Pollution Mixture Affect Air Quality, Exposure, and Health Effects?

Health impacts in cities in developing countries of Asia result from exposure to a mixture of pollutants, both particles and gases, which are derived in large part from combustion sources (Harrison 2006). This is true in Europe and North America as well, but the specific sources and their proportional contributions in Asia are different, with a larger share from open burning of biomass and solid waste materials, combustion of lower-quality fuels including coal, and use of two- and three-wheeled vehicles with two-stroke engines. These conditions affect the size distribution and chemical composition of PM, among other aspects of air pollution. (The proportional contribution of these sources is also changing over time in major cities in Asia.) Finally, time–activity patterns, building characteristics, and proximity of susceptible populations to pollution sources in the region also differ from those in Western countries in ways that may affect human exposure and health effects (Janssen and Mehta 2006).

Our current knowledge of these issues is rudimentary, and additional research is needed to inform effective and sustainable control strategies. Without such studies, epidemiologists will have a difficult time assessing the relative effects of various pollution mixtures or specific pollution sources or even interpreting patterns of variation.

What Are the Effects of Long-Term Exposure to Air Pollution?

Although time-series studies will continue to be important potential drivers of environmental and public policy, additional study designs, such as cohort studies like the ACS and Six Cities studies (Pope et al. 2002; Laden et al. 2006), are needed in Asia to estimate the effects of long-term exposure on annual average rates of mortality from chronic cardiovascular or respiratory diseases and impacts on life expectancy, the metrics that may be the most meaningful and relevant to policy. Conducting such studies will be challenging, not least because of rapidly changing air pollution concentrations and exposures in developing Asia; as some U.S. studies suggest, however, if chronic effects are due to recent exposure, this problem may not be severe. Studies of adverse reproductive outcomes and effects on the health of young children will be easier to conduct because the relevant exposures will be more recent. A detailed quantitative review of the larger Asian literature (including cross-sectional studies of chronic respiratory disease and studies of lung cancer and adverse reproductive outcomes) may provide support for extrapolations of the Western studies, but long-term Asian studies will provide the most direct evidence.

It may be possible to "retrofit" existing Asian cohort studies, originally designed to address issues other than air pollution, with estimates of air pollution exposure. This approach was used in the ACS study, but it is not a simple process, as HEI's experience with the pilot study in Guangzhou makes clear (Lam et al. 2009). Retrofitting studies requires the building of multidisciplinary teams of investigators, with commitment of adequate, long-term resources, to work in collaboration with government officials, their industrial counterparts, and local stakeholders. In order to assess the current potential for such studies, HEI's PAPA program issued a Request for Information and Qualifications for teams of investigators to conduct such studies in developing countries of Asia (Health Effects Institute 2009). Based on the responses, it was evident that the potential existed in several locations.

What Do Results of Current Time-Series Studies in a Subset of Asian Cities Tell Us About Health Effects of Air Pollution Exposure in Other, As Yet Unstudied, Asian Locales?

The number of time-series studies being reported from across Asia is growing; the first Indian studies of short-term exposure to RSP and daily mortality, part of the PAPA

research program, will soon be published. Even so, almost all current studies have been conducted in mainland China, Taipei,China, and South Korea. Major population centers in South and Southeast Asia (India, Pakistan, Vietnam, Philippines, Indonesia, and Malaysia) are still largely understudied, with the exception of Bangkok. Differences in the relative prevalence of urban air pollution sources (such as open burning) and urban poverty may modify the effects of exposure. Expanded coordinated multi-city studies, designed and analyzed consistently and conducted across the region, could provide more definitive answers. In some cases, nonmortality outcomes, such as hospital admissions, may also be studied, enabling policy makers to better quantify the health impacts of air pollution.

What Role Does Indoor Air Pollution Play in the Health Effects of Outdoor Air Pollution?

The magnitude and prevalence of exposure to indoor air pollution are high in Asian cities, especially among people living in poverty. We need a better understanding of how air pollution from indoor sources contributes to concentrations of outdoor air pollution and how indoor exposure to air pollution from indoor sources affects risk estimates for outdoor air pollution. Coordinated measurements of exposure and coordinated epidemiologic studies will be needed to address these questions.

What Role Does Poverty Play in the Health Effects of Air Pollution?

Limited evidence, largely from studies in Europe and North America, suggests that economic deprivation increases the rates of morbidity and mortality related to air pollution. One reason may be the higher air pollution exposures experienced by people of lower socioeconomic status. Nevertheless, increased vulnerability can also be caused by factors related to socioeconomic status, such as health status, nutritional status, and access to medical services. Studies of these issues are relatively rare in Asia, where extreme poverty is more prevalent than in the West; therefore, results of Western studies cannot be simply extrapolated, though the recent results of the PAPA studies in Shanghai and Hong Kong are welcome and much-needed additions (Kan H et al. 2008, 2010; Wong CM et al. 2008a, 2010b). Some analyses of U.S. cohort studies suggested that low levels of attained education were associated with larger estimated RRs of air pollution–related mortality (Krewski et al. 2000), but more recent analyses based on extended follow-up of the largest cohort have not upheld this pattern (Krewski et al. 2009). Studies in Asia that examine the effect of exposure on morbidity and mortality from diseases associated with poverty (such as acute lower respiratory infection [ALRI] in children and TB) and studies that estimate effects of exposure across socioeconomic strata are needed. HEI's study of hospital admissions for ALRI in children in Ho Chi Minh City is, to our knowledge, the only example of such a study.

What Are the Health Consequences of Changes in Air Pollution Resulting from Climate Change and Efforts to Reduce Emissions of Climate-Forcing Agents?

Changes in air pollution resulting from climate change and from efforts to reduce emissions of climate-forcing agents may have important consequences for health in the region, especially in low- and middle-income countries. However, major unknowns remain, including the quantitative association between reductions in the concentrations of GHGs such as CO_2 and toxic air pollutants such as $PM_{2.5}$, the relative toxicity of short-lived GHGs with different climate-forcing potentials, such as SO_4^{2-} and BC, and the impact of policy choices. There is also a need to understand more fully how concentration–response functions for air pollution may vary with regard to global and within-region differences in climate, demographics, and pollutant mixes.

Finally, although the ability to conduct research on the health effects of air pollution in developing Asia is improving, it is still constrained by limitations in environmental and public health infrastructures. Air quality monitoring has increased in the region, but there is a need for more extensive systems for monitoring urban air quality that are designed to support health effects studies and impact assessments. There is also a corresponding need for more highly trained professionals in air quality monitoring, exposure assessment, and environmental epidemiology. Equally important, there remain considerable deficiencies in registration of vital statistics in Asia, especially regarding accurate and comprehensive assignment of causes of death. There is also a need to encourage cooperation and collaboration in health effects research between health and environmental scientists and public agencies. These deficiencies constitute a major impediment to environmental health research and, more broadly, to the development of appropriate, evidence-based public health policy.

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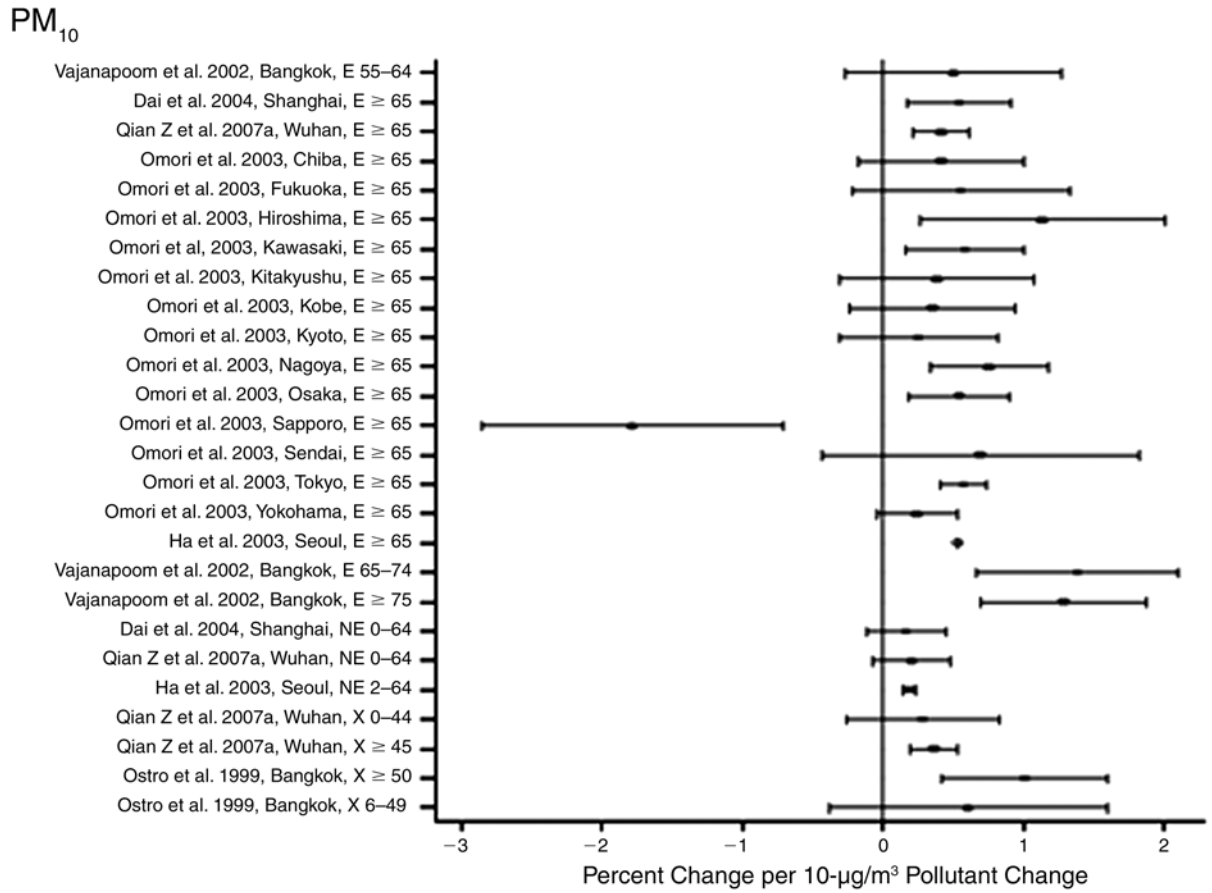
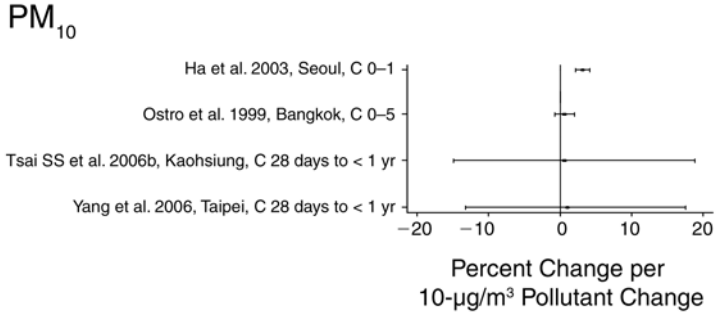
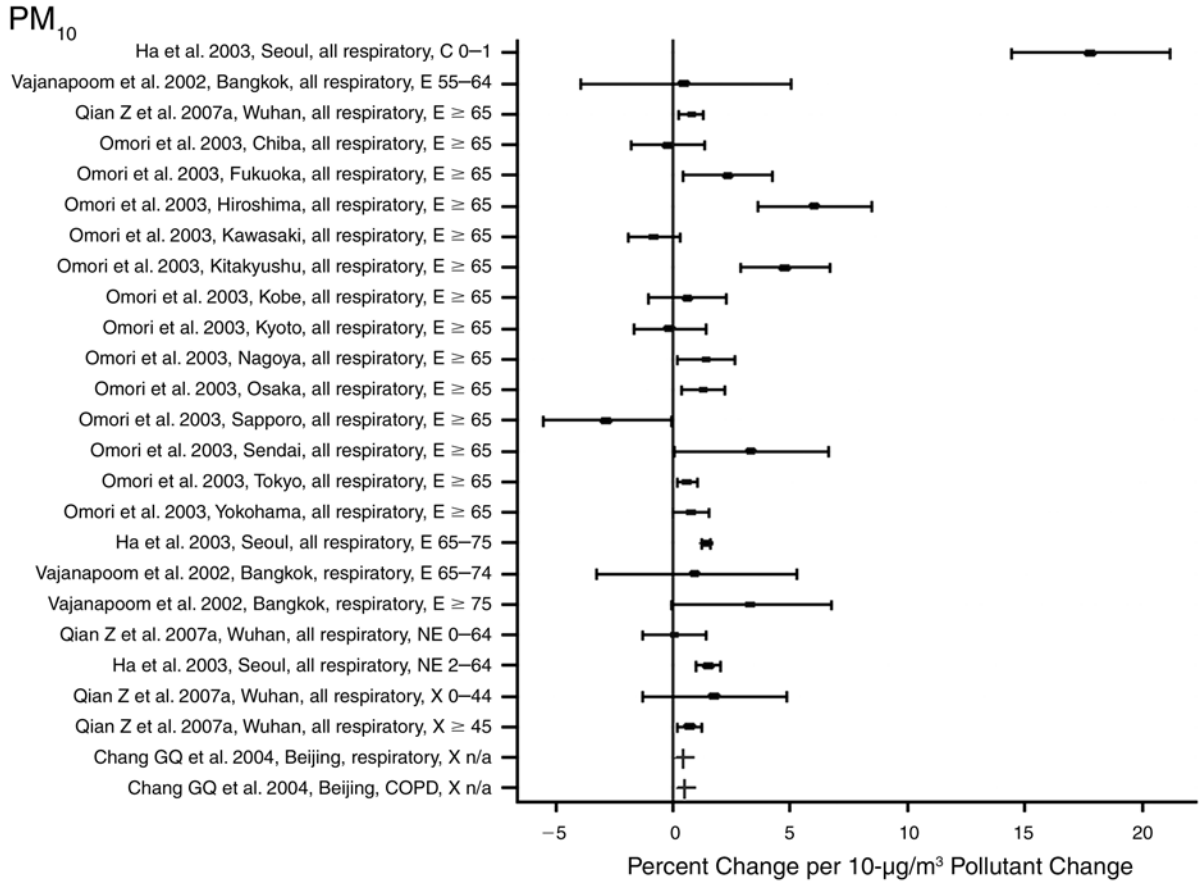
APPENDIX A. Additional Analyses of Daily Time-Series Studies



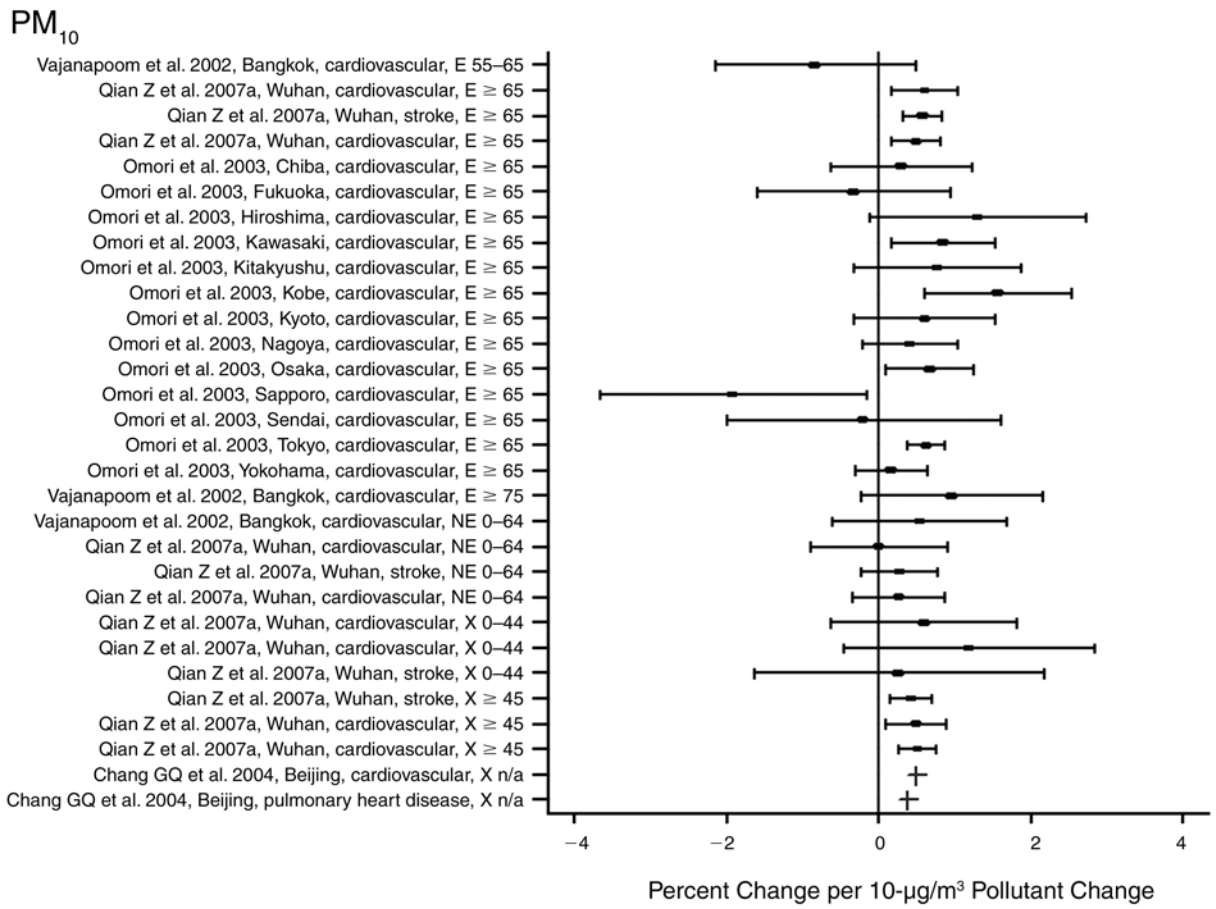
Appendix Figure A.1a. Percent change in the mean number of daily deaths from all natural causes per 10-µg/m³ change in 24-hr mean PM_{10} concentration among adults only. Studies of children and infants were excluded. Y-axis labels give study information in the following sequence: reference citation, study location, and age group: Elderly (E), Not Elderly (NE), or other (X) and years of age.



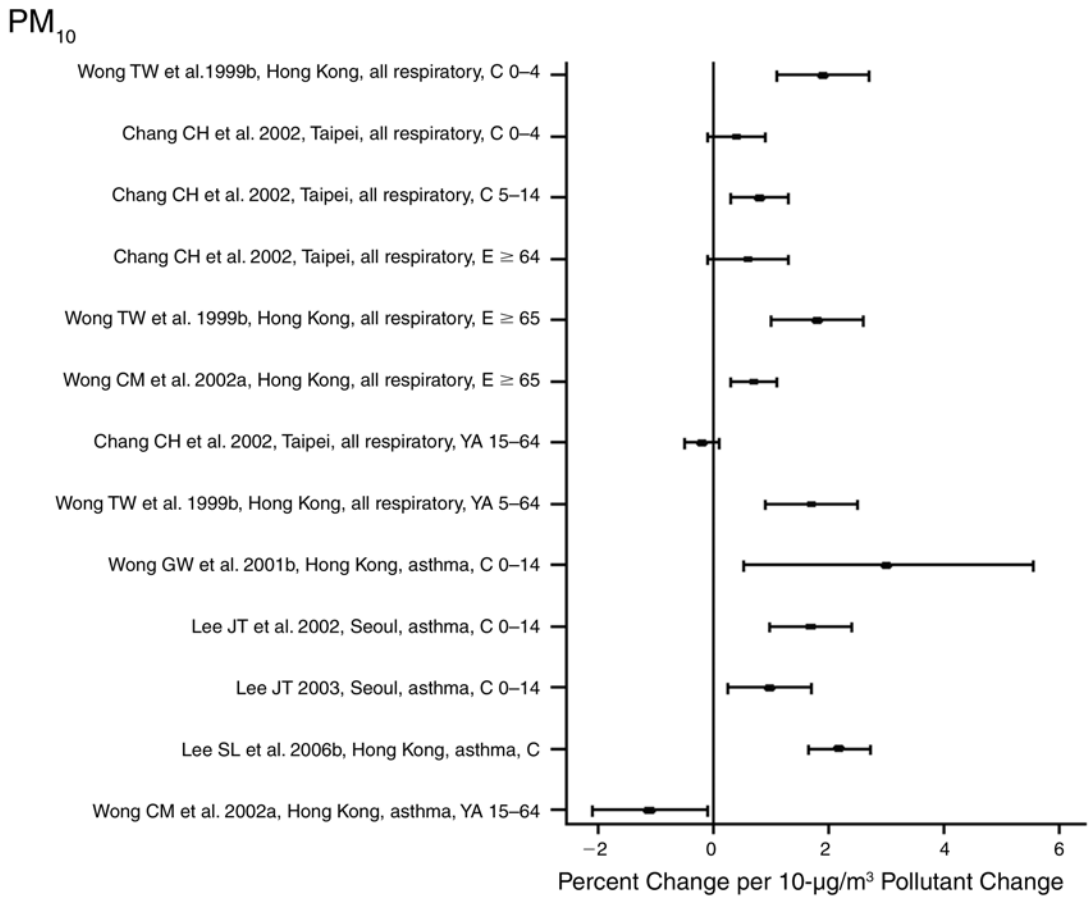
Appendix Figure A.1b. Percent change in the mean number of daily deaths from all natural causes per 10-µg/m³ change in 24-hr mean PM_{10} concentration among children and infants. Y-axis labels give study information in the following sequence: reference citation, study location, and age group. All study populations consisted of children (C).



