Impact of heat on all-cause and cause-specific mortality: A multi-city study in Texas

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Abstract

Studies on the health effects of heat are particularly limited in Texas, the U.S. state with the top ten highest number of annual heat-related deaths per capita from 2018-2020. This study aims to assess the effects of heat on all-cause and cause-specific mortality in 12 metropolitan statistical areas (MSAs) across Texas from 1990-2011. First, we determined the heat thresholds for each MSA above which the relation between temperature and mortality is linear. We then conducted a distributed lag non-linear model for each MSA, followed by a random effects meta-analysis to estimate the pooled effects for all MSAs. We repeated this process for each mortality cause and age group to achieve the effect estimates. We found a 1°C temperature increase above the heat threshold is associated with an increase in the relative risk of all-cause mortality of 0.60% (95%CI [0.39%, 0.82%]) and 1.10% (95%CI [0.65%, 1.56%]) for adults older than 75. For each MSA, the relative risk of mortality for a 1°C temperature increase above the heat threshold ranges from 0.10% (95%CI [0.09%, 0.10%]) to 1.29% (95%CI [1.26%, 1.32%]). Moreover, high temperature had a negative but not statistically significant effect on cardiovascular mortality (-0.37%, 95%CI [-0.35%, 1.09%]) and respiratory disease (-1.97%, 95%CI [-0.11%, 4.08%]). Our study found that high temperatures can significantly impact all-cause mortality in Texas, and effect estimates differ by MSA, age group, and cause of death. Our findings generate critical information on the impact of heat on mortality in Texas, providing insights for policymakers on resource allocation and strategic intervention to reduce heat-related health effects.

Keywords: Urban Climate; High Temperature; Mortality; Distributed Lag Model

1. Introduction

Climate change has increased the intensity, frequency, and duration of extreme heat events (i.e., heat waves) (EPA, 2022), and studies have shown the adverse effect of exposure to high temperatures on mortality and morbidity across geographic locations and population groups (Anderson & Bell, 2009; Anderson & Bell, 2011; Chien et al., 2016; Haines et al., 2006; Martiello & Giacchi, 2010; O'Neill et al., 2005). From 2004-2018, there were 702 heat-related deaths on average each year in the U.S. (Vaidyanathan et al., 2020). The City of Houston, Texas, recorded a substantial increase in emergency department visits, especially among older adults during a heat wave in 2011 (Zhang et al., 2015). Heat exposure can result in cardiovascular disease, respiratory disease, and death (Michelozzi et al., 2009). The World Health Organization has warned that the overall health impacts of climate change are serious and urgent, especially for regions and population groups lacking adequate health service systems and cooling measures (World Health Organization, 2017).

Texas has more than 60 extreme heat days a year on average, which is projected to double by 2050 (States at Risk, 2022; Vaidyanathan et al., 2020). The number of annual heat-related deaths per capita ranks in the top 10 highest among all states from 2018-2020 (ValuePenguin, 2022). Previous studies on the association between heat and mortality in Texas focused on a single city or population group (Chien et al., 2016; Mallen et al., 2019; Marsha et al., 2018; Zhang et al., 2015; Zottarelli et al., 2021). For example, Zhang et al. (2015) and Marsha et al. (2018) focused on the effect of heat on emergency department visits and mortality in Houston. Mallen et al. (2019) and Zottarelli et al. (2021) concentrated on vulnerable groups during extreme heat in Dallas and San Antonio. Chen et al. (2017) used county-level data in Texas to determine the effect of ambient temperature on mortality yet only concentrated on cold weather and older adults. No studies have analyzed the overall mortality risk caused by heat across all major metropolitan statistical areas (MSAs) in Texas.

