

Integrated Assessment of the Spatial Variability of Ozone Impacts from Emissions of Nitrogen Oxides

DANIEL Q. TONG,[†]
NICHOLAS Z. MULLER,[‡]
DENISE L. MAUZERALL,^{†,§} AND
ROBERT O. MENDELSON^{*,‡}

*Science, Technology and Environmental Policy Program,
Woodrow Wilson School of Public and International Affairs,
Princeton University, Princeton, New Jersey 08544,
School of Forestry and Environmental Studies,
Yale University, 230 Prospect Street,
New Haven, Connecticut 06511, and Geosciences Department,
Princeton University, Princeton, New Jersey 08544*

This paper examines the ozone (O₃) damages caused by nitrogen oxides (NO_x) emissions in different locations around the Atlanta metropolitan area during a summer month. We calculate O₃ impacts using a new integrated assessment model that links pollution emissions to their chemical transformation, transport, population exposures, and effects on human health. We find that increased NO_x emissions in rural areas around Atlanta increase human exposure to ambient O₃ twice as much as suburban emissions. However, increased NO_x emissions in central city Atlanta actually reduce O₃ exposures. For downtown emissions, the reduction in human exposures to O₃ from titration by NO in the central city outweighs the effects from increased downwind O₃. The results indicate that the marginal damage from NO_x emissions varies greatly across a metropolitan area. The results raise concerns if cap and trade regulations cause emissions to migrate toward higher marginal damage locations.

Introduction

Tropospheric ozone (O₃) is an oxidant formed from photochemical oxidation of hydrocarbons and carbon monoxide in the presence of nitrogen oxides (NO_x = NO + NO₂). Tropospheric O₃ damages human health, agriculture, ecosystems, and materials. NO_x is currently being controlled using cap and trade programs that allow firms to exchange emissions freely across the Eastern United States in order to comply with aggregate emission limits set by the U.S. Environmental Protection Agency (USEPA). Such trading programs increase the efficiency of these regulatory limits on aggregate emissions (1–4). However, the trading programs do not consider the variability in the marginal damages of each ton of NO_x. It is possible that some trades may move pollution from a location where the pollution causes little damage to a location where the pollution causes more damage. Fully efficient trading must weigh the damages per

ton and trade in damage adjusted tons. This would then equate marginal cost per ton to marginal damage per ton at every location (5).

We use a new Integrated Assessment (IA) model to evaluate whether the marginal O₃ damage caused by an additional emission of NO_x is the same across several locations around Atlanta. This new model, the Air Pollution Impact Model (APIM), follows the consequences of emissions by predicting local concentrations, population exposures, and a set of health effects. The model captures the complexity of what each discipline currently understands about air pollution. APIM relies upon the state-of-the-science air quality model (Community Multiscale Air Quality (CMAQ) model), to examine the transport, chemical transformation, and deposition of atmospheric pollutants. The CMAQ model results are then coupled with geographically detailed population distributions to calculate exposures. The most recent epidemiological evidence is used to estimate mortality and morbidity effects.

Past IA studies have suggested that the damages from pollution might vary depending on location (e.g., 5–7). For example, a study using the Comprehensive Air quality Model (CAMx) simulated the ozone produced from NO_x emissions from large point sources in the eastern United States and showed that a shift of a unit of NO_x emissions from one place or time to another could result in large changes in ozone and ozone related health effects (7). This study follows in the vein of these earlier studies but makes a few innovations. It is the first attempt to see whether NO_x emissions across a single metropolitan area may have different regional O₃ impacts depending solely upon where in the metropolitan area they are emitted. This study is also the first attempt to use CMAQ within an IA framework to study the link between a marginal emission of NO_x, O₃, and human health.

We begin by estimating the baseline ambient O₃ concentrations that result from the actual emissions of O₃ precursors in the United States as developed by the USEPA for July 1996. We then test the effect of incremental increases of NO_x emissions in rural, suburban, and center city locations within and around the Atlanta metropolitan area. Taking one site at a time, we increase emissions by 0.5 mol/sec to test how exposures and health impacts change from the baseline level. By comparing the outcomes across nine different emission sites, we examine the differences in incremental damages caused by these incremental emissions from each site.

Method

This paper presents a new IA model, APIM, which links air pollutant emissions to a set of health effects. APIM relies on CMAQ to model emissions, transport, chemical transformation, and deposition of atmospheric pollutants and/or their precursors on both local and regional scales (8). APIM uses the air pollutant concentrations predicted by CMAQ along with spatially disaggregated population data to calculate human exposures to specific air pollutants. Concentration–response relationships from the epidemiological literature are used to convert population exposures into a set of physical health effects. Although not shown in this paper, APIM also calculates exposures to agricultural crops, materials, visibility, and forests, places a dollar value on each effect, and predicts the aggregate dollar damages resulting from the emissions.

