The Yangtze River Delta (YRD), which generally refers to southern Jiangsu Province, eastern and northern Zhejiang Province, and the municipality of Shanghai, is the fastest growing economic development region in China and one of the most densely populated regions in the world. Shanghai is one of the world’s largest cities, with >18 million long-term residents and a population density of >40,000 people/km² in some districts. Accompanying this economic development has been a dramatic increase in energy consumption and air pollution emissions. For example, although the Shanghai metropolitan area and the provinces of Jiangsu and Zhejiang constitute only 2% of the area of China, their emissions of sulfur dioxide (SO₂), nitrogen oxides (NOₓ), and fine particulate matter [aerodynamic diameter ≤ 2.5 μm (PM₂.₅)] account for 12%, 15%, and 12%, respectively, of total emissions in China in 2006, which increased by 36%, 55%, and 14%, respectively, from 2001 to 2006 (Zhang et al. 2009). NOₓ emissions are of particular concern because they have increased the fastest and are forecasted to increase even more (Chen et al. 2006). Several studies (Kan et al. 2004; Li et al. 2004; Streets et al. 1999) have evaluated the health benefits of air pollution control in Shanghai, primarily SO₂ and PM₁₀, and occasionally sulfate particles. Similar studies have been conducted in other parts of China, such as a recent estimate of annual deaths attributable to air pollution in the Pearl River Delta (Loh et al. 2008). In another study in the Pearl River Delta area, Wang et al. (2005) investigated how the emissions from different sectors influenced the concentrations of gaseous pollutants including ozone. And Wang and Mauzerall (2006) quantified the total health damages from PM due to anthropogenic emissions from Zaozhuang, Shandong Province. Besides these studies on regional air pollution, both the magnitude of the air pollution problem in China at the national level and the contribution from the power plants have been estimated by several studies, including one of the first to quantify the national burden of air pollution (World Bank 1997). In another study, Wang and Smith (1999) focused on the electric power sector in the context of determining the secondary benefits of greenhouse gas reductions. In addition, a large-scale study conducted (Ho and Nielsen 2007) assessed the health damages of air pollution in China and examined several pollution control policies and how they might affect economic performance.

Researchers have pointed out that emission reductions in different sectors may have different levels of effectiveness on reducing human exposure (Li et al. 2004; Streets et al. 1999) and that the benefits of many pollution control measures likely far exceed the costs; however, the variance by sector and its policy implications for future pollution control have not been investigated systematically in China. For example, none of the previous studies included both ozone and PM₂.₅ in assessing the health damages, although exposures have been associated with increased mortality and a variety of other health outcomes (Bell et al. 2006, 2007; Levy et al. 2005; Pope and Dockery 2006). The exclusion of ozone is partly because of past emphasis on the power sector, where SO₂ and PM receive greater attention, but is also attributable to limitations in the atmospheric models used in previous studies (Li et al. 2004). Also, most studies used PM₁₀ and total suspended particles (TSPs) to estimate population exposure to PM in China, whereas epidemiologic studies in the United States and worldwide have demonstrated more robust associations with PM₂.₅.

Our study will fill this gap by comparing how emission control strategies across different sectors (e.g., power generation, mobile sources, industry) influence population exposures and health risks related to PM₂.₅ and ozone in the YRD area. The sectoral details will help guide development strategies that are economically and environmentally optimal, providing the basis for policy makers to determine how to...
prioritize future control efforts among the different sectors in the YRD.

**Materials and Methods**

In this study, we applied air pollution health impact assessment methods, following approaches articulated elsewhere (National Research Council 2002; World Health Organization 2000). Briefly, this entailed estimating baseline emissions and the marginal contribution from individual source categories, use of a chemistry-transport model to characterize population exposures associated with source category emissions, and application of concentration-response (C-R) functions from the epidemiological literature along with characterization of population patterns to quantify health impacts.

**CMAQ modeling and emission inventory.** A state-of-the-science Eulerian grid model—the Community Multiscale Air Quality (CMAQ) modeling system [version 4.6; U.S. Environmental Protection Agency (EPA), Research Triangle Park, NC, USA] was applied to an emission inventory we developed to estimate the baseline concentrations as well as the marginal concentration change associated with hypothetical control strategies for multiple sectors in the YRD. CMAQ has capabilities to simulate the various chemical and physical processes important for understanding atmospheric processes and thus allowed us to model population exposure to pollutants such as ozone and PM$_{2.5}$. Although CMAQ is currently used in analyses in the United States (Cohan et al. 2007) and in some air quality assessments in developing countries, we used the fifth-generation mesoscale model with 27-km (covering all of China), 9-km (covering eastern China), and 3-km (covering the YRD) grid resolutions, respectively [see Supplemental Material, Figure S1 (doi:10.1289/ehp.1001991)]. Outputs from the 27-km and 9-km domains were used as boundary conditions for the 3-km domain. To develop meteorological inputs for CMAQ, we used the fifth-generation mesoscale model (MM5) developed by the U.S. National Center for Atmospheric Research. For more detailed information on CMAQ and MM5 configurations, see Supplemental Material, Table S1.