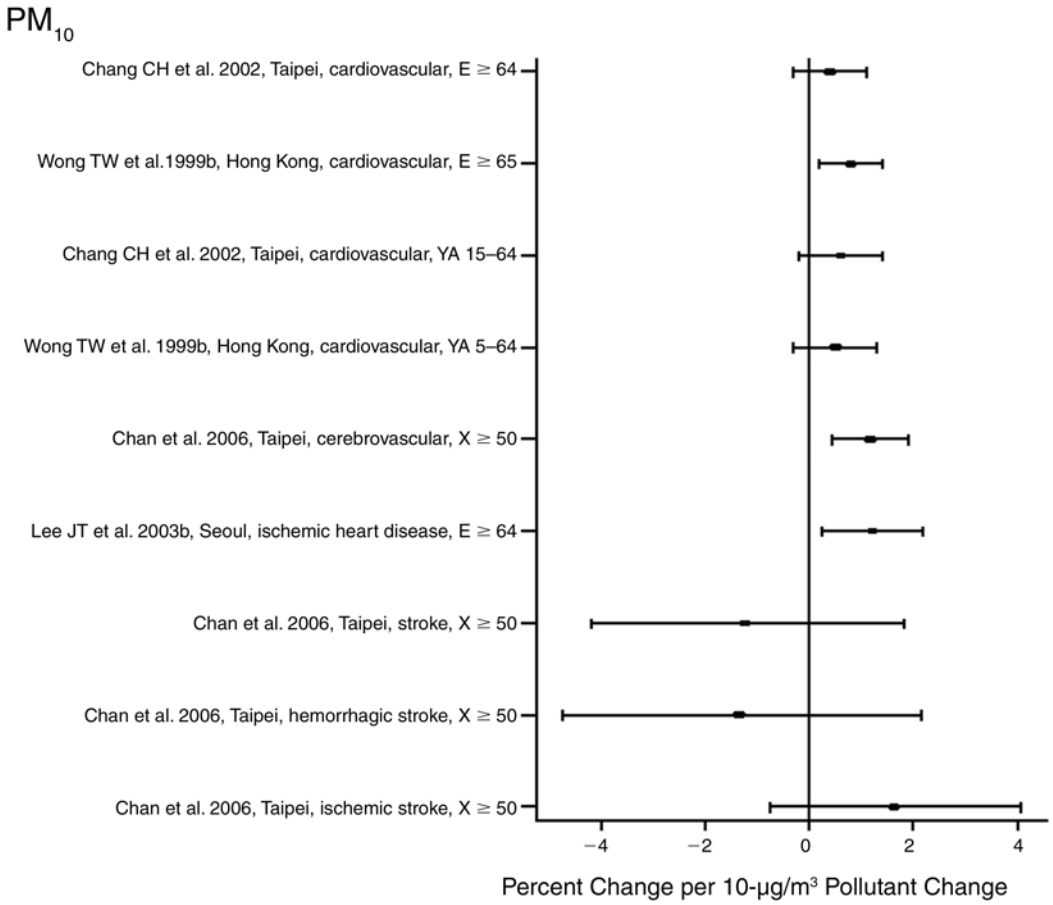
Appendix Figure A.2. Percent change in the mean number of daily deaths from respiratory causes per 10-μg/m^3 change in 24-hr mean PM_{10} concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: Children (C), Elderly (E), Not Elderly (NE), or other (X) and years of age or not applicable (n/a). A data point of "+" indicates the paper reported only a single point estimate with no standard error or confidence interval; the value could not be used in the meta-analysis and only the point estimate is presented.



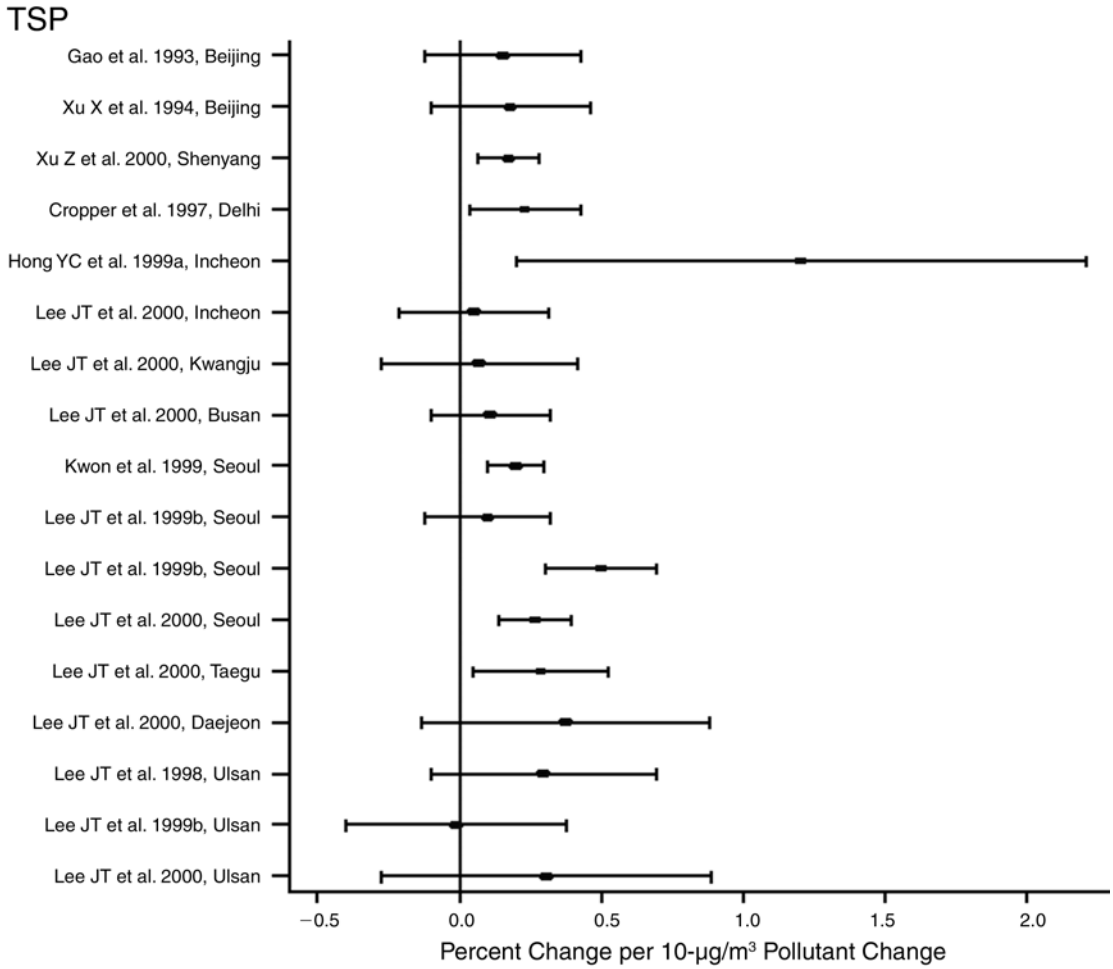
Appendix Figure A.3. Percent change in the mean number of daily deaths from cardiovascular causes per 10-µg/m^3 change in 24-hr mean PM_{10} concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: Elderly (E), Not Elderly (NE), or other (X) and years of age (or not applicable [n/a]). A data point of "+" indicates the paper reported only a single point estimate with no standard error or confidence interval; the value could not be used in the meta-analysis and only the point estimate is presented.



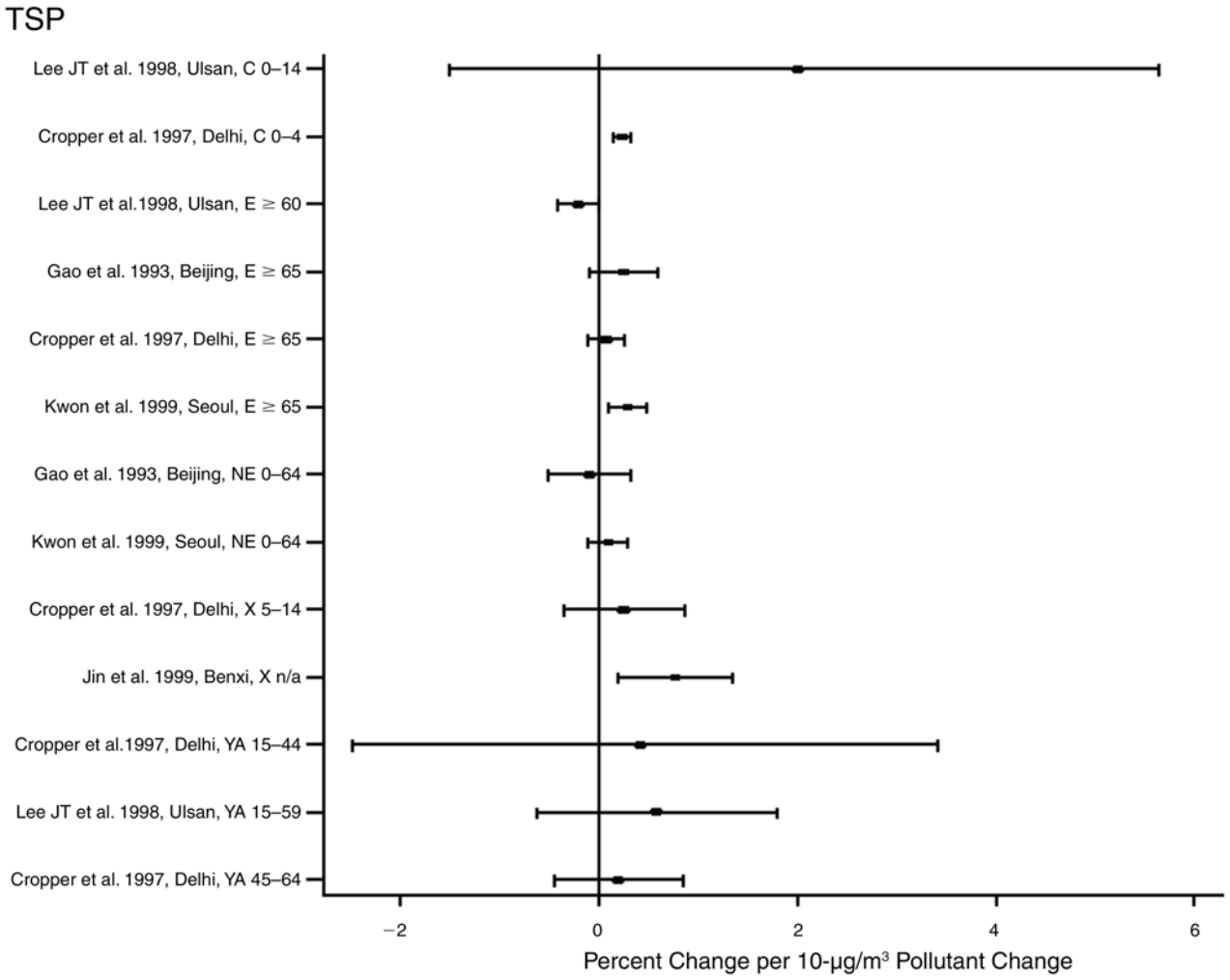
Appendix Figure A.4. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-µg/m^3 change in 24-hr mean PM_{10} concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of hospital admission, and age group: Children (C), Elderly (E), or Young Adult (YA) and years of age.



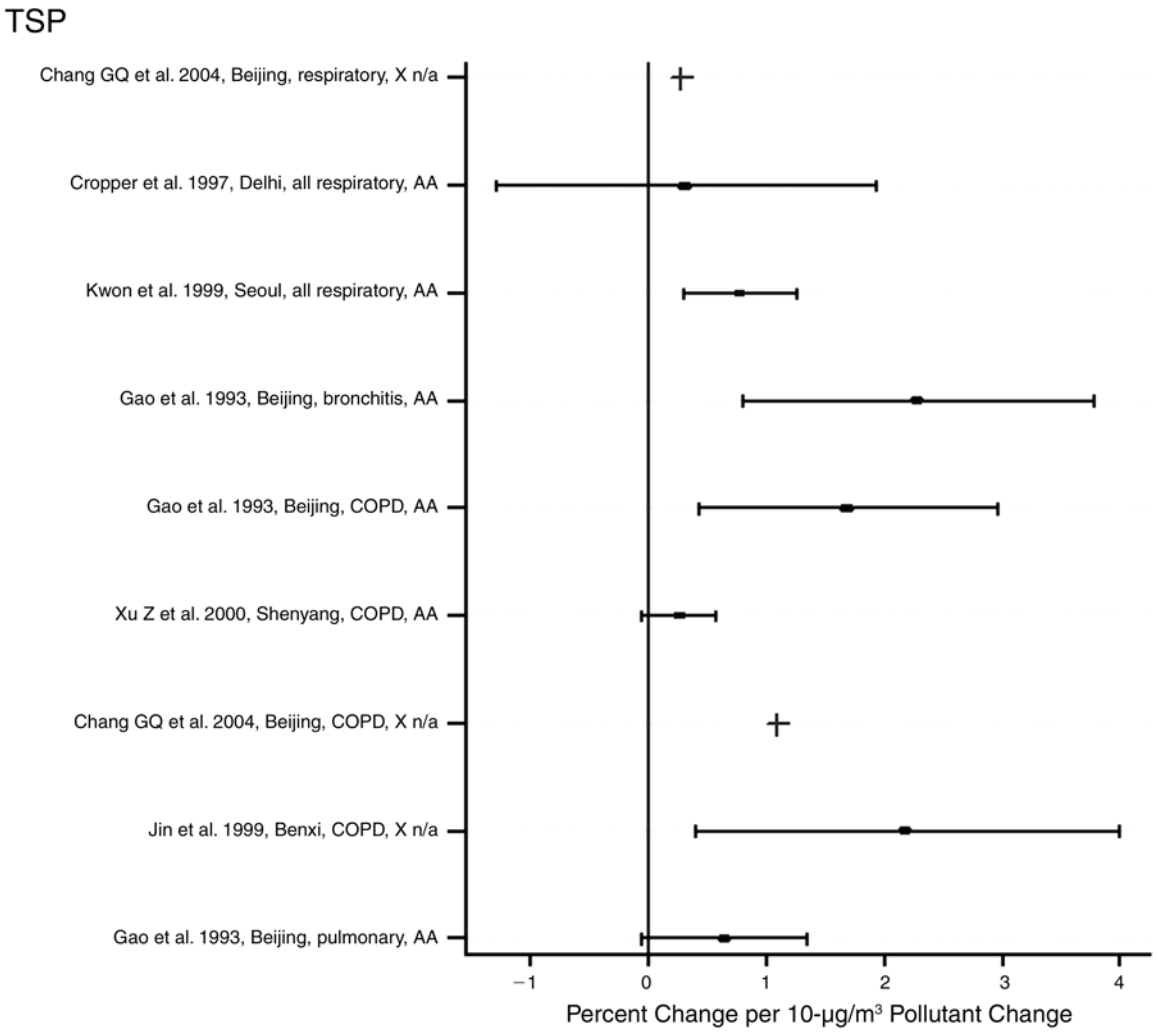
Appendix Figure A.5. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 10-µg/m^3 change in 24-hr mean PM_{10} concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of admission, and age group: Elderly (E), Young Adult (YA), or other (X) and years of age.



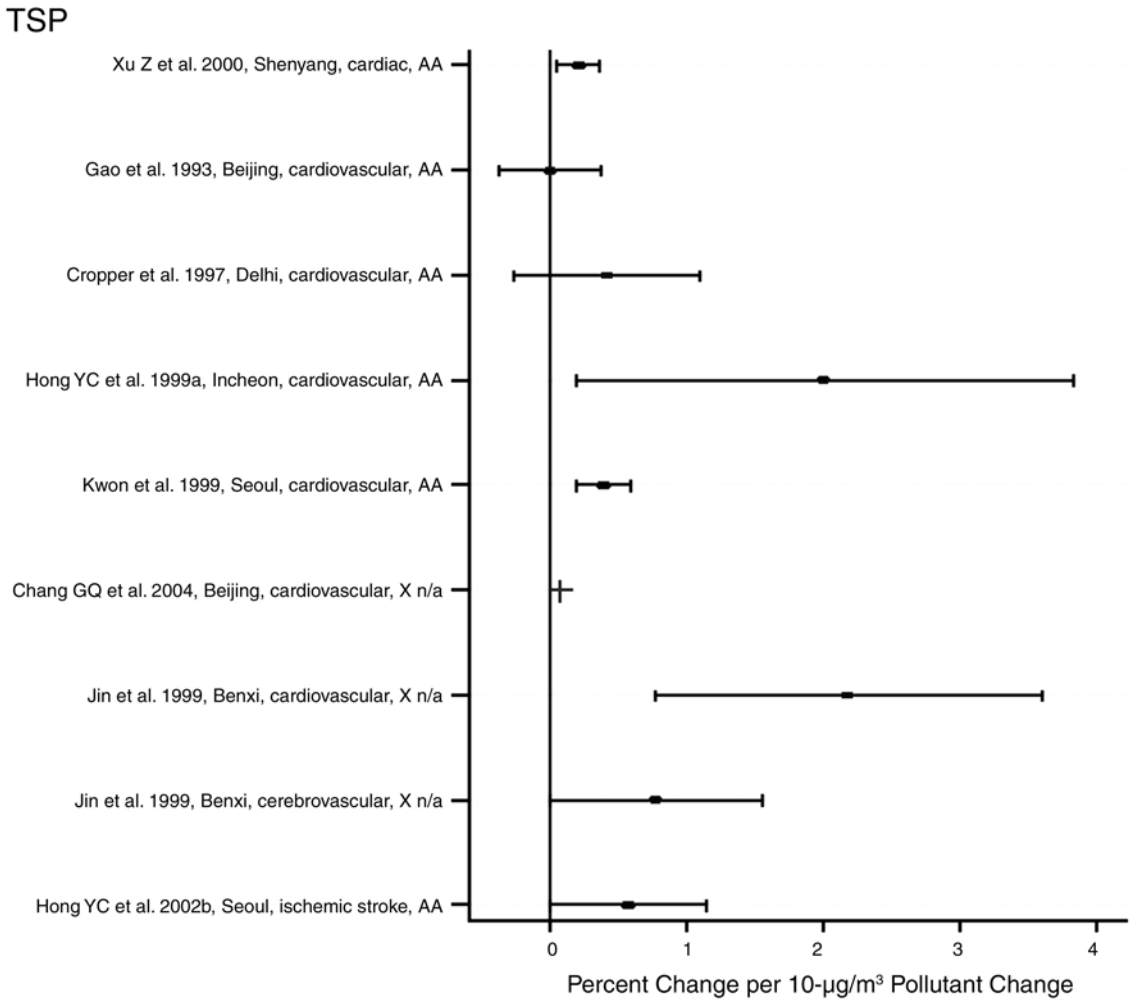
Appendix Figure A.6. Percent change in the mean number of daily deaths from all natural causes per 10-µg/m^3 change in 24-hr mean TSP concentration. Y-axis labels give study information in the following sequence: reference citation and study location.



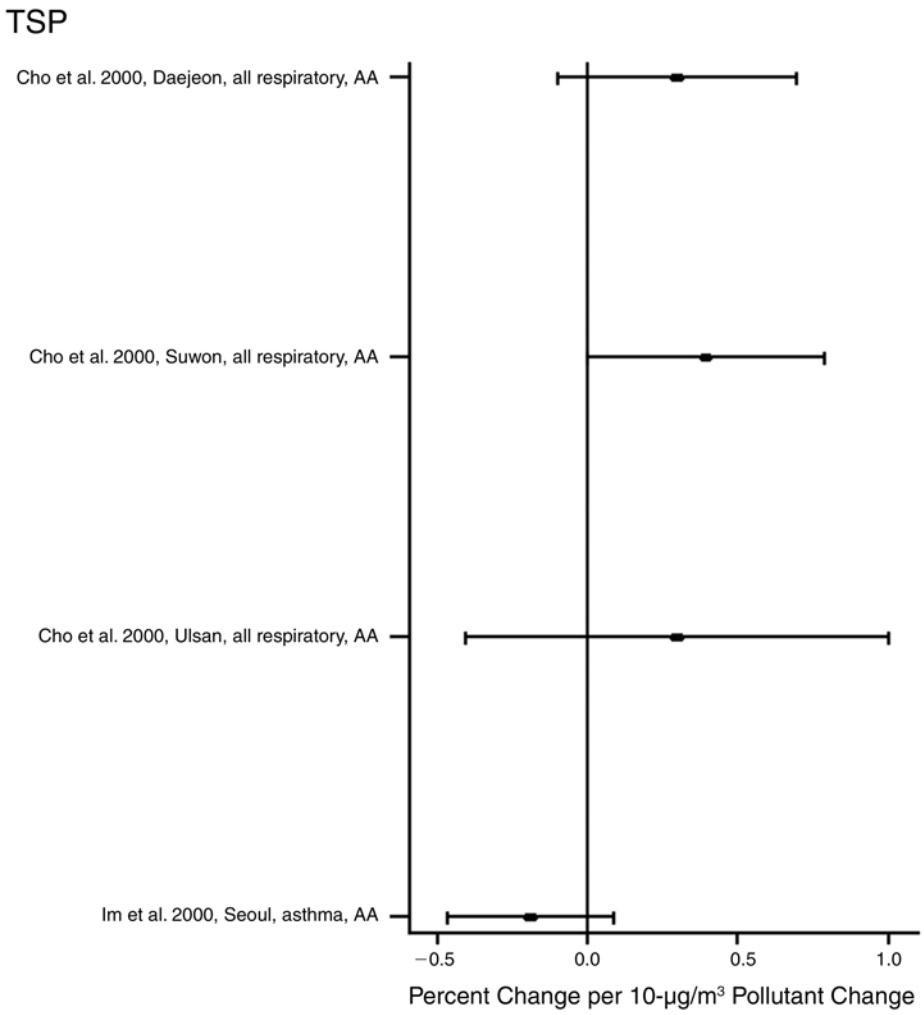
Appendix Figure A.7. Percent change in the mean number of daily deaths from all natural causes per 10-μg/m³ change in 24-hr mean TSP concentration. Y-axis labels give study information in the following sequence: reference citation, study location, and age group: Children (C), Elderly (E), Not Elderly (NE), other (X), or Young Adult (YA) and years of age (or not applicable [n/a]).



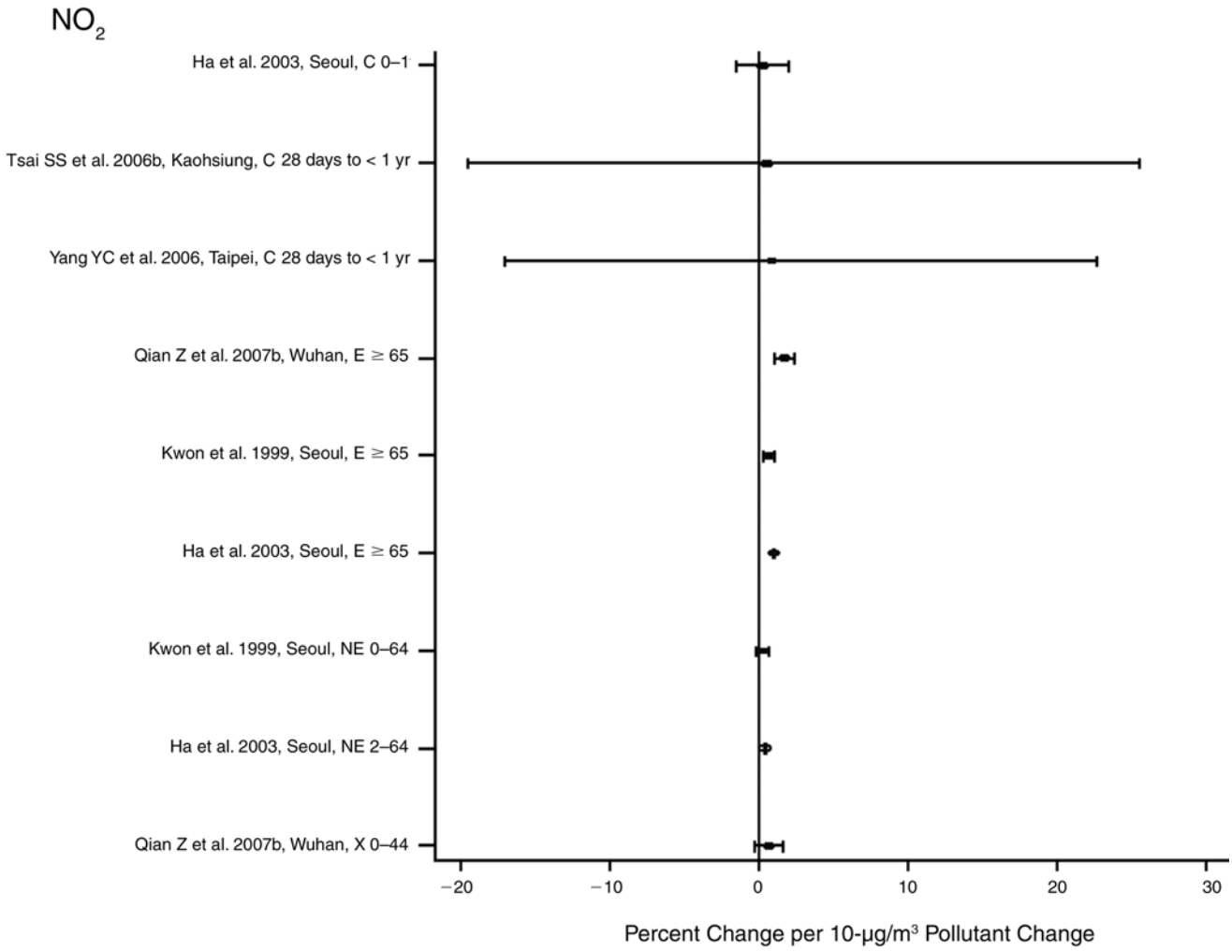
Appendix Figure A.8. Percent change in the mean number of daily deaths from respiratory causes per 10-µg/m^3 change in 24-hr mean TSP concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: other (X, n/a) or All Ages (AA). A data point of "+" indicates the paper reported only a single point estimate with no standard error or confidence interval; the value could not be used in the meta-analysis and only the point estimate is presented.



