

# **U.S. EPA—South Carolina Collaboration to Develop a Multi-Pollutant, Risk-Based Air Quality Management Strategy for the Upstate South Carolina Region**

---



*May 26, 2016*

*(This page left intentionally blank)*

# ACKNOWLEDGEMENTS

## *Authors*

SC Department of Health and Environmental Control:

Tommy Flynn  
Maeve Mason  
Andy Hollis

US Environmental Protection Agency:

Kimber Scavo  
Neal Fann  
Julia Gamas  
Ali Kamal  
Mark Morris  
Ted Palma

We gratefully acknowledge the work of CAU/TATT and Dean Hybl, Elzbieta Covington, Paul Martin, Greg Quina, Leslie Coolidge, Brian Barnes, Gregory Green, Martha Keating, Tyler Fox, Alison Eyth, David Misenheimer, Marc Houyoux, Sharon Phillips, Laura Bunte, Regina Chappell, Brad Akers, Lynorae Benjamin, Jane Spann, Karen Wesson, Rudy Kapichak, and Kelly Sheckler.



South Carolina Department of Health  
and Environmental Control



## **U.S. EPA—South Carolina collaboration to develop a multi-pollutant, risk-based air quality management strategy for the Upstate South Carolina Region**

---

### **Table of Contents**

<b>ACKNOWLEDGEMENTS .....</b>	<b>iii</b>
Authors .....	iii
<b>Executive Summary.....</b>	<b>1</b>
<b>Overview .....</b>	<b>1</b>
<b>Section I: Project Summary.....</b>	<b>3</b>
Choose an Area of Study and Set Goals.....	4
Assess Current Air Quality Issues in the Upstate Region.....	4
Decide on the Parameters for the Emissions Inventory .....	5
Develop a Control Strategy .....	5
Process Emissions for Modeling .....	6
Conduct Air Quality Modeling.....	7
Assess Risk from Air Toxics .....	7
Assess Ozone and PM <sub>2.5</sub> Benefits .....	7
<b>Section II: Results &amp; Recommended Next Steps .....</b>	<b>9</b>
Results .....	9
BenMAP Risk Assessment .....	17
Overarching Conclusions .....	18
Key Take Away/Lessons Learned .....	19
Recommended Next Steps.....	21
<b>Section III: Project Template.....</b>	<b>23</b>
Multi-Pollutant Analysis Template.....	23
<b>Appendix A: Original Project Description -- November 2013.....</b>	<b>25</b>
Overview .....	25
Demographic.....	26
Air Quality Issues in the Upstate Region .....	26
Tools and Data.....	28
Emissions Inventory and Baseline and Regional Modeling.....	28

<i>Control Measure Information and Additional Data</i> .....	29
Air Quality Modeling .....	30
Air Toxics Risk Assessment.....	31
Ozone and PM <sub>2.5</sub> Benefits Assessment .....	31
Insights on Development of the Local Control Strategy .....	32
Other Planning/Policy Considerations .....	34
<b>Appendix B: Background on Air Quality Management: Working toward a Multi-Pollutant Approach.....</b>	<b>38</b>
<b>Appendix C: 2011 NATA Risk Reduction Analysis - South Carolina Ten at the Top Counties .....</b>	<b>40</b>
Background.....	40
CAU/TATT Air Toxic Emissions .....	40
CAU/TATT Estimated Cancer Risks .....	42
Emission Reductions and Estimated Cancer Risk Reductions .....	44
<b>Appendix D: South Carolina Ten at the Top Counties Cost Analysis.....</b>	<b>46</b>
Introduction.....	46
CAU/TATT Criteria Emissions Profile .....	47
CAU/TATT CoST Analysis Results .....	49
Geographic Distribution of CAU/TATT Emissions Reductions.....	50
<b>Appendix E: South Carolina Ten at the Top Counties CMAQ Modeling.....</b>	<b>59</b>
Introduction.....	59
Modeled PM <sub>2.5</sub> Reductions .....	60
Ozone Reductions .....	64
Other Pollutants .....	65
<b>Appendix F: Additional Information Regarding Health-Related Benefits .....</b>	<b>69</b>
Introduction to Benefits Analysis Methods .....	69
Health Impact Assessment.....	71
Economic Valuation of Health Impacts .....	73
Uncertainty Characterization .....	75
Benefits Analysis Data Inputs .....	77
Demographic Data.....	77
Effect Coefficients .....	78
Baseline Incidence Estimates .....	82

<i>Economic Valuation Estimates</i> .....	85
<i>Growth in WTP Reflecting National Income Growth Over Time</i> .....	92
<b>References</b> .....	<b>95</b>

## **U.S. EPA—South Carolina collaboration to develop a multi-pollutant, risk-based air quality management strategy for the Upstate South Carolina Region**

---

### **Executive Summary**

This report describes a collaborative effort between the U.S. EPA, the South Carolina Department of Health and Environmental Control, and local community and business leaders in ten upstate South Carolina counties to develop and analyze a multi-pollutant, risk-based air quality management strategy. A primary goal was to identify and evaluate a local control strategy targeting emissions of ozone and PM<sub>2.5</sub> and their precursors while at the same time reducing air toxics of concern for communities to maximize both health benefits and air quality improvements. This report provides an overview of the data and analytical steps needed for such an analysis. The results of this project demonstrate that improving air quality in areas already attaining the NAAQS can yield significant health benefits. This project can also inform and help attainment areas assess actions to keep ozone and particulate matter levels below the level of the NAAQS to ensure continued health protection for their citizens, better position such areas to remain in attainment, and help all areas efficiently direct available resources toward a more cost-effective strategy. Perspectives from each of the partners in this study are also provided in this report. In general, local area perspective and expertise play a large role in successfully implementing any voluntary emissions reduction program. Additionally, this collaborative effort between federal and state technical staff allowed for knowledge transfer and feedback on new and innovative tools developed during the course of this project.

### **Overview**

The U.S. Environmental Protection Agency (EPA) and the State of South Carolina's Department of Health and Environmental Control (DHEC) share an interest in exploring multi-pollutant analysis and planning as a means to improve air quality effectively, and as a way to make the most efficient use of available resources. In 2012, the Office of Air Quality Planning and Standards launched the voluntary Ozone Advance Program. This program provides assistance to areas throughout the country that want to create a better buffer against future violations of the ozone national ambient air quality standard (NAAQS) by helping identify and implement pollution reductions strategies designed to help areas from falling into nonattainment status. The Ozone Advance program was soon followed by the Particulate Matter (PM) Advance program, which was similarly designed to help areas take steps to reduce local PM levels. Several areas, including South Carolina joined both programs and EPA soon realized this was an excellent opportunity to implement and further study multi-pollutant planning.

The EPA's Detroit multi-pollutant pilot project<sup>1</sup> provided a framework for analyzing air quality management programs capable of realizing multiple policy goals. In particular, the Detroit project demonstrated that it is possible to achieve air quality improvements among an array of pollutants while also reducing air pollution risk to both the general population and those most vulnerable to air pollution-related health impacts.

---

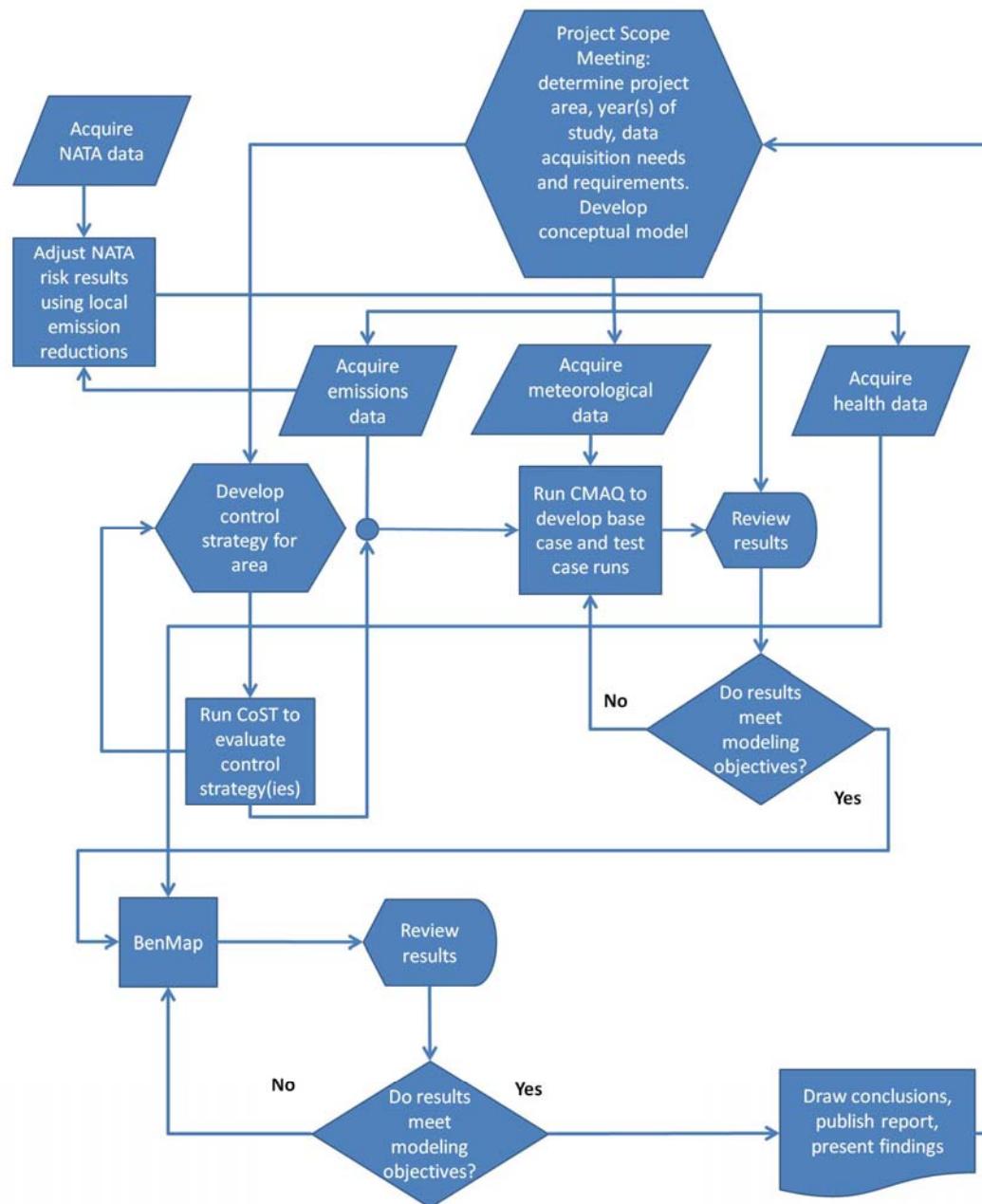
<sup>1</sup> Wesson, K., Fann, N., Morris, M., Fox, T., Hubbell, B., 2010. A multipollutant, risk-based approach to air quality management. Case study for Detroit. *Atmospheric Pollution Research*, 1, 296– 304.

A team at the EPA's Office of Air Quality Planning and Standards, EPA Region 4 in Atlanta, DHEC, and South Carolina's Upstate Region (Upstate) which includes the nonprofit group Clean Air Upstate Coalition/Ten at the Top (CAU/TATT) began work together in 2013 on a multi-pollutant, risk-based air quality management strategy building upon the information learned in the Detroit pilot project. Section I of this document is the project summary that documents the work conducted throughout the collaboration. Section II describes the results and recommended next steps from the analyses and Section III includes a template which outlines the practical steps we took throughout the process and highlights additional points to consider. There are several appendices which include details of the analyses and collaboration.

## Section I: Project Summary

The following flow chart (Figure 1) provides an overview of the process used in the development of this multi-pollutant, risk-based air quality management strategy and serves as a guide to describe the various steps.

**Figure 1:**



### Choose an Area of Study and Set Goals

In the summer of 2013, EPA and DHEC had several conference calls to scope out the parameters for working together including deciding on the area of study. DHEC, in consultation with their local partners, recommended studying the CAU/TATT area (<http://tenatthetop.org/>). It is a group of 10 counties in the upstate of SC that were very proactive in taking steps to implement local measures. The 10 counties are Abbeville, Anderson, Cherokee, Greenville, Greenwood, Laurens, Oconee, Pickens, Spartanburg, and Union counties. Over the next few months, the team worked together to develop a project plan and description (See Appendix A) which included the following goals:

- (1) identify local emission reduction measures for the Upstate that address multiple pollutants, that are harmonized with existing or planned federal/state/local measures,<sup>2</sup> that are quantifiable, and whose implementation by DHEC and/or Upstate is achievable;
- (2) maintain compliance with the National Ambient Air Quality Standards (NAAQS);
- (3) demonstrate that the selected strategy(ies) can reduce population risk from exposure to ozone, PM<sub>2.5</sub>, and selected air toxics in the Upstate and can reduce exposure among populations at greatest level of baseline risk;
- (4) transition to a multi-pollutant air quality management strategy; and
- (5) foster a spirit of collaboration among EPA, the Upstate, and DHEC that highlights the importance of a coalition approach.

### Assess Current Air Quality Issues in the Upstate Region

In order to understand the air quality issues in the area, DHEC presented the current air quality data to EPA (2010-12) for both ozone and PM<sub>2.5</sub> and EPA provided its most recent projected air quality for the year 2020. The EPA also presented the 2005 National Air Toxics Assessment (NATA) data to assess air toxics in the region and updated that analysis to reflect the 2011 NATA towards the end of the project.

*PM<sub>2.5</sub> air quality levels.* 2010-2012 air quality data indicated that the Upstate attained the current annual PM<sub>2.5</sub> NAAQS by a narrow margin. Anderson, Greenville, and Spartanburg Counties were designated as unclassifiable for the 1997 PM<sub>2.5</sub> NAAQS (70 FR 944, January 5, 2005). The Upstate was attaining the 2006 and 2012 PM<sub>2.5</sub> standards for daily and annual PM<sub>2.5</sub>. 2010-2012 design values for PM<sub>2.5</sub> monitors in the Upstate indicated that Greenville had a 10.9 µg/m<sup>3</sup> design value (12 µg/m<sup>3</sup>) for the annual standard and a 23 µg/m<sup>3</sup> (35 µg/m<sup>3</sup>) design value for the 24-hr standard.

*Ozone air quality levels.* The Upstate was attaining both the 1997 (0.08 ppm) and 2008 (0.075 ppm) ozone. 2010-2012 design values indicated that Abbeville (0.064 ppm), Anderson (0.073 ppm), Greenville (0.069 ppm), Pickens (.071 ppm), and Spartanburg (0.075 ppm) are all in a range of concern for attaining any future more stringent NAAQS.

---

<sup>2</sup> See the 2004 National Academy of Sciences (NAS) report describing the elements of a multi-pollutant air quality management plan (AQMP).

*Projected ozone and PM<sub>2.5</sub> air quality levels.* EPA provided 2010-2012 design values for the Upstate counties as well as projected 2020 design values based on EPA's photochemical modeling used in the Regulatory Impact Analysis (RIA) for the PM<sub>2.5</sub> NAAQS Final Rule (Please note that this modeling used a 2007-based modeling platform with projections from the 2007-2009 design values and not all monitors were included in the model run. Furthermore, some areas had projected design values, but no operating monitor due to monitor shutdowns). Based on EPA's regulatory modeling, the Upstate counties realized reductions in their projected design values due to Federal rules that are expected to be in place in 2020 including multiple mobile source rules and the Mercury and Air Toxics (MATS) final rule. Table I in Appendix A displays this information.

*Air toxics.* Based on the 2011 National Air Toxic Assessment (NATA) nearly 28,000 tons of air toxics are emitted each year from the Upstate. According to NATA, the average cancer risk in the Upstate associated with inhalation of air toxics is about 47 in a million. A majority of this risk is associated with formaldehyde (62 percent) with acetaldehyde (14 percent) and benzene (8 percent) also key contributing pollutants. Formaldehyde and acetaldehyde are generally formed with photochemical activity along the I-85 corridor in the southeast US, while benzene emissions are associated with mobile traffic along the many interstates in the Upstate. NATA estimates that the about 3,000 people who live in the area are exposed to cancer risks greater than 60 in a million, with the highest risks in the urban areas of Greenville. Please see Appendix C for additional details on the 2011 NATA results.

### *Decide on the Parameters for the Emissions Inventory*

The EPA used the 2011 National Emissions Inventory (NEI) Version 2 for ozone and PM<sub>2.5</sub> and the 2011 NATA data for air toxics emissions, concentrations, and risk. Emissions sources that are known to be of concern, especially if they are likely to be candidates for reductions, will be important to characterize well. For these sources, such data as emissions factors and stack parameters could be further evaluated to assure that the source is well characterized, with particular attention being paid to inventorying all pollutants emitted. Emission summaries for all sources of concern would be valuable, including: (1) pollutant and sector by the 10-county area, by county, and by any seasonal patterns and particular geographic areas of interest; and (2) for particular sources/sectors, a more detailed characterization across pollutants and what controls may be available or planned. For hazardous air pollutants (HAPs), DHEC worked to improve their inventory for the toxic species that are leading the cancer and non-cancer risk in the area and engaged in the state review process for the 2011 NATA in 2014-15.

### *Develop a Control Strategy*

DHEC and EPA held several conference calls to assess the emission inventories information in order to identify those sources affecting potential areas of interest (e.g., monitor locations; populations of concern) within the 10-county area with a focus on those that are in need of control to reduce emissions and associated risks. As a first step, EPA used its Control Strategy Tool (CoST - <http://www.epa.gov/ttn/ecas/cost.htm>). This tool provided a good place to start, having control effectiveness and cost information for many criteria pollutant control measures. This tool electronically connects this control measures information directly to sources listed in the emissions inventory using the Emissions Modeling Framework (EMF - <http://www.ie.unc.edu/cempd/projects/emf/install/>). EPA applied CoST to the 2011 NEI, Version 2. To estimate reductions of HAPs, EPA used a feature of CoST that allows it to calculate co-benefit reductions of volatile organic and metal HAPs from reductions in

the selected VOC and PM criteria pollutants. Controls to HAPs were not applied directly. This information gave DHEC insight on the sources they thought were important to analyze. The decision was to focus the analysis on the non-EGU point source sector which included all area sources, and all non-EGU point sources. EGU point sources were not considered since there are no coal-fired EGUs in the study area, and the current natural gas fired units are well controlled. The goal of analyzing a maximum controls strategy on the non-EGU sector was to see the magnitude of potential reductions. This would then provide context for the local measures that CAU/TATT was considering implementing. While two of the CAU/TATT measures were part of the CoST maximum controls run, they were considered separate from the rest of the measures in that CoST run or "robust strategy." See Appendix D which includes details on the CoST analysis.

In spring 2014, DHEC held meetings with stakeholders representing CAU/TATT to identify available control options for those sources to develop a local control strategy that targets "multi-pollutant" reductions, i.e., those that will focus on the toxics of concern for communities within the 10-county area but maximize those ozone and PM<sub>2.5</sub> precursor emissions reductions to gain health benefits and further reductions in future design values for ozone and PM<sub>2.5</sub>. This process consisted of a general introduction to the project, followed by a brainstorming session. DHEC worked with CAU/TATT and considered various measures to reduce congestion and unnecessary idling (e.g., Right Turn on Red, Roundabouts, Light Synchronization, Anti-Idling Programs). The strategies identified in this session were then prioritized based on several criteria, including: availability of data used to quantify the results of each strategy and the perceived or realized support for each initiative in the CAU/TATT area. Three particular control strategies of interest to South Carolina emerged.

DHEC and the EPA held several discussions on potential local CAU/TATT measures. While reductions from these measures may not be at a scale that would make a substantial difference in a regional inventory, local measures are still important in the overall scheme of multi-pollutant risk-based planning. See Appendix B for more details. Three local CAU/TATT strategies were analyzed to assess their potential effectiveness: new gas stoves and gas logs, open-burning curtailment and anti-idling. Two of the local measures were included in the CoST maximum control (new gas stoves and gas logs and open burning curtailment) and were part of the resulting control strategy CoST run, but were not considered part of the robust strategy. DHEC analyzed the anti-idling measure separately.

It is important to consider seasonality when choosing control measures. In general, areas in nonattainment of an ozone NAAQS may only require control measures to be operated during certain times of the year when ozone is highest. This will affect the design of a control measure, how it will normally operate during a typical year, capital and operation and maintenance costs. Another example is PM control of heating devices such as wood stoves. Most PM problems from wood smoke occur in the winter as wood stoves are used much more during that season than the rest of the year. In most cases, the cost of controls will be less if allowed to idle, but there will be costs to reactivate these controls. For controls that are inherently seasonal such as wood stove controls, most of the costs related to seasonality are built in to the CoST control measures.

### *Process Emissions for Modeling*

A "base case" 2011 emissions inventory was provided by the EPA to DHEC. A "test case" emissions inventory which took into account the reductions identified with CoST was also provided. The Sparse

Matrix Operator Kernel Emissions (SMOKE) was used to process and merge these emissions inventories for each source category (e.g., onroad mobile, nonpoint, point, etc.) into the gridded, hourly, speciation emissions needed for an air quality model. Note that most onroad mobile sector control strategies would require the Motor Vehicle Emissions Simulator (MOVES) to be run to develop emission factors, which are combined with activity data by SMOKE to provide air quality model-ready emissions.

### Conduct Air Quality Modeling

The modeled predictions of air quality changes are data that can be used to gauge the success of a given control strategy. These assessments are essential for predicting the effects on local and regional air quality, attainment of NAAQS standards, and risk and exposure. EPA recommended the CMAQ photochemical model (Community Multiscale Air Quality Model - [www.cmaq-model.org/](http://www.cmaq-model.org/)) at a horizontal scale of 12x12 km for predicting changes in ozone and PM<sub>2.5</sub> concentrations.

EPA and DHEC decided that a "brute-force" model run comparing the base case emissions inventory and a test case emissions inventory would be most appropriate for this project. The 2011 control strategy test case included the aforementioned robust strategy and the three local CAU/TATT strategies (new gas stoves and gas logs and open burning curtailment included in the CoST run; anti-idling was analyzed by DHEC separately). EPA provided base case model-ready emissions files at a grid resolution of 12km, test case point and area source emission files, and boundary condition files. This provided the needed source emissions and characteristics for a refined air quality photochemical modeling "brute-force" exercise. DHEC conducted the CMAQ modeling for this project, and the results are presented below in Section 3. Given the lack of financial resources for this project, we were unable to run CMAQ at a finer grid resolution or model a future year, though this could be useful. In addition, ideally, we would have included air toxics in the CMAQ model run. We also would have used CMAQ combined with HAP dispersion modeling results from the AERMOD ([http://www.epa.gov/ttn/scram/dispersion\\_prefrec.htm#aermod](http://www.epa.gov/ttn/scram/dispersion_prefrec.htm#aermod)) dispersion model to provide modeled estimates at the census tract for air toxics. See results below in Section II and Appendix E for more details.

### Assess Risk from Air Toxics

To predict the effect of the proposed emissions reductions on air toxic risks, we started with the 2011 NATA county level risks for each of the CAU/TATT counties. We assumed that a reduction in emissions would result in a similar reduction in risk for a given pollutant. Because the inventory used for NATA (2011 NEI) and that developed for the CAU/TATT reduction effort are not the same, we could not directly apply the tonnage of reductions to the NATA analysis. Instead, we applied the percentage reductions from the CAU/TATT inventory to NATA point and nonpoint risk results on a pollutant by pollutant basis. There were no estimated emissions reductions from other source types, so there were no estimated risk reductions from those. This approach assumes that reductions are equal across all NATA point and nonpoint source categories. Nevertheless, we feel this approach will provide an approximate estimate of potential reductions in risk associated with the proposed emissions reductions. See results below in Section II and Appendix C for more details.

### Assess Ozone and PM<sub>2.5</sub> Benefits

South Carolina DHEC and EPA staff worked collaboratively to estimate the human health benefits of improving ozone and PM<sub>2.5</sub> air quality projected to result from implementing the robust strategy as well

as the three local CAU/TATT strategies (new gas stoves, anti-idling and open burning curtailment. We applied the environmental Benefits Mapping and Analysis Program—Community Edition (BenMAP-CE), <http://www.epa.gov/benmap>, to assess the number and economic value of the avoided PM<sub>2.5</sub> and ozone-related health impacts. Calculating the health impacts required four key sources of data described in Table I below.

**Table I: Key Data Inputs for BenMAP-CE Used to Estimate Avoided Health Impacts**

Data Input	Source
Air quality changes	DHEC modeled PM <sub>2.5</sub> and ozone changes
Population counts	U.S. Census data projected to the year 2011
Risk coefficients	Concentration-response relationships from U.S. air pollution epidemiological studies
Baseline rates of death and disease	Centers for Disease Control and Prevention-provided death rates and Healthcare Cost and Utilization Program provided hospital visit rates for all other areas

After surveying the epidemiological literature, EPA determined that there were no epidemiological studies well matched to the 10-county area, and so the project team applied the default studies the Agency uses for its national-scale benefits assessments to quantify the changes in PM and ozone-related premature deaths and illnesses (USEPA, 2009; USEPA, 2013). See results below in Section II and Appendix F for more details.

## Section II: Results & Recommended Next Steps

### Results

A suite of area and point source control measure were identified using the CoST. The control case included the following reductions:

- **For select non-point (area) sources:** The major reduction control technologies were: Low NOx Burners (1997 AQMD, and RACT to 25 TPY); conversion to low NOx burners in water and space heaters; open burning curtailment program; conversion of wood fireplaces and stoves to gas; and reformulation (Ozone Transport Commission or OTC Rule, Phase II, and process modification). Other controls include application of Control Technology Guidelines (CTGs); low pressure/vacuum or LPV relief valve use; and solvent utilization changes.
- **For select point sources:** The major reduction technologies were: low emission combustion; dry injection/fabric filter system utilization; wet scrubber installation; and permanent total enclosure installation. Other controls included low NOx burner conversion; Selective Catalytic Reduction (SCR); add-on controls, work practices and material reformulation/substitution; and solvent recovery system installation.

It is important to note that CoST applies controls that are mostly "end of pipe." Opportunities for emissions reductions from emerging renewable energy, energy efficiency measures, and fuel switching (for example, to natural gas), and additional local measures have not been considered. Furthermore, mobile source controls were not applied in CoST. A separate strategy to address anti-idling was supplied by DHEC. Also, strategies for EGU reductions were not actively sought in CoST since there are no coal fired EGUs in the region and current natural gas units are well controlled.

Criteria pollutant reductions were quantified using the 2011 Modeling Platform in CMAQ at a horizontal scale of 12 x 12 km on a domain 100 x 100 cell grid centered around the Upstate. A "brute-force" emission reduction evaluation method was used, comparing base case and test case model runs. Boundary conditions for the test domain were derived from the EPA's 2011 12 km NATA model runs.

CoST results indicated that the following reductions in each pollutant would be expected.

NO <sub>x</sub>	-	1587 tons
PM <sub>2.5</sub>	-	222 tons
SO <sub>2</sub>	-	766.32 tons
VOCs	-	2727 tons

The total cost of controls was estimated at \$20,000,000.

### *PM Reductions*

The following results show the modeled PM<sub>2.5</sub> reductions that took place between base case and test case strategies at the PM<sub>2.5</sub> monitors in the Upstate. PM<sub>2.5</sub> reductions are at around a 2 percent (%) reduction at the monitors for the annual standard (Table 2). Temporal reductions are much higher than average in colder months (quarters 1 and 4)(Table 3). Speciated reductions show higher reductions in organic carbon (Table 4). These results, taken together indicate that the wood stove conversion to natural gas reduction strategy may be effective at reducing annual PM<sub>2.5</sub> emissions.

**Table 2: PM<sub>2.5</sub> Annual Standard Reductions at Upstate Monitors**

Monitor ID	Base DV	Future DV	% Reduction
450450009	10.6	10.39	1.9
450450015	10.9	10.64	2.4

**Table 3: Quarter I and Quarter 4 Reductions in PM<sub>2.5</sub> Concentrations**

Monitor ID	Date	Base DV	Future DV	% Reduction
450450015	Q4	10.44	9.944	4.8
450450015	Q1	10.15	9.701	4.4
450450009	Q4	9.929	9.5	4.3
450450009	Q1	9.551	9.199	3.7

**Table 4: Speciated PM<sub>2.5</sub> Relative Reduction Factors**

Crustal	Elemental Carbon	NH4	Organic Carbon	SO4	NO3	Water	Salt
0.999	0.9851	0.9972	0.9615	0.9982	0.9764	0.9985	0.9955
0.9991	0.9843	0.9971	0.9558	0.9982	0.9753	0.9986	0.9944

#### Ozone Reductions

The following results show the modeled ozone reductions between base case and test case that took place at ozone monitors in the Upstate area (Table5). While maximum daily ozone reductions can be as high as approximately 2 ppb. Design value reductions are typically less than 1 ppb at the monitors (less than a 1 percent (%) reduction).

**Table 5: Modeled Design Value Ozone Reductions at Upstate Monitors**

Monitor_ID	Monitor_Name	Base_DV	Future_DV	% Reduction
450010001	Due West	62	61.7	0.48
450070005	Big Creek	70	69.8	0.29
450210002	Cowpens	67.3	67.2	0.15
450450016	Hillcrest	68	67.3	1.03
450451003	Famoda Farms	65.3	65.2	0.15
450730001	Long Creek	64.5	64.4	0.16
450770002	Clemson	69.7	69.5	0.29
450770003	Wolf Creek	69	68.8	0.29
450830009	North Spartanburg	73.7	73.3	0.54

*Estimated Benefits of the Air Quality Management Plan*

The control strategy listed above was paired with health incidence and mortality data for the CAU/TATT area for 2010 – 2012. Changes in air quality were modeled against health impact functions available in the BenMAP program and were tabulated to estimate the number of avoided deaths and avoided number of non-mortality related end-points. BenMAP was then used to estimate the valuation of these avoided health endpoints for the entire modeling domain and then the CAU/TATT area. Tables 6 - 7 shows the number of avoided deaths and avoided non-mortality endpoints for the CAU/TATT area. Two studies are typically used to estimate the range of monetary savings from these avoided health endpoints. Table 8 presents a statistical estimate of the monetary benefits of these avoided health endpoints.