The emission inventory for the YRD consisted of emissions mainly from four sectors: power plants, industrial sources (e.g., metallurgical, mineral, cement, chemical industries, small industries such as coke and brick production), mobile sources, and domestic life (e.g., livestock, residential, and biomass burning). Power plants and emissions from production processes of major industrial sources were modeled as point sources. Mobile sources mainly included on-road vehicle emissions in the major cities. Emissions from domestic life and the fugitive emissions from industry were modeled as area sources.

In developing our emission inventory, we used the Asian emission inventory for 2006 that was developed for the INTEX-B project of the U.S. National Aeronautics and Space Administration (Zhang et al. 2009). The resolution of the INTEX-B emission inventory is 30 min × 30 min (~55 km × ~55 km). To capture finer resolution within our emission inventory, we incorporated various sources of input, using collaborations with local agencies, Google Earth (version 5.1.3535.3218; Google Inc., Mountain View, CA, USA) to identify the locations of large point sources based on their addresses, updated road networks and LandScan population data to characterize the distribution of mobile source emissions, and the statistical yearbook of the major cities in the YRD for domestic fuel emissions from the consumption of coal, liquefied petroleum gas, coal gas, and natural gas.

After conducting the base-case simulation using CMAQ, we validated it using available monitoring data for ozone and PM$_{2.5}$ collected in Shanghai before proceeding with additional model runs. Because PM$_{2.5}$ is not currently a criteria air pollutant in China and there are no publicly available ozone monitoring data, the monitoring data we had access to for model validation were all from research monitoring sites located in Shanghai and operated by Fudan University [see Supplemental Material, Figure S2 (doi:10.1289/ehp.1001991)]. Hourly ozone monitoring data were available from three stations for 4 weeks (2–9 May, 12–19 June, 10–17 July, and 14–21 August 2006). Daily average PM$_{2.5}$ monitoring data were available from one station for 1–29 April, 1–26 August, and 1–28 November 2006.

Once the validation of the baseline run was satisfactory, we designed scenarios to model the marginal contribution of selected emission sectors in the YRD to the baseline concentrations in the 3-km resolution domain. Although some underestimation of the total health benefits would have resulted, given the significant regional transport of PM$_{2.5}$ and ozone, the size of the 3-km domain provides YRD environmental authorities with the most relevant information for control strategy prioritization and decision making.

For this study, we modeled sectoral impacts in total (e.g., all power plants in the YRD simultaneously). Table 1 lists the emission reduction scenarios in more detail. In designing the scenarios, we considered several factors.

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Sector</th>
<th>Pollutants reduced</th>
<th>Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Power</td>
<td>NO$_x$ alone (SCR alone)</td>
<td>85%</td>
</tr>
<tr>
<td>2</td>
<td>Power</td>
<td>NO$_x$ + SO$_2$ (SCR + FGD)</td>
<td>85% for NO$_x$ + 90% for SO$_2$</td>
</tr>
<tr>
<td>3</td>
<td>Mobile</td>
<td>NO$_x$ alone</td>
<td>20%</td>
</tr>
<tr>
<td>4</td>
<td>Mobile</td>
<td>VOC alone</td>
<td>20%</td>
</tr>
<tr>
<td>5</td>
<td>Mobile</td>
<td>NO$_x$ + VOC + PM</td>
<td>20%</td>
</tr>
<tr>
<td>6</td>
<td>Mobile</td>
<td>NO$_x$ + VOC + PM</td>
<td>50%</td>
</tr>
<tr>
<td>7</td>
<td>Industry</td>
<td>NO$_x$ alone</td>
<td>20%</td>
</tr>
<tr>
<td>8</td>
<td>Industry</td>
<td>VOC alone</td>
<td>20%</td>
</tr>
<tr>
<td>9</td>
<td>Industry</td>
<td>NO$_x$ + VOC + PM</td>
<td>20%</td>
</tr>
<tr>
<td>10</td>
<td>Domestic</td>
<td>NO$_x$ alone</td>
<td>20%</td>
</tr>
</tbody>
</table>

Abbreviations: FGD, fluidized gas desulfurization; SCR, selective catalytic reduction.
exposure under different scenarios, which includes changes in multiple constituents. To estimate the pollutant-specific benefits, we used the incremental population exposure change between scenarios. For example, the difference in population exposure between scenario 1 and the base-case allowed us to calculate exposure reductions associated with NO_x emissions, whereas the difference between scenarios 2 and 1 allowed us to approximate the exposure reductions associated with SO_2 emissions.

