Extreme Heat, Vulnerable Populations and Adverse Health Outcomes: Informing Targeted Climate Change Adaptation Planning

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Dedication

I dedicate this work to my amazingly supportive and loving parents, Paul and Mary, who have always encouraged me to think beyond myself. By leading through example, they taught me that perseverance, compassion, and inquisition can lead to you to the most rewarding of places.

Thank you for reminding me, often times daily, that the benefits of my research will outlast any living legacy

And, most importantly, that

Today is your day! Your mountain is waiting So ... Get on your way!

- Dr. Seuss

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Chapter 1.

Introduction

1.1. Background and motivation

The Intergovernmental Panel on Climate Change (IPCC) concludes that global regional increases in hot extremes and heat waves are 'very likely' [1] and are anticipated to result in the increase in the intensity, frequency and duration of heat waves [1]. Recent climatological studies that incorporate numerous climate models have shown that heat events which, on average, now occur only once every 20 years will, by the end of the century, occur about every other year across much of the United States (U.S.)[2] These changes will come with significant implications to human health and require swift responses to prepare and respond to the changing climate.

1.2. Heat, human health, and heat-related vulnerability

In the U.S., the largest proportion of weather-related deaths is attributed to heat [3, 4]. Research has shown that short-term increases in mortality occur during periods of high heat [5, 6], and several biological explanations for these increases exist, predominately related to the concept of thermoregulation, the ability to manage one's individual temperature. When an individual is exposed to extreme heat, the body responds by reallocating blood flow from the vital organs in the central portion of the body to below the skin's surface, believed to be a mechanism to cool the overall system. When a person is unable to thermoregulate effectively, due to aging, medication use, chronic health conditions such as diabetes, or other impairments, the body reallocates too much blood from the vital organs, which results in increased stress on the heart and lungs [7] which can contribute to fatal health events.

The association between heat and mortality has been well researched, indicating that vulnerable populations, such as young children and the elderly, are at particular risk of hospitalization or death during heat events. The most notable

events where population health was affected by short term increases in temperature include the 1995 Chicago heat wave, where nearly 500 excess deaths were attributed to extreme heat [8], and the 2003 European heat wave that was responsible for, conservatively estimated, 35,000 excess deaths [9]. Extensive epidemiologic investigation of these particular heat events have provided insight into determining individual and community-level characteristics that are associated with risk of death when exposed to extreme heat.

The term 'vulnerability' is used in public health to reflect an individual's or population's adverse response to an environmental hazard. Similarly, the US National Research Council (NRC) defines vulnerability as it pertains to climate change as "the capacity to be harmed" via the magnitude of changes, underlying factors that contribute to sensitivity, and the ability to avoid, prepare for, and response to impacts at various levels [10]. As epidemiologic research suggests that heat-related mortality is dependent on the severity of the heat event and the health status of the affected population at [11-16], the general increasing trends of disease morbidity in the US population suggests that even more individuals may be considered specifically at risk when exposed to extreme heat. A recent report from the Center for Medicare and Medicaid Services, which is the administration responsible for health care for nearly all US residents over the age of 65, stated that the prevalence of multiple chronic conditions, including cardiovascular disease, among individuals over the age of 65 is steadily increasing [17]. Trends of increasing disease prevalence do not bode well for the future when considering evidence that the associations of morbidity and adverse heat-related outcomes, including death, are elevated among those suffering chronic health conditions.

The epidemiologic literature identifies both individual and community-level characteristics that increase one's vulnerability to heat. People's ability to respond to exposure to heat can be inhibited, depending on their health status. Pre-existing health conditions such as cardiovascular-, respiratory-, renal-, neurological-diseases, as well as diabetes and mental health conditions contribute to heat vulnerability [15, 18-20]. Individuals 65 and older are the most

vulnerable to heat, possibly as a result of their reduced ability to thermoregulate [21] and an increased likelihood of impaired renal failure [22]. Additionally, as individuals age, the risk of having chronic diseases increases, along with the likelihood that medication, such as anticholinergics, will inhibit the body's ability to initiate a thermoregulatory response [23]. Socioeconomic and demographic factors such as older age [24, 25], minorities [8, 24, 26], low-income, high school educated or less [19, 24, 27-29], being unmarried [20, 21], and social factors such as living alone, having access to transportation [18, 30] have been associated with increased risk of death during extreme heat events. Further, housing characteristic like central air conditioning [28] are protective characteristics for individuals exposed to extreme heat or heatwaves [19]. Recently, the role of area level green space has garnered attention in assessing the heat-health relationship under the hypothesis that increased green space is protective from extreme heat as it reduces ambient air temperatures [31]. Green space characterizations range from measurements of area level vegetation [32] to calculation of percent impervious surface [33] to having access to green space [34].

Urban Heat Island

Local climate is directly impacted by the physical structure and land cover composition of a city [35] and is often characterized by the concept of the urban heat island (UHI). Higher observed ambient temperatures in urban areas compared to the surrounding suburban and rural areas define the UHI effect [36]. Much of a cityscape is comprised of impervious surfaces such as concrete, brick, asphalt, which retain ambient heat, and thus contribute to the increase in the air temperature. Additionally, increased air temperatures result from heat that is emitted from buildings and vehicles, increased overall consumption of energy per capita, and lack of green space or vegetation [37, 38]. There is a much larger risk of annual extreme heat events occurring in the sprawling cities compared to most compact metropolitan areas [39]

Although numerous individual and community-level characteristics could be used to evaluate a person's vulnerability to heat, as presented in the literature

review above, populations who are most consistently identified as being at risk to adverse health events when exposed to extreme heat are the elderly, people with lower socioeconomic status, and urban populations. These characteristics can be useful markers of vulnerability, but are still fairly general, making it difficult to develop targeted intervention and response plans for extreme heat exposure.

Much of what is known about heat-related morbidity and mortality stems from retrospective analyses in which the data were extracted from death certificates, archived temperature data, census, and other broad population-based surveys (e.g., Behavioral Risk Factor Surveillance System, American Housing Survey). Analyses using these data sources must often limit the spatial and temporal resolutions to the coarsest spatial scale across variables, resulting in estimates reflecting the impacts of heat on populations of thousands to millions (e.g., county-level). Risk estimates of heat-related mortality should reflect temperature variability within cities and as well as consider individual-level factors that contribute to vulnerability.

Ultimately, the challenge in determining the populations who will most likely fare worse in the face of extreme heat is rooted in the complex interactions between human health, behavior and the built environment. The current state of research does not establish clear links or provide specific intervention points, but there is room to begin the process of identifying where and how efforts to reduce vulnerability can be most effective.

1.3. Adaptation

Adaptation to the changing climate will be necessary because, due to the substantial emissions of climate change-accelerating greenhouse gases in previous years, the increasing global temperatures will continue to climb even under the most protective of climate change mitigation scenarios [40]. The US National Climate Assessment (NCA) is developing suggested strategies and approaches to for local governments to plan for future heat events or increase adaptation to heat events. The NRC's *America's Climate Choices: Adapting to the*

Impacts of Climate Change (2010) defines adaptation as the 'adjustment in natural and human systems to a new or changing environment that exploits beneficial opportunities or moderates negative effects" [10]. As regions, states and municipalities are faced with responding to and preparing for the impacts of a changing climate on human health, research that can contribute to decision-making policy and development for climate change adaptation at a scale relevant to specific populations and communities is needed. Some public health literature discusses or focuses on the theoretical [41, 42] and analytical [43] implications of a changing climate on human health. Although such investigations have contributed to the overall discussion on the impacts of climate change on human health, there is a move towards utilizing public health research as a way to inform lasting adaptation measures.