The impact of heat on health differs by individual sociodemographic characteristics, spatial location, and mortality causes. Gronlund (2014), Xu et al. (2014), and Basagaña et al. (2011) have shown that age, income, and education impact the effect of heat on health. Zhang et al. (2015) found older adults to be more susceptible than younger adults to extreme temperatures. Further, Chien et al. (2016) found the effect of heat waves on mortality of older adults to be most pronounced in Northwestern Texas and partially Western Texas. Anderson and Bell (2009) revealed that cardiorespiratory mortality risk increased by 8.8% during heat wave days compared with non-heat wave days in 107 U.S. communities. Cheng et al. (2019) reviewed the mechanism of cardiorespiratory risk caused by heat exposure. Zhang et al.(2018) projected the number of deaths caused by cardiovascular diseases resulting from heat exposure under varying climate change, population, and adaptation scenarios. Basagaña et al. (2011) estimated the effect of heat on 66 mortality causes, finding that the risk of mortality increases for individuals with pre-existing illness (e.g., cardiovascular and respiratory diseases).

O'Neill et al. (2005) suggested that race was associated with the magnitude of the effect of heat on mortality, which may be related to differences in air conditioning prevalence across cities. Texas has a diverse racial composition, with about 40% of residents identifying as Hispanic and about 12% as Black (Texas Demographic Center, 2020). In Texas, Black and Hispanic adults are more likely to report being not in good health compared with White adults (Turner et al., 2021). No studies have focused on Texas when estimating the impact of heat on mortality by race and ethnicity.

Herein, we provided a comprehensive analysis of the effects of heat on mortality for all major MSAs in Texas using a consistent statistical methodology. To our knowledge, our study covers the longest time period and the widest geographic area in Texas compared to previous research. We conducted a heat-mortality analysis for each MSA separately and then performed a meta-analysis. Our analysis culminated in an assessment of the association between heat and mortality by age and mortality causes. We also discuss the impact of heat on mortality by race and ethnicity. Our findings on the overall effect of heat on mortality in Texas can be referenced by state regulators when developing policies to mitigate heat-related health effects. Further, our quantification of the heat-mortality relationship for each major MSA in Texas can serve as input for future projections of health risks due to climate change.

2. Data and Methods

2.1 Study Setting

This study focused on the summer season (May 1st to September 30th) in Texas, a state located in the South-Central region of the United States that is the second largest state in the country by both geographic area and population. Of the 25 total MSAs in Texas, we selected 12 MSAs for analysis based on data consistency in population, mortality, weather, and air pollution information from 1990-2011: Austin-Round Rock, Beaumont-Port Arthur, Brownsville-Harlingen, Corpus Christi, Dallas-Plano-Irving, El Paso, Houston-The Woodlands-Sugar Land, Killeen-Temple, Lubbock, McAllen-Edinburg-Mission, San Antonio-New Braunfels, and Waco. During the study period, the population size of the selected MSAs remained consistently above 200,000. Figure 1 showed the map of 12 selected Texas MSA and their population sizes.

2.2 Mortality and Population Data

We obtained all-cause mortality data for residents living in 12 MSAs in Texas from the Texas Department of State Health Services. We aggregated the raw data by daily and MSA levels from 1990-2011. To aggregate the data, we first classified the mortality data into different age groups (0-65, 65-75, 75+) determined by the observation counts in the data. We then classified the data into two groups of different mortality causes based on the International Classification of Disease Ninth Revision (ICD-9) and Tenth Revision (ICD-10) (World Health Organization, 1975, 1992). The first group was cardiovascular diseases (CVD, ICD-9 390-429; ICD-10 I01-I52), which included ischemic heart disease (IHD, ICD-9 410-414; ICD-10 I20-I52), myocardial infarction disease (MI, ICD-9410; ICD-10 I21, I22), and stroke (ICD-9 430-438; ICD-10 I60-I69). The second group was respiratory diseases (RESP, ICD-9 460-519; ICD-10 J00-J99), which included chronic obstructive pulmonary disease (COPD, ICD-9 490-496 except 493; ICD-10 J40-J44, J47) and pneumonia (PNEU, ICD-9 480-486; ICD-10 J12-J18). The dataset for the population size in each MSA was from the Texas A&M University Texas Real Estate Research Center (2022).