**Population data.** To calculate the population exposure and subsequent health impacts, we collected population data from two different sources. First, we used LandScan 2007 as the primary basis for estimating exposures. LandScan is a worldwide population database compiled on a 30-second × 30-second latitude/longitude grid. Census counts (at subnational level) were apportioned to each grid cell based on likelihood coefficients, which are based on proximity to roads, slope, land cover, nighttime lights, and other information (Dobson et al. 2000; Oak Ridge National Laboratory 2009). Similar to Census data, LandScan provides a single population estimate for each location, although these estimates include diurnal movements and collective travel habits, whereas most censuses count people at their nighttime residences. In addition, LandScan includes annual updating of data inputs, as well as global coverage, which potentially allows for easy comparisons with other parts of the world in the future.

However, LandScan does not provide the same level of detail as Census data in terms of population demographics, so we obtained additional information necessary for subsequent calculation of health benefits (e.g., baseline mortality rates) from China Census 2000 (China Data Center 2003). To calculate the health benefits, we applied spatially variable baseline mortality rates, to reflect the fact that the YRD area includes both urban and rural areas and that their residents may have different disease patterns, socioeconomic status, and life expectancy. For counties in the YRD, the average baseline mortality rate is 0.6%, with the 5th and 95th percentile rate at 0.34% and 0.84%, respectively. We assumed these estimates based on Census 2000 are applicable to population estimates from LandScan 2007 data.

**Population exposure.** The population exposure change under different scenarios was calculated by combining population in each location with the corresponding concentration change. Geographical information system (ArcGIS) software (version 9.3; ESRI, Redlands, CA, USA) was used to convert population data to match the grid structure of CMAQ. Because the emissions of different pollutants under study vary significantly by sector, we focused our analyses on the marginal benefits per ton of emission reductions. We facilitated these comparisons by using the concept of intake fraction (iF), the fraction of a material released from a source that is inhaled or ingested (Bennett et al. 2002). We calculated iFs as \((\sum C_i \times P_i) \times (BR_i/Q_i)\), where \(C_i\) is the marginal concentration in grid cell \(i\) (micrograms per cubic meter) associated with source emission rate \(Q\) (micrograms per day, noting that \(Q\) can be the same pollutant as \(C\) or a precursor to \(C\), \(P_i\) is the population count in the grid cell, and \(BR_i\) is a nominal breathing rate of 20 m^3/day. We calculated iFs for primary PM as well as secondary PM associated with various particle precursors (SO_2, NO_x, VOCs) and ozone (defined as the mass of ozone inhaled per unit mass of NO_x or VOC emissions). Because numerous particle constituents are influenced by precursor emission changes in CMAQ, we did not focus on iFs for individual constituents but discuss the dominant constituents for all secondary PM iFs. In addition, because emissions of multiple pollutants can influence PM_{2.5} and ozone concentrations, we estimated the pollutant-specific and sector-specific iFs by comparing the population exposures among scenarios.

**Health effects.** Although detailed characterization of health risks is beyond the scope of our investigation, we developed C-R functions for PM_{2.5} and ozone mortality to allow our CMAQ outputs to be integrated into a common metric and to allow for initial evaluation of the magnitude of health benefits, the dominant pollutants, and the key uncertainties. For both PM_{2.5} and ozone, we determined a central estimate, plausible lower bound, and plausible upper bound. These are not meant as formal 95% CIs but were used to construct uncertainty distributions when combining population exposures among scenarios.

As a general point, there are multiple limitations in applying C-R functions largely derived from the United States or Europe to China. There are differences in baseline disease patterns and age distributions, health care systems, pollutant levels and composition, and exposure modifiers. There are also complexities given the more recent focus on PM_{2.5} in U.S. and European studies but the use of PM_{10} or TSP in China given available monitoring data. To develop applicable C-R functions, we used a combination of evidence from the global literature and the Chinese literature.

First we considered PM: Two studies that developed C-R functions applicable to China (Aunan and Pan 2004; Levy and Greco 2007) concluded that the Chinese time-series mortality literature yielded estimates on the order of 0.3–0.4% increases in all-cause mortality per 10 μg/m^3 increase in daily PM_{10} concentrations, slightly lower than the global literature. In a recent study that examined three cities in China (Wuhan, Shanghai, and Hong Kong), Wong et al. (2008) found a pooled C-R function for time-series mortality of 0.37% (95% CI, 0.21–0.54%), similar to the values reported above. C-R functions in China were higher for cardiovascular and respiratory mortality, with patterns similar to those seen in the global literature.

However, health risk assessments for PM generally apply evidence from cohort mortality studies (National Research Council 2002; World Health Organization 2000), given the strength of available studies and supporting evidence for mortality risks from long-term exposure (e.g., evidence that PM contributes to accelerated atherosclerosis; Floyd et al. 2009). Recent syntheses of the cohort mortality literature (Levy et al. 2009) and expert elicitation studies (Industrial Economics 2006) found that a 1% increase in mortality per 1 μg/m^3 increase in annual PM_{2.5} concentrations was a reasonable central estimate, falling between estimates from the Harvard Six Cities Study (Laden et al. 2006; Schwartz et al. 2008) and the American Cancer Society study (Jerrett et al. 2009; Pope et al. 2002). There is no cohort mortality evidence available in China, but earlier cross-sectional studies yielded C-R functions roughly comparable to those from the U.S. cohort studies (Levy and Greco 2007).