**Table 6: Estimated Incidence of Avoided PM<sub>2.5</sub> and Ozone-Related Premature Deaths and Illnesses**

Author	<i>Impacts as summed across regions</i>		
	10 TOP	SC	Interstate
Premature Mortality (30-99)	10	11	16
Krewski et al. (2009)	(7.5-13)	(7.8-14)	(12-20)
Premature Mortality (25-99)	23	24	37
Lepeule et al. (2012)	(13-33)	(14-35)	(21-52)
Premature Mortality (<1)	0.030	0.032	0.046
Woodruff et al. (2006)	(.015-.045)	(.015-.048)	(.022-.069)
All Respiratory Hospital Admissions (65-99)	1.60	1.70	2.70
Zanobetti et al. (2009), Kloog et al. (2012)	(-.70-3.2)	(-.074-3.3)	(-1.1-5.1)
All Cardiovascular Hospital Admissions (65-99)	1.9	2.0	3.0
Zanobetti et al. (2009), Bell et al. 2008, Peng et al. (2009)	(.88-3.7)	(.92-3.9)	(1.4-5.7)
Emergency Rooms Visits for Asthma (0-99)	5.5	5.8	8.6
Glad et al. Mar et al. Slaughter et al. (2012)	(-.95-11)	(-1.0-11)	(-1.5-17)
Acute Bronchitis (8-12)	14	15	24
Dockery et al. (2006)	(-.52-29)	(-.55-31)	(-.85-48)
Lower Respiratory Symptoms (7-14)	180	194	300
Schwartz and Neas (2000)	(88-280)	(93-290)	(140-450)
Upper Respiratory Symptoms (9-11)	260	280	430
Pope et al. (1991)	(82-440)	(87-470)	(130-720)

Asthma Exacerbation (6-18)	271	290	440
<i>Ostro et al. (2001), Mar et al. (2004)</i>	(34-540)	(36-570)	(55-880)
Lost Work Days (18-64)	1200	1300	2000
<i>Ostro (1987)</i>	(1100- 1400)	(1100- 1400)	(1700- 2200)
Minor Restricted Activity Days	7200	7600	12000
<i>Ostro and Rothschild (1989)</i>	(6100- 8400)	(6500- 8800)	(9900- 14000)

**Table 7: Estimated Dollar Values of Avoided PM<sub>2.5</sub> and Ozone-Related Premature Deaths and Illnesses (millions of 2011\$, discounted at 3 percent)**

Health Effect	Pollutant	<i>Impacts as summed across region</i>		
		Ten at the Top	South Carolina	Interstate Domain
Premature Mortality (Krewski et al. 2009 & Bell et al. 2004 (Ozone))	PM <sub>2.5</sub> & O <sub>3</sub>	\$100 (9.4-280)	\$110 (9.9-300)	\$160 (15-450)
Premature Mortality (Lepeule et al. 2012 & Levy et al. 2005 (Ozone))	PM <sub>2.5</sub> & O <sub>3</sub>	\$230 (20-650)	\$240 (21-680)	\$370 (32-1,000)
Non-fatal heart attacks (Peters et al. v1)	PM <sub>2.5</sub>	\$1.3 (0.23-3.3)	\$1.4 (0.24-3.5)	\$2.1 (0.37-5.3)
Hospital admissions - respiratory (Zanobetti et al Kloog et al. & Katsouyanni et al. (Ozone))	PM <sub>2.5</sub> & O <sub>3</sub>	\$0.057 (-0.024-0.11)	\$0.062 (-0.025-0.12)	\$0.1 (-0.040-0.20)
Hospital admissions - cardiovascular (Zanobetti et al Bell et al. Peng et al.)	PM <sub>2.5</sub>	\$0.073 (0.0041-0.017)	\$0.076 (0.035-0.15)	\$0.11 (0.051-0.22)
Emergency room visits for asthma (Glad et al. Mar et al. Slaughter et al. & Sarnat et al. Peel et al.)	PM <sub>2.5</sub>	\$0.0027 (-0.00043-0.0061)	\$0.0029 (-0.00044-0.0068)	\$0.0047 (-0.00062-0.011)
Glad et al. Wilson et al. Mar and Koenig Ito et al. (Ozone))				

Acute bronchitis		\$0.007	\$0.0073	\$0.011
(Dockery et al.)	PM <sub>2.5</sub>	(-0.00034- 0.019)	(-0.00036- 0.020)	(-0.00055- 0.031)
Lower respiratory symptoms		\$0.0039	\$0.0041	\$0.0063
(Schwartz and Neas)	PM <sub>2.5</sub>	(0.0013- 0.0078)	(0.0013- 0.0083)	(0.0021- 0.013)
Upper respiratory symptoms		\$0.0088	\$0.0092	\$0.014
(Pope et al.)	PM <sub>2.5</sub>	(0.0019- 0.021)	(0.0020- 0.023)	(0.0031- 0.035)
Asthma exacerbation		\$0.028	\$0.034	\$0.069
(Ostro et al. Mar et al.)	PM <sub>2.5</sub>	(0.0099- 0.081)	(-0.014- 0.097)	(-0.036- 0.20)
Lost work days		\$0.19	\$0.18	\$0.32
(Ostro)	PM <sub>2.5</sub>	(0.16- 0.21)	(0.16- 0.21)	(0.28- 0.37)
Minor restricted-activity days	PM <sub>2.5</sub> & O <sub>3</sub>	\$0.53	\$0.57	\$0.93
(Ostro and Rothschild)		(0.28- 0.81)	(0.29- 0.88)	(0.47- 0.15)

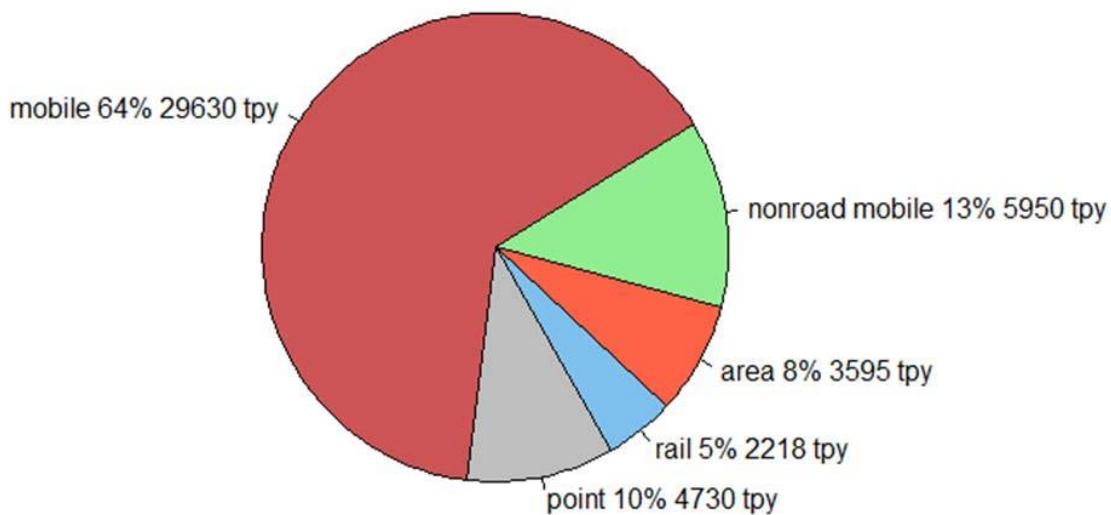
**Table 8: Predicted Air Quality Change, Estimated Economic Value of Avoided Deaths and Illnesses and Net Benefits**

Total Air Quality Benefits	PM <sub>2.5</sub> ( $\mu\text{g m}^{-3}$ )	Ozone (ppb)
Maximum Air Quality Change at Zip Code (PM: 29601; Ozone: 29374)	0.388	0.202
Maximum Air Quality Change at a Monitor (PM: 450450015; Ozone: 450830009)	0.260	0.400
Maximum Air Quality Change at a County (PM: Greenville; Ozone: Spartanburg)	0.251	0.106
TATT Change in Population-Weighted Exposure	0.131	0.068
<hr/>		
<b>Cost-Benefits Analysis (in Millions of 2010 Dollars)</b>		
TATT Cost (in 2010 Dollars) (PM: per $\mu\text{g m}^{-3}$ reduced; Ozone: per ppb reduced)	\$150	\$290
Total Benefits of Avoided Mortality and Morbidity (PM: Krewski-Lepeule; Ozone: Bell-Levy)	\$99-220	\$3.1-4.4
Total Control Strategy Cost	\$20	
Net Total Benefits	\$82-210	
Benefit-Cost Ratio	4.1-10	

The small reductions in ozone concentrations are likely due to the minor contributions of ozone forming NOx from stationary sources in the area. A majority of NOx emissions in the Upstate area are from mobile sources. Based on the 2011 NEI, these sources were responsible for approximately 77 percent of all NOx emission in the 10 Upstate counties (Figure 2)

**Figure 2:**

## Upstate NOx Emissions by Source Category



---

This data highlights the importance of working with local governments to encourage transportation planning which reduces mobile source emissions to meet our state's air quality goals.

### BenMAP Risk Assessment

Using upstate-specific epidemiological data, partners used the Environmental Benefits Mapping and Analysis Program – Community Edition (BenMap–CE) to examine population risk exposure between base case and test case scenarios. The expected risk reduction for each county in the CAU/TATT is characterized below (Table 9).

*Air Toxics Reduction's Effect on Cancer Rates.*

Cancer risk reductions from the air pollution control strategies were quantified using the EPA's 2011 NATA data<sup>3</sup>. These reductions are as follows:

---

<sup>3</sup> <http://www.epa.gov/national-air-toxics-assessment>

**Table 9:**

County	Cancer Risk (in a million)	
	2011 NATA Risk	Expected Risk Reduction
Abbeville	44	0.01
Anderson	46	2
Cherokee	45	1
Greenville	48	3
Greenwood	47	0.01
Laurens	45	0.02
Oconee	42	0.01
Pickens	43	0.03
Spartanburg	48	3
Union	47	0.01

### Overarching Conclusions

**Criteria Pollutants:** Implementing local control strategies in an area with a mix of sources is an important component of successful air quality management programs. Control strategies that are implemented based on analyzing air quality and health data together result in a reduction of risk to certain populations in the area. As a result, there are cost savings and air quality improvement to attain and maintain clean air quality and health benefits.

**Toxics:** The highest air toxics reductions are about 3 percent in both Greenville and Spartanburg counties. It's important to note that this analysis does not include potential reductions from "anti-idling" efforts. The NATA model results for mobile sources do not segregate out the idling emissions, thus we cannot estimate that portion of the risk that would be reduced. However, we would expect further risk reductions from this program. Because a majority of the above risks are from secondary formed pollutants (mainly formaldehyde), reduction efforts to reduce precursors such as nitrogen oxides and other criteria pollutants will have a co-benefit in reducing risks from air toxics.

**Establishing partnerships:** Bringing together local, state and federal partners to address important multi-pollutant air pollution challenges results in improvements to air quality and public health. It is important to include local organizations into this type of project because they are able to provide perspective on what will and will not work in their communities to reduce emissions in an effective manner.

## Key Take Away/Lessons Learned

Throughout this process the partners for this project have learned a great deal. Below are some key points and lessons learned from each of the primary partner's perspectives:

### **DHEC**

- Environmental justice (EJ) concerns are and will continue to be a focus in the future. Not only has the federal government and the EPA demonstrated this via Executive Orders, policy initiatives, and major rulemaking; the states too are experiencing this in increased involvement with the permitting process. DHEC has always prided itself on striving to work with and encourage EJ community leaders to have a seat at the table and will continue to do this into the future. In addition, SC has a rich history in very active EJ community groups and leaders. Together DHEC and these EJ communities and leaders have worked together on several projects that have resulted in successfully mitigating environmental harms to at-risk communities across the state. The results of this project need to be shared with local EJ community leaders so that DHEC can continue its work engaging and educating local EJ communities and leaders to reduce environmental and health risks.
- This project has been instrumental in developing DHEC's understanding of EPA's BenMAP-CE. Understanding risk communication is becoming increasingly important in developing relationships with local community representatives. Enhancing the Department's awareness and understanding or tools such as BenMAP will enhance the Department's ability to respond to questions and concerns. In addition, this experience will also aid in the Department's own understanding of NAAQS development at the national level and how tools like BenMAP are used to make policy decisions.
- Local, voluntary efforts at developing collaborative approaches to problem solving are an effective and necessary step in transition to multi-pollutant air quality management. As evidenced by Early Action Compacts, EPA Advance, and local air quality coalitions, time and time again DHEC has experienced the benefit of involving all interested parties in problem solving. While these efforts are often time and resource intensive, the benefits are great. Experience has taught us the value that local community perspective and expertise can play in helping to make decisions that serve to promote and protect the health of SC citizens and the environment.

### **EPA**

Addressing air pollution challenges through a collaborative partnership is crucial for any successful air quality management approach. This project demonstrated how a multi-disciplinary team comprised of local, state and federal air quality modelers, health scientists, policy analysts and others could develop a cost-effective approach to managing air quality. The EPA found it very rewarding to establish and maintain relationships between parties who shared a common goal. Through this project we were able to establish a framework that will help focus future similar multi-pollutant risk-based analyses (see Section III). The framework and this report provide a structure for conducting local risk-based analyses with the goal of identifying specific actions that can reduce emissions, help areas improve air quality and

continue to meet the NAAQS while focusing on cost-effective actions that would provide additional public health protection for communities.

The results of this project demonstrate that improving air quality in areas already attaining the NAAQS will yield significant health benefits – and thus should encourage many areas to reduce emissions, either on their own or in the context of the Ozone and PM Advance<sup>4</sup> programs, which promote emission reductions in attainment areas. The results of this project will inform and help other attainment areas (1) assess actions to keep ozone and particulate matter levels below the level of the NAAQS to ensure continued health protection for their citizens, (2) better position areas to remain in attainment, and (3) help areas efficiently direct available resources toward a more cost-effective strategy. In addition, reductions in attainment areas may also help reduce the transport of air pollution to downwind nonattainment areas. Nonattainment areas should also benefit from reviewing these results, which demonstrate that a risk-based approach to addressing air pollution and population exposure in a given local area is important to ensuring the public health protection of its citizens.

Key lessons learned include: staff involvement at all levels and throughout the various agencies and stakeholder groups made continued progress possible; it is important to gather local health and population data to effectively analyze local control strategies; and localized emissions reductions are still important even in areas that are in attainment. While the air quality benefits of local strategies may not show an air quality benefit when a broader-scale attainment demonstration for a SIP is conducted, this risk-based analysis demonstrates that local regulatory and voluntary control measures are an important part of an air quality management program. By using a multi-pollutant approach, some strategies with multi-pollutant benefits might not have been considered if pollutants had been examined one at a time; a strategy that might seem too expensive to reduce a single pollutant or too difficult to implement might emerge as cost-effective once all the cobenefits are factored into the analysis.

In addition, EPA benefitted from this working relationship beyond providing analytical support and gaining insight from the results of the analysis. The EPA staff also benefited from feedback from South Carolina on the CoST database and how to reflect local conditions in the tool with more accuracy and from the opportunity to deliver BenMAP-CE training to South Carolina staff, which prepared them for subsequent domestic and international training classes. Through the collaborative working relationship, EPA was able to train a new staff member in how to run the tool with location-specific health data. Working together in this way helped EPA to better understand the strengths and limitations to the BenMAP-CE program, and improved the ability of staff to provide similar assistance to other analysts throughout the U.S.

---

<sup>4</sup> The EPA continues to encourage state and local air agencies to join the Ozone and PM Advance program (<http://www3.epa.gov/ozoneadvance/basic.html>). This project started within the context of that program. The Advance program promotes local actions to reduce ozone and particulate matter and encourages states, tribes and local governments to take proactive steps to keep their air clean. EPA is hopeful that the information gathered during this process will help facilitate additional local emissions reductions and health benefits in the CAU/TATT Region of South Carolina, and serves as an example of localities nationwide.

While there are opportunities for continued analyses in the Upstate area that would provide more insight into the most cost-effective multi-pollutant control strategy, the results of this analysis demonstrate that multi-pollutant control programs can save money and time, and achieve significant health, environmental and economic benefits while reducing costs and burdens on sources of air pollution. An integrated air quality control strategy that reduces multiple pollutants can help ensure that reductions will be efficiently achieved while producing the greatest overall air quality and public health benefits.

## **CAU/TATT**

CAU/TATT supports a multi-pollutant perspective. CAU/TATT is interested at looking at how actions impact the total sphere of pollutants, instead of looking at them individually and in ways to easily communicate this information to the general public.

CAU/TATT also supports the development of tools and resources for local coalitions aimed at allowing them to easily understand the effect that certain activities will have on air quality and various pollutant levels. This analysis is helpful in determining whether programs (like Breath Better at Schools) and their financial costs are supported by the likely outcomes. CAU/TATT also supports the need for better data tracking tools to determine whether programs have measurable impacts.

Finally, CAU/TATT is interested in learning more about the connection between air quality and health, especially asthma. As pollution levels continue to decrease, they believe that it becomes increasingly important to understand how/why certain health outcomes fail to show improvement. CAU/TATT feels that learning what potential contributing factors can be understood and mitigated is an important next step.

### Recommended Next Steps

In addition to evaluating and summarizing these results, the DHEC and EPA will seek future opportunities to share these results with other interested parties. These opportunities might include seeking publication of these results in academic journals, poster and oral presentations at local and national conferences, etc. In addition to focusing on the publication of these results the following have been identified potential next steps to include options related to future modeling efforts and the development of tools to enhance similar type pilot projects.

#### Modeling:

- A potential 4 km CMAQ or CAMx model run to better assess community level PM<sub>2.5</sub> and ozone reductions. This resolution may also be appropriate for showing the benefits of an anti-idling program.

Future year baseline and projected model runs to assess the impact of several planned and on-the-way rules and regulations (utility MACT, NESHAPs, NSPS, 2015 Ozone NAAQS, etc.). This will provide directional information on the effect of regional and national controls on the Upstate area. In addition, as deemed necessary as part of additional air quality modeling to be conducted to assess the impact of the local control strategy, any local enhancements to the emissions inventory to create the future-year base inventory will need to be determined by DHEC. This could mean collecting data on any closings or shut-downs of industrial sources, mobile fleet turnover, and installation of potential control measures and determining which to include in the future base year projection.

- A more targeted control strategy to include options for potential implementation (e.g., local measures for the mobile source sector) to determine effect.
- Periodic rerun of the modeling and BenMAP to see how the control strategy responds to future improvements in emissions inventory and modeling.
- To estimate air toxics exposure concentrations for a given risk assessment, we could use the CMAQ/AERMOD hybrid approach that uses the grid cell modeled concentrations from CMAQ with the finer-scale gradient provided by AERMOD, which is the methodology used in the Detroit project. The hybrid ambient pollutant concentrations would be used as surrogates for the inhalation exposure concentrations of the populations in the study locations. The default assumption in this approach is that the population of interest is breathing outdoor air continuously, which overestimates exposure because people are not always at the study location due to their daily activities, and because indoor air concentrations are expected to be the same or lower than the outdoor concentrations (when the indoor concentrations are produced solely by inflow from outside air). The HEM-3 model ([http://www.epa.gov/ttn/fera/fera\\_download.html](http://www.epa.gov/ttn/fera/fera_download.html)), which was used in the Detroit project, is based on this approach.

**Planning:**

- Both DHEC and CAU/TATT are eager to share the results of this pilot with other local air quality coalitions. Sharing these results is important on many levels, including, but not limited to increasing understanding and participation at the local level, while supporting/facilitating collaboration with state and local community and environmental justice leaders as well as business and educational institutions to come together to develop tailored approaches to reducing pollution and protecting health. As a result of this study, DHEC will be conducting CAMx source apportionment modeling to quantify pollution contributions from mobile sources in different regions of the state and using the results to highlight the importance of mobile source emissions reduction programs. Providing this information to local governments and air quality coalitions will hopefully lead to transportation planning and policy decisions which reduce mobile source emissions and improve air quality.

**Tools & Expansion:**

- In addition, DHEC is interested in working with EPA and academia to develop tools that might better analyze the effect of mobile source controls, like anti-idling campaigns and environmentally focused transportation planning.
- Processes to communicate the findings of a multi-pollutant control strategy to the public.
- Finally, DHEC has also considered whether this pilot project could/should be replicated in another area of the state – perhaps the Central Midlands area around Columbia or in the coastal region near Charleston.
- Partner with academia to encourage future epidemiological studies that represent either or both smaller urban and/or regional rural research areas to produce results that can be used to replace national scale benefit assessments.

## Section III: Project Template

### *Multi-Pollutant Analysis Template*

The following list provides the steps and key questions to explore in completing a project similar to the USEPA-SC project. The timing specified is an amount of time we suggest if your project plan has a 15-month timeframe. Each project's timeframe will vary depending on the goal, staff and resources available.

**Step 1:** The initiating agency should determine the project's scope and objectives and convene appropriate partners (federal, state, local, industry, communities, NGOs) to get agreement on goals and the project scope. What geographical area do you want to study? Nonattainment area status, jurisdictional boundaries in your state and/or local government, population density, mix of sources and current air quality issues are important factors to consider. This includes considering air toxics, greenhouse gases, transportation, energy and land-use planning and environmental justice considerations. What are the year(s) of study and the team's data acquisition needs and requirements? It is also helpful to develop a conceptual model (e.g., workplan) that includes a description of the area of study and the problems and issues to address.

*Timing: 1-2 months*

**Step 2:** Acquire meteorological, emissions and NATA data

*Timing: 1 month*

**Step 3:** Develop control strategy(ies) to analyze and compare (consider all pollutants of interest, geographic area, and potential non-end-of-pipe measures that could be applied that are not in CoST). An end-of-pipe measure is typically a control that is applied to a unit or process to reduce its output of emissions.

*Timing: 2-3 months*

**Step 4:** Run CoST and calculate the cost per ton of any measures of interest for which CoST may not have information, such as local measures and non-end-of-pipe measures (energy efficiency, renewable energy and fuel switching). Evaluate the cost-effectiveness of the strategy(ies). Steps 3 and 4 can be iterative.

*Timing: 1 month*

**Step 5:** Process emissions for modeling and run CMAQ to develop base case and test case (and future year) runs & review results. For a more robust air toxics analysis, also run AERMOD and perform a CMAQ/AERMOD hybrid analysis. The CMAQ/AERMOD hybrid air quality model combines the results of a chemical transport model (CMAQ) and a Gaussian dispersion model (AERMOD) to take advantage of the chemistry and long-range transport provided by CMAQ and the local-scale gradient provided by AERMOD. If this approach is taken for air toxics, step 6 is not needed.

-determine if the modeling results meet the objectives; if not, additional modeling and/or other analyses may be warranted

*Timing: 1-2 months*

**Step 6:** Adjust NATA risk results using local emission reductions

*Timing: Concurrently with CMAQ run*

**Step 6:** Acquire health data

*Timing: 2-4 months to occur simultaneously with steps 2-5*

**Step 7:** Run BenMAP-CE. Incorporate results of CMAQ test case and future year and health data in BenMAP

If multiple strategies were analyzed, compare the air quality and BenMAP-CE results with the cost of controls to determine the most cost-effective control strategy. Take into consideration cost and benefits of the strategy options. In particular, consider both the magnitude and distribution of benefits, assessing the extent to which air quality benefits occur among susceptible and vulnerable subgroups. Not all benefits are quantifiable (e.g., environmental justice and ecosystems services). Such unquantifiable benefits should be factored into the strategy selection if applicable.

*Timing: 2 months*

**Step 8:** Review results, draw conclusions, and write a report

*Timing: 2 months*

**Step 9:** Implement the selected strategy.

*Timing: As appropriate for each state and local agency's adoption process*

## Appendix A: Original Project Description -- November 2013

### Overview

The U.S. Environmental Protection Agency (EPA) and the State of South Carolina's Department of Health and Environmental Control (DHEC) share an interest in exploring multi-pollutant analysis and planning as a means to improve air quality effectively, and as a way to make most efficient use of available resources.

EPA's Detroit multi-pollutant pilot project provides a framework for analyzing air quality management programs capable of realizing multiple policy goals. In particular, the project demonstrates that it is possible to achieve air quality improvements among an array of pollutants while also reducing air pollution risk to both the general population and those most prone to air pollution-related health impacts. Two key factors contributed to the success of the project: (1) careful planning that involved profiling the Detroit metropolitan area, rigorously formulating the overall "air quality problem" to be addressed, and identifying the data and tools to be utilized; and (2) EPA and the Michigan Department of Environmental Quality (DEQ) collaborated closely, ensuring that all parties understood the project goals and were willing to share data. In May 2013, EPA approached the DHEC with the opportunity to work together to develop a multi-pollutant analysis modeled after the Detroit pilot.

EPA, DHEC, and South Carolina's Upstate Region (Upstate) which includes the nonprofit group Clean Air Upstate Coalition (CAU)/Ten at the Top (TATT) are each interested in collaborating to develop and use a multi-pollutant, risk-based analysis for the region which builds upon the lessons learned in Detroit while addressing air quality issues unique to the Upstate.

EPA's and DHEC's goals for this project are:

- (1) identify local emission reduction measures for the Upstate that address multiple pollutants, that are harmonized with existing or planned federal/state/local measures,<sup>5</sup> that are quantifiable, and whose implementation by DHEC and/or Upstate is achievable;
- (2) maintain compliance with the National Ambient Air Quality Standards (NAAQS);
- (3) demonstrate that the selected strategy(ies) can reduce population risk from exposure to ozone, PM<sub>2.5</sub>, and selected air toxics in the Upstate and can reduce exposure among populations at greatest level of baseline risk;
- (4) transition to a multi-pollutant air quality management strategy; and
- (5) foster a spirit of collaboration among EPA, the Upstate, and DHEC that highlights the importance of a coalition approach.

EPA, DHEC, and the Upstate will work together to develop a multi-pollutant risk-based analysis. The conclusions will inform choices that DHEC and the Upstate make as to which specific strategies may be

---

<sup>5</sup> See the 2004 National Academy of Sciences (NAS) report describing the elements of a multi-pollutant air quality management plan (AQMP).

implemented and documented as a supplement to the combined ozone/fine particulate matter (PM<sub>2.5</sub>) Advance plan (“path forward”) for South Carolina.<sup>6</sup>

Below we summarize the questions to be answered by this analysis; the tools and data available; the project schedule; and the roles and responsibilities of EPA, DHEC, and the Upstate. Because we expect to refine each component of the analytical plan iteratively, this document will evolve over time.

### *Demographic*

South Carolina’s Upstate is comprised of Abbeville, Anderson, Cherokee, Greenville, Greenwood, Laurens, Oconee, Pickens, Spartanburg, and Union counties. Clean Air Upstate Coalition (CAU)/Ten at the Top (TATT) is a nonprofit group founded in 2005 which is focused on fostering a spirit of cooperation and collaboration among Upstate public, private, and nonprofit leaders to build a comprehensive picture of what is important to people in the Upstate as they look at the future. CAU/TATT has facilitated and coordinated the Upstate Air Quality Advisory Committee and the Clean Air Upstate initiative in an effort to reduce emissions and stay within federal air quality standards with representatives from stakeholder groups across the Upstate (including Upstate Forever, Piedmont Natural Gas, WSPA-TV, BMW Manufacturing, Duke Energy, City of Greenville, and Michelin NA).

### *Air Quality Issues in the Upstate Region*

The Upstate faces a confluence of air quality management challenges.

1. *Current PM<sub>2.5</sub> air quality levels.* Recent air quality data indicate that the Upstate attains the current annual PM<sub>2.5</sub> NAAQS by a narrow margin. Anderson, Greenville, and Spartanburg Counties were designated as unclassifiable for the 1997 PM<sub>2.5</sub> NAAQS (70 FR 944, January 5, 2005) and that designation remains in effect. The Upstate is attaining the 2006 and 2012 PM<sub>2.5</sub> standards for daily and annual PM<sub>2.5</sub>. 2010-2012 design values for PM<sub>2.5</sub> monitors in the Upstate indicate that Greenville has a 10.9 µg/m<sup>3</sup> design value (12 µg/m<sup>3</sup>) for the annual standard and a 23 µg/m<sup>3</sup> (35 µg/m<sup>3</sup>) design value for the 24-hr standard.
2. *Current ozone air quality levels.* With the help of Early Action Compacts<sup>7</sup> and local stakeholder involvement, the Upstate is attaining both the 1997 (0.08 ppm) and 2008 (0.075 ppm) ozone standards but is likely to exceed a more stringent NAAQS (0.070 ppm or less). 2010-2012 design values indicate that Abbeville (.064), Anderson (.073), Greenville (.069), Pickens (.071), and Spartanburg (.075) are all in a range of concern for attaining any future more stringent NAAQS.
3. *Projected ozone and PM<sub>2.5</sub> air quality levels.* Table I provides the 2010-2012 design values for the Upstate counties as well as projected 2020 design values based on EPA’s photochemical modeling used in the Regulatory Impact Analysis (RIA) for the PM<sub>2.5</sub> NAAQS Final Rule (Please note that this modeling used a 2007-based modeling platform with projections from the 2007-2009 DVs and not all monitors operating today were included in the model run. Furthermore, some areas have projected Design Values, but no currently operating monitor due to monitor

<sup>6</sup> [www.epa.gov/ozonepmadvance](http://www.epa.gov/ozonepmadvance)

<sup>7</sup> For more information, see: <http://www.scdhec.gov/environment/baq/EarlyActionPlan/index.asp>.

shutdowns.). Based on EPA's regulatory modeling the Upstate counties realize reductions in their projected design values due to Federal rules that are expected to be in place from now to 2020 including multiple mobile source rules and the Mercury and Air Toxics (MATS) final rule.