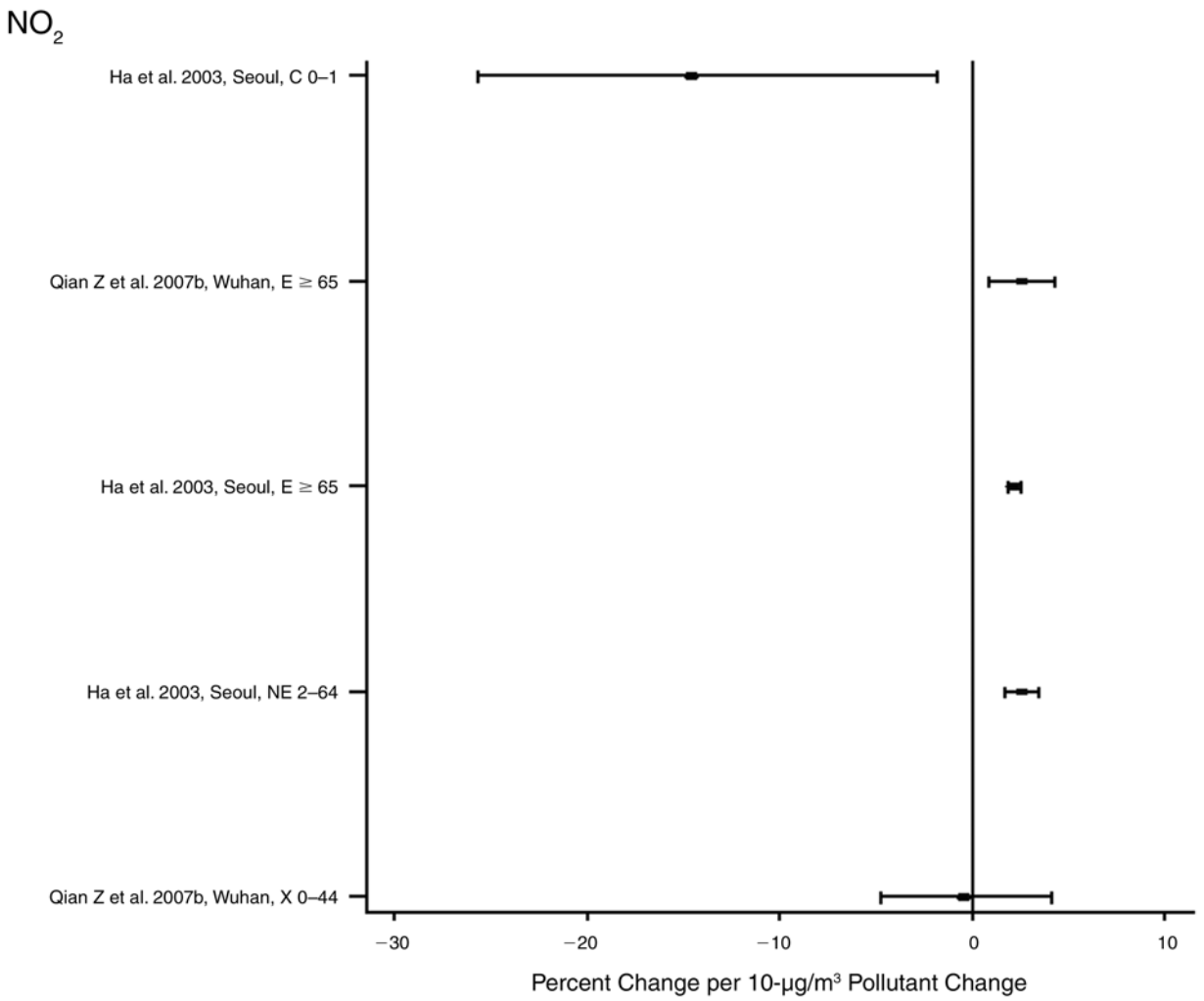
Appendix Figure A.9. Percent change in the mean number of daily deaths from cardiovascular causes per 10-µg/m^3 change in 24-hr mean TSP concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: All Ages (AA) or other (X, n/a). A data point of "+" indicates the paper reported only a single point estimate with no standard error or confidence interval; the value could not be used in the meta-analysis and only the point estimate is presented.



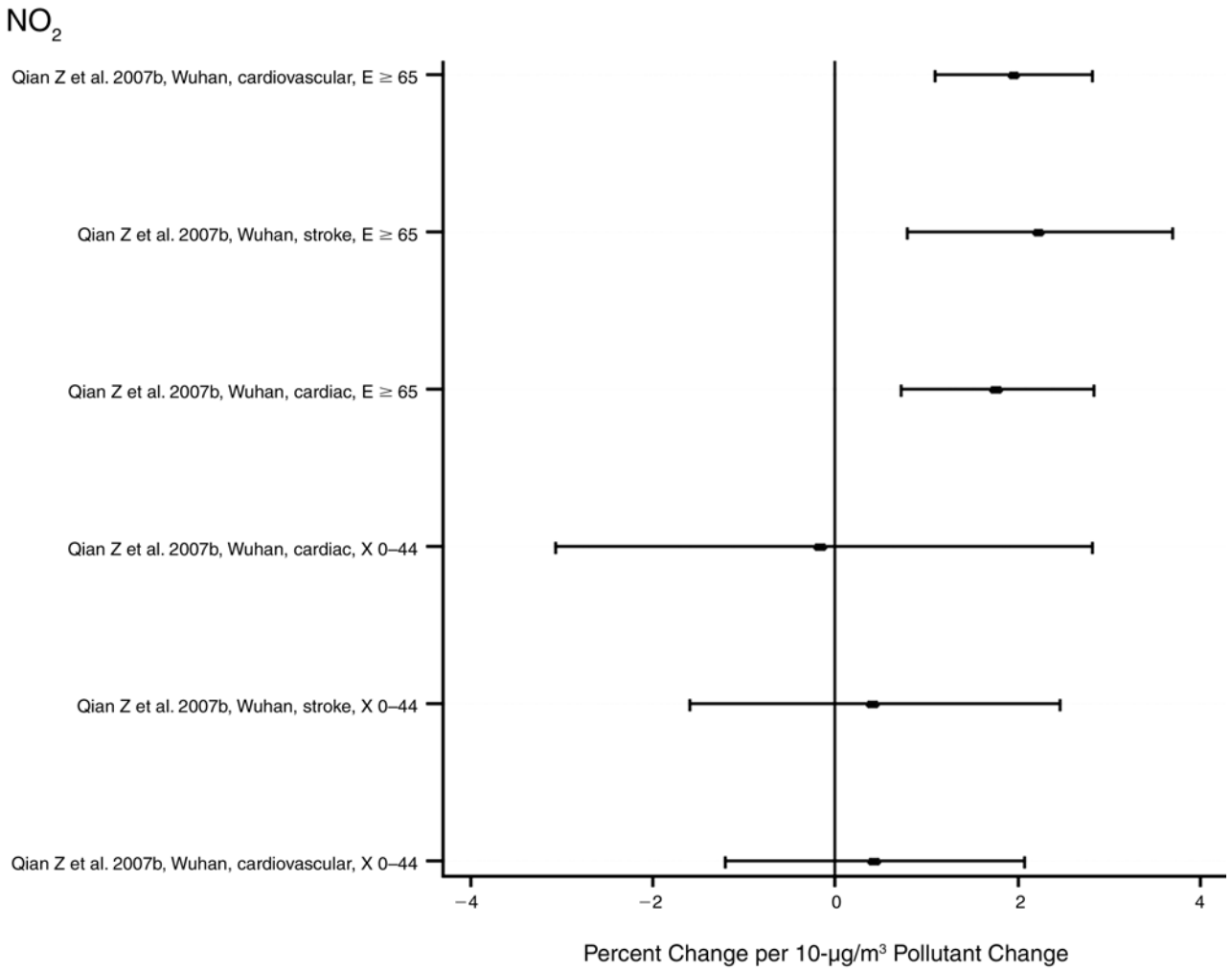
Appendix Figure A.10. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-µg/m³ change in 24-hr mean TSP concentration among persons of all ages. Y-axis labels give study information in the following sequence: reference citation, study location, cause of admission, and age group: All Ages (AA).



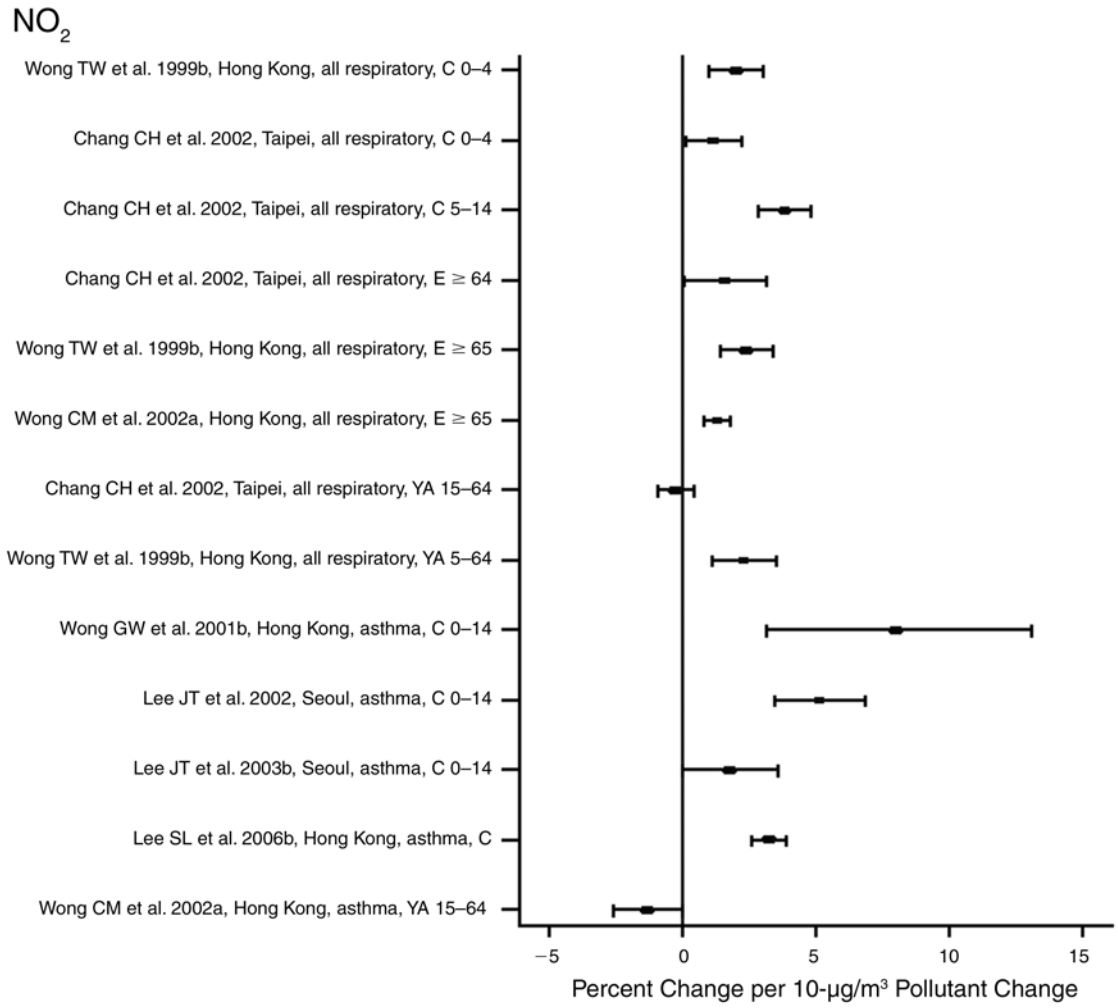
Appendix Figure A.11. Percent change in the mean number of daily deaths from all natural causes per 10-µg/m^3 change in 24-hr mean NO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, and age group: Children (C), Elderly (E), Not Elderly (NE), or other (X) and days or years of age.



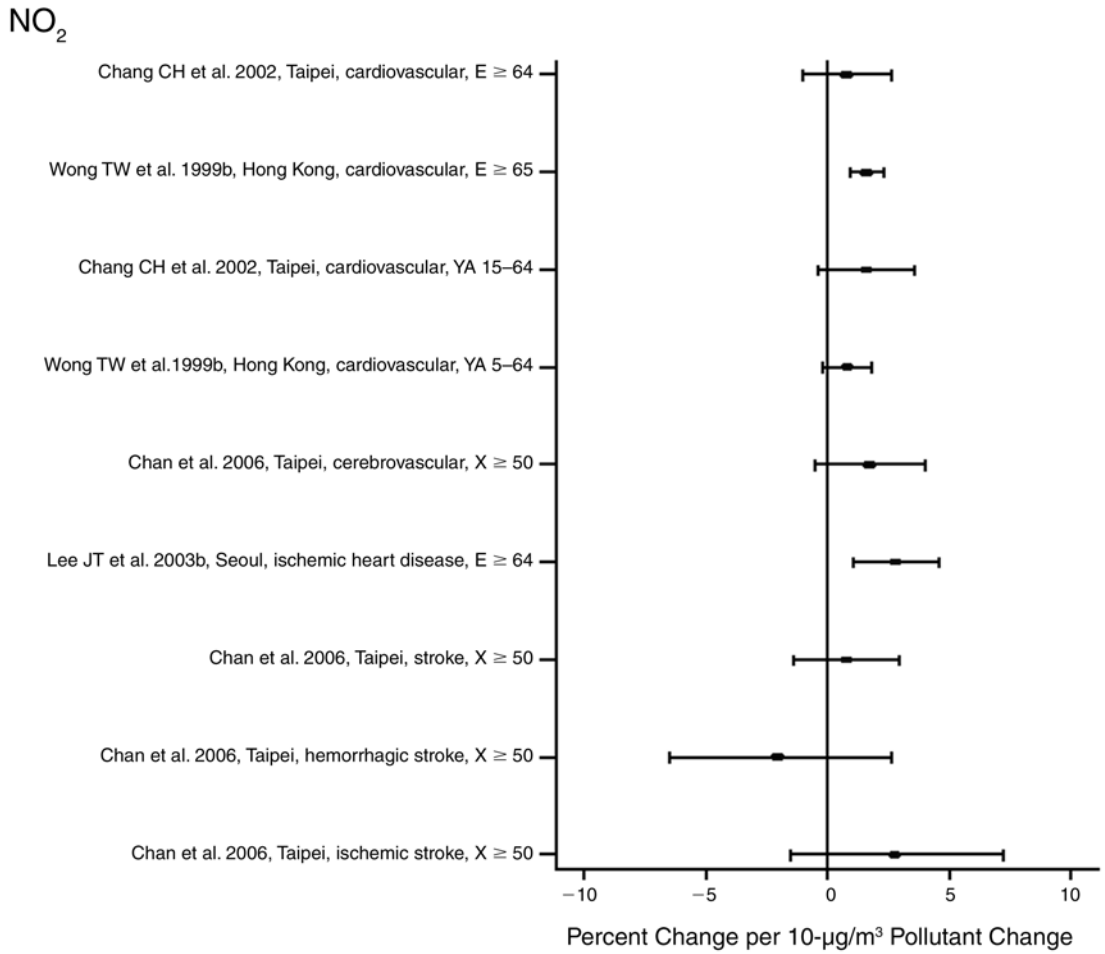
Appendix Figure A.12. Percent change in the mean number of daily deaths from all respiratory causes per 10-µg/m^3 change in 24-hr mean NO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, and age group: Children (C), Elderly (E), Not Elderly (NE), or other (X) and years of age.



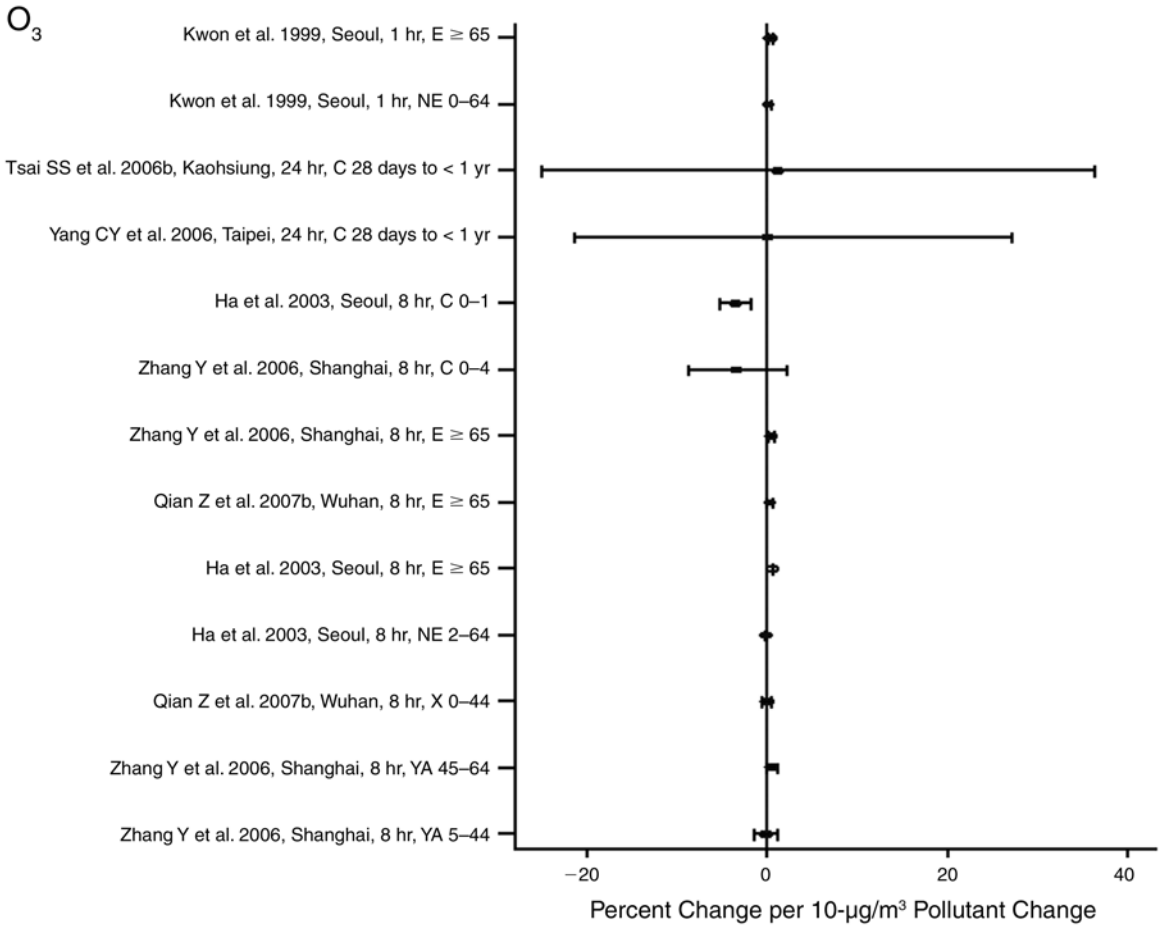
Appendix Figure A.13. Percent change in the mean number of daily deaths from cardiovascular causes per 10-μg/m³ change in 24-hr mean NO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: Elderly (E) or other (X) and years of age.



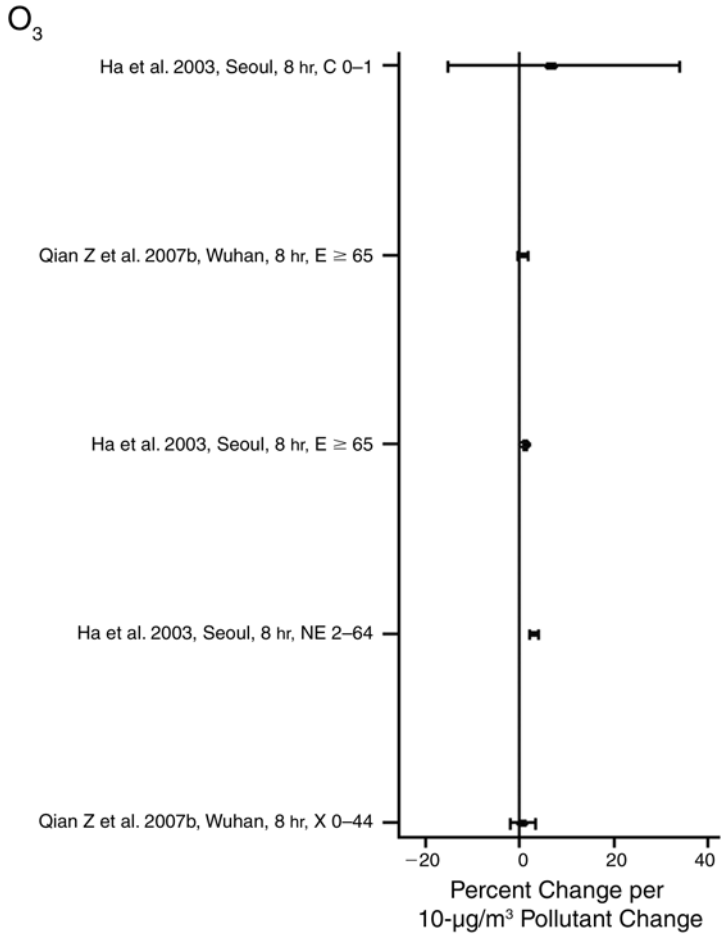
Appendix Figure A.14. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-µg/m^3 change in 24-hr mean NO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of admission, and age group: Children (C), Elderly (E), or Young Adult (YA) and years of age.



