Charging NO_x Emitters for Health Damages: An Exploratory Analysis

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This paper is dedicated to David Bradford, a colleague, mentor and friend. David enthusiastically brought his expertise as a tax economist to environmental economics. His keen desire to strengthen the interconnections between environmental science and economics led to his finding insightful, constructive and valuable ways to bring economic analysis to bear on air pollution and climate change projects. His untimely passing is a terrible loss for those who knew him as well as to everyone in the field who was looking forward to addressing today's pressing environmental problems in ways which are both scientifically rigorous and economically sound.

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ABSTRACT

We present a proof-of-concept analysis of the measurement of the health damage of ozone (O_3) produced from nitrogen oxides (NO_x = NO + NO_2) emitted by individual large point sources in the eastern United States. We use a regional atmospheric model of the eastern United States, the Comprehensive Air Quality Model with eXtensions (CAMx), to quantify the variable impact that a fixed quantity of NO_x emitted from individual sources can have on the downwind concentration of surface O_3, depending on temperature and local biogenic hydrocarbon emissions. We also examine the dependence of resulting ozone-related health damages on the size of the exposed population. The investigation is relevant to the increasingly widely used "cap and trade" approach to NO_x regulation, which presumes that shifts of emissions over time and space, holding the total fixed over the course of the summer O_3 season, will have minimal effect on the environmental outcome. By contrast, we show that a shift of a unit of NO_x emissions from one place or time to another could result in large changes in resulting health effects due to ozone formation and exposure. We indicate how the type of modeling carried out here might be used to attach externality-correcting prices to emissions. Charging emitters fees that are commensurate with the damage caused by their NO_x emissions would create an incentive for emitters to reduce emissions at times and in locations where they cause the largest damage.

Key words: surface ozone, NO_x emissions, point sources, health impacts, mortality, morbidity, cap-and-trade.

1. Introduction

Pollution in economic theory is essentially defined by externalities, negative impacts of one economic agent on others that are not reflected in market prices. An important input to corrective policy is the size of the externality, an estimation of the amount of damage done by the externality-causing action. In this paper we lay out a method of estimating the damage to human health due to exposure to ozone (O_3) formed as a result of emission of nitrogen oxides ($NO_x = NO + NO_2$) from individual large stationary sources in the eastern United States. We also present quantitative estimates for cases selected to draw attention to sources of variation in health damage that the present thrust of NO_x emissions regulation neglects. We apply the modifier "proof of concept" to the estimates to reflect the need for further refinement of the modeling components and for incorporating effects other than health damages resulting from O_3 -exposure. The longer term objective is to associate an estimate of external costs due to emissions from any point source as a function of the prevailing weather conditions, other emissions in the region, and populations, ecosystems and materials exposed. The initial calculations suggest the possibility of substantial gains (ie. lower total damages for a given quantity of emissions) from regulatory redesign.

NO_x contributes to a wide range of environmental effects including the formation of acid rain (via formation of nitric acid), PM2.5 (via formation of secondary particulates such as ammonium nitrate) with resulting health impacts, and contributions to regional haze, eutrophication of aquatic ecosystems (via addition of excess nitrogen), and elevated O_3 concentrations (via reaction with hydrocarbons and carbon monoxide) with resulting impacts on health and agriculture. Due to recent regulatory emphasis on controlling O_3 through NO_x "cap-and-trade" programs, we focus here on the contribution of NO_x emissions from point sources to ambient O_3 concentrations and the resultant damage to human health.

"Cap and trade" is a mechanism for regulating NO_x emissions, whereby a limit, or cap, is placed on total emissions from large point sources (typically during the "ozone season" from May to October each year). Regulated sources (mostly electric power generating plants) are obliged to have an allowance for each unit (ton) of NO_x emitted. In practice, allowances are

initially allocated to sources based on their historical operating levels. A source that does not own sufficient allowances for its needs can purchase them from others at a price agreed upon by the trading parties. The trading of allowances is expected to lower the total cost of whatever level of emissions control is embodied in the cap relative to a prescriptive type of regulatory control. A premise of such a system is that, because the total emissions are unaffected, such an exchange has no environmental significance. However, this premise may not be realized in any given situation and when it fails an exchange of allowances may have either positive or negative environmental effects. Our damage estimates allow us to isolate instances where a shift in NO_x emissions can have large effects on environmental damage.

To estimate the damages to human health resulting from NO_x emissions we use a model of the chemical and transport processes in the troposphere to calculate the increase in O_3 concentration, at each time and location, that results from a given incremental quantity of NO_x emitted from a power plant under different conditions. These O_3 concentration changes are combined with geographically specific demographic data to estimate the increase in mortality and respiratory illness that results from that increase in O_3. Specifically, we examine how temperature variations within a single summer month can have a significant effect on the quantity of O_3 produced from a unit of NO_x. We also examine how the same quantity of NO_x emissions from a power plant can result in very different O_3 production depending on the quantity of biogenic hydrocarbons emitted in the surrounding region. Finally, we show how the health damage that elevated O_3 concentrations cause increases as the size of the exposed population increases.

In the section relating these exercises to the design of policy, we draw attention to the standard economic argument for presenting firms with the "right" prices of the resources they use. In this case the resource used is NO_x emissions, for which, in the locations we study, the price charged is a combination of the price of emission allowances and the implicit price effect of applicable environmental regulations. The current regulatory regime diverges from one that would connect NO_x emission choices with their economic consequences in at least two respects. First, the present regulatory regime gives little systematic incentive to shift NO_x emissions during the summer from times and places where it has a large negative externality in the form of health impacts to times and places where the externality is smaller. Our analysis describes the

benefits of considering this variability. Second, the regulatory system is focused on avoiding exceedences of limits on ambient concentrations in order to comply with the National Ambient Air Quality Standards (NAAQS). The epidemiological studies on which we base our estimates of damages indicate that health impacts increase linearly with increasing concentration [*EPA*, 2003; *Steib et al.*, 2003]. A threshold limit on ambient O_3 concentration is a related but distinct objective to the avoidance of mortality and morbidity that a standard economic approach would imply because it is not concerned with damages which result from exposures to concentrations below the NAAQS.

In section 2 we provide background on the science of O_3 formation from NO_x emissions and on the regulatory history of NO_x and O_3. In section 3 we describe CAMx (Comprehensive Air Quality Model with Extensions), the regional air quality model we use to quantify the O_3 produced from NO_x emitted from point sources, evaluate its ability to reproduce observed O_3 concentrations, and describe the method we use to quantify morbidities and mortalities resulting from O_3 exposure. Section 4 uses CAMx to demonstrate the differences in O_3 production resulting from variability in local temperature and biogenic hydrocarbon emissions, as well as the importance of the location of high population densities relative to emission sites in determining resulting mortality and respiratory morbidity. These exercises demonstrate the potential environmental impact of an exchange of emissions from one time, place, or weather condition to another, expressed in terms of lives lost or sickness experienced. In the fifth section we estimate the monetary value of these environmental impacts, using valuation factors taken from the literature. Section 6 discusses the policy advantages of a damage based incentive system to control NO_x emissions from point sources. Section 7 summarizes our conclusions and highlights needed future research.

2. Scientific and Regulatory Background

2.1. Ozone Chemistry

O_3 is a pollutant that is formed in the lower atmosphere (troposphere) from a complex series of sunlight-driven reactions between NO_x, carbon monoxide (CO), volatile organic

compounds (VOCs) which are largely hydrocarbons, and methane (CH_4). The primary source of NO_x to the troposphere is fossil-fuel combustion. In 1998 in the United States, electric utilities accounted for 25% of total NO_x emissions, transportation for 53% and industrial sources for 12% [*EPA*, 2000]. VOCs are emitted from a range of human activities, including fossil-fuel combustion (excepting high-temperature combustion), direct evaporation of fuel and solvents, and chemical manufacturing. Terrestrial vegetation also provides a large natural source of hydrocarbons in summer with isoprene being the biogenic hydrocarbon emitted in the largest quantity. The emission of biogenic hydrocarbons varies greatly by location as indicated in Figure 4. Difficulty regulating O_3 occurs because in regions of high NO_x (primarily urban centers and power plant plumes), O_3 formation is limited by the availability of hydrocarbons. In regions of low NO_x (primarily rural areas with abundant emission of natural hydrocarbons), O_3 formation is limited by the availability of NO_x [*Sillman et al.*, 1990].

