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Michelle L. Bell,[1] Roger D. Peng,[2] Francesca Dominici[2]

1 Yale University

School of Forestry & Environmental Studies

205 Prospect St.

New Haven, CT, 06511 USA

2 Johns Hopkins Bloomberg School of Public Health

Dept. of Biostatistics

615 North Wolfe St.

Baltimore, MD, 21205 USA

Corresponding author: M.L. Bell

Phone (203) 432-9869

Fax: 203.432.3817

E-mail: michelle.bell@yale.edu

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Abstract

Time-series analyses have shown that ozone is associated with increased risk of premature mortality, however little is known about how ozone impacts health at low concentrations. A critical scientific and policy question is whether a threshold level exists below which ozone does not adversely impact mortality. We developed and applied several statistical models to data on air pollution, weather, and mortality for 98 U.S. urban communities for the period 1987 to 2000 to estimate the exposure-response curve for tropospheric ozone and risk of mortality and to evaluate whether a "safe" threshold level exists. Methods include a linear approach, and subset, threshold, and spline models. All results indicate that any threshold would exist at very low concentrations, far below current U.S. and international regulations and nearing background levels. For example, under a scenario in which the U.S. Environmental Protection Agency 8-hour regulation is met every day in each community, there was still a 0.30% increase in mortality per 10 ppb increase in the average of the same and previous days' ozone levels (95% posterior interval 0.15, 0.45%). Our findings indicate that even low levels of tropospheric ozone are associated with increased risk of premature mortality. Interventions to further reduce ozone pollution would benefit public health, even in regions that meet current regulatory standards and guidelines.

Tropospheric ozone is a common urban area pollutant linked to numerous harmful health effects, including reduced lung function, increased frequency of respiratory symptoms, and development of asthma (Broeckaert et al. 1999; Brunekreef and Holgate 2002; McConnell et al. 2002; U.S. EPA 1996). Recent meta-analysis and time-series studies have linked short-term ozone exposure to premature mortality (Anderson et al. 2004; Bell et al. 2004 and 2005; Huang et al. 2005; Ito et al. 2005; Levy et al. 2005), however the exposure-response curve for ozone remains unknown. Over 100 million people in the United States live in areas that exceed the current health-based National Ambient Air Quality Standard (NAAQS) for ozone (U.S. EPA 2004). Elevated concentrations of ozone is also a growing concern for rapidly developing nations with rising emissions of ozone precursors from expanding transportation networks.

The U.S. Environmental Protection Agency (U.S. EPA) is currently reviewing the scientific evidence on ozone and health to determine if the current NAAQS (80 ppb for the daily 8-hour maximum) should be revised in order to meet the Clean Air Act mandated goal of protecting human health with an "adequate margin of safety" (U.S. EPA 1997). There are several critical questions regarding the association between ozone and mortality as the current NAAQS is re-examined: a) can ozone impact mortality even at low levels? b) are current regulations sufficiently stringent to prevent premature mortality? and c) is there an attainable threshold ozone level that does not affect mortality, and if so is it below current regulatory limits? Evidence relevant to these questions can be obtained by estimating the exposure-response curve for ozone and mortality. The shape of this curve can provide a basis for: 1) understanding the impacts of

low levels of ozone pollution on health; 2) assessing the adequacy of regulatory standards; 3) designing other health-based studies on ozone; 4) estimating the health consequences associated with emissions scenarios and policies (e.g., Hubbell et al. 2005); and 5) assessing how climate change might impact human health through altered ozone levels (e.g., Knowlton et al. 2004).

Materials and Methods

Data and hierarchical model. To investigate the exposure-response relationship between ozone and mortality, we applied several modeling structures to daily time-series data on all-cause non-accidental mortality, weather (temperature and dew point), and ozone pollution levels for the period 1987 to 2000 for 98 large U.S. urban communities (Figure 1). The communities are listed in the Appendix, and consist of urban areas based a county or a set of contiguous counties. Our database includes over 40% of the total U.S. population and is part of the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) (Daniels et al. 2000 and 2004; Dominici et al. 2000; Samet et al. 2000a, 2000b, and 2000c). Air pollution data were obtained from the U.S. EPA and weather data from the U.S. National Climatic Data Center.