**Table I. Current and Projected Design Values for Ozone and PM<sub>2.5</sub>: SC Upstate**

County	2010-2012 Design Value			2020 Projected Design Value		
	Ozone (ppb)	Annual PM <sub>2.5</sub> ( $\mu\text{g}/\text{m}^3$ )	Daily PM <sub>2.5</sub> ( $\mu\text{g}/\text{m}^3$ )	Ozone (ppb)	Annual PM <sub>2.5</sub> ( $\mu\text{g}/\text{m}^3$ )	Daily PM <sub>2.5</sub> ( $\mu\text{g}/\text{m}^3$ )
Abbeville	64			60.5		
Anderson	73			NV <sup>8</sup>		
Cherokee	70			55.4		
Greenville	69	10.9	23		9.86	21.7
Greenwood					8.88	18.4
Laurens						
Oconee	64	NV	NV		6.40	13.4
Pickens	71					
Spartanburg	75	10.7	21	63.1	8.36	17.5
Union				59.5		

4. **Air toxics.** Based on the 2005 National Air Toxic Assessment (NATA) nearly 16,000 tons of air toxics are emitted each year from the Upstate. According to NATA, the average cancer risk in the Upstate associated with inhalation of air toxics is about 46 in a million. A majority of this risk is associated with formaldehyde (56 percent) with benzene (12 percent) and acetaldehyde (10 percent) also key contributing pollutants. Formaldehyde and acetaldehyde are generally formed with photochemical activity along the I-85 corridor in the southeast US, while benzene emissions are associated with mobile traffic along the many interstates in the Upstate. NATA estimates that the nearly 21,000 people who live in the area are exposed to cancer risks greater than 60 in a million, with the highest risks in the urban areas of Greenville and Spartanburg. Please see Attachment I for additional details on the 2005 NATA results.
5. **Environmental Justice.** Previous local-scale analyses suggest that there are pronounced gradients to intra-city air quality—particularly for PM<sub>2.5</sub>. Moreover, the public health literature indicates that certain population groups may be more susceptible to air pollution impacts. This portion of

<sup>8</sup> No value

the analysis will consider how air pollution levels correspond to such population subgroups, and whether policies can specifically target such populations. For example, children, the elderly, and people with respiratory or heart diseases are at higher risk of being vulnerable to the effects of air pollution. Communities that are low-income and/or minority can bear a disproportionate burden of environmental harm and risks. These overburdened communities can have high unemployment, low income, and limited access to healthcare and are often located in urban areas that may have environmental pollution from multiple active or abandoned industrial facilities. In 2008, EPA conducted an evaluation of communities in SC using social demographics, environmental, compliance, and health data to identify areas with disproportionately high and adverse environmental and public health burdens. The evaluation identified several areas in the Upstate that were overburdened. One specific area was in Greenville, SC. DHEC identified and contacted stakeholders to engage in collaborative problem-solving to address air-related issues. The stakeholder group developed and implemented an education and outreach plan for this area. These efforts not only helped to reduce air emissions, but also established stronger working relationships with the stakeholders in that area.

6. Key *multi-pollutant* sources. EPA and DHEC will use the 2005 NATA information and the CoST tool to provide information regarding which sources may be particularly important contributors to the emissions in the area and offer potential co-control opportunities from a multi-pollutant perspective. The NATA and CoST information will assist DHEC in identifying the specific sources (e.g., mobile, inland port, prescribed burns) that will be added to this section and that can become the focus of DHEC and the Upstate's efforts to design a local control strategy.

Given the nature of the air quality problem in the Upstate, the main objectives of a “multi-pollutant, risk-based” control strategy for the local area might be:

- Attainment and maintenance of the recently revised PM<sub>2.5</sub> NAAQS and current and potentially more stringent ozone NAAQS;
- Lowering emissions, ambient levels, and exposures to key air toxics of concern; and
- Maximizing health benefits among those populations at greatest risk of air pollution-related health impacts.

### *Tools and Data*

This section describes some of the technical tools and data that might be needed to develop a multi-pollutant, risk-based control strategy. The information offered below is mostly based on what was done for the Detroit project and the lessons learned from it.

#### Emissions Inventory and Baseline and Regional Modeling

Having an emissions inventory that can support a multi-pollutant, risk-based analysis is important. For this project, we will use the 2011 National Emissions Inventory (NEI) for ozone and PM<sub>2.5</sub> and the 2011 NATA data for air toxics emissions, concentrations, and risk as it becomes available. Below is a list of additional items to consider when determining where to make adjustments to the current emissions inventory.

- *Source characteristics:* Emissions sources that are known to be of concern, especially if they are likely to be candidates for reductions, will be important to characterize well. For these sources, such data as emissions factors and stack parameters could be further evaluated to assure that the source is well characterized, with particular attention being paid to inventorying all pollutants emitted. Emission summaries for all sources of concern would be valuable, including: (1) pollutant and sector by the 10-county area, by county, and by any seasonal patterns and particular geographic areas of interest; and (2) for particular sources/sectors, a more detailed characterization across pollutants and what controls may be available or planned. (Workplan Item 3)
- *Hazardous air pollutants (HAPs):* Sometimes there are gaps in emissions inventories with respect to HAPs since it can be difficult to inventory these data from many sources. For example, in many cases emissions are reported as total tons of VOC and are not speciated into the component pollutants. Trying to inventory all 187 HAPs for all sources may not be possible. Instead, it may be a better use of resources to focus on improving the inventory for the toxic species that are leading the cancer and non-cancer risk in the area. Monitoring data and information from any special studies performed in the area may be useful to identify the pollutants that are of the greatest concern and could be the focus of emissions inventory improvements. (Workplan Item 3) South Carolina should engage in the current state review process for the 2011 NATA inventory since EPA plans to use the 2011 NATA platform to run modeling analyses.
- *Baseline modeling:* For this study, EPA will provide the 2011 NATA inventories and air quality and risk analyses for an appropriate multi-pollutant baseline. This will provide the needed source emissions and characteristics, refined air quality concentrations (census track receptors for toxics and 12km CMAQ run for ozone and PM<sub>2.5</sub>), and the air toxic risk assessment that will allow source attribution of risk drivers for the 10-county area. (Workplan Item 4)
- *Future year baseline modeling and emissions projection:* EPA will also provide projected emissions and air quality for 2020 based on the PM<sub>2.5</sub> NAAQS RIA for ozone and PM<sub>2.5</sub>. This will provide directional information on the effect of regional and national controls on the Upstate area. Table I, above, also provides directional impact of future controls. In addition, as deemed necessary as part of additional air quality modeling to be conducted to assess the impact of the local control strategy, any local enhancements to the emissions inventory to create the future-year base inventory will need to be determined by DHEC. This could mean collecting data on any closings or shut-downs of industrial sources, mobile fleet turnover, and installation of potential control measures and determining which to include in the future base year projection. (Workplan Item 4)

#### Control Measure Information and Additional Data

In order to maximize potential co-control opportunities for direct and precursor emissions for PM<sub>2.5</sub>, ozone, and air toxics, DHEC will identify those sources affecting potential areas of interest (e.g., monitor locations; populations of concern) within the 10-county area with a focus on those that are in need of control to reduce emissions and associated risks. As part of this effort, with assistance from EPA, DHEC will identify available control options for those sources to develop a local control strategy that targets

“multi-pollutant” reductions, i.e., those that will focus on the toxics of concern for communities within the 10-county area but maximize those ozone and PM<sub>2.5</sub> precursor emissions reductions to gain health benefits and further reductions in future design values for ozone and PM<sub>2.5</sub>.

- To assist DHEC in development of their local control strategy, EPA makes available (not including any state specific databases) its Control Strategy Tool (COST - <http://www.epa.gov/ttn/ecas/cost.htm>). This tool provides a good place to start, having multi-pollutant information on many sources and allowing the user to electronically connect directly to the emissions inventory using the Emissions Modeling Framework (EMF - <http://www.ie.unc.edu/cempd/projects/emf/install/>). EPA will offer the DHEC CoST-related support. Most likely it will still be important to add additional information and “multipollutantize” some of the control measures in this database for the sources of most concern, e.g., it may be necessary to enhance the database with reduction efficiencies for air toxics for those controls that gain ozone and PM<sub>2.5</sub> precursor emissions reductions (VOCs and metal HAPs). (Workplan Item 2)
- *Health and population data:* DHEC will gather the appropriate necessary refined health data for input to BenMAP-CE<sup>9</sup> in order for the benefits analysis to be reflective of the demographics and susceptibility of the underlying population in the 10-country area. This will help determine the location of vulnerable and susceptible populations and correlations with higher concentrations of pollutants of concern and quantify the health benefits of emissions reductions. EPA will assist DHEC with running BenMAP-CE.
- See *Health Impact Assessment below.* (Workplan Item 3)

### Air Quality Modeling

The modeled predictions of air quality changes are data that can be used to gauge the successfullness of the control strategy. These data are essential for predicting the effects on local and regional air quality, attainment of NAAQS standards, and risk and exposure. As in the Detroit project and with leveraging EPA’s 2011 NATA effort, EPA expects to apply the CMAQ photochemical model (Community Multiscale Air Quality Model - [www.cmaq-model.org/](http://www.cmaq-model.org/)) at a horizontal scale of 12x12 km for predicting ozone and PM<sub>2.5</sub> and combine the HAP results from the AERMOD ([http://www.epa.gov/ttn/scram/dispersion\\_prefrec.htm#aermod](http://www.epa.gov/ttn/scram/dispersion_prefrec.htm#aermod)) dispersion model to provide horizontal resolution at the census tract. Further discussion will determine the best choice of additional air quality modeling for this work.

Once control options for specific sources are defined and a local control strategy for the area is designed, then EPA and DHEC will work together to decide on and conduct an appropriate local air quality assessment to inform the toxics risk assessment (i.e., inform Human Exposure Model (HEM-3)) and the ozone and PM<sub>2.5</sub> health assessment (i.e., inform BenMAP). There are several options for the assessment that EPA will conduct with input from DHEC:

- I. Conduct specific air quality modeling of the local control strategy for a small modeling domain that includes the 10-county area. This would be illustrative so “projections” would be a scaled

---

<sup>9</sup> BenMap-CE is the Environmental Benefits Mapping and Analysis Program-Community Edition.

version of the 2011 NATA inventory to serve as a “future base” and then assess the local control strategy as the “future control” scenario.

2. Adjust the 2011 NATA inventory based on appropriate adjustment factors to translate emissions changes to air quality concentration changes for the 10-county area (and beyond, if expect downwind PM<sub>2.5</sub> or ozone benefits outside the area).
3. Conduct a qualitative assessment if the emission reductions are not deemed significant or consider a more localized assessment of specific communities if the control scenarios are largely focused on single facilities (e.g., separately conduct dispersion modeling of an individual source).

If additional “fine-scale” modeling is deemed necessary, EPA and DHEC will have additional discussions about source locations, spatial and temporal scales and future year projections at the “fine-scale.” We define “fine-scale” modeling to be modeling of a photochemical model with a horizontal grid resolution of 4x4 km or smaller and/or application of a dispersion model. (Work Plan Item 5)

The modeled concentrations of air quality can be used to evaluate the impact of the control strategy on the future year design values (DVs) and on the air quality in the urban area, as well as in the region. Visualizing the results in programs like ArcGIS is extremely helpful to analyze the geographical impact of the strategy. Using GIS, one can also overlay the population to better understand the population-weighted air quality changes, as well as analyze areas of remaining high concentrations with respect to emission sources.

It will also be important to calculate the change in the predicted, future year design values based on the local control strategy. To do this more efficiently, EPA has created the Model Attainment Tool Software (MATS) ([http://www.epa.gov/ttn/scram/modelingapps\\_mats.htm](http://www.epa.gov/ttn/scram/modelingapps_mats.htm)). MATS takes the inputs of modeled and monitored data to predict the 8-hr ozone, the 24-hr PM<sub>2.5</sub>, and annual PM<sub>2.5</sub> DVs. MATS can also be used to create the spatial fields of ozone and PM<sub>2.5</sub> air quality to input into BenMAP for the health impact assessment.

### Air Toxics Risk Assessment

To estimate exposure concentrations for a given risk assessment for this project, we would use ambient pollutant concentrations as surrogates of the inhalation exposure concentrations for the populations in the study locations. The default assumption in this approach is that the population of interest is breathing outdoor air continuously, which is conservative because people are not always at the study location due to their daily activities, and because indoor air concentrations are expected to be the same or lower than the outdoor concentrations (when the indoor concentrations are produced solely by inflow from outside air). The HEM-3 model ([http://www.epa.gov/ttn/fera/fera\\_download.html](http://www.epa.gov/ttn/fera/fera_download.html)), which was used in the Detroit project, is based on this approach.

Further discussion between EPA, DHEC, and the Upstate would be beneficial for this assessment.

### Ozone and PM<sub>2.5</sub> Benefits Assessment

As in the Detroit project, we would recommend relying upon the environmental Benefits Mapping and Analysis Program (BenMAP) to assess the avoided PM<sub>2.5</sub> and ozone-related health impacts and associated monetized benefits of alternate policy scenarios. The calculation of health impacts requires four key

sources of data: air quality changes, population estimates, risk coefficients, and the baseline incidence rate for each health endpoint quantified. As described in Hubbell et al. (2009), performing a city-level health impact analysis calls for local-scale input data to reduce the risk of biasing the analysis with health data that does not characterize the health status of populations within the Upstate. Spatially resolved incidence rates and effect coefficients will also be useful to any EJ analysis performed for the 10-county area, as they will enable us to identify vulnerable and susceptible populations and estimate health impacts among these at-risk populations. However, we were unable to obtain age-stratified population data needed to run this analysis correctly so we resolved to use the BenMAP county-level incidence rates. Air quality changes for ozone and PM<sub>2.5</sub> based on the local control strategy emissions reductions are provided outside of the BenMAP program (standard file format from CMAQ is established and can be generated for input). However, while EPA can generate population projections for the study area, the identification of effect coefficients and incidence rates will require more effort. See Table 3: Health and Socioeconomic Data Inputs.

The EPA will survey the epidemiological literature to determine whether existing or new PM or ozone studies have been conducted for South Carolina or the Upstate. It will also be critical to use ZIP or tract-level baseline incidence rates where available. Below we have detailed the health endpoints and age ranges for which we need these data. In general, we need rates stratified by patient sex, ZIP or census tract FIPS, year, age category, and principal diagnosis.<sup>10</sup> If available, race-stratified rates would be useful to performing a more reliable EJ or distributional analysis.

If appropriate, distributional and EJ impacts could be quantified using a combination of BenMAP and ArcGIS. EPA has developed approaches for using baseline health data in conjunction with air quality levels to identify populations at greatest risk of air pollution impacts. The identification of “at-risk” populations might be a useful first step to developing the air quality management strategies.

#### Insights on Development of the Local Control Strategy

While the sections above discuss what data is needed to implement and analyze the results of a multi-pollutant, risk-based control strategy, this section tries to discuss some of the important things to consider when designing the control strategy. In general, based on the Detroit project results and “lessons learned,” we recommend trying to incorporate these basic goals:

- Aim to achieve population oriented emission reductions, particularly for susceptible and vulnerable populations;
- Consider selecting control measures that reduce multiple pollutants whenever possible;
- Focus on reducing the toxic pollutants that are driving the cancer and non-cancer incident rates;
- When making decisions based on costs, try to consider the resultant \$ per ppb or  $\mu\text{g}/\text{m}^3$  of air quality improvement or \$ per health benefit a control measure will potentially supply rather than simply looking at \$ per tons of emissions reduced;

---

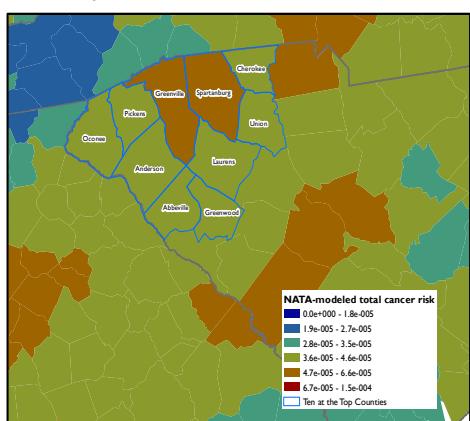
<sup>10</sup> If possible, age stratified by the following bins would be most useful: 0-1, 2-6, 7-14, 15-17, 18-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85-99.

- Where possible, combine base air quality with health information to better inform decisions, especially as they relate to EJ issues.

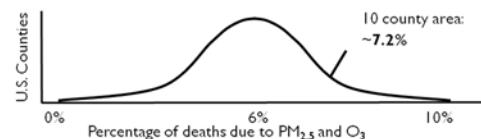
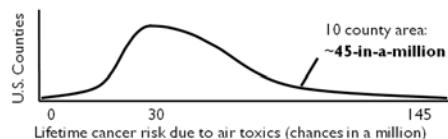
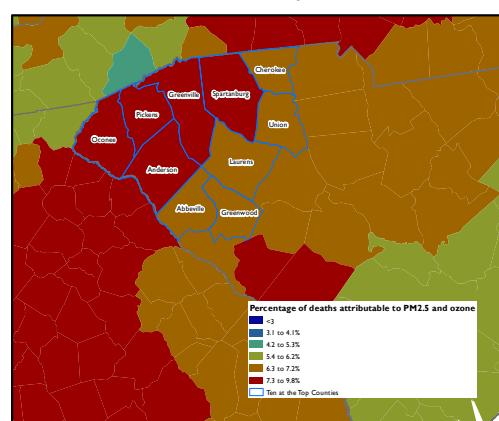
We realize that policy considerations and costs constrictions will also need to be considered when determining what control measures to include. When possible, we recommend using the basic guidelines listed above to develop a first draft of the strategy and then analyzing impacts on air quality, DVs, toxic risk, and health benefits to refine the strategy to better fit the goals of the work. (Work Plan Item 2). Information regarding the confluence of air toxic and criteria pollutant risk and the location of emitting sources may prove helpful to constructing an emissions control strategy that satisfies the criteria above (Figures 1, 2 and 3).

**Figure 1. Air toxic lifetime cancer risk and criteria pollutant annual mortality risk at each county**

2005 NATA-predicted lifetime cancer risk

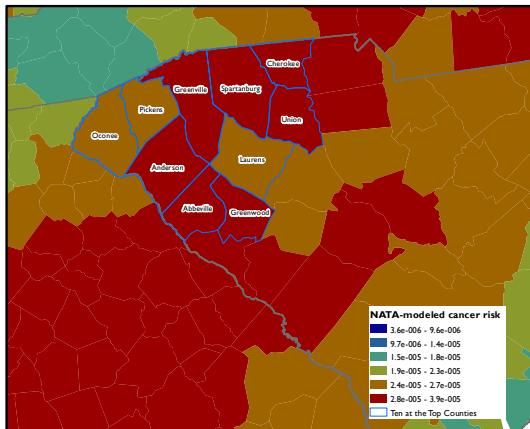


2005 PM<sub>2.5</sub> and ozone mortality risk

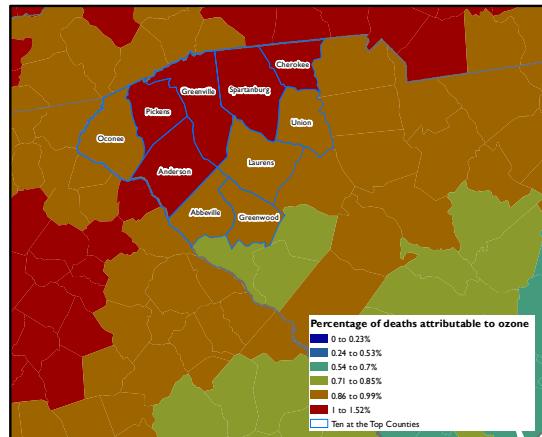


**Figure 2. Lifetime cancer risk from air toxics formed in the atmosphere and annual ozone mortality at each county**

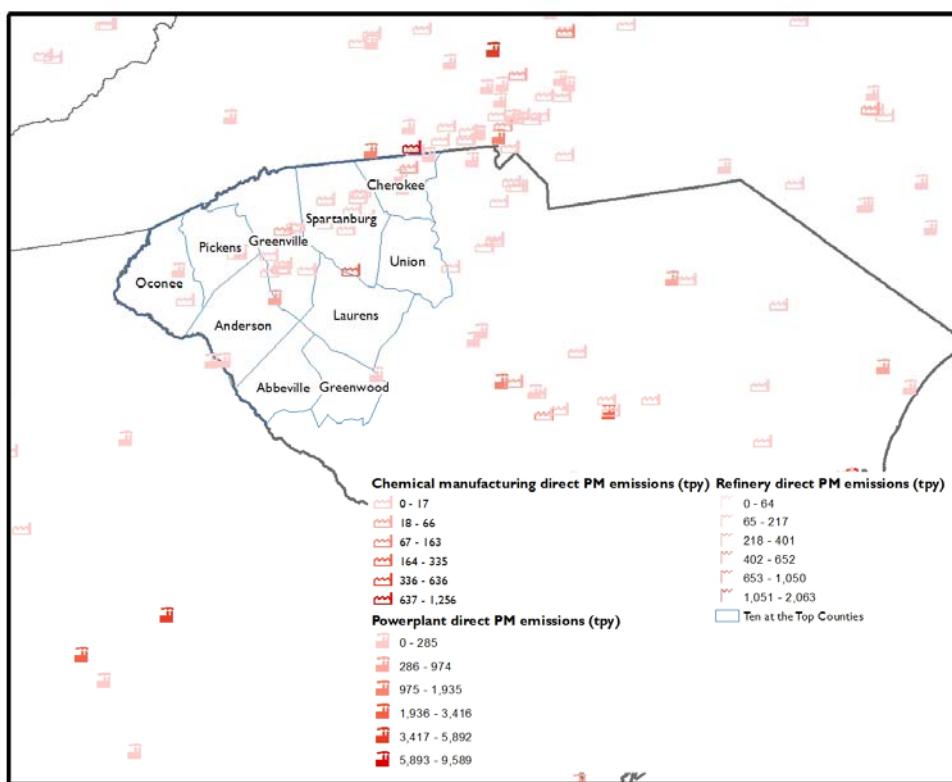
2005 NATA-predicted lifetime cancer risk from air toxics formed in the atmosphere



2005 ozone mortality risk



**Figure 3. Sources of directly emitted PM<sub>2.5</sub>**



#### Other Planning/Policy Considerations

- Energy Planning (Energy Efficiency/Renewable Energy)
- Environmental Justice assessment?/Transportation & Land-use planning and Climate Change? (Work Plan Item 2)

*Roles and responsibilities:*

The success of each stage of the project will hinge upon the close collaboration between EPA, DHEC, and the Upstate. Table 2 includes a workplan and summarizes roles and responsibilities for the project with a draft schedule.

*Work Plan Key Items:*

**Table 2. Work Plan** (EPA and DHEC agree to meet to discuss status on the last Monday of each month at 1 pm.)

	<b>Schedule</b>	<b>Coordination</b>	<b>Resources</b>
(1) Problem Formulation & Work Plan	August/ September	DHEC with EPA providing technical guidance	EPA/DHEC
(2) Brainstorm/Develop Control information and control strategies	August/ September/ October/ November	DHEC and CAU/TATT with EPA providing technical assistance	Covered by state's current SIP funds. EPA could help with running CoST.
(3) Data Acquisition for Emissions Inventories and Health Data	December	DHEC and CAU/TATT	Covered by state's current SIP funds
(4) Emission Inventory (base and future projection) using 2011 data and additional CoST run with control strategy	December-May	DHEC & EPA	Expect to leverage existing EPA inventories and projections. SC may make local improvements as appropriate.
(5) AQ modeling and post processing	December-May	EPA leads with assistance from DHEC	EPA would conduct in consultation with DHEC. Details and options noted above.
(6) Risk and benefits assessment	December-May	DHEC and EPA jointly perform assessment	DHEC would conduct in consultation with EPA and with CDC involvement
(7) Compilation of results of risk-based analysis, and potential selection of measures to be implemented and added to SC Advance path forward	May-June	DHEC leads with EPA providing technical guidance	Covered by state's current SIP funds

(8) Information for public outreach	June	DHEC leads with EPA providing guidance	Covered by state's current SIP funds
-------------------------------------	------	--	--------------------------------------

**Table 3. Health and Socioeconomic Data Inputs**

Unless indicated below, each data set representing total counts (or prevalence) will be segmented accordingly:

- By gender
- By age group: 0-5, 6-11, 12-19, 20-39, 40-59, 60+
- ZIP level data for three most recent years (to be averaged across years)

DISEASE	ICD 9 CODE	DATA SET
Acute myocardial infarction	410-414	ER and hospital admissions
Conduction disorders	426	ER and hospital admissions
Cardiac dysrhythmias	427	ER and hospital admissions
Congestive heart failure	428	ER and hospital admissions
All cardiovascular	390-429	ER and hospital admissions
All cardiovascular (less myocardial infarctions)	390-409, 411-429	ER and hospital admissions
Heart disease complications	429	ER and hospital admissions
Cerebrovascular disease	430-438	ER and hospital admissions
Hemorrhagic stroke	430-432	ER and hospital admissions
Stroke	430-434	ER and hospital admissions
Ischemic stroke	433-434	ER and hospital admissions
Peripheral vascular disease	440-448	ER and hospital admissions
Respiratory disease	460-519 466, 480-486, 490-493	ER and hospital admissions
Respiratory illness	464-466, 480-487, 490-492	ER and hospital admissions

	464-466, 480-487	
Chronic lung disease or COPD	490-496 490-492, 494-496 490-492, 494,	ER and hospital admissions
Chronic lung disease (less asthma)	490-492 491,492, 494, 496	ER and hospital admissions
Pneumonia	480-486	ER and hospital admissions
Asthma	493	ER and hospital admissions
Lower respiratory infection	466.1, 466.0, 480-487, 490, 510-511	ER and hospital admissions
<b>Other Effects (if available)</b>		
Acute bronchitis	466	Prevalence only
Chronic bronchitis	491	Prevalence only
Cough variant asthma	493.82	Prevalence only
Asthma: any exacerbation or attack	493-493.9	Prevalence only
Days of work lost due to any cause	Asthma and/or bronchitis	Incidence data
<b>Socioeconomic data</b>		
<b>Poverty data</b>	<ul style="list-style-type: none"> <li>• Fraction of individuals and households (by race) below the poverty line (ZIP or tract)</li> <li>• Fraction of individuals and households (by race) at below 1.5 x the poverty line (ZIP or tract)</li> </ul>	
<b>Education</b>	<ul style="list-style-type: none"> <li>• Fraction of individuals (by race) with less than a grade 12 education (ZIP or tract)</li> <li>• Fraction of individuals (by race) with a grade 12 education (ZIP or tract)</li> <li>• Fraction of individuals (by race) with greater than a grade 12 education (ZIP or tract)</li> </ul>	

## **Appendix B: Background on Air Quality Management: *Working toward a Multi-Pollutant Approach***

In 1955, the first federal air pollution control law was promulgated primarily to fund research into the scope and sources of air pollution. Since that time, air quality management has evolved in many ways to include the first Federal Clean Air Act (CAA or Act) in 1963. However, it wasn't until 1970 that the previous iterations were amended creating what some consider to be the first modern day CAA. The 1970 amendments increased authority of the newly created Environmental Protection Agency (EPA) and established the basic structure of our nation's present air quality management program. This Act authorized the establishment of National Ambient Air Quality Standards (NAAQS), the New Source Performance Standards (NSPS) for new and modified stationary sources, the establishment of National Emission Standards for Hazardous Air Pollutants (NESHAPs), increased enforcement authority, and authorized requirements for the control of motor vehicle emissions. The 1970 CAA also established requirements for State Implementation Plans (SIPs) to achieve the NAAQS and address air quality concerns.

In June 1989, then President Bush proposed significant revisions to the CAA. The resulting 1990 amendments were enacted in large part to deal with urban air pollution or NAAQS. The NAAQS are air quality standards set by the EPA for six "criteria pollutants" which are among the most harmful to public health and the environment. With the 1990 amendments, EPA is required to set NAAQS for each of the criteria pollutants and review these standards once every five years to determine if they are appropriate or if new standards are needed to protect public health.<sup>11</sup> Since these last major amendments, technology and science have continued to evolve such that many now recognize the importance of a more holistic approach to air quality management. A number of task forces, work groups, and studies have looked at the current air quality management system and made recommendations for improvements. In 2004, the National Research Council issued a report that described some of the challenges that will be faced in future air quality management efforts. They recommended a multipollutant approach to reducing emissions for both criteria and hazardous air pollutants.<sup>12</sup>

It is with these thoughts in mind that the EPA and states have sought opportunities to work together to seek out opportunities for collaboration; to take an integrated multi-pollutant approach to managing air quality that is based on risk assessment and simultaneous review of multiple interrelated pollutants. In response to the need to further explore and understand the technical needs and challenges presented by implementing a multi-pollutant, risk-based approach to air quality management, the EPA's Office of Air Quality Standards and Planning (OAQPS) embarked on a case study in 2010 centered on the urban area of Detroit, Michigan. As part of this case study, two contrasting air quality control strategies were assessed and compared; known as the 'status quo' and a "multi-pollutant, risk-based" approach aimed at further reducing population risk from exposure to ozone, PM<sub>2.5</sub> and selected air toxics.

In 2013, the EPA again sought to partner with another state to further explore a multi-pollutant approach to air quality management in the Southeastern United States. Working with staff of the South Carolina Department of Health and Environmental Control as well as local community leaders in the

---

<sup>11</sup> 42 U.S.C. § 7409 (2011).