2.3 Weather and Air Pollution Data

Hourly weather data originated from the Integrated Surface Database provided by the National Climate Data Center. The original dataset contained hourly temperature and dew point temperature data from multiple monitoring stations. To aggregate the dataset to the MSA- and day-level, we used the weather records from one representative monitoring station in the most populated area within each MSA following the method in Zanobetti et al. (2012). Next, we aggregated the hourly data into daily mean, minimum, and maximum temperature, as well as dew point temperature. In our estimation, we used daily mean temperature to represent the daily temperature exposure (Chen et al., 2017).

We included ground-level ozone in our analysis since it directly correlates with temperature (Coates et al., 2016) and ozone exposure is significantly associated with health outcomes (Reid et al., 2012). We retrieved ozone information from the Texas Air Monitoring Information System Database (Texas Commission on Environmental Quality). We aggregated the ozone data from the hour- to day-level for each MSA by taking the daily average results from all monitors within each MSA.

2.4. Statistical Analysis

We combined mortality, population, weather, and air pollution data to conduct a multi-city time series analysis consisting of two stages, following Chen et al. (2017). First, we conducted an MSA-specific estimate of the effect of heat on mortality using Poisson regression, which allows for overdispersion in the model. Second, we performed a meta-analysis on the effect of heat on mortality by pooling the estimated results from the Poisson regression. We repeated this two-stage process to estimate the effect for each age group and mortality cause.

2.4.1 MSA-Specific Models

The first stage—construction of MSA-specific models—involved two steps. First, we determined the presence of thresholds of heat (i.e., when temperature reaches a certain value, a linear relationship exists between temperature and mortality). We plotted the relationship between temperature and mortality counts using generalized additive modeling (GAM) with a spline function of temperature for each MSA. The dependent variable in the GAM was lag 0-6 (i.e., average temperature of the previous six days) to account for the delayed effect of heat. We included days of the week and year to account for time trends and seasonality. We also adjusted models for mean dew point temperature and population size, per previous studies (Lee et al., 2016; Marsha et al., 2018). We controlled dew point temperature for the purpose of controlling relative humidity because the relative humidity level can be calculated given dew point temperature and temperature. In our estimation, most results from MSA-specific analyses indicated a U-shaped or J-shaped relationship between temperature and mortality. Because of the nonlinear relationship between temperature and mortality and the purpose for the use in the future projection of heat-related risk, we need to determine an optimal threshold temperature above that there is an approximate linear association. Given the possible range of thresholds of heat, we determined the exact threshold used in the model by minimizing the Akaike information criterion for regressions using quasi-Poisson distribution.

For the second step in constructing MSA-specific models, we used single threshold distributed lag non-linear models to quantify the effect of heat on mortality. We estimated a Poisson regression model for each MSA:

$$\log[E(Y_t)] = \alpha + cb\left(meTMP_{t,l}\right) + \delta\ DOW_t + s(DOY_t, 4) + s(meDWP_t, 3) + \lambda \ln\left(Population_y\right) \qquad (1)$$

Y_t is the number of deaths on day t. $meTMP$ refers to daily mean temperature and is our primary exposure variable. We modeled meTMP from two dimensions: exposure-response and lag. $cb(meTMP_{t,l})$ is a cross-basis function of $meTMP_{t,l}$, which is the mean temperature on lag day l of day t; l represents natural spline function with maximum six lag days and two degrees of freedom; DOW_t is a factor variable representing the day of the week for day t; $s(DOY_t, 4)$ represents a smooth function (natural cubic spline) of DOY_t , which represents which day of the year for day t with four degrees of freedom; $meDWP_t$ represents mean dew point temperature with three degrees of freedom; $\ln(Population_t)$ represents the natural logarithm of the population size in year y; α, δ, λ are the estimated parameter in the regression.

Additionally, we conducted the same procedure for each age group and mortality cause. The estimated results can be explained as the change in relative risk of mortality with a 1°C increase in temperature above the threshold of heat.