Measurements from ambient monitors were used as a surrogate for community-level exposure. The measure of exposure was the average of the same and previous days' ozone levels (lag $\overline{01}$). First 24-hour averages were calculated for each day within each community, and then the lag $\overline{01}$ concentrations were calculated. The use of any single day's ozone level as the exposure metric would underestimate the relationship between

ozone and mortality (Bell et al. 2004). Measurements from multiple monitors within a community were aggregated using a 10% trimmed mean to estimate a community-level exposure.

Mortality data were obtained from the National Center for Health Statistics. The mortality outcome is the number of daily deaths within the community excluding non-residents and excluding those caused by injuries and other external causes corresponding to International Classification of Diseases, Ninth Revision (ICD-9) codes 800 and above, and International Classification of Diseases, 10th Revision (ICD-10) codes S and above. Additional information on the generation of the air pollution dataset is available at http://www.ihapss.jhsph.edu/data/NMMAPS/FlowCharts/pollution.htm. Our entire database is available through the Internet-based Health & Air Pollution Surveillance System (iHAPSS) at http://www.ihapss.jhsph.edu/.

We used a Bayesian hierarchical model to evaluate the relationship between ambient ozone levels and mortality rates within each community (community-specific relative rate estimate) and to combine information across communities to produce a national average relative rate estimate, accounting for the uncertainty of each community's relative rate (Dominici et al 2000; Everson and Morris 2000). The first stage estimates the relationship between short-term exposure to ozone and daily non-accidental mortality rates within each community, using a Poisson regression model (McCullagh and Nelder 1989) of the form:

$$\log(\mu_t^c) = \beta^c x_t^c + \gamma^c DOW_t + ns(time_t, 7/yr) + ns(T_t^c, 6) + ns(T_{t-1,t-3}^c, 6) \quad [1]$$

$$+ ns(D_t^c, 3) + ns(D_{t-1,t-3}^c, 3) + interaction\ terms\ for\ age\ and\ time$$

where:

μ_t^c = expected number of deaths for community *c* on day *t*, based on an over-dispersed Poisson distribution

x_t^c = average of the same and previous days' daily ozone concentrations in community *c* on day *t*

DOW_t = categorical variable for day of the week on day *t*

$ns(time_t, 7/yr)$ = natural cubic spline function of calendar time with 7 degrees of freedom per year

$ns(T_t^c, 6)$ = natural cubic spline function for temperature with 6 degrees of freedom

$ns(T_{t-1,t-3}^c, 6)$ = natural cubic spline function of the average of the three previous days' temperature (adjusted for current day temperature)

$ns(D_t^c, 3)$ = natural cubic spline function for dew point with 3 degrees of freedom

$ns(D_{t-1,t-3}^c, 3)$ = natural cubic spline function of the average of the three previous days' dew point (adjusted for current day dew point)

interaction terms for age and time = interaction terms between natural cubic spline functions of time and age-specific indicators (< 65, 65 to 74, and $\geq$ 75 years)

In the first stage, we estimated the effect of ozone on mortality for each community, $\hat{\beta}^c$, (an estimate of the true community-specific relative rate, β^c), and the corresponding variance $\hat{v}^c$. We assume:

$$\hat{\beta}^c \mid \beta^c, \hat{v}^c \sim N\left(\beta^c, \hat{v}^c\right) \quad [2]$$

$$\beta^c \mid \mu, \tau^2 \sim N\left(\mu, \tau^2\right) \quad [3]$$

where μ is the true national average relative rate and τ^2 is the variance of the true community-specific relative rates, β^c. Sensitivity analyses and characteristics of the first stage statistical model for confounding adjustment have been explored for particulate matter (PM), with results indicating that national average estimates are robust to model specification for weather and seasonal confounding (Peng et al. 2005; Welty and Zeger 2005). Earlier analysis showed national average and community-specific estimates for ozone and mortality to be robust to inclusion of PM_{10} (PM with an aerodynamic diameter less than 10 microns) in the first stage model (Bell et al. 2004). Results were also robust to exclusion of days with high temperature (Bell et al. 2004).

As a second stage, we generated a national relative rate estimate that accounts for the statistical uncertainty of each community's relative rate estimate and for the variability across communities of the true relative rates. We fit this two-stage normal-normal model by use of a Two-Level Normal independent sampling estimation (TLNise) with non-informative priors (Everson and Morris 2000). TLNise is available online at http://www.swarthmore.edu/NatSci/peverso1/TLNise/tlnise.htm.