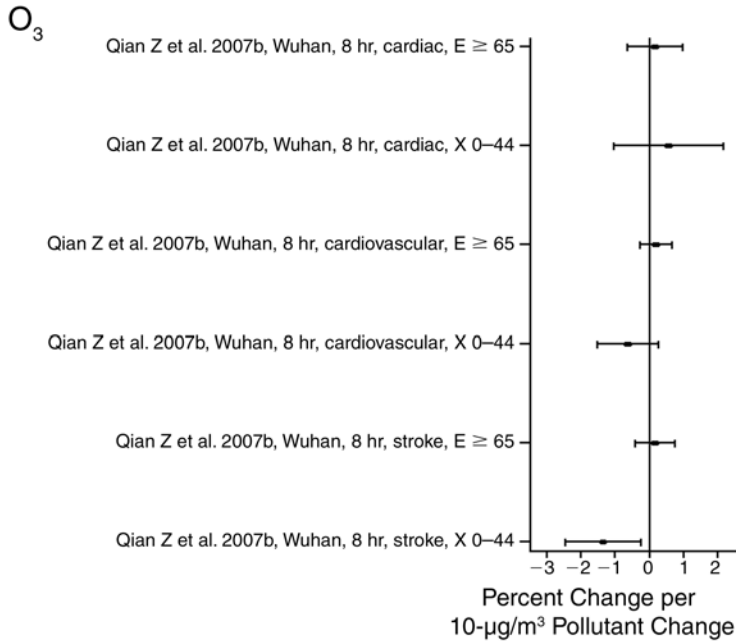
Appendix Figure A.15. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 10-µg/m^3 change in 24-hr mean NO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of admission, and age group: Elderly (E), Young Adult (YA), or other (X) and years of age.



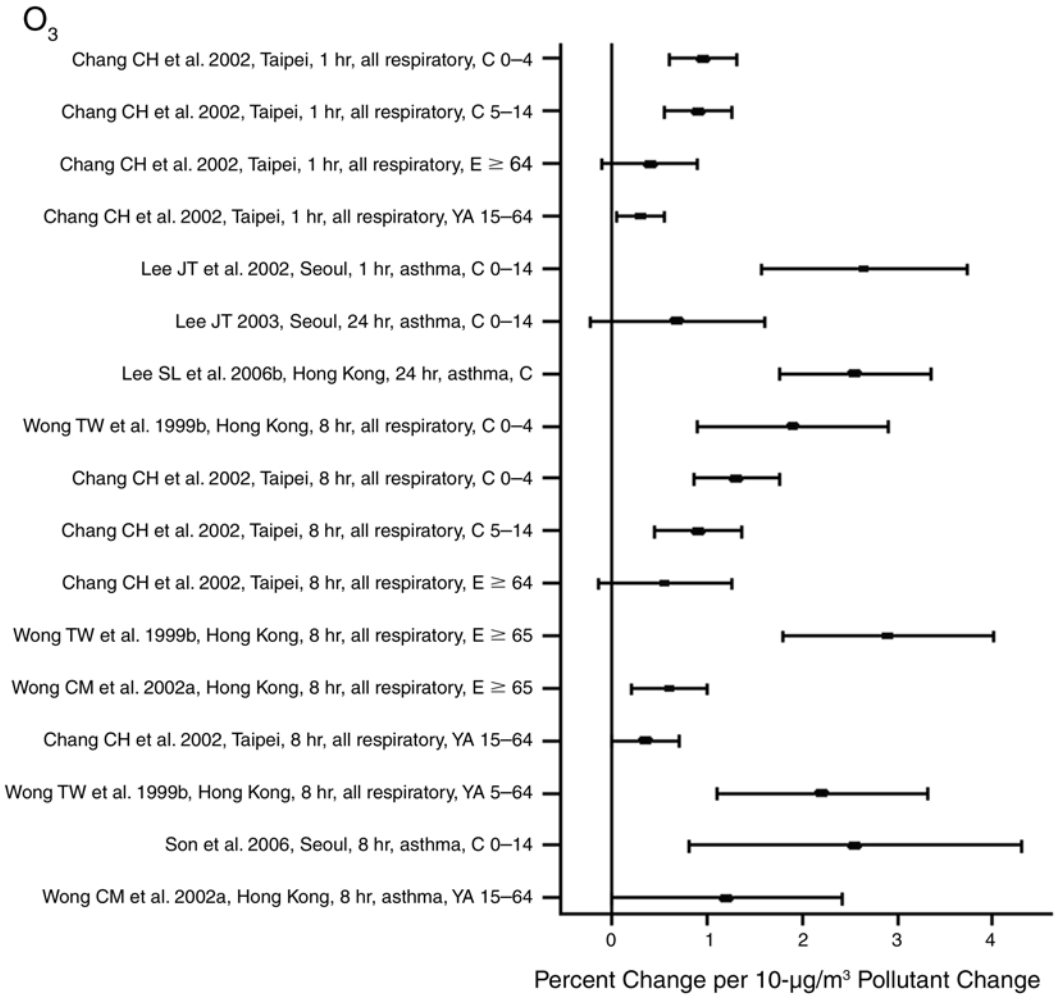
Appendix Figure A.16. Percent change in the mean number of daily deaths from all natural causes per 10-µg/m^3 change in mean O_3 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, and age group: Elderly (E), Not Elderly (NE), Children (C), other (X), or Young Adult (YA) and years of age.



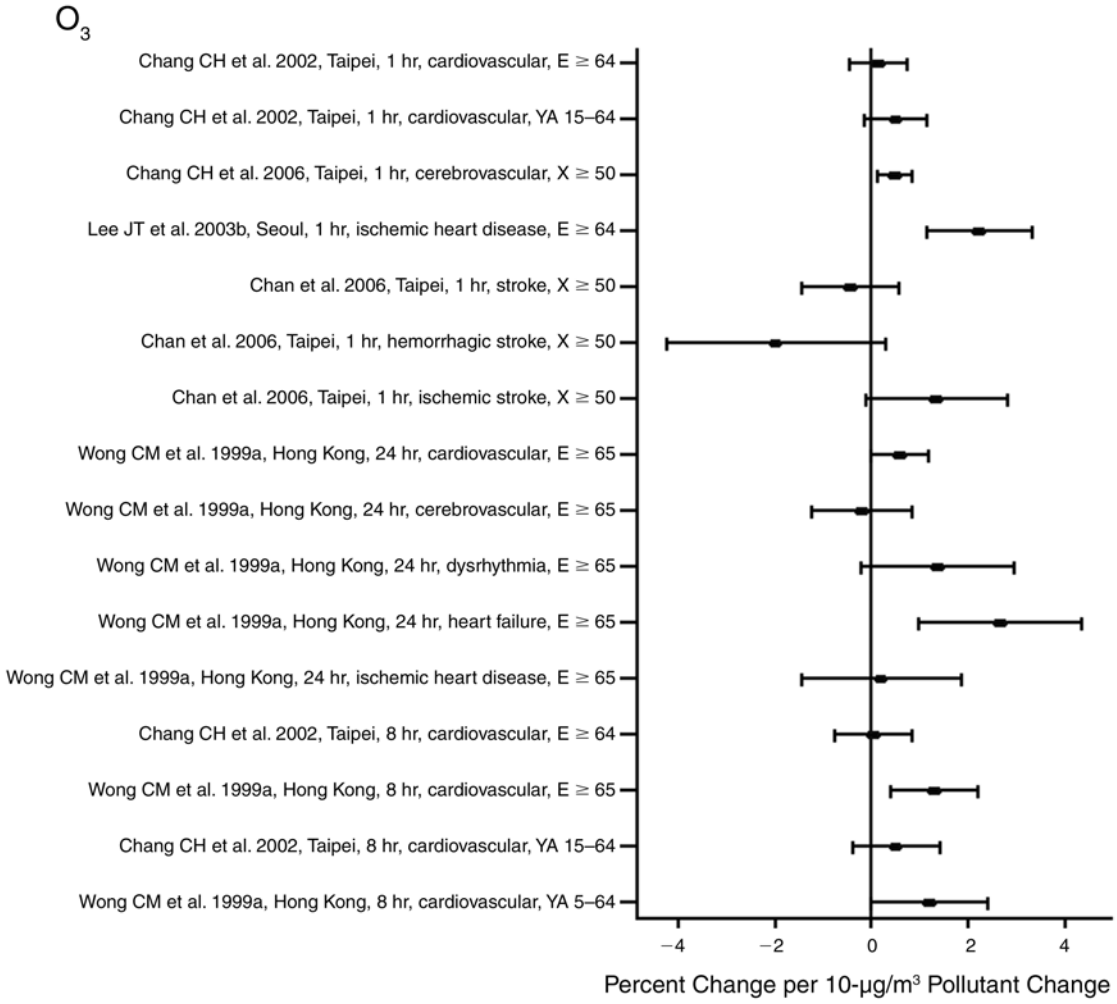
Appendix Figure A.17. Percent change in the mean number of daily deaths from all respiratory causes per 10-µg/m^3 change in 8-hr O_3 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, and age group: Children (C), Elderly (E), Not Elderly (NE), or other (X) and years of age.



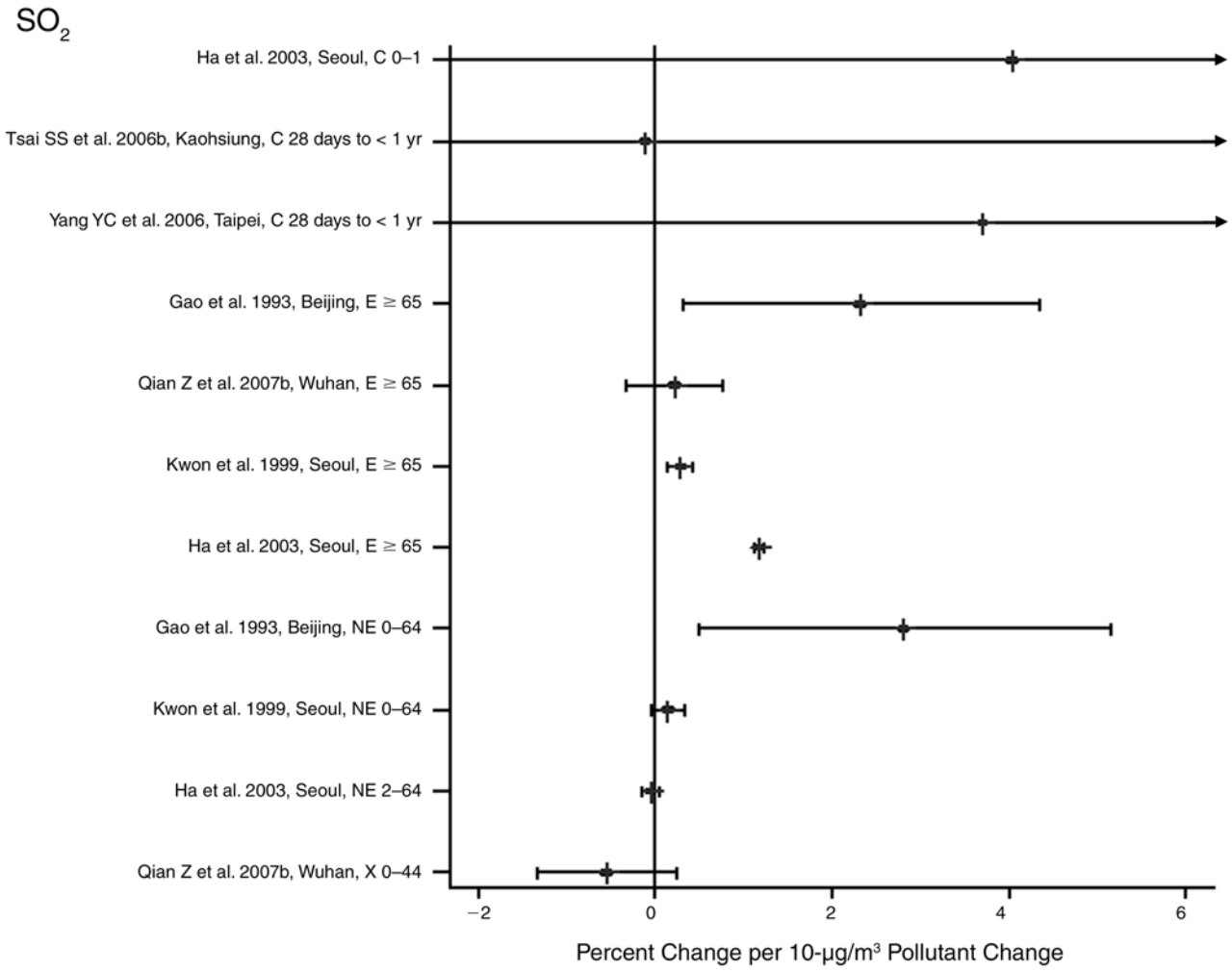
Appendix Figure A.18. Percent change in the mean number of daily deaths from cardiovascular causes per 10-µg/m^3 change in 8-hr O_3 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, cause of death, and age group: Elderly (E) or other (X) and years of age.



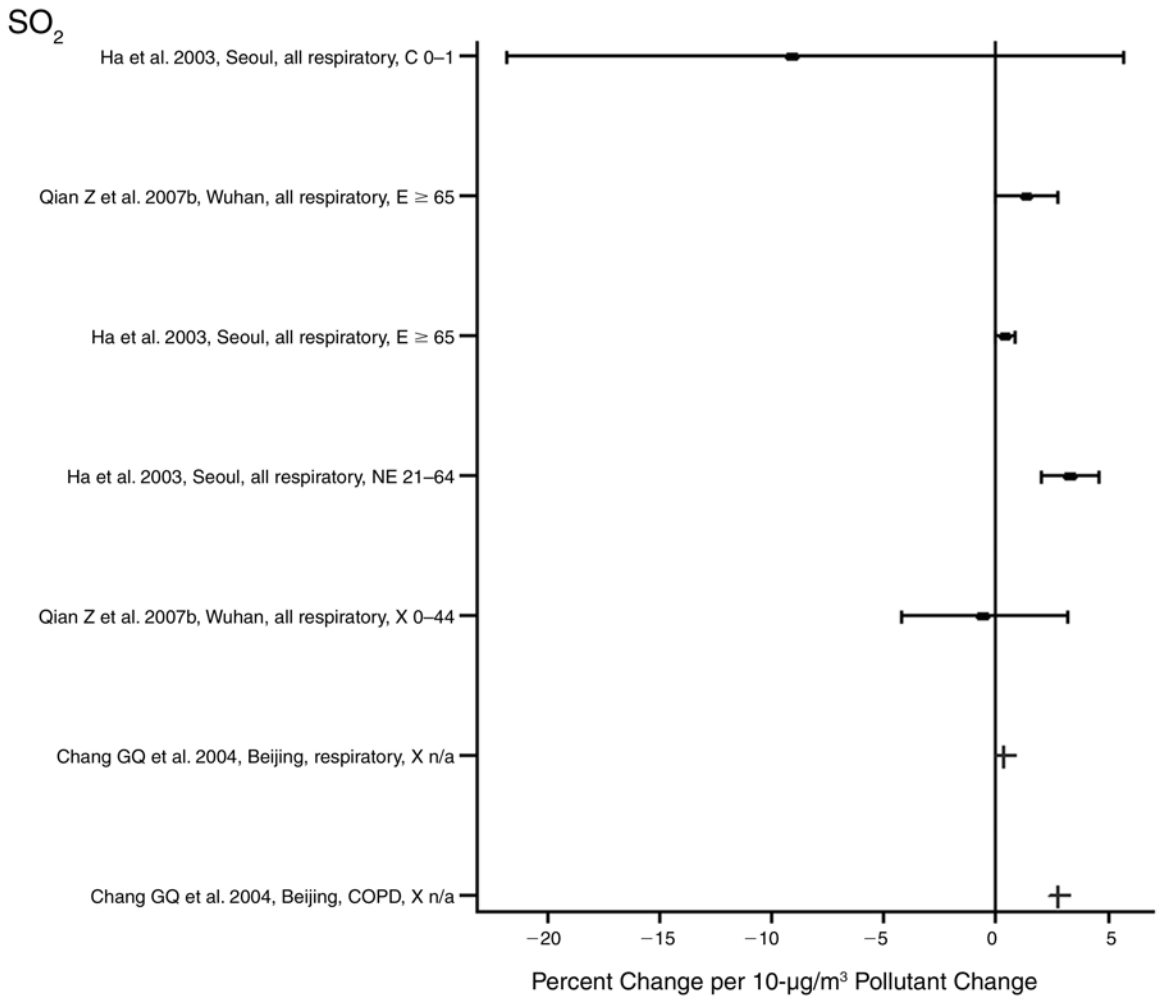
Appendix Figure A.19. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-µg/m^3 change in mean O_3 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, cause of admission, and age group: Children (C), Elderly (E), Young Adult (YA) and years of age.



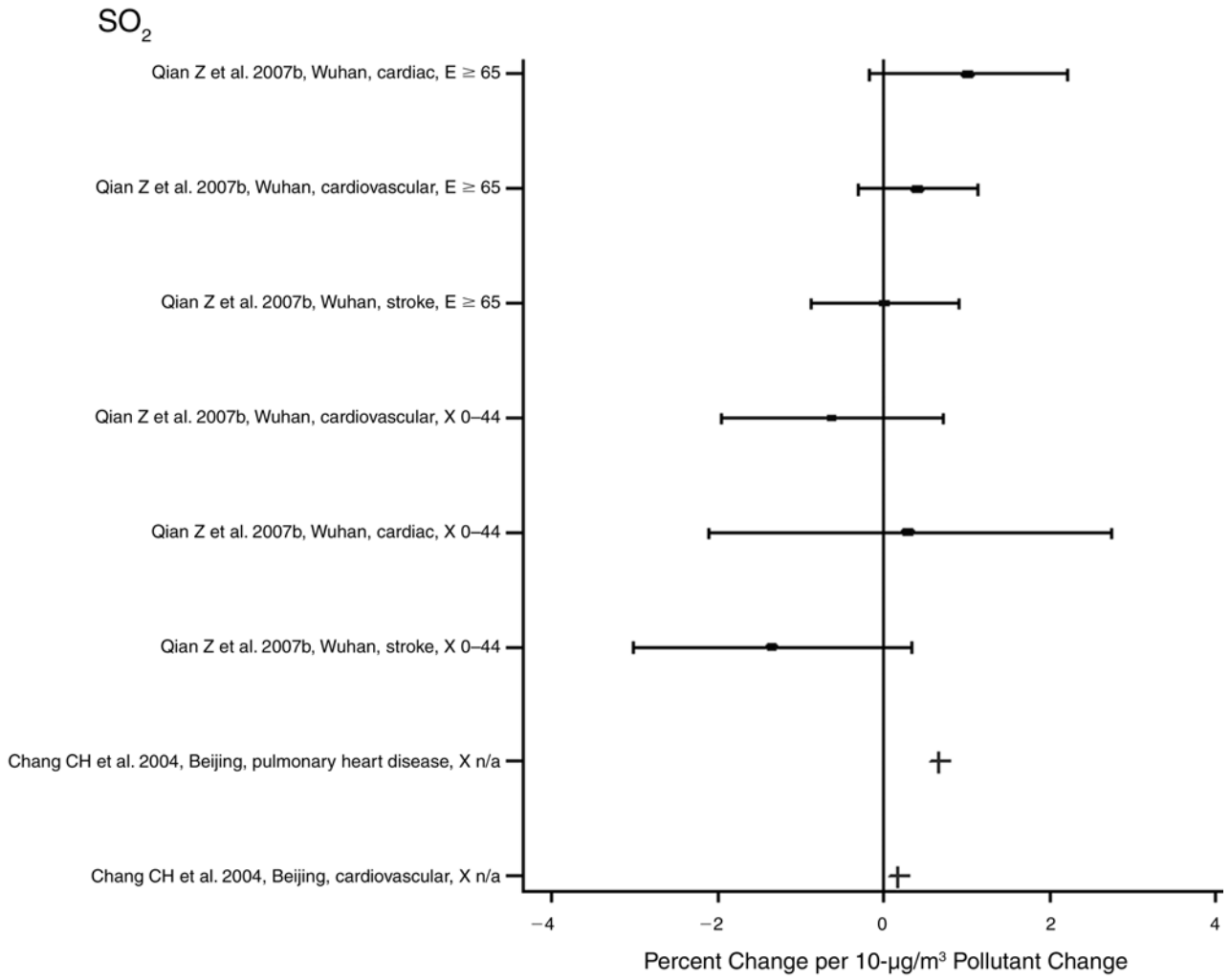
Appendix Figure A.20. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 10-µg/m³ change in mean O_3 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, O_3 averaging time, cause of admission, and age group: Elderly (E), Young Adult (YA), or other (X) and years of age.



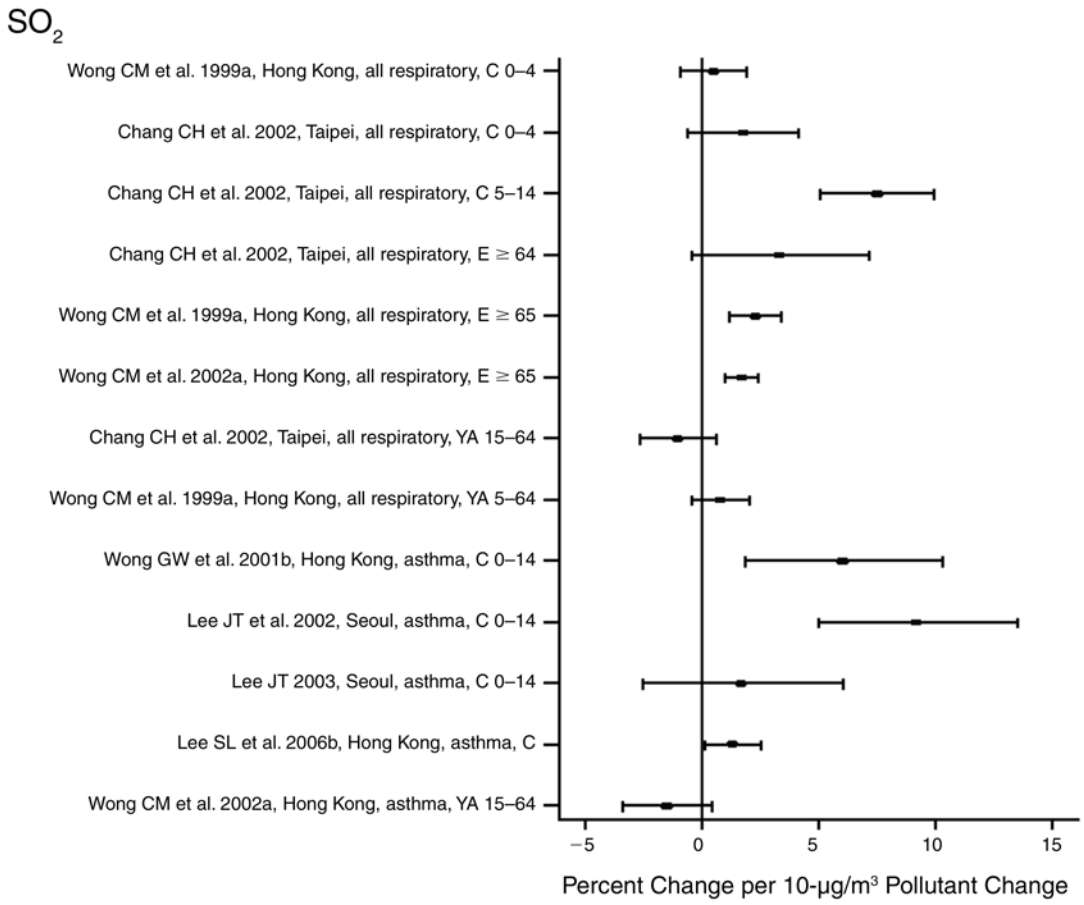
Appendix Figure A.21. Percent change in the mean number of daily deaths from all natural causes per 10-µg/m^3 change in 24-hr mean SO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, and age group: Children (C), Elderly (E), Not Elderly (NE), or other (X) and years of age.



