Steps towards assessing sources of ozone damages to human health and ecosystems with the CMAQ adjoint

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I. Motivation

The Clean Air Act supports the establishment of a national standard for ambient concentrations of atmospheric pollutants to protect human health and public welfare (CAA, 1990). The primary standard has been viewed as sufficient for also protecting public welfare. We seek to explore how emissions affect these regulatory endpoints differently in the CMAQ chemical transport modeling framework.

Distinct spatial distributions

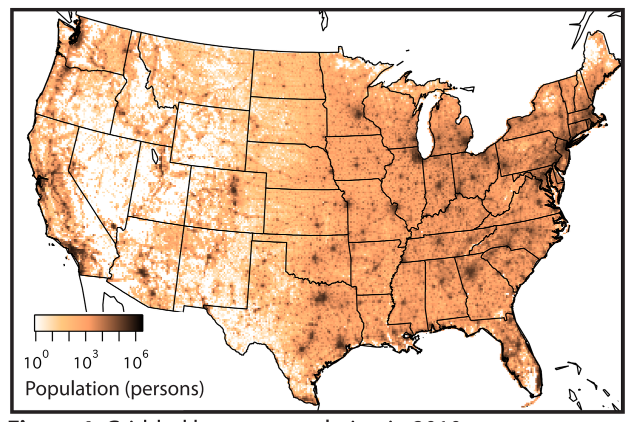


Figure 1. Gridded human population in 2010

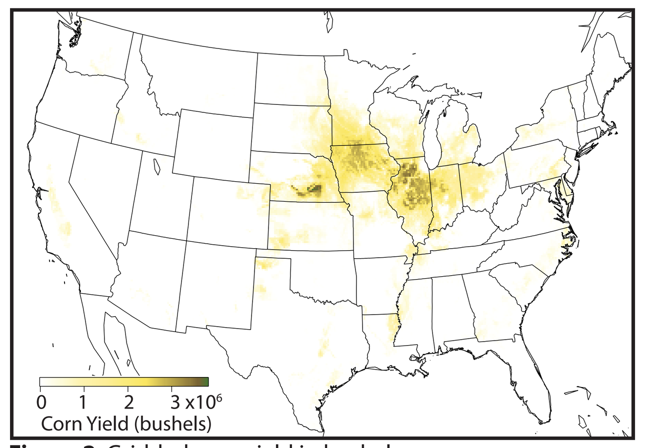


Figure 2. Gridded corn yield in bushels.

Generally, dense human populations are located separately from sensitive ecosystems. Urban non-attainment areas often contain vegetation, but the majority of crops and timber are located in more rural areas where ozone monitors may be more scarce. The separation of these vulnerable populations in space allows the possibility that emissions influences on each endpoint are unique.

Unique response regimes

Epidemiological studies have revealed association between peak ozone concentrations and increased mortality rates (Bell et al., 2004; Schwartz, 2005; Jerrett et al., 2009); therefore, reducing peak ozone concentrations has been the focus of the primary standard, which is formulated as a limit on the 4th highest daily maximum 8-hr average ozone concentration. Over the last three decades, a 25% reduction in this metric has been achieved nationally.

Plants also demonstrate reduced productivity when exposed to elevated ozone concentrations (e.g., Lesser et al., 1990, Mills et al., 2007). However, cumulative exposures to lower concentrations have been shown to reduce yield of crops (and decrease the biomass production of trees (EPA REA, 2012). In addition to responses varying with ozone concentration, the water vapor concentration, to which stomata respond, also affects the influence of ozone on plant health.