In this study, APIM simulates the chemistry and transport of O₃ and its precursors using July 1996 emission data. Point

* Corresponding author e-mail: robert.mendelsohn@yale.edu; phone: 203-432-5128.

[†] Woodrow Wilson School of Public and International Affairs, Princeton University.

[‡] Yale University.

[§] Geosciences Department, Princeton University.

and area source estimates of anthropogenic emissions of NO_x, volatile organic compounds (VOCs), sulfur dioxide (SO₂), carbon monoxide (CO), and ammonia (NH₃) come from the county level USEPA 1996 National Emissions Trends inventory and are processed by the Sparse Matrix Operator Kernel Emissions (SMOKE) model (9). Mobile emissions of NO_x, VOC, CO, and primary particulate matter (PM) are prepared using the vehicle emission model MOBILE5. Biogenic emissions, including NO_x and VOC, are obtained using the Biogenic Emissions Inventory System, version 3 (BEIS3) (10). BEIS3 accounts for variability in temperature and solar radiation when estimating biogenic emissions.

APIM relies on the 3-D 5th Generation Mesoscale Model (MM5) (11) to obtain time-dependent meteorological parameters. The meteorological data are calculated for July 1996 as well. These meteorological data include wind speed and direction, temperature, humidity, surface pressure, and solar radiation. Hourly meteorological data from MM5 are processed using the Meteorology–Chemistry Interface Processor (MCIP) (version 2.2) to drive SMOKE and CMAQ. The transport, chemical transformation, and deposition of O₃ and related chemical species in APIM are simulated using CMAQ (version 4.2). CMAQ (8) uses the emission inputs from SMOKE and meteorology inputs from MM5 (after processing by MCIP) to predict hourly concentrations of each pollutant over space and time.

In this study, CMAQ is configured to include detailed implementation of horizontal and vertical advection, turbulent diffusion based on K-theory, gas, liquid, and particulate chemistry using a modified version of the CBM-IV chemical mechanism (12). CMAQ also includes dry deposition, cloud physics, and chemistry (8). The domain is divided into 132 columns and 90 rows with a horizontal grid resolution of 36 × 36 km². There are 12 vertical layers extending from the surface to approximately 15 km. Vertical layers are unevenly distributed with 6 layers in the lowest kilometer, and a surface layer (layer 1 thickness) of approximately 38 m.

The first 7 days of each month-long simulation are used for spin-up and are excluded from subsequent analysis. Lateral boundary conditions for 19 chemical species are derived from a multi-year simulation of the global chemical transport Model of Ozone and Related Tracers, version 2 (MOZART-2) (13). The top of the model domain is at 200 mbar, and CMAQ makes a zero-flux assumption at the top boundary; the contribution of stratosphere–troposphere exchange (STE) to tropospheric O₃ is not included in this study.

Tropospheric O₃ concentrations predicted by this version of CMAQ have been compared with surface measurements from 987 Air Quality System (AQS) (the former AIRS) and 123 Clean Air Status and Trends Network (CASTNet) networks as well as with vertical O₃ profiles from ozonesonde data (14). For July 1996, CMAQ reproduces surface O₃ for a wide range of conditions (30–80 ppbv) with a normalized mean error (NME) less than 35% and normalized mean bias (NMB) lying between ±15% for the whole domain (14). Although systematically over-predicting O₃ in the eastern United States and under-predicting it in the western United States, CMAQ is able to reproduce 1-hour and 8-hour daily maxima with a cross-domain mean bias (MB) of 1 ppbv and 8 ppbv, or NMB of 8% and 25%, respectively (14).

APIM calculates human exposures by multiplying predicted O₃ concentrations (ppmv) by the human population in each county in the continental United States. A similar approach can be used to calculate exposure of agricultural crops and tree species. For human exposures, we use the U.S. Census Bureau's 1996 county population data (U.S. Census Bureau; <http://www.census.gov>). The county population data are broken down by age group.

APIM then calculates a set of physical health effects for each age group resulting from the change in human O₃

exposures between the baseline scenario and each perturbation scenario. The calculation depends on the accuracy of the concentration–response function in the vicinity of the baseline concentrations. The concentration–response functions in APIM come from empirical relationships estimated in the epidemiological literature.

The concentration–response function for premature mortality was calculated by examining the relationship between mortality rates and O₃ concentrations in 95 American cities (15). This relationship has recently been supported by three additional studies (16–18). APIM relies on the log-linear form used in the epidemiological literature. The mortality rate is regressed on a vector of control variables, X, and the level of pollution, [O₃]

$$\ln(H/\text{Pop}) = \sum \alpha X + \beta[\text{O}_3] \quad (1)$$

where (H/Pop) is the mortality rate (deaths, H, per population, Pop). α and β are estimated coefficients. The O₃ concentrations in most of the human studies rely on actual concentrations.