Ozone production from NO_x emitted in power plant plumes varies depending on the availability of hydrocarbons, the magnitude of the NO_x emission rate, and meteorological conditions. A power plant plume contains large quantities of NO_x, little CO and virtually no VOCs. Hence O_3 production from NO_x in a power plant plume is limited by the availability of VOCs in the surrounding environment as it dilutes. Data from aircraft transects of power plant plumes indicate that higher availability of biogenic hydrocarbons, principally isoprene, result in higher quantities of O_3 produced from the NO_x in the plume [*Ryerson et al.*, 2001]. In addition, as the size of the plume increases, the ozone production efficiency (the number of molecules of O_3 produced per molecule of NOx consumed [*Liu et al.*, 1987]) decreases. Therefore, large concentrated plumes can result in less net O_3 formation than small plumes per NO_x molecule emitted because of the more rapid conversion of NO_x in concentrated plumes to HNO_3 [*Ryerson et al.*, 2001]. Hence, in our later analysis all of our cases use large power plants which emit approximately the same quantity of NO_x.

In addition, O_3 production occurs more rapidly under conditions of stagnant high pressure which are frequently accompanied by high temperatures and sunny conditions [*Logan*, 1989; *Vukovich*, 1995]. As a result, the probability of exceeding the NAAQS for O_3 increases with daily maximum temperature [*Lin et al.*, 2001]. In our analysis temperature is used as a proxy for stagnant meteorological conditions.

2.2. NO_x and O_3 regulatory history

Legislative and regulatory efforts to control ambient O_3 concentrations started with the passage of the 1970 Clean Air Act (CAA). In 1971 EPA established a NAAQS for O_3 of a 1-hour maximum daily average concentration not to exceed 0.08ppm which in 1979 was revised to 0.12ppm [*EPA*, 2004a]. Based on scientific evidence of the adverse effect of O_3 on health, in 1997 EPA again revised the O_3 NAAQS and required that a 3-year average of the annual fourth highest daily 8-hour average concentration not exceed 0.08 ppm [*EPA*, 2004a; *NRC*, 2004]. Under the CAA, a state with monitoring stations that fail to meet the NAAQS is regarded as "out of attainment" and is obliged to specify in its State Implementation Plan (SIP) how it will attain the NAAQS in the future. No incentive presently exists to reduce O_3 further if a state is in compliance. O_3 has a lifetime of approximately 2 days in the boundary layer of the eastern United States in summer [*Fiore et al.*, 2002] and hence may be transported across state boundaries. Inter-state transport can make it difficult for states that fail to meet the NAAQS to become compliant by simply reducing in-state emissions.

Economists have been encouraging the use of market-based policies for controlling pollution as an alternative to more traditional command-and-control approaches (such as uniform emission standards or specific technology requirements) for some time. In the late 1960s, discussion of the concept of markets in pollution rights began in academic circles and entered the U.S. policy world in 1975 [*Atkinson and Tietenberg*, 1987]. Economic theory predicts that tradeable emission permits will induce emitters to find a cost-minimizing emission allocation when a cap on total emissions is specified [*NRC*, 2004]. Emitters that can reduce emissions easily do so and sell permits to those for whom reducing emissions is more costly.

In an attempt to control regional O_3 concentrations and to address inter-state transport of O_3 and its precursors to facilitate the compliance of down-wind states with the O_3 NAAQS, a coalition of 13 northeastern states formed the Ozone Transport Commission (OTC) and created a regional NO_x cap-and-trade program. The OTC "NO_x Budget program" ran from 1999-2002 during which time the total NO_x emissions from large point sources within the 13-state coalition were reduced approximately 50% from 1990 baseline levels [EPA, 2004b]. The period of NO_x

emission reduction from power plants coincided with a period of economic growth during which emissions from other sources (ex. vehicles) may have increased.

We compared surface NO concentrations from the EPA-AIRS measurement network between 10am and 2pm over the summer season pre- and post- the 1999 cap and do not find the expected decrease in NO concentrations. Nor does a similar comparison of O_3 concentrations over the summer season pre- and post- the 1999 cap show a decrease in O_3 concentrations resulting from a decrease in NO_x emissions from the power plants as a result of the cap (analysis not shown).

In 2004 the OTC program was replaced by the NO_x State Implementation Plan (SIP) Call budget trading program, developed in response to the EPA's call for SIPs to reduce the transport of O_3 over large regions of the country. Under the SIP Call program the trading region was expanded to 19 eastern states and total summer emissions from electric power generators are planned to be reduced by approximately 80% from 1990 levels [*EvoMarkets*, 2003].

The theoretical advantages of an emissions cap and trade system depend on the assumption that only the total amount emitted matters. Under this condition, a shift of emissions from one time or place to another will have no environmental consequences and minimizing the cost of abatement is the object of the plan. Recognizing the general problem, the "NO_x Budget program" incorporated "flow control" to require that allowances banked from one summer season for use in subsequent summers be surrendered on a 2:1 basis if the bank became larger than 10 percent of the regional budget. The flow control rule inhibits excessively large emissions in any single summer but it is not clear whether the net environmental result (measured by health and mortality impacts) is likely to be positive. Our analysis suggests the importance of designing rules to take into account the environmental effects of variations in the time or location of emissions in a single summer season, let alone between seasons.

3. Methods

3.1. CAMx model description

We utilize the Comprehensive Air Quality Model with extensions (CAMx) version 3.0, a regional Eulerian photochemical grid model [*ENVIRON*, 2000]. CAMx simulates the emission, dispersion, chemical and photochemical reactions and removal of tropospheric ozone and its precursors in the lower troposphere by solving the continuity equation for each chemical species in a three-dimensional grid. The Eulerian continuity equation relates the time dependency of the average species concentration in each grid cell volume to the sum of all physical and chemical processes operating on that volume.

We use the CAMx model in a configuration previously used by the Ozone Transport Assessment Group (OTAG) to solve for the concentration distribution of ozone and its primary precursors. Model resolution is 0.33° latitude by 0.5° longitude extending in 5 vertical layers (100, 500, 1500, 2500, and 4000 meters above ground level) from the surface to 4 km. CAMx includes a plume-in-grid sub-model to simulate the chemistry of NO_x plumes emitted from point sources such as power plants. The plume-in-grid sub-model tracks individual plume segments, accounting for dispersion and inorganic chemical evolution, until the puff mass can be adequately resolved by the grid box in which it resides at which time the remaining NO_x is released to the grid. This CAMx configuration uses the Carbon Bond IV (CB-IV) chemical mechanism with updated radical termination and isoprene oxidation mechanisms based on [*Carter*, 1996] including 25 gas species and 96 reactions. Winds are generated by the Regional Atmospheric Modeling System (RAMS) with horizontal advection provided by the diffusive-corrected algorithm of [*Smolarkiewicz*, 1983]. Boundary conditions are set to clean background concentrations for all constituents on all four sides and at the top of the domain (0_3 = 35ppbv) with a day/night diurnal cycle imposed on short-lived species. Hourly surface emissions are included from elevated point sources, gridded low-level point, mobile, biogenic (from the BEIS2 model) and anthropogenic area emissions in the eastern United States. The BEIS2 model includes the dependence of isoprene emissions on temperature, solar radiation, ecosystem extent and leaf area [*Geron et al.*, 1994; *Guenther et al.*, 1994; *Pierce et al.*, 1998]. However, [*Palmer*

et al., 2003] inferred from GOME satellite measurements of formaldehyde that BEIS2 under-predicts isoprene emissions by a factor of two. CAMx also includes wet and dry deposition [*Wesley*, 1989] of gases. Emissions and meteorological fields for the model were obtained from the New York State Department of Environmental Conservation (NYSDEC) [Sistla and Hao, personal communication, 2001]. Model simulations were conducted for July 7-17, 1995, with the first two days of the simulation used for model spin-up.

3.2. CAMx model / EPA-AIRS O_3 data comparison

To evaluate the ability of this version of CAMx to accurately simulate ambient O_3 concentrations within the region, we compared concentrations of O_3 measured as part of the EPA-AIRS surface measurement network with O_3 concentrations obtained from the CAMx simulation. Locations of the EPA-AIRS O_3 measurement sites for July 1995 are shown in Figure 1. Figure 2 shows a comparison of 1-hour daily maximum O_3 concentrations calculated by the CAMx model for July 9-17, 1995 and measured on the same dates by the EPA-AIRS network. A comparison of 1-hour maximum concentrations is shown because the epidemiological dose-response functions used later in this paper to estimate both mortality and morbidity use this value.