One way to characterize climate change adaptation is to describe it as either short or long term. Short-term measures of heat adaptation could include the implementation of cooling centers, extreme heat warnings, water distribution stations or education programs [44]. While such interventions and programs can reduce the impacts of extreme heat in our most vulnerable populations, they provide temporary protection. Longer-term adaptation strategies, which would provide lasting protection and potentially numerous co-benefits [45] would require more extensive interventions, such as tree planting [46], removing impervious surface [47], installing green or white roofs [48], and residential weatherization efforts such as increased wall and window insulation, and weather stripping [49].

Where there is vulnerability to a specific exposure such as extreme heat, there is an opportunity to develop and implement methods and interventions that are specifically designed to reduce the vulnerability. Public health and adaptation research can inform each other in response to what is expected to be a continually changing climate.

1.4. Research objective and hypothesis

There is an urgent need to refine and evaluate methodologies for accurately identifying populations most vulnerable to heat, as 2012 was the hottest year on record worldwide (http://www.nasa.gov/topics/earth/features/2012-temps.html) and there is scientific consensus that global temperatures will continue to increase [50]. Such methodologies will be critical in the development and implementation of climate adaptation efforts. The objectives of this research are to address gaps in the methodologies and applications commonly used to evaluate the impacts of climate change-related exposures- using excessive heat events as a case example — on populations believed to be most likely to experience morbidity or mortality. Within this broad topic, this dissertation addresses three aims:

- The development an innovative methodology that 'downscales' epidemiologic effect estimates of the relationship between extreme heat and mortality at the census tract level;
- 2). Examination of whether the creation of a heat vulnerability index, comprised of fine-scale demographic, environmental, health and socoioeconomic variables, provides an explicit and consistent pattern of heat-related vulnerability;
- 3). Evaluation of the ability of a fine-scale heat vulnerability index to reliably predict the association of extreme heat and mortality.

These aims are investigated by employing epidemiologic methodologies, statistical modeling and analyses, and remote sensing and geographic information system (GIS) applications within a framework that accommodates translating the results to support the development of programs and policies specific to climate change adaptation.

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Chapter 2.

Calculating and displaying downscaled estimates of heat-related cardiorespiratory mortality in 20 U.S. cities at the census tract level

2.1 ABSTRACT

BACKGROUND: The expectation that the intensity, frequency and duration of extreme heat events will increase in coming years as a result of climate change establishes an urgency to prepare for such events. Municipalities developing climate adaptation and preparedness plans seek to identify populations vulnerable to health effects during extreme heat. Although health outcome data is often available in geographic units containing large numbers of people (e.g., counties), research on heat-health associations that contributes to decision-making and policy development at a scale relevant to specific local populations is needed.

OBJECTIVES: This paper demonstrates a method for 1) downscaling U.S. county-level effect estimates of the odds of cardiorespiratory death during extreme heat to reflect the census tract level proportions of populations known to be vulnerable to heat, and 2) displaying the odds of cardiorespiratory death related to extreme heat exposure for 20 large U.S. cities at the census tract scale.

METHODS: Time-stratified case crossover analysis was conducted for 20 U.S. cities to investigate the association between extreme heat (days $> 95^{th}$ percentile of month- and location-specific daily apparent temperature in May-September) and cardiorespiratory deaths from 1990-2006 among specific demographic subgroups defined by age, race and gender, as recorded on death certificates. Then, demographic data from the 2010 U.S. census was used to 'downscale' these estimates by computing, for each census tract,

a weighted average of the county-level, sub-group specific odds ratios of cardiorespiratory death associated with heat. Weights were assigned according to census tract-specific population characteristics. The weighted odds ratios were then mapped using a geographic information system. Five cities that represent each of five climate zones in the U.S. are presented in detail to evaluate model results and spatial patterns.

RESULTS: Extreme heat was significantly associated with cardiorespiratory mortality most frequently among: white males under the age of 65; and white males, white females and nonwhite females over the age of 75. Distinct patterns of elevated risk of heat-related cardiorespiratory death were not seen across subpopulations, although heat was positively associated with mortality in most cities. Nonwhite males and females over the age of 65 in Chicago observed the highest risk of cardiorespiratory death associated with heat, with odds ratios ranging between 1.29 and 1.38 (p-value <0.0001).

CONCLUSION: Downscaled estimates of the associations between extreme heat and cardiorespiratory mortality displayed spatial heterogeneity across the 20 study cities. Fine-scale analyses of heat-related mortality can inform programs to target the populations most likely to experience deleterious effects during hot weather.

2.2 Keywords

downscaling, case-crossover, vulnerability, extreme heat

2.3 Abbreviations and definitions

EH₉₅ Indicator variable for extreme heat, defined as apparent

temperature for lag days 0,1 above the month-specific

average of apparent temperature at lag days 0,1.

MSA Metropolitan Statistical Area

Mortality_{CVDRESP} Deaths due to cardiorespiratory disease

2.4 Introduction and background

The association between weather and mortality has been documented in many epidemiological studies [1-7]. Different methodological and computational approaches for characterizing the typically non-linear J- or U-shaped relation between daily temperature and mortality have been employed [7-9]. Despite these differences, consensus that daily mortality often increases after short-term exposure to high ambient temperatures [10-12] directs attention to the need for public health interventions to reduce this risk.

The expectation that the intensity, frequency and duration of extreme heat events will increase in coming years as a result of climate change establishes an urgency to prepare for such events [13]. The United States (U.S.) National Climate Assessment reports that early actions — those that result from preparedness and planning - will likely have the largest impact in protecting human health[14]. Cities across the U.S. have adopted efforts to prepare for and respond to extreme heat through a variety of mechanisms including preparedness planning, heat wave health warning systems, education and outreach, and establishing cooling centers. However, these efforts have been moderately successful in terms of behavioral changes among populations considered most vulnerable to extreme heat. In many cases, members of vulnerable populations misperceive appropriate behavioral responses and often underestimate their own risk [15, 16]. As local governments continue to be called upon to implement or strengthen heat-related programs, targeted assessments can inform decision makers on how to prepare for and respond during extreme heat events [17, 18].

Quantifying the number of deaths due directly to heat exposure is a challenge for epidemiologic studies because of inconsistent definitions of heat-specific death [19, 20], and likely underreporting [21, 22]. Individuals with preexisting health conditions - such as cardiovascular and respiratory disease - are considered susceptible to extreme heat as the heart and lungs can experience increased stress as blood is pumped away from vital organs during thermoregulation [21]. Deaths due to cardiovascular and respiratory disease increase during periods of high temperatures, defined as both heat waves and

extreme heat events [10, 12, 19, 23-25]. In addition to preexisting health conditions, key individual-level markers of vulnerability to extreme heat include older age, minority race/ethnicity, lower educational attainment, and being female [4, 5, 26, 27]. Further, spatial heterogeneity of extreme heat effects suggests that place-based assessments are appropriate, rather than assuming a one-size-fits-all approach [3, 4, 7, 8, 28-32].

Despite the complex relationship between ambient environmental conditions and health, sufficient understanding of the association between heat exposure and mortality exists [33] to use this knowledge to inform public health interventions. However, multicity temperature-health studies often calculate effect estimates that are not easily translatable for targeted interventions or preparedness planning for extremely hot weather, as they lack specific estimates for vulnerable populations in particular locations. Further, to our knowledge, no analyses have visually captured the intra-urban spatial heterogeneity of the calculated health burden of extreme heat, although proxy measures of heat vulnerability have been mapped [34-37].

We propose a method for displaying the spatial heterogeneity of the odds of death associated with extreme heat among vulnerable populations living in 20 large U.S. Cities. We calculate the relative risk of cardiorespiratory deaths associated with extreme heat exposure among twelve demographic subpopulations within these cities defined by age, race and gender. Using demographic data from the 2010 U.S. Census, we calculate a weighted, or 'downscaled', odds ratio of heat-related cardiorespiratory death according to tract-specific subpopulation characteristics. We then present a chloropleth map of the downscaled odds ratios to display spatial patterns of vulnerability to extreme heat.