2.4.2 Meta-Analysis

We pooled estimation results from the first stage through multivariate meta-analysis using random effect modeling in the second stage. Following Chen et al. (2017), we assumed that the estimated effect of heat on mortality followed a normal distribution with the real effect as the mean. The variance of distribution was the summation of the within-MSA variance of estimated effect and between-MSA variance of true effect. We estimated the between-MSA variance of true effect using restricted maximum likelihood. Each MSA included in the meta-analysis was assumed to be randomly selected. We also conducted the same two-stage modeling for the effect estimate for each mortality cause and age group.

2.4.3 Sensitivity Analysis

We conducted a sensitivity analysis to check how the effect of heat on mortality differed based on different ranges of lag days. We first changed the lag range for temperature control to lag 0-1, lag 0-2 and lag 0-10. We then repeated the analysis with the inclusion of ozone pollution in the model.

3. Results

Across the 12 major MSAs in Texas during warm seasons from 1990-2011 (Table 1), the daily mean temperature and average daily mean temperature ranged from 6-37℃ and from 25-30℃, respectively. Average daily mean temperature was highest in McAllen-Edinburg-Mission and lowest in Lubbock. Dallas-Plano-Irving was the most populous MSA and had 58 deaths each day on average, the second largest rate of all study MSAs. Waco was the least populous and had six deaths each day on average. Houston-The Woodlands-Sugar Land had 74 deaths each day on average, the largest rate among all MSAs, and Brownsville-Harlingen had the least daily deaths on average. Heat thresholds in each MSA ranged from 24-32℃.

The pooled estimation result for the effect of heat on all-cause mortality equaled 0.60% and was statistically significant for all MSAs (Figure 2). Pooled estimation results for each MSA were statistically significant, with Brownsville-Harlingen having the largest effect of heat on mortality (1.29%, 95%CI [1.26, 1.32]) and Houston-The Woodlands-Sugar Land having the smallest effect (0.10%, 95%CI [0.09, 0.10]).

For age-specific pooled estimation of the effect of heat on mortality (Figure 3), heat tended to have a more significant impact on older adults. For adults older than 75 years, 1℃ of temperature increase above the heat threshold was associated with a 1.10% (95%CI [0.65, 1.56]) increase in relative risk of mortality, on average. As for the cause-specific estimation, we tested two major systems of diseases: cardiovascular diseases (0.37%) and respiratory diseases (1.97%). The effect of heat is the highest on the mortality risk due to COPD, but this result was not statistically significant.

In the sensitivity analysis for ranges of lag days and ozone pollution in our models (Figure 4), extending the lag length in the model resulted in an increase in the estimated effect of heat on mortality. The highest estimated effect (0.82%, 95%CI [0.43, 1.2]) occurred when using lag 0-10. After controlling for ozone, the highest estimated effect (0.73%, 95%CI [-1.15, 2.65]) occurred with the model using lag 0-6 but was insignificant.

4. Discussion

Previous investigations of the impact of heat on human health were not focused on warm climate regions but rather on cold climates or at the national level (Anderson & Bell, 2009; Anderson & Bell, 2011; Basu & Ostro, 2008; Gronlund, 2014; O'Neill & Ebi, 2009; Ye et al., 2012). The limited studies in Texas concentrated on one city or a specific population (Chen et al., 2017; Chien et al., 2016; Zhang et al., 2015). To our knowledge, this is the first study that systematically explored the effect of heat on all-cause mortality across all major MSAs in Texas, which provides critical information for decision-makers that can support preparation for projected increases in temperature with climate change.

The pooled estimate for all MSAs was 0.60% (95%CI [0.39, 0.82]), which suggested that a 1°C temperature increase above the heat threshold increases the relative mortality risk by 0.60% on average. Anderson and Bell (2009) conducted a time series study on associations between weather and mortality using data from 43 U.S. cities from 1987-2005. Their estimate of the average effect of heat in Dallas was 1.0%, which is slightly higher than our result of 0.74% (95%CI [0.74, 0.75]). The slight difference between our results and earlier work may be related to the different study periods: advances in technology and knowledge over time may better equip society for the health threat of heat.