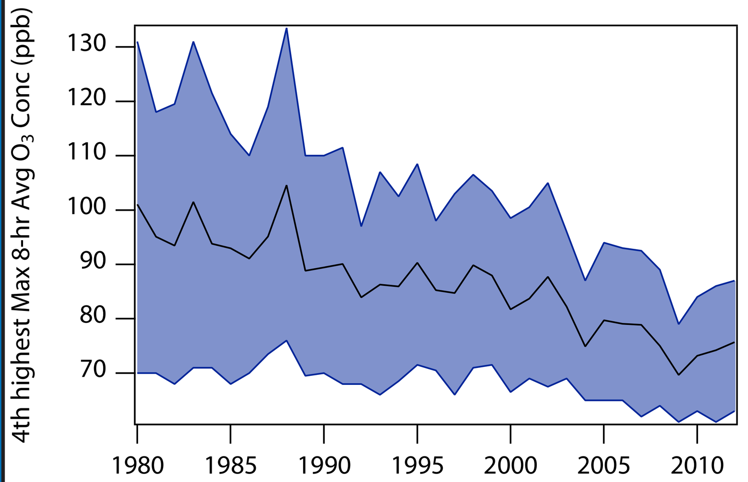


Figure 3. 4th greatest maximum 8-hr average monitored O_3 concentrations have declined over past decades due to emissions controls.

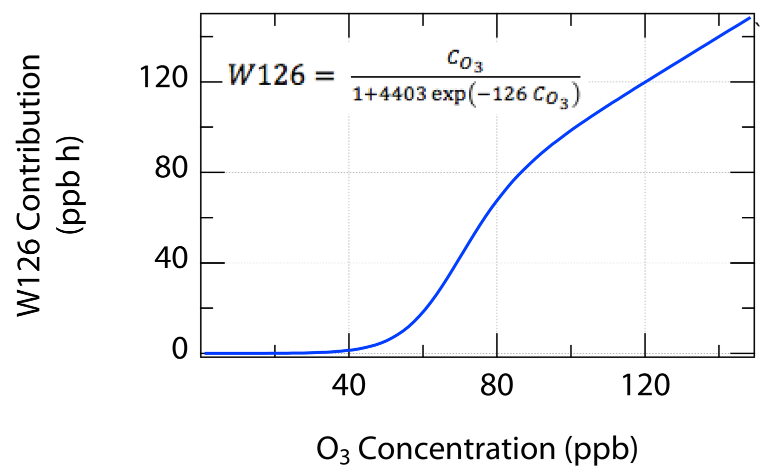
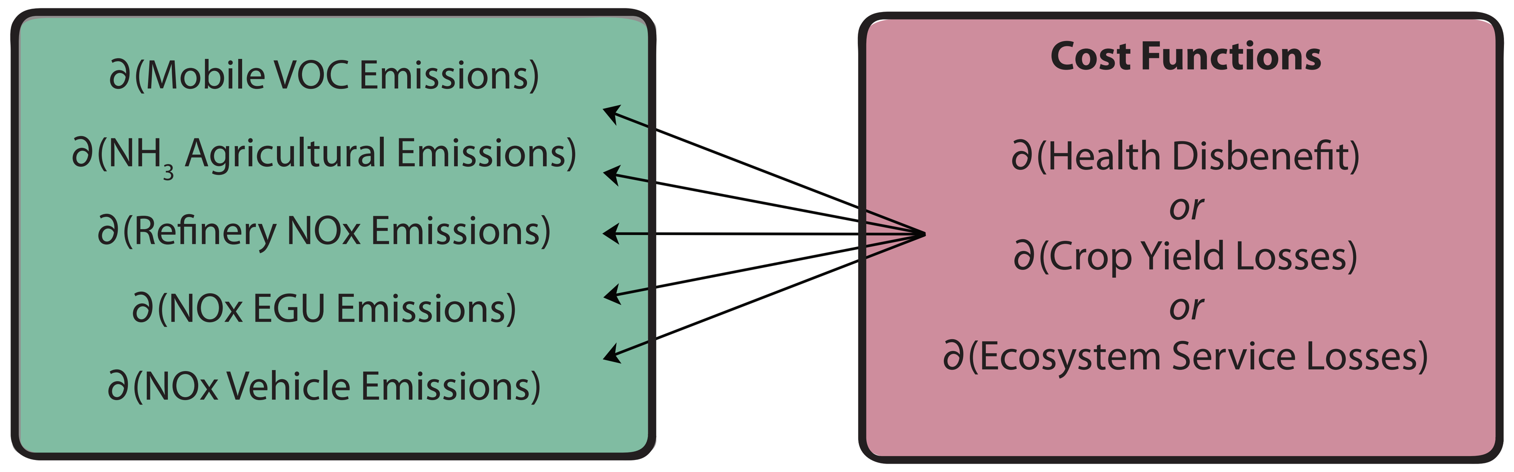


Figure 4. Method for weighting the effect of ozone concentration on plant life, which can be summed over daylight hours in growing season.