2.5 Methods

2.5.1. Mortality data and city selection

Daily mortality records were obtained from the National Center for Health Statistics for the years 1990 – 2006 for 20 largest U.S. cities, as identified by the 2000 US Census (http://www.census.gov/statab/ccdb/cit1020r.txt). Individual-level data extracted from these records included primary cause of death; decedent's county of residence; date of

death; age; race; gender and education. A city was defined to include all urban counties comprising its Metropolitan Statistical Area (MSA). County-specific daily deaths with primary causes being cardiovascular (International Classification of Diseases 9th revision (ICD9): 390 – 429; International Classification of Diseases 10th revision (ICD10)) ICD10: I01 – I59) or respiratory (ICD 9: 460 – 519; ICD10: J00 – J99) diseases were aggregated to create county-specific daily deaths due to cardiorespiratory causes for use in this analysis. Individuals who were determined to have both lived and died in the same county were included in this analysis. We further created categories for the individual characteristics recorded on death certificates that were previously shown to be associated with vulnerability to heat: white race and non-white race; less than 65 years of age, between 65 and 75 years of age, and older than 75 years of age; and male and female.

2.5.2. Demographic data

City-specific census tract level demographic data was collected from the 2010 US Census (www.census.gov). The variables of interest for this analysis were the tract-level proportions of subpopulations in the same age groups and race and gender categories defined for the individual-level mortality data, as above. We calculated the census tract-specific proportions of the twelve combinations of age, race, and gender. By applying the most recent tract-specific demographic proportions, the downscaled estimates will reflect the current populations' risk related to extreme heat.

2.5.3. Weather data

Hourly weather and dew point data from airport weather stations nearest to the 20 study cities were obtained from the National Climatic Data Center (NCDC). A two-day moving average of mean daily apparent temperature (AT °C), a composite of temperature and dewpoint [6], was calculated for each city (Equation (EQ) 1), for summer months (May – September, 1990 - 2006). These represent the average of mean apparent temperatures occurring on the date of death and the previous day (Lag01) during the time period of interest for this research. Lag01 has been shown to best capture the effect of heat on mortality in a short time frame [1].

EQ 1. AT = -2.653 + (0.994 x ambient temperature) + (0.0153 x dew point temperature)

Extreme heat (EHO1 $_{95}$) was defined as being a day during the study period that exceeded the two-day mean apparent temperature for lagO1 (AT $_{01}$), based on month-and city-specific 95 th percentiles. An indicator variable was constructed with a value of 1 on days that exceeded the month-specific 95 th AT $_{01}$, and zero otherwise. We examined temperature exposure during summer months (1 May - 30 September), as the impacts of extreme heat are most likely to occur during this time period.

2.5.4. Study design and analysis

Descriptive statistics on the mortality, demographic and weather data were calculated. To evaluate the association between extreme temperature and cardiorespiratory deaths in the study cities, a time stratified case-crossover analysis was used. The case-crossover approach, similar to a matched-case control study where the cases serve as their own controls, estimates acute, transient effects that result shortly after an exposure [38] and is commonly employed to study extreme heat exposure and cause-specific mortality [39]. The study design controls for known and unknown time-invariant confounders, as well as minimizes the effects of time trend and seasonality. Control days were selected on the same day of the week in the same calendar month as cases. [40]. A conditional logistic regression model is fit for case-crossover studies, allowing calculation of the odds ratio of cardiorespiratory death on summer days when extreme heat occurred versus more moderate temperature days and its corresponding 95% confidence interval (CI).

One of our key goals was to evaluate whether the associations between heat and mortality differed according to age, race and gender. We evaluated whether those individual-level characteristics were effect modifiers of the extreme heat and mortality relationship by using interaction terms between the demographic indicator variables and the indicator variable for extreme heat in the case crossover model [41]. Crude models (Model 1) provided a baseline, descriptive estimate of the association between extreme heat and all cardiorespiratory mortality for the 20 cities.

```
Model (1). logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95}
```