Using this two-stage approach, we performed four analyses that make different modeling assumptions about the community-specific exposure-response curve for ozone and mortality. Under each analysis, we estimated a national relative rate and/or a national exposure-response curve by combining information across the 98 communities.

Linear approach. For the first analysis, the *linear approach*, we estimated a linear association between the log of the expected mortality rate and ozone levels as described in Equation [1]. This model assumes that any change in ozone concentration, even at very low levels, can be associated with mortality. For example, a 10 ppb increase in ozone levels from 5 to 15 ppb would lead to the same percent increase in mortality as a 10 ppb rise from 50 to 60 ppb. This is the modeling approach used in most epidemiological studies of air pollution and health and impact assessments of air pollution policies. We then relax this assumption of linearity across the entire range of ozone levels with the three approaches described below.

Subset approach. Under the second analysis, the *subset approach*, we estimated a linear relationship between the log of the expected mortality rate and ozone levels as in Equation [1] but using a subset of the data including only days with lag $\overline{01}$ ozone levels below a specified concentration, *s*. We performed this analysis for values of *s* ranging from 5 to 60 ppb. Under this approach, we assume that "safe" ozone levels are those lower than the specific *s* value that leads to lack of evidence of an association between ozone and mortality.

We also used the *subset approach* to assess the relationship between ozone and mortality under several idealized policy scenarios in which various ozone regulations and guidelines were met every day in each community. Because ozone regulations are expressed in different metrics, we proceeded in three steps. First, we used hourly ozone concentrations to calculate daily ozone levels under the same metric specified by the standard (e.g., daily 8-hour maximum or daily 1-hour maximum). Second, we constructed

a subset of the dataset that includes only days that meet the regulatory standard or guideline. For example, for the U.S. EPA ozone standard we first calculated a daily time series of 8-hour maximum ozone levels, and then we constructed a subset of the dataset that only includes days with an 8-hour maximum ozone level lower than 84 ppb (U.S. EPA 1997). Third, using only days that met the standard, we estimated the percent increase in mortality associated with a 10 ppb increase in lag $\overline{01}$ ozone levels on average across the 98 communities, with the 95% posterior interval, which is the Bayesian analogue of the 95% confidence interval. This strategy allows us to analyze the subset of days that meet a regulatory requirement using the metric specified in the standard, but to present results with a single metric for the exposure variable (lag $\overline{01}$) to maintain a common interpretation of the relative rate estimates.

The U.S. NAAQS for ozone is "80 ppb" for the daily 8-hour maximum, however U.S. EPA regulations specify that values between 80 and 84 ppb can be rounded down and are not considered exceedances (U.S. EPA 1997). Thus, for our analysis of the U.S. NAAQS we considered a standard of 84 ppb for the daily 8-hour maximum. Regulations generally do not require every monitor to meet the standard every day. For example, a standard can allow a specified number of exceedances and require that a certain percentile (e.g., 98^{th}) meet the requirement on a 3-year average. In actual compliance with a regulatory standard for a given area, the levels of pollution would follow an uneven spatial distribution (U.S. EPA 2005). Our analysis considers a more stringent application in that it incorporates only days with ozone levels at or below the specific standard for both the same and previous days. However, the regulatory standard requires

compliance from every monitor, whereas this analysis considers averages across communities rather than individual monitor exceedances.

Threshold approach. If a threshold (h) exists, we would expect to detect an association between ozone and mortality for ozone levels > h but not for ozone levels < h. Our *threshold approach* has the same structure of Equation [1], but with the pollution term replaced by:

$$\left(x_t^c - h\right)_+ \qquad [4]$$

where:

$$\left(x_t^c - h\right)_+ = \left(x_t^c - h\right) \text{ if } x_t^c \geq h \qquad [5]$$

$$= 0 \text{ otherwise}$$

Under this model we assume no association between ozone and mortality for days with ozone concentrations below h and a linear relationship for days with ozone levels above h. We performed this analysis for values of h ranging from 0 to 60 ppb at increments of 5 ppb. For each community-specific model and threshold level (h), we calculated the Akaike Information Criterion as:

$$AIC^c(h) = \text{Deviance} + 2 \times (\#\text{ of parameters}) \qquad [6]$$

Note that the number of parameters can differ by urban community due to the varying frequencies with which ozone is measured and the variables for time. We then calculated the average *AIC* for each h value as:

$$\overline{AIC}(h) = \frac{1}{N}\sum_{c=1}^{N} AIC^c(h) \qquad [7]$$

where:

N = number of communities (98)

The rationale for this approach is that if an ozone threshold exists, the *threshold approach* with the appropriate value for *h* will have the best fit and therefore the minimum $\overline{AIC}(h)$ (Akaike 1973).