Exponentiating eq 1 and multiplying by population yields an estimate of deaths

$$H = \text{Pop}(\exp^{\sum \alpha X + \beta[\text{O}_3]}) \quad (2)$$

The change in deaths (H₁ – H₀) from a change in ozone concentrations ([O₃]₁ – [O₃]₀) would be

$$H_1 - H_0 = \text{Pop}(\exp^{\sum \alpha X + \beta[\text{O}_3]_1}) - \text{Pop}(\exp^{\sum \alpha X + \beta[\text{O}_3]_0}) \quad (3)$$

The change in the mortality rate from pollution is proportional to the underlying mortality rate. For example, according to eq 3, the elderly will suffer more additional deaths from pollution than the young simply because they have a higher underlying mortality rate. For acute mortality, coefficient β is 0.00052 (15) (this parameter is interpreted as the change in mortality rate corresponding to a 1 ppbv change in the daily average O₃ concentration.)

We also examine two morbidity effects associated with O₃ exposure: hospital admissions for respiratory ailments (19) and emergency room visits for asthma (20). Both respiratory ailments and emergency room visits have the same functional form as above. For respiratory hospital admissions β is 0.00715 (19). For emergency-room visits for asthma, β is 0.0035 (20).

We then convert all three health effects into a single index of health days lost. We assume emergency room visits for asthma constitute a loss of 1 day. Hospital admissions for respiratory ailments are modeled as a loss of 6 days, reflecting the mean length of stay for hospital admissions due to respiratory disorders (Agency for Healthcare Research and Quality, Healthcare Utilization Project: <http://hcup.ahrq.gov/Hcup-net.asp>). Premature mortality is valued as the expected days of life lost given a person's age. Expected days of life are calculated by summing the number of remaining days a person would live times the probability of survival. These expected days of life lost reflect the underlying mortality rates by age in each county. On average, an American newborn in 2003 is expected to live approximately 77 years (21).

Results

Using July 1996 meteorological conditions and emissions for the entire country, we utilized CMAQ to predict O₃ concentrations over every county in the continental United States. Of course, only the counties that are downwind from Atlanta are relevant to the results. This is the baseline simulation for the rest of our comparisons. All of our simulations used the same July 1996 meteorology. The paper focused strictly on the variability in summer impacts of NO_x emissions. We then

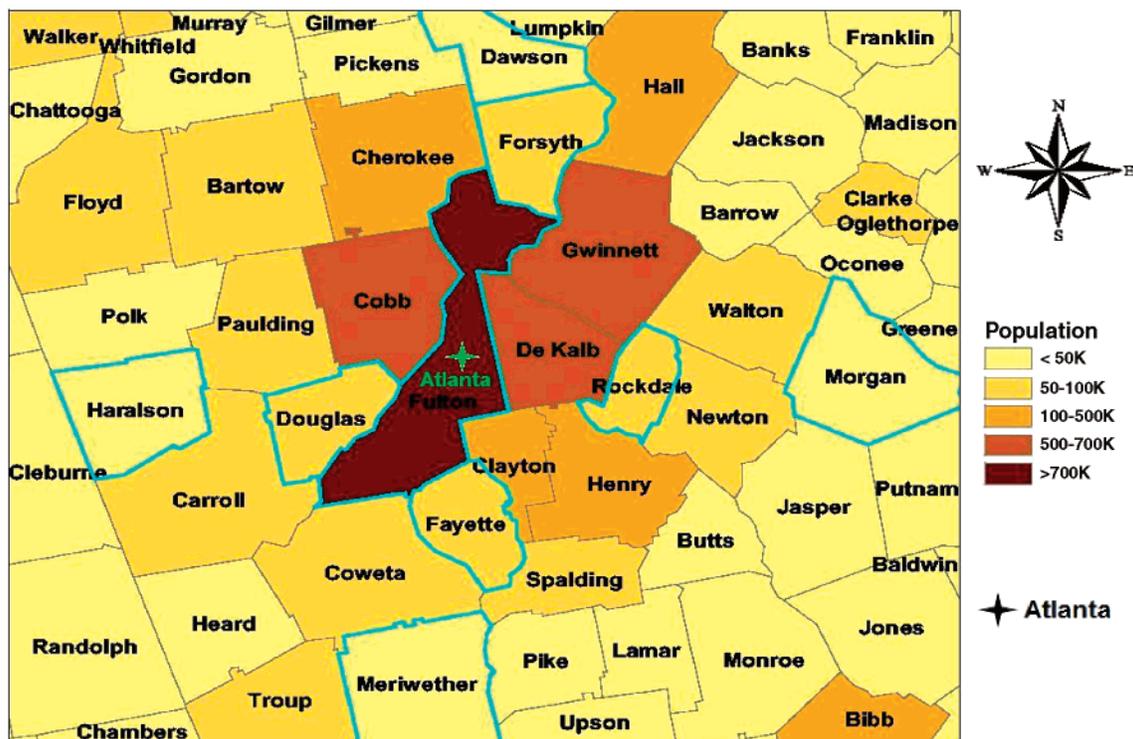


FIGURE 1. Map of Atlanta metropolis and the surrounding area. Selected counties (with blue boundaries) designate locations where additional NO_x emissions are added.