Despite the lack of Chinese cohort mortality evidence, the literature is sufficiently compelling to indicate that mortality risk due to long-term exposure would be expected, and comparisons of the time-series estimates indicate reasonable concordance between the Chinese and U.S. literature despite the large differences in ambient concentrations and other factors. Thus, for our central estimate, we used a 1% increase in all-cause mortality per 1 μg/m^3 increase in annual PM_{2.5} concentrations. For our bounds, we note that a recent study (Levy et al. 2009) used 0.3% as a lower bound and 2.0% as an upper bound, representing the median values across experts for the 5th and 95th percentiles of the uncertainty distribution in the recent expert elicitation study. We maintained this upper bound (which slightly exceeds the central estimate from the Harvard Six Cities Study) but used a lower bound of 0.1%, reflecting a value similar to the time-series evidence and the uncertainties in determining a cohort mortality effect in China without direct evidence.

These C-R functions were applied identically to all particle constituents, given a lack of systematic information to support quantitative differential toxicity, especially with respect to atmospheric conditions in China.

Ozone had not been characterized in previous studies developing C-R functions for China, in part because of a lack of systematic evidence in the global literature at the time of those investigations. However, three recent meta-analyses and multicity studies (Bell et al. 2010, 2011, 2012) provided a more compelling basis for characterizing ozone health effects. These studies used a range of methods to combine evidence from different observational studies, and the meta-analyses included several studies from China. Bell et al. (2010) found a pooled C-R function for time-series mortality of 0.3% (95% CI, 0.12–0.48%), with the upper bound of 0.48% corresponding to China. These results are consistent with earlier meta-analyses of cohort mortality studies, which found that the Chinese time-series mortality literature was not as clear as the U.S. literature in showing a consistent pattern of increasing mortality with ozone exposure (Bell et al. 2004, 2006). Bell et al. (2011, 2012) also found that the ozone-disease relationship was stronger for cardiovascular and respiratory mortality than for all-cause mortality.

### Exposure-Response Functions

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Exposure Metric</th>
<th>RH Factor</th>
<th>Baseline Mortality</th>
<th>Mortality Risk</th>
<th>Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM_{2.5}</td>
<td>PM_{2.5}</td>
<td>0.5</td>
<td>0.2%</td>
<td>0.3%</td>
<td>0.21–0.54%</td>
</tr>
<tr>
<td>Ozone</td>
<td>Ozone</td>
<td>0.7</td>
<td>0.6%</td>
<td>0.3%</td>
<td>0.12–0.48%</td>
</tr>
</tbody>
</table>

### Emissions and Concentrations

- **Primary PM:**
  - PM_{10} emissions from various sources.
  - PM_{2.5} concentrations estimated from CMAQ outputs.
- **Secondary PM:**
  - SO_2, NO_x, VOCs concentrations calculated from precursor emissions.
- **Ozone:**
  - Ozone concentrations estimated from CMAQ outputs.

### Health Effects

- **Cardiovascular:**
  - Increased risk with PM_{2.5} and ozone exposure.
  - Central estimate: 1% increase in mortality per 1 μg/m^3.
  - Lower bound: 0.1%.
  - Upper bound: 2.0%.
- **Respiratory:**
  - Similar risks as cardiovascular.
  - Central estimate: 1% increase in mortality per 1 μg/m^3.
  - Lower bound: 0.1%.
  - Upper bound: 2.0%.
- **All-cause:**
  - Central estimate: 1% increase in mortality per 1 μg/m^3.
  - Lower bound: 0.1%.
  - Upper bound: 2.0%.

### Summary

Our analysis of health impacts from air pollution in the YRD region indicated significant potential benefits from reducing emissions of PM_{2.5} and ozone. The central estimates suggest a 1% increase in mortality per 1 μg/m^3 increase in annual concentrations of these pollutants, which is similar to findings from previous studies in the United States and Europe. Further research is needed to refine these estimates and to understand the long-term effects of exposure to these pollutants in China.
2005; Ito et al. 2005; Levy et al. 2005) found evidence of an independent ozone effect in the time-series literature; ozone mortality was recently evaluated in multiple Chinese cities (Wong et al. 2008), and recent evidence from the American Cancer Society cohort study (Jerrett et al. 2009) provides some indication of a long-term ozone effect on respiratory mortality. Thus, ozone mortality merits inclusion in our investigation.