To estimate the effect of extreme heat on specific subpopulations, we included higher order interaction terms for the effect of extreme heat by age, race and gender, thus accounting for the differential effects of exposure to extreme heat previously observed in such subpopulations [27, 42]. The final model (Model 2) used to estimate the subpopulation-specific odds of cardiorespiratory death associated with extreme heat was:

```
Model (2).  \begin{aligned} & \text{logit}(\text{mortality}_{\text{CVDRESP}}) = \beta_1 \text{EHO1}_{95} + \beta_2 (\text{EHO1}_{95} \bullet \text{Race}) + \beta_3 (\text{EHO1}_{95} \bullet \text{Gender}) + \\ & \beta_4 (\text{EHO1}_{95} \bullet \text{Age}_{65\text{-}75}) \ + \beta_5 (\text{EHO1}_{95} \bullet \text{Age}_{\geq 75}) + \beta_6 (\text{EHO1}_{95} \bullet \text{Race} \bullet \text{Gender}) + \end{aligned}
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 $\beta_7(EHO1_{95} \bullet Race \bullet Age_{65-75}) + \beta_8(EHO1_{95} \bullet Race \bullet Age_{\geq 75}) + \beta_9(EHO1_{95} \bullet Gender \bullet Age_{65-75}) + \beta_8(EHO1_{95} \bullet Gender \bullet Age_{\geq 75})$

Because we were interested in three demographic variables with three categories for age, and two for race and gender, Model 2 yielded twelve subpopulation-specific odds ratios and their corresponding 95% CI's.

2.5.5. Downscaling and mapping estimates

The proportions of the twelve subpopulations within each census tract were calculated for the 20 cities. The twelve subpopulation-specific odds ratios and their respective census tract proportions were multiplied and then added to yield an overall weighted odds ratio for the census tract. The idea was that if extreme heat were associated with higher excess mortality in specific sub-populations than in others, a census tract with a large proportion of people in those vulnerable sub-populations would have a higher weighted heat-mortality odds ratio than a census tract in which proportionally fewer of those more vulnerable people resided. This method was applied for all census tracts in each of the 20 cities. Next, the downscaled census tract-level odds ratios were mapped to display the spatial variation of the odds of cardiorespiratory death associated with exposure to extreme heat. Five cities representative of distinct US climate zones [43] are

presented and discussed in detail. Because no specific measure of spatial heterogeneity could be calculated in this analysis, descriptive statistics of the weighted odds ratios for each of the 20 study cities are presented and discussed.

All analyses were carried out using SAS Version 9.3, R and ArcGIS 10.

2.6 Results.

Table 2.1 shows descriptive statistics of the study cities, including the number of cardiorespiratory deaths observed. The distribution of the age, race and gender characteristics that are the focus of this study were mostly consistent across decedents in the study cities. In general, there was an equal distribution of male and female cases, while the majority of cases in every city were characterized as white. Table 2.2 shows that the month-specific means of daily cardiorespiratory deaths for each city during the study period were relatively stable. Month-specific apparent temperatures for lag 01 followed expected increasing patterns during the summer months, decreasing around September.

Census tract level subpopulation descriptive statistics are shown in Table 2.3. Table 2.3 illustrates that over half the total population in each city was below the age of 65, with the percentage of people in the age 65 - 75, and 75 and older groups ranging from less than 1% to about 7% of the population. A slightly larger proportion of white females were found in the older age categories, which is expected as women live longer than men. More white women lived in most cities than women of other races.

Table 2.1. Descriptive statistics for cardiorespiratory mortality in 20 large U.S. cities, 1990 - 2006

	Counties	Number of		Age		F	Race	Gender	
City	comprising cities	cardiorespiratory	< 65 years	65 - <i>7</i> 5 years	≥ 75 years	White	Nonwhite	Male	Female
	comprising cities	cases	(%)	(%)	(%)	(%)	(%)	(%)	(%)
Austin, TX	Travis	6,191	23.0	21.5	55.5	82.4	17.6	52.6	47.4
Baltimore, MD	Anne Arundel, Baltimore, Baltimore City, Harford	20,665	29.6	29.3	41.1	58.3	41.7	52.5	47.5
Boston, MA	Essex, Middlesex, Norfolk, Plymouth, Suffolk	17,234	29.8	31.4	38.8	81.0	19.0	52.5	47.5
Chicago, IL	Cooke, DuPage, Kane, Lake, McHenry, Will	28,450	33.O	31.7	35.3	52.9	47.1	51.3	48.7
Columbus, OH	Franklin	11,637	25.9	27.3	46.8	76.6	23.4	51.8	48.2
Dallas, TX	Dallas	16,412	30.9	27.3	41.8	67.4	32.6	52.8	47.2
Detroit, MI	Macomb, Oakland, Wayne	26,141	31.9	30.9	37.2	54.7	45.3	52.0	48.0