chose nine locations in and around Atlanta and increased NO_x emissions in each selected location by 0.5 mol/sec for 23 days holding conditions in the rest of the country constant. These increases in emissions over this time period result in the total release of 31.4 tons of NO_x. In each case, CMAQ is rerun for the same 23 day period. Each perturbation run pertains to additional emissions from a different site. The difference between a perturbation run and the baseline run represents the predicted change in O₃ concentrations resulting from the incremental NO_x emissions. We calculate the effect of a single ton of emissions by dividing the total change in exposures and health consequences by the 31.4 tons added.

NO_x emissions are added to a source county in proportion to the original emissions profile within that county as follows:

$$\Delta\text{NO}_{ij} = \frac{E_T \times \text{NO}_{ij} \times f_i}{\sum_{i=1}^M \sum_{j=1}^N [(\text{NO}_{ij} + \text{NO}_{2ij})f_i]} \quad (4)$$

$$\Delta\text{NO}_{2ij} = \frac{E_T \times \text{NO}_{2ij} \times f_i}{\sum_{i=1}^M \sum_{j=1}^N [(\text{NO}_{ij} + \text{NO}_{2ij})f_i]} \quad (5)$$

where ΔNO_{ij} and ΔNO_{2ij} are the increases of NO and NO₂ emissions in the source county, E_T is the total increase in NO_x emissions from that county (in this case E_T equals 0.5 mol/sec), NO_{ij} and NO_{2ij} are the baseline emissions from the source county, f_i is the fraction of grid cell i located inside the county (f ranges from 0 to 1), M is the number of grid cells inside the county, and N is the number of layers into which NO_x is emitted. In this way, the additional NO_x emissions are distributed across the entire source county, with each included grid cell receiving a portion of the 0.5 mol/sec in proportion to the baseline emissions profile. The additional NO_x is continuously emitted for the month long simulation period. The increased NO_x emissions represent

approximately 2% of the total amount of NO_x emitted from Fulton County (urban center). Except in the rural Haralson county, in which elevated sources dominate, surface sources (mobile, area, and biogenic sources) contribute over three quarters of total NO_x emissions in these selected counties (Table 1).

The nine locations include the county containing Atlanta, 4 suburban counties near Atlanta with populations between 50 000 and 100 000 persons, and 4 rural counties with populations less than 50 000 which are shown in Figure 1. The source counties surrounding Atlanta are distributed north, south, east and west of the city. We use APIM to test whether the incremental O₃ impacts from the NO_x emissions are the same regardless of the location of the emission source.

The change in O₃ concentrations due to the additional NO_x emissions in a rural county 75 km west of Atlanta is shown in Figure 2 (Haralson County). NO_x emissions have a complex spatial effect on O₃ concentrations, first depleting O₃ (titration by NO), and then catalyzing its production in downwind regions (22). The model predicts a small territory surrounding the source where NO_x emissions reduce O₃ concentrations (titration) surrounded by a much larger region in which O₃ levels increase. The increase in O₃ concentrations spreads to the northeast due to the prevailing winds. The summertime circulation in the Southeast United States is dominated by the Bermuda high pressure system, as depicted in Figure 3 by the monthly average surface wind in July 1996. Similar meteorological conditions have been reported for the same period in other years.

The resulting changes in human O₃ exposure in this case closely mirror the spatial distribution of O₃ changes. The small zone near the source has reduced O₃ exposures because the increased NO_x emissions cause local titration. A much larger area further downwind experiences an increase in human O₃ exposures. With rural emissions, the reduction of exposures in the small titration zone is overwhelmed by the much larger increase in exposures further downwind. The net effect is an increase in O₃ exposures.