Using standard units conversions and an assumption of an approximate 3:4 ratio between 8-hr maximum and 1-hr maximum concentrations (Levy et al. 2005), the three meta-analyses (Bell et al. 2005; Ito et al. 2005; Levy et al. 2005) yield C-R functions for all-cause mortality of 0.21% (95% CI, 0.15–0.32%), 0.27% (95% CI, 0.18–0.35%), and 0.28% (95% CI, 0.21–0.35%), respectively, per 10-μg/m³ increase in 8-hr maximum ozone. The recent multi-city study in Asia (Wong et al. 2008) found that, for the three Chinese cities combined, the C-R function for all-cause mortality was 0.31% (95% CI, 0.13–0.48%). Central estimates were very similar for cardiovascular and respiratory mortality (0.29% and 0.23%, respectively, although both estimates lacked statistical significance), and the all-cause mortality estimate for Shanghai is identical to the three-city pooled estimate. As for PM, little evidence exists that the ozone time-series mortality C-R function differs systematically between the Chinese cities and the global literature.

Similar to PM$_{2.5}$, there is no evidence of ozone cohort mortality for China. In the U.S. cohort literature, an ozone effect on mortality was significant in one recent publication (Jerrett et al. 2009), but only for respiratory mortality in models including PM$_{2.5}$. The C-R function for respiratory mortality corresponded to a 2.7% increase per 10-μg/m³ increase in 8-hr maximum ozone following the conversions above. This is significantly greater than the time-series estimates (albeit for respiratory mortality, only a fraction of all-cause mortality), but because of the lack of an impact for all-cause mortality or corroboration from other studies, we do not use this evidence for our primary C-R functions.

Combining this evidence, we considered 0.3% reduction in all-cause mortality per 10-μg/m³ reduction in 8-hr maximum ozone as a reasonable central estimate (reflecting the Chinese three-city study and two of the meta-analyses), with 0.15% as a lower bound (reflecting the lower confidence limits of the various studies) and 0.45% as an upper bound (reflecting the upper confidence limit of the Chinese three-city study as well as modest weight on the emerging cohort mortality evidence).

**Health benefit estimation.** Although we did not develop the upper and lower bound C-R functions as specific percentiles of uncertainty distributions, we wished to estimate net health benefits across PM$_{2.5}$ and ozone, necessitating some combination of distributions. To approximate the overall net mortality change considering the uncertainty in C-R functions, we assumed that the C-R functions for PM$_{2.5}$ and ozone each follows a triangular distribution with central estimate as the mode and the lower and upper bounds as the minimum and maximum values for the distribution. We performed Monte Carlo analysis using SAS 9.1 (SAS Institute Inc., Cary, NC, USA) to combine the distributions, noting that uncertainty in other risk assessment components was not considered.

**Results**

**Comparison between CMAQ modeling and monitoring data.** To validate the performance of CMAQ, we compared ozone and PM$_{2.5}$ modeling results in the base-case scenario with observation data for part of 2006 at four monitoring stations in Shanghai, using U.S. EPA guidance (U.S. EPA 2007). The mean normalized bias (MNB) between model and observational data was relatively low for both daily average PM$_{2.5}$ (0.5%) and daily maximum 8-hr ozone (~4.3%), indicating a lack of systematic model bias. For hourly ozone, the model underestimated concentrations somewhat, with MNB of ~25.3% and normalized mean error of 29%. In general, model performance was considered adequate for our application.

**Base-case emissions and ambient concentrations.** In Table 2, we show the estimated emission rates by sector and pollutant in the base-case scenario. Of note, emissions of many pollutants are high in the industry sector, in part due to the active manufacturing industry in the YRD and the lower penetration rate for pollution control technologies compared with the power sector.

![Figure 1](image) Estimated annual average PM$_{2.5}$ concentration (μg/m³) in the YRD domain in the base-case scenario.
Figure 1 shows the annual average PM$_{2.5}$ concentration in the YRD domain in the base-case scenario. The estimated mean annual PM$_{2.5}$ concentration in the YRD domain was 38.4 μg/m$^3$, although with significant variation across the study domain (range, 12.7–132.8 μg/m$^3$). Similarly, Figure S4 in Supplemental Material (doi:10.1289/ehp.1001991) shows the annual average 8-hour maximum ozone concentration, which ranged from 17 to 54 ppb.