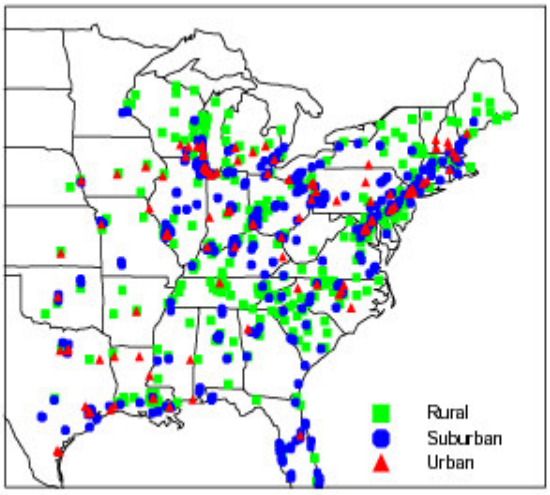

Figure 1. EPA-AIRS O_3 measurement site locations within the CAMx domain, July 1995.

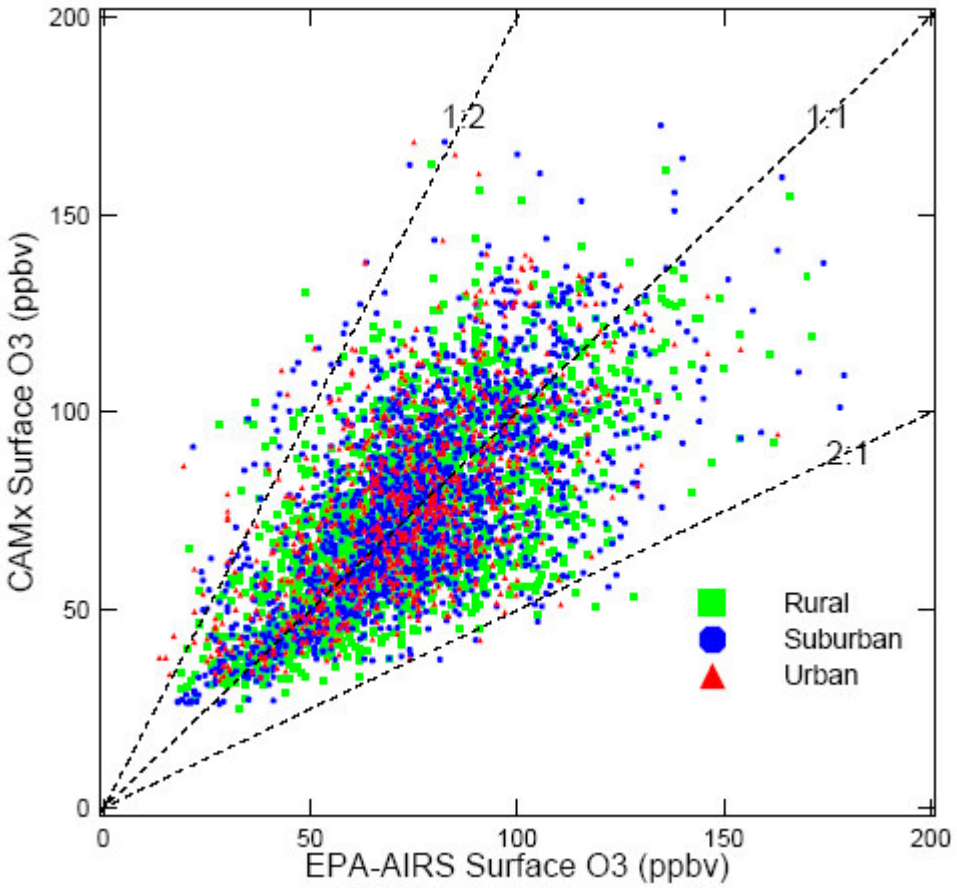

Figure 2. Comparison of 1-hr. daily maximum O_3 surface concentrations for July 9-17, 1995 from the EPA-AIRS measurement network and CAMx simulation. Rural, suburban and rural labels refer to the EPA-AIRS designation for individual measurement sites. O_3 concentrations simulated in a surface CAMx grid box are compared with the measured O_3 concentrations at the EPA-AIRS network stations that fall inside the grid box. Multiple stations within one category (urban, suburban or rural) that fall within one grid box are averaged together for the comparison.

Statistical analysis of the results in Figure 2, with P_i indicating the predicted CAMx O_3 values and O_i indicating the observed EPA-AIRS O_3 concentrations, result in the following summary statistics:

$$\text{Root mean square error (RMSE)} = \left[\frac{1}{N}\sum_{i=1}^{N}(P_i - O_i)^2\right]^{\frac{1}{2}} = 19.6 \text{ ppb};$$

Mean Bias (MB) = $\frac{1}{N}\sum_{i=1}^{N}(P_i - O_i)$ = 0.62 ppb;

Mean Normalized Bias (MNB) = $\frac{1}{N}\sum_{i=1}^{N}\frac{(P_i - O_i)}{O_i}$ = 6.0 %

This evaluation indicates that although there is random error in the maximum daily O_3 concentrations calculated by CAMx the model is without significant bias.

We use the model to predict the change in O_3 concentration (measured by the 1-hour daily maximum) over time over the whole model domain that would result from a specified increase in NO_x emissions over a specified time at a given power plant location. A simple regression of O_i on P_i in the data in Figure 1, yields the slope and intercept estimates of 0.646 (56.43) and 26.0 (28.54), where the figures in parentheses are the t statistics for the difference from zero (adjusted R-square is 0.42). An apparent implication of the regression is that a unit difference in the O_3 prediction of the model corresponds on average to a 0.646 difference in measured O_3 concentration between two time or location points. The slope coefficient (change in measured per unit change in model-generated ozone concentration) in these regression analyses are of central interest to us. As we explain, we focus on the difference between the O_3 concentrations generated by CAMx for all receptor locations in a standard run (which uses the input data underlying Figure 1) and in a set of alternative runs (which perturb the input emissions at strategically chosen times and places). The underlying assumption is that the differences in model-generated O_3 concentrations are good predictors of the differences in measured concentrations that would have been experienced, had the real world input data been different in the prescribed manner. The regression analysis of the use of model results in predicting measured outcomes would suggest, instead, taking as the basis for predicting the impacts the model differences multiplied by the slope coefficient (.646). The relationship, however, is likely to vary across the domain because of variation in physical and chemical conditions between the source and receptors. Because we have found in discussions with colleagues that there is no consensus on the validity of such an adjustment, and because, in any case, it is just one of many refinements to what we emphasize is a proof-of-concept study, for the remainder of this paper we do not make any adjustment of the model results.

3.3. Model Simulations

The two goals of the CAMx simulations presented in this paper are first to demonstrate the spatial and temporal heterogeneity of tropospheric ozone production and the variability in resulting health damages (morbidity and mortality) that may result from a given amount of NO_x emitted from the same location under different meteorological conditions or from different locations with varying biogenic hydrocarbon emissions or downwind populations. The second objective is to provide a proof-of-concept prototype of the feasibility of conducting a full integrated assessment that links NO_x emissions to O_3 concentrations to human exposure, to morbidity, mortality and damage valuations.

To demonstrate the spatial and temporal heterogeneity of tropospheric ozone production, we conduct a set of CAMx regional simulations for seven cases. First, we conduct a standard simulation that includes all regional emissions. This is followed by six perturbation simulations. In order to quantify the impact of NO_x emissions from individual power plants on O_3 concentrations, we reduce the NO_x emissions from each plant by 62.5 tons (125,000 lbs) in a 24-hour period (1.77×10^6 moles where NO_x emissions from power plants were assumed to be 90% NO and 10% NO_2) and calculate the difference in maximum O_3 concentration between the standard simulation and each perturbation simulation. The power plants used for this experiment are among the largest 5% of power plants in the United States; each emits approximately 100 tons of NO_x per day. 62.5 tons of NO_x was chosen for the analyses because it is sufficiently large to generate a discernable effect and sufficiently small that a single large power plant may emit this quantity or more in a 24-hour period. The perturbation simulations were chosen to demonstrate the variability in O_3 production resulting from identical NO_x emissions from power plants produced during conditions of high and low temperature at the same location and in locations of high and low biogenic hydrocarbon emissions. They were also chosen to demonstrate the dependence of total health effects on the population density in the regions downwind of the emissions. Figures 3, 5 and 7 show the increase in maximum surface O_3 concentrations over the model domain between the standard simulation and perturbation simulations summed over the day of emission and the subsequent 3 days. After 4 days the power plant emissions from the 24-hour period have a non-discernable effect on regional O_3

concentrations. The total increase in 1-hour maximum O_3 is shown because it is incorporated in the dose-response functions used to estimate the mortality and morbidity that result from exposure to O_3.