TABLE 1. July 1996 Average NO_x Emissions from Surface and Elevated Emissions Sources from Nine Source Counties Near the Atlanta Metropolitan Area

source county	surface emissions (mobile, area and biogenic sources) (mol/sec)	elevated emissions (power plants) (mol/sec)	percentage of NO _x emitted from surface sources	relation to Atlanta
Fulton	26.02	3.07	89%	Atlanta, center city
Douglas	3.90	0.90	81%	west, adjacent suburb
Fayette	6.35	0.90	88%	south, adjacent suburb
Forsyth	2.45	0.07	97%	north, suburb
Rockdale	1.49	0.01	99%	east, suburb
Dawson	0.75	0.01	97%	north, rural
Haralson	1.80	2.14	46%	west, rural
Meriwether	1.95	0.60	76%	south, rural
Morgan	1.28	0.05	96%	east, rural

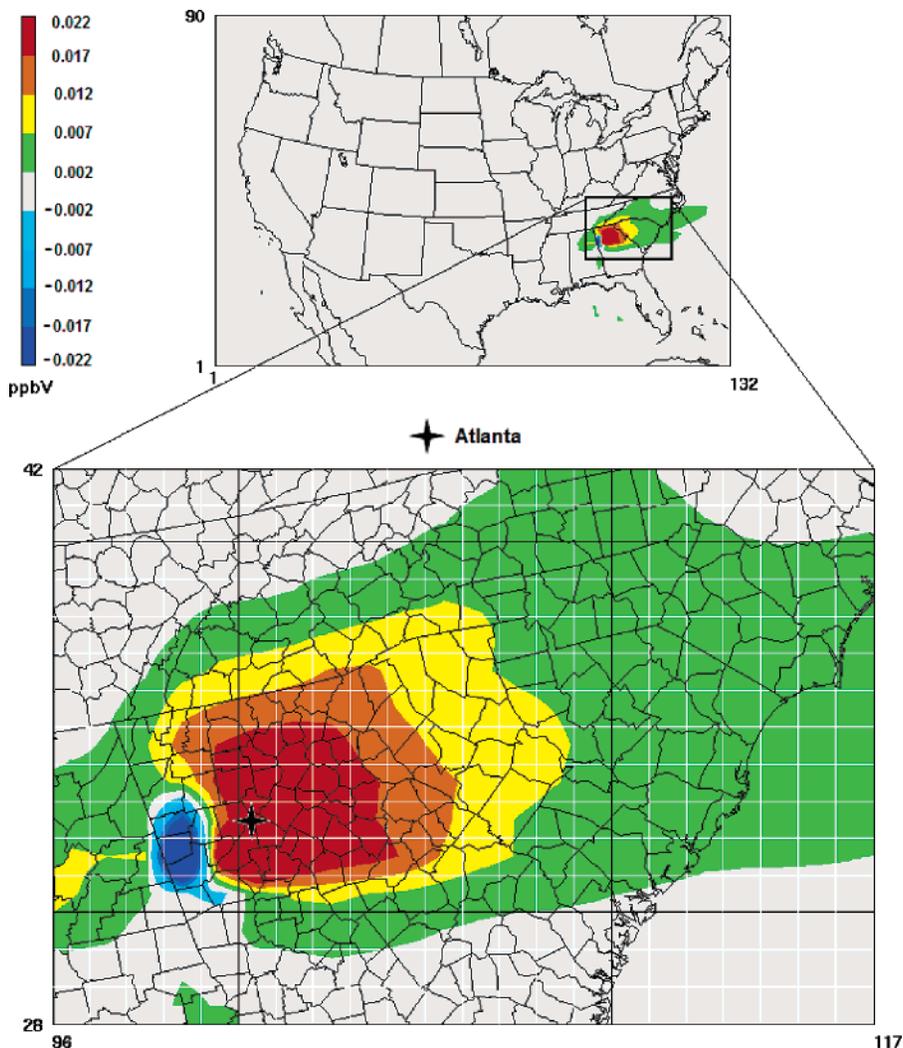


FIGURE 2. Change in monthly average concentrations of surface O₃ (ppbv) due to added emissions of 0.5 mol/sec NO_x in Haralson County, Georgia.

In our second example, shown in Figure 4, we look at the changes in O₃ due to increased NO_x emissions in Atlanta (Fulton County). Note that the area of titration that causes reduced O₃ is larger in Figure 4 than Figure 2. NO_x emissions are already sufficiently high in Atlanta that the local O₃ photochemistry is limited by the availability of VOCs. Adding more NO_x in this environment reduces inner-city O₃ concentrations. However, what is especially important in the downtown Atlanta example is that the human population is dense in this titrated zone. The aggregate reduction in human O₃ exposure in the titrated zone is larger than the aggregate

increase in human O₃ exposures further downwind. Hence, aggregate exposures in the Atlanta emission scenario fall below the baseline level.