Although further refinement of and mechanistic explanations for each dose-response relationship are active areas of research in both human and plant populations, current understanding reveals that both cumulative, lower-concentration and acute, higher-concentration ozone exposure can degrade human health and public welfare. Thus, the relative roles of emissions sources in each endpoint may very well be distinct, potentially warranting consideration of unique regulatory treatment.

II. CMAQ adjoint framework



$$\partial \boldsymbol{x} = (F')^T(x, \partial \boldsymbol{J})$$

The CMAQ adjoint framework of Hakami et al., (2007) facilitates the assessment of relative contributions of each modeled emissions source with respect to a concentration-based metric. Specifically, the derivative of the mathematical relationship between emissions and concentrations is established by the adjoint model. When an adjoint is provided an adjoint forcing based on an end point of interest (i.e., estimated mortality due to ozone exposure), the relative influence of each emissions parameter on the cost function are efficiently determined.

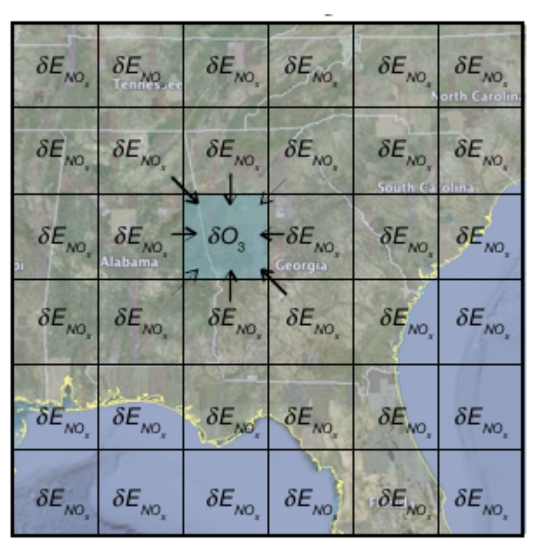


Figure 5. Depiction of the spatial specificity adjoint sensitivities provide when relating the influence of emissions on concentration-based metrics.

When applied, the CMAQ adjoint transforms the adjoint forcing through the chemical and physical processes in the same manner as the forward model treats emitted species. In order to use the CMAQ adjoint to assess the influence of emissions on distinct regulatory endpoints, we must define the cost functions that represent the degradation caused by ozone exposure. Here, we discuss the formulation of human health and ecological cost functions based on 2007 modeled ozone concentrations. One can consider adjoint forcing functions as input to the adjoint model; by analogy to the forward modeling framework, the spatial and temporal resolution is similar to emissions.

III. Evaluating Degradation of Human Health

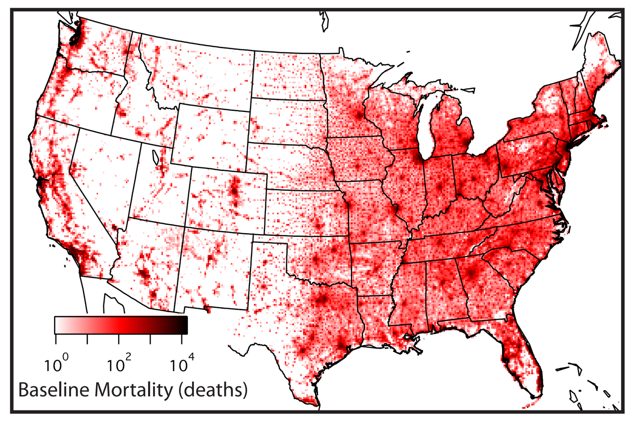


Figure 6. Baseline mortality rate.