Spline approach. Under the fourth analysis, the *spline approach*, we allow the relationship between ozone and mortality to fluctuate for different ranges of pollution levels, using a non-linear function of ozone. This model can be defined as Equation [1] but replacing $\beta^c x_t^c$ with $ns(x_t^c)$, where *ns* is a natural cubic spline of ozone levels (Daniels et al. 2000 and 2004; Dominici et al. 2002). Boundary knots were specified at 0 and 80 ppb, with interior knots at 20 and 40 ppb. The *spline approach* extends the *linear approach* because here the relative rate corresponding to a 10 ppb increase in ozone levels from 5 to 15 ppb is allowed to differ from the relative rate corresponding to a 10 ppb increase from 50 to 60 ppb. Visual inspection of the estimated exposure-response curve can provide evidence about whether a safe level exists and at what concentration.

Results

We found that daily increases in ambient ozone levels were significantly associated with daily increases in the number of deaths, on average across the 98 U.S. communities. Specifically, under the *linear approach,* we found that the percent increase in all-cause mortality associated with a 10 ppb increase in the lag $\overline{01}$ ozone levels was 0.32% (95% posterior interval 0.17, 0.46%). We also found that the largest relative rate estimates

occur on more recent days: the percent increases in all-cause mortality associated with a 10 ppb increase in lag $\overline{01}$ daily ozone levels were 0.25% (0.12, 0.38%); 0.18% (0.07, 0.30%); 0.14% (0.03, 0.26%); and 0.04% (-0.07, 0.16%) for single-day lags of 0, 1, 2, and 3 days, respectively. The community-specific maximum likelihood estimates under the linear approach displayed no association with the communities' long-term ozone concentrations over the study period, as tested by correlation and weighted second-stage regression.

Our results show that daily increases in ambient ozone were significantly associated with daily increases in the number deaths, on average across the 98 U.S. communities for the idealized policy scenarios under which every community meets current ozone regulatory standards and guidelines (California Environmental Protection Agency 2005; Canadian Council of Ministers of the Environment 2000; European Commission 2002; U.S. EPA 1997; WHO 2000) for every day of the study period, 1987 to 2000 (Table 1). For example, the percent increase in all-cause mortality associated with a 10 ppb increase in lag $\overline{01}$ ozone levels was 0.30% (0.15, 0.45%) when we used a dataset including only days with a daily 8-hour maximum ozone concentration lower than U.S. ozone regulations. We also found that daily increases in ambient ozone exposure are linked to premature mortality under compliance with other ozone regulations, including some more stringent than the U.S. standards. In summary, these results indicate that current regulations, even California's new, more stringent standards, are not sufficiently low to provide complete protection against the risk of premature mortality from ozone.

Daily changes in ambient ozone were significantly associated with daily changes in the number of deaths, on average across the 98 U.S. communities, even when we used data that includes only days with lag $\overline{01}$ average ozone levels lower than 15 ppb. Figure 2 shows the estimated percent increase in mortality for a 10 ppb increase in the lag $\overline{01}$ ozone level for different values of *s*. National relative rate estimates for *s* values ranging from 35 to 60 ppb are similar to the ones obtained by using all data. The 95% posterior interval increases as *s* is lowered due to the decreasing sample size. For example, at an *s* value of 40 ppb, 30% of days are excluded from analysis, on average across the 98 communities. At an *s* of 20 ppb, 73% of days are excluded. The estimates decline and lose significance only when *s* is equal to very low concentrations ($\leq$ 10 ppb). Therefore the *subset approach* suggests that a safe ozone level would be lower than approximately 10 ppb, for the lag $\overline{01}$ daily ozone level, which is roughly 15 to 19 ppb for the maximum 8-hour average. However, relative rate estimates for *s* at or below 10 ppb have large statistical uncertainty because of the very small number of days with ozone concentrations so low. In fact 73 communities were excluded entirely at an *s* of 5 ppb due to insufficient data.