Houston, TX	Harris	20,424	32.2	28.1	39.7	61.1	38.9	53.1	46.9
Indianapolis, IN	Marion	11,808	25.1	26.3	48.6	<i>7</i> 2.3	27.7	51.7	48.3
Jacksonville, FL	Duval	10,783	25.6	25.1	49.3	69.1	30.9	51.2	48.8
Los Angeles, CA	Los Angeles	28,064	31.8	31.9	36.3	53.9	46.1	51.8	48.2
Memphis, TN	Shelby	13,226	27.5	24.3	48.2	51.4	48.6	50.3	49.7
Milwaukee, WI	Milwaukee, Waukesha	12, <i>7</i> 11	25.4	27.7	46.9	79.6	20.4	54.1	45.9
New York, NY	Bronx, Kings, New York, Queens, Richmond, Westchester	28,100	32.3	32.5	35.2	48.6	51.4	50.8	49.2
Philadelphia, PA/NJ	Bucks, Burlington*, Camden*, Chester, Delaware, Glouchester, Montgomery, Philadelphia	25,731	31.5	31.5	37.0	56.5	43.5	51.8	48.2
Phoenix, AZ	Maricopa	15,141	29.9	30.8	39.3	87.3	12.7	53.9	46.1
San Antonio, TX	Bexar	11.986	25.0	27.4	47.6	83.8	16.2	53.3	46.7
San Diego, CA	San Diego	16,635	26.6	30.2	43.2	78.3	21.7	54.3	45.7
San Francisco, CA	San Francisco, San Mateo	14,638	21.3	25.0	53.7	62.8	37.2	55.6	44.4
San Jose, CA	Santa Clara	11,247	22.8	24.6	52.6	<i>7</i> 8.4	21.6	54.6	45.4

^{*} Denotes New Jersey counties

Table 2.2. Month-specific descriptive statistics of cardiorespiratory deaths and apparent temperature (Lag01), by study city (1990-2006)

	Mean	daily numl	ber of cardi	orespirator	y deaths,	Month-specific 95th percentile apparent temperature,						
		by m	onth (1990	-2006)		Lago1 ([©] C) (1990 - 2006)						
	May	June	July	August	September	May	June	July	August	September		
Austin, TX	2.47	2.36	2.42	2.34	2.32	32.98	35.64	35.77	35.58	34.58		
Baltimore, MD	8.09	7.90	8.01	7.98	7.73	26.42	31.45	33.90	32.81	29.46		
Boston, MA	6.65	6.68	6.71	6.55	6.55	20.50	28.47	31.37	30.22	25.41		
Chicago, IL	10.98	10.98	10.89	10.88	10.96	24.28	29.74	32.74	31.22	26.86		
Columbus, OH	4.53	4.57	4.42	4.36	4.50	25.35	30.05	32.09	31.92	27.54		
Dallas, TX	6.39	6.36	6.33	6.20	6.06	32.18	35.25	36.36	35.93	33.94		
Detroit, MI	10.09	10.14	10.04	9.97	10.00	24.27	29.83	31.27	30.55	26.10		
Houston, TX	7.98	7.94	7.80	7.70	7.85	33.54	36.24	36.54	36.43	35.06		
Indianapolis, IN	4.64	4.47	4.51	4.49	4.59	25.75	30.37	32.86	32.11	27.73		
Jacksonville, FL	4.18	4.14	4.16	4.13	4.12	31.14	34.46	35.65	35.09	33.81		
Los Angeles, CA	10.91	10.77	10.77	10.73	10.77	21.13	22.77	26.24	26.96	26.09		
Memphis, TN	5.29	5.18	4.79	5.10	5.07	30.34	34.41	36.15	35.93	33.28		
Milwaukee, WI	5.01	4.96	4.83	4.81	4.82	22.78	28.92	31.73	30.70	26.31		
New York, NY	10.85	10.88	10.78	10.78	10.72	24.00	30.58	33.83	33.08	29.13		
Philadelphia, PA	9.98	10.01	9.91	9.72	9.85	26.07	31.65	34.87	33.95	29.54		
Phoenix, AZ	6.01	5.80	5.76	5.83	5.71	30.49	35.41	38.31	38.14	35.82		
San Antonio, TX	4.65	4.68	4.57	4.55	4.60	33.33	35.47	35.59	35.67	34.44		
San Diego, CA	6.11	6.00	6.06	5.84	6.04	21.41	23.65	26.50	27.58	27.49		
San Francisco, CA	5.87	5.78	5.61	5.49	5.39	18.65	19.99	20.69	21.36	21.73		
San Jose, CA	4.59	4.58	4.36	4.02	4.07	22.07	25.08	25.89	25.95	25.33		

Table 2.3. Mean proportion of 2010 census tract-specific subpopulation for 20 U.S. cities

City (no. of census tracts)	White males, < 65 years	White males, 65 - 75 years	White males, ≥ 75 years	Nonwhite males, < 65 years	Nonwhite males, 65 - 75 years	Nonwhite males, ≥ 75 years	White females, < 65 years	White females, 65 - 75 years	White females, ≥ 75 years	Nonwhite females, < 65 years	Nonwhite females, 65 - 75 years	Nonwhite females, ≥ 75 years
Austin, TX	0.33	0.02	0.01	0.15	0.00	0.00	0.31	0.02	0.02	0.14	0.00	0.00
(217)	0.33	0.02	0.01	0.15	0.00	0.00	0.31	0.02	0.02	0.14	0.00	0.00
Baltimore, MD (572)	0.24	0.02	0.02	0.18	0.01	0.01	0.24	0.02	0.03	0.20	0.01	0.01
Boston, MA (909)	0.32	0.03	0.02	0.11	0.00	0.00	0.32	0.03	0.04	0.12	0.01	0.00
Chicago, IL (1,967)	0.26	0.02	0.01	0.18	0.01	0.01	0.26	0.02	0.02	0.19	0.01	0.01
Columbus, OH (284)	0.29	0.02	0.01	0.15	0.01	0.00	0.29	0.03	0.02	0.16	0.01	0.01
Dallas, TX	0.25	0.02	0.01	0.21	0.01	0.00	0.24	0.02	0.02	0.21	0.01	0.01
(528) Detroit, MI	0.26	0.02	0.02	0.17	0.01	0.01	0.26	0.03	0.03	0.18	0.01	0.01
(1,158) Houston, TX	0.26	0.02	0.01	0.20	0.01	0.00	0.25	0.02	0.02	0.20	0.01	0.01
(786) Indianapolis, IN	0.26	0.02	0.01	0.18	0.01	0.00	0.26	0.02	0.02	0.19	0.01	0.01
(224) Jacksonville, FL												
(173)	0.26	0.02	0.02	0.18	0.01	0.00	0.25	0.02	0.03	0.19	0.01	0.01
Los Angeles, CA (2,334)	0.22	0.02	0.01	0.22	0.01	0.01	0.21	0.02	0.02	0.23	0.01	0.01
Memphis, TN (220)	0.16	0.01	0.01	0.28	0.01	0.01	0.15	0.02	0.02	0.30	0.02	0.01
Milwaukee, WI (382)	0.28	0.02	0.02	0.16	0.00	0.00	0.27	0.02	0.03	0.17	0.01	0.00
New York, NY (2,361)	0.19	0.02	0.01	0.23	0.01	0.01	0.19	0.02	0.02	0.26	0.02	0.01
Philadelphia, PA (1,297)	0.28	0.02	0.02	0.14	0.01	0.00	0.28	0.03	0.03	0.15	0.01	0.01
Phoenix, AZ (913)	0.31	0.03	0.02	0.13	0.00	0.00	0.31	0.03	0.03	0.13	0.00	0.00
San Antonio, TX (365)	0.32	0.02	0.02	0.13	0.00	0.00	0.32	0.03	0.03	0.13	0.01	0.00