3.4. Estimating Mortality and Morbidity

The relationship between O_3 exposure and resulting health effects can be estimated from epidemiological studies. Most epidemiological research has made use of time-series analyses that have focused on the effects of short-term exposures to a pollutant by using records of hospital admissions and deaths that occur following periods of elevated concentrations. These studies are unable to include the negative effects of chronic or long-term exposure and hence presumably underestimate the total health damage due to exposure. Cohort studies, which follow a population over time and do include both acute and chronic impacts, have been conducted for PM2.5 [Dockery et al., 1993; Pope et al., 2002; Pope et al., 1995] but have not focused on O_3. Hence, in relying in this analysis on time-series studies only (specifically, on meta-analyses that pool the results of many regional time-series studies [*Stieb* et al., 2003]) we use an arguably conservative estimate of the mortality and respiratory morbidity that results from elevated O_3 exposure.

The change in the incidence of mortality or morbidity due to exposure to air pollution (here O_3) is estimated with the following function [*EPA*, 1999]:

$$\Delta M = Y_o\left(1 - e^{-\beta \Delta O_3}\right) * population \qquad (1)$$

ΔM is the change in the number of mortalities during a 24-hour period, or change in incidence of hospital admissions for respiratory disease, relative to a baseline number of deaths or hospital admissions for respiratory disease. For mortality, Y_0 is the baseline incidence of daily non-accidental deaths per person of any age. For morbidity, Y_0 is the daily average hospital admission rate for diseases of the respiratory system. Epidemiological studies often report a relative risk (RR) (the percentage increase in mortality or morbidity) due to a specific increase in pollutant concentration. The concentration-response coefficient, β, can be derived from the reported relative risk and the change in pollutant concentration relative to a baseline (here ΔO_3), by solving:

$$\beta = \frac{\ln(RR)}{\Delta O_3} \qquad (2)$$

We discuss the β values we use for mortality and morbidity in the following sub-sections. We obtain the population in equation (1) from 1995 world population data gridded at 0.04167 latitude by longitude resolution [*CIESIN et al.*, 2000]. Population data for the eastern United States is regridded at the CAMx resolution of 0.3 degrees latitude by 0.5 degrees longitude (approximately $(36\ km)^2$ in the United States). The population within each CAMx grid box is used to calculate the mortality and morbidity that result from the change in O_3 concentrations in each grid box. Mortality and morbidity incidence is summed over all affected grid boxes over all days in which the O_3 concentration was enhanced due to (or, in some locations, reduced by) the NO_x emitted from the individual power plants.

3.4.1. O_3 Mortality

To obtain the relative risk of mortality for an increase in O_3 concentration resulting from individual power plant emissions, we use a pooled estimate of percent excess mortality derived from a compilation of 25 epidemiological time-series analyses of daily 1-hour maximum O_3 concentrations during the warm season [*Stieb et al.*, 2003]. The pooled estimate of RR for single pollutant studies is 1.014 for a 31.2 ppb increase in 1 hour maximum daily O_3 concentration, which translates into a β value of 0.000446 [*Stieb et al.*, 2003]. We estimate Y_0 from the total U.S. population in 1995 and calculate an average daily mortality rate of 2.4×10^{-5} (2,312,132 deaths/ 261,638,000 persons / 365 days) [*CDC-NCHS*, 2003; *USCB*, 2000].

3.4.2. O_3 Morbidity

Hospital admissions for respiratory ailments have been found to be substantially and consistently associated with O_3 exposure. A synthesis of O_3 hospital admission time-series studies, compiled by *Thurston and Ito* [1999] is used here to estimate the effect of O_3 on morbidity. Data on hospital admissions for respiratory disease in the United States were not available. Therefore, to calculate Y_0 we assume that the average daily rate of hospital admissions for respiratory disease is approximately equal to the rate of hospital discharge following a

diagnosis of a respiratory ailment which was 3.41 x 10^{-5} discharges per day in 1998 (124.6 discharges / 10,000 population / 365 days), the year for which such data are available [*CDC-NCHS*, 1998]. This value is of similar magnitude to values obtained in regional studies of daily hospital admission for respiratory diseases [*EPA*, 1999]. The estimate for hospital admissions for major respiratory ailments due to O_3 exposure for all ages was a RR = 1.18 per 100 ppb increase in daily 1-hour maximum O_3 (95% CI, 1.10-1.26) [*Thurston and Ito*, 1999] which translates into a β value of 0.00166 . In cases where studies in the meta-analysis used other measures of O_3 exposure besides a daily 1-hour maximum concentration (such as an 8-hour average O_3 concentration) the ratio of the daily 1-hour maximum to the other measures used in the original analyses were used to compute the increment comparable to a 100 ppb change in daily 1-hour maximum O_3 concentrations [*Thurston and Ito*, 1999].

4. Results

Our results demonstrating the impacts of variability in temperature and biogenic hydrocarbon emissions on O_3 production, and the importance of population density in determining resulting mortalities and respiratory morbidity are summarized below. These factors were chosen for examination because of the expected size of their influence and because of the ability of our model to simulate them. Additional factors may also influence O_3 production from large point sources such as the concentration of NO_x in the plume, cloudiness, time of day of emission, proximity of the NO_x emission to other anthropogenic emissions, and other factors. Analysis of these additional factors is left for the future.

4.1. Temperature variability -- Effect on O_3 production and resulting mortality and morbidity

First, to examine the temporal heterogeneity of tropospheric ozone production due to temperature variability within a short period of time, a point source was selected which experienced a range of temperatures between July 9 and 17, 1995. The effects on O_3 production of identical emissions of NO_x from a power plant in eastern Pennsylvania on July 9, 1995 and

July 14, 1995 when the 24-hour period temperature ranged over 72 ± 7.7°F and 83.5 ± 6.3°F, respectively are shown in Figures 3a and 3b. The average increase in 1-hour maximum surface O_3 over the model domain between the standard simulation and perturbation simulations on the day of emission and the subsequent 3 days, was 0.021 ppbv during the cool period and 0.036 ppbv during the warm period. The model predicts that the same amount of NO_x emitted in the warmer period results in more O_3 which leads to twice the mortalities as in the cooler period -- 0.185 versus 0.354 persons as shown in Figures 3c and 3d. Likewise, the number of cases of respiratory morbidity due to O_3 exposure was also nearly twice as high during the warm period -- 0.866 versus 1.654 cases as is shown in Figures 3e and 3f. Due to the use of the concentration response function for O_3, calculated mortalities and morbidities isolate the effect of increased O_3 and do not include possible direct effects of increased temperature on mortality and morbidity.