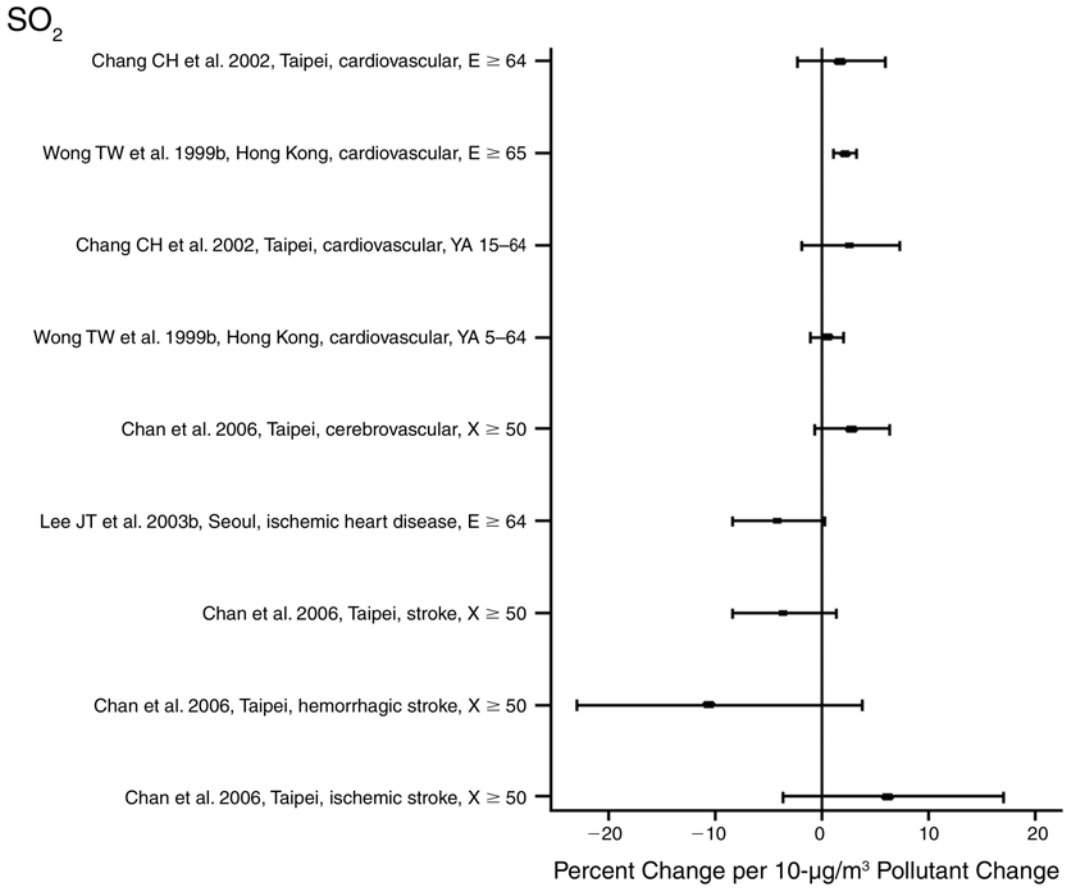
Appendix Figure A.22. Percent change in the mean number of daily deaths from respiratory causes per 10-µg/m^3 change in 24-hr mean SO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: Children (C), Elderly (E), Not Elderly (NE), or other (X) and years of age or not applicable (n/a). A data point of "+" indicates the paper reported only a single point estimate with no standard error or confidence interval; the value could not be used in the meta-analysis and only the point estimate is presented.



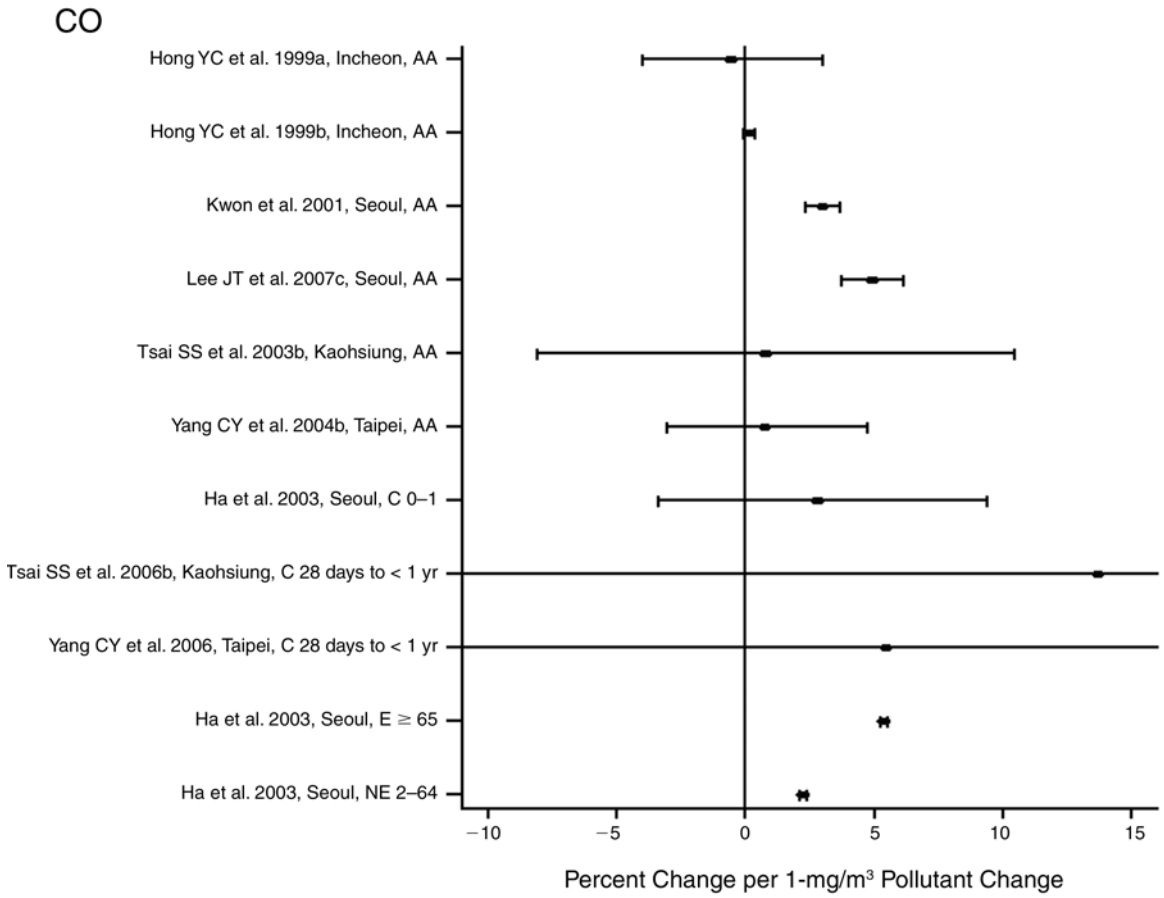
Appendix Figure A.23. Percent change in the mean number of daily deaths from cardiovascular causes per 10-µg/m^3 change in 24-hr mean SO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: Elderly (E) or other (X) and years of age or not applicable (n/a). A data point of "+" indicates the paper reported only a single point estimate with no standard error or confidence interval; the value could not be used in the meta-analysis and only the point estimate is presented.



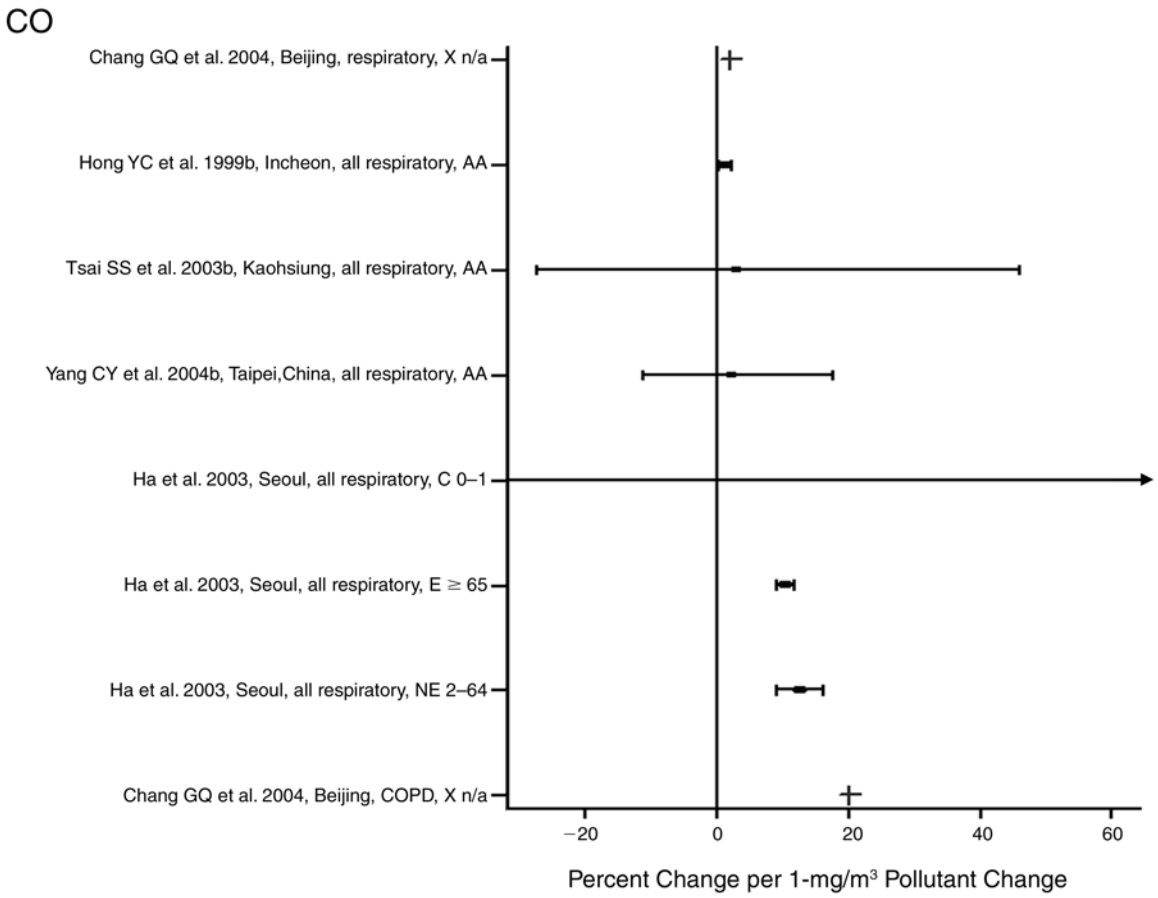
Appendix Figure A.24. Percent change in the mean number of daily hospital admissions from respiratory causes per 10-μg/m^3 change in 24-hr mean SO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of admission, and age group: Children (C), Elderly (E), or Young Adult (YA) and years of age.



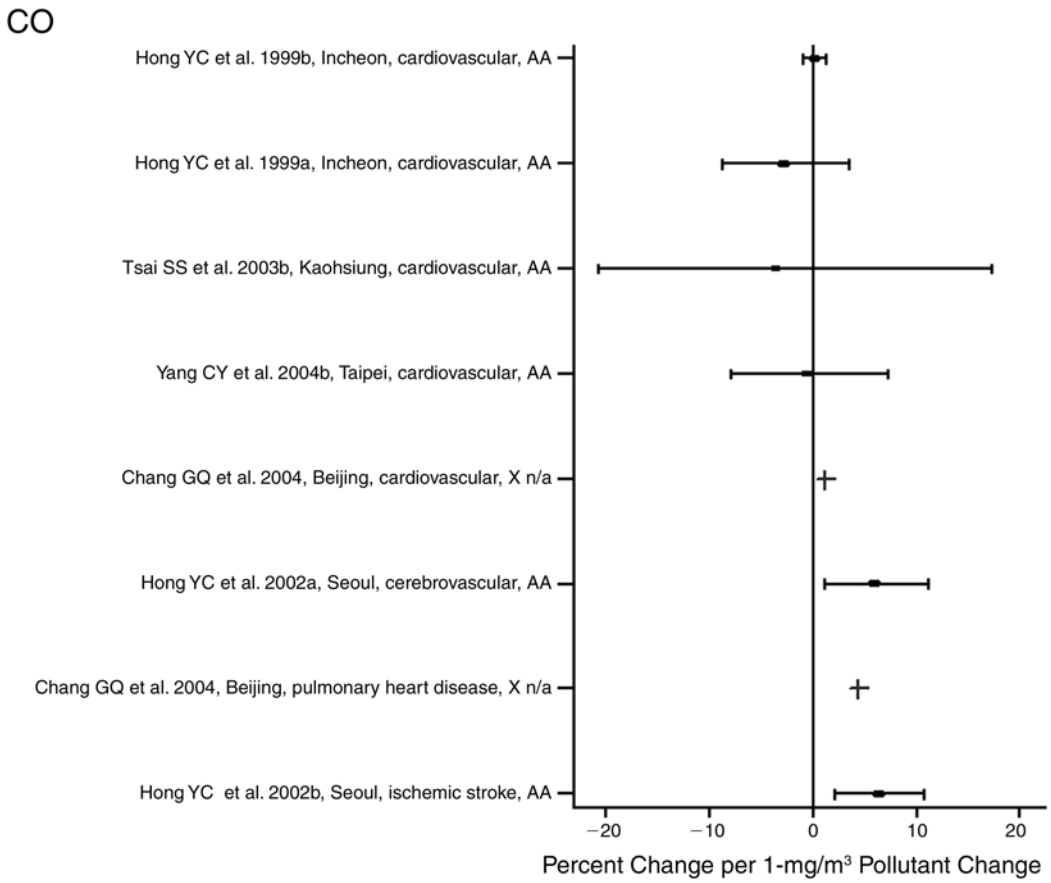
Appendix Figure A.25. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 10-µg/m^3 change in 24-hr mean SO_2 concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of admission, and age group: Elderly (E), Young Adult (YA), other (X) and years of age.



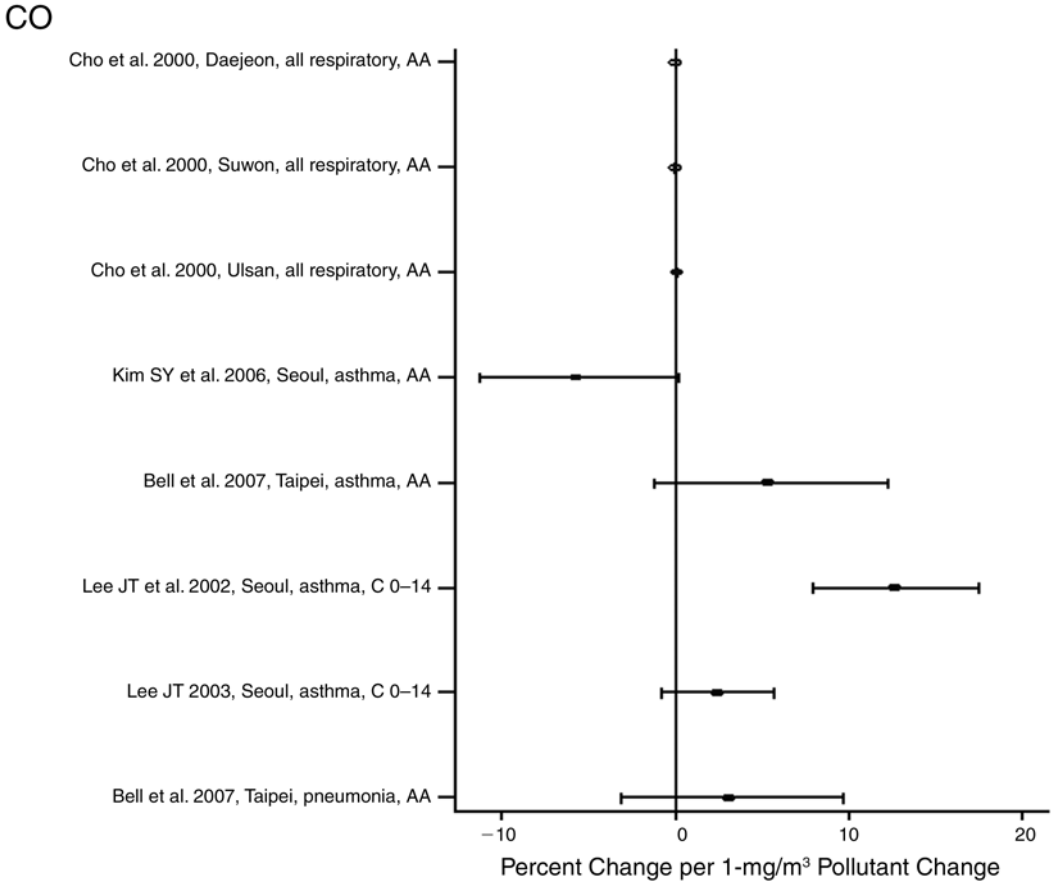
Appendix Figure A.26. Percent change in the mean number of daily deaths from all natural causes per 1-mg/m^3 change in 24-hr mean CO concentration. Y-axis labels give study information in the following sequence: reference citation, study location, and age group: All Ages (AA), Children (C), Elderly (E), or Not Elderly (NE) and years of age, if applicable.



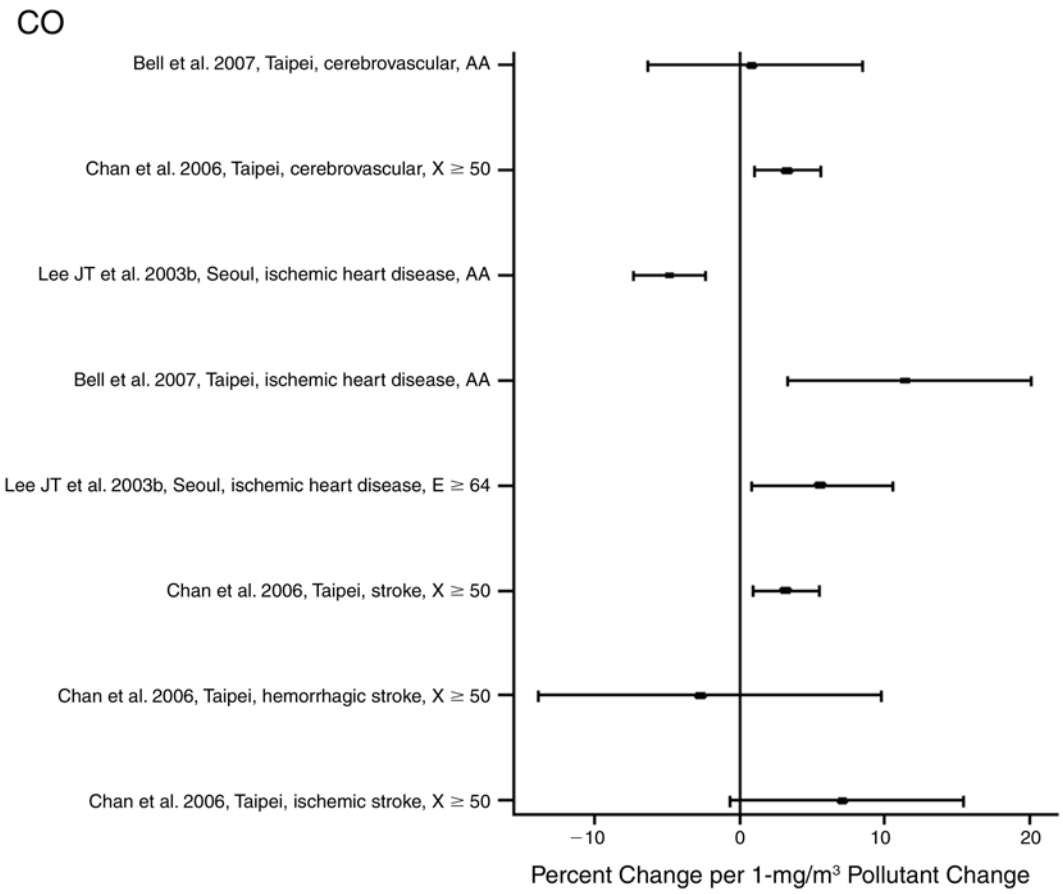
Appendix Figure A.27. Percent change in the mean number of daily deaths from respiratory causes per 1-mg/m^3 change in 24-hr mean CO concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: other (X), All Ages (AA), Children (C), Elderly (E), or Not Elderly (NE) and years of age or not applicable (n/a). A data point of "+" indicates the paper reported only a single point estimate with no standard error or confidence interval; the value could not be used in the meta-analysis and only the point estimate is presented. The estimate for children in Ha et al. 2003 was outside the scale of the plot.



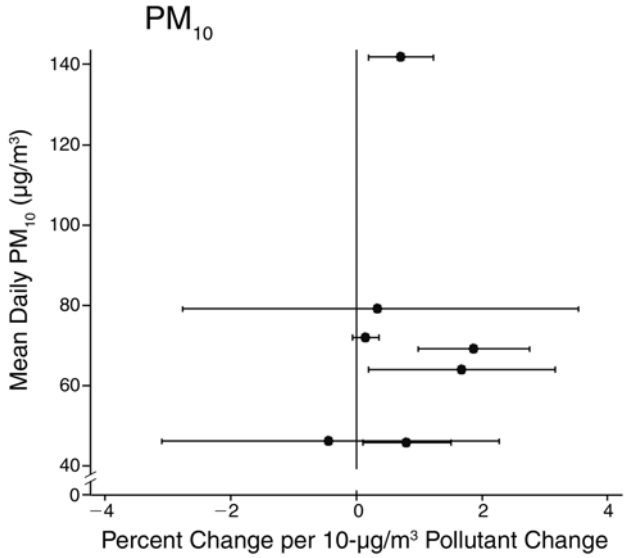
Appendix Figure A.28. Percent change in the mean number of daily deaths from cardiovascular causes per 1-mg/m³ change in 24-hr mean CO concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of death, and age group: All Ages (AA) or other (X, n/a). A data point of "+" indicates the paper reported only a single point estimate with no standard error or confidence interval; the value could not be used in the meta-analysis and only the point estimate is presented.



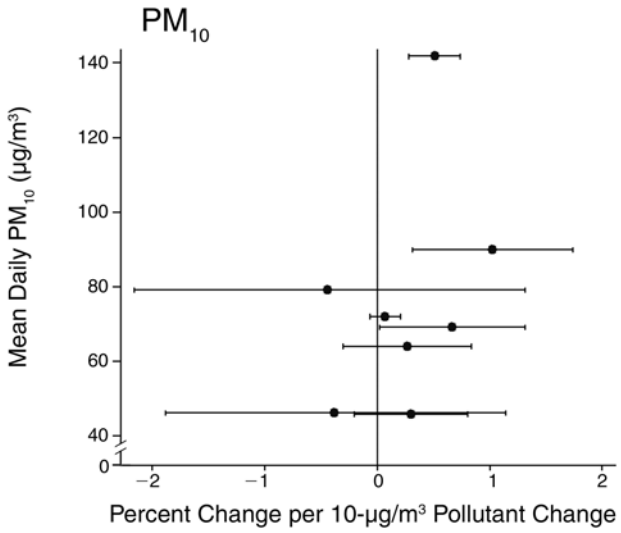
Appendix Figure A.29. Percent change in the mean number of daily hospital admissions from respiratory causes per 1-mg/m^3 change in 24-hr mean CO concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of admission, and age group: All Ages (AA) or Children (C) and years of age, if applicable.