Results from the *threshold* and *spline approaches* are consistent with those from the *subset approach* and provide evidence that a safe ozone level can only exist at very low concentrations. We found that the model fit under the *threshold approach* for values of *h* from 5 to 60 ppb never provides more than a nominal improvement (<1% difference in the $\overline{AIC}$) over the model fit under the *linear approach* (analogous to the *threshold approach* with $h = 0$) for the national average and each individual community. In other

words, a model that allows for a safe ozone level fit the data only marginally better than a model that assumes any level of ozone pollution, even low concentrations, can be associated with mortality. The *spline approach* indicates that the national average exposure-response curve obtained using natural cubic splines is near horizontal, indicating the lack of evidence for an association, only at the very low concentrations (from 0 to ~10 ppb) and then becomes approximately linear at higher concentrations (Figure 3).

Discussion

In summary, our nationwide study provides strong and consistent evidence that daily changes in ambient ozone exposure are linked to premature mortality, even at very low pollution levels, including an idealized scenario of complete adherence to current ozone regulations. We also found robust evidence of this relationship between ozone exposure and mortality when we used data that included only ozone levels nearing background concentrations, which typically range from 10 to 25 ppb (Fiore et al. 2003). Therefore, any anthropogenic contribution to ambient ozone, however slight, still presents an increased risk for premature mortality.

Results from this multi-site national study are consistent with single-site time-series studies that found no evidence of a "safe" ozone level at concentrations higher than background levels. Consistent with the results obtained under our *spline approach*, Kim et al. (2004) found that a spline model indicated a threshold around 20 to 30 ppb for the daily 1-hour maximum, which is approximately equal to 8 to 12 ppb for the 24-hour

average, using five years of data for Seoul, Korea. Hoek et al. (1997) found that relative risk estimates of mortality associated with daily changes in ozone were robust to exclusion of days with a 24-hour average $\geq$ 40 $\mu g/m^3$ (about 20 ppb) in a study of Rotterdam, the Netherlands, and concluded that should a threshold exist, it may be at a low concentration. Adverse health responses such as decreases in pulmonary function and alterations in the respiratory tract and declines in lung function have been observed at ozone levels close to background concentrations (Chan and Wu 2005; WHO 2000). Ozone levels below U.S. EPA regulations have been associated with increased frequency of respiratory symptoms in children with asthma (Gent et al. 2003).

Pollution levels below air quality regulatory standards should not be misinterpreted as safe for human health. For instance, the San Joaquin Valley Air Pollution Control District refers to the standards as the "highest level of ozone that can be present without adverse health effects" (San Joaquin Valley Air Pollution Control District 2006). However, decision-makers and the public should distinguish between the complete absence of harm and a lessened or acceptable risk. In fact, the interpretation of an "adequate margin of safety" and what is a "safe" level could depend on the individual, as people may differ in their susceptibility to air pollutants, as well as on the evolving knowledge about the health impacts of air pollution at low levels (American Thoracic Society 2000). This research shows that any reduction in ambient ozone levels, such as through transportation planning in urban areas, should yield important benefits to public health, even in areas that meet current regulatory standards. Persons may be adversely

affected by ozone pollution, even at very low levels including days that meet current regulatory requirements.

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Table 1. National effect estimates under the scenario that a specific regulation or guideline is met every day in each community

Note: The numbers in parentheses represent the 95% posterior interval.

Organization/government	*Regulation/guideline*	*Increase in mortality for 10 ppb increase in lag* $\overline{01}$ *ozone*
U.S. EPA	84 ppb daily 8-hour max	0.30% (0.15, 0.45%)
World Health Organization (guideline)	120 μg/m^3 (~61 ppb) daily 8-hour max	0.25% (0.06, 0.43%)
European Commission (target value for 2010)	120 μg/m^3 (~61 ppb) daily 8-hour max	0.25% (0.06, 0.43%)
Canada (to be achieved by 2010)	65 ppb daily 8-hour max	0.28% (0.11, 0.45%)
California	70 ppb daily 8-hour max	0.30% (0.14, 0.46%)
	90 ppb daily 1-hour max	0.29% (0.14, 0.44%)
	Both of California's above standards	0.31% (0.14, 0.47%)
n/a	All of the above standards and guidelines	0.24% (0.06, 0.42%)
Considering all days	n/a	0.32% (0.17, 0.46%)

Figure Legends

Figure 1. Locations of the 98 U.S. urban communities

Figure 2. Percent increase in daily non-accidental mortality per 10 ppb increase in lag $\overline{01}$ ozone obtained by using the *subset approach*.