**iF variation by pollutant and sector.** To estimate the pollutant-specific iFs, we compared the population exposures among scenarios (Table 3). iFs ranged significantly across pollutants, with more modest differences across sectors. Primary PM$_{2.5}$ from the industry and mobile source sectors have the highest iF of $1.4 \times 10^{-5}$, which means that for every metric ton of primary PM$_{2.5}$ emitted from either sector, 14 g is eventually inhaled by the total population in the YRD domain. For secondary PM$_{2.5}$, the iF was greatest for SO$_2$ emissions from the power sector, with a value of $1.2 \times 10^{-6}$. Among the different species of PM$_{2.5}$ modeled by CMAQ, 98% of the PM$_{2.5}$ concentration reduction was attributable to sulfate and ammonium particles. For secondary PM$_{2.5}$ from NO$_x$ emissions, the iFs from power plants, mobile sources, and industry are nearly identical ($3.9 \times 10^{-7}$). In each case, the concentration reduction was driven by nitrate and ammonium reductions with an offsetting increase in sulfate (35% of the magnitude of the nitrate and ammonium reductions for the power sector, 20% for mobile sources, and 19% for industry). In contrast, the domestic emissions sector has a negative iF for secondary PM$_{2.5}$ from NO$_x$ emissions, potentially attributable to two factors. The low NO$_x$ emissions within the domestic sector translated into a small reduction in secondary nitrate population exposure. Second, when compared with other scenarios, the domestic sector had the greatest increase in ozone concentrations per unit NO$_x$ emission reductions, which contributed to greater oxidizing power of the atmospheric and subsequent increases in other PM$_{2.5}$ species (e.g., secondary sulfate and secondary organic aerosols). As a result, there is an increase in overall PM$_{2.5}$ concentration corresponding to the NO$_x$ emission reductions within the domestic sector.

Across all scenarios, reducing NO$_x$ emissions alone led to an increase in ozone population exposure (as indicated by the negative values in Table 3, "Ozone from NO$_x$"). However, when VOC emissions are reduced alone, ozone population exposures are reduced and the iF is on the order of $1 \times 10^{-6}$ (as shown in Table 3, "Ozone from VOC"). This indicates that the YRD model domain is VOC limited in terms of ozone formation, explainable by the high baseline NO$_x$ emissions and low biogenic VOCs. Findings were similar using 1-hr maximum ozone concentrations, with iFs approximately 10–30% higher. Of note, within scenarios with concurrent NO$_x$ and VOC controls, we observed only modest differences from the sum of NO$_x$ and VOC controls applied separately.

**Health benefit variation by pollutant and sector.** Although the health benefits per ton of emission reductions are approximately proportional to the iF values in Table 3 (when baseline mortality rates are about constant in different parts of the domain), the absolute benefits of the control scenarios will also depend on the magnitude of emission reductions. As indicated in Table 4, the greatest mortality reduction is achieved by controlling primary PM$_{2.5}$ emissions from the industry sector and by controlling SO$_2$ emissions from the power sector, with approximately 12,000 fewer deaths per year (lower bound of 1,200, upper bound of 24,000). This is attributable to the high primary PM$_{2.5}$ iF and relatively high magnitude of emission reductions from the industry sector, whereas the SO$_2$ emissions from the power sector have an order of magnitude lower iF but an order of magnitude higher emission reduction.

For control scenarios addressing NO$_x$ emissions, the health benefits from secondary PM$_{2.5}$ reductions (which are themselves reduced by offsetting increases in sulfate concentrations) are blunted by adverse health impacts associated with ozone increases (Table 4). Although the NO$_x$ control scenarios still have positive net benefits (excluding the domestic sector), the net benefits are small relative to the aforementioned benefits for primary PM$_{2.5}$ and SO$_2$ controls.

**Discussion**

To maximize the health benefits of emission reductions among different pollutants from different sectors, there are two major factors to consider. First, NO$_x$ and VOC reductions are required since they are the major multicompound pollutants driving air pollution and health outcomes. Second, it is important to maximize the benefits achievable by each of these pollutants since the benefits from one pollutant are offsetting the other.

---

**Table 3.** iFs for primary PM$_{2.5}$, secondary PM$_{2.5}$, and 8-hour maximum ozone by emissions sector.

<table>
<thead>
<tr>
<th>Sector</th>
<th>Power</th>
<th>Mobile</th>
<th>Industry</th>
<th>Domestic</th>
</tr>
</thead>
<tbody>
<tr>
<td>iF</td>
<td>Scenarios compared</td>
<td>iF</td>
<td>Scenarios compared</td>
<td>iF</td>
</tr>
<tr>
<td>Primary PM$_{2.5}$</td>
<td>1.4 × 10$^{-5}$</td>
<td>5, 4, 3</td>
<td>1.4 × 10$^{-5}$</td>
<td>9, 8, 7</td>
</tr>
<tr>
<td>Secondary PM$_{2.5}$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>From SO$_2$</td>
<td>1.2 × 10$^{-6}$</td>
<td>2, 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>From NO$_x$</td>
<td>3.9 × 10$^{-7}$</td>
<td>1</td>
<td>3.9 × 10$^{-7}$</td>
<td>7</td>
</tr>
<tr>
<td>From VOC</td>
<td>2.4 × 10$^{-7}$</td>
<td>4</td>
<td>1.3 × 10$^{-7}$</td>
<td>8</td>
</tr>
<tr>
<td>Ozone</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>From NO$_x$</td>
<td>–6.8 × 10$^{-7}$</td>
<td>1</td>
<td>–6.9 × 10$^{-7}$</td>
<td>7</td>
</tr>
<tr>
<td>From VOC</td>
<td>1.7 × 10$^{-6}$</td>
<td>4</td>
<td>1.4 × 10$^{-6}$</td>
<td>8</td>
</tr>
</tbody>
</table>

Blank cells indicate values not estimated in any scenario runs. iF results reported are unitless. To calculate pollutant-specific iFs for primary PM$_{2.5}$, secondary PM$_{2.5}$, and 8-hr maximum ozone by emissions sector.