The estimated effect of heat on mortality in our study was lower compared to previous studies in northern cities in the U.S. For example, Anderson and Bell (2009) found that the effect of heat on mortality was about 4% in New York City and 3% in Chicago. Gasparrini et al. (2012) showed the relative risk of increasing temperature on mortality was 2.1% (95% CI [1.6, 2.6]) in England and Wales. This difference could be potentially attributed to the high prevalence of air conditioning use in Texas (O'Neill et al., 2005; Zhang et al., 2015), which could mitigate the impact of heat on human health (Davis et al., 2003; Rogot et al., 1992). According to the Residential Energy Consumption Survey, about 96% of households in Texas use air conditioning (U.S. Energy Information Administration, 2009), while northern states have a much lower usage. For example, about 76% of residents in Michigan use air conditioning. Zhang et al. (2015) found that a heat wave in 2011 had no impact on mortality in Houston because of widespread air conditioning use. In estimating the correlation between air temperature and heat-related deaths in 28 MSAs in the U.S., Davis et al. (2003) found a decline in heat-related mortality rates from 1964-1998, which they associated with an increased prevalence of air conditioning use in households.

We found that a 1℃ temperature increase above the heat threshold increases the relative mortality risk for adults older than 75 by 1.10% (95%CI [0.65, 1.56] on average. This finding is consistent with Chien et al. (2016) that found the relative mortality risk of older adults caused by heat waves was 1.0% in Texas. Generally, older adults are more susceptible to heat than younger individuals (Centers for Disease Control and Prevention, 2017). Kenney et al. (2014) explained that it was because they have a higher risk of excess central cardiovascular strain, impaired thermoregulation, and lower evaporative heat loss due to attenuated blood flow and cardiac output.

Our finding of an insignificant effect of 1°C temperature increase above the heat threshold on most causes of mortality may be explained by the small number of non-zero observations after dividing total mortality into specific causes, ultimately diminishing the variation for each estimated parameter. Although the findings were not statistically significant, the magnitude of estimation results corroborates previous findings. Our results suggested that the effects of heat on mortality risk caused by respiratory diseases (i.e., chronic obstructive pulmonary disease and pneumonia) tended to be higher than those caused by cardiovascular diseases (i.e., ischemic heart disease, myocardial infarction disease, and stroke). Basagaña et al. (2011) investigated 66 mortality causes and found that respiratory diseases were one of the most pronounced heat-related mortality causes (1.21%) on extreme heat days. Anderson and Bell (2009) found that cardiorespiratory mortality risk increased by 8.8% on heat wave days compared to non-heat wave days in 107 U.S. communities. In 50 U.S. cities from 1989-2000, Medina-Ramón et al. (2006) found that extreme heat increased the mortality risk of pneumonia by 1.00% and stroke by 1.03%.

Our estimations from sensitivity analysis suggested that the effect of heat on mortality became less significant when using a shorter lag range for temperature, which is reasonable since the acute effect of heat on mortality is generally trivial with a short lag period (Anderson & Bell, 2009). In our models that adjusted for ozone, results were insignificant because of the direct association between temperature and ground-level ozone (Bloomer et al., 2009), the latter of which is especially harmful to the human respiratory system (Norval et al., 2011). The observation of ozone for some days are missing, which shrink the sample size for estimation when adding in ozone. Therefore, the confidence interval of the estimation results without ozone is much smaller than the results with ozone added in the model.