An additional ton of NO_x emission in Atlanta (Fulton) spread over July 1996 reduces aggregate human exposures to O₃ by approximately 96 people ppmv (see Table 2). The suburban counties Fayette and Douglas are slightly upwind of Atlanta so that their zone of titration includes the high Atlanta populations. Emissions in these counties cause a small reduction in exposure. The titration zone for the more distant suburbs of Forsyth and Rockdale, however, does not

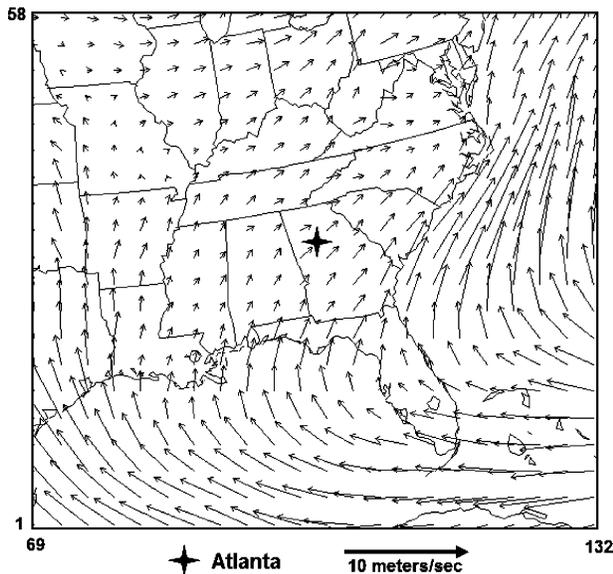


FIGURE 3. Monthly mean surface wind in July 1996 over the southeast United States. Data are from the MM5 simulations which provide the meteorological fields for CMAQ.

include Atlanta. A ton of NO_x emitted from these suburbs increases O_3 exposures by 57 people ppmv. Finally, in each of the rural areas, a ton of NO_x emitted over the course of July increases O_3 exposures by an aggregated 157–176 people ppmv. Merriwether exposures are slightly higher because it is upwind of Atlanta. For the rural and distant suburban emission scenarios, the reduction of human exposures in the titrated areas is small compared to the increased exposures downwind. Note that all these changes in aggregate exposures reflect small changes in concentrations across millions of people.

We use the change in O_3 exposures and eq 3 to estimate the premature mortalities, hospital admissions for respiratory ailments, and emergency room visits for asthma. These effects are combined in a single measure of health days lost. The

TABLE 2. Ozone (O_3) Exposures and Health Impacts Resulting from an Additional Emitted Ton of NO_x from Different Counties In and Around Atlanta^a

source county	change in human exposures (people × ppmv of O_3)	change in health days lost	relation to Atlanta
Fulton	-96	-2	center city
Douglas	-21	-1	west, adjacent suburb
Fayette	-11	-0.3	south, adjacent suburb
Forsyth	57	2	north, suburb
Rockdale	57	2	east, suburb
Dawson	158	4	north, rural
Haralson	167	4	west, rural
Meriwether	176	5	south, rural
Morgan	157	4	east, rural

^a The table reflects the marginal effect of adding an emission of 1 ton/month NO_x to each county spread across July. Exposures are calculated by multiplying the change in concentration by population exposed. Health is calculated using concentration response functions and changes in concentrations times populations.

mortality effects represent over 90% of these health days lost. The health effects are similar to the exposure results in Table 2 but they are not exactly proportional because the sensitivity of the population depends on the age of those exposed. We can calculate what happens to each age group because the population exposures in each county are broken down by age. Because the elderly have much higher baseline mortality and morbidity rates, and because pollution effects are proportional to the baseline, the elderly suffer a higher fraction of the health days lost. For example, with the 4 rural emission perturbations, the elderly suffer almost half of all the deaths.

NO_x emissions in downtown Atlanta reduce O_3 exposures and thus health days lost by a total of 2 days per ton per month. The additional NO_x emission reduces O_3 concentrations in a high population zone. The reduction in human exposures in the titration zone is larger than the increase in