<sup>12</sup> John Bachmann (2007) Will the Circle Be Unbroken: A History of the U.S. National Ambient Air Quality Standards, Journal of the Air & Waste Management Association, 57:6, 652-697 (<http://dx.doi.org/10.3155/1047-3289.57.6.652>)

South Carolina upstate, a pilot project was developed to assess the effects of examining a control reduction strategy aimed at addressing multiple pollutants and air toxics to improve air quality and health. Perspectives from each of the partners in this study are provided in the report. In general local area perspective and expertise play a big role in successfully implementing any voluntary emissions reduction program. Additionally, a collaborative effort between federal and State technical staff allowed for knowledge transfer and feedback on new and innovative tools developed during the course of this project.

## Appendix C: 2011 NATA Risk Reduction Analysis - South Carolina Ten at the Top Counties

### Background

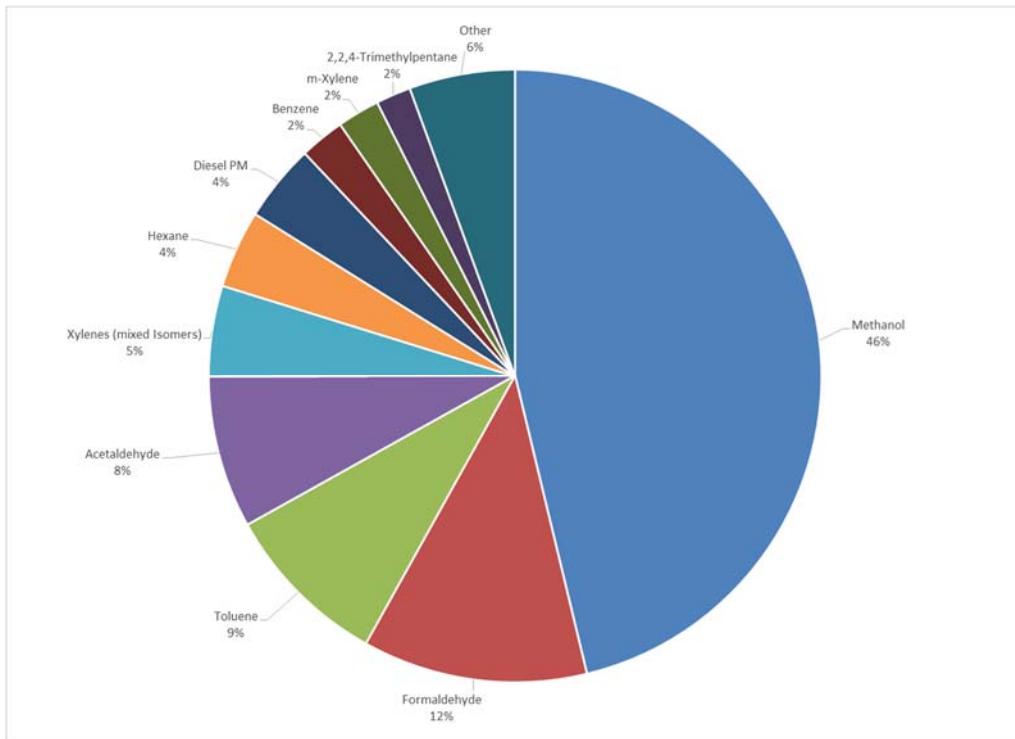
The South Carolina Clean Air Upstate Coalition (CAU)/Ten at the Top (TATT) is a group of ten counties in the northwest corner of South Carolina and consists of the following counties: Abbeville, Anderson, Cherokee, Greenville, Greenwood, Laurens, Oconee, Pickens, Spartanburg, and Union. The following air toxic analysis is based on county level air toxic risk and emission estimates from the 2011 *National Air Toxic Assessment (NATA)* as well as emissions reduction data provided by South Carolina as part of this program.

The 2011 NATA is a risk analysis based on an emissions inventory, the 2011 National Emissions Inventory (NEI) of major, area, and mobile source emissions for the calendar year 2011. The analysis assumes that these emissions occur for 70 years and does not take into account yearly variability in emissions. The reader is referred to the NATA Technical support document (<http://www.epa.gov/national-air-toxics-assessment/2011-nata-technical-support-document>) and the NATA website ([www.epa.gov/NATA](http://www.epa.gov/NATA)) for a more complete compendium of the approach, as well as the uncertainties and limitations of the NATA analysis.

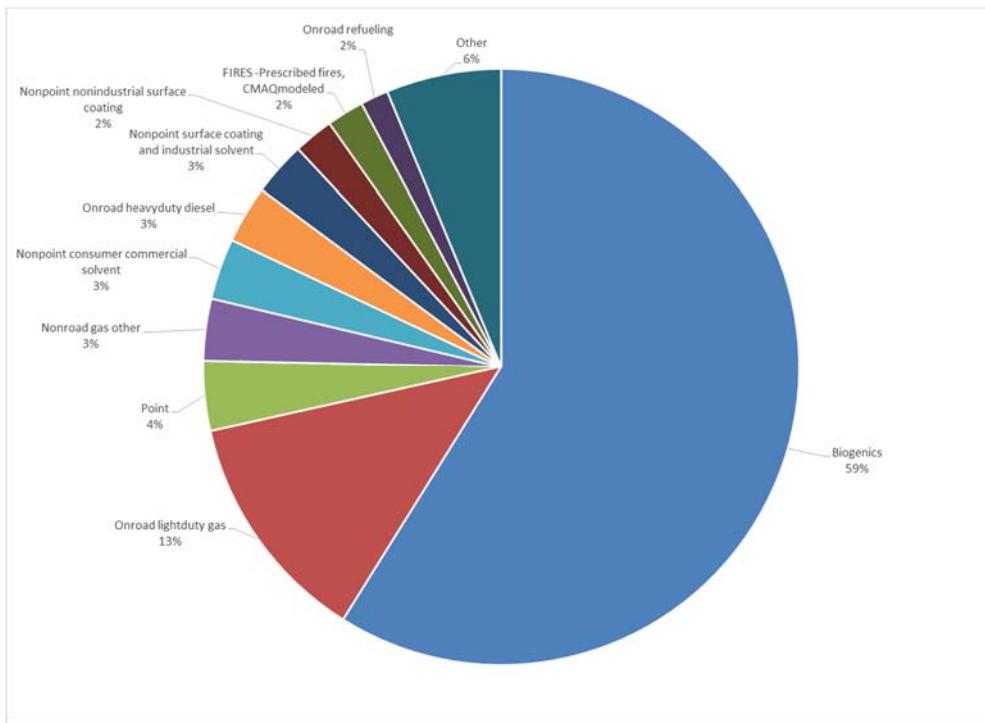
### CAU/TATT Air Toxic Emissions

Based on the 2011 NEI, nearly 28,000 tons of air toxics are emitted each year from the South Carolina CAU/TATT counties. In comparison, statewide emissions of air toxics in South Carolina are estimated by the 2011 NEI to be nearly 160,000 tons/year. Methanol emissions comprise nearly half the CAU/TATT emissions, with formaldehyde, toluene, and acetaldehyde emissions comprising over a quarter of the emissions in the CAU/TATT counties. Figure 1 depicts the air toxic emissions by pollutant for the SC CAU/TATT counties. One-third of these emissions are from onroad mobile sources, and almost another third is from smaller area sources. Major point sources are responsible for only 4 percent of the emissions in the SC CAU/TATT counties. Figure 2 depicts the air toxic emissions for each source sector for the SC CAU/TATT counties.

**Figure 1. 2011 NEI – Pollutant contributions to SC CAU/TATT county air toxic emissions (28,000 TPY).**



**Figure 2. 2005 NATA – Source Sector contributions to SC CAU/TATT county air toxic emissions (28,000 TPY).**



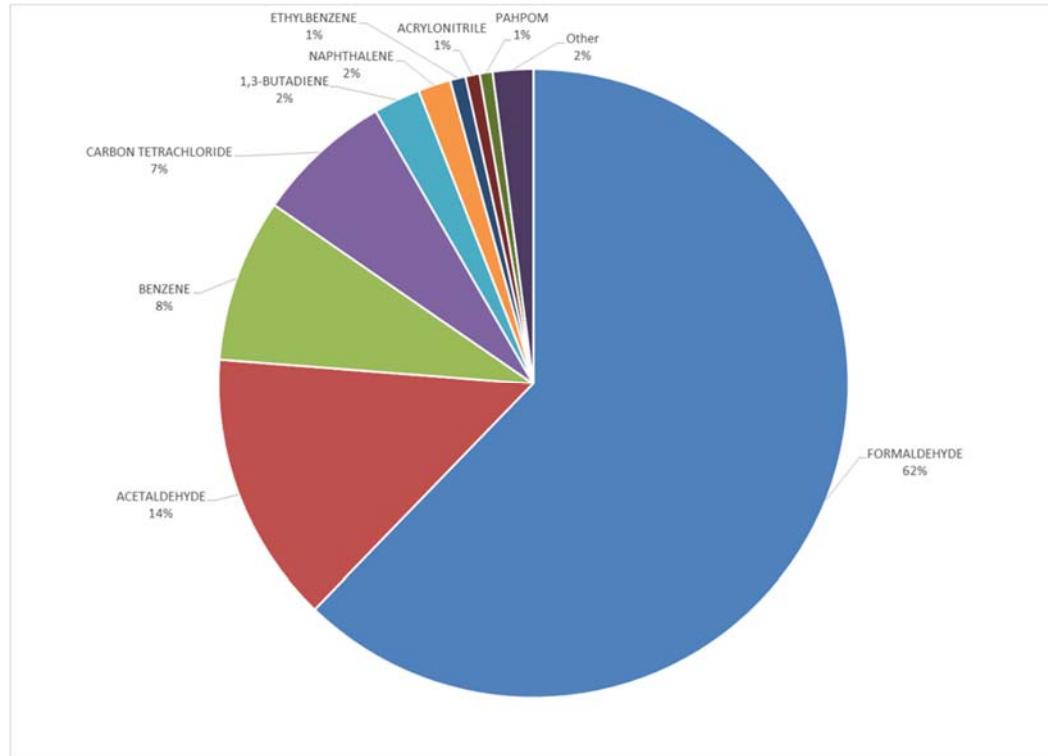
## CAU/TATT Estimated Cancer Risks

Based on the 2011 NATA, the average excess cancer risk in the SC CAU/TATT counties associated with inhalation of air toxics is about 47 in one million. This risk is slightly above the statewide cancer risk estimate of 44 in one million and the national average cancer risk predicted by NATA of 40 in one million. A majority of the risks in the CAU/TATT counties is associated with formaldehyde (62 percent), with acetaldehyde (14 percent) and benzene (8 percent) also key contributing pollutants. While not directly emitted in large quantities (see above), formaldehyde and acetaldehyde are mostly formed through photochemical activity. In the Southeast US, such activity is most prevalent along the I-85 corridor. Figure 3 depicts the primary pollutants contributing to the average cancer risks in the CAU/TATT counties.

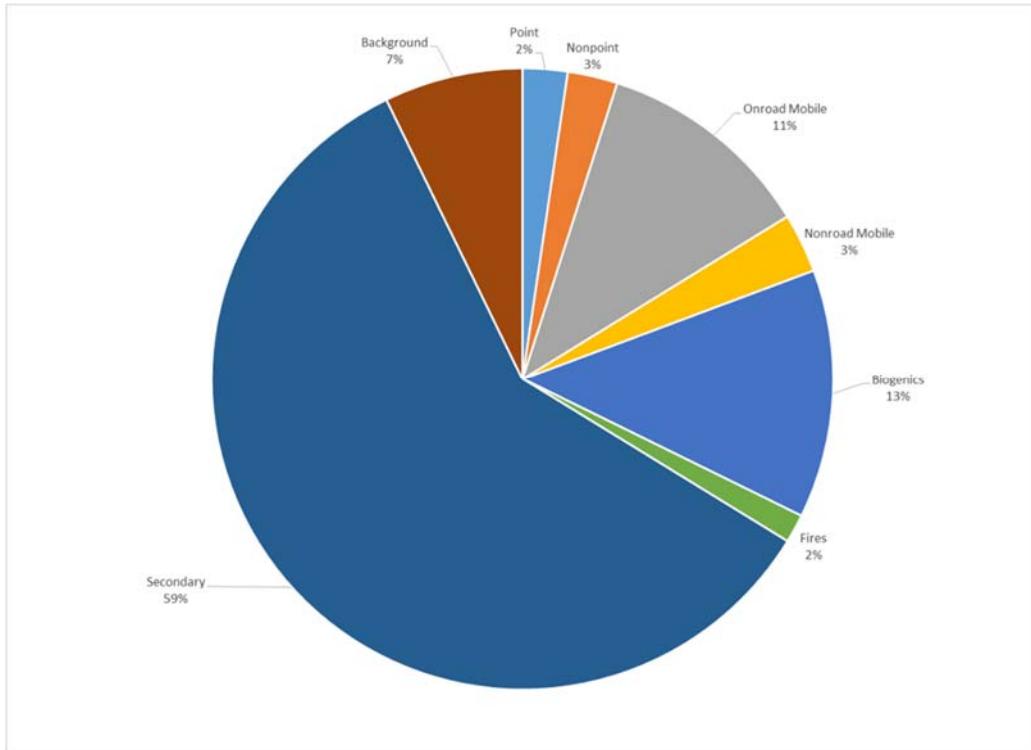
NATA estimates that a majority of the risks in the CAU/TATT counties is from secondarily formed pollutants and from background sources or transported emissions into the CAU/TATT region. Figure 4 depicts the primary source sectors contributing to the average cancer risks in the CAU/TATT counties.

While NATA estimates that the entire population of the SC CAU/TATT counties is exposed to cancer risks greater than 30 in one million, about 3,000 residents are exposed to cancer risks greater than 60 in one million at the census tract level, with the highest risk of 66 in one million in the urban core areas of Greenville. Figure 5 depicts the cancer risks for all the census tracts in South Carolina.

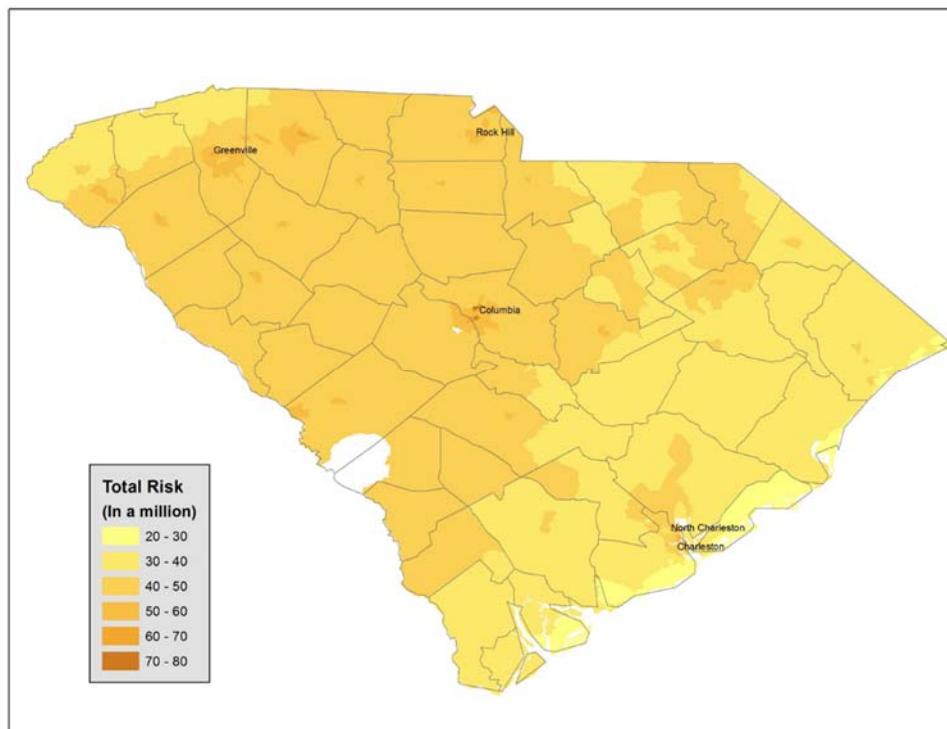
**Figure 3. 2011 NATA – Pollutant contributions to SC CAU/TATT county cancer risks (47 in one million).**



**Figure 4. 2011 NATA –Source Sector contributions to SC CAU/TATT county cancer risks (47 in one million).**



**Figure 5. 2011 NATA - Census Tract Cancer Risks- South Carolina.**



## *Emission Reductions and Estimated Cancer Risk Reductions*

To predict the effect of the proposed emissions reductions on air toxic risks, we started with the 2011 NATA county level risks for each of the CAU/TATT counties. We assumed that a reduction in emissions would result in a similar reduction in risk for a given pollutant. Because the inventory used for NATA (2011 NEI) and that developed for the CAU/TATT reduction effort are not the same, we could not directly apply the tonnage of reductions to the NATA analysis. Instead, we applied the percentage reductions from the CAU/TATT inventory to NATA point and nonpoint risk results on a pollutant by pollutant basis. There were no estimated emissions reductions from other source types, so there were no estimated risk reductions from those.

This approach assumes that reductions are equal across all NATA point and nonpoint source categories. Nevertheless, we feel this approach will provide an approximate estimate of potential reductions in risk associated with the proposed emissions reductions.

The emissions reductions were focused on six air toxic pollutants from point and nonpoint source sectors. Table I depicts the expected reductions for each pollutant for all the CAU/TATT counties.

**Table I. Proposed Air Toxic Emissions Reductions.**

	Pre-CAU/TATT (TPY)	Post-CAU/TATT (TPY)	% Reductions
Formaldehyde	12	2	-85%
Acetaldehyde	6	1	-84%
Benzene	15	2	-86%
1,3-Butadiene	3	0.3	-87%
Chromium (VI) compounds	0.001	0.00001	-99%
Ethyl benzene	28	11	-60%

We estimated risk reductions from the proposed program by applying the percentage emissions reductions to the county wide risk averages for each of the CAU/TATT counties. Table 2 depicts the county risk averages from the 2011 NATA and the expected reductions. The highest reductions are about 3 percent in both Greenville and Spartanburg counties. It's important to note that this analysis does not include potential reductions from "anti-idling" efforts. The NATA model results for mobile sources do not segregate out the idling emissions, thus we cannot estimate that portion of the risk that would be reduced. However, we would expect further risk reductions from this program.

**Table 2. 2011 NATA – CAU/TATT Average County Total Cancer Risks and Expected Risk Reductions.**

	2011 County Cancer NATA Risk	Expected % Risk Reduction
Abbeville	44	0.01
Anderson	46	2
Cherokee	45	1
Greenville	48	3
Greenwood	47	0.01
Laurens	45	0.02
Oconee	42	0.01
Pickens	43	0.03
Spartanburg	48	3
Union	47	0.01

## Appendix D: South Carolina Ten at the Top Counties Cost Analysis

### *Introduction*

Emissions reductions opportunities were identified for South Carolina's Clean Air Upstate Coalition (CAU)/Ten at the Top (TATT) counties using the CoST tool (<http://www.epa.gov/ttnecas1/cost.htm>). CoST provides information on potential control options to reduce emissions and how much they would cost to implement. The tool is applied to emissions inventories, and controls are identified and applied at the unit-Standard Classification Code (SCC) level for point sources and at the county-SCC level for non-point and mobile sources. CoST can be applied to reduce a single pollutant, or, as in the case of South Carolina's CAU/TATT to reduce multiple pollutants simultaneously. For South Carolina, CoST was applied using the 2011 NEI and the pollutants of interest were: NOx, PM<sub>2.5</sub>, SO<sub>2</sub> and VOCs. Two options exist to reduce emissions using CoST, one is at minimum cost to achieve a target emissions reduction, and the other is by applying maximum reductions. For the CAU/TATT, the maximum emissions reductions method was chosen to see what was potentially available in terms of controls and emissions reductions. While it is possible to use CoST to apply controls to non-EGU point, non-point, mobile and electricity generating units (EGUs), the group decided to apply CoST to non-EGU point and nonpoint sources, so the CoST analysis did not include EGUs, mobile sources and open burning SCCs. An emissions strategy for school bus idling was conducted by South Carolina's Department of Health and Environmental Control (DHEC) outside of CoST.

States can use CoST to get a sense of the types of controls they could apply and design and to compare potential control strategies and their emissions reductions. States can also provide updated information on controls given their knowledge of local conditions. The U.S. EPA welcomes any such updated local information that can be incorporated into CoST. Currently the tool includes mostly end-of-pipe controls, although there are ongoing efforts to include opportunities for reductions from renewable energy, energy efficiency and fuel switching. Emissions control strategies from CoST should be viewed as illustrative or hypothetical, because each State has a better idea of what controls and emissions reductions strategies are feasible for them.

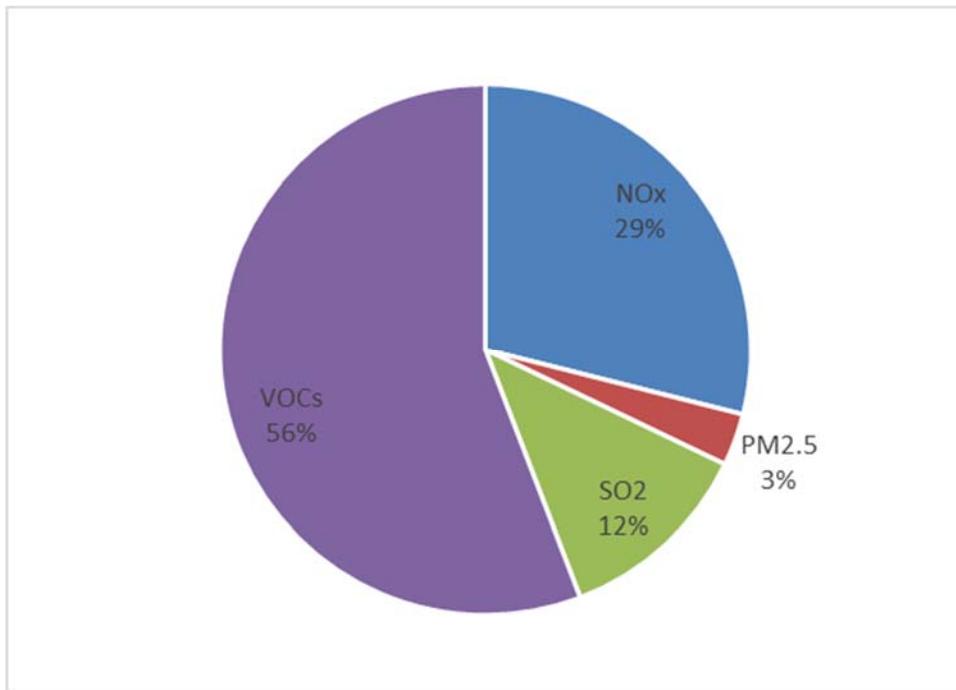
As with any tool or model uncertainties exist in results derived from CoST. Data may not always be available to reflect the specific characteristics of a facility or source group, and thus the variation in emissions control requirements and resulting emissions. Also, emissions inventories used with CoST are a reflection of how accurately data was reported by the reporting entities (e.g., industries reporting to state air agencies, states reporting to EPA). There is also uncertainty regarding which production and control technologies will become cheaper in the future, so CoST results should not be expected to provide a prediction of these emerging technologies.

Software to run CoST can be downloaded from the Community Modeling and Analysis System (CMAS) website. A user manual is available. The Contact person for CoST is David Misenheimer ([misenheimer.david@epa.gov](mailto:misenheimer.david@epa.gov)) at the Air Economics Group in the Office of Air Quality Planning and Standards.

## CAU/TATT Criteria Emissions Profile

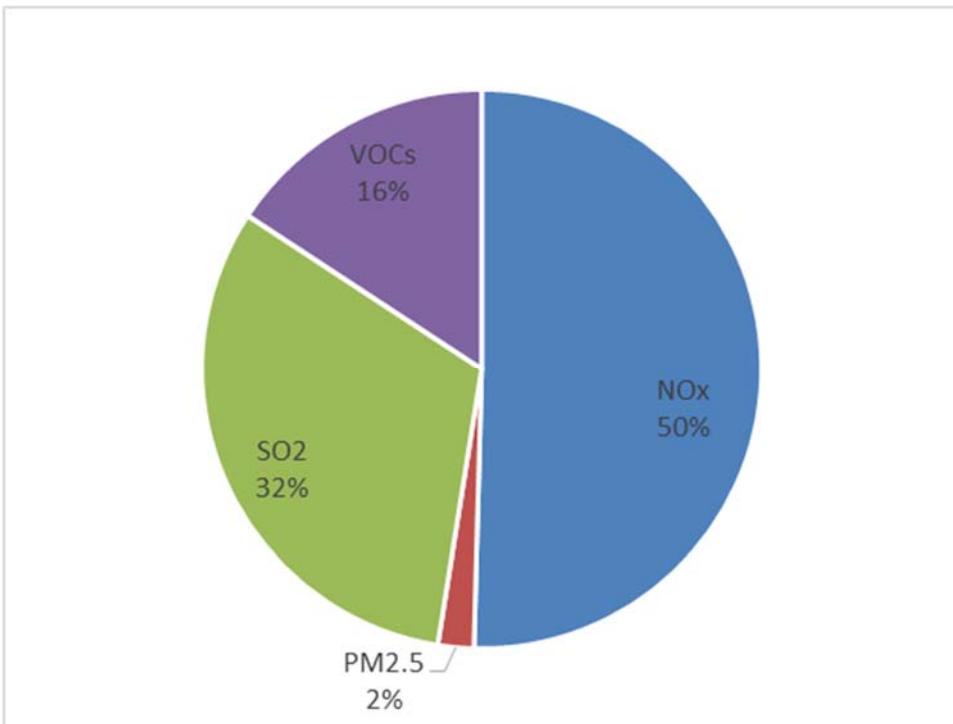
Data from the 2011 NEI shows that there were 8,000 tons of NOx, PM<sub>2.5</sub>, SO<sub>2</sub> and VOCs being emitted in CAU/TATT counties. Figure 1 shows the breakdown by pollutant, with 2,300 tons of NOx, 257 tons of PM<sub>2.5</sub>, less than 1000 tons of SO<sub>2</sub> and about 5000 tons of VOCs.

**Figure 1. 2011 NEI - Criteria Pollutant Emissions in CAU/TATT Counties**



Point source emissions were about 3,000 tons: 1,500 tons of NOx, 60 tons of PM<sub>2.5</sub>, 1,000 tons of SO<sub>2</sub> and about 500 tons of VOCs. These emissions came mostly from IC engines and coal fired boilers. Non-point source emissions were about 5,000 tons: 780 tons of NOx coming mostly from residential, commercial and institutional Natural gas use; about 200 tons of PM<sub>2.5</sub> mostly from fireplaces, hydronic heaters and woodstoves; and almost 4,000 tons of VOCs mostly from architectural, motor vehicle and other coatings. Figures 2 and 3 show emissions for point and non-point sources as a percentage of total source emissions respectively.

**Figure 2. 2011 NEI - Criteria Pollutant Point Source Emissions in CAU/TATT Counties as a Percentage of Total Point Source Emissions**



**Figure 3. 2011 NEI - Criteria Pollutant Non-Point Source Emissions in CAU/TATT Counties as a Percentage of Total Non-Point Source Emissions**

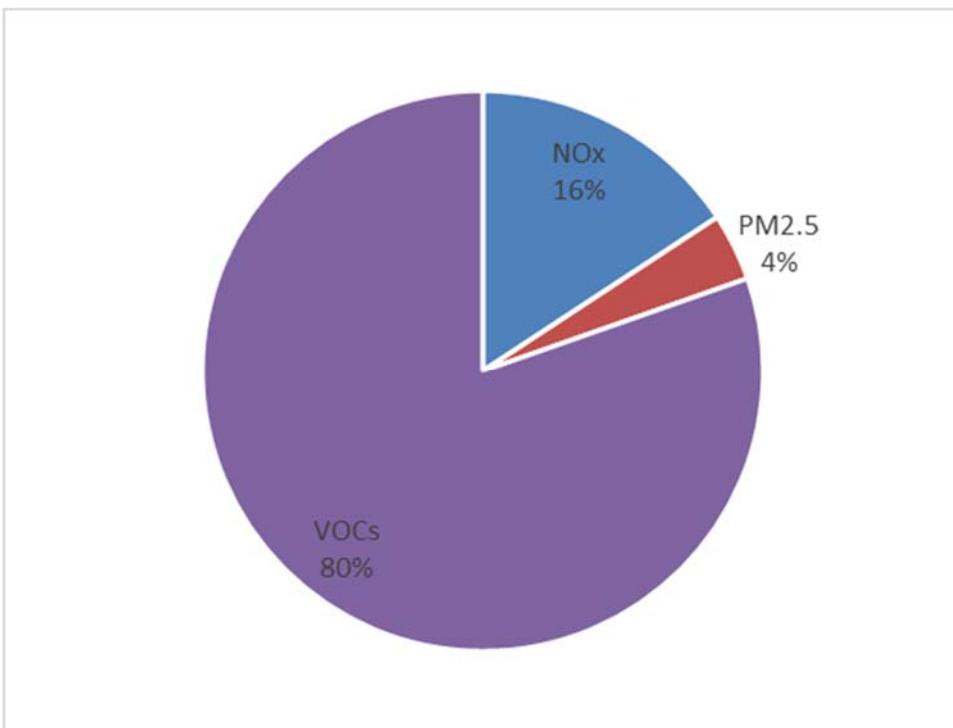


Table I is a summary of emissions information from the CAU/TATT. It shows that the largest contribution to NOx is from internal combustion (IC) engines and boilers, the largest contribution of PM<sub>2.5</sub> is from fireplaces, hydronic heaters and woodstoves, and the largest contribution of VOCs is from architectural, motor vehicle and other coatings. This last source is also the largest contributor to non-point sources, and to total emissions in general, whereas IC engines and boilers are a substantial contributor to non-EGU point sources, and to emissions in general contributing almost 40 percent of total emissions.