**Table 4.** Mortality change estimates for control scenarios by sector and pollutant.

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Sector</th>
<th>Pollutant controlled</th>
<th>Emission reductions from base-case (1,000 tons/year)</th>
<th>PM-related mortality change per year</th>
<th>Ozone-related mortality change per year</th>
<th>Net mortality change (PM and ozone) per year</th>
<th>Net mortality change per year per ton of emissions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Power</td>
<td>NO$_x$</td>
<td>610</td>
<td>2,000 (200, 4,000)</td>
<td>–420 (–210, –630)</td>
<td>1,600 (350, 2,900)</td>
<td>2.7 × 10$^{-3}$</td>
</tr>
<tr>
<td>2</td>
<td>Power</td>
<td>SO$_2$</td>
<td>1,300</td>
<td>12,000 (1,200, 24,000)</td>
<td>0</td>
<td>12,000 (1,200, 24,000)</td>
<td>9.2 × 10$^{-3}$</td>
</tr>
<tr>
<td>3</td>
<td>Mobile</td>
<td>NO$_x$</td>
<td>83</td>
<td>260 (26, 520)</td>
<td>–60 (–30, –90)</td>
<td>210 (41, 380)</td>
<td>2.5 × 10$^{-3}$</td>
</tr>
<tr>
<td>4</td>
<td>Mobile</td>
<td>VOC</td>
<td>200</td>
<td>380 (38, 750)</td>
<td>38 (19, 57)</td>
<td>430 (190, 680)</td>
<td>2.1 × 10$^{-3}$</td>
</tr>
<tr>
<td>5</td>
<td>Mobile</td>
<td>Primary PM</td>
<td>8</td>
<td>(500, 62, 1,200)</td>
<td>0</td>
<td>20 (62, 1,200)</td>
<td>1.0 × 10$^{-3}$</td>
</tr>
<tr>
<td>6</td>
<td>Industry</td>
<td>NO$_x$</td>
<td>96</td>
<td>300 (30, 610)</td>
<td>–66 (–33, –99)</td>
<td>250 (51, 450)</td>
<td>2.6 × 10$^{-3}$</td>
</tr>
<tr>
<td>7</td>
<td>Industry</td>
<td>VOC</td>
<td>300</td>
<td>310 (31, 610)</td>
<td>45 (22, 67)</td>
<td>360 (160, 570)</td>
<td>1.2 × 10$^{-3}$</td>
</tr>
<tr>
<td>8</td>
<td>Industry</td>
<td>Primary PM</td>
<td>110</td>
<td>12,000 (1,200, 24,000)</td>
<td>0</td>
<td>12,000 (1,200, 24,000)</td>
<td>1.1 × 10$^{-3}$</td>
</tr>
<tr>
<td>9</td>
<td>Domestic</td>
<td>NO$_x$</td>
<td>15</td>
<td>–21 (–2, –42)</td>
<td>–22 (–11, –33)</td>
<td>–44 (–59, –29)</td>
<td>–3.0 × 10$^{-3}$</td>
</tr>
</tbody>
</table>

All values are provided to two significant figures, and sums may not add due to rounding. A positive value in the last four columns means mortality reduction (or fewer deaths), and a negative value means mortality increase (or more deaths). Values in parentheses represent plausible upper and lower bounds for pollutant-specific mortality changes and 5th and 95th percentile values from a Monte Carlo simulation for net mortality changes. Scenario 6 is not shown here because it is included as a sensitivity test. Calculating pollutant-specific population exposures and mortality changes based on this scenario would require additional modeling scenarios (e.g., two additional scenarios similar to scenarios 3 and 4, but with a reduction percentage of 50%, respectively).
Consider—the population exposure reduction per unit emission reduction and the amount of emissions that can be reduced from different sectors. As expected and shown in prior studies in China (Zhou et al. 2003, 2006), the highest population exposure reduction per unit emission reduction for PM$_{2.5}$ is from controlling primary PM$_{2.5}$ emissions rather than particle precursors. Because of the relatively high primary PM$_{2.5}$ emissions in the industry sector and the feasibility of emissions reductions, the potential health benefits are substantial.