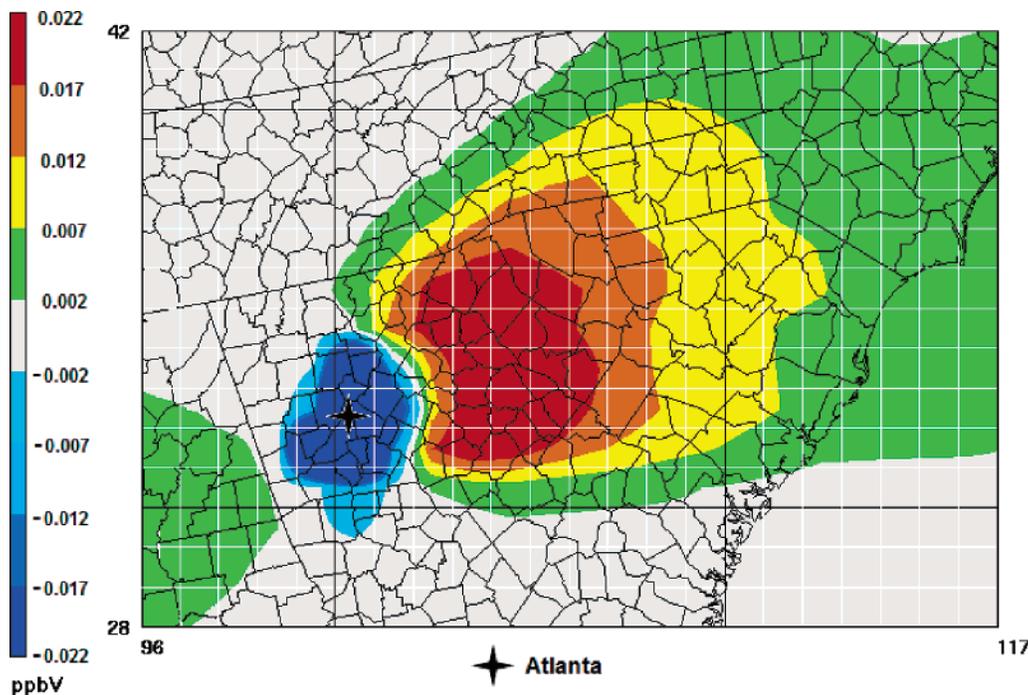


FIGURE 4. Change in monthly average concentrations of surface O_3 (ppbv) due to added emissions of 0.5 mol/sec NO_x in Fulton County, Georgia.

human exposure further downwind. The net result is fewer health days lost from O₃ exposures. Emissions in the adjacent suburban counties also reduce health days lost by 1 day per ton. However, emissions in more distant suburbs increase health days lost by 2 days/ton and emissions in rural areas increase health days lost by 4 to 5 days/ton. Merriwether health effects are slightly higher because it is upwind of Atlanta. In the more rural areas, there are fewer people living in the titration zone. Consequently, the increased human exposure downwind dominates the net effect. Most of these health days lost are from premature mortality.

Discussion and Summary

This paper describes an experiment to test whether the damages from incremental emissions of NO_x are the same across a metropolitan area. We utilize APIM, a new IA model built around a state-of-the-science air quality modeling system (SMOKE/MM5/CMAQ), to test this hypothesis in and around Atlanta. By adding NO_x emissions in different locations one by one, we are able to compute the marginal impacts of the resulting change in surface O₃ on public health. The results reveal that the regional O₃ damages from NO_x emissions vary considerably across a metropolitan area. Whereas NO_x emissions generally cause summertime O₃ damages, especially in rural areas, excessive emissions in the center city actually reduce the health damages from O₃. The marginal damage of a ton of emissions varies considerably across space.

The fact that the damages from emissions are not the same across space is an important policy finding. Recent O₃ regulations have set in place a cap and trade program to control summertime NO_x emissions in the Eastern and Midwestern United States. This market setting places the same price on all tons of NO_x. The policy does not differentiate whether a ton reduces or increases O₃ damages.

Given that the O₃ damages of NO_x emissions do vary across space, it is important that the pollutant trading scheme get the prices right. Consideration should be given to weighting each ton by the damage it causes. If ton A causes twice the damage of ton B, it would be appropriate for polluters to reduce two tons of B in order to emit one more ton of A. Increased emissions of harmful tons should not be purchased with reductions in emissions of beneficial tons.

Of course, further work must be completed before developing a complete pollutant price index. First, this study examines only the effect of NO_x emissions on O₃. The harm caused by NO_x emissions through NO₂ and especially through the formation of secondary particulates must also be calculated. These other damages also vary across space although they are not expected to have the same spatial pattern as O₃ damages. Second, the experiment was conducted over a single summer month. Simulations over longer time-periods would provide valuable insight into the temporal variability of the results. Third, similar analyses need to be conducted in other locations to get a more complete picture of the damages caused across the country. Finally, a horizontal resolution of 36 km is used in this study. Model simulations at a finer resolution might result in different damage estimates, but are not expected to change the major findings of this study.

Acknowledgments

We thank the Glaser Progress Foundation for the financial resources to conduct this study and their enthusiastic support. We thank Princeton University for computational resources. We also thank Professors William Nordhaus, David Bradford, and Michelle Bell for their guidance and encouragement.