**Table I. Summary of NEI 2011 Criteria Emissions for CAU/TATT Counties**

Pollutant	Sector	NEI 2011 Emissions (tons)	Percent from Pollutant	Percent from Sector	Percent from Total	Major Sources
<b>NOx</b>	<b>Non-point</b>	782	22%	16%	10%	Residential, commercial and institutional NG use
<b>PM<sub>2.5</sub></b>	<b>Non-point</b>	193	74%	4%	2%	Fireplaces, hydronic heaters and woodstoves
<b>VOCs</b>	<b>Non-point</b>	3,990	86%	80%	50%	Architectural, motor vehicle and other coatings
<b>NOx</b>	<b>Point-non-EGU</b>	1,535	78%	50%	19%	IC engines and boilers*
<b>PM<sub>2.5</sub></b>	<b>Point-non-EGU</b>	63	26%	2%	1%	ICI coal powered boilers**
<b>SO<sub>2</sub></b>	<b>Point-non-EGU</b>	966	100%	32%	12%	ICI coal powered boilers**
<b>VOCs</b>	<b>Point-non-EGU</b>	479	14%	16%	6%	Paper coating
<b>NOx</b>	<b>Total</b>	2,317	100%		29%	
<b>PM<sub>2.5</sub></b>	<b>Total</b>	256	100%		3%	
<b>SO<sub>2</sub></b>	<b>Total</b>	966	100%		12%	
<b>VOCs</b>	<b>Total</b>	4,470	100%		56%	
<b>Total</b>	<b>Non-point</b>	4,965		100%	62%	
<b>Total</b>	<b>Point-non-EGU</b>	3,044		100%	38%	
<b>Total</b>	<b>Total</b>	8,009			100%	

Note: \*IC engines are internal combustion engines. \*\* ICI coal powered boilers are Industrial/Commercial/Institutional coal powered boilers.

## CAU/TATT CoST Analysis Results

The result of applying CoST to the 2011 NEI towards maximum emissions reductions of all four criteria pollutants (NOx, PM<sub>2.5</sub>, SO<sub>2</sub> and VOC) was about \$20 million dollars (in 2011 dollars). Table 2 summarizes the results. Costs of reductions by pollutant were as follows: NOx at \$2 million (10 percent of total cost) and 1,600 tons reduced; PM<sub>2.5</sub> at \$2 million (10 percent of total cost) and 200 tons reduced; SO<sub>2</sub> at \$3 million (14 percent of total cost) and 800 tons reduced; and VOC at \$13 million (66 percent of total cost) and almost 3,000 tons reduced. Non-EGU point source reductions were almost \$8 million (40 percent of the total cost). Non-point source reductions amounted to \$12 million dollars (60 percent of total cost).

**Table 2. Summary of CoST Results for CAU/TATT Counties**

Pollutant	Sector	Annual Cost (2011 \$)	Percent of total cost	Emissions Reductions (tons)	Average Cost per Ton	Percent emissions reductions	Percent reductions from Pollutant	Major Reduction Technologies
NOx	Non-point	\$ 551,431	3%	347	\$ 1,591	44%	22%	Low NOX burner
PM <sub>2.5</sub>	Non-point	\$ 1,335,943	7%	166	\$ 8,069	86%	74%	New gas stoves, Burn ban
VOCs	Non-point	\$ 10,213,138	51%	2,344	\$ 4,357	59%	86%	Reformulation
NOx	Point-non-EGU	\$ 1,389,517	7%	1,241	\$ 1,119	81%	78%	Low emission combustion
PM <sub>2.5</sub>	Point-non-EGU	\$ 755,964	4%	57	\$ 13,321	90%	26%	Dry injection / Fabric filters
SO <sub>2</sub>	Point-non-EGU	\$ 2,857,715	14%	766	\$ 3,729	79%	100%	Dry injection, fabric filters, wet scrubbers
VOCs	Point-non-EGU	\$ 2,941,515	15%	383	\$ 7,676	80%	14%	Permanent total enclosures
NOx	Total	\$ 1,940,948	10%	1,588	\$ 1,222	69%	100%	
PM <sub>2.5</sub>	Total	\$ 2,091,907	10%	222	\$ 9,409	87%	100%	
SO <sub>2</sub>	Total	\$ 2,857,715	14%	766	\$ 3,729	79%	100%	
VOCs	Total	\$ 13,154,653	66%	2,728	\$ 4,823	61%	100%	
Total	Non-point	\$ 12,100,512	60%	2,857	\$ 4,236	58%		
Total	Point-non-EGU	\$ 7,944,711	40%	2,448	\$ 3,246	80%		
Total	Total	\$ 20,045,223	100%	5,304	\$ 3,779	66%		

Note: This cost is annualized at a 3 percent discount rate.

For NOx point sources almost 70 percent of the emissions reductions came from Low Emission Combustion at 3 percent of the total cost. About 20 percent of non-point NOx reductions came from Low NOx burners. Point source PM<sub>2.5</sub> reductions from dry injection and fabric filters were 26 percent of the reductions at 4 percent of the total cost. For non-point PM<sub>2.5</sub>, new gas stoves were 38 percent of the reductions at 3 percent of the overall cost, while open burning curtailment was 36 percent of reductions at 4 percent of total cost. SO<sub>2</sub> point source emissions reductions came from dry injection, fabric filter systems, and wet scrubbers, and were 15 percent of total costs. No SO<sub>2</sub> non-point source controls were applied. Finally, for VOCs, permanent total enclosures were 12 percent of point source reductions at 14 percent of the costs, and reformulation control measures were 43 percent of non-point source reductions at 42 percent of costs.

School bus anti-idling emission reductions were also estimated using county level school bus fleet numbers and documented emission rates for diesel buses<sup>13</sup>. The total reductions for this pollution reduction strategy were relatively small (NOx emissions reductions totaled ~3 TPY and PM<sub>2.5</sub> reductions totaled ~.1 TPY, for instance). These reductions, though small, can be significant when looking at nearby areas around schools where there are potentially sensitive populations. However, due to the small scale of these reductions, they were not included in the photochemical modeling.

## Geographic Distribution of CAU/TATT Emissions Reductions

Figure 4 shows NOx reductions in CAU/TATT counties. Almost 1,300 tons of NOx reductions came from Spartanburg County, with Greenville County following at 200 tons. The remaining counties had 50 or less tons reductions.

<sup>13</sup> EPA - OTC report, Average In-Use Emissions from Urban Buses and School Buses. October 2008. <https://www3.epa.gov/otaq/consumer/420f08026.pdf>

**Figure 4. NOx Emissions Reductions in CAU/TATT Counties from Point and Non-point Sources**

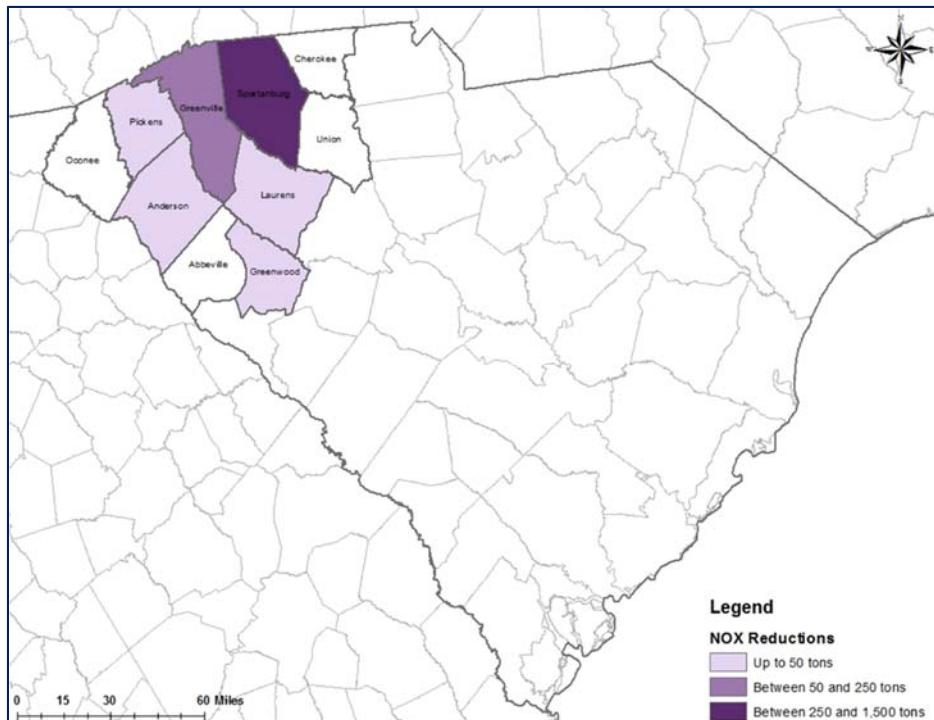
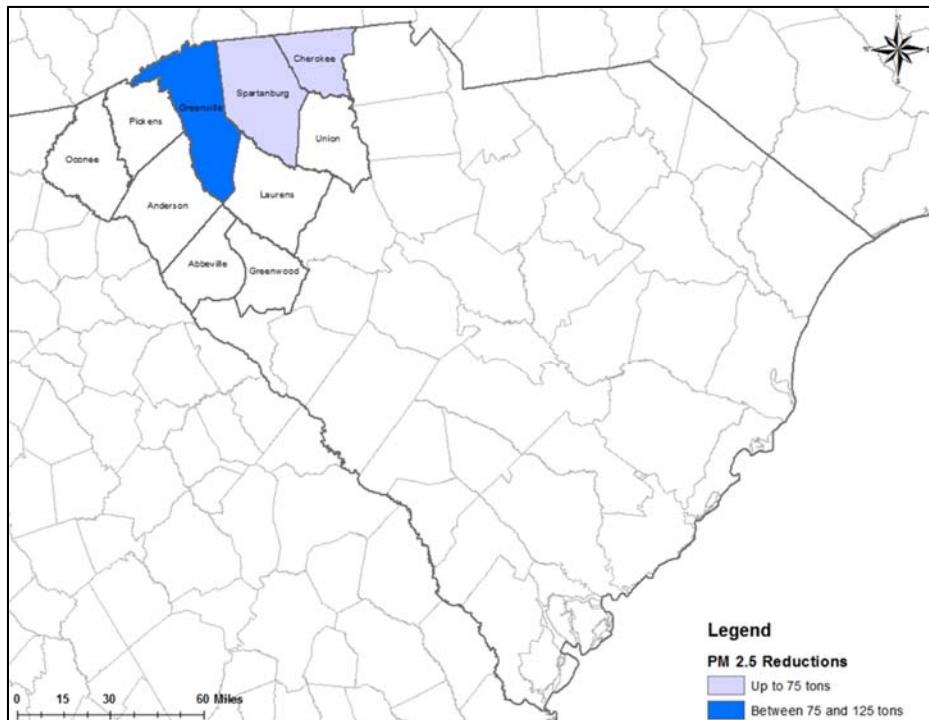


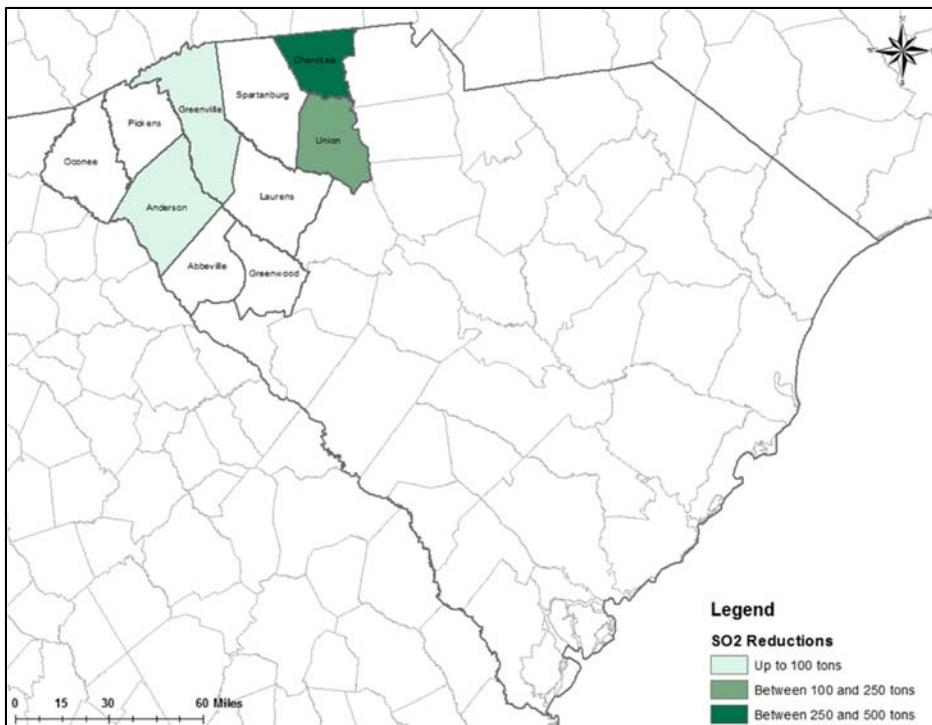
Figure 5 shows that PM<sub>2.5</sub> reductions took place in only three counties. The highest reductions of almost 115 tons happened in Greenville County, while Cherokee and Spartanburg Counties saw reductions close to 60 and 50 tons respectively.

**Figure 5. PM<sub>2.5</sub> Emissions Reductions in CAU/TATT Counties from Point and Non-point Sources**



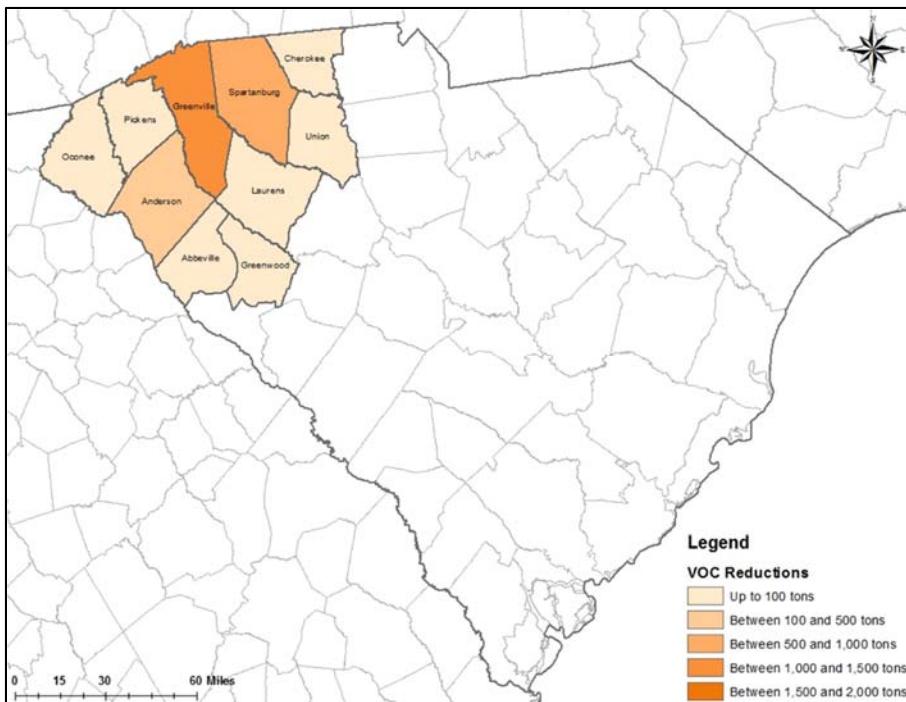
SO<sub>2</sub> Reductions are shown in Figure 6. Cherokee (460 tons) and Union Counties had the highest reductions at 460 and 120 tons respectively. Anderson County had reductions of 100 tons whereas Greenville County had 90 tons of PM<sub>2.5</sub> reductions.

**Figure 6. SO<sub>2</sub> Emissions Reductions in CAU/TATT Counties from Point and Non-point Sources**



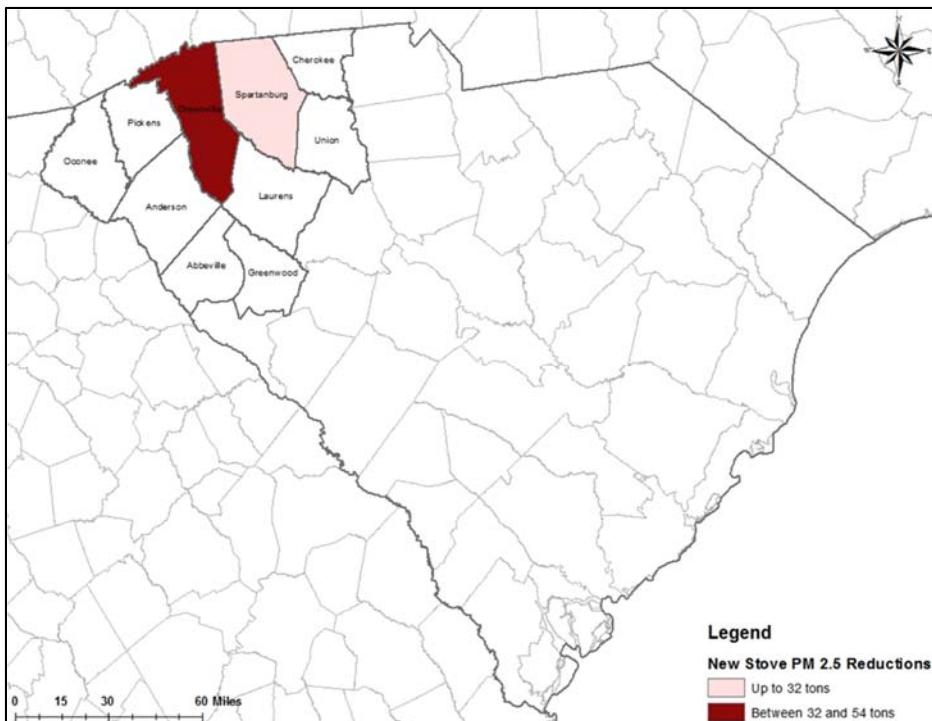
All CAU/TATT counties saw VOC reductions in the analysis. Greenville and Spartanburg Counties had the highest reductions at 1,200 tons and 700 tons respectively. Anderson County had almost 500 tons reductions. The remaining counties had between less than 200 and more than 30 tons reductions. VOC reductions are shown in Figure 7.

**Figure 7. VOC Emissions Reductions in CAU/TATT Counties from Point and Non-point Sources**

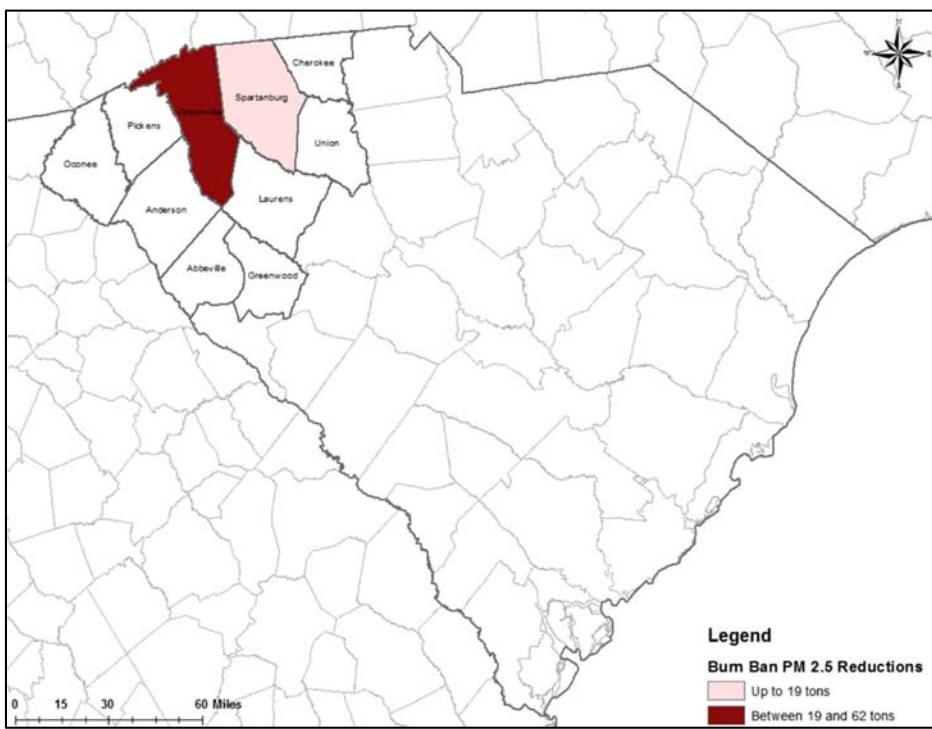


New gas stoves or gas logs and open burning curtailment were emissions controls considered as options by DHEC. Figures 8 and 9 show emissions reductions from their application in CAU/TATT counties. In both cases emissions reductions took place in Greenville and Spartanburg Counties, although reductions were small as compared to other controls. Greenville County saw about 50 tons of PM<sub>2.5</sub> reductions from new stoves or gas logs and about 60 tons reductions from open burning curtailment. For Spartanburg PM<sub>2.5</sub> reductions were 30 and 20 tons respectively.

**Figure 8. PM<sub>2.5</sub> Emissions Reductions in CAU/TATT Counties from New Gas Stoves or Gas Logs**

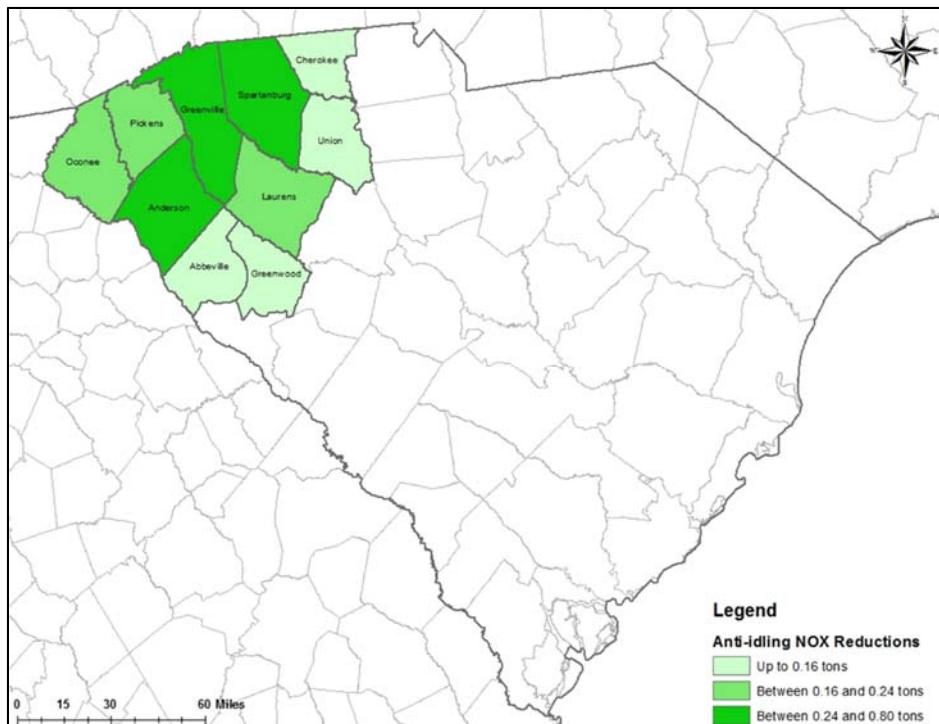


**Figure 9. PM<sub>2.5</sub> Emissions Reductions in CAU/TATT Counties from Open Burning Curtailment**



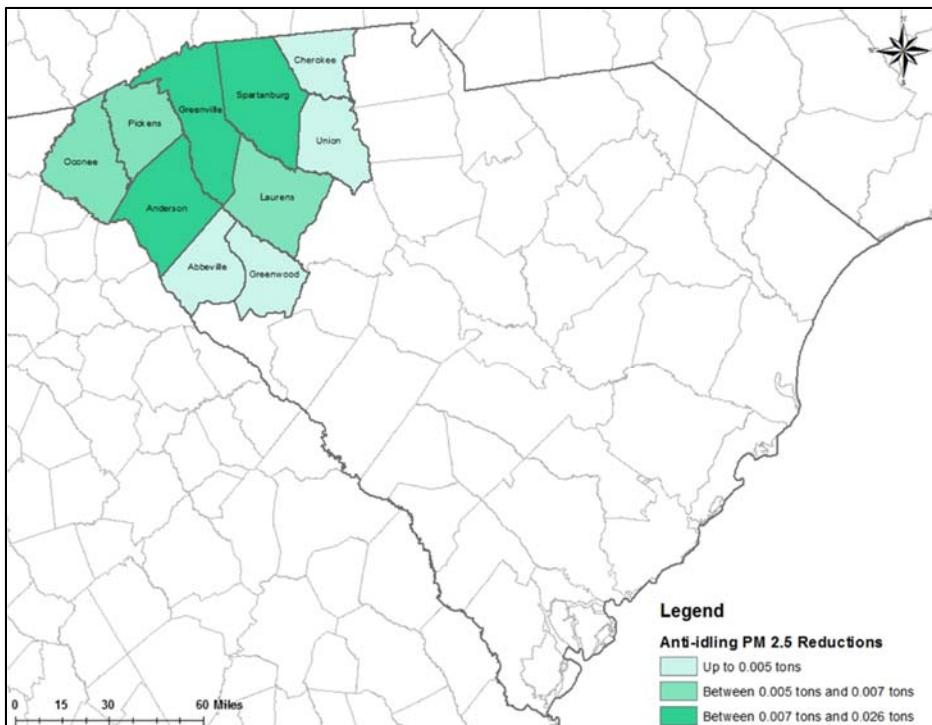
As explained in the previous section, South Carolina conducted a separate emissions reductions analysis for school bus anti-idling programs. Figures 10 to 12 show NOx, PM<sub>2.5</sub>, and VOC reductions from this measure, with all ten counties showing reductions of all three pollutants. Emissions reductions ranged between 0.6 and 0.16 tons of NOx with Greenville, Spartanburg and Anderson Counties showing the highest reductions of almost 0.8, .60 and almost 0.5 tons respectively.

**Figure 10. NOx Emissions Reductions in CAU/TATT Counties from School Bus Anti-Idling**



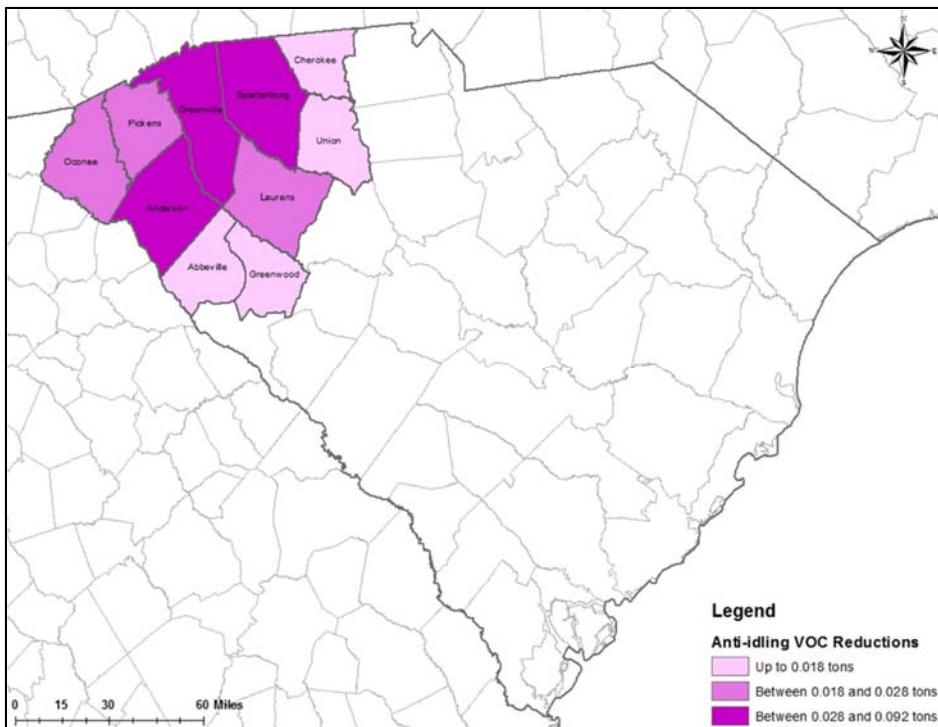
PM<sub>2.5</sub> reductions from bus anti-idling happened mostly in Greenville, Spartanburg and Andersonville Counties, of 0.025, 0.020, and 0.015 tons reduced respectively (Figure 11). Other county reductions ranged between 0.002 and 0.004 tons.

**Figure 11. PM<sub>2.5</sub> Emissions Reductions in CAU/TATT Counties from School Bus Anti-Idling**



VOC emissions reductions from school bus anti-idling also happened, in their majority, in Greenville (0.090 tons), Spartanburg (0.07 tons) and Anderson (almost 0.06 tons) Counties. Other county VOC reductions ranged between 0.008 and almost 0.03 tons. See Figure 12.

**Figure 12. VOC Emissions Reductions in CAU/TATT Counties from School Bus Anti-Idling**



## Appendix E: South Carolina Ten at the Top Counties CMAQ Modeling

### Introduction

The photochemical model simulations for this emission reduction strategy used the Community Multiscale Air Quality Model (CMAQ) version 5.0.2 which is a three-dimensional air quality model designed to simulate the formation photochemical and secondarily formed pollutants such as ozone and PM<sub>2.5</sub> over regional spatial scales (<https://www.cmascenter.org/cmaq/>). This simulation used the 2011 National Emissions Inventory (NEI) Modeling Platform Version 2 applied at a horizontal scale of 12 x 12 km on 100 x 100 cell grid centered around the "Upstate" of South Carolina to assess primary and secondary formed criteria pollutants and was run for the entire 2011 year. A "brute-force" emission reduction evaluation method was used to assess criteria pollutant reductions. This method compares the difference between the base case and a test case which includes emission reduction strategies using two model runs.