Our findings contribute to research on health disparities. MSAs wherein more than 50% of individuals identify as Latino or Black (e.g., Brownsville-Harlingen, Corpus Christi, and San Antonio-New Braunfels) tended to have a higher risk of heat-related mortality compared to MSAs with a smaller percentage of population identifying as Latino or Black (e.g., Austin-Round Rock, Waco, and Killeen-Temple), on average. In addition, MSAs with lower levels of median household income (e.g., Corpus Christi and Beaumont-Port Arthur) had higher heat-related mortality risk than higher-income MSAs (e.g., Dallas-Plano-Irving and Houston-The Woodlands-Sugar Land). This is consistent with findings from O'Neill et al. (2005) in which Black and Latino adults had higher heat-related mortality risk, which correlated to disparities in air conditioning use. Air conditioning has been shown to be the most important factor affecting indoor temperatures (Larsen et al., 2022). Our results illustrate that communities of color and low-income communities have a higher likelihood of being exposed to heat, which adds to the literature that has shown individuals identifying as low-income, Latino, and Black are more likely to live in areas with less tree canopy—the likes of which lower temperatures—than their higher-income and White counterparts (Lanza et al., 2019).

This study has some noteworthy limitations. First, our modeled estimates did not account for human behavior to mitigate heat exposure, such as using air conditioning and staying indoors during extreme heat days (O'Neill et al., 2005). Second, we measured temperature at fixed sites in each MSA, which can misclassify the real extent of heat exposure (Kuras et al., 2017). Air temperatures can vary within an MSA, with temperatures often higher in downtown areas than surrounding areas due to high amounts of energy-absorbing building materials, waste heat emissions, urban form, and a lack of trees (Stone Jr et al., 2019). For example, at the microscale, Lanza et al. (2021) has measured daily air temperatures to differ by 4°C, on average, between two sites (i.e., unshaded playground and playground under tree shade) in Central Texas less than 50m apart. Future analyses using higher resolution urban microclimate and community-level sociodemographic data can reduce potential misclassification of heat exposure and evaluate the modifying effects of community-level factors on heat-mortality associations.

5. Conclusion

Our study provided evidence that high temperatures can significantly impact all-cause mortality in Texas during the warm season, with effects differing by MSA, age group, and cause of death. Findings contribute to our understanding of the impact of heat on human health in warm climates, and can serve as evidence for policymakers to inform resource allocation and strategic intervention to safeguard the public—especially older adults and individuals who are Black, Latino, or low-income—from the adverse impact of heat, a hazard set to be more commonplace in our changing climate.

Tables and Figures

Table 1. Summary of daily mean temperature, daily count of all-cause mortality, population size, and heat threshold in 12 major metropolitan statistical areas (MSAs) in Texas, 1990-2011.

Texas MSA	Daily Mean Temperature (◦C) [1]	Daily Count of All-Cause Mortality	Population Size [2]	Heat Threshold
	Mean (Min, Max)	Mean (Min, Max)	(#)	(◦C)
Austin-Round Rock	27 (11, 34)	17 (2, 40)	1,716,289	25
Beaumont-Port Arthur	27 (16, 33)	11 (1, 23)	388,745	25
Brownsville-Harlingen	29 (17, 33)	5 (0, 15)	406,220	28
Corpus Christi	28 (16, 33)	8 (0, 20)	405,027	29
Dallas-Plano-Irving	27 (9, 37)	58 (30, 95)	6,366,542	26
El Paso	27 (12, 36)	10 (0, 23)	804,123	24
Houston-The Woodlands-Sugar Land	28 (16, 35)	74 (40, 129)	5,920,416	30
Killeen-Temple	28 (12 ,37)	5 (0, 17)	405,300	30
Lubbock	25 (6, 36)	5 (0, 14)	290,805	30
McAllen-Edinburg-Mission	30 (12, 35)	7 (0, 19)	774,769	32
San Antonio-New Braunfels	28 (13, 36)	32 (7, 56)	2,142,508	26
Waco	27 (11, 36)	6 (0, 15)	252,772	30

[1] Average daily mean temperature throughout the study period
[2] Based on 2010 Census data