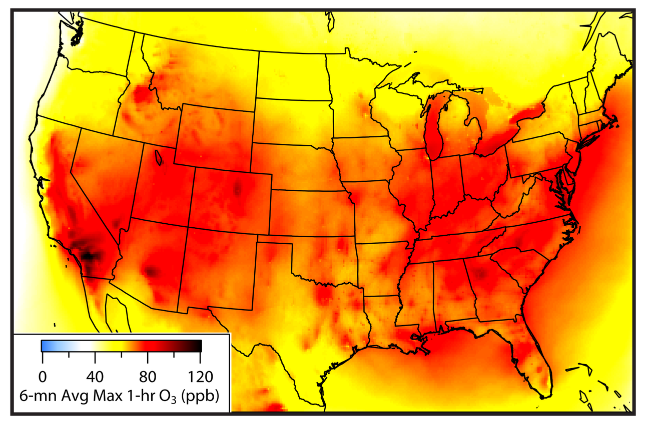


Figure 7. Six-month average maximum hourly ozone concentrations used in Jerrett et al. (2009) mortality function based on CMAQ model output for 2007.

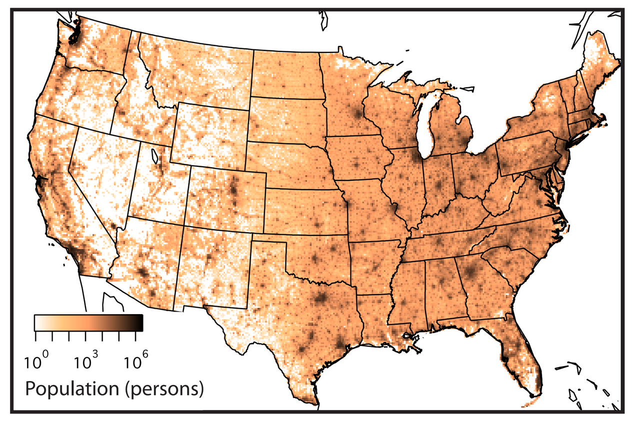


Figure 8. Distribution of human population above 30 years of age.

Recent work by Jerrett et al. (2009) has associated long-term exposure to ozone with death from respiratory causes. The following equation provides a relationship between modeled ozone concentrations and increased mortality rates.

$$\frac{\partial(\text{Mortality})}{\partial(\text{Conc}_i)} = M_0 \ \text{Pop}_{(>30)} \ ß(\exp[-ß\ \text{Conc}_i])$$

where M_0 represents the baseline mortality rate in each grid cell (Figure 6). Conc_i is the maximum six-month mean of the hourly maximum concentration of ozone in each grid cell (Fig. 7). $\text{Pop}_{(>30)}$ represents the humans in each grid cell above 30 years of age. ß is a coefficient determined in the study (0.04 increased mortalities due to respiratory illness per 10 ppb increase in ozone metric) (Jerrett et al., 2009). The offline manner of calculating the adjoint forcing function is particularly useful in this and similar cases where the variable of interest is a function of concentration over a long period of time.

To assess the relative contribution of emissions throughout the episode to the mortality associated with long-term ozone exposure, we prepare an adjoint forcing array that spans the spatial and temporal extent of the modeled domain. Similar to the method of Pappin et al. (2013), we distribute the forcing, ***∂J***, in a manner commensurate with the mortality calculation.

$$\frac{\partial(J)}{\partial([O_3])} = \left[\frac{\partial(\text{Mortality})}{182\,\partial(\text{Conc}_i)}\right]\Bigg|_{\text{max 1-hr}\,[O_3]}$$