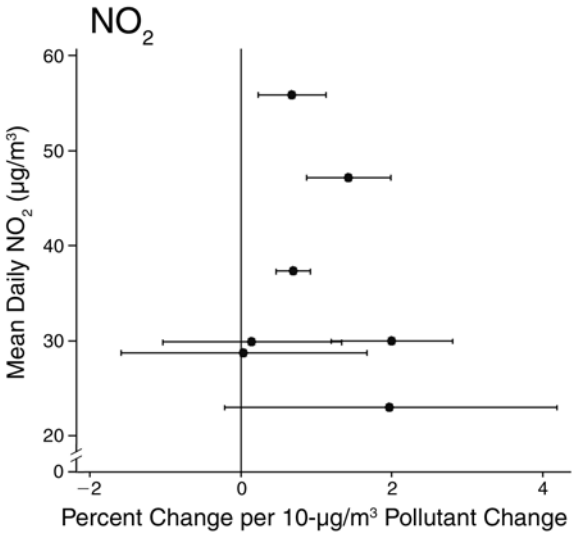
Appendix Figure A.30. Percent change in the mean number of daily hospital admissions from cardiovascular causes per 1-mg/m^3 change in 24-hr mean CO concentration. Y-axis labels give study information in the following sequence: reference citation, study location, cause of admission, and age group: All Ages (AA), other (X), or Elderly (E) and years of age, if applicable.



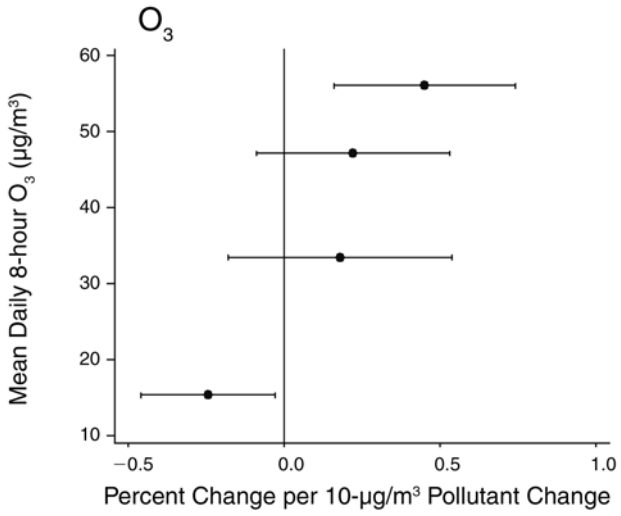
Appendix Figure A.31. Effect estimates for respiratory mortality per 10-µg/m^3 change in 24-hr mean PM_{10} concentration, among persons of all ages, according to daily mean PM_{10} concentration.



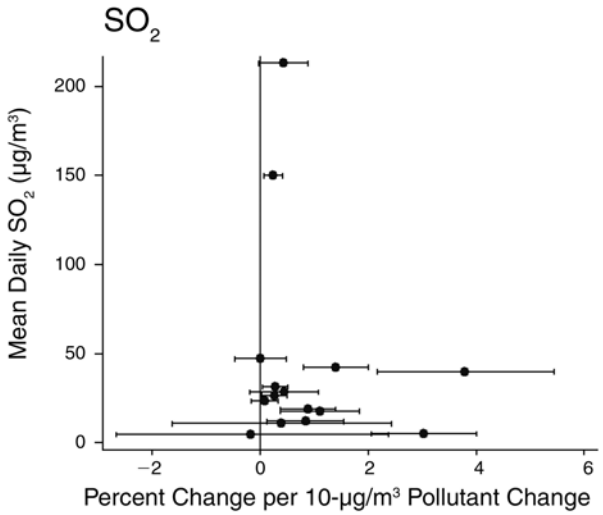
Appendix Figure A.32. Effect estimates for all-natural-cause mortality per 10-µg/m³ change in 24-hr mean PM_{10} concentration, among persons of all ages, according to daily mean PM_{10} concentration.



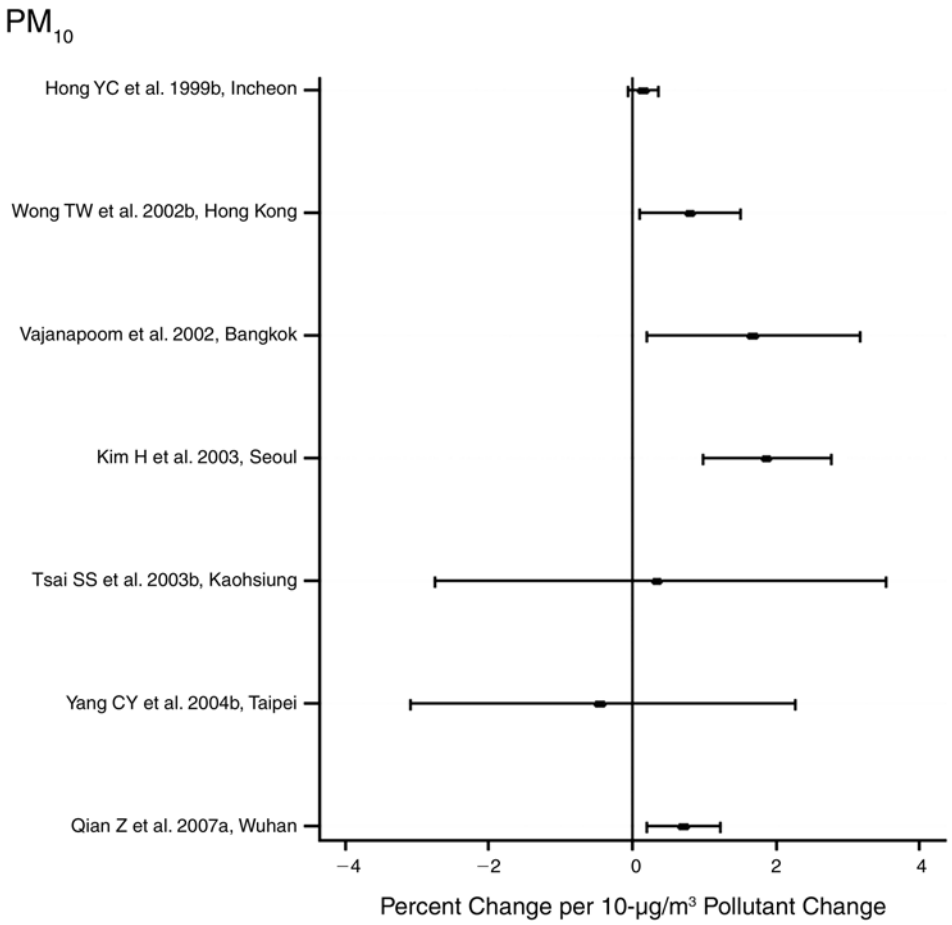
Appendix Figure A.33. Effect estimates for all-natural-cause mortality per 10-µg/m³ change in 24-hr mean NO_2 concentration, among persons of all ages, according to daily mean NO_2 concentration.



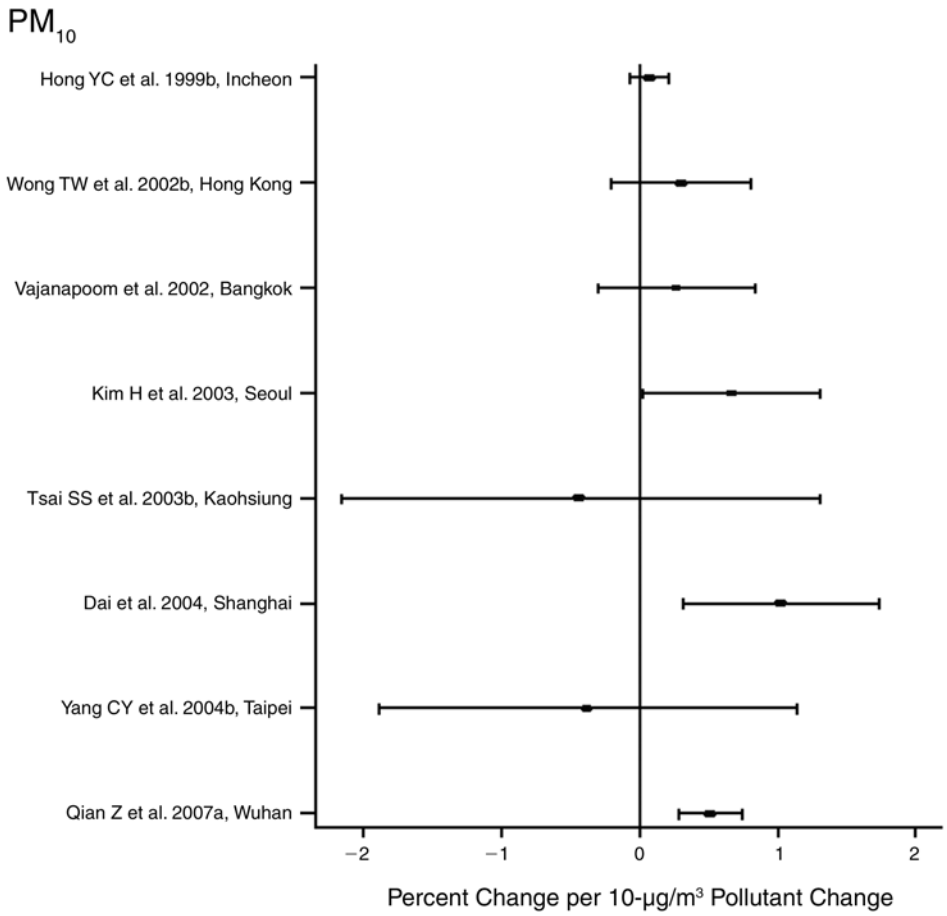
Appendix Figure A.34. Effect estimates for all-natural-cause mortality per 10-µg/m³ change in mean O_3 concentration, among persons of all ages, according to 8-hr O_3 concentration.



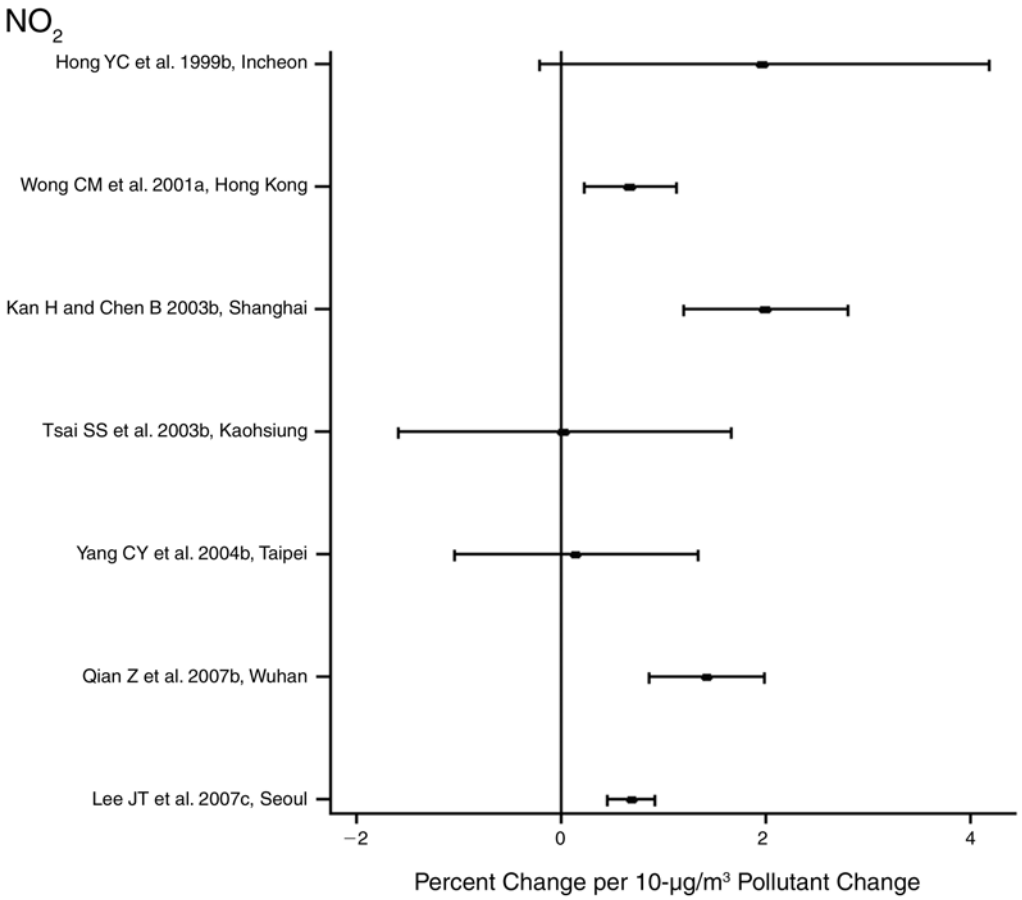
Appendix Figure A.35. Effect estimates for all-natural-cause mortality per 10-µg/m³ change in 24-hr mean SO_2 concentration, among persons of all ages, according to daily mean SO_2 concentration.



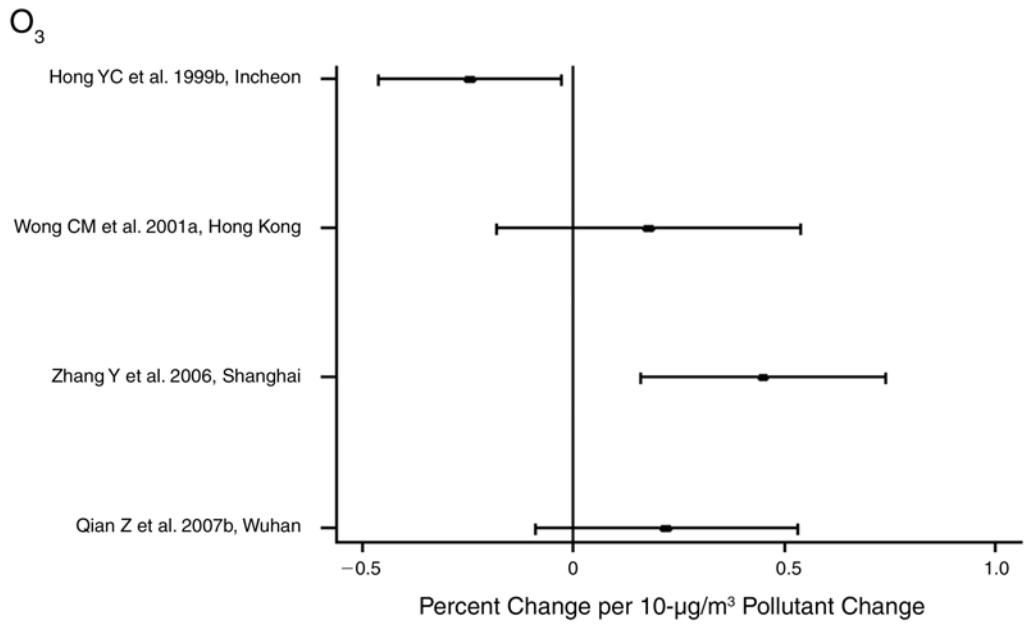
Appendix Figure A.36. Effect estimates for all respiratory mortality per 10-µg/m^3 change in 24-hr mean PM_{10} concentration, among persons of all ages, according to year of publication. Y-axis labels give study information in the following sequence: reference citation and study location.



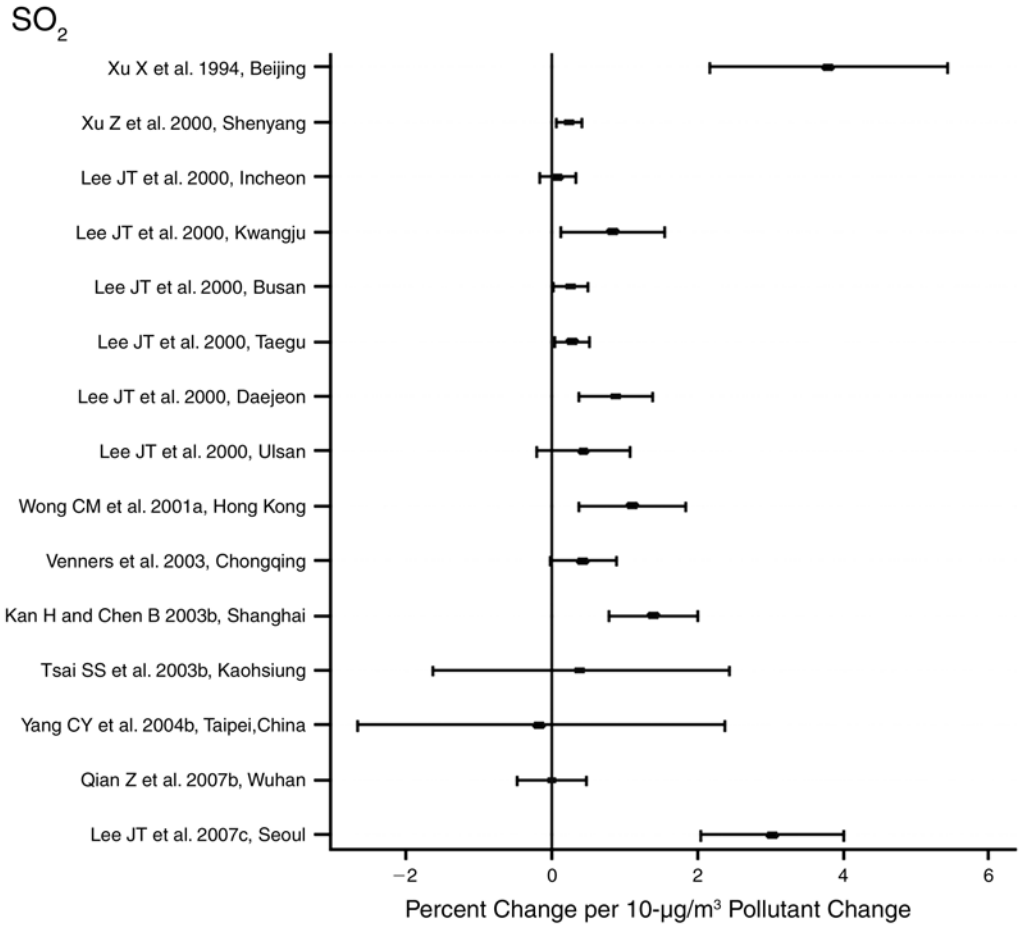
Appendix Figure A.37. Effect estimates for all cardiovascular mortality per 10-µg/m³ change in 24-hr mean PM_{10} concentration, among persons of all ages, according to year of publication. Y-axis labels give study information in the following sequence: reference citation and study location.



Appendix Figure A.38. Effect estimates for all-natural-cause mortality per 10-μg/m³ change in 24-hr mean NO_2 concentration, among persons of all ages, according to year of publication. Y-axis labels give study information in the following sequence: reference citation and study location.



Appendix Figure A.39. Effect estimates for all-natural-cause mortality per 10-μg/m³ change in 8-hr O_3 concentration, among persons of all ages, according to year of publication. Y-axis labels give study information in the following sequence: reference citation and study location.



Appendix Figure A.40. Effect estimates for all-natural-cause mortality per 10-μg/m³ change in 24-hr mean SO_2 concentration, among persons of all ages, according to year of publication. Y-axis labels give study information in the following sequence: reference citation and study location.

Appendix Table A.1. Asian Daily Time-Series Studies Identified Through the Systematic Literature Search and Included in the Quantitative Analysis

Country / Citation	Study Location	Study Period	Outcome	Disease	Pollutant
China					
Chang GQ et al. 2004	Beijing	1998–2000	Mortality	Respiratory disease, cardiovascular disease, coronary heart disease, cerebrovascular disease, COPD	TSP, PM_{10}, SO_2, NO_x, CO
Chang GQ et al. 2003b	Beijing	1998–2000	ER visits	Colds, pneumonia, bronchitis	TSP, PM_{10}, SO_2, NO_x, CO
Dai et al. 2004	Shanghai	2002–2003	Mortality	All causes, cardiovascular disease, respiratory disease	PM_{10}, $PM_{2.5}$
Dong et al. 1995	Beijing	1990–1991	Mortality	All causes	TSP, SO_2
Gao et al. 1993	Beijing	1989	Mortality	All causes, respiratory disease	TSP, SO_2
Jin et al. 1999	Benxi	1993–1994	ER visits	All causes, COPD, cardiovascular disease, cerebrovascular disease	TSP, SO_2
Kan H and Chen B 2003b	Shanghai	2000–2001	Mortality	All causes, cardiovascular disease, COPD (incl. asthma)	PM_{10}, SO_2, NO_2
Kan H and Chen B 2003a	Shanghai	2000–2001	Mortality	All causes, cardiovascular disease, COPD (incl. asthma)	PM_{10}, SO_2, NO_2
Kan H et al. 2004b	Shanghai	2001–2002	Mortality	Diabetes	PM_{10}, SO_2, NO_2
Kan H et al. 2003	Shanghai	2001–2002	Mortality	Stroke	PM_{10}, SO_2, NO_2
Kan H et al. 2004a	Shanghai	2000–2001	Mortality	All causes, cardiovascular disease, COPD	PM_{10}, SO_2, NO_2
Kan H et al. 2007	Shanghai	2004–2005	Mortality	All causes, cardiovascular disease, respiratory disease	PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$
Ko FW et al. 2007	Hong Kong	2000–2004	Admissions	COPD	PM_{10}, $PM_{2.5}$, NO_2, SO_2, O_3
Lee SL et al. 2006b	Hong Kong	1997–2002	Admissions	Asthma	PM_{10}, $PM_{2.5}$, NO_2, SO_2, O_3
Qian Z et al. 2007a	Wuhan	2001–2004	Mortality	All causes, cardiovascular disease, stroke, cardiac disease, respiratory disease, cardiopulmonary disease	PM_{10}
Qian Z et al. 2007b	Wuhan	2001–2004	Mortality	All causes, cardiovascular disease, stroke, cardiac disease, respiratory disease, cardiopulmonary disease	NO_2, SO_2, O_3
Venners et al. 2003	Chongqing	1995	Mortality	Respiratory disease, cardiovascular disease, cancer, other	$PM_{2.5}$, SO_2
Wong CM et al. 1999a	Hong Kong	1994–1995	Admissions	Respiratory disease, cardiovascular disease	PM_{10}, NO_2, SO_2, O_3
Wong CM et al. 2001a	Hong Kong	1995–1997	Mortality	All causes, cardiovascular disease, respiratory disease	PM_{10}, NO_2, SO_2, O_3
Wong CM et al. 2002a	Hong Kong, London	1995–1997 (Hong Kong), 1992–1994 (London)	Admissions	Asthma, respiratory disease, cardiac disease, ischemic heart disease	PM_{10}, NO_2, SO_2, O_3

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Appendix Table A.1 (*Continued*). Asian Daily Time-Series Studies Identified Through the Systematic Literature Search and Included in the Quantitative Analysis