Note: The circle denotes the point estimate, and the vertical line represents the 95% posterior interval. Each estimate is obtained by including in the analysis only days with 24-hour average lag $\overline{01}$ ozone levels below the *s* value specified on the x-axis. Not all communities had sufficient data for analysis at all *s* values: * = 25 communities; ** = 74 communities; and *** = 92 communities. All other estimates used 98 communities. The estimate at the far right marked by a square uses all data.

Figure 3. Exposure response curve for ozone and mortality using the *spline approach*

Note: This shows the percent increase in daily non-accidental mortality at various ozone concentrations using the *spline approach.* The black line represents the central estimate, and the green lines represent the 95% posterior interval.

Figure 1. Locations of the 98 U.S. urban communities

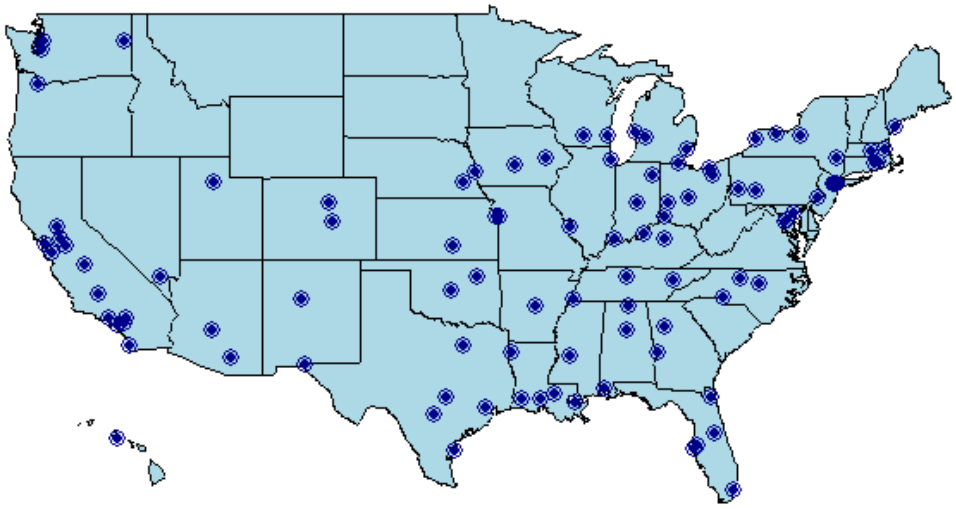


Figure 2. Percent increase in daily non-accidental mortality per 10 ppb increase in lag $\overline{01}$ ozone obtained by using the *subset approach*.

Note: The circle denotes the point estimate, and the vertical line represents the 95% posterior interval. Each estimate is obtained by including in the analysis only days with 24-hour average lag $\overline{01}$ ozone levels below the *s* value specified on the x-axis. Not all communities had sufficient data for analysis at all *s* values: * = 25 communities; ** = 74 communities; and *** = 92 communities. All other estimates used 98 communities. The estimate at the far right marked by a square uses all data.