Mortality-based Adjoint Forcing

June-July-August average

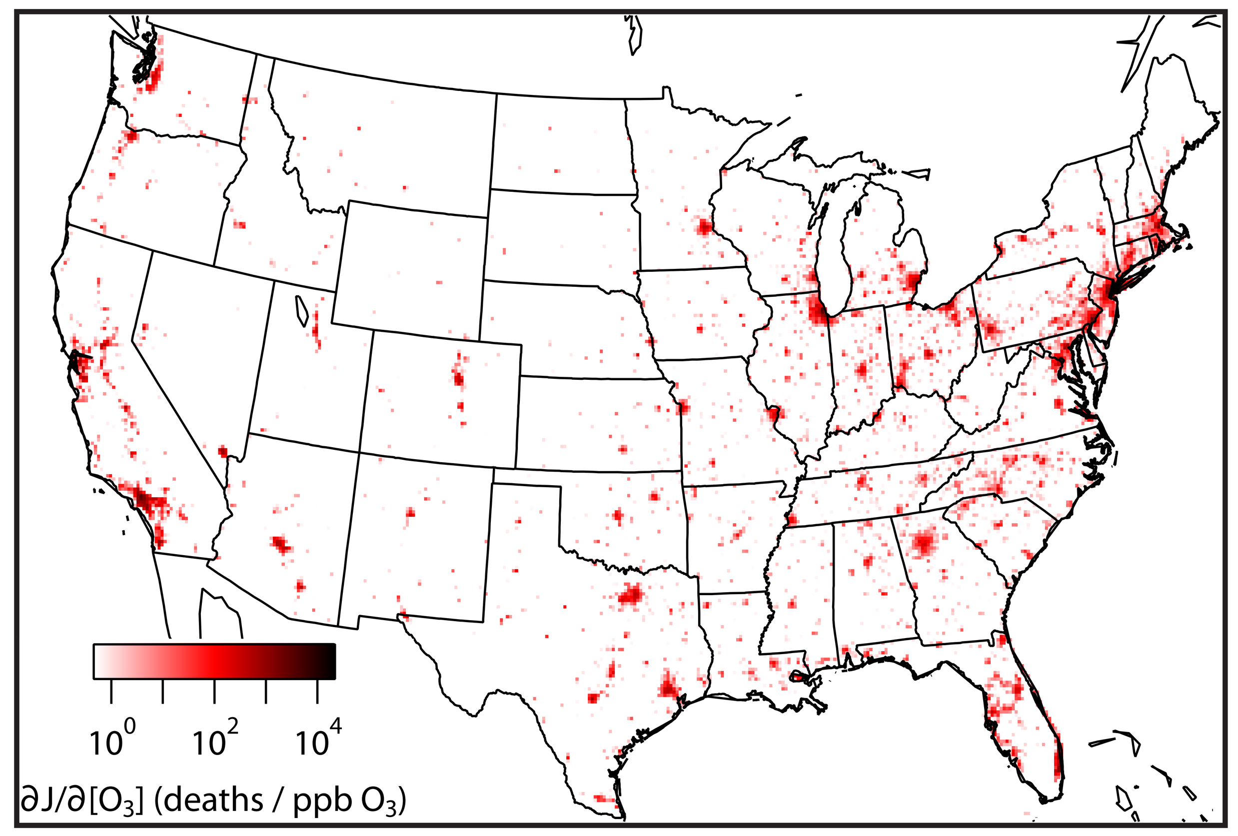


Figure 9. The adjoint forcing function attributes the increased mortality throughout the episode to each grid cell. The forcing is non-zero only in the hour during which the maximum ozone concentration for the day occurs.

IV. Assessing Influence on Crop Yield

In a manner similar to human exposure-response calculations, we consider the effect of ozone exposure on crop yield. Although a number of different ozone metrics exist that are relevant to ecosystem health, the cumulative peak-weighted index, W126, is the most widely accepted metric in the U.S. The seasonal value is calculated as follows:

$$W126_{90\,\text{day}} = \left[\sum_{i=1}^{90}\left(\frac{[O_3]}{1+\left(4403e^{-126[O_3]}\right)}\right)\Bigg|_{i,\text{8am-8pm (LST)}}\right]$$