Country / Citation	Study Location	Study Period	Outcome	Disease	Pollutant
China (*Continued*)					
Wong GW et al. 2001b	Hong Kong	1993–1994	Admissions	Asthma	PM_{10}, NO_2, SO_2,
Wong TW et al. 2002c	Hong Kong	2000	General practitioner consultations	Respiratory disease, upper respiratory tract infection	PM_{10}, NO_2, SO_2, O_3
Wong TW et al. 2006	Hong Kong	2000–2002	General practitioner consultations	Upper respiratory tract infection	PM_{10}, $PM_{2.5}$, NO_2, SO_2, O_3
Wong TW et al. 1999b	Hong Kong	1994–1995	Admissions	Cardiovascular disease	PM_{10}, NO_2, SO_2, O_3
Wong TW et al. 2002b	Hong Kong	1995–1998	Mortality	Respiratory disease, cardiovascular disease	PM_{10}, NO_2, SO_2, O_3
Xu X et al. 1995a	Beijing	1990	Outpatient visits	Surgery or nonsurgery	TSP, SO_2
Xu X et al. 1994	Beijing	1994	Mortality	All causes, cardiovascular disease, cardiopulmonary disease, cancer	TSP, SO_2
Xu X et al. 1995b	Beijing	1990	Outpatient and ER visits		TSP, SO_2
Xu Z et al. 2000	Shenyang	1992	Mortality	All causes, cardiovascular disease, cardiopulmonary disease, COPD, cancer	TSP, SO_2
Zhang Y et al. 2006	Shanghai	2001–2004	Mortality	All causes, cardiovascular disease, respiratory disease	O_3
Zhang YP et al. 2007	Taiyuan	2004–2005	Mortality	All causes, cancer, cardiovascular disease, respiratory disease	PM_{10}
India					
Cropper et al. 1997	Delhi	1991–1994	Mortality	All causes, respiratory disease, cardiovascular disease	TSP, SO_2, NO_x
Japan					
Omori et al. 2003	13 Largest cities	1990–1994	Mortality	All causes, cardiovascular disease, respiratory disease	SPM
Yamazaki et al. 2007	13 Urban areas	1990–1994	Mortality	Stroke, intracerebral hemorrhage	PM_7, NO_2, photochemical oxidants
Ye et al. 2001	Tokyo	1980–1995	Emergency transports	Cardiovascular (angina, cardiac insufficiency, hypertension, myocardial infarction) and respiratory (asthma, acute and chronic bronchitis, pneumonia)	PM_{10}, NO_2, SO_2, CO, O_3
Malaysia					
Sastry 2002	Kuala Lumpur	1997	Mortality	All causes	PM_{10}
Singapore					
Chew et al. 1999	Singapore	1990–1994	ER visits	Asthma	TSP, SO_2, NO_2, O_3

Table continues next page

Appendix Table A.1(*Continued*). Asian Daily Time-Series Studies Identified Through the Systematic Literature Search and Included in the Quantitative Analysis

Country / Citation	Study Location	Study Period	Outcome	Disease	Pollutant
South Korea					
Cho et al. 2000	Ulsan, Daejeon, Suwon	1996	Admissions	Respiratory (bronchial asthma, COPD, bronchitis)	TSP, NO_2, SO_2, CO, O_3
Ha et al. 2003	Seoul	1995–1999	Mortality	All causes, respiratory disease	TSP, SO_2, NO_2, CO, O_3
Hong YC et al. 2002b	Seoul	1991–1997	Mortality	Stroke (hemorrhagic or ischemic)	TSP, NO_2, SO_2, CO, O_3
Hong YC et al. 2002a	Seoul	1995–1998	Mortality	Stroke	PM_{10}, NO_2, SO_2, O_3, CO
Hong YC et al. 1999b	Incheon	1995–1996	Mortality	All causes, cardiovascular disease, respiratory disease	PM_{10}, NO_2, SO_2, CO, O_3
Hong YC et al. 1999a	Incheon	1995	Mortality	All causes	TSP, PM_{10}, NO_2, SO_2, CO, O_3
Im et al. 2000	Seoul	1996–1997	ER visits	Asthma	TSP, NO_2, SO_2, CO, O_3
Ju and Cho 2001	Seoul	1994–1997	ER visits	Asthma	TSP, PM_{10}, O_3
Kim H et al. 2003	Seoul	1995–1999	Mortality	All causes, respiratory disease, cardiovascular disease, cerebrovascular event	PM_{10}
Kim H et al. 2004a	Seoul	1997–2001	Mortality	All causes	PM_{10}
Kim SY et al. 2006b	Seoul	2002	Admissions	Asthma	PM_{10}, NO_2, SO_2, CO, O_3
Kim SY et al. 2004b	Seoul	1995–1999	Mortality	All causes	PM_{10}, NO_2, SO_2, O_3, CO
Kwon et al. 2001	Seoul	1994–1998	Mortality	All causes	PM_{10}, NO_2, SO_2, CO, O_3
Kwon and Cho 1999	Seoul	1991–1995	Mortality	All causes, cardiovascular disease, respiratory disease	TSP, NO_2, SO_2, O_3
Lee JT et al. 2003b	Seoul	1997–1999	Admissions	Ischemic heart disease	PM_{10}, NO_2, SO_2, CO, O_3
Lee JT et al. 2000	7 Cities	1991–1997	Mortality	All causes	TSP, SO_2, O_3
Lee JT et al. 2002	Seoul	1997–1999	Admissions	Asthma	PM_{10}, NO_2, SO_2, CO, O_3
Lee JT et al. 1998	Ulsan	1991–1994	Mortality	All causes	TSP, SO_2
Lee JT and Schwartz 1999	Seoul	1991–1995	Mortality	All causes	TSP, SO_2, O_3
Lee JT et al. 1999b	Seoul, Ulsan	1991–1995	Mortality	All causes	TSP, SO_2, O_3
Lee JT et al. 2007c	Seoul	2000–2004	Mortality	All causes	PM_{10}, NO_2, SO_2, CO, O_3
Lee JT 2003	Seoul	1997–1999	Admissions	Asthma	PM_{10}, NO_2, SO_2, CO, O_3
Lee YJ et al. 2001	Seoul	1995–1996	ER visits	Respiratory disease	PM_{10}, TSP, NO_2, SO_2, CO, O_3
Leem et al. 1998	Seoul	1995–1996	ER visits	Respiratory disease	PM_{10}, O_3

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Appendix Table A.1 (*Continued*). Asian Daily Time-Series Studies Identified Through the Systematic Literature Search and Included in the Quantitative Analysis

Country / Citation	Study Location	Study Period	Outcome	Disease	Pollutant
South Korea (*Continued*)					
Park H et al. 2002	Seoul	1996–1999	School absences	Illness-related causes	PM_{10}, NO_2, SO_2, CO, O_3
Son et al. 2006	Seoul	2002	Admissions	Asthma	O_3
Song HI 2001	Seoul	1992–1993	Doctor's visits	Asthma	TSP, SO_2
Sung et al. 2001	12 Provinces	1991–1995	Mortality	Cardiopulmonary disease	TSP, SO_2, O_3
Taipei,China					
Bell et al. 2007	Taipei	1995–2002	Admissions	IHD, cerebrovascular disease, asthma, pneumonia	PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, NO_2, SO_2, CO, O_3
Chan et al. 2006	Taipei	1997–2002	Admissions	Cerebrovascular	PM_{10}, $PM_{2.5}$, NO_2, SO_2, CO, O_3
Chang CC et al. 2005	Taipei	1997–2001	Admissions	Cardiovascular	PM_{10}, NO_2, SO_2, O_3, CO
Chang CH et al. 2002	Taipei	1997–1999	Admissions	Respiratory disease, cardiovascular disease	PM_{10}, NO_2, SO_2, O_3
Hwang JS and Chan 2002	Taipei	1998	Clinic visits	Lower respiratory tract illness	PM_{10}, NO_2, SO_2, CO, O_3
Lee IM et al. 2007b	Kaohsiung	1996–2003	Admissions	COPD	PM_{10}, NO_2, SO_2, CO, O_3
Tsai SS et al. 2006b	Kaohsiung	1996–2003	Admissions	Asthma	PM_{10}, NO_2, SO_2, CO, O_3
Tsai SS et al. 2003a	Kaohsiung	1997–2000	Admissions	Stroke	PM_{10}, NO_2, SO_2, CO, O_3
Tsai SS et al. 2003b	Kaohsiung	1994–2000	Mortality	All causes, respiratory disease, cardiovascular disease	PM_{10}, NO_2, SO_2, CO, O_3
Tsai SS et al. 2006a	Kaohsiung	1994–2000	Mortality	Postneonatal (27 days–1 yr) causes	PM_{10}, NO_2, SO_2, CO, O_3
Yang CY et al. 2004c	Kaohsiung	1997–2000	Admissions	Cardiovascular disease	PM_{10}, NO_2, SO_2, CO, O_3
Yang CY et al. 2004b	Taipei	1994–1998	Mortality	All causes, cardiovascular disease, respiratory disease	PM_{10}, NO_2, SO_2, CO, O_3
Yang CY and Chen CJ 2007	Taipei	1996–2003	Admissions	COPD	PM_{10}, NO_2, SO_2, CO, O_3
Yang CY et al. 2007	Taipei	1996–2003	Admissions	Asthma	PM_{10}, SO_2, NO_2, CO, O_3
Yang CY et al. 2006	Taipei	1994–2000	Mortality	Postneonatal (27 days–1 yr) causes	PM_{10}, SO_2, NO_2, CO, O_3
Thailand					
Ostro et al. 1999	Bangkok	1992–1995	Mortality	All causes	PM_{10}
Vajanapoom et al. 2002	Bangkok	1992–1997	Mortality	All causes, respiratory disease, cardiovascular disease, other	PM_{10}, poor visibility

Appendix Table A.2. Time-Series Studies Excluded from Quantitative Analysis

Country / Citation	Study Location	Study Period	Outcome	Disease	Pollutant	Reason for Exclusion	Comments
China							
Dong et al. 1996	Beijing	1991	Outpatient visits	All causes	TSP, SO_2	Study period < 1 yr	
Kan HD et al. 2005	Beijing	2003	Mortality	Severe acute respiratory syndrome	PM_{10}, SO_2, NO_2	Rare outcome	
Tseng et al. 1992	Hong Kong	1983–1989	Hospital discharges	Asthma	TSP, RSP, SO_2, NO_2, NO_x, O_3	No numerical estimates	
Tseng and Li 1990	Hong Kong	1983–1987	Admissions	Asthma episodes	TSP, RSP, SO_2, NO_2, NO, O_3	Quarterly data	
Wang H et al. 2003	Shenyang	1996–2000	Mortality	Cardiovascular disease	TSP	Monthly data	
Xia and Tong 2006	Hong Kong	1994–1997	Admissions	Circulatory and respiratory diseases	PM_{10}, SO_2, NO_2, O_3	365-Day lag	
Zhou YR et al. 1997	Chongqing	1991–1992	Admissions	Respiratory disease, COPD, cancer, injury	TSP, SO_2, NO_x	Monthly data	
India							
Pande et al. 2002	New Delhi	1997–1998	ER visits	COPD, asthma, acute coronary event	TSP, SO_2, NO_x, CO	No numerical estimates	
Japan							
Honda et al. 2003	Tokyo	1976–1990	Mortality	All causes	SO_2, NO_2, NO, CO, oxidant	No numerical estimates	SPM and NO_2 concentrations were associated with numbers of hospital admissions for asthma attacks in children < 6 yr. There was no such association in children 7–20 yr.
Murakami Y and Ono 2006	Tokyo	1990–1994	Mortality	Myocardial infarction	SPM	Analysis of peak SPM hourly only	
Ono et al. 2007	13 Largest cities	1990–1994	Mortality	All causes, cardiovascular disease, respiratory disease	SPM	No seasonal control	
Piver et al. 1999	Tokyo	1980–1995	Emergency transport	Heatstroke	PM_{10}, NO_2, O_3	Rare outcome	
Sawaguchi et al. 1997	47 Prefectures	1988–1994	Mortality	Sudden infant death syndrome	SO_2, NO_2	No numerical estimates	
Shimizu et al. 2001	Yokohama	1990–1991	ER visits	Asthma	SPM, SO_2, NO_2, NO	No numerical estimates	

Table continues next page

Appendix Table A.2 (*Continued*). Time-Series Studies Excluded from Quantitative Analysis

Country / Citation	Study Location	Study Period	Outcome	Disease	Pollutant	Reason for Exclusion	Comments
Japan (*Continued*)							
Shinkura et al. 1999	Yamashita public health district of Kagoshima City	1978–1988	Mortality	All causes	SO_2	Monthly data	
Singapore							
Emmanuel 2000	Singapore	1997	Mortality, ER visits	Respiratory disease	PM_{10}, SO_2, NO_2, CO, O_3, haze	Episode study of Asian dust storm	Effects of dust storms on clinic visits for allergic rhinitis were marked 2 days after event but were not statistically significant.
South Korea							
Choi and Paek 1995	Seoul	1993	Admissions	Asthma	TSP, SO_2, CO	Study < 1 yr in duration	
Hwang SS et al. 2005b	Seoul	2002	Mortality	All causes, respiratory disease, cardiovascular disease, other	PM_{10}	Episode study of Asian dust storm	Results suggested a causal relationship between dust storm and mortality from all causes, and cardiovascular and respiratory disease.
Jang et al. 2006	Bucheon, Gyenggi-do	2003	Admissions	Asthma, COPD, cardiovascular disease	PM_{10}, SO_2, NO_2, O_3	Monthly data	
Kim J and Yang HE 2005	Seoul	1999	Mortality	Respiratory and cardiovascular disease	TSP, PM_{10}, SO_2, NO_2, CO, O_3	No seasonal control	
Kim SY et al. 2007	Seoul	2002	Admissions	Asthma	PM_{10}, SO_2, NO_2, CO, O_3	Subject cohort was a subset with previous asthma	
Kwon et al. 2002	Seoul	1995–1998	Mortality	All causes, respiratory disease, cardiovascular disease	Asian dust storm (PM_{10}, SO_2, NO_2, CO, O_3)	Episode study of Asian dust storm	Effects of dust storms on cardiovascular disease were marked 1 day after the event but were not statistically significant.

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Appendix Table A.2 (*Continued*). Time-Series Studies Excluded from Quantitative Analysis

Country / Citation	Study Location	Study Period	Outcome	Disease	Pollutant	Reason for Exclusion	Comments
Taipei,China							
Chang CC et al. 2006	Taipei	1997–2001	Clinic visits	Allergic rhinitis	Asian dust storm	Episode study of Asian dust storm	During several months of haze from forest fires, an increase in the PM_{10} concentrations from 50 to 150 μg/m^3 was significantly associated with increases in outpatient visits for upper respiratory illness (12%), asthma (19%), and rhinitis (26%). Neither hospital admissions nor mortality increased significantly.
Chen CH et al. 2006b	Taipei	1998–2001	Admissions	Asthma, chronic bronchitis	PM_{10}, SO_2, NO_2, CO, O_3	Monthly data	
Chen YS et al. 2004	Taipei	1995–2000	Mortality	All causes, respiratory disease, cardiovascular disease	PM_{10}, SO_2, NO_2, CO, O_3	Episode study of Asian dust storm	Asian dust events were found to be weakly associated with mortality from all causes. However, the association between dust events and deaths from respiratory causes was stronger.
Chen YS and Yang CY 2005	Taipei	1996–2001	Admissions	Cerebrovascular disease	PM_{10}, SO_2, NO_2, CO, O_3	Episode study of Asian dust storm	Asian dust events were positively, but not significantly, associated with respiratory and cardiovascular mortality. The relationship was stronger in people ≥ 65 yr.
Hwang JS et al. 2000	Taipei	1994–1995	School absences	All causes	NO_x	No seasonal control	
Knöbel et al. 1995	Taipei	1981–1991	Mortality	SID or suffocation	PM_{10}, SO_2, CO, pollutant standard index, visibility	No numerical estimates	
Yang CY et al. 2005a	Taipei	1996–2001	Admissions	Stroke	Asian dust storm (PM_{10}, SO_2, NO_2, CO, O_3)	Episode study of Asian dust storm	Statistically significant associations between Asian dust storm and daily primary intracerebral hemorrhagic stroke and ischemic stroke were found.
Yang CY et al. 2005b	Taipei	1996–2001	Admissions	Asthma	Asian dust storm (PM_{10}, SO_2, NO_2, CO, O_3)	Episode study of Asian dust storm	Effects of dust storms on daily asthma admissions were not found to be statistically significant, possibly owing to inadequate sample size for detecting association.
Yang CY 2006	Taipei	1997–2001	ER visits	Conjunctivitis	Asian dust storm (PM_{10}, SO_2, NO_2, CO, O_3)	Episode study of Asian dust storm	The effects of dust storms on clinic visits for conjunctivitis were marked 4 days after the event. However, the association was not statistically significant.

Appendix Table A.3. Number of Studies Reporting Mortality and Admissions Results by Outcome and Pollutant[a]

Disease	Number of Studies with Numerical Estimates	Pollutant						
		TSP	PM_{10}	$PM_{2.5}$	SO_2	NO_2	CO	O_3
Mortality								
All natural causes	38	13	22	3	23	14	9	17
Respiratory disease	20	5	15	2	11	9	6	10
COPD	10	4	5		7	3	1	2
Cardiovascular disease	24	7	17	2	13	11	5	10
Cardiac disease	2		1		1			
Cerebrovascular event	10	2	6		6	6	2	5
Ischemic heart disease	3		2		1	1		2
Cancer	4	2	1		3			
Admissions								
Respiratory disease	4	1	3		4	4	1	4
Asthma	13	2	11	2	10	10	6	11
COPD	4		4	1	4	4	2	4
Cardiovascular disease	3		2		2	2		3
Cardiac disease	3		3		3	3	2	3
Cerebrovascular event	6		5	2	5	4	3	6
Ischemic heart disease	5		4	1	4	4	2	5
Heart failure	2		1		1	1		2
Dysrhythmia								1

[a] Studies that included more than one pollutant were counted with each pollutant.

Appendix Table A.4. Number of Studies Reporting Results by Age Group[a]

Age Group	Years	Number of Studies
Children	0–20	20
Young adults	5–64	7
Adults	11–64	4
Older adults	≥ 30	58
Not elderly	0–65	6
Elderly	≥ 65	20
Miscellaneous		9

[a] Studies that included analyses of more than one age group were counted in each group.

APPENDIX B. Additional Analyses of Effects of Long-Term Exposure

Appendix Table B.1. ORs (and 95% CIs) for Asthma from Qualitative Comparisons Between Areas with Differing Pollutant Levels in Studies of Urban and Rural Locations or Between Cities[a]

Citation	Study Details[b]	OR (95% CI)
Ho et al. 2007	Taipei,China, A+W-r, high vs. low CO, female	1.98 (1.54–2.56)
Ho et al. 2007	Taipei,China, A+W-r, high vs. low CO, male	1.78 (1.38–2.30)
Ho et al. 2007	Taipei,China, A+W-r, high vs. low O_3, female	1.00
Ho et al. 2007	Taipei,China, A+W-r, high vs. low O_3, male	1.02 (1.00–1.03)
Ho et al. 2007	Taipei,China, A+W-r, high vs. low PM_{10}, female	0.99 (0.99–1.00)
Ho et al. 2007	Taipei,China, A+W-r, high vs. low PM_{10}, male	1.00
Kuo et al. 2002	Taipei,China, A+W-rns, high vs. low NO_2, female + male	1.69 (1.16–2.48)
Kuo et al. 2002	Taipei,China, A+W-rns, high vs. low O_3, female + male	0.75 (0.32–1.77)
Kuo et al. 2002	Taipei,China, A+W-rns, high vs. low PM_{10}, female + male	0.95 (0.64–1.40)
Kuo et al. 2002	Taipei,China, A+W-rns, high vs. low SO_2, female + male	1.20 (0.67–2.13)
Lin RS et al. 2001c	Taipei,China, A+W-r, high vs. low CO, female + male	1.30 (1.18–1.43)
Lin RS et al. 2001c	Taipei,China, A+W-r, med vs. low CO, female + male	1.10 (1.03–1.17)
Qian Z et al. 2000	China, W-r, high- vs. low-pollution districts, female + male	1.48 (0.95–2.31)
Qian Z et al. 2004	China, W-r, high- vs. low-pollution districts, female + male	2.33 (1.71–3.17)
Qian Z et al. 2004	China, A-rns, high- vs. low-pollution districts, female + male	2.30 (1.26–4.20)
Wang TN 1999	Taipei,China, W-r, high vs. low CO, female + male	1.15 (1.10–1.20)
Wang TN 1999	Taipei,China, W-r, high vs. low NO_2, female + male	1.08 (1.04–1.12)
Wang TN 1999	Taipei,China, W-r, high vs. low O_3, female + male	1.11 (1.07–1.15)
Wang TN 1999	Taipei,China, W-r, high vs. low PM_{10}, female + male	1.00 (0.96–1.04)
Wang TN 1999	Taipei,China, W-r, high vs. low SO_2, female + male	0.98 (0.95–1.01)
Wang TN 1999	Taipei,China, W-r, high vs. low TSP, female + male	1.29 (1.24–1.34)
Yang CY et al. 1997	Taipei,China, W-r, industrial vs. non-industrial, female + male	1.65 (0.88–3.09)
Aggarwal et al. 2006	India, A-r, urban vs. rural, female + male	1.34 (1.19–1.51)
Chen PC et al. 1998	Taipei,China, A+W-rns, urban vs. rural, female + male	1.68 (1.11–2.54)
Hong CY et al. 2004a	Indonesia, W-rns, urban vs. rural, female + male	1.18 (0.46–3.03)
Hong SJ et al. 2004b	South Korea, W-r, industrial vs. rural in 1995, female + male	1.38 (0.80–2.38)
Hong SJ et al. 2004b	South Korea, W-r, industrial vs. rural in 2000, female + male	0.71 (0.31–1.62)
Hong SJ et al. 2004b	South Korea, W-r, urban vs. rural in 1995, female + male	1.25 (0.93–1.68)
Hong SJ et al. 2004b	South Korea, W-r, urban vs. rural in 2000, female + male	0.82 (0.58–1.16)
Yang CY et al. 1998	Taipei,China, W-r, industrial vs. rural, female + male	1.22 (0.69–2.16)

[a] The study details and ORs correspond to those shown in Figure 66.

[b] The study details consist of the location, outcome, pollutant levels or areas compared, and sex of subjects. Female + male indicates the data were combined. The six outcome categories were recent wheezing (W-r); recent asthma (A-r); wheezing, degree of recency not specified (W-rns); asthma, degree of recency not specified (A-rns); and asthma and wheeze together (A+W-r and A+W-rns).