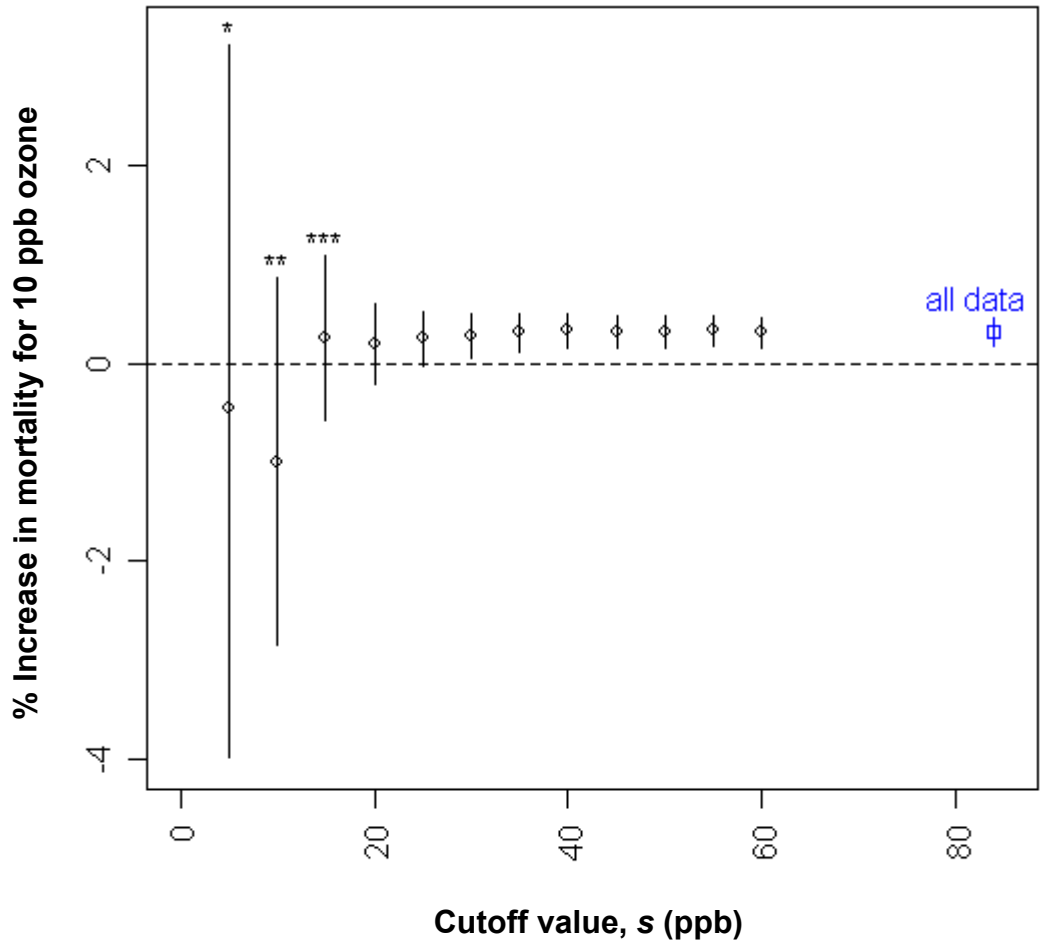
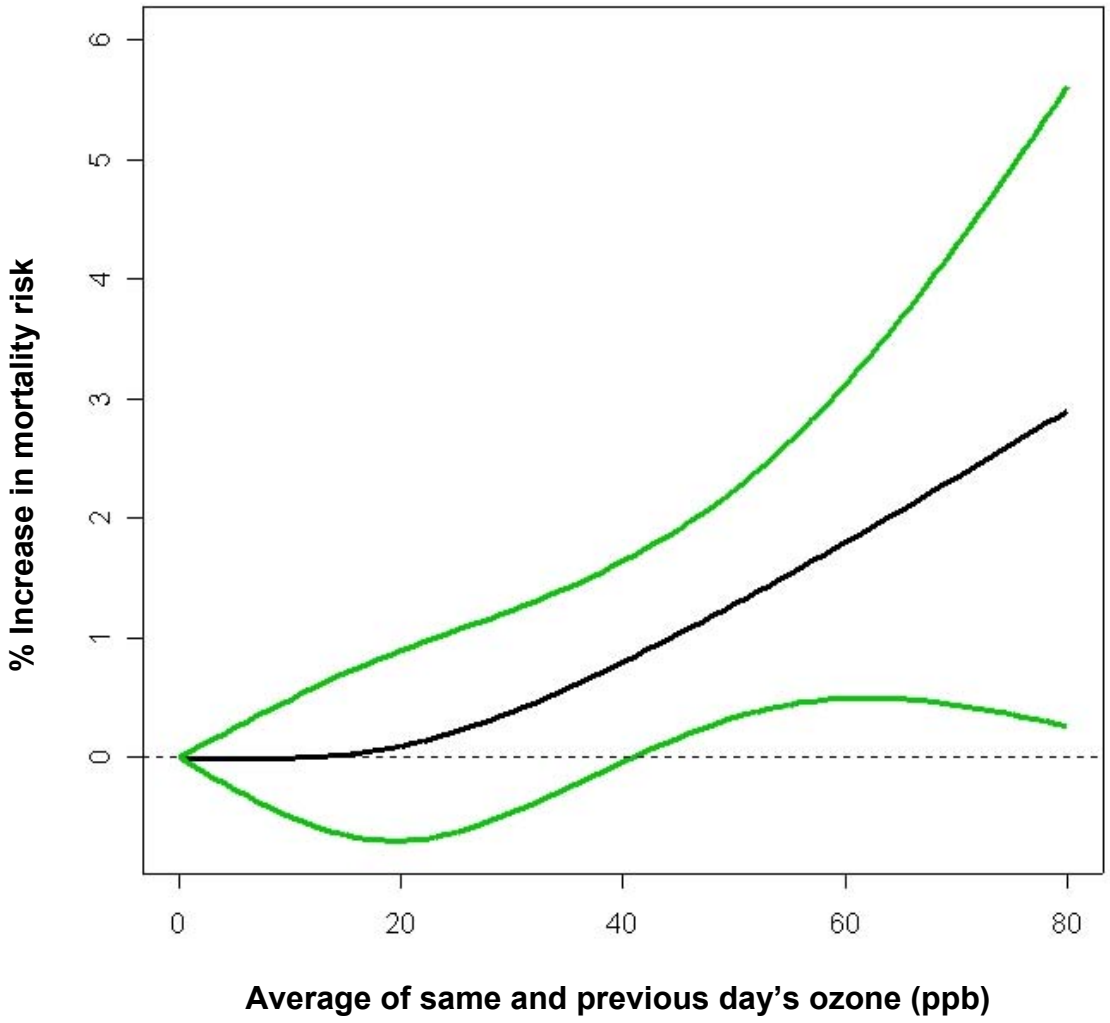