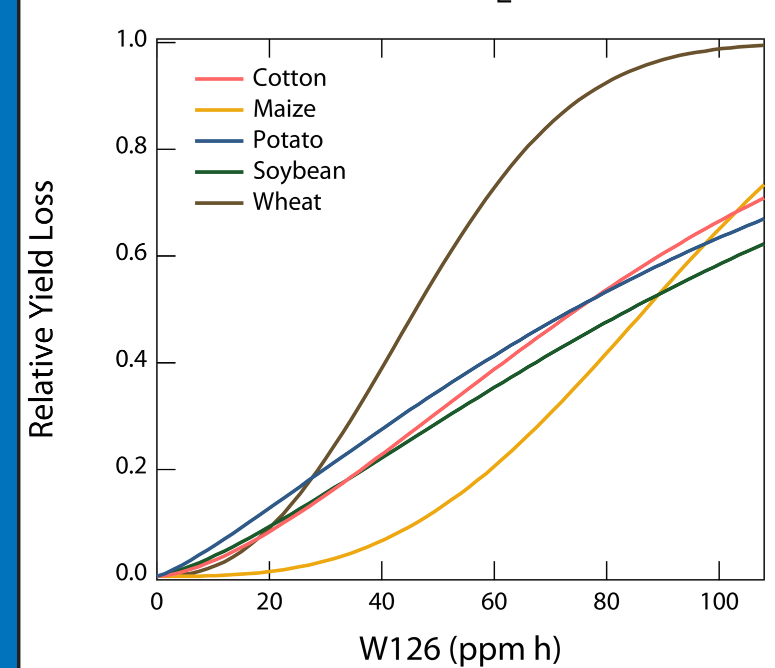


Figure 10. Estimates of the relative yield loss caused by cumulative exposure of crops over the summer months.

In laboratory and field studies, exposure to different W126 values has been correlated with loss of yield in a variety of plants. We focus on maize (corn) and soybean here as they represent a significant portion of the economic gain from plants primarily grown in the summer months, which typically have higher ozone concentrations. The relative yield loss to W126 relationships are shown (Fig. 10) and calculated as follows

$$RYL = 1 - \exp\left[-\left(\frac{W126}{A_i}\right)^{B_i}\right]$$

where A_i and B_i are crop-specific parameters that are empirically determined (Lehrer et al., 2007).

$$\frac{\partial(\text{Crop Loss})}{\partial([O_3])} = \frac{\partial W126}{\partial C_{O_3}}\,\frac{\partial RYL}{\partial W126}\,\frac{\partial YL}{\partial RYL}$$

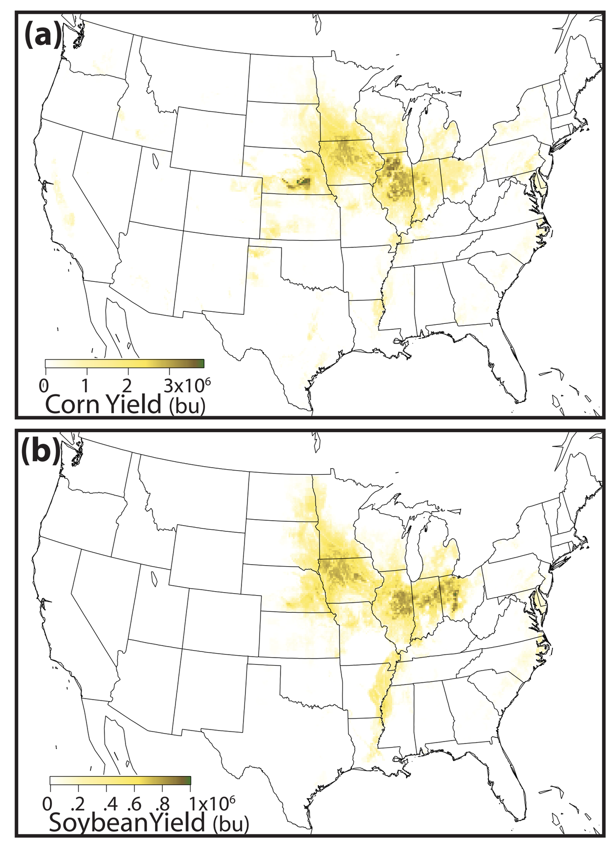
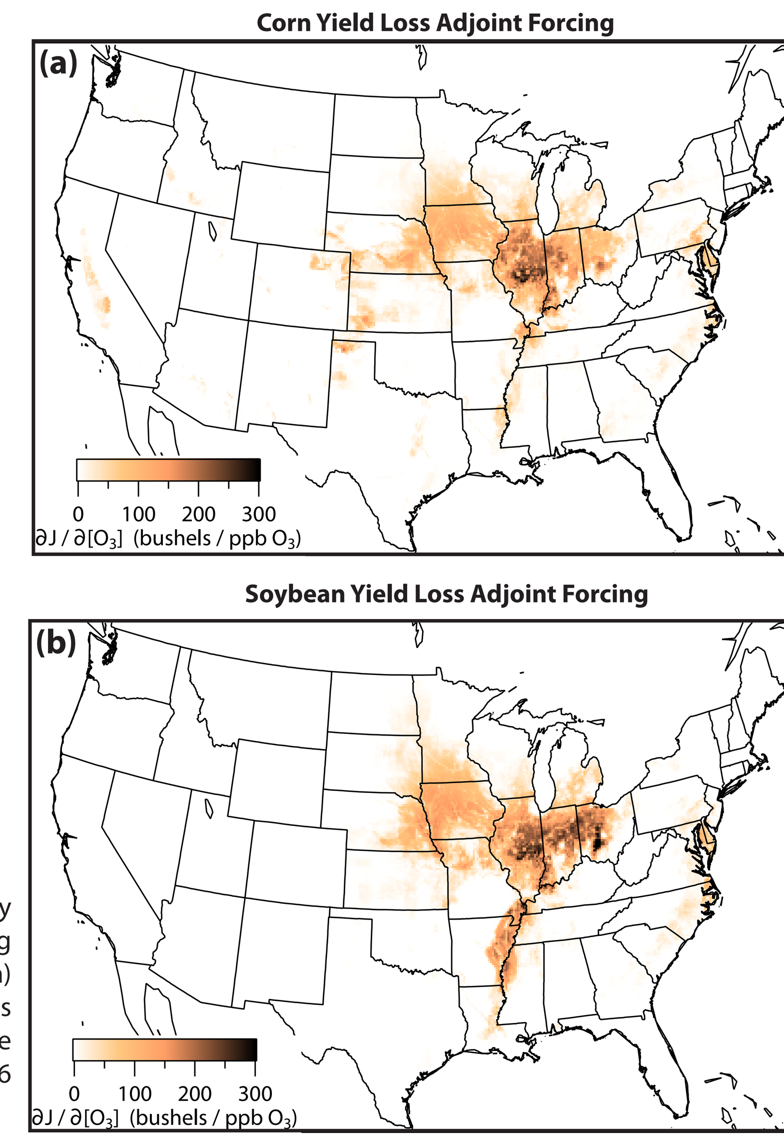