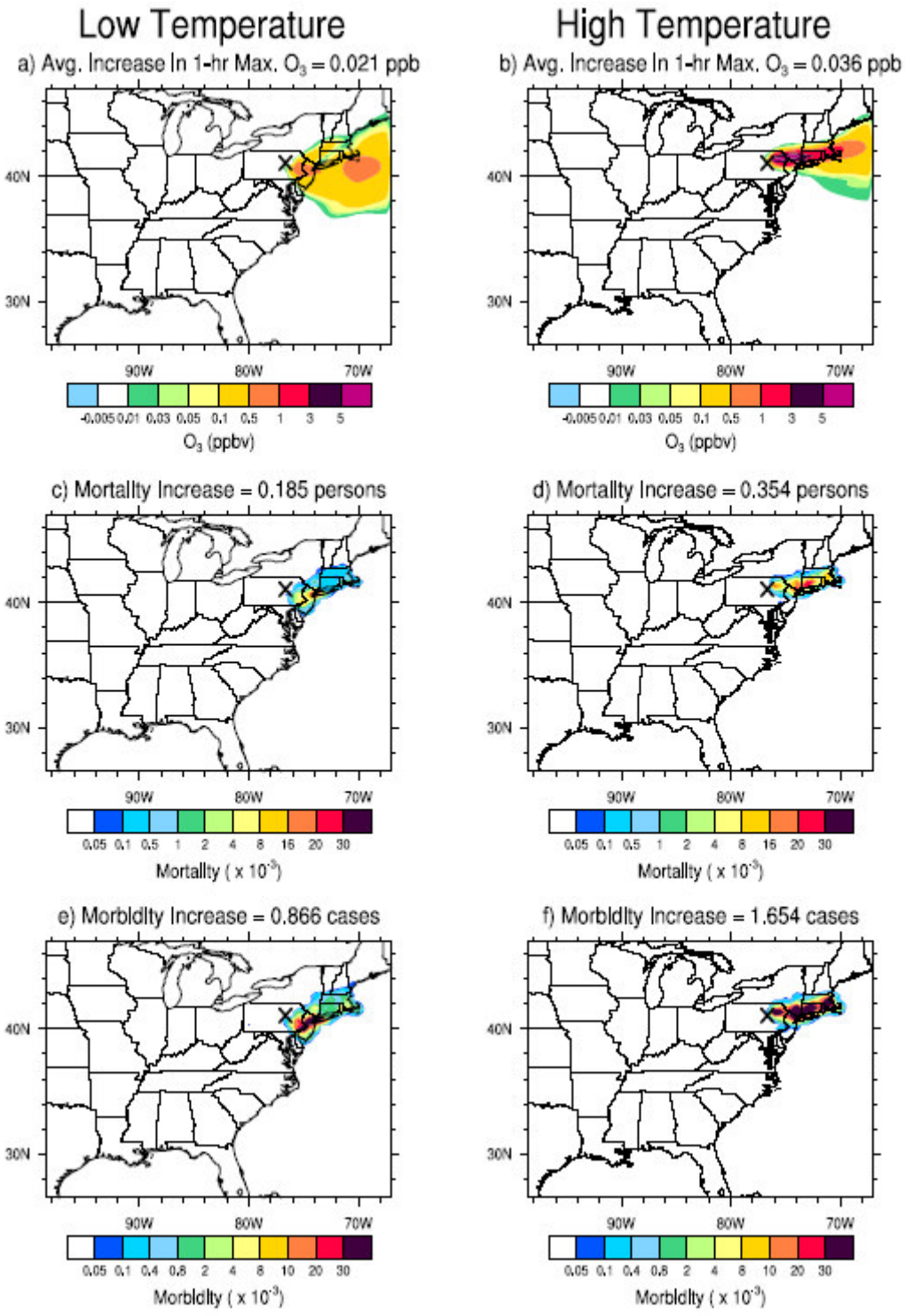

Figure 3. O_3 produced by an identical amount of NO_x emitted from the same power plant in eastern Pennsylvania in a 24-hour period on a) 9 July 1995, a cool day (296°K ± 4.3°K; 72° ± 7.7°F) and b) 14 July 1995, a warm day (302°K ± 3.5°K; 83.5 ° ± 6.3 ° F) and the

subsequent 3 days until O_3 production from these NO_x emissions cease. The "X" in each figure marks the power plant location. The 'average increase in 1-hour maximum O_3' is the total increase in maximum surface O_3 concentrations over the model domain between the standard simulation and perturbation simulations on the day of emission and the subsequent 3 days. Beyond 3 days the power plant emissions cease to have a significant influence. Here figures c) and d) show the mortalities and e) and f) show the morbidities that are estimated to result from the increase in daily maximum O_3 occurring in a) and b), respectively.

These simulations show that, in violation of the preconditions for the use of a cap and trade system, a shift of a fixed amount of NO_x emissions from a single source between two closely separated time points could have a substantial effect on the quantities of O_3 that are produced, and the consequent health effects. In fact, since energy demand is usually larger when temperatures are high, emissions are also likely to be larger during periods of high temperature when the model indicates that more O_3 is produced per unit NO_x emitted. An emitter using emission allowances at two different times presently has no reason to favor the time when the resulting damage is lower. Trading allowances (or variability in the timing of the use of allowances from a single source) to emit at a price that does not reflect the difference in damage will lead to too much emission in the high-damage conditions relative to the low-damage conditions. In the example, the temperature at the emission source and at downwind locations makes an important difference. The question is naturally raised, whether an alternative regulatory system might be developed that provides an appropriate incentive for emitters to reduce their emissions during times of elevated temperature within the ozone season.

4.2. Biogenic hydrocarbon emission variability -- Effect on O_3 production and resulting mortality and morbidity

Isoprene is a hydrocarbon that is naturally emitted from deciduous tree species, particularly oak, poplar and gum trees. In the United States these species are most abundant in the south-eastern part of the country and are absent in most of the west. This results in large spatial variability in biogenic VOC emissions in the United States, as is shown in Figure 4. In addition, isoprene emissions increase with increasing temperature and sunlight. Biogenic

hydrocarbon emissions reach a maximum at approximately 40°C and decrease following a bell shaped curve on either side of 40°C [*Pierce et al.*, 1998]. A unit of NO_x emitted from a power plant in a region of high isoprene emissions can result in larger O_3 production than the same amount emitted in a region of low isoprene emissions [*Ryerson et al.*, 2001]. To examine the impact that biogenic hydrocarbon emissions, particularly isoprene, can have on O_3 production, we examined the quantity of O_3 produced from identical emissions of NO_x by two different power plants, one in the south (Mississippi) and the other in the northern mid-west (Indiana) of the U.S. To control for the effect of temperature on isoprene emissions and on O_3 production, we chose 24-hour periods during which the mean temperature at the two sites was nearly identical: 87.2° F ± 5.9° F in the high isoprene region and 87.9° F ± 6.3° F in the low isoprene region. The regional average increase in 1-hour maximum O_3, as shown in Figure 5a and 5b, was 0.033 ppbv in the region with low emissions and over three times higher at 0.109 ppbv in the region of high emissions. Due to the apparent factor of two under-prediction of isoprene emissions by BEIS2 [*Palmer et al.*, 2003], the quantity of O_3 produced in our simulation in the high-isoprene region may actually be under-predicted.

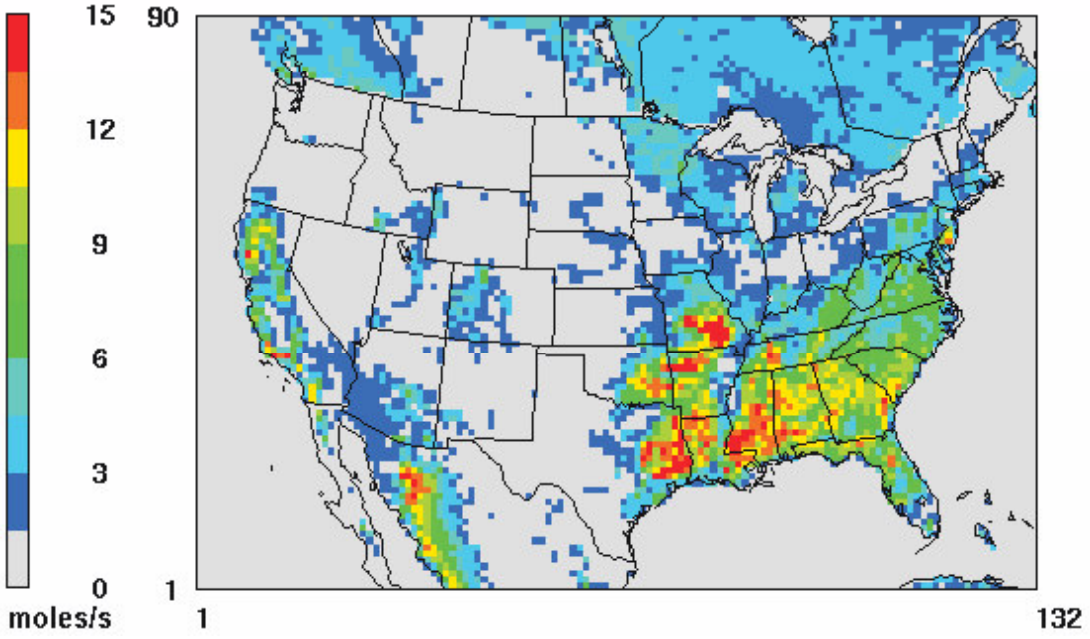

Figure 4. July emissions of isoprene in the United States (monthly mean of 24-hour daily averages). This figure was created from a simulation using the Community Multi-scale Air Quality Modeling System (CMAQ) [*Byun and Ching*, 1999].

