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Effect modification of ozone-related mortality risks by temperature in 97 US cities

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ABSTRACT

Many time-series studies have characterized the relationship between short-term ozone exposure and adverse health outcomes, controlling for temperature as a confounder. Temperature may also modify ozone effects, though this has been largely under-investigated. In this study, we explored whether temperature modifies the effect of short-term ozone exposure on mortality. We used the database developed for the National Morbidity and Mortality Air Pollution Study to estimate ozone mortality risks in 97 US cities in May through September, 1987–2000. We treated temperature as a confounder as well as an effect modifier by estimating risks at low, moderate, and high temperature categories. When temperature was treated as a confounder, a 10-ppb increase in daily 24-h ozone was associated with a 0.47% (95% CI: 0.19%–0.76%) increase in mortality. When we assessed effect modification by temperature, the interaction between ozone and temperature was not statistically significant. However, there was a U-shaped pattern in mortality risk, which was greater at the low (<25th percentile) and high (>75th percentile) temperature levels than moderate temperature levels. At the high temperature category, a 10% increase in AC prevalence mitigated mortality risk associated with 10-ppb of ozone exposure by −0.18% (95% CI: −0.35%, −0.02%). Furthermore, ozone mortality risk in the high temperature category increased as we restricted our analyses to hotter days. On days where temperatures exceeded the 75th, 90th, and 95th percentile temperatures, a 10-ppb increase in ozone was associated with a 0.65% (95% CI: 0.20%–1.09%), 0.83% (95% CI: 0.17%–1.48%), and 1.35% (95% CI: 0.44%–2.27%) increase in mortality, respectively. These results suggested that high temperatures may exacerbate physiological responses to short-term ozone exposure.

1. Introduction

Epidemiological studies have demonstrated links between short-term ozone exposure and a host of adverse health effects, which include respiratory illnesses, acute respiratory symptoms, emergency department visits, hospital admissions, and premature mortality (Bell et al., 2004; Ostro and Rothschild, 1989; Peel et al., 2005; Schwartz, 1994; US EPA, 2010a). Ozone-related mortality risk estimates are particularly sensitive to the model specification of weather parameters, especially temperature (Thurston and Ito, 2001). This is largely attributable to the fact that ozone and temperature are highly correlated, as temperature influences the rate at which ozone forms.

Ozone mortality risk estimates vary widely in the epidemiological literature and yield a broad range of mortality estimates when they are applied in ozone health impact assessments. For example, a previous study estimated 4700 to 19,000 premature deaths to be attributable to ozone exposure in the continental US in 2005 by applying risk estimates from a time-series and a long-term cohort epidemiological study (Fann et al., 2011). In addition, a 2010 US Environmental Protection Agency (EPA) analysis estimated between 890 and 4000 premature deaths avoided due to reductions in short-term exposure to ozone resulting from attaining a more stringent ozone standard (US EPA, 2010b). In these and many other studies characterizing the ozone health impacts, the authors apply risk estimates from epidemiological studies that model temperature as a confounder.

Few time-series studies have investigated the potential for temperature to modify ozone-related health effects (Ren and Tong, 2006; Ren et al., 2006, 2008, 2009; Roberts, 2004). Toxicology studies conducted in mice have shown evidence that temperature modulates health responses to air pollution exposure (Watkinson, 2003). Furthermore, epidemiologists and risk assessors are increasingly interested in characterizing the joint impacts of multiple stressors to account for both the interactive and individual effects, as assessing risks from multiple pollutants or multiple stressors simultaneously may yield a more holistic view of air pollution health impacts (NARSTO, 2004; National Research Council, 2004).

In this study, we estimate ozone-related mortality risk and assessed temperature as an effect modifier in 97 communities in the continental US from 1987 to 2000. As noted above, while the epidemiological literature on ozone-related mortality risk is extensive, only a few studies

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have assessed temperature as an effect modifier and even fewer have explicitly compared risk estimates with and without accounting for effect modification by temperature. In addition, to our knowledge, no study has estimated the impact of air conditioning (AC) prevalence on ozone-related mortality risk at different temperature levels. Here, we build on existing literature and estimate ozone mortality risks by treating temperature as a confounder as well as an effect modifier by estimating risks at low, moderate, and high temperature categories.

2. Methods

2.1. Data collection

We obtained daily data on non-accidental mortality, air pollution, and weather condition for 97 communities in the continental US in 1987–2000 from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) database (Fig. 1) (iHAPPS, 2007; Samet et al., 2000). A detailed description of this extensively employed database can be found elsewhere (Samet et al., 2000). The daily mortality counts represented the number of cause-specific (non-accidental) deaths for individuals of ages 0–99 (excluding non-residents) at a county level. Consistent with previous studies that utilized this database, we used the 10% trimmed mean of ozone concentrations to mitigate the influence of very high or very low ozone concentrations (Bell et al., 2004; Ren et al., 2009). From the NMMAPS database, we obtained data on daily 24-h ozone concentrations during the warm months (May to September), 24-h temperature, and 24-h dew point temperature. We also obtained data on prevalence of AC use during our study period for 72 of the 97 cities from the Census Bureau's American Housing Survey.