EPA provided merged and unmerged 2011 NEI CMAQ ready emissions files for the base case. For the test case, emissions inventory files produced by the CoST tool were provided by EPA for non-EGU point source and area source sectors. For a summary of total reductions between the base case and test case, see Appendix D: South Carolina Ten at the Top Counties Cost Analysis. Source sectors involved in the emissions reductions test case strategy were processed by DHEC using the Sparse Matrix Operating Kernel Emissions (SMOKE) (<https://www.cmascenter.org/smoke/>) program. Meteorological data used for the simulation was processed using Weather and Research Forecasting (WRF) model version 3.4 and the Meteorology-Chemistry Interface Processor (MCIP) version 4.2. In order to reduce run time and file sizes, a sub-CONUS domain was chosen (shown in Figure I below). Modeling smaller domains can sometimes be less accurate if boundary conditions are not represented effectively, so boundary conditions for the project domain were extracted from the EPA's 2011 12 km NATA model runs. The base case modeling run was evaluated for model performance by comparing outputs to observational data and was found to be acceptable based on recent air quality policy applications.<sup>14,15</sup> Summary information for mean bias (MB), mean gross error (MGE), normalized mean bias (NMB), normalized mean gross error (NMGE), root mean squared error (RMSE) and the Pearson correlation coefficient (r) for hourly ozone and quarterly PM<sub>2.5</sub> data is included in Tables I and 2. A bubble plot for mean bias for ozone, evaluated at monitor sites within the domain is included as Figure I.

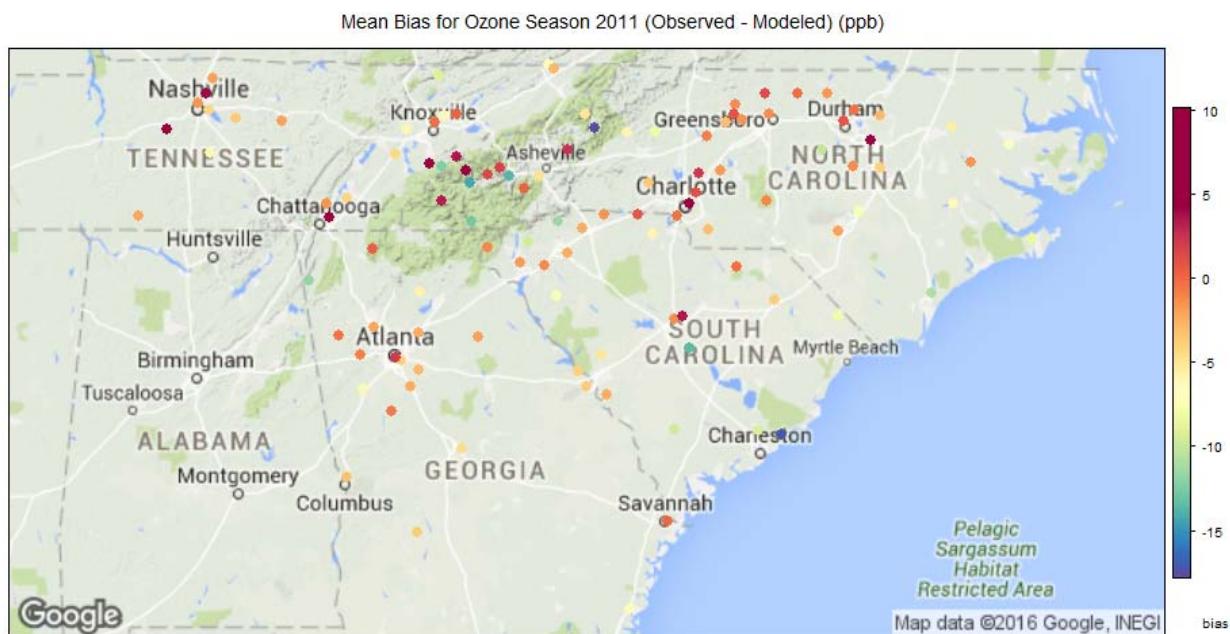
**Table I. Performance Statistics for Hourly Ozone**

MB	MGE	NMB	NMGE	RMSE	r
<b>3.298263</b>	10.05897	0.097767	0.298166	13.08218	0.731095

<sup>14</sup> Simon, H., K.R. Baker, and S.B. Phillips, 2012. Compilation and Interpretation of Photochemical Model Performance Statistics Published between 2006 and 2012, Atmospheric Environment, 61, 124-139.

<sup>15</sup> EPA, Air Quality Modeling Technical Support Document for the 2008 Ozone NAAQS Cross-State Air Pollution Rule Proposal, November 2015

**Figure 1. Ozone Mean Bias at Monitors in the Test Domain**



**Table 2. Performance Statistics for Quarterly PM<sub>2.5</sub>**

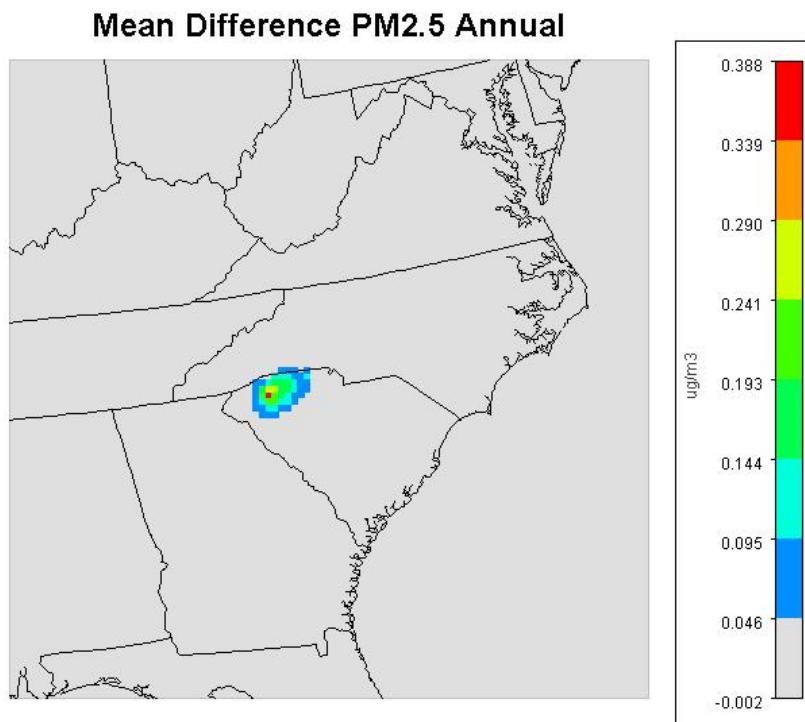
MB	MGE	NMB	NMGE	RMSE	r
<b>-0.07738</b>	0.745125	-0.00714	0.068779	0.825169	0.940994

Base case and test case reductions of ozone and PM<sub>2.5</sub> were quantified using the Modeled Attainment Test Software (MATS) ([https://www3.epa.gov/scram001/modelingapps\\_mats.htm](https://www3.epa.gov/scram001/modelingapps_mats.htm)) which calculates the relative reduction factors of design values at target monitoring locations.

### Modeled PM<sub>2.5</sub> Reductions

The following results show the modeled PM<sub>2.5</sub> reductions that took place between base case and test case strategies at the PM<sub>2.5</sub> monitors in the Upstate. A spatial representation of reductions of mean annual PM<sub>2.5</sub> is included as Figure 2.

**Figure 2. Mean Annual Reductions in PM<sub>2.5</sub> Concentrations**



PM<sub>2.5</sub> reductions for the annual standard were calculated using MATS. These reductions are at around 2 percent (%) at the two monitors operating in the study area (Table 3).

**Table 3. PM<sub>2.5</sub> Annual Standard Reductions at CAU/TATT Monitors**

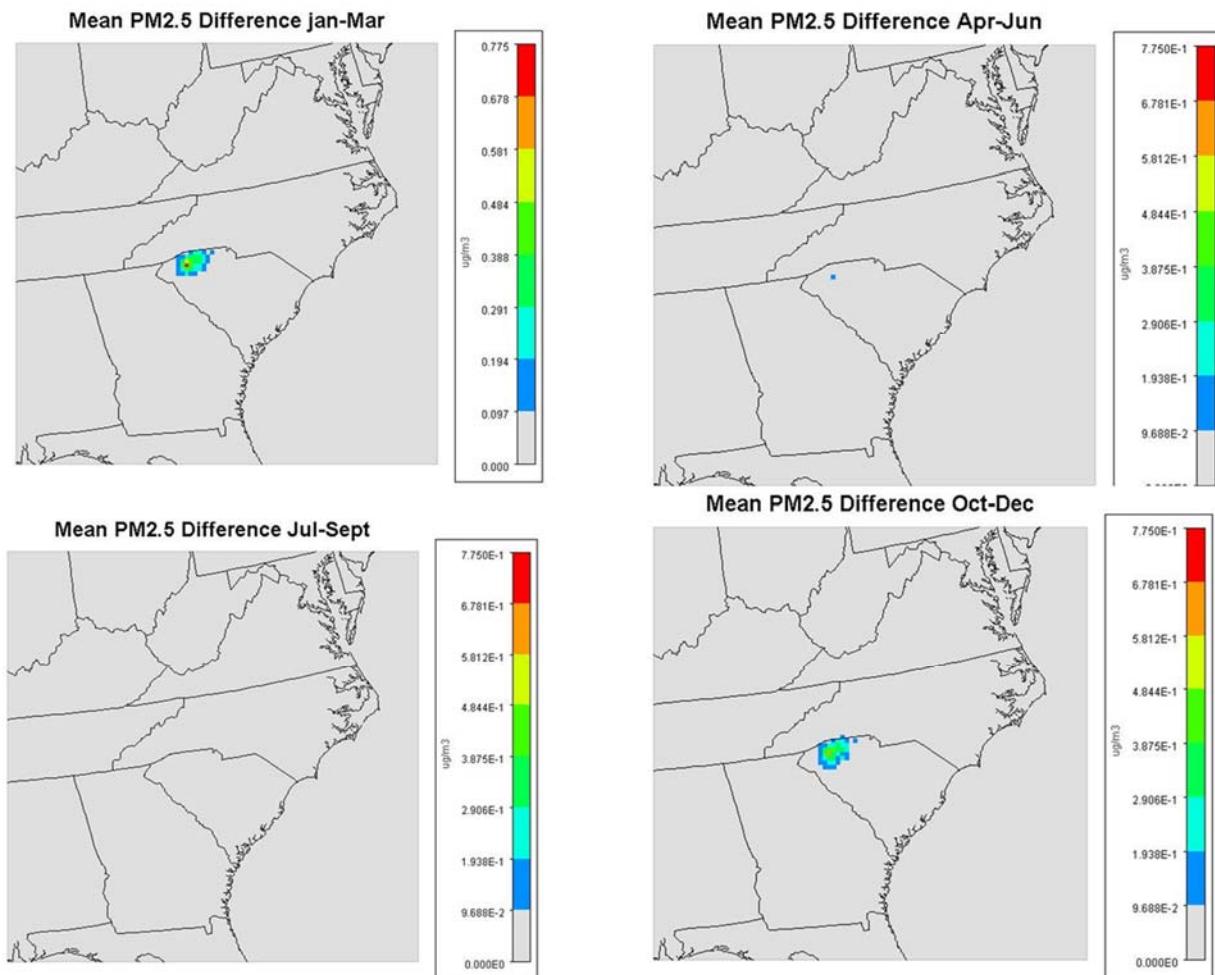
Monitor ID	Base DV	Future DV	% Reduction
450450009	10.6	10.39	1.9
450450015	10.9	10.64	2.4

The MATS software also evaluates quarterly reductions of PM<sub>2.5</sub>. Of note, temporal reductions are much higher than average in colder months (quarters 1 and 4)(Table 4 and Figure 3).

**Table 4. Quarterly Reductions in PM<sub>2.5</sub> Concentrations**

Monitor ID	Date	Base DV	Future DV	% Reduction
450450015	Q1	10.15	9.701	4.4
450450015	Q2	11.04	10.96	0.7
450450015	Q3	11.98	11.96	0.2
450450015	Q4	10.44	9.944	4.8
450450009	Q1	9.551	9.199	3.7
450450009	Q2	11.12	11.04	0.7
450450009	Q3	11.84	11.83	0.1
450450009	Q4	9.929	9.5	4.3

**Figure 3. Mean Quarterly Reductions in PM<sub>2.5</sub> Concentrations**



MATS also provides PM<sub>2.5</sub> speciated reductions. The relative reduction factors between test case and base case are higher reductions in organic carbon (Table 5).

**Table 5. Speciated PM<sub>2.5</sub> Relative Reductions Factors**

Crustal	Elemental Carbon	NH4	Organic Carbon	SO4	NO3	Water	Salt
0.999	0.9851	0.9972	0.9615	0.9982	0.9764	0.9985	0.9955
0.9991	0.9843	0.9971	0.9558	0.9982	0.9753	0.9986	0.9944

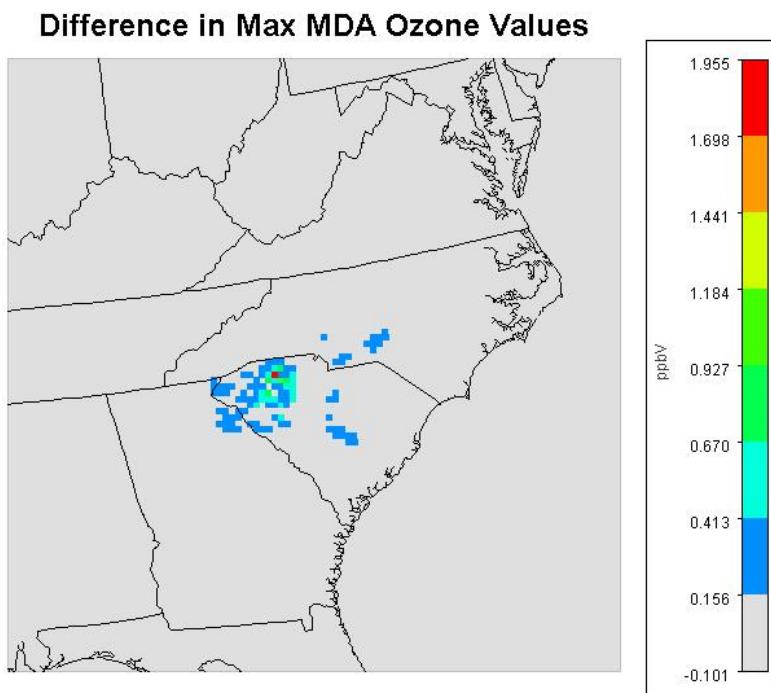
These results, taken together, may indicate that the wood stove conversion to natural gas reduction strategy may be driving the annual PM<sub>2.5</sub> emission reductions since wood combustion is linked to

atmospheric organic carbon<sup>16</sup>, and wood stoves and fireplaces are used primarily in the colder months of quarters 1 and 4. Programs which encourage wood stove and fireplace conversions to natural gas could prove to be a useful strategy to pursue to reduce PM<sub>2.5</sub> concentrations in the area.

## Ozone Reductions

The following results show the modeled ozone reductions that took place between base case and test case strategies at the ozone monitors in the Upstate. A spatial representation of reductions of maximum 8-hour daily max (MDA8) ozone concentrations, during ozone season (April 1 through October 31), is included as Figure 4.

**Figure 4. Maximum MDA8 Ozone Reductions**



The largest domain wide reduction in ozone was approximately 2 ppb, but most of the area saw less than a 1 ppb reduction in ozone. This is the maximum difference; the form of the standard is 4th high maximum value.

Ozone reductions using the MATS software use the 4th high maximum value consistent with the standard. The following results show the modeled ozone reductions between base case and test case that took place at ozone monitors in the Upstate area (Table 6).

---

<sup>16</sup> EPA, Particulate Matter (PM<sub>2.5</sub>) Speciation Guidance Document, July 22, 1998.  
<http://www3.epa.gov/ttnamtl/files/ambient/pm25/spec/specpln2.pdf>

**Table 6. 4th High MDA8 Reductions at CAU/TATT Monitors**

Monitor_ID	Monitor_Name	Base_DV	Future_DV	% Reduction
450010001	Due West	62	61.7	0.48
450070005	Big Creek	70	69.8	0.29
450210002	Cowpens	67.3	67.2	0.15
450450016	Hillcrest	68	67.3	1.03
450451003	Famoda Farms	65.3	65.2	0.15
450730001	Long Creek	64.5	64.4	0.16
450770002	Clemson	69.7	69.5	0.29
450770003	Wolf Creek	69	68.8	0.29
450830009	North Spartanburg	73.7	73.3	0.54

While maximum daily ozone reductions can be as high as approximately 2 ppb, design value reductions are typically less than 1 ppb at the monitors (less than a 1 percent (%) reduction). As such, the reduction strategies tested in this exercise do not seem to be very effective at reducing ozone concentrations, and other reductions strategies should be identified in the case of demonstration attainment.

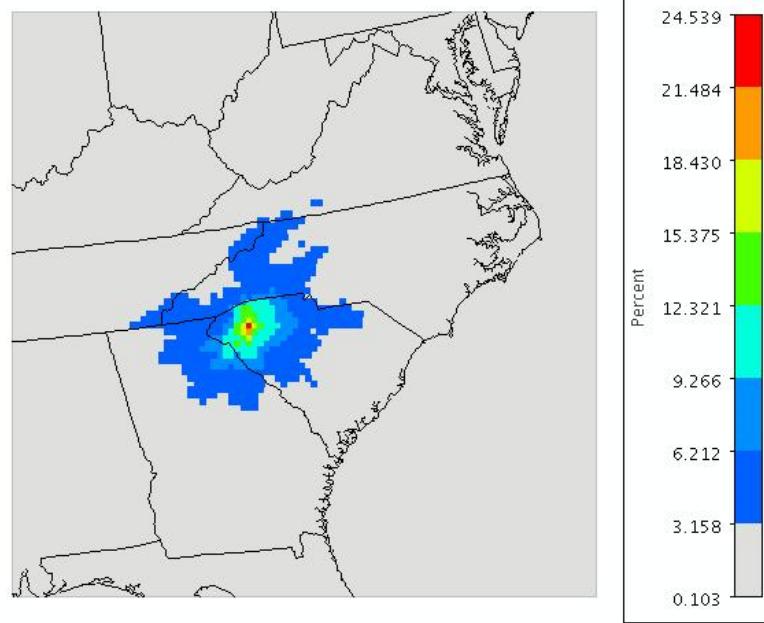
## *Other Pollutants*

### *Volatile Organic Compounds*

The following two figures (Figures 5 and 6) show the maximum and mean percent decreases in VOC between the base case and the test case, respectively.

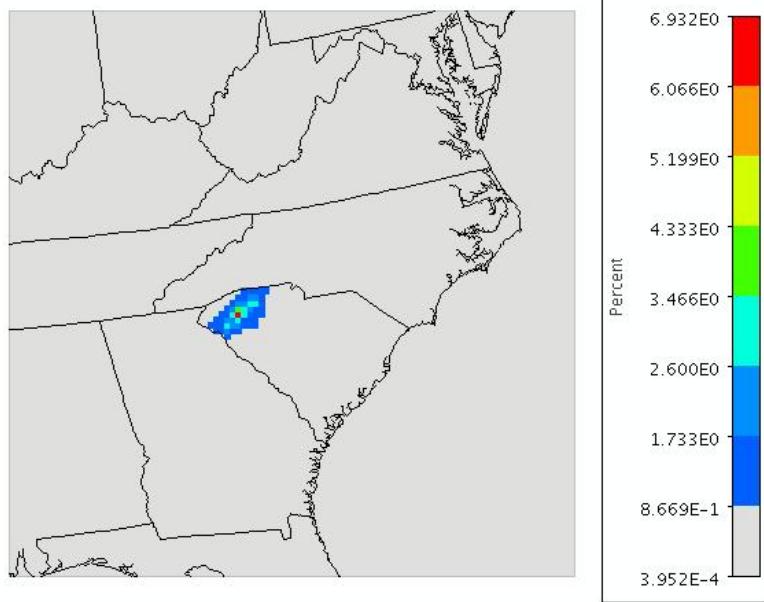
**Figure 5. Maximum Percent Decrease in VOCs**

**Maximum Percent Decrease in VOCs**



**Figure 6. Mean Percent Decrease in VOCs**

**Mean Percent Decrease in VOCs**

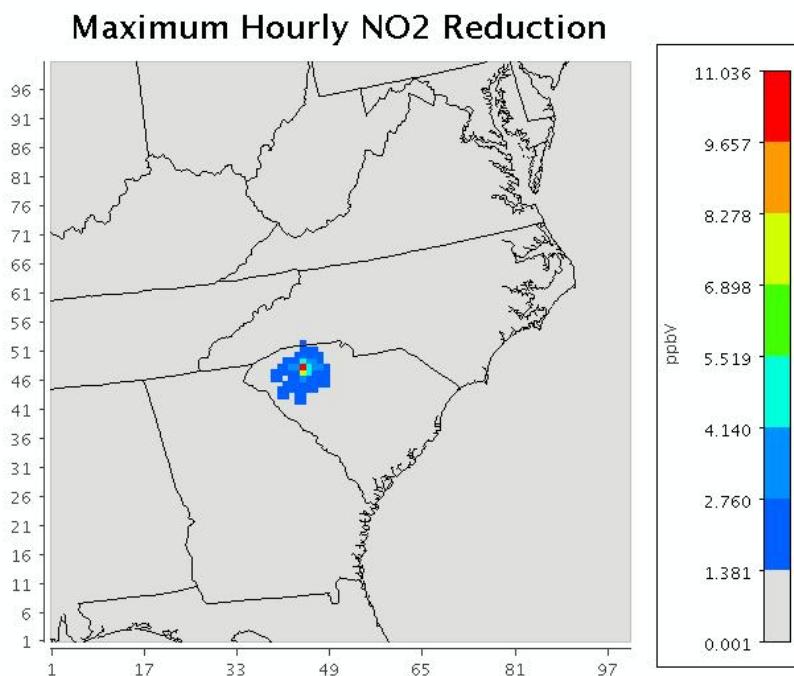


Anthropogenic ozone production in SC is primarily driven by NOx emissions<sup>17</sup>, so these reductions in VOCs have little effect on ozone reductions. However, the reduction of VOCs translates to a reduction in air toxics, which is important from a public health standpoint.

#### NO<sub>2</sub> and SO<sub>2</sub>

The following two figures (Figures 7 and 8) show the maximum decreases in NO<sub>2</sub> and SO<sub>2</sub> between the base case and the test case, respectively.

**Figure 7. Maximum Hourly NO<sub>2</sub> Reduction**

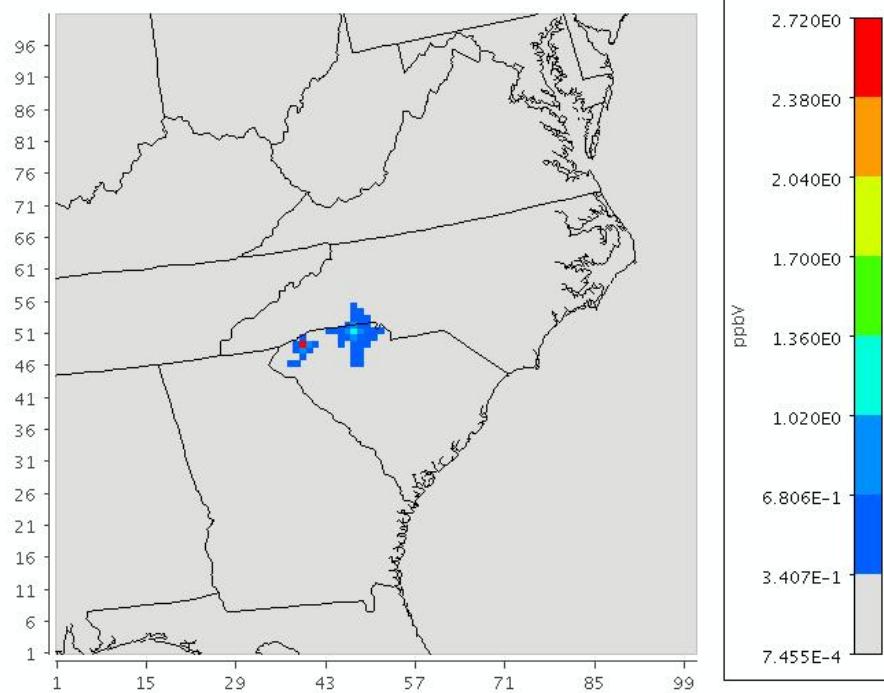


<sup>17</sup> Southeastern States Air Resource Managers, Inc., *Emissions and Air Quality Modeling for SEMAP, Final Report*. October 15, 2014.

[https://epd.georgia.gov/air/sites/epd.georgia.gov.air/files/related\\_files/document/appendix\\_d.pdf](https://epd.georgia.gov/air/sites/epd.georgia.gov.air/files/related_files/document/appendix_d.pdf)

**Figure 8. Maximum Hourly SO<sub>2</sub> Reduction**

### Maximum Hourly SO<sub>2</sub> Reduction



There were modest reductions in these criteria pollutants, with a maximum hourly reduction of around 11 ppb of NO<sub>2</sub> and just under 3 ppb of SO<sub>2</sub>.

## Appendix F: Additional Information Regarding Health-Related Benefits

### *Introduction to Benefits Analysis Methods*

In the “Ten at the Top” (TATT) analysis we follow a “damage-function” approach in calculating health co-benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare endpoints (specific effects that can be associated with changes in air quality) and estimates values of those changes assuming independence between the values of individual endpoints. Total benefits are calculated simply as the sum of the values for all non-overlapping health and welfare endpoints. The “damage-function” approach is the standard method for assessing costs and benefits of environmental quality programs and has been used in several recent published analyses (Levy et al., 2009; Hubbell et al., 2009; Tagaris et al., 2009).

Tables 1 and 2 summarize the human health and environmental benefits categories contained within the total monetized benefits estimate and those categories that were not quantified due to limited data. The list of unquantified benefit categories is not exhaustive, and neither is the quantification of each effect complete. In order to identify the most meaningful human health and environmental co-benefits, we excluded effects not identified as having at least a causal, likely causal, or suggestive relationship with the affected pollutants in the most recent comprehensive scientific assessment, such as an Integrated Science Assessment (ISA). This does not imply that additional relationships between these and other human health and environmental co-benefits and the affected pollutants do not exist. Due to this decision criterion, some effects that were identified in previous lists of unquantified benefits in other EPA Regulatory Impact Assessment (RIA) have been dropped (e.g., UVb exposure). In addition, some quantified effects represent only a partial accounting of likely impacts due to limitations in the currently available data (e.g., climate effects from CO<sub>2</sub>, etc).

**Table I. Human Health Effects of Pollutants Affected by CAU/TATT Emissions Reductions**

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	Source of More Information
Improved Human Health				
Reduced incidence of premature mortality from exposure to ozone	Premature mortality based on short-term exposure (all ages)	✓	✓	ozone ISA <sup>d</sup>
	Premature respiratory mortality based on long-term exposure (age 30–99)	—	—	
Reduced incidence of morbidity from exposure to ozone	Hospital admissions—respiratory (age > 65)	✓	✓	ozone ISA <sup>d</sup>
	Emergency department visits for asthma (all ages)	✓	✓	
	Asthma exacerbation (age 6–18)	✓	✓	
	Minor restricted-activity days (age 18–65)	✓	✓	
	School absence days (age 5–17)	✓	✓	
	Decreased outdoor worker productivity (age 18–65)	—	—	
	Other respiratory effects (e.g., medication use, pulmonary inflammation, decrements in lung functioning)	—	—	
	Cardiovascular (e.g., hospital admissions, emergency department visits)	—	—	
	Reproductive and developmental effects (e.g., reduced birthweight, restricted fetal growth)	—	—	
Reduced incidence of premature mortality from exposure to PM <sub>2.5</sub>	Adult premature mortality based on cohort study estimates (age >25 or age >30)	✓	✓	ozone ISA <sup>d</sup>
	Infant mortality (age <1)	✓	✓	
Reduced incidence of morbidity from exposure to PM <sub>2.5</sub>	Non-fatal heart attacks (age > 18)	✓	✓	ozone ISA <sup>d</sup>
	Hospital admissions—respiratory (all ages)	✓	✓	
	Hospital admissions—cardiovascular (age >20)	✓	✓	
	Emergency department visits for asthma (all ages)	✓	✓	
	Acute bronchitis (age 8–12)	✓	✓	
	Lower respiratory symptoms (age 7–14)	✓	✓	
	Upper respiratory symptoms (asthmatics age 9–11)	✓	✓	
	Asthma exacerbation (asthmatics age 6–18)	✓	✓	
	Lost work days (age 18–65)	✓	✓	
	Minor restricted-activity days (age 18–65)	✓	✓	
	Chronic Bronchitis (age >26)	—	—	

Benefits Category	Specific Effect	Effect Has Been Quantified	Effect Has Been Monetized	Source of More Information
	Emergency department visits for cardiovascular effects (all ages)	—	—	
	Strokes and cerebrovascular disease (age 50–79)	—	—	
	Other cardiovascular effects (e.g., other ages)	—	—	
	Other respiratory effects (e.g., pulmonary function, non-asthma ER visits, non-bronchitis chronic diseases, other ages and populations)	—	—	PM ISA <sup>c</sup>
	Reproductive and developmental effects (e.g., low birth weight, pre-term births, etc.)	—	—	
	Cancer, mutagenicity, and genotoxicity effects	—	—	PM ISA <sup>c,d</sup>

The benefits analysis in this chapter relies on an array of data inputs—including air quality modeling, health impact functions and valuation functions among others—which are themselves subject to uncertainty and may also contribute to the overall uncertainty in this analysis. As a means of characterizing this uncertainty we use Monte Carlo methods for characterizing random sampling error associated with the concentration response functions from epidemiological studies and economic valuation functions. Second. While the contributions from additional data inputs to uncertainty in the results are not quantified here, this analysis employs best practices in every aspect of its development.