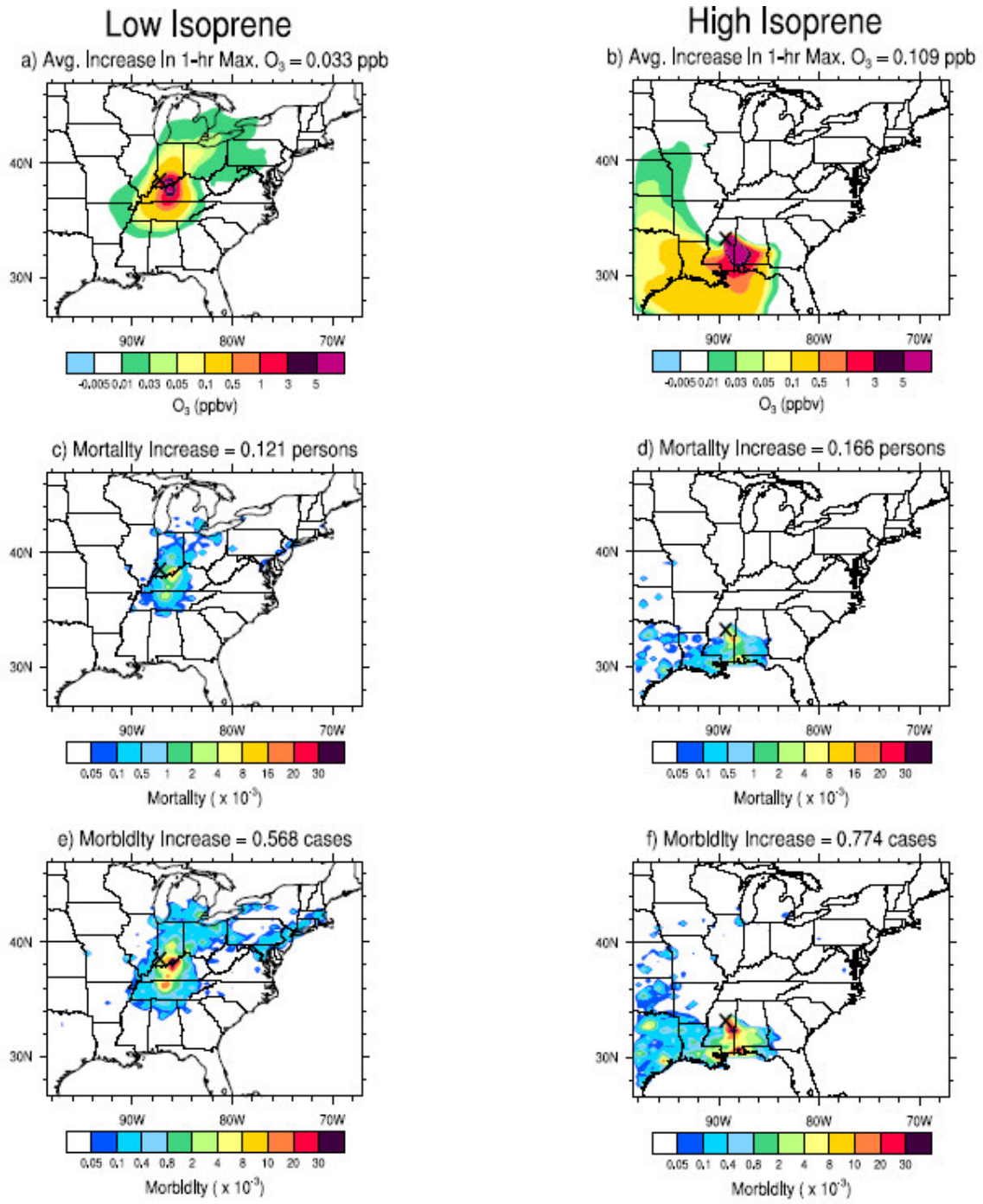

Figure 5. O_3 produced by an identical amount of NO_x emitted on 10 July 1995 from a power plant in a) a region of relatively low isoprene emissions and b) a region of relatively high isoprene emissions. The temperatures at both locations were nearly identical (87.9° F ± 6.3° F and 87.2° F ± 5.9° F in the low and high isoprene regions respectively). The rest of the notation is as described in Figure 3.

In this example, the region of higher isoprene emissions was also a region of lower population density (see Figure 6). Despite this fact, the 3.5 times larger quantity of O_3 produced in the region of higher isoprene emissions resulted in both 30% more mortalities (0.166 versus 0.121 deaths) and morbidities (0.774 versus 0.568 cases) than resulted from the same increase in NO_x emissions in the region of lower isoprene emissions (see Figures 5c-f). By choosing locations where the region of high isoprene corresponds to a region of lower population, this scenario is intentionally conservative and is hence understating the potential importance of variability of isoprene concentration on resulting O_3 mortalities.

A regulatory scheme that ignores the spatial variability of biogenic hydrocarbon emissions when regulating NO_x may miss an opportunity to decrease O_3 concentrations and could inadvertently increase mortalities and morbidities by trading from a region of relatively low isoprene emissions (north-east) to high emissions (south-east). In particular, trades between regions under a cap-and-trade program in which all NO_x emissions from large point sources in a region are treated identically, could have a substantial effect (up or down, depending on the direction of the exchange) on the quantities of O_3 that are produced, and the consequent health effects. A regulatory system that provides incentives for emitters to reduce their emissions in regions of high isoprene emissions would be more effective at reducing ambient O_3 concentrations [*Ryerson et al.*, 2001]. If these reductions coincided with high population densities the reductions would also be more effective at reducing adverse effects on exposed populations than would a regulatory program that assumes emissions in all locations have identical effects. We discuss the effect of downwind population density next.

4.3. Downwind Population Density -- Effect on resulting mortality and morbidity

Population density in the eastern United States is shown in Figure 6. The north-eastern Washington D.C. to Boston corridor has among the highest population densities in the United States. To examine the impact that equivalent reductions of NO_x would have from point sources upwind of regions of high and low population density, point sources in the high-population density northeast (Maryland) and lower-population density mid-Atlantic region (North Carolina)

were selected. Average isoprene emissions are substantially larger in North Carolina than in Maryland, however. Thus, despite controlling for temperature, the average increase in 1-hour maximum O_3, as shown in Figures 7a and 7b, was more than twice as great (0.081 ppbv versus 0.035 ppbv) when emissions originated from lowly populated North Carolina (Figure 7a) than from highly populated Maryland (Figure 7b). Despite higher maximum O_3 concentrations in the low population region, the resulting incidence of mortalities shown in Figures 7b and 7d (0.592 versus 0.268 deaths) and respiratory morbidity shown in Figure 7c and 7e (2.771 versus 1.252 cases), is much lower. Thus, for the same NO_x emission and a smaller O_3 increase, the damage (i.e., higher mortality and morbidity) was substantially greater in the high population region.

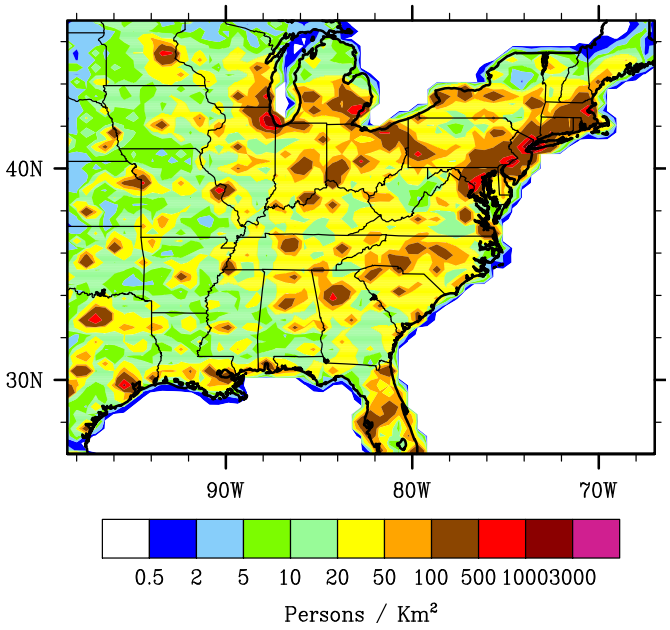

Figure 6. Population density in the eastern United States [*CIESIN et al.*, 2000].