Literature Cited

- (1) Dales, J. H. *Pollution, Property & Prices: An Essay in Policy-making and Economics*; University of Toronto Press: Toronto, ON, 1968.
- (2) Tietenberg, T. H. *Emissions Trading: An Exercise in Reforming Pollution Policy*; Resources for the Future: Washington, DC, 1985.
- (3) Baumol, W. J.; Oates, W. E. *The Theory of Environmental Policy*, 2nd ed.; Cambridge University Press: New York, 1988.
- (4) Hahn, R. W.; Stavins, R. N. Incentive-based environmental regulation: A new era from an old idea? *Ecol. L. R.* **1991**, *18*, 8–10.
- (5) Mendelsohn, R. Regulating Heterogeneous Emissions. *J. Environmental Economics and Management* **1986**, *13*, 301–312.
- (6) Mendelsohn, R. An Economic Analysis of Air Pollution from Coal-Fired Power Plants. *J. Environmental Economics Management* **1980**, *7*, 30–43.
- (7) Mauzerall, D. L.; Sultan, B.; Kim, N.; Bradford, D. NO_x Emissions: Variability in Ozone Production, Resulting Health Damages and Economic Costs. *Atmos. Environ.* **2005**, *39*, 2851–2866.
- (8) Byun, D. W., Ching, J. K. S., Eds. *Science Algorithms of the EPA Models-3 Community Multiscale Air Quality (CMAQ) Modeling System*; EPA Report EPA-600/R-99/030; Office of Research and Development: Research Triangle Park, NC, 1999. <http://www.epa.gov/asmdner/CMAQ>.
- (9) Carolina Environmental Program. *Sparse Matrix Operator Kernel Emission (SMOKE) Modeling System*; University of North Carolina, Carolina Environmental Programs: Research Triangle Park, NC, 2003. <http://www.unc.edu/cep/emdp/products/smoke>.
- (10) Pierce, T. E. *BEIS3 v. 0.9*; USEPA, Research Triangle Park, NC, 2001. <ftp://ftp.epa.gov/amd/asmd/beis3v09/>.
- (11) Grell, G. A.; Dudhia, J.; Stauffer, D. R. *A Description of the Fifth-Generation Penn State/NCAR Mesoscale Model (MM5)*; NCAR Technical Note, NCAR/TN-398+IA; National Center for Atmospheric Research, Boulder, CO, 1993.
- (12) Gery, M. W.; Whitten, G. Z.; Killus, J. P.; Dodge, M. C. A Photochemical Kinetic Mechanism for Urban and Regional Scale Computer Modeling. *J. Geophys. Res.* **1994**, *99*, 925–956.
- (13) Horowitz, L. W.; Walters, S.; Mauzerall, D. L.; Emmons, L. K.; Rasch, P. J.; Granier, C.; Tie, X.; Lamarque, J. F.; Schultz, M. G.; Tyndall, G. S.; Orlando, J. J.; Brassuer, G. P. A Global Simulation of Tropospheric Ozone and Related Tracers: Description and Evaluation of MOZART, version 2. *J. Geophys. Res.* **2003**, *108*, 4784. doi: 10.1029/2002JD002853.
- (14) Tong, D. Q.; Mauzerall, D. L. Spatial Variability of Summertime Tropospheric Ozone over the Continental United States: Implications of an Evaluation of the CMAQ Model. *Atmos. Environ.*, in press.
- (15) Bell, M. L.; McDermott, A.; Zeger, S. L.; Samet, J. M.; Dominici, F. Ozone and Short-term Mortality in 95 US Urban Communities 1987–2000. *J. Am. Med. Assoc.* **2004**, *292*, 2372–2378.
- (16) Bell, M. L.; Dominici, F.; Samet, J. M. A Meta-Analysis of Time-Series Studies of Ozone and Mortality With Comparison to the National Morbidity, Mortality, and Air Pollution Study. *Epidemiology* **2005**, *16*, 436–445.
- (17) Ito, K.; De Leon, S. F.; Lippman, M. Associations Between Ozone and Daily Mortality: Analysis and Meta-Analysis. *Epidemiology* **2005**, *16*, 446–457.
- (18) Levy, J. I.; Chemerynski, S. M.; Sarnat, J. A. Ozone Exposure and Mortality: An Empiric Bayes Metaregression Analysis. *Epidemiology* **2005**, *16*, 458–468.
- (19) Schwartz, J. Short-Term Fluctuations in Air Pollution and Hospital Admissions of the Elderly for Respiratory Disease. *Thorax* **1995**, *50*, 531–538.
- (20) Steib, D. M.; Burnett, R. T.; Beveridge, R. C.; Brook, J. R. Association between Ozone and Asthma Emergency Department Visits in Saint John, New Brunswick, Canada. *Environ. Health Perspect.* **1996**, *104*, 12.
- (21) Hoyert, D. L.; Kung, H.-C.; Smith, B. L. Death: Preliminary Data for 2003. *Natl. Vital Statistics Rep.* **2005**, *53*, 15.
- (22) Chameides, W. L.; Saylor, R. D.; Cowling, E. B. Ozone pollution in the rural United States and the new NAAQS. *Science* **1997**, *276*, 916.

Received for review December 6, 2005. Accepted December 16, 2005.

ES052452N