To assess economic value in a damage-function framework, the changes in environmental quality must be translated into effects on people or on the things that people value. In some cases, the changes in environmental quality can be directly valued, as is the case for changes in visibility. In other cases, such as for changes in ozone and PM, a health and welfare impact analysis must first be conducted to convert air quality changes into effects that can be assigned dollar values.

We note at the outset that EPA rarely has the time or resources to perform extensive new research to measure directly either the health outcomes or their values for regulatory analyses. Thus, similar to Kunzli et al. (2000) and other recent health impact analyses, our estimates are based on the best available methods of benefits transfer. Benefits transfer is a means of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Adjustments are made for the level of environmental quality change, the socio-demographic and economic characteristics of the affected population, and other factors to improve the accuracy and robustness of benefits estimates.

#### *Health Impact Assessment*

Health Impact Assessment (HIA) quantifies changes in the incidence of adverse health impacts resulting from changes in human exposure to specific pollutants, such as PM<sub>2.5</sub>. HIAs are a well-established approach for estimating the retrospective or prospective change in adverse health impacts expected to result from population-level changes in exposure to pollutants (Levy et al. 2009). PC-based tools such as the environmental Benefits Mapping and Analysis Program (BenMAP) can systematize health impact analyses by applying a database of key input parameters, including health impact functions and population projections. Analysts have applied the HIA approach to estimate human health impacts resulting from

hypothetical changes in pollutant levels (Hubbell et al. 2005; Davidson et al. 2007, Tagaris et al. 2009). EPA and others have relied upon this method to predict future changes in health impacts expected to result from the implementation of regulations affecting air quality (e.g. U.S. EPA, 2015a). For this assessment, the HIAs are limited to those health effects that are directly linked to ambient PM<sub>2.5</sub> concentrations. There may be other indirect health impacts associated with implementing emissions controls, such as occupational health impacts for coal miners.

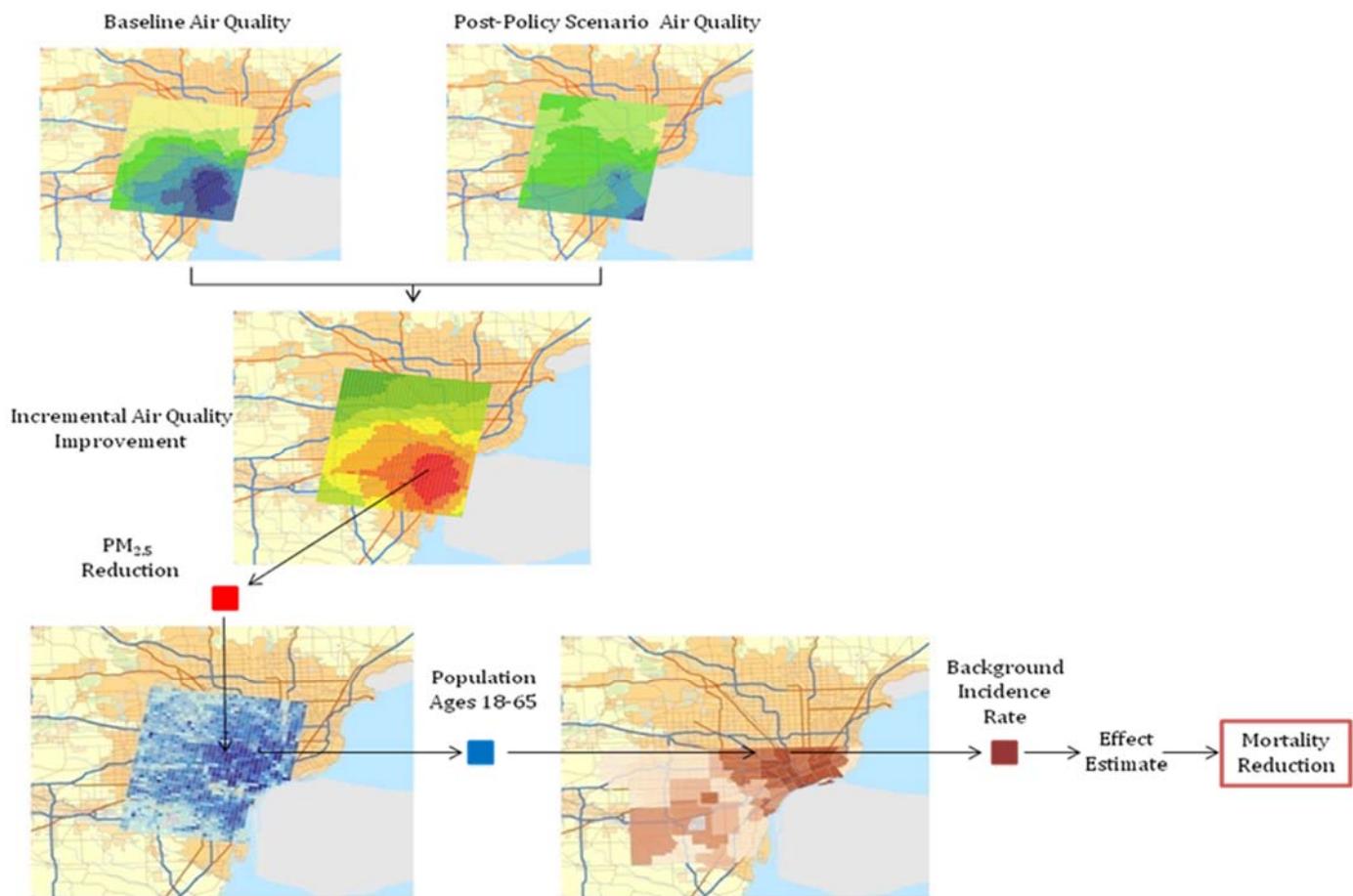
The HIA approach used in this analysis involves three basic steps: (1) utilizing CMAQ-generated projections of PM<sub>2.5</sub> and ozone air quality and estimating the change in the spatial distribution of the ambient air quality; (2) determining the subsequent change in population-level exposure; (3) calculating health impacts by applying concentration-response relationships drawn from the epidemiological literature (Hubbell et al. 2009) to this change in population exposure.

A typical health impact function might look as follows:

$$\Delta y = y_0 \cdot (e^{\beta \cdot \Delta x} - 1) \cdot Pop$$

where  $y_0$  is the baseline incidence rate for the health endpoint being quantified (for example, a health impact function quantifying changes in mortality would use the baseline, or background, mortality rate for the given population of interest);  $Pop$  is the population affected by the change in air quality;  $\Delta x$  is the change in air quality; and  $\beta$  is the effect coefficient drawn from the epidemiological study. Tools such as BenMAP can systematize the HIA calculation process, allowing users to draw upon a library of existing air quality monitoring data, population data and health impact functions. Figure A-1 provides a simplified overview of this approach.

**Figure 1. Illustration of BenMAP Approach**



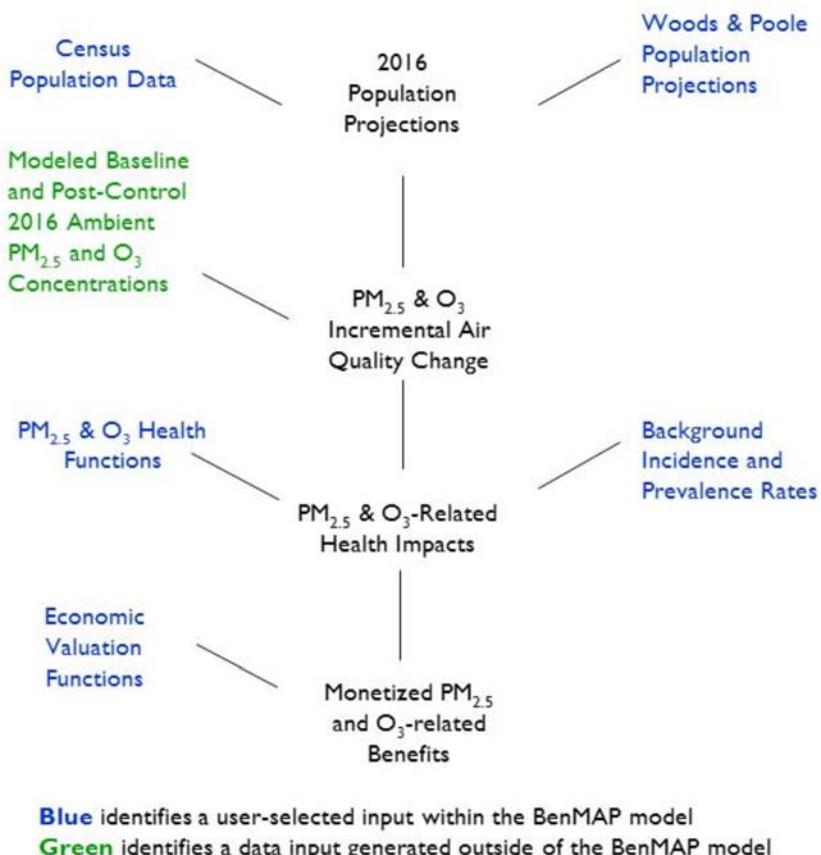
#### *Economic Valuation of Health Impacts*

After quantifying the change in adverse health impacts, the final step is to estimate the economic value of these avoided impacts. The appropriate economic value for a change in a health effect depends on whether the health effect is viewed *ex ante* (before the effect has occurred) or *ex post* (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects by a small amount for a large population. The appropriate economic measure is therefore *ex ante* Willingness to Pay (WTP) for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is \$100, then the WTP for an avoided statistical premature mortality amounts to \$1 million ( $\$100/0.0001$  change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These cost of illness (COI) estimates generally (although not in every case) underestimate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect.

We use the BenMAP-CE tool version 1.1 (U.S. EPA, 2015b) to estimate the health impacts and monetized health co-benefits for the Mercury and Air Toxics Standards. Figure A-2 shows the data inputs and outputs for the BenMAP-CE tool.

**Figure 2. Data Inputs and Outputs for the BenMAP-CE Tool**



## *Uncertainty Characterization*

As for any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty affecting estimated results, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, estimates of values (both from WTP and COI studies), population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Each of these inputs may be uncertain and, depending on its role in the co-benefits analysis, may have a disproportionately large impact on estimates of total monetized co-benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emission levels can lead to large impacts on total monetized co-benefits.

The National Research Council (NRC) (2002, 2008) highlighted the need for EPA to conduct rigorous quantitative analysis of uncertainty in its benefits estimates and to present these estimates to decision makers in ways that foster an appropriate appreciation of their inherent uncertainty. In general, the NRC concluded that EPA's methodology for calculating the benefits of reducing air pollution is reasonable and informative in spite of inherent uncertainties. Since the publication of these reports, EPA continues to improve the characterization of uncertainties for both health incidence and benefits estimates. We use a Monte Carlo analysis to assess uncertainty quantitatively, as well as to provide a qualitative assessment for those aspects that we are unable to address quantitatively.

We used Monte Carlo methods to characterize both sampling error and variability across the economic valuation functions, including random sampling error associated with the concentration response functions from epidemiological studies and random effects modeling. Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables, such as incidence of premature mortality. Specifically, we used Monte Carlo methods to generate confidence intervals around the estimated health impact and dollar benefits. The reported standard errors in the epidemiological studies determined the distributions for individual effect estimates.

In benefit analyses of air pollution regulations conducted to date, the estimated impact of reductions in premature mortality has accounted for 85 percent to 95 percent of total monetized benefits. Therefore, it is particularly important to attempt to characterize the uncertainties associated with reductions in premature mortality. The health impact functions used to estimate avoided premature deaths associated with reductions in ozone have associated standard errors that represent the statistical errors around the effect estimates in the underlying epidemiological studies. In our results, we report credible intervals based on these standard errors, reflecting the uncertainty in the estimated change in incidence of avoided premature deaths. We also provide multiple estimates, to reflect model uncertainty between alternative study designs. EPA estimates PM-related mortality without assuming a health effect threshold at low concentrations, based on the current body of scientific literature (U.S. EPA-SAB, 2009a, U.S. EPA-SAB, 2009b).

Key sources of uncertainty in the PM<sub>2.5</sub> health impact assessment include:

- gaps in scientific data and inquiry;
- variability in estimated relationships, such as epidemiological effect estimates, introduced through differences in study design and statistical modeling;

- errors in measurement and projection for variables such as population growth rates;
- errors due to misspecification of model structures, including the use of surrogate variables, such as using PM<sub>10</sub> when PM<sub>2.5</sub> is not available, excluded variables, and simplification of complex functions;
- biases due to omissions or other research limitations; and
- additional uncertainties from benefits transfer method using BPT estimates.

In Table 4, we summarize some of the key uncertainties in the benefits analysis.

**Table 4. Primary Sources of Uncertainty in the Benefits Analysis**

*1. Uncertainties Associated with Impact Functions*

- The value of the ozone or PM effect estimate in each impact function.
- Application of a single impact function to pollutant changes and populations in all locations.
- Similarity of future-year impact functions to current impact functions.
- Correct functional form of each impact function.
- Extrapolation of effect estimates beyond the range of ozone or PM concentrations observed in the source epidemiological study.
- Application of impact functions only to those subpopulations matching the original study population.

*2. Uncertainties Associated with CMAQ-Modeled Ozone and PM Concentrations*

- Responsiveness of the models to changes in precursor emissions from the control policy.
- Projections of future levels of precursor emissions, especially ammonia and crustal materials.
- Lack of ozone and PM<sub>2.5</sub> monitors in all rural areas requires extrapolation of observed ozone data from urban to rural areas.

*3. Uncertainties Associated with PM Mortality Risk*

- Limited scientific literature supporting a direct biological mechanism for observed epidemiological evidence.
- Direct causal agents within the complex mixture of PM have not been identified.
- The extent to which adverse health effects are associated with low-level exposures that occur many times in the year versus peak exposures.
- The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study.
- Reliability of the PM<sub>2.5</sub> monitoring data in reflecting actual PM<sub>2.5</sub> exposures.

*4. Uncertainties Associated with Possible Lagged Effects*

- The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels that would occur in a single year is uncertain as well as the portion that might occur in subsequent years.

*5. Uncertainties Associated with Baseline Incidence Rates*

- Some baseline incidence rates are not location specific (e.g., those taken from studies) and therefore may not accurately represent the actual location-specific rates.
- Current baseline incidence rates may not approximate well baseline incidence rates in 2016.
- Projected population and demographics may not represent well future-year population and demographics.

*6. Uncertainties Associated with Economic Valuation*

- Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them.
- Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates because of differences in income or other factors.

*7. Uncertainties Associated with Aggregation of Monetized Benefits*

- Health and welfare benefits estimates are limited to the available impact functions. Thus, unquantified or unmonetized benefits are not included.

$\text{PM}_{2.5}$  mortality benefits represent a substantial proportion of total monetized co-benefits (over 90 percent), and these estimates have following key assumptions and uncertainties.

1. We assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because  $\text{PM}_{2.5}$  produced via transported precursors emitted from EGUs may differ significantly from direct  $\text{PM}_{2.5}$  released from diesel engines and other industrial sources, but the scientific evidence is not yet sufficient to allow differential effects estimates by particle type.
2. We assume that the health impact function for fine particles is linear within the range of ambient concentrations under consideration. Thus, the estimates include health co-benefits from reducing fine particles in areas with varied concentrations of  $\text{PM}_{2.5}$ , including both regions that are in attainment with fine particle standard and those that do not meet the standard down to the lowest modeled concentrations.

## *Benefits Analysis Data Inputs*

In Figure 5-2, we summarized the key data inputs to the health impact and economic valuation estimate. Below we summarize the data sources for each of these inputs, including demographic projections, effect coefficients, incidence rates and economic valuation. Our approach here is generally consistent with the Regulatory Impact Analysis for the Ozone NAAQS RIA (U.S. EPA, 2015a).

### *Demographic Data*

Quantified and monetized human health impacts depend on the demographic characteristics of the population, including age, location, and income. We use projections based on economic forecasting models developed by Woods and Poole, Inc. (Woods and Poole, 2008). The Woods and Poole (WP) database contains county-level projections of population by age, sex, and race out to 2030. Projections in each county are determined simultaneously with every other county in the United States to take into account patterns of economic growth and migration. The sum of growth in county-level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates (Hollman et al., 2000). According to WP, linking county-level growth projections together and constraining to a national-level total growth avoids potential errors introduced by forecasting each county independently. County projections are developed in a four-stage process:

1. First, national-level variables such as income, employment, and populations are forecasted.
2. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an “export-base” approach, which relies on linking industrial-sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-based approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector.
3. Third, population is projected for each economic area based on net migration rates derived from employment opportunities and following a cohort-component method based on fertility and mortality in each area.
4. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or

county are determined by aging the population by single year of age by sex and race for each year through 2016 based on historical rates of mortality, fertility, and migration.

## *Effect Coefficients*

The first step in selecting effect coefficients is to identify the health endpoints to be quantified. We base our selection of health endpoints on consistency with EPA's Integrated Science Assessments (which replace the Criteria Document), with input and advice from the EPA Science Advisory Board - Health Effects Subcommittee (SAB-HES), a scientific review panel specifically established to provide advice on the use of the scientific literature in developing benefits analyses for air pollution regulations (<http://www.epa.gov/sab/>). In general, we follow a weight of evidence approach, based on the biological plausibility of effects, availability of concentration-response functions from well conducted peer-reviewed epidemiological studies, cohesiveness of results across studies, and a focus on endpoints reflecting public health impacts (like hospital admissions) rather than physiological responses (such as changes in clinical measures like Forced Expiratory Volume (FEV1)).

There are several types of data that can support the determination of types and magnitude of health effects associated with air pollution exposures. These sources of data include toxicological studies (including animal and cellular studies), human clinical trials, and observational epidemiology studies. All of these data sources provide important contributions to the weight of evidence surrounding a particular health impact. However, only epidemiology studies provide direct concentration-response relationships which can be used to evaluate population-level impacts of reductions in ambient pollution levels in a health impact assessment.

For the data-derived estimates, we relied on the published scientific literature to ascertain the relationship between PM and adverse human health effects. We evaluated epidemiological studies using the selection criteria summarized in Table 5. These criteria include consideration of whether the study was peer-reviewed, the match between the pollutant studied and the pollutant of interest, the study design and location, and characteristics of the study population, among other considerations. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility. In general, the use of results from more than a single study can provide a more robust estimate of the relationship between a pollutant and a given health effect. However, there are often differences between studies examining the same endpoint, making it difficult to pool the results in a consistent manner. For example, studies may examine different pollutants or different age groups. For this reason, we consider very carefully the set of studies available examining each endpoint and select a consistent subset that provides a good balance of population coverage and match with the pollutant of interest. In many cases, either because of a lack of multiple studies, consistency problems, or clear superiority in the quality or comprehensiveness of one study over others, a single published study is selected as the basis of the effect estimate.

**Table 5. Criteria Used When Selecting C-R Functions**

Consideration	Comments
Peer-Reviewed Research	Peer-reviewed research is preferred to research that has not undergone the peer-review process.
Study Type	Among studies that consider chronic exposure (e.g., over a year or longer), prospective cohort studies are preferred over ecological studies because they control for important individual-level confounding variables that cannot be controlled for in ecological studies.
Study Period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and lifestyle over time. However, when there are only a few studies available, studies from all years will be included.
Population Attributes	The most technically appropriate measures of benefits would be based on impact functions that cover the entire sensitive population but allow for heterogeneity across age or other relevant demographic factors. In the absence of effect estimates specific to age, sex, preexisting condition status, or other relevant factors, it may be appropriate to select effect estimates that cover the broadest population to match with the desired outcome of the analysis, which is total national-level health impacts. When available, multi-city studies are preferred to single city studies because they provide a more generalizable representation of the C-R function.
Study Size	Studies examining a relatively large sample are preferred because they generally have more power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population or through repeated observations on a smaller population (e.g., through a symptom diary recorded for a panel of asthmatic children).
Study Location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior, and lifestyle.
Pollutants Included in Model	When modeling the effects of ozone and PM (or other pollutant combinations) jointly, it is important to use properly specified impact functions that include both pollutants. Using single-pollutant models in cases where both pollutants are expected to affect a health outcome can lead to double-counting when pollutants are correlated.
Measure of PM	For this analysis, impact functions based on PM <sub>2.5</sub> are preferred to PM <sub>10</sub> because of the focus on reducing emissions of PM <sub>2.5</sub> precursors, and because air quality modeling was conducted for this size fraction of PM. Where PM <sub>2.5</sub> functions are not available, PM <sub>10</sub> functions are used as surrogates, recognizing that there will be potential downward (upward) biases if the fine fraction of PM <sub>10</sub> is more (less) toxic than the coarse fraction.
Economically Valuable Health Effects	Some health effects, such as forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are not quantified in this analysis.
Non-overlapping Endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double-counting of benefits.

When several effect estimates for a pollutant and a given health endpoint have been selected, they are quantitatively combined or pooled to derive a more robust estimate of the relationship. The BenMAP-CE Manual Appendices provides details of the procedures used to combine multiple impact functions (U.S. EPA, 2015b). In general, we used fixed or random effects models to pool estimates from different studies of the same endpoint. Fixed effects pooling simply weights each study's estimate by the inverse variance, giving more weight to studies with greater statistical power (lower variance). Random effects pooling accounts for both within-study variance and between-study variability, due, for example, to differences in population susceptibility. We used the fixed effects model as our null hypothesis and then determined whether the data suggest that we should reject this null hypothesis, in which case we would use the random effects model. Pooled impact functions are used to estimate hospital admissions and asthma exacerbations. For more details on methods used to pool incidence estimates, see the BenMAP-CE Manual Appendices (U.S. EPA, 2015b), which are available with the BenMAP-CE software at <http://www2.epa.gov/benmap.html>.

Effect estimates selected for a given health endpoint were applied consistently across all locations nationwide. This applies to both impact functions defined by a single effect estimate and those defined by a pooling of multiple effect estimates. Although the effect estimate may, in fact, vary from one location to another (e.g., because of differences in population susceptibilities or differences in the composition of PM), location-specific effect estimates are generally not available.

The specific studies from which effect estimates for the primary analysis are drawn are included in Tables A-6 and A-7. In all cases where effect estimates are drawn directly from epidemiological studies, standard errors are used as a partial representation of the uncertainty in the size of the effect estimate. We refer readers interested in further details regarding each study to the Regulatory Impact analysis for the Ozone NAAQS RIA and the Regulatory Impact Analysis for the PM RIA (EPA, 2012; EPA, 2015).

#### *PM<sub>2.5</sub> Premature Mortality Effect Coefficients*

Both long- and short-term exposures to ambient levels of PM<sub>2.5</sub> air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most significant health endpoint quantified in this analysis.

**Table 6. Health Endpoints and Epidemiological Studies Used to Quantify PM<sub>2.5</sub>-related Health Impacts**

Endpoint	Study	Study Population	Relative Risk or Effect Estimate ( $\beta$ ) (with 95th Percentile Confidence Interval or SE)
Premature Mortality			
Premature mortality— cohort study, all-cause	Krewski <i>et al.</i> (2009) Lepeule <i>et al.</i> (2012)	> 29 years > 24 years	RR = 1.06 (1.04–1.06) per 10 µg/m <sup>3</sup> RR = 1.14 (1.07–1.22) per 10 µg/m <sup>3</sup>
Premature mortality— all-cause	Woodruff <i>et al.</i> (1997)	Infant (< 1 year)	OR = 1.04 (1.02–1.07) per 10 µg/m <sup>3</sup>
Chronic Illness			

Nonfatal heart attacks	Peters et al. (2001) Pooled estimate: <i>Pope et al. (2006)</i> <i>Sullivan et al. (2005)</i> <i>Zanobetti et al. (2009)</i> <i>Zanobetti and Schwartz (2006)</i>	Adults (> 18 years)	OR = 1.62 (1.13–2.34) per 20 µg/m <sup>3</sup> $\beta = 0.00481$ (0.00199) $\beta = 0.00198$ (0.00224) $\beta = 0.00225$ (0.000591) $\beta = 0.0053$ (0.00221)
<b>Hospital Admissions</b>			
Respiratory	<i>Zanobetti et al. (2009)</i> —ICD 460-519 (All respiratory) <i>Kloog et al. (2012)</i> —ICD 460-519 (All Respiratory) <i>Moolgavkar (2000)</i> —ICD 490-496 (Chronic lung disease) <i>Babin et al. (2007)</i> —ICD 493 (asthma) <i>Sheppard (2003)</i> —ICD 493 (asthma)	> 64 years 18–64 years < 19 years < 18	$\beta=0.00207$ (0.00446) 1.02 (1.01–1.03) per 36 µg/m <sup>3</sup> $\beta=0.0007$ (0.000961) RR = 1.04 (1.01–1.06) per 11.8 µg/m <sup>3</sup>
Cardiovascular	Pooled estimate: <i>Zanobetti et al. (2009)</i> —ICD 390-459 (all cardiovascular) <i>Peng et al. (2009)</i> —ICD 426-427; 428; 430-438; 410-414; 429; 440-449 (Cardio-, cerebro- and peripheral vascular disease) <i>Peng et al. (2008)</i> —ICD 426-427; 428; 430-438; 410-414; 429; 440-449 (Cardio-, cerebro- and peripheral vascular disease) <i>Bell et al. (2008)</i> —ICD 426-427; 428; 430-438; 410-414; 429; 440-449 (Cardio-, cerebro- and peripheral vascular disease) <i>Moolgavkar (2000)</i> —ICD 390-429 (all cardiovascular)	> 64 years 20–64 years	$\beta=0.00189$ (0.000283) $\beta=0.00068$ (0.000214) $\beta=0.00071$ (0.00013) $\beta=0.0008$ (0.000107) RR=1.04 (t statistic: 4.1) per 10 µg/m <sup>3</sup>
Asthma-related emergency department visits	Pooled estimate: <i>Mar et al. (2010)</i> <i>Slaughter et al. (2005)</i> <i>Glad et al. (2012)</i>	All ages	RR = 1.04 (1.01–1.07) per 7 µg/m <sup>3</sup> RR = 1.03 (0.98–1.09) per 10 µg/m <sup>3</sup> $\beta=0.00392$ (0.002843)
<b>Other Health Endpoints</b>			
Acute bronchitis	Dockery et al. (1996)	8–12 years	OR = 1.50 (0.91–2.47) per 14.9 µg/m <sup>3</sup>
Asthma exacerbations	Pooled estimate: <i>Ostro et al. (2001)</i> (cough, wheeze, shortness of breath) <sup>b</sup> <i>Mar et al. (2004)</i> (cough, shortness of breath)	6–18 years <sup>b</sup>	OR = 1.03 (0.98–1.07) OR = 1.06 (1.01–1.11) OR = 1.08 (1.00–1.17) per 30 µg/m <sup>3</sup> RR = 1.21 (1–1.47) per RR = 1.13 (0.86–1.48) per 10 µg/m <sup>3</sup>
Work loss days	Ostro (1987)	18–65 years	$\beta=0.0046$ (0.00036)

Acute respiratory symptoms (MRAD)	Ostro and Rothschild (1989) (Minor restricted activity days)	18–65 years	$\beta=0.00220$ (0.000658)
Upper respiratory symptoms	Pope et al. (1991)	Asthmatics, 9–11 years	1.003 (1–1.006) per 10 $\mu\text{g}/\text{m}^3$
Lower respiratory symptoms	Schwartz and Neas (2000)	7–14 years	OR = 1.33 (1.11–1.58) per 15 $\mu\text{g}/\text{m}^3$

**Table 7. Health Endpoints and Epidemiological Studies Used to Quantify Ozone-Related Health Impacts <sup>a</sup>**

Endpoint	Study	Study Population	Relative Risk or Effect Estimate ( $\beta$ ) (with 95 <sup>th</sup> Percentile Confidence Interval or SE)
Premature Mortality			
Premature mortality—short-term	<i>Smith et al. (2009)</i> <i>Zanobetti and Schwartz (2008)</i>	All ages	$\beta = 0.00032$ (0.00008) $\beta = 0.00051$ (0.00012)
Premature respiratory mortality-long-term	<i>Jerrett et al. (2009)</i>	>29 years	$\beta = 0.003971$ (0.00133)
Hospital Admissions			
Respiratory	Pooled estimate: <i>Katsouyanni et al. (2009)</i>	> 65 years	$\beta = 0.00064$ (0.00040) penalized splines
Asthma-related emergency department visits	Pooled estimate: <i>Glad et al. (2012)</i>		$\beta = 0.00306$ (0.00117)
	<i>Ito et al. (2007)</i>		$\beta = 0.00521$ (0.00091)
	<i>Mar and Koenig (2010)</i>	0-99 years	$\beta = 0.01044$ (0.00436) (0-17 yr olds) $\beta = 0.00770$ (0.00284) (18-99 yr olds)
	<i>Peel et al. (2005)</i>		$\beta = 0.00087$ (0.00053)
	<i>Sarnat et al. (2013)</i>		$\beta = 0.00111$ (0.00028)
	<i>Wilson et al. (2005)</i>		RR = 1.022 (0.996 – 1.049) per 25
Other Health Endpoints			
Asthma exacerbation	Pooled estimate: <sup>a</sup> <i>Mortimer et al. (2002)</i> <i>Schildcrout et al. (2006)</i>	6–18 years	$\beta = 0.00929$ (0.00387) $\beta = 0.00222$ (0.00282)
	Pooled estimate: <i>Chen et al. (2000)</i> <i>Gilliland et al. (2001)</i>	5-17 years	$\beta = 0.015763$ (0.004985) $\beta = 0.007824$ (0.004445)
Acute respiratory symptoms (MRAD)	Ostro and Rothschild (1989)	18–65 years	$\beta = 0.002596$ (0.000776)

<sup>a</sup> The original study populations were 5 to 12 years for Schildcrout et al. (2006) and 5-9 years for the Mortimer et al. (2002) study. Based on advice from the SAB-HES, we extended the applied population to 6-18 years for all three studies, reflecting the common biological basis for the effect in children in the broader age group. See: U.S. EPA-SAB (2004a) and NRC (2002).