Regulation of NO_x emissions that ignores the variability of downwind population density misses an opportunity to decrease the health effects of emissions. As in the case of variations in biogenic hydrocarbon emissions from place to place, trades between regions under a cap-and-trade program in which all NO_x emissions from large point sources in a region are treated identically could have a substantial effect (up or down, depending on the direction of the exchange) on health outcomes. A regulatory system that provides incentives for emitters to reduce their emissions in regions of high downwind population density would be more effective

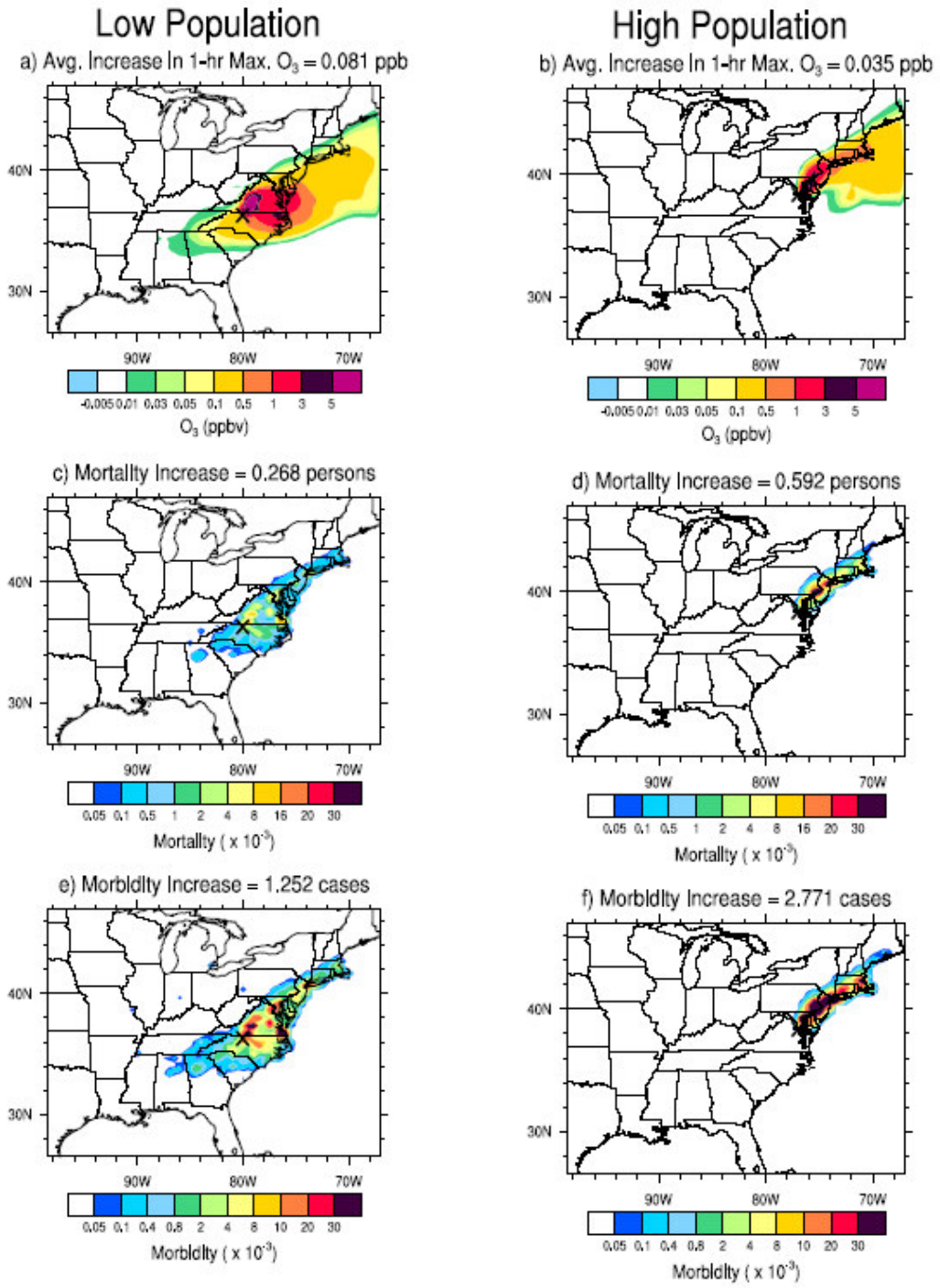

Figure 7. O_3 produced by an identical amount of NO_x emitted on 10 July 1995 from a power plant a) upwind from a relatively low population region and b) upwind of a high population region. The mean temperature at the two sites was nearly identical: 83.9°F ± 6.3°F in the high population region and 83.4°F ± 5.8°F in the low population region. The rest of the notation is as described in Figure 3.

at reducing the adverse effects on exposed populations than would a regulatory program that assumes emissions in all locations will have identical effects. Variations in daily wind direction can determine whether a particular NO_x source is upwind or downwind from a large population center.

Ideally, a chemical weather forecasting system integrated with a health damage model could be used to predict a few days in advance what the likely damage that NO_x emitted today would cause. Emitters could then take that information into account when making decisions as to whether to control their emissions, import power from elsewhere, or purchase or sell permits to cover the damage that their emissions were projected to cause.

5. Monetizing health costs resulting from O_3 produced from point source NO_x emissions

In the standard economic analysis of externalities, the problem that externalities cause is an inefficient allocation of resources. Much as a producer of electricity would be expected to use "too much" coal if it were provided free of charge (and as a result, in a competitive system, the price of electricity would be "too low"), a producer of electricity who is charged nothing for NO_x emissions may be expected to emit "too much" NO_x. A standard corrective in the economic theory of pollution is to determine the "right" price for the unpriced input (here NO_x emissions), and to charge the firms that use NO_x emissions in production via a fee or tax. The argument is that the producing firm "should" pay the price for these inputs, just as the firm pays for other inputs, not as a moral matter but as a condition for avoiding a use of resources that result in a loss of potential value to the ultimate consumers of goods and services and to the general public.

Our analysis indicates potentially large differences in the damage done by incremental NO_x emissions from sources at different locations and under different regional VOC and meteorological conditions, where the impacts have been expressed in terms of change in mortality and morbidity risks. The logic of correcting a negative externality calls for expressing these effects in monetary terms, so that they can be compared with the cost of corrective actions. In the case of health impacts, the needed valuation is the willingness of people to pay for reduced risks of suffering them.

A large literature addresses the estimation of the monetary value of health effects. A deeper analysis than the one we undertake here would consider issues such as whether the willingness to pay for reduced risk of health effects depends on the age of the affected people, their incomes, number of dependents, etc. For purposes of the present analysis, we take off-the-shelf figures for the value of a statistical life or an illness. Mortality valuation estimates, obtained from 26 different studies that use both contingent valuation and labor market valuation methods to approximate the willingness to pay to avoid the occurrence of a premature death, give values of a statistical life that range from $0.6 million - $13.5 million with a mean of $4.8 million in 1990 U.S. dollars [*EPA*, 1999; *Viscusi*, 1992]. The model simulations represent O_3 production during July 1995, so in order to calculate the value of the damages caused by the NO_x emissions in our 1995 simulations in contemporary terms, we use the 17% increase in the consumer price index (CPI) between 1990 and 1995 to adjust the mean value of a death to 1995$5.6 million [*DoL*, 2004]. Due to the increase in CPI between 1995 and 2004 these same deaths, had they occurred in 2004, would have a valuation 24 % higher than the 1995 value [*DoL*, 2004].

For the specified identical quantities of NO_x emitted from individual power plants over 24-hour periods, mortalities resulting from the O_3 produced over the subsequent several days ranged from a minimum of approximately 0.1 deaths in the low isoprene region to 0.6 deaths when emitted upwind from a region of high population density. Using the mean inflation-adjusted value of 1995$5.6 million/life, the monetary cost of the deaths in our six cases varies between a low of $670,000 to a high of $3.3 million. The NO_x emission change (62.5 tons) that gives rise to these impacts is comparable to shutting down a large power plant for a day. For an emission of this magnitude, the estimated value of the impact per incremental ton emitted ranges in the six cases from $10,700 to $52,800.

The literature on the cost of morbidity due to an illness indicates substantial variation depending on the illness. Chronic bronchitis is usually estimated to have the highest cost and a restricted activity day among the lowest. We utilize a morbidity dose-response function to estimate the relative risk of a hospital admission due to an increase in daily 1-hour maximum O_3 concentrations. We therefore use a health valuation for all respiratory hospital admissions of 1990$6100/case [*EPA*, 1997] which adjusted for inflation is 1995$7020/case. Thus, for the

identical quantities of NO_x used in our exercises, emitted from individual power plants over 24-hour periods, the calculated morbidities range from 0.57 cases for the low isoprene region to 2.8 cases for the high population region. The implied estimated costs of total respiratory effects range from 1995$4000 to 1995$19,700 or from $64. to $315. per ton NO_x emitted. Given valuations found in the literature, the costs of the morbidity impacts of NO_x emissions are tiny when compared with the costs of the mortality risk effects.