Figure 11. The yield of corn (a) and soybean (b) in bushels. The distribution and amount were reconstructed from 2007 NASS production statistics and BELD land use data.

The seasonal yield loss is distributed across grid cells in accordance with the contribution the ozone in each cell and hour made to the total W126. For instance, the forcing is always zero at night because of the construction of W126. Additionally, the relative yield loss associated with the W126 metric is allocated according to the actual yield in the grid cell.

The differences in the spatial patterns and magnitude between corn and soybeans arise from unique yields, W126 dose-responses, and ozone concentrations at the two different times.

Figure 12. The 90-day average adjoint forcing representing yield loss of (a) corn and (b) soybeans yields due to ozone exposure according to the W126 metric.



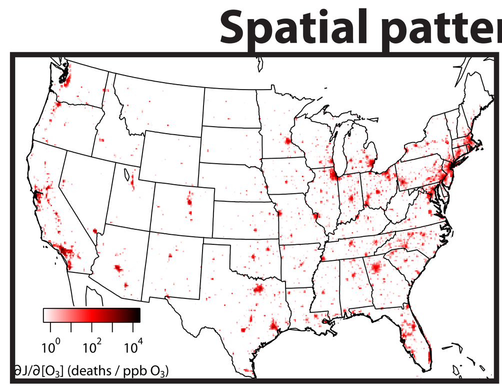
V. Current Message & Next Steps

Modeling developments

Jerrett et al. (2009) based mortality and W126-based crop yield loss adjoint forcing calculation implemented in Python-based CMAQ adjoint preprocessor

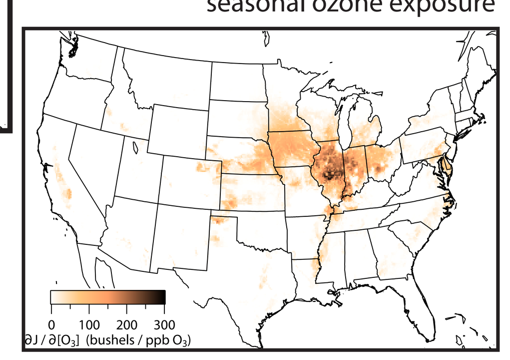
Spatially-distributed primary crop yields constructed for 2007