Appendix Table B.2. ORs (and 95% CIs) for Asthma from Qualitative Comparisons Between Areas with Differing Pollutant Levels or Proximities to Traffic Within Cities[a]

Citation	Study Details[b]	OR (95% CI)
Langkulsen et al. 2006[c]	Thailand, A-r, high1 vs. low, female + male	2.60 (1.38–4.90)
Langkulsen et al. 2006[c]	Thailand, A-r, high2 vs. low, female + male	2.44 (1.21–4.92)
Langkulsen et al. 2006[c]	Thailand, A-r, high3 vs. low, female + male	1.57 (0.76–3.24)
Xu X and Wang L 1993	China, W-r, high vs. low PM, female + male	2.10 (1.20–3.68)
Xu X and Wang L 1993	China, W-r, mod vs. low PM, female + male	1.40 (0.80–2.45)
Yu TS et al. 2001	Hong Kong, W-r, high vs. low pollution, female + male	1.31 (0.65–2.64)
Yu TS et al. 2001	Hong Kong, A-rns, high vs. low pollution, female + male	1.98 (1.24–3.16)
Zhang J et al. 1999	China, A-rns, urban vs. suburban, female	0.84 (0.14–5.04)
Zhang J et al. 1999	China, W-r, urban vs. suburban, female	0.85 (0.41–1.76)
Zhang J et al. 1999	China, A-rns, urban vs. suburban, male	3.59 (1.36–9.48)
Zhang J et al. 1999	China, W-r, urban vs. suburban, male	0.71 (0.38–1.33)
Nakai et al. 1999	Japan, W-r, 20–150 m vs. residential, female	0.85 (0.45–1.61)
Nakai et al. 1999	Japan, W-r, < 20 m vs. 20–50 m, female	1.17 (0.59–2.32)
Nakai et al. 1999	Japan, W-r, < 20 m vs. residential, female	1.00 (0.48–2.08)
Nitta et al. 1993	Japan, W-r, 20–50 m vs. 50–150 m in 1982, female	1.17 (0.69–1.98)
Nitta et al. 1993	Japan, W-r, < 20 m vs. 20–150 m in 1979, female	2.75 (1.65–4.58)
Nitta et al. 1993	Japan, W-r, < 20 m vs. 20–150 m in 1983, female	0.94 (0.61–1.45)
Nitta et al. 1993	Japan, W-r, < 20 m vs. 50–150 m in 1982, female	1.52 (0.91–2.54)
Sekine et al. 2004	Japan, A-r, high vs. low traffic density, female	2.66 (0.98–7.22)
Sekine et al. 2004	Japan, W-r, high vs. low traffic density, female	1.34 (0.85–2.11)
Sekine et al. 2004	Japan, W-r, medium vs. low traffic density, female	1.39 (0.95–2.03)
Sekine et al. 2004	Japan, A-r, medium vs. low traffic density, female	1.99 (0.82–4.83)
Shima et al. 2003[d]	Japan, A-r, 0–49 m vs. rural, female	4.03 (0.90–18.05)
Shima et al. 2003	Japan, W-r, 0–49 m vs. rural, female	0.76 (0.08–7.22)
Shima et al. 2003	Japan, W-r, 0–49 m vs. rural, male	1.35 (0.34–5.36)
Shima et al. 2003[d]	Japan, A-r, 0–49 m vs. rural, male	3.77 (1.00–14.21)
Shima et al. 2003[d]	Japan, A-r, 50 m vs. rural, female	1.74 (0.63–4.81)
Shima et al. 2003	Japan, W-r, 50 m vs. rural, female	0.79 (0.26–2.40)
Shima et al. 2003[d]	Japan, A-r, 50 m vs. rural, male	1.99 (0.79–5.01)
Shima et al. 2003	Japan, W-r, 50 m vs. rural, male	1.02 (0.44–2.36)
Yang CY et al. 2002c	Taipei,China, W-r, high vs. low pollution, female + male	1.01 (0.84–1.21)
Yang CY et al. 2002c	Taipei,China, A-r, high vs. low pollution, female + male	0.94 (0.78–1.13)

[a] The study details and ORs correspond to those shown in Figure 67.

[b] The study details consist of the location; outcome; pollutant levels or areas or distances (m) from traffic compared; and sex of subjects. The six outcome categories were recent wheezing (W-r); recent asthma (A-r); wheezing, degree of recency not specified (W-rns); asthma, degree of recency not specified (A-rns); and asthma and wheeze together (A+W-r and A+W-rns).

[c] For Langkulsen et al. 2006, high1 vs. low means highly polluted general area vs. control area; high2 vs. low means highly polluted roadside area vs. control area; high3 vs. low means moderately polluted roadside area vs. control area.

[d] The ORs for Shima et al. 2003 are estimates of the relative risk of new occurrences of asthma.

Appendix Table B.3 ORs (and 95% CIs) for Asthma from Quantitative Comparisons[a]

Citation	Study Details[b]	OR per 10-μg/m^3 Change in Pollutant Concentration (95% CI)
Hwang BF et al. 2005a	Taipei,China, A-rns, CO, female + male	1.00 (1.00–1.01)
Hwang BF et al. 2005a	Taipei,China, A-rns, NO_x, female + male	1.00 (0.98–1.03)
Hwang BF et al. 2005a	Taipei,China, A-rns, O_3, female + male	1.07 (1.00–1.14)
Hwang BF et al. 2005a	Taipei,China, A-rns, PM_{10}, female + male	0.93 (0.91–0.96)
Hwang BF et al. 2005a	Taipei,China, A-rns, SO_2, female + male	0.95 (0.89–1.02)
Mi et al. 2006[c]	China, A-rns, NO_2, female + male	1.44 (1.06–1.96)
Mi et al. 2006[c]	China, W-r, NO_2, female + male	1.00 (0.74–1.35)
Mi et al. 2006[c]	China, W-r, O_3, female + male	0.43 (0.16–1.16)
Mi et al. 2006[c]	China, A-rns, O_3, female + male	0.54 (0.21–1.39)
Shima and Adachi 2000[d]	Japan, W-r, NO_2, female	1.08 (0.92–1.26)
Shima and Adachi 2000[d]	Japan, A-r, NO_2, female	1.05 (0.87–1.26)
Shima and Adachi 2000[d]	Japan, A-r, NO_2, female + male	1.47 (1.05–2.07)
Shima and Adachi 2000[d]	Japan, W-r, NO_2, female + male	1.34 (1.02–1.77)
Shima and Adachi 2000[d]	Japan, W-r, NO_2, male	1.13 (0.99–1.30)
Shima and Adachi 2000[d]	Japan, A-r, NO_2, male	1.13 (0.97–1.31)
Shima et al. 2002	Japan, A+W-r, NO_2, female	1.00 (0.83–1.20)
Shima et al. 2002	Japan, A+W-r, PM_{10}, female	1.03 (0.79–1.35)
Shima et al. 2002	Japan, A+W-r, NO_2, female + male	0.94 (0.78–1.14)
Shima et al. 2002[e]	Japan, A+W-r (incidence), NO_2, female + male	1.32 (1.02–1.71)
Shima et al. 2002	Japan, A+W-r, PM_{10}, female + male	1.00 (0.75–1.35)
Shima et al. 2002[e]	Japan, A+W-r (incidence), PM_{10}, female + male	1.50 (0.93–2.40)
Shima et al. 2002	Japan, A+W-r, NO_2, male	0.88 (0.66–1.17)
Shima et al. 2002	Japan, A+W-r, PM_{10}, male	0.98 (0.63–1.52)
Zhang JF et al. 2002	China, W-r, NO_x, female + male	0.97 (0.91–1.04)
Zhang JF et al. 2002	China, A-rns, NO_x, female + male	0.99 (0.93–1.06)
Zhang JF et al. 2002	China, W-r, PM_{10}, female + male	1.01 (0.94–1.09)
Zhang JF et al. 2002	China, A-rns, PM_{10}, female + male	1.03 (0.97–1.10)
Zhang JF et al. 2002	China, A-rns, $PM_{10–2.5}$, female + male	1.06 (0.96–1.17)
Zhang JF et al. 2002	China, W-r, $PM_{10–2.5}$, female + male	1.03 (0.90–1.17)
Zhang JF et al. 2002	China, W-r, $PM_{2.5}$, female + male	1.01 (0.87–1.18)
Zhang JF et al. 2002	China, A-rns, $PM_{2.5}$, female + male	1.05 (0.92–1.20)
Zhang JF et al. 2002	China, A-rns, SO_2, female + male	1.00 (0.97–1.04)
Zhang JF et al. 2002	China, W-r, SO_2, female + male	1.01 (0.98–1.05)
Zhang JF et al. 2002	China, W-r, TSP, female + male	1.01 (0.99–1.03)
Zhang JF et al. 2002	China, A-rns, TSP, female + male	1.01 (1.00–1.02)

[a] The study details and ORs correspond to those shown in Figure 68 [VI.5a].

[b] The study details consist of the location, outcome, pollutant, and sex of subjects. The six outcome categories were recent wheezing (W-r); recent asthma (A-r); wheezing, degree of recency not specified (W-rns); asthma, degree of recency not specified (A-rns); and asthma and wheeze together (A+W-r and A+W-rns).

[c] Mi et al. 2006 was conducted within a single city.

[d] The ORs from Shima and Adachi 2000 are for 4th, 5th, and 6th grades combined.

[e] Two of the ORs for females and males from Shima et al. 2002 were calculated from asthma incidence data.

Abbreviations and Other Terms

A-r	recent asthma
A-rns	asthma, recency not specified
A+W-r	recent asthma and wheeze
A+W-rns	asthma and wheeze, recency not specified
ACS	American Cancer Society
AHSMOG	Seventh-Day Adventist Health Study of Smog
ALRI	acute lower respiratory infection
APED	Air Pollution Epidemiology Database
APHEA	Air Pollution and Health: A European Approach
APHENA	Air Pollution and Health: A European and North American Approach
ATS	American Thoracic Society
BC	black carbon
BHR	bronchial hyperresponsiveness
BMRC	British Medical Research Council
CAI-Asia	Clean Air Initiative for Asian Cities
CI	confidence interval
CMB	chemical mass balance
CNG	compressed natural gas
CO	carbon monoxide
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
DALYs	disability-adjusted life-years
EC	elemental carbon
ECRHS	European Community Respiratory Health Services
ER	emergency room
ETS	environmental tobacco smoke
FEF	forced expiratory flow
$FEF_{25–75}$	forced expiratory flow during the middle half of FVC
FEV_1	forced expiratory volume in 1 second
FVC	forced vital capacity
GAM	generalized additive model
GDP	gross domestic product
GHG	greenhouse gas
GMAPs	Global Model for Ambient Particulates
GNI	gross national income
HEART	Health Effects of Air Pollution Reduction Trial
HVAC	heating, ventilation, and air-conditioning
IHD	ischemic heart disease
ISAAC	International Study of Asthma and Allergies in Childhood
ISOC	International Scientific Oversight Committee
IUATLD	International Union Against Tuberculosis and Lung Disease
LPG	liquefied petroleum gas
$MMEF_{25–75}$	maximum midexpiratory flow during the middle half of FVC
NAAQS	National Ambient Air Quality Standards
NAE	National Academy of Engineering
NHLBI	National Heart, Lung, and Blood Institute (U.S.)
NMMAPS	National Morbidity, Mortality, and Air Pollution Study
NMVOCs	non-methane volatile organic compounds
NO	nitric oxide
NO_3^-	nitrates
NO_2	nitrogen dioxide
NO_x	oxides of nitrogen
NRC	National Research Council
O_3	ozone
OC	organic carbon
OECD	Organisation for Economic Co-operation and Development
OR	odds ratio
PACF	partial autocorrelation function
PAHO	Pan American Health Organization
PAHs	polycyclic aromatic hydrocarbons
PAPA	Public Health and Air Pollution in Asia

PAPA–SAN	Public Health and Air Pollution in Asia — Science Access on the Net
PCA	principal-components analysis
PEFR	peak expiratory flow rate
PFT	pulmonary function test
PM	particulate matter
$PM_{2.5}$	PM with an aerodynamic diameter ≤ 2.5 μm
PM_{10}	PM with an aerodynamic diameter ≤ 10 μm
$PM_{10-2.5}$	PM with an aerodynamic diameter between 10 and 2.5 μm
PMF	positive matrix factorization
ppmV	parts per million by volume
PPP	purchasing power parity
REAS	Regional Emissions Inventory in Asia
RR	relative risk
RSP	respirable suspended particles
SE	standard error
SEPA	Chinese State Environmental Protection Administration
SO_2	sulfur dioxide
SO_4^{2-}	sulfate
SPM	suspended particulate matter
TB	tuberculosis
TSP	total suspended particles
UNEP	United Nations Environment Programme
UNFPA	United Nations Population Fund
UN-Habitat	United Nations Human Settlements Programme
U.S. EPA	U.S. Environmental Protection Agency
VO_2max	maximal oxygen uptake
VOCs	volatile organic compounds
W-r	recent wheeze
W-rns	wheeze, recency not specified
WHO	World Health Organization

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GLOSSARY

EPIDEMIOLOGIC STUDY DESIGNS AND BIOSTATISTICAL TERMS COMMONLY USED IN AIR POLLUTION STUDIES

Case–control study In a case–control study, persons in a population who develop particular diseases, or who die from those diseases, are identified and then classified according to their exposure to air pollution and other factors related to disease occurrence, such as age and cigarette-smoking status. These are the cases. A sample of members of the study population who were free of the disease at the time that the case subjects became ill or died (the controls) is also selected and classified in terms of the same risk factors. An estimate of the relative risk of disease or death that is associated with a particular factor, often termed an **odds ratio,** can then be calculated. The case–control approach provides an efficient alternative to a cohort study (see description below), because the relative risk of disease or death can be estimated in a population without having to collect information on all its members.

Case–crossover study In a case–crossover study, cases of, or deaths from, particular diseases that occur in a population are studied to provide an estimate of the effect of short-term exposure to air pollution. For each person who has the disease or who has died, exposure to air pollution is determined for a period near the time of diagnosis or death (the case period) and one or more periods during which the health event did not occur (control periods). The relative rate or relative risk can then be estimated using standard methods of analysis for matched case–control data.

Cohort study Cohort studies provide estimates of the effects of prolonged exposure to air pollution on mortality and morbidity due to chronic disease. In a cohort study, members of an entire population who are initially free of disease are observed over an extended period of time. Each member of the population is classified according to his or her exposure to air pollution, often determined on the basis of place of residence, and according to other factors related to the occurrence of disease, such as age and cigarette-smoking status. The rate or the risk of disease or death that occur among the exposed and unexposed persons are then compared in order to estimate the effects of exposure. Differences in these rates are often presented as a **rate ratio** or **relative risk**.

Confounder or confounding Confounding refers to confusing the effect of the variable of interest with the effect of another variable, the confounder. The confusion leads to a biased estimate of the effect of the variable of interest. For example, the effect of exposure to an air pollutant is our variable of interest; but other factors that affect human health (e.g., season, weather, tobacco smoking, indoor burning of solid fuels, and access to medical care) may be correlated in the same place and time as the exposure and their effects could be confused with those of the air pollutant. Confounding may be prevented in either the study design or the analysis of data, for example, by restricting study populations to those who are unexposed to potential confounders (e.g., nonsmokers) or through multivariable regression techniques.

Cross–sectional study Cross–sectional studies are typically used to study the effects of long-term exposure to air pollution on the prevalence of chronic respiratory symptoms and disease or on chronic impairment of pulmonary function. In a cross-sectional study, the disease status and exposure to air pollution of population members at a fixed point in time are determined. The effect of exposure on the prevalence of disease can then be calculated. In contrast to case–control or cohort studies (see above), however, in a cross-sectional study it is often not possible to determine the temporal relationship between exposure and disease.

Ecologic study In an ecologic study, also called an aggregate-level study, information is not collected on individual members of a population; instead, the relationship between average rates of disease or death and average exposure to air pollution in the population is analyzed. Such studies often make use of routinely collected data on both health outcomes and levels of air pollution. When ecologic studies compare incidence or mortality rates for whole populations in geographic regions with different average levels of air pollution, they can provide estimates of the relative risk of disease or death due to air pollution exposure. These estimates are often more difficult to interpret than those from case–control or cohort studies because they are not based on data on individual subjects.

Health impact study Health-impact studies, or assessments, are often used to estimate the potential benefits in terms of public health of actions taken to improve air quality. In a health impact study, the amount of disease, disability, or death attributable to exposure to air pollution in a specific locale is quantified. Such analyses make use

of data on the levels of air pollution and the rates of disease or death in that locale and estimates of the effect of air pollution on the incidence of disease or on mortality, such as relative risks from epidemiologic studies. The effect estimates may come from studies of the local population, but when such studies are not available, estimates from studies in other populations may also be used.

Lag time The interval between exposure to a given pollutant or pollutants and the subsequent measurement of health outcomes, for purposes of estimating the independent contribution of the pollutant levels on those health outcomes. The lag time can be a single interval (often a day), a cumulative period (usually 2 or more days) over which pollutant data are averaged, or a variable interval permitting the comparison of a given time point with pollutant data at each of several previous time points (called a "distributed lag"). The contributions of pollutant levels on outcomes yielded in lag-time models are called "lagged estimates." Lag times of 0 and 1 day are commonly used and are termed "lag 0" and "lag 1," respectively.

Meta-analysis Meta-analysis refers to the analysis of multiple studies, including statistical techniques for merging and contrasting results across studies. (Rothman, Greenland and Lash 2008 p. 652)

Panel study Panel studies are often used to estimate the effects of short-term exposure to air pollution on acute respiratory symptoms or changes in pulmonary function or other biomarkers. In a panel study, a small group, or "panel," of individuals is followed for a short period of time. During that period, health outcomes and exposure to air pollution are ascertained for each subject on several occasions, and the longitudinal relationship between exposure and outcome is analyzed.

Time-series study A time-series study provides an estimate of the effects of short-term exposure to air pollution. Typically, daily counts of health events, such as deaths or hospital admissions, in one or more locales are analyzed in relation to contemporaneous data on daily concentrations of air pollutants and other risk factors, such as weather, that vary over time. Statistical techniques, typically Poisson regression, are then applied to these data to estimate the proportional increase in the rate of disease or death that is associated with a given increment in exposure to air pollution.

POLLUTANTS COMMONLY STUDIED

Benzene A clear, colorless, volatile, highly flammable liquid with a characteristic odor. It is found in gasoline fumes, vehicle emissions, and tobacco smoke. Benzene is derived from petroleum and is used extensively as a raw material or solvent in many manufacturing processes.

Carbon monoxide (CO) A colorless, odorless, flammable gas that is toxic at high concentrations. It is produced by the incomplete combustion of fossil fuels and is emitted by motor vehicles and industrial activities.

Criteria pollutant An air pollutant targeted for regulation by the USEPA. Lead, particulate matter, sulfur dioxide, carbon monoxide, nitrogen oxides, ozone, are all criteria pollutants that the USEPA is required by the United States Congress in the Clean Air Act to regulate under national ambient air quality standards.

Lead (Pb) A soft, heavy, malleable toxic metallic element. It is found naturally as well as in certain manufactured products, such as lead-based paint and lead solder in water pipes. Historically, most ambient lead emissions were produced by motor vehicles and industrial sources (e.g., mining, smelting, and manufacturing). Today, lead in gasoline is banned in many Asian countries and throughout North America and Europe.

Nitrogen oxides (NO_x) A group of highly reactive gases containing nitrogen and oxygen in various proportions. Nitrogen dioxide (NO_2) is an example of NO_x and is a common pollutant. It is produced by the combustion of fossil fuels and is emitted by motor vehicles and industrial sources, such as waste incineration. In many urban locations, motor vehicles are the principal source.

Ozone (O_3) A strong-smelling, extremely reactive toxic form of oxygen. It is produced both naturally and in photochemical processes involving NO_x and volatile organic compounds emitted by motor vehicles and industrial activities.

Particulate matter (PM) Very small solid particles and liquid droplets suspended or carried in air, such as soot, dust, fumes, and mist. It is produced both naturally and by motor vehicles and industrial activities, which emit PM directly and emit other pollutants that react in the atmosphere to form additional PM. (Nitrogen and sulfur oxides, for example, react to form nitrates and sulfates, respectively.) PM gives smog its color and affects visibility. It comes in a wide range of sizes, including:

- Total suspended particulates (TSP) in all sizes
- PM 10 μm or smaller in aerodynamic diameter (PM_{10}) or respirable suspended particulates (RSP)
- PM 2.5 μm or smaller in aerodynamic diameter ($PM_{2.5}$)

Sulfur dioxide (SO_2) A heavy, colorless, pungent-smelling toxic gas. It is produced naturally and by the combustion of fossil fuels containing sulfur (such as gasoline, diesel, fuel oil, certain coals, and natural gas). Sulfur dioxide is emitted by motor vehicles and industrial activities.

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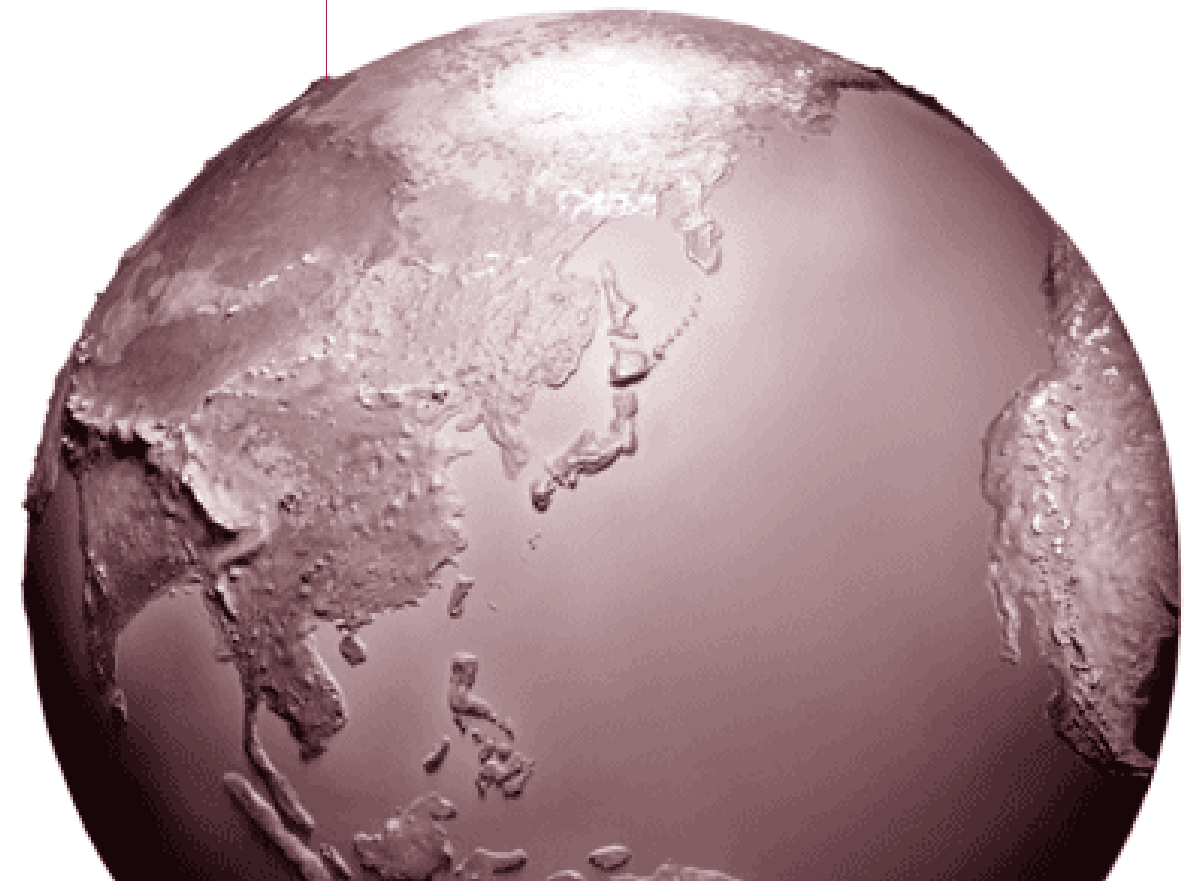

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SPECIAL REPORT 18

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