Figure 3. Exposure response curve for ozone and mortality using the *spline approach*

Note: This shows the percent increase in daily non-accidental mortality at various ozone concentrations using the *spline approach.* The black line represents the central estimate, and the green lines represent the 95% posterior interval.



Appendix. List of 98 U.S. Urban Communities

Descriptive statistics for each community are available at:

http://www.ihapss.jhsph.edu/data/NMMAPS/descriptives/frame.htm.

Akron, Ohio	Greensboro, North Carolina	Oklahoma City, Oklahoma
Albuquerque, New Mexico	Honolulu, Hawaii	Omaha, Nebraska
Arlington, Virginia	Houston, Texas	Orlando, Florida
Atlanta, Georgia	Huntsville, Alabama	Philadelphia, Pennsylvania
Austin, Texas	Indianapolis, Indiana	Phoenix, Arizona
Bakersfield, California	Jackson, Mississippi	Pittsburgh, Pennsylvania
Baltimore, Maryland	Jacksonville, Florida	Portland, Oregon
Baton Rouge, Louisiana	Jersey City, New Jersey	Providence, Rhode Island
Biddeford, Maine	Johnstown, Pennsylvania	Raleigh, North Carolina
Birmingham, Alabama	Kansas City, Kansas	Riverside, California
Boston, Massachusetts	Kansas City, Missouri	Rochester, New York
Buffalo, New York	Kingston, New York	Sacramento, California
Cedar Rapids, Iowa	Knoxville, Tennessee	Salt Lake City, Utah
Charlotte, North Carolina	Lafayette, Louisiana	San Antonio, Texas
Chicago, Illinois	Lake Charles, Louisiana	San Bernardino, California
Cincinnati, Ohio	Las Vegas, Nevada	San Diego, California
Cleveland, Ohio	Lexington, Kentucky	San Jose, California
Colorado Springs, Colorado	Lincoln, Nebraska	Santa Ana / Anaheim, California

Columbus, Georgia
Columbus, Ohio
Corpus Christi, Texas
Coventry, Rhode Island
Dallas / Fort Worth, Texas
Dayton, Ohio
Denver, Colorado
Des Moines, Iowa
Detroit, Michigan
District of Columbia
El Paso, Texas
Evansville, Indiana
Fort Wayne, Indiana
Fresno, California
Grand Rapids, Michigan
Little Rock, Arkansas
Louisville, Kentucky
Low Angeles, California
Madison, Wisconsin
Memphis, Tennessee
Miami, Florida
Milwaukee, Wisconsin
Mobile, Alabama
Modesto, California
Muskegon, Michigan
Nashville, Tennessee
New Orleans, Louisiana
New York, New York
Newark, New Jersey
Oakland, California
Seattle, Washington
Shreveport, Louisiana
Spokane, Washington
St. Louis, Missouri
St. Petersburg, Florida
Stockton, California
Syracuse, New York
Tacoma, Washington
Tampa, Florida
Toledo, Ohio
Tucson, Arizona
Tulsa, Oklahoma
Wichita, Kansas
Worcester, Massachusetts