2.2. Statistical methods

We utilized a two-stage statistical approach to estimate national estimates of ozone-related mortality risks. In the first stage, we performed a time-series analysis using a Poisson regression model to estimate community-specific mortality risk from exposure to same-day ozone (R Development Core Team, 2011). The time series of daily mortality counts were modeled against ozone and other covariates using a generalized linear regression model with a quasi-Poisson link function to account for overdispersion. The model controlled for long-term patterns and seasonality using natural splines with 3 degrees of freedom (*df*) for each warm season (May to September). We included indicator variables to allow mortality rates to vary by day of the week. We also controlled for potential confounding due to weather conditions by including smoothing functions of 24-h dew point and 24-h temperature in the baseline model, which does not account for interaction between ozone and temperature (see Supplemental information for additional details on model construction). This model can be represented as:

$$\log[E(Y_t)] = \alpha + \beta_1 \text{ozone}_t + ns(\text{temp}_t, df = 4) + ns(\text{dptemp}_t, df = 4) + ns(\text{season}_t,\ df = 3 \text{ per season}) + \gamma \text{dow}_t + \varepsilon_t \quad (1)$$

where subscript t denotes the time of the observation and β_1 is the main effect of ozone; $E(Y_t)$ is the expected value of the daily count of deaths (Y_t); $ns()$ is the natural smoothing spline function; α is the intercept. Ozone_t, temp_t, and dptemp_t are daily 24-h ozone, temperature, and dew point temperature, respectively. Season_t is based on the day of the calendar year and controls for seasonal and long-term variation of mortality. Dow_t is the day of week at time t and γ is a vector of coefficients; ε_t is the residual.

To assess effect modification, we designated daily observations into low, moderate, and high temperature levels by applying the 25th and 75th percentiles of daily 24-h temperature in May–September of 1987–2000 within each community as cutoff values (see Appendix A). This relative, city-specific temperature categorization method was utilized to characterize regional differences in the ability of populations to adapt to temperature changes. Using this categorical temperature term, we estimated ozone risk with

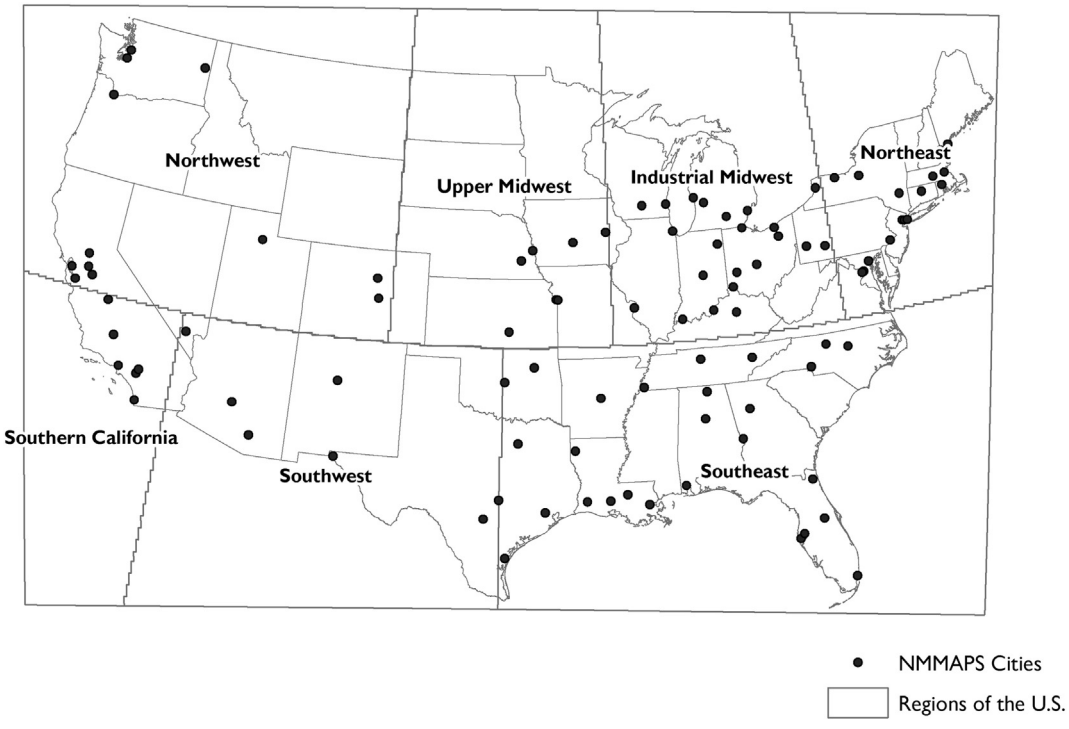

Fig. 1. Location of 97 NMMAPS cities.