Spatial patterning of health & crop forcings is distinct



Adjoint forcing of ozone reflecting mortality from respiratory causes due to long-term ozone exposure

Adjoint forcing of ozone reflecting reduced yield of corn crops due to seasonal ozone exposure



First, we will ***apply these adjoint forcings within the CMAQ adjoint framework*** to observe any distinction between emissions influences on each regulatory endpoint.

Then, we will develop similar adjoint forcing calculators for ***additional ecological endpoints*** including timber (based on relationships in Fig. 13 from Lehrer et al. (2007)) and sensitive vegetation.

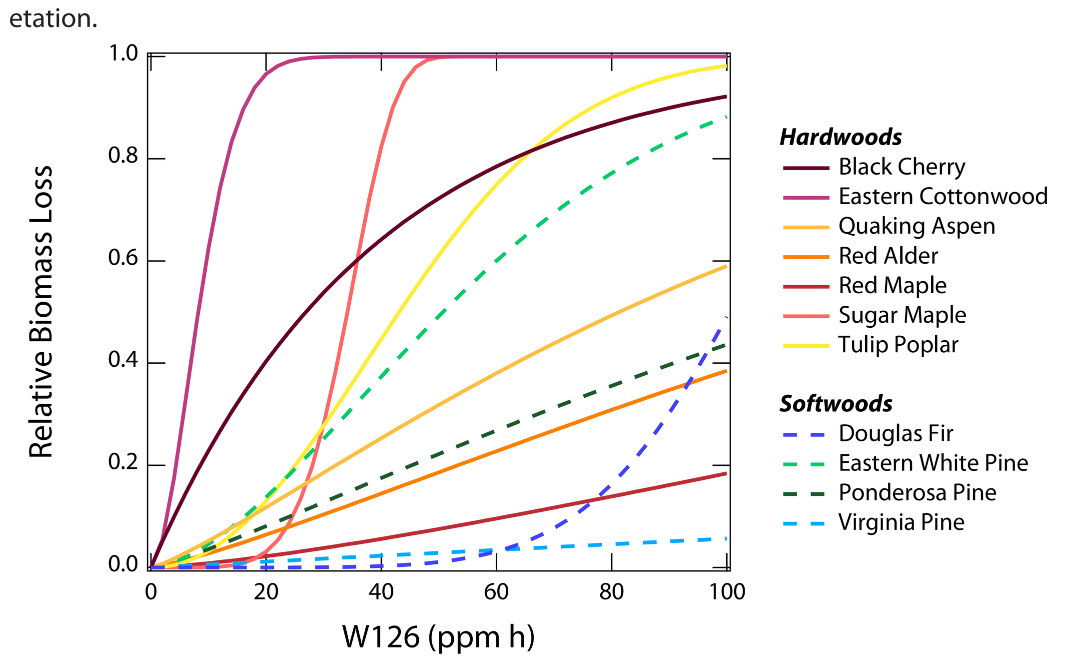


Figure 13. Estimates of the relative biomass loss caused by cumulative exposure over the summer months.

VI. References & Acknowledgements

Bell et al., JAMA, 292(19):2372-2378 (2004).
Clean Air Act Amendments of 1990, P.L. 101-549, 104 Stat. 2399, 1990-11-15.
Hakami et al., Environ Sci Tech, 41(22): 7807–7817 (2007).
Jerrett et al., N Engl J Med, 360:1085-1095 (2009).
Lehrer et al., EPA 452/R-07-002 (2007).
Lesser et al., Crop Sci, 30:148-155 (1990).
Mills et al., Atmos Env, 41:2630-2643 (2007).
Pappin and Hakami, Environ Health Perspect, 121 (5), S. 572–579 (2013).
Schwartz, Am J Respir Crit Care Med ,171: 627-631 (2005).
U.S. EPA, Ozone Standards, Welfare Risk and Exposure Assessment (First External Draft) (2012).

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