Figure Legends

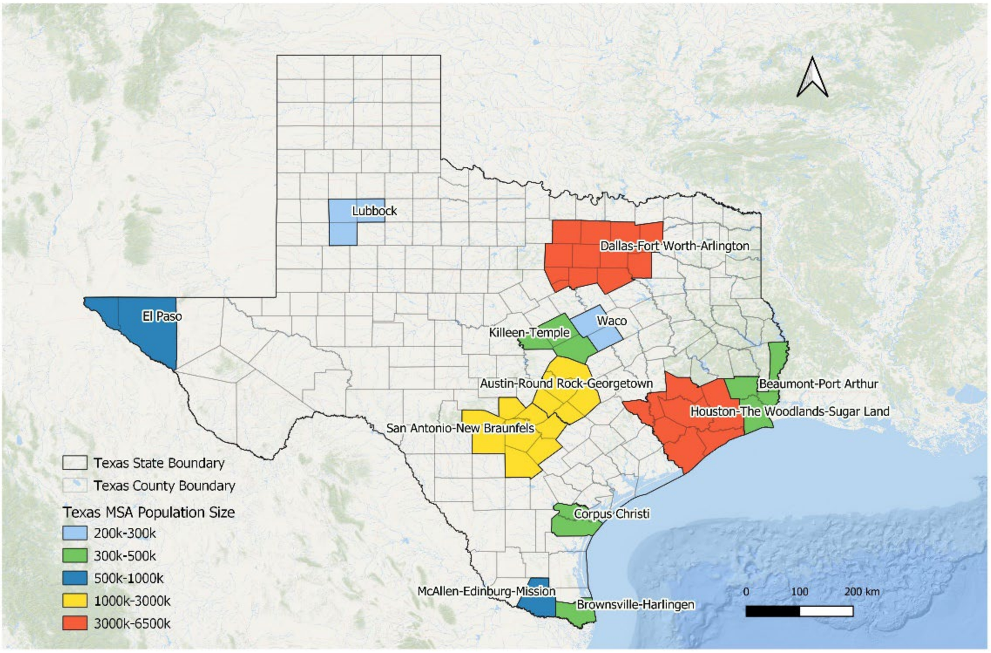

Figure 1. Map of 12 selected Texas Metropolitan Statistical Areas (MSAs). The selection of these MSAs was based on their population size and the availability of weather and air pollution data during the 10-year study period.

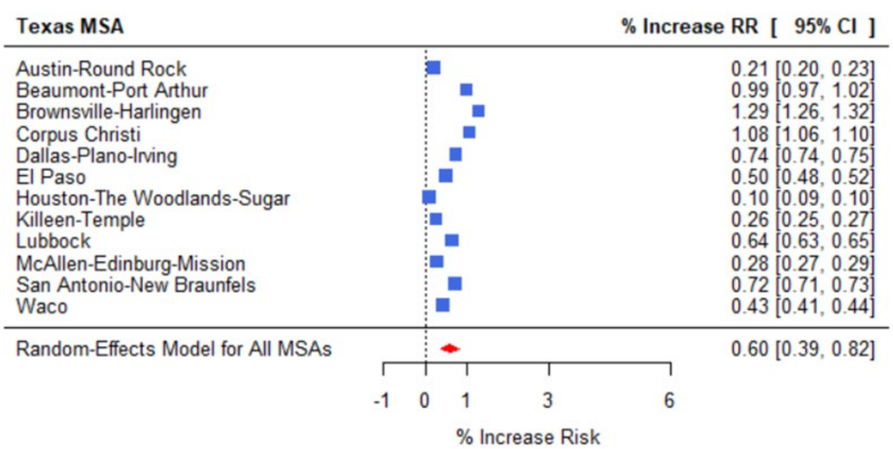

Figure 2. Meta-Analysis for the effect of heat on all-cause mortality at lag 0-6 days in 12 major metropolitan statistical areas in Texas, 1990-2011.