San Diego, CA (627)	0.28	0.02	0.02	0.16	0.01	0.00	0.27	0.03	0.03	0.16	0.01	0.01
San Francisco, CA	0.24	0.02	0.02	0.20	0.01	0.01	0.21	0.02	0.02	0.21	0.02	0.02
(351) San Jose, CA (372)	0.21	0.02	0.01	0.24	0.01	0.01	0.20	0.02	0.02	0.24	0.01	0.01

Odds ratios and 95% CI's for the 20 cities' subpopulations are presented in Figures 2.1 a-s. Odds ratios are interpreted as a measure of the association between the exposure to extreme heat and cardiorespiratory death, specifically the odds of cardiorespiratory death on a day defined as being extremely hot, versus a more moderate temperature day, in a particular subpopulation. The 95% confidence intervals are a measure of precision of the estimate, with the narrower CI around the OR being the more precise estimate, and where CIs containing an OR = 1 are not statistically significant, and thus consistent with the possibility of no association.

Significant positive associations between extreme heat and daily mortality were most frequently observed in the following subpopulations: white males under the age of 65, and white males, white females and nonwhite females over the age of 75. Nine of the twelve subpopulations in Chicago observed statistically significant increased odds of death associated with extreme heat. Nonwhite males and females over the age of 65 had significantly higher odds of cardiorespiratory death compared to their white male and female counterparts under the age of 65" (Nonwhite males 65-75: 1.38 (1.21, 1.56); (Nonwhite males > 75: 1.30 (1.16, 1.45); Nonwhite females 65-75: 1.32 (1.15, 1.51); (Nonwhite females > 75: 1.29 (1.18, 1.41)). Most cities showed increased odds of death associated with heat across subpopulations, although distinct patterns were not apparent. Conversely, point estimates for the association of heat and mortality in Columbus, OH; Indianapolis, IN; Jacksonville, FL and Memphis, TN were protective, but significantly so only among white males over 75 in Memphis and nonwhite males under 65 in Milwaukee. Associations between cardiorespiratory mortality and extreme heat among young white females were never statistically significant. Complete cityspecific model results are shown in Appendix A.

Figures 2.1.a-s. Subpopulation odds ratios (OR's) and 95% confidence intervals (CI's) for cardiorespiratory mortality associated with extreme heat (>95th percentile in month) for the 20 study cities, from a case-crossover analysis, May-September, 1990-2006.

Figure 2.1.b.

May - September, 1990 - 2006

Subpopulation ORs, 95% CIs for cardiorespiratory mortality

and extreme heat (>95th percentile in month) for Baltimore, MD;

Figure 2.1.a. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Austin, TX; May – September, 1990 - 2006

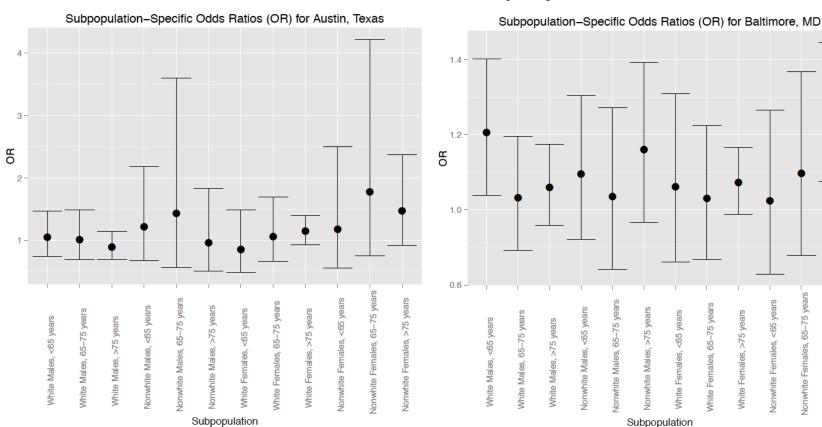


Figure 2.1.c. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Boston, MA May - September, 1990 - 2006

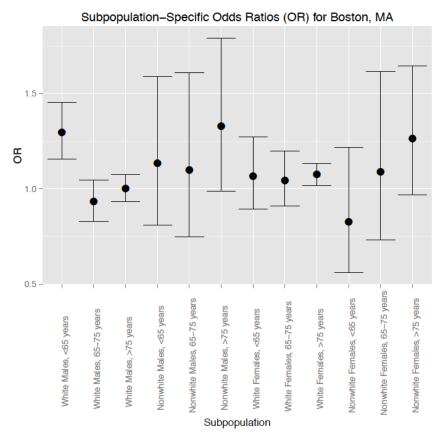


Figure 2.1.d. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Chicago, IL; May - September, 1990 - 2006

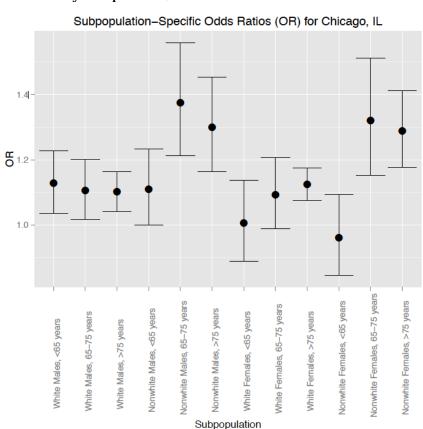


Figure 2.1.e. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Columbus, OH May - September, 1990 - 2006

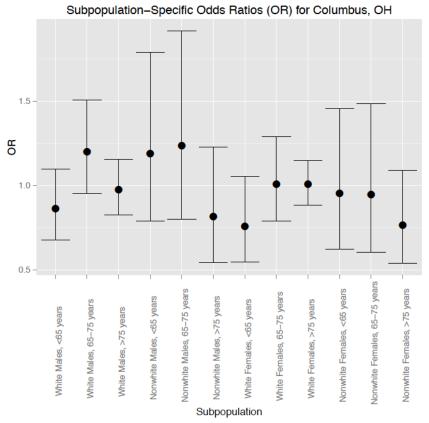


Figure 2.1.f. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Dallas, TX; May - September, 1990 - 2006

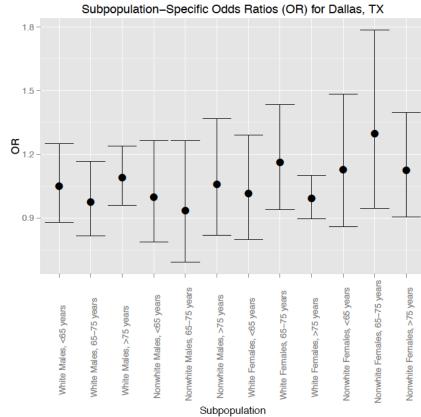


Figure 2.1.g.
Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Detroit, MI May – September, 1990 - 2006

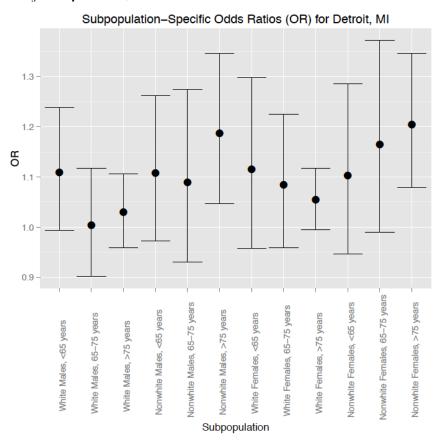


Figure 2.1.h . Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Houston, TX; May - September, 1990 - 2006

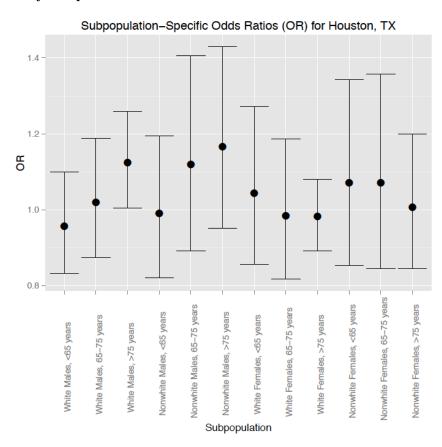


Figure 2.1.i. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Indianapolis, IN; May – September, 1990-2006

Figure 2.1.j.
Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Jacksonville, FL; May – September, 1990 - 2006

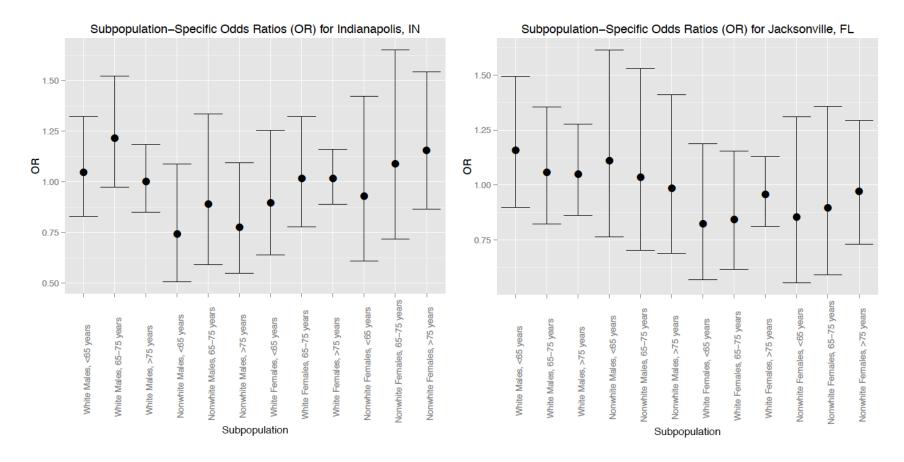


Figure 2.1.k. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Los Angeles, CA; May - September, 1990 - 2006

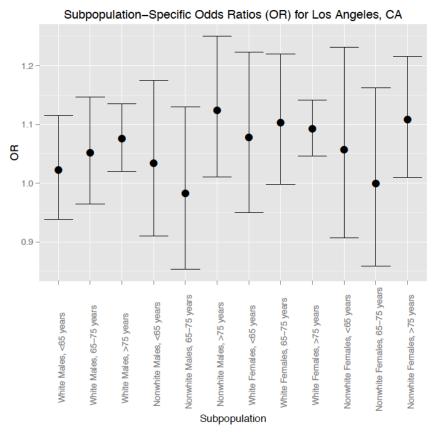


Figure 2.1.l. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Memphis, TN; May – September, 1990 - 2006

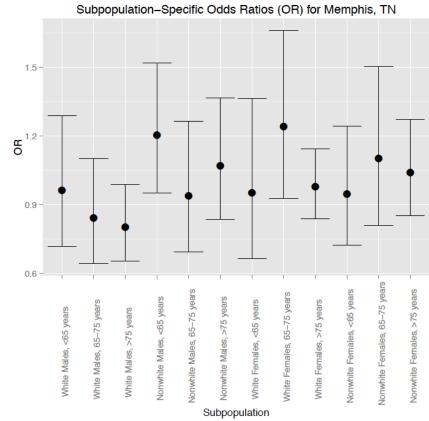


Figure 2.1.m. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Milwaukee, WI; May - September, 1990 - 2006

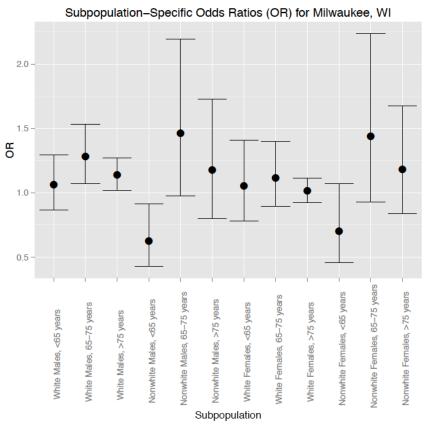


Figure 2.1.n.
Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for New York City, NY; May – September, 1990 - 2006

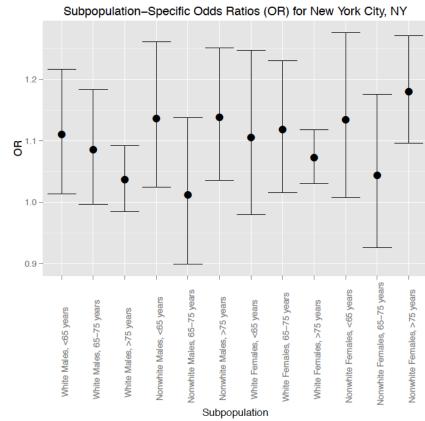


Figure 2.1.o. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Philadelphia, PA; May - September, 1990 - 2006