6. Policy Implications

The implications of our investigation for policy design are potentially wide-ranging. Focusing only on the mortality effects, which our calculations suggest are of greater policy significance, the estimated impact of an incremental ton of emitted ranges from 0.0019 to 0.0095 fatalities (a factor of about 5) depending on ambient temperature and emission location. By shifting 11 tons of NO_x emissions per day from a (relatively) high-damage to lower-damage location over a ten-day period one could avoid the loss of approximately one life (on average).

The figures also suggest a significant influence of meteorological conditions on the health impact of incremental NO_x emissions. The mortality impact of emissions from a single location may vary by a factor of nearly two as the temperature varies within a short span of time. In our simulations we calculated 0.00566 and 0.00296 deaths per incremental ton of NO_x emitted for the high and low temperature scenarios, respectively. Reducing O_3 generation and thus saving human lives might be brought about, for example, by reallocating electricity production in space to reduce NO_x emissions under high-temperature stagnant weather conditions and by utilizing pollution control devices more intensely in locations of high population exposure. The use of a chemical weather forecast system would assist in predicting damages given current weather conditions.

Placing monetary value on the damages that result from incremental NO_x emissions (taking into account, as we do here, only the contribution to downwind O_3 formation and neglecting the health damages from secondary PM formation) suggests that the magnitudes are significant in relation to the costs of abating the emissions. Still considering only fatality effects, and using mean off-the-shelf estimates of the value of a statistical life saved, the estimated

damage per incremental ton of emissions in our scenarios ranges from 1995$10,700 to 1995$52,800. To get a sense of what these figures mean, we compare them to the price of NO_x emission allowances under the SIP Call cap and trade system. One allowance permits an affected source to emit one ton of NO_x between May 1 and September 30 for a given year. As reported at http://www.evomarkets.com/ on 22 July 2004, the last price for NO_x emission allowances for 2004 was $2,550, for 2005, $3,750, and for 2006, $3,200. Using a CPI adjustment of 0.8 [*DoL*, 2004], these prices are equivalent to 1995$2040., 1995$3000., 1995$2560., respectively. These figures can be read as measures of the cost of incremental NO_x abatement during the ozone season (*given* the existing regulations governing power plants, which have complex implications for the marginal cost of abatement). Economic theory predicts that the marginal cost of abatement of NO_x will be driven to the market price of allowances, with the important proviso that the marginal cost may exceed the allowance price for all levels of abatement. Although the allowance prices cannot be directly compared with our estimate of the marginal damage due to NO_x emissions (since both the year and the underlying emissions picture is different), the figures certainly suggest the possibility that net gains are possible from more stringent, and as significantly, more effective, NO_x control.

The textbook remedy for a defect in the allocation of resources that results in an environmental externality is a corrective tax or fee (usually called a Pigovian tax after the British economist A.C. Pigou, who first elaborated the idea) on the action that gives rise to the externality. The damage done by a marginal unit of NO_x emissions under a variety of conditions would, under ideal competitive conditions, be exactly the emission fees called for in theory to correct for the polluting effect. Taking those fees into account would induce NO_x emitters to make decisions about how much, when, and where to emit that are "correct" in the usual sense used in economics, that is, in the same sense that the wages of workers and prices of fuel induce correct decisions about the quantity of these goods and services to use. The marginal damages from our calculations can be thought of as preliminary estimates of the needed Pigovian fees.

An important question, which we do not address here, is how much difference it would make if NO_x emitters had to pay for the damage they cause. In economists' jargon, a lot depends on elasticities. If, for example, raising the price of electricity to reflect the health costs implied by our analysis would not affect the amount of electricity demanded (the price elasticity of

demand is low); if raising the cost to producers of emitting NO_x, perhaps varying it substantially over short periods of time, would have little effect on their decisions about how much or where to produce (the price elasticity of producer demand for NO_x emissions as an input to producing electricity is low), then the substitutions of the sort described above as possible tradeoffs would not occur, or would not occur to significant degree. If, however, as experience suggests is true in most areas of economic activity, there is substantial flexibility in the time and place of production, and substantial flexibility in the commercial and household use of electricity, especially given time to adjust to a different price regime, then large gains are to be had from a regulatory regime that provides the "right" incentives to respond to differences at the margins we have identified. These differences include: meteorological conditions from the perspective of a given source, differences in the biogenic VOC emissions in source areas, and differences in the population density downwind of emission sources.

The tendency in the recent evolution of NO_x regulation has been to rely on cap and trade systems. In particular, under the recent OTC and NO_x SIP Call trading regimes implemented in the northeastern United States, fixed totals of emissions are set for the entire ozone season. Economic theory predicts that trading among sources will tend to equalize the marginal cost of abatement across emitters, where abatement is measured by reductions in emissions at any point in the ozone season. This system misses the distinctions identified in this paper that determine the downstream health impacts of marginal emissions: differences in meteorological conditions within the ozone season, variations in biogenic hydrocarbon emissions and downwind population densities. Variations on cap and trade systems that would address these shortcomings are possible. For example, a modified cap-and-trade program with "zones" could be adopted in which trading in only one direction is permitted (eg. from regions upwind to downwind of high population centers, from regions of high to low emissions of isoprene, etc.). The same sort of modeling work as is carried out in this paper could be used in the design of such alternatives to guide producers to reduce their emissions at times and in places where the emissions cause the most damage.

7. Conclusions and Future Work

In this paper we present a proof-of-concept use of an integrated assessment that combines an atmospheric model with economic and demographic information to estimate the health damage externality due to ozone (O_3) formation from nitrogen oxide (NO_x = NO + NO_2) emissions from large point sources. We use a regional atmospheric model of the eastern United States (CAMx) to examine the variation in the amount of O_3 produced as a result of locating, at times and places with strategically chosen differences in temperature and local biogenic hydrocarbon emissions, a fixed reduction in the nitrogen oxides emitted from power plants within a 9-day period in July 1995. Results of the CAMx model indicate that for the same NO_x emission the O_3 produced can vary by more than a factor of five. In addition, we showed that the variation in health damages that the resulting O_3 causes depends strongly on the size of the exposed population. Within our span of days and conditions examined, variation in O_3 production and downwind population can together result in a factor of five difference in resulting mortalities for an identical change in the quantity of NO_x emitted.

The increasingly widely adopted emission cap-and-trade approach has been very successful at reducing total NO_x emissions from large point sources [*EPA*, 2004b]. However, because it does not control for the location or time during the summer that emissions take place nor for the resulting damages, it is less successful at minimizing the damages that result from the emissions permitted under the cap. To reduce total damage, consideration could be given to a system of fees for emitters to provide incentives for them to reduce NO_x emissions at times and in locations where health damages are greatest. This could be achieved by coupling a regional atmospheric chemistry model that calculates O_3 concentrations obtained from NO_x emitted from individual point sources with estimates of resulting health damages and their economic cost. Such a system would involve a transformation of air quality regulation as it would create an incentive system to reduce total damages in an economically efficient manner rather than simply requiring a reduction in total emissions or the achievement of a particular uniform air quality goal regardless of location or resulting damage.

Although we have suggested the possibility of using model-derived estimates for purposes of setting emission fees, the details of such a system merit examination. Since the fees

are to depend on weather and other conditions, would they be set after the fact (making the emitters, in effect, use the model in forecast mode to make their decisions), or would forecasts be used by the regulator to set fees in advance (based on expected damages)? Regardless of which approach were used, refinements in chemical weather forecasting systems would clearly be beneficial to enable the regulatory system to estimate more accurately appropriate corrective fees, providing emitters with the information they need to adjust their power production and NO_x emission plans to minimize total costs which include mortality and morbidity risks as well as abatement measures.

In addition, to account more fully for the health effects of power plant NO_x emissions it is necessary to include the formation of secondary particulates (PM) and their impact on mortality and morbidity. Secondary PM, in addition to O_3, are formed from NO_x emissions in winter as well as summer. Using an integrated assessment model without chemistry, analyses of the benefits of an annual (rather than just summer) NO_x emissions cap were found to yield substantial net benefits due to reduction of PM2.5 concentrations and its detrimental health effects [*Burtraw et al.*, 2003; *Burtraw et al.*, 2001]. Additional analyses that include both gas and aerosol chemistry and an examination of the effects that spatial and temporal variability in emissions have on PM2.5 formation and resulting health damages and costs would be useful. Such an analysis would permit a determination of the total health damage NO_x emissions cause which in turn would allow a calculation of the fees that an emitter might be charged for the total damage its emissions cause.

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