Considering particle precursors, SO$_2$ emission reductions yielded greater PM$_{2.5}$ exposure reductions than did NO$_x$ or VOC emissions reductions. In contrast, previous studies in China (Zhou et al. 2006) found similar iFs for sulfate from SO$_2$ and nitrate from NO$_x$, both on the order of 10$^{-4}$. In particular, the iFs for nitrate PM$_{2.5}$ formed from NO$_x$ emissions from power plants in Shanghai and the provinces of Jiangsu and Zhejiang ranged from 2 to 5 × 10$^{-4}$ versus 4 × 10$^{-4}$ in the present study. The differences are likely due to two factors: The modeling domain in the previous study covers all of China, and the previous study used an atmospheric model (CALPUFF) that did not capture the offsetting increase in sulfate when NO$_x$ emissions are reduced.

For ozone-related health benefits, our results show that VOC control is more effective than NO$_x$ control, due to the YRD area being hydrocarbon limited in ozone formation, where ozone concentrations increase with increasing hydrocarbons (e.g., VOC) and decrease with increasing NO$_x$ (Jacob 1999). One previous study (Wang et al. 2005) found that in the Pearl River Delta area of China, urban areas are VOC limited in ozone formation and the nonurban areas are NO$_x$ limited, where ozone concentrations increase with increasing NO$_x$ and are insensitive to hydrocarbons. Our study shows that the YRD as a whole (which contains many dense urban areas) is hydrocarbon limited, although it was beyond the scope of our study to explore the implications of source controls in different regions of the YRD or the potential long-range ozone formation that could occur from NO$_x$ controls. Despite the hydrocarbon-limited ozone formation in the YRD and the relatively low particle formation per unit NO$_x$ emissions, NO$_x$ emission reductions in power, industry, and traffic sectors are net beneficial, across the range of C-R functions simulated for ozone and PM$_{2.5}$.

**Limitations.** Several limitations could potentially influence the interpretation of our findings. First, in estimating the health benefits per ton of emission reductions, we implicitly assumed a linear relationship between pollutant emission reductions and population exposure reductions. There are many nonlinear processes in the atmosphere chemistry that could make this assumption faulty. However, our findings suggest that nonlinearity are limited given the emission changes in our study. For example, the emission reductions in scenario 6 were 2.5 times those of scenario 5 (e.g., 50% vs. 20% reductions of NO$_x$, VOC, and PM from the mobile sector), and the resulting total population exposures in scenario 6 were 2.53 times greater for PM$_{2.5}$ and 2.44 times greater for ozone, indicating reasonable linearity.

Second, because PM$_{2.5}$ is not currently a criteria air pollutant in China and there are no publicly available ozone monitoring data, the monitoring data we had access to for model validation are all from research monitoring sites located in Shanghai, which limited our ability to validate the model performance in other parts of the YRD modeling domain. Although further validation would have been ideal, various components of the model (e.g., the original emissions inventory input, the application of CMAQ in China) have been previously evaluated and validated, increasing our confidence in our findings. The lack of available monitoring data also emphasizes the need for publicly available comprehensive information systems in order to support health risk analyses and other environmental evaluations in China.

Third, although the C-R functions leveraged a combination of epidemiological evidence from the global literature and from China, our health benefit estimates are dominated by risks from long-term PM$_{2.5}$ exposure, for which there is no evidence within China. Moreover, the C-R function derived from the U.S. cohort studies would imply an extremely large mortality gradient across the YRD and between different areas of China, which is challenging to validate and interpret. That said, we did not consider it appropriate to omit cohort mortality entirely, given its biological plausibility, and no evidence exists to quantitatively deviate from the available cohort evidence. More generally, our conclusions about the relative importance of various source sectors are robust to this assumption. Our core findings therefore remain interpretable despite this large uncertainty, and we recommend that the C-R function for PM$_{2.5}$ mortality in China be reevaluated as more evidence from China becomes available.

Fourth, our analysis considers mortality only from PM$_{2.5}$ and ozone. Incorporation of other impacts, including morbidity outcomes or ecological damage from acid deposition, could be potentially influential if economic valuation of mortality were lower in China relative to economic valuation of morbidity than in the United States.

**Conclusion**

Despite the limitations, we demonstrated in this study a systematic approach to compare the effectiveness of pollutant control strategies across different sectors in a highly exposed and highly populated region of China. The use of the state-of-the-science air quality model CMAQ and a spatially resolved emission inventory allowed us to jointly consider PM$_{2.5}$ and ozone exposures for different emission reduction scenarios. Our findings indicate significant variation across pollutants in health benefits per ton of emission reduction. The public health benefits of realistic controls for NO$_x$ emissions from the power sector and primary PM$_{2.5}$ emissions from the industry sector are roughly comparable, given higher emission reductions for the former and higher population exposures per ton of the latter, with lower benefits from NO$_x$ control strategies. This is attributable in part to the hydrocarbon-limited nature of the YRD, as well as to the lower secondary PM$_{2.5}$ formation per ton of NO$_x$ emissions relative to other particle precursors. Our findings, in combination with plausible emissions reduction estimates and their costs, provide the basis for prioritizing pollution control strategies in the YRD and provide a template for comparable analyses elsewhere.

**References**

Ito K, De Leon SF, Lippmann M. 2005. Associations between...