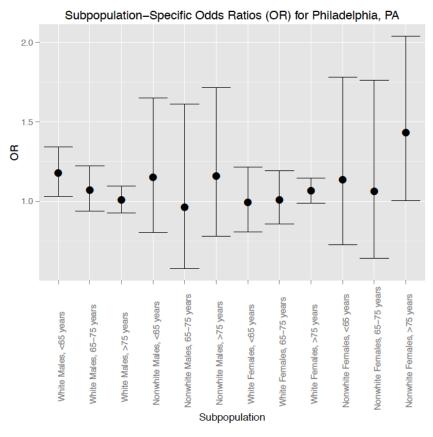


Figure 2.1.p. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for Phoenix, AZ; May – September, 1990 - 2006

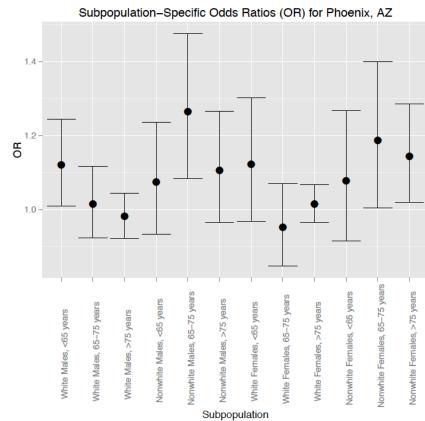


Figure 2.1.q. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for San Antonio, TX; May - September, 1990 - 2006

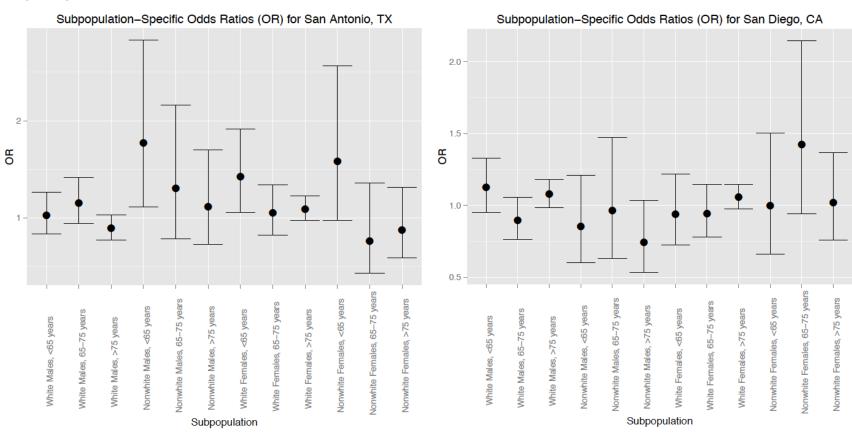


Figure 2.1.r.

May – September, 1990 - 2006

Subpopulation ORs, 95% CIs for cardiorespiratory mortality

and extreme heat (>95th percentile in month) for San Diego, CA;

Figure 2.1.s. Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for San Francisco, CA; May – September, 1990 - 2006

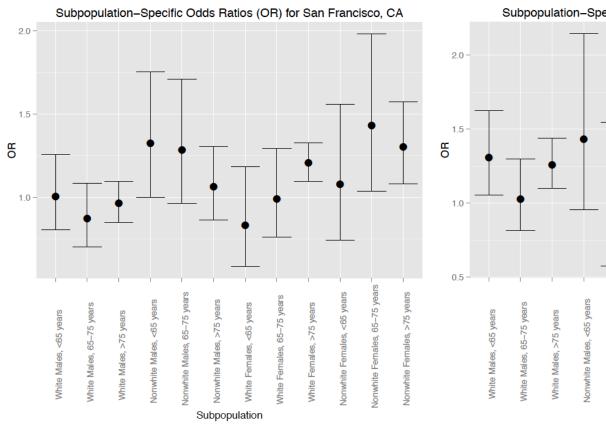
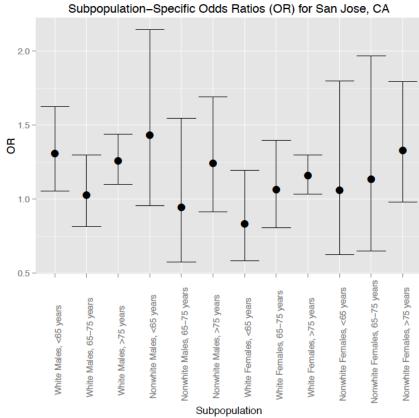
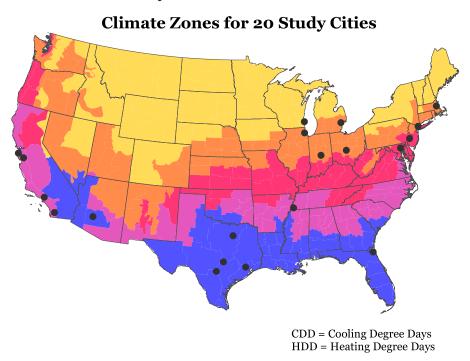


Figure 2.1.t.
Subpopulation ORs, 95% CIs for cardiorespiratory mortality and extreme heat (>95th percentile in month) for San Jose, CA; May – September, 1990 - 2006



Based on the climate zone designations presented in Figure 2.2, we selected five cities (Milwaukee, WI, Chicago, IL, Baltimore, MD, San Francisco, CA and Houston, TX) that represented each climate zone to present maps displaying the downscaled, census-tract specific odds ratios for the association of extreme heat and cardiorespiratory mortality. Cooling and heating degree days are a descriptive way to relate each day's temperature to the energy demand necessary to either heat or cool a building. This characterization has been utilized in previous heat-health studies to categorize cities and their prevailing climates [43].

Figure 2.2. U.S. climate zones, determined by cooling and heating degree days, and locations of the 20 study cities within each zone



The study cities and their respective weighted odds ratios descriptive statistics are presented by climate zone in Table 2.4.

Table 2.4. Descriptive statistics of estimated census tract level weighted odds ratios for heat related mortality for 20 U.S. cities, by climate zone.

	Census tracts (n)	Mean	Median Standard Deviation		Range
Climate Zone 1					
Milwaukee, WI	382	0.94	1.01	0.12	0.70, 1.06
Climate Zone 2					
Boston, MA	909	1.12	1.13	0.04	0.98, 1.27
Chicago, IL	1,967	1.07	1.0 7	0.01	1.03, 1.15
Columbus, OH	284	0.92	0.89	0.06	0.84, 1.10
Detroit, MI	1,158	1.11	1.10	0.01	1.08, 1.12
Indianapolis, IN	224	0.93	0.94	0.03	0.87, 0.98
New York, NY	2,361	1.12	1.12	0.01	1.07, 1.14
Climate Zone 3					
Baltimore, MD	572	1.10	1.10	0.02	1.06, 1.16
Philadelphia, PA	1,29 7	1.10	1.09	0.02	1.05, 1.16
Climate Zone 4					
Los Angeles, CA	2,334	1.05	1.05	0.00	1.03, 1.09
Memphis, TN	220	1.03	1.04	0.04	0.97, 1.13
San Diego, CA	62 7	1.00	1.00	0.02	0.95, 1.07
San Francisco, CA	351	1.0 7	1.0 7	0.06	0.96, 1.21
San Jose, CA	372	1.1 7	1.1 7	0.03	1.09, 1.25
Climate Zone 5					
Austin, TX	217	1.04	1.03	0.04	0.97, 1.16
Dallas, TX	528	1.05	1.05	0.01	1.03, 1.08
Houston, TX	7 86	1.02	1.01	0.01	0.98, 1.04
Jacksonville, FL	173	0.98	0.98	0.01	0.96, 1.06
Phoenix, AZ	913	1.09	1.10	0.02	1.01, 1.11
San Antonio, TX	365	1.32	1.32	0.05	1.03, 1.49

Variation in the mean weighted odds ratio was observed for cities in climate zones 2, 4 and 5. In climate zone 2, Boston Detroit and New York have similar means that reflect an increased risk of heat-related cardiorespiratory death, but Columbus and Indianapolis have similarly decreased risks of heat-related cardiorespiratory death. In climate zones 4 and 5, respectively, San Jose and San Antonio had much larger weighted odds ratios than the other cities in their climate zone. However, despite these differences, the only city that observed a relatively high standard deviation — which would indicate very distinct spatial heterogeneity — was Milwaukee.

Table 2.5 presents the subpopulation-specific odds ratios and their corresponding confidence intervals for Milwaukee, Chicago, Baltimore, San Francisco and Houston. We observed large odds ratios for white males, and nonwhite males and females between 65 and 75 years old in Milwaukee; however, these odds ratios were accompanied by large confidence intervals.

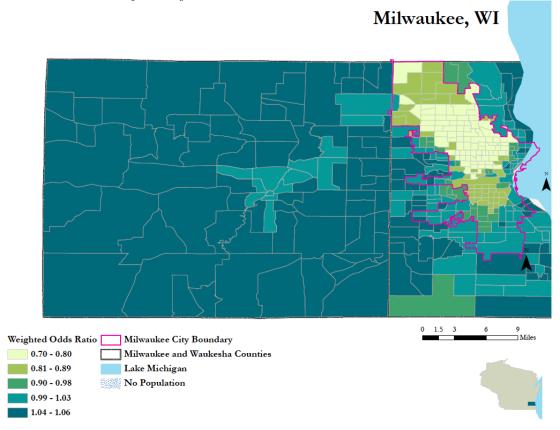
Table 2.5. Subpopulation-specific odds ratios and 95% confidence intervals for associations between extreme heat and cardiorespiratory mortality in cities representative of 5 U.S. climate zones

	Climate Zone 1	Climate Zone 2	Climate Zone 3	Climate Zone 4	Climate Zone 5
Sub-population	Milwaukee, WI	Chicago, IL	Baltimore, MD	San Francisco, CA	Houston, TX
White males, <65 years	1.06 (0.87, 1.3)	1.13 (1.04, 1.23)	1.21 (1.04, 1.40)	1.01 (0.81, 1.26)	0.96 (0.83, 1.10)
White males, ≥ 65 - < 75 years	1.28 (1.07, 1.53)	1.11 (1.02, 1.20)	1.03 (0.89, 1.20)	0.88 (0.71, 1.09)	1.02 (0.87, 1.19)
White males, ≥ 75 years	1.14 (1.02, 1.27)	1.10 (1.04, 1.16)	1.06 (0.96, 1.17)	0.97 (0.85, 1.10)	1.12 (1.00, 1.26)
Nonwhite males, <65 years	0.63 (0.43, 0.91)	1.11 (1.00, 1.23)	1.10 (0.92, 1.30)	1.33 (1.00, 1.76)	0.99 (0.82, 1.20)
Nonwhite males, $\geq 65 - < 75$ years	1.46 (0.97, 2.19)	1.38 (1.21, 1.56)	1.04 (0.84, 1.27)	1.29 (0.96, 1.71)	1.12 (0.89, 1.41)
Nonwhite males, ≥ 75 years	1.17 (0.80, 1.73)	1.30 (1.16, 1.45)	1.16 (0.97, 1.39)	1.06 (0.87, 1.31)	1.17 (0.95, 1.43)
White females, <65 years	1.05 (0.78, 1.41)	1.01 (0.89, 1.14)	1.06 (0.86, 1.31)	0.83 (0.59, 1.18)	1.04 (0.86, 1.27)
White females, ≥ 65 - < 75 years	1.12 (0.89, 1.40)	1.09 (0.99, 1.21)	1.03 (0.87, 1.22)	0.99 (0.76, 1.30)	0.98 (0.82, 1.19)
White females, ≥ 75 years	1.01 (0.92, 1.11)	1.12 (1.08, 1.17)	1.07 (0.99, 1.17)	1.21 (1.10, 1.33)	0.98 (0.89, 1.08)
Nonwhite females, <65 years	0.70 (0.46, 1.07)	0.96 (0.84, 1.09)	1.02 (0.83, 1.26)	1.08 (0.75, 1.56)	1.07 (0.85, 1.34)
Nonwhite females, $\geq 65 - < 75$ years	1.44 (0.93, 2.23)	1.32 (1.15, 1.51)	1.10 (0.88, 1.37)	1.43 (1.04, 1.98)	1.07 (0.84, 1.36)
Nonwhite females, ≥ 75 years	1.18 (0.84, 1.67)	1.29 (1.18, 1.41)	1.25 (1.08, 1.44)	1.31 (1.08, 1.58)	1.01 (0.85, 1.20)

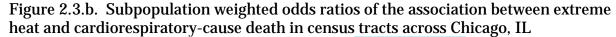
Nonwhite males between 65 and 75 in Chicago experienced a 38% excess of cardiorespiratory death during an extreme heat event (95% CI: (1.21, 1.56) compared to a more moderate temperature day, an excess substantially higher than that observed for their white, male counterparts under the age of 65 OR 1.13 95%CI (1.04, 1.23). Similarly, nonwhite females older than 75 in Milwaukee, Chicago, Baltimore and San Francisco experienced significantly higher odds of cardiorespiratory death associated with extreme heat, with point estimates for the OR's of 1.18, 1.29, 1.25, and 1.31, respectively in these cities. In more temperate climate zones, zones 4 and 5, we observed a larger effect of extreme heat in subpopulations between 65 and 75, but patterns differed depending on race and gender. In San Francisco, large effects were observed among nonwhite males and females, although confidence intervals were wide. Houston's population of nonwhite males and females also experienced higher heat associations than their white counterparts, but these associations were not statistically significant nor as large as those observed in cities characterized as having temperate climate zones.

Maps for Milwaukee, Chicago, Baltimore, San Francisco and Houston (Figures 2.3.a-e) display the census tract-level downscaled odds ratios for the association between extreme heat and cardiorespiratory disease.

Figure 2.3.a. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Milwaukee, WI



The spatial distribution of the odds of cardiorespiratory death due to extreme heat exposure is uniform across the Milwaukee-designated MSA. A clear pattern of increasing heat-mortality associations is observed when moving west from the central portion of the city (located along Lake Michigan) to the surrounding areas. Odds ratios suggesting adverse effects (1.03-1.06) are distributed in the western three-quarters of the Milwaukee study area, never within the Milwaukee city boundary.



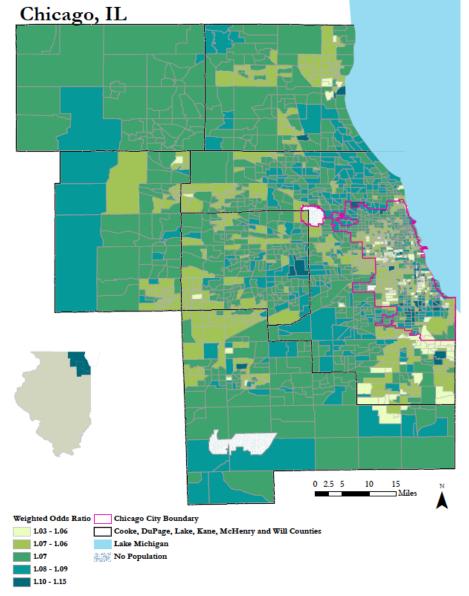
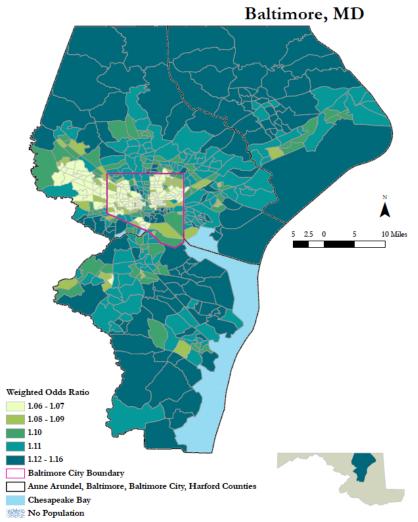
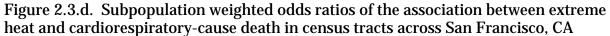


Figure 2.3.b presents mapped odds ratios of census tracts in the Chicago study area. Census tracts where the highest odds ratios were estimated are sprinkled through the study area. Large census tracts in the west, southwest, and central portions of the study area show high (1.10-1.15) odds of cardiorespiratory death linked to heat. Notably, neighboring census tracts within the city boundary, as well as in the southeastern portion of the study area, indicate contrasting effects of the risk of death associated with heat. Within the city boundary, the highest odds ratios are concentrated together in the southernmost portion. Lowest odds ratios were observed in the southeastern study area.

Figure 2.3.c. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Baltimore, MD



The highest weighted odds ratios in Baltimore, MD were observed in the areas surrounding the Baltimore city boundary. The spatial pattern observed was similar to that of Milwaukee, where the lowest odds of cardiorespiratory death associated with extreme heat exposure were observed in the central portion of the metropolitan area, with increasing odds of death observed as you move away from the city center. As shown in Figure 2.3.c, a distinct corridor of lower odds extended from the city boundary northeastward. The range of weighted odds ratios was comparable to those observed in climatologically similar Chicago and San Francisco.



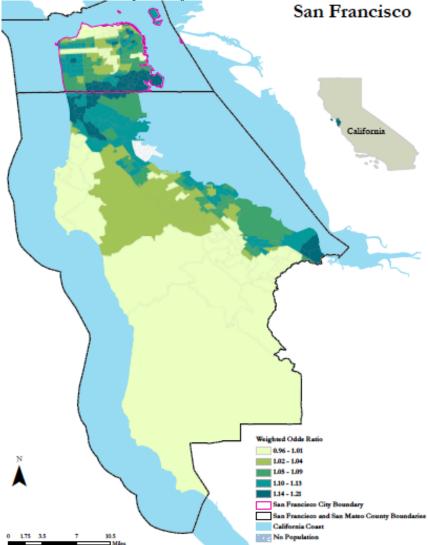