## Baseline Incidence Estimates

Epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the relative risk of a health effect, rather than estimating the absolute number of avoided cases. For example, a typical result might be that

a 10 ppb decrease in daily ozone levels might, in turn, decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. A baseline incidence rate is the estimate of the number of cases of the health effect per year in the assessment location, as it corresponds to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number. For example, if the baseline incidence rate is the number of cases per year per million people, that number must be multiplied by the millions of people in the total population.

Table 8 summarizes the sources of baseline incidence rates and provides average incidence rates for the endpoints included in the analysis. For both baseline incidence and prevalence data, we used age-specific rates, where available. We applied concentration-response functions to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits.

**Table 8. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population**

Endpoint	Parameter	Rates	
		Value	Source
Mortality	Daily or annual mortality rate projected to 2015	Age-, cause-, and county-specific rate	CDC Wonder (2004–2006) U.S. Census bureau
Hospitalizations	Daily hospitalization rate	Age-, region-, state-, county- and cause-specific rate	2007 HCUP data files <sup>a</sup>
Asthma ER Visits	Daily asthma ER visit rate	Age-, region-, state-, county- and cause-specific rate	2007 HCUP data files <sup>a</sup>
Chronic Bronchitis	Annual prevalence rate per person		1999 NHIS (American Lung Association, 2002b, Table 4)
	• Aged 18–44	0.0367	
	• Aged 45–64	0.0505	
	• Aged 65 and older	0.0587	
	Annual incidence rate per person	0.00378	Abbey et al. (1995, Table 3)
Non-fatal Myocardial Infarction (heart attacks)	Daily non-fatal myocardial infarction incidence rate per person, 18+	Age-, region-, state-, and county- specific rate	2007 HCUP data files <sup>a</sup> ; adjusted by 0.93 for probability of surviving after 28 days (Rosamond et al., 1999)
Asthma Exacerbations	Incidence among asthmatic African-American children		Ostro et al. (2001)
	• daily wheeze	0.076	
	• daily cough	0.067	
	• daily dyspnea	0.037	

Acute Bronchitis	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002c, Table 11)
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children <sup>b</sup>	0.0012	Schwartz et al. (1994, Table 2)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children	0.3419	Pope et al. (1991, Table 2)
Work Loss Days	Daily WLD incidence rate per person (18–65) <ul style="list-style-type: none"> <li>• Aged 18–24</li> <li>• Aged 25–44</li> <li>• Aged 45–64</li> </ul>	1996 HIS (Adams, Hendershot, and Marano, 1999, Table 41); U.S. Bureau of the Census (2000)	
<b>Rates</b>			
Endpoint	Parameter	Value	Source
School Loss Days	Rate per person per year, assuming 180 school days per year	9.9	National Center for Education Statistics (1996) and 1996 HIS (Adams et al., 1999, Table 47);
Minor Restricted-Activity Days	Daily MRAD incidence rate per person	0.02137	Ostro and Rothschild (1989, p. 243)

<sup>a</sup> Healthcare Cost and Utilization Program (HCUP) database contains individual level, state and regional-level hospital and emergency department discharges for a variety of ICD codes.

<sup>b</sup> Lower respiratory symptoms are defined as two or more of the following: cough, chest pain, phlegm, and wheeze.

The baseline incidence rates for hospital and emergency department visits that we applied in this analysis are an improvement over the rates we used in the proposal analysis in two ways. First, these data are newer, and so are a more recent representation of the rates at which populations of different ages, and in different locations, visit the hospital and emergency department for illnesses that may be air pollution related. Second, these newer data are also more spatially refined. For many locations within the U.S., these data are resolved at the county- or state-level, providing a better characterization of the geographic distribution of hospital and emergency department visits. Newer and more spatially resolved incidence rates are likely to yield a more reliable estimate of air pollution-related hospitalizations and emergency department visits. Consistent with the proposal RIA, we continue to use county-level mortality rates. We have projected mortality rates such that future mortality rates are consistent with our projections of population growth (U.S. EPA, 2015b).

For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Table 9 lists the prevalence rates used to determine the applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years.

**Table 9. Asthma Prevalence Rates Used for this Analysis<sup>a</sup>**

Population Group	Value	Asthma Prevalence Rates
		Source
All Ages	0.0780	American Lung Association (2010, Table 7)
< 18	0.0941	
5–17	0.1070	
18–44	0.0719	
45–64	0.0745	
65+	0.0716	
African American, 5 to 17	0.1776	American Lung Association (2010, Table 9)
African American, <18	0.1553	American Lung Association <sup>b</sup>

<sup>a</sup> See [ftp://ftp.cdc.gov/pub/Health\\_Statistics/NCHS/Datasets/NHIS/2000/](ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000/).

<sup>b</sup> Calculated by ALA for U.S. EPA, based on NHIS data (CDC, 2009)

## Economic Valuation Estimates

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects for a large population. Therefore, the appropriate economic measure is WTP for changes in risk of a health effect rather than WTP for a health effect that would occur with certainty (Freeman, 1993). Epidemiological studies generally provide estimates of the relative risks of a particular health effect that is avoided because of a reduction in air pollution. We converted those to units of avoided statistical incidence for ease of presentation. We calculated the value of avoided statistical incidences by dividing individual WTP for a risk reduction by the related observed change in risk.<sup>18</sup>

WTP estimates generally are not available for some health effects, such as hospital admissions. In these cases, we used the cost of treating or mitigating the effect as a primary estimate. These cost-of-illness (COI) estimates generally understate the true value of reducing the risk of a health effect, because they reflect the direct expenditures related to treatment, but not the value of avoided pain and suffering (Harrington and Portney, 1987; Berger, 1987). We provide unit values for health endpoints (along with

<sup>18</sup> To comply with Circular A-4, EPA provides monetized benefits using discount rates of 3 percent and 7 percent (OMB, 2003). These benefits are estimated for a specific analysis year (i.e., 2016), and most of the PM benefits occur within that year with two exceptions: acute myocardial infarctions (AMIs) and premature mortality. For AMIs, we assume 5 years of follow-up medical costs and lost wages. For premature mortality, we assume that there is a “cessation” lag between PM exposures and the total realization of changes in health effects. Although the structure of the lag is uncertain, EPA follows the advice of the SAB-HES to assume a segmented lag structure characterized by 30 percent of mortality reductions in the first year, 50 percent over years 2 to 5, and 20 percent over the years 6 to 20 after the reduction in PM<sub>2.5</sub> (U.S. EPA-SAB, 2004c). Changes in the lag assumptions do not change the total number of estimated deaths but rather the timing of those deaths. Therefore, discounting only affects the AMI costs after the analysis year and the valuation of premature mortalities that occur after the analysis year. As such, the monetized benefits using a 7 percent discount rate are only approximately 10 percent less than the monetized benefits using a 3 percent discount rate.

information on the distribution of the unit value) in Table 10. All values are in constant year 2006 dollars, adjusted for growth in real income out to 2016 using projections provided by Standard and Poor's. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real income increases. Many of the valuation studies used in this analysis were conducted in the late 1980s and early 1990s. Because real income has grown since the studies were conducted, people's willingness to pay for reductions in the risk of premature death and disease likely has grown as well. We did not adjust cost of illness-based values because they are based on current costs. Similarly, we did not adjust the value of school absences, because that value is based on current wage rates. For these two reasons, these cost of illness estimates may underestimate the economic value of avoided health impacts in 2016. Readers interested in learning more about the basis for the economic value estimates below may refer to the Ozone and PM NAAQS Regulatory Impact Analyses (EPA, 2012; EPA, 2015).

**Table 10. Unit Values for Economic Valuation of Health Endpoints (2010\$)<sup>a</sup>**

<b>Health Endpoint</b>	<b>Central Estimate of Value Per Statistical Incidence, Income Level</b>		<b>Derivation of Distributions of Estimates</b>
	<b>2000</b>	<b>2016</b>	
Premature Mortality (Value of a Statistical Life)	\$6,800,000	\$9,300,000	EPA currently recommends a central VSL of \$6.3m (2000\$) based on a Weibull distribution fitted to 26 published VSL estimates (5 contingent valuation and 21 labor market studies). The underlying studies, the distribution parameters, and other useful information are available in Appendix 5B of EPA's current Guidelines for Preparing Economic Analyses (U.S. EPA, 2000).
Chronic Bronchitis (CB)	\$370,000	\$510,000	The WTP to avoid a case of pollution-related CB is calculated as where x is the severity of an average CB case, WTP <sub>13</sub> is the WTP for a severe case of CB, and \$ is the parameter relating WTP to severity, based on the regression results reported in Krupnick and Cropper (1992). The distribution of WTP for an average severity-level case of CB was generated by Monte Carlo methods, drawing from each of three distributions: (1) WTP to avoid a severe case of CB is assigned a 1/9 probability of being each of the first nine deciles of the distribution of WTP responses in Viscusi et al. (1991); (2) the severity of a pollution-related case of CB (relative to the case described in the Viscusi study) is assumed to have a triangular distribution, with the most likely value at severity level 6.5 and endpoints at 1.0 and 12.0; and (3) the constant in the elasticity of WTP with respect to severity is normally distributed with mean = 0.18 and standard deviation = 0.0669 (from Krupnick and Cropper [1992]). This process and the rationale for choosing it is described in detail in the Costs and Benefits of the Clean Air Act, 1990 to 2010 (U.S. EPA, 1999).

**Table 10. Unit Values for Economic Valuation of Health Endpoints (2010\$) (continued)**

Health Endpoint	Central Estimate of Value Per Statistical Incidence, Income Level		Derivation of Distributions of Estimates
	2000	2016	
Non-fatal Myocardial Infarction (heart attack)			No distributional information available. Age-specific cost-of-illness values reflect lost earnings and direct medical costs over a 5-year period following a non-fatal MI. Lost earnings estimates are based on Cropper and Krupnick (1990). Direct medical costs are based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990).
<u>3% discount rate</u>	\$86,190	\$86,190	Lost earnings: Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings: age of onset: at 3% at 7% 25–44 \$8,774 \$7,855 45–54 \$12,932 11,578 55–65 \$74,746 66,920 Direct medical expenses: An average of: 1. Wittels et al. (1990) (\$102,658—no discounting) 2. Russell et al. (1998), 5-year period (\$22,331 at 3% discount rate; \$21,113 at 7% discount rate)
Age 0–24	\$96,238	\$96,238	
Age 25–44	\$101,562	\$101,562	
Age 45–54	\$181,208	\$181,208	
Age 55–65	\$86,190	\$86,190	
<u>7% discount rate</u>	\$84,117	\$84,117	
Age 0–24	\$94,238	\$94,238	
Age 25–44	\$99,033	\$99,033	
Age 45–54	\$170,332	\$170,332	
Age 55–65	\$84,117	\$84,117	
<b>Hospital Admissions</b>			
Chronic Obstructive Pulmonary Disease (COPD)	\$17,961	\$17,961	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality (2000) ( <a href="http://www.ahrq.gov">www.ahrq.gov</a> ).
Asthma Admissions	\$9,627	\$9,627	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality (2000) ( <a href="http://www.ahrq.gov">www.ahrq.gov</a> ).

**Table 10. Unit Values for Economic Valuation of Health Endpoints (2010\$) (continued)**

<b>Health Endpoint</b>	<b>Central Estimate of Value Per Statistical Incidence, Income Level</b>		<b>Derivation of Distributions of Estimates</b>
	<b>2000</b>	<b>2016</b>	
All Cardiovascular	\$26,682	\$26,682	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality (2000) ( <a href="http://www.ahrq.gov">www.ahrq.gov</a> ).
All respiratory (ages 65+)	\$26,632	\$26,632	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 ( <a href="http://www.ahrq.gov">www.ahrq.gov</a> ).
All respiratory (ages 0–2)	\$11,233	\$11,233	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 ( <a href="http://www.ahrq.gov">www.ahrq.gov</a> ).
Emergency Room Visits for Asthma	\$415	\$415	No distributional information available. Simple average of two unit COI values: (1) \$311.55, from Smith et al. (1997); and (2) \$260.67, from Stanford et al. (1999).

(continued)

**Table 10. Unit Values for Economic Valuation of Health Endpoints (2010\$) (continued)**

<b>Health Endpoint</b>	<b>Central Estimate of Value Per Statistical Incidence, Income Level</b>		<b>Derivation of Distributions of Estimates</b>
	<b>2000</b>	<b>2016</b>	
<b><i>Respiratory Ailments Not Requiring Hospitalization</i></b>			
Upper Respiratory Symptoms (URS)	\$32	\$32	Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, we assumed a uniform distribution between \$9.2 and \$43.1.
Lower Respiratory Symptoms (LRS)	\$17	\$21	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS. In the absence of information surrounding the frequency with which each of the 11 types of LRS occurs within the LRS symptom complex, we assumed a uniform distribution between \$6.9 and \$24.46.
Asthma Exacerbations	\$47	\$57	Asthma exacerbations are valued at \$45 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between \$15.6 and \$70.8.

(continued)

**Table 10. Unit Values for Economic Valuation of Health Endpoints (2010\$) (continued)**

<b>Health Endpoint</b>	<b>Central Estimate of Value Per Statistical Incidence, Income Level</b>			<b>Derivation of Distributions of Estimates</b>
	<b>2000</b>	<b>2016</b>		
Acute Bronchitis	\$389	\$476		Assumes a 6-day episode, with the distribution of the daily value specified as uniform with the low and high values based on those recommended for related respiratory symptoms in Neumann et al. (1994). The low daily estimate of \$10 is the sum of the mid-range values recommended by IEC (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high daily estimate was taken to be twice the value of a minor respiratory restricted-activity day, or \$110.
Work Loss Days (WLDs)	Variable (U.S. median = \$141)	Variable (U.S. median = \$141)		No distribution available. Point estimate is based on county-specific median annual wages divided by 52 and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.
Minor Restricted Activity Days (MRADs)	\$55	\$67		Median WTP estimate to avoid one MRAD from Tolley et al. (1986). Distribution is assumed to be triangular with a minimum of \$22 and a maximum of \$83, with a most likely value of \$52. Range is based on assumption that value should exceed WTP for a single mild symptom (the highest estimate for a single symptom—for eye irritation—is \$16.00) and be less than that for a WLD. The triangular distribution acknowledges that the actual value is likely to be closer to the point estimate than either extreme.

## Growth in WTP Reflecting National Income Growth Over Time

Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. There is substantial empirical evidence that the income elasticity<sup>19</sup> of WTP for health risk reductions is positive, although there is uncertainty about its exact value. Thus, as real income increases, the WTP for environmental improvements also increases. Although many analyses assume that the income elasticity of WTP is unit elastic (i.e., a 10 percent higher real income level implies a 10 percent higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefits estimates in two different ways: through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the Science Advisory Board (SAB) advised EPA to adjust WTP for increases in real income over time but not to adjust WTP to account for cross-sectional income differences “because of the sensitivity of making such distinctions, and because of insufficient evidence available at present” (U.S. EPA-SAB, 2000). A recent advisory by another committee associated with the SAB, the Advisory Council on Clean Air Compliance Analysis, has provided conflicting advice. While agreeing with “the general principle that the willingness to pay to reduce mortality risks is likely to increase with growth in real income (U.S. EPA-SAB, 2004b, p. 52)” and that “The same increase should be assumed for the WTP for serious non-fatal health effects (U.S. EPA-SAB, 2004b, p. 52),” they note that “given the limitations and uncertainties in the available empirical evidence, the Council does not support the use of the proposed adjustments for aggregate income growth as part of the primary analysis” (U.S. EPA-SAB, 2004b, p. 53). Until these conflicting advisories have been reconciled, EPA will continue to adjust valuation estimates to reflect income growth using the methods described below, while providing sensitivity analyses for alternative income growth adjustment factors.

Based on a review of the available income elasticity literature, we adjusted the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, we applied estimates derived from cross-sectional studies in our analysis. Details of the procedure can be found in Kleckner and Neumann (1999). An abbreviated description of the procedure we used to account for WTP for real income growth between 1990 and 2016 is presented below.

Reported income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP. As such, we use different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. Note that because of the variety of empirical sources used in deriving the income elasticities, there may appear to be inconsistencies in the

---

<sup>19</sup> Income elasticity is a common economic measure equal to the percentage change in WTP for a 1 percent change in income.

magnitudes of the income elasticities relative to the severity of the effects (*a priori* one might expect that more severe outcomes would show less income elasticity of WTP). We have not imposed any additional restrictions on the empirical estimates of income elasticity. One explanation for the seeming inconsistency is the difference in timing of conditions. WTP for minor illnesses is often expressed as a short term payment to avoid a single episode. WTP for major illnesses and mortality risk reductions are based on longer term measures of payment (such as wages or annual income). Economic theory suggests that relationships become more elastic as the length of time grows, reflecting the ability to adjust spending over a longer time period. Based on this theory, it would be expected that WTP for reducing long term risks would be more elastic than WTP for reducing short term risks. The elasticity values used to adjust estimates of benefits in 2016 are presented in Table II.

**Table II. Elasticity Values Used to Account for Projected Real Income Growth<sup>a</sup>**

Benefit Category	Central Elasticity Estimate
Minor Health Effect	0.14
Severe and Chronic Health Effects	0.45
Premature Mortality	0.40

<sup>a</sup> Derivation of estimates can be found in Kleckner and Neumann (1999) and Chestnut (1997). COI estimates are assigned an adjustment factor of 1.0.

In addition to elasticity estimates, projections of real gross domestic product (GDP) and populations from 1990 to 2020 are needed to adjust benefits to reflect real per capita income growth. For consistency with the emissions and benefits modeling, we used national population estimates for the years 1990 to 1999 based on U.S. Census Bureau estimates (Hollman, Mulder, and Kallan, 2000). These population estimates are based on application of a cohort-component model applied to 1990 U.S. Census data projections (U.S. Bureau of Census, 2000). For the years between 2000 and 2016, we applied growth rates based on the U.S. Census Bureau projections to the U.S. Census estimate of national population in 2000. We used projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010.<sup>20</sup> We used projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's (2000) for the years 2010 to 2016.<sup>21</sup>

Using the method outlined in Kleckner and Neumann (1999) and the population and income data described above, we calculated WTP adjustment factors for each of the elasticity estimates

<sup>20</sup> U.S. Bureau of Economic Analysis, Table 2A (1992\$) (available at <http://www.bea.doc.gov/bea/dn/0897nip2/tab2a.htm>) and U.S. Bureau of Economic Analysis, Economics and Budget Outlook. Note that projections for 2007 to 2010 are based on average GDP growth rates between 1999 and 2007.

<sup>21</sup> In previous analyses, we used the Standard and Poor's projections of GDP directly. This led to an apparent discontinuity in the adjustment factors between 2010 and 2011. We refined the method by applying the relative growth rates for GDP derived from the Standard and Poor's projections to the 2010 projected GDP based on the Bureau of Economic Analysis projections.

listed in Table 12. Benefits for each of the categories (minor health effects, severe and chronic health effects, and premature mortality) are adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. Note that, for premature mortality, we applied the income adjustment factor to the present discounted value of the stream of avoided mortalities occurring over the lag period. Also note that because of a lack of data on the dependence of COI and income, and a lack of data on projected growth in average wages, no adjustments are made to benefits based on the COI approach or to work loss days and worker productivity. This assumption leads us to underpredict benefits in future years because it is likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

**Table 12. Adjustment Factors Used to Account for Projected Real Income Growth<sup>a</sup>**

Benefit Category	2016
Minor Health Effect	1.15
Severe and Chronic Health Effects	1.29
Premature Mortality	1.25

<sup>a</sup> Based on elasticity values reported in Table 11, U.S. Census population projections, and projections of real GDP per capita.

## References

- Agency for Healthcare Research and Quality (AHRQ). 2000. HCUPnet, Healthcare Cost and Utilization Project.
- American Lung Association (ALA). 2010. Trends in Asthma Morbidity and Mortality. American Lung Association Epidemiology and Statistics Unit, Research and Program Services Division.
- Burnett RT, Smith-Doiron M, Stieb D, Raizenne ME, Brook JR, Dales RE, et al. 2001. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol* 153(5):444-452.
- Chen L, Jennison BL, Yang W, Omaye ST. 2000. Elementary school absenteeism and air pollution. *Inhal Toxicol* 12(11):997-1016.
- Cropper, M. L. and A. J. Krupnick. 1990. The Social Costs of Chronic Heart and Lung Disease. Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.
- Davidson K, Hallberg A, McCubbin D, Hubbell BJ. 2007. Analysis of PM<sub>2.5</sub> Using the Environmental Benefits Mapping and Analysis Program (BenMAP). *J Toxicol Environ Health* 70: 332—346.
- Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne, and F.E. Speizer. 1996. Health Effects of Acid Aerosols on North American Children-Respiratory Symptoms. *Environmental Health Perspectives* 104(5):500-505.
- Eisenstein, E.L., L.K. Shaw, K.J. Anstrom, C.L. Nelson, Z. Hakim, V. Hasselblad and D.B. Mark. 2001. Assessing the Clinical and Economic Burden of Coronary Artery Disease: 1986-1998. *Medical Care* 39(8):824-35.
- Gilliland FD, Berhane K, Rappaport EB, Thomas DC, Avol E, Gauderman WJ, et al. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology* 12(1):43-54.
- Holloman, F.W., T.J. Mulder, and J.E. Kallan. January 2000. Methodology and Assumptions for the Population Projections of the United States: 1999 to 2100. Population Division Working Paper No. 38, Population Projections Branch, Population Division, U.S. Census Bureau, Department of Commerce.
- Hubbell BJ, Hallberg A, McCubbin D, Post, E. 2005. Health-Related Benefits of Attaining the 8-Hr Ozone Standard. *Environ Health Perspect* 113: 73—82.
- Ito, K. 2003. Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan. In Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute, Boston, MA.
- Jerrett M, Burnett RT, Pope CA, III, et al. 2009. Long-Term Ozone Exposure and Mortality. *N Engl J Med* 360:1085-95.

- Kleckner, N., and J. Neumann. June 3, 1999. Recommended Approach to Adjusting WTP Estimates to Reflect Changes in Real Income. Memorandum to Jim Democker, U.S. EPA/OPAR.
- Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi, Y, et al. 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. HEI Research Report, 140, Health Effects Institute, Boston, MA.
- Kunzli, N., R. Kaiser, S. Medina, M. Studnicka, O. Chanel, P. Filliger, et al. 2000. Public-health impact of outdoor and traffic-related air pollution: A European Assessment. *The Lancet* 356(9232):795-801.
- Laden, F., J. Schwartz, F.E. Speizer, and D.W. Dockery. 2006. Reduction in Fine Particulate Air Pollution and Mortality. *American Journal of Respiratory and Critical Care Medicine* 173:667-672.
- Levy JI, Baxter LK, Schwartz J. 2009. Uncertainty and variability in health-related damages from coal-fired power plants in the United States. *Risk Anal.* doi: 10.1111/j.1539-6924.2009.01227.x [Online 9 Apr 2009]
- Moolgavkar SH, Luebeck EG, Anderson EL. 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis St. Paul and Birmingham. *Epidemiology*. 8(4):364-370.
- Moolgavkar, S.H. 2000. Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas. *Journal of the Air and Waste Management Association* 50:1199-1206.
- Moolgavkar, S.H. 2003. Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties. In Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Boston, MA: Health Effects Institute.
- National Research Council (NRC). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. Washington, DC: The National Academies Press.
- National Research Council (NRC). 2008. Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution. National Academies Press. Washington, DC.
- Ostro, B., M. Lipsett, J. Mann, H. Braxton-Owens, and M. White. 2001. Air Pollution and Exacerbation of Asthma in African-American Children in Los Angeles. *Epidemiology* 12(2):200-208.
- Ostro, B.D. 1987. Air Pollution and Morbidity Revisited: A Specification Test. *Journal of Environmental Economics Management* 14:87-98.
- Ostro, B.D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity: An Observational Study of Multiple Pollutants. *Environmental Research* 50:238-247.
- Peel, J. L., P. E. Tolbert, M. Klein, et al. 2005. Ambient air pollution and respiratory emergency department visits. *Epidemiology*. Vol. 16 (2): 164-74.

- Peters, A., D.W. Dockery, J.E. Muller, and M.A. Mittleman. 2001. Increased Particulate Air Pollution and the Triggering of Myocardial Infarction. *Circulation* 103:2810-2815.
- Pope, C.A., III, D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. Respiratory Health and PM<sub>10</sub> Pollution: A Daily Time Series Analysis. *American Review of Respiratory Diseases* 144:668-674.
- Ransom, Michael, and C. Arden Pope. 1992. M.R. Ransom and C.A. Pope, III, Elementary school absences and PM<sub>10</sub> pollution in Utah Valley. *Environ. Res.* 58, pp. 204–219.
- Russell, M.W., D.M. Huse, S. Drowns, E.C. Hamel, and S.C. Hartz. 1998. Direct Medical Costs of Coronary Artery Disease in the United States. *American Journal of Cardiology* 81(9):1110-1115.
- Samet, J.M., S.L. Zeger, F. Dominici, F. Curriero, I. Coursac, D.W. Dockery, J. Schwartz, and A. Zanobetti. 2000. The National Morbidity, Mortality and Air Pollution Study: Part II: Morbidity, Mortality and Air Pollution in the United States. Research Report No. 94, Part II. Health Effects Institute, Cambridge MA. June.
- Schwartz J. 1994a. PM(10) Ozone, and Hospital Admissions For the Elderly in Minneapolis St Paul, Minnesota. *Arch Environ Health*. 49(5):366-374.
- Schwartz J. 1994b. Air Pollution and Hospital Admissions For the Elderly in Detroit, Michigan. *Am J Respir Crit Care Med.* 150(3):648-655.
- Schwartz J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. *Thorax*. 50(5):531-538.
- Schwartz, J. 1993. Particulate Air Pollution and Chronic Respiratory Disease. *Environment Research* 62:7-13.
- Schwartz, J. 2005. How sensitive is the association between ozone and daily deaths to control for temperature? *Am J Respir Crit Care Med.* Vol. 171 (6): 627-31.
- Schwartz, J., and L.M. Neas. 2000. Fine Particles are More Strongly Associated than Coarse Particles with Acute Respiratory Health Effects in Schoolchildren. *Epidemiology* 11:6-10.
- Sheppard, L. 2003. Ambient Air Pollution and Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994. In Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Boston, MA: Health Effects Institute.
- Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista, and W.B. Saunders. 1997. A National Estimate of the Economic Costs of Asthma. *American Journal of Respiratory and Critical Care Medicine* 156(3 Pt 1):787-793.
- Tagaris E, Liao KJ, Delucia AJ, et al. 2009. Potential impact of climate change on air-pollution related human health effects. *Environ. Sci. Technol.* 43: 4979—4988.
- Tolley, G.S. et al. 1986. Valuation of Reductions in Human Health Symptoms and Risks. University of Chicago. Final Report for the U.S. Environmental Protection Agency. January

- U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2009b. Review of EPA's Integrated Science Assessment for Particulate Matter (First External Review Draft, December 2008). EPA-COUNCIL-09-008. May. Available on the Internet at <[http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fc85256ead006be86e/73ACCA834AB44A10852575BD0064346B/\\$File/EPA-CASAC-09-008-unsigned.pdf](http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fc85256ead006be86e/73ACCA834AB44A10852575BD0064346B/$File/EPA-CASAC-09-008-unsigned.pdf)>.
- U.S. Environmental Protection Agency (U.S. EPA), 2015b. Environmental Benefits Mapping and Analysis Program—Community Edition (Version 1.1). Research Triangle Park, NC. Available on the Internet at <<http://www2.epa.gov/benmap>>.
- U.S. Environmental Protection Agency (U.S. EPA), 2015a. Regulatory Impact Analysis, 2015 National Ambient Air Quality Standards for Ground-level Ozone, Chapter 6. Office of Air Quality Planning and Standards, Research Triangle Park, NC. October. Available at <<http://www.epa.gov/ttn/ecas>>.
- U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 2009a. Consultation on EPA's Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment. EPA-COUNCIL-09-009. May. Available on the Internet at <[http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fc85256ead006be86e/723FE644C5D758DF852575BD00763A32/\\$File/EPA-CASAC-09-009-unsigned.pdf](http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fc85256ead006be86e/723FE644C5D758DF852575BD00763A32/$File/EPA-CASAC-09-009-unsigned.pdf)>.
- Vedal, S., J. Petkau, R. White, and J. Blair. 1998. Acute Effects of Ambient Inhalable Particles in Asthmatic and Nonasthmatic Children. *American Journal of Respiratory and Critical Care Medicine* 157(4):1034-1043.
- Wilson, A. M., C. P. Wake, T. Kelly, et al. 2005. Air pollution, weather, and respiratory emergency room visits in two northern New England cities: an ecological time-series study. *Environ Res.* Vol. 97 (3): 312-21.
- Wittels, E.H., J.W. Hay, and A.M. Gotto, Jr. 1990. Medical Costs of Coronary Artery Disease in the United States. *American Journal of Cardiology* 65(7):432-440.
- Woodruff TJ, Parker JD, Schoendorf KC. 2006. Fine particulate matter ( $PM_{2.5}$ ) air pollution and selected causes of postneonatal infant mortality in California. *Environmental Health Perspectives* 114(5):786-90.
- Woods & Poole Economics Inc. 2008. Population by Single Year of Age CD. CD-ROM. Woods & Poole Economics, Inc. Washington, D.C.