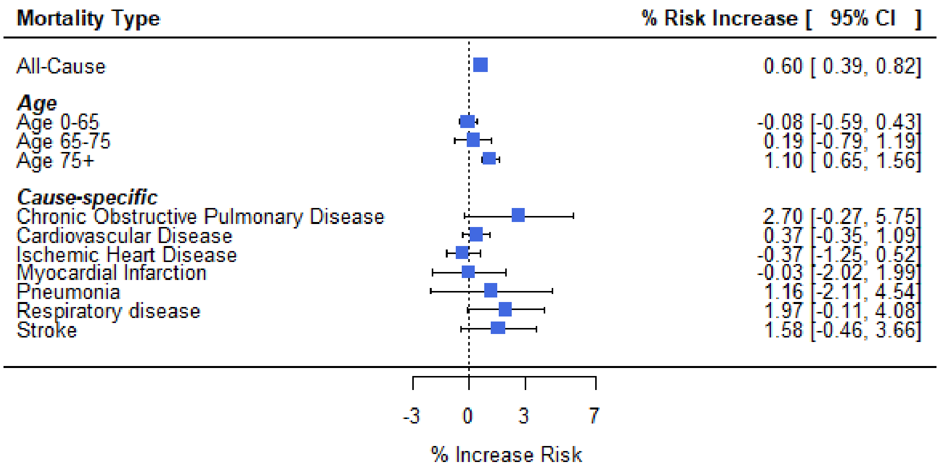

Figure 3. Pooled estimations for the effect of heat on health by age and cause-specific mortality at the metropolitan statistical area-level in Texas, 1990-2011.

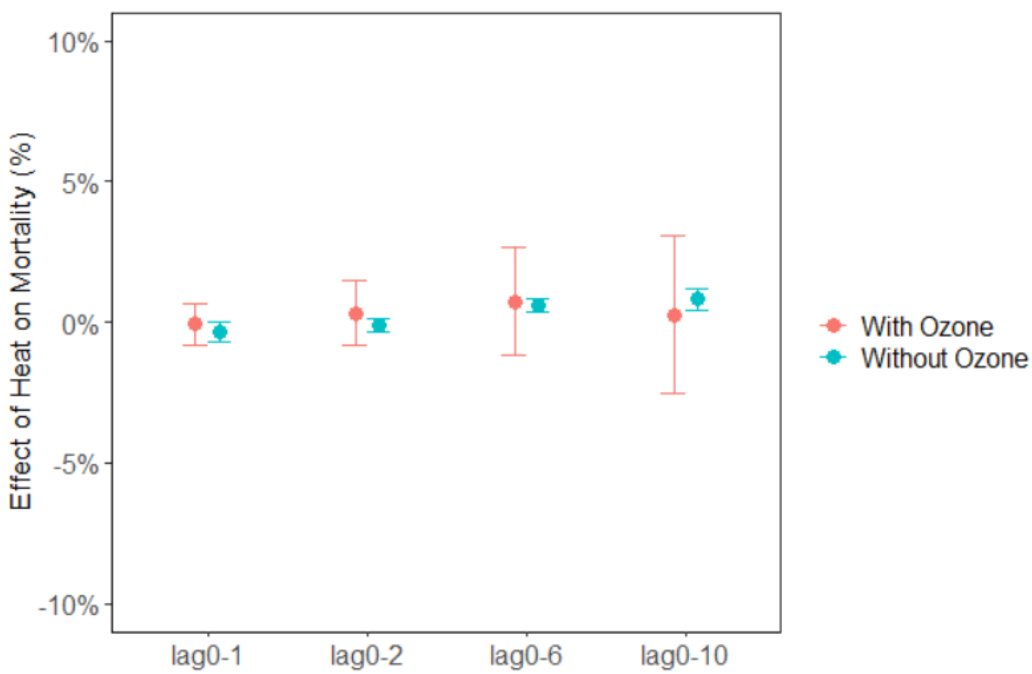

Figure 4. Sensitivity analysis based on changing lag range and adding ground-level ozone to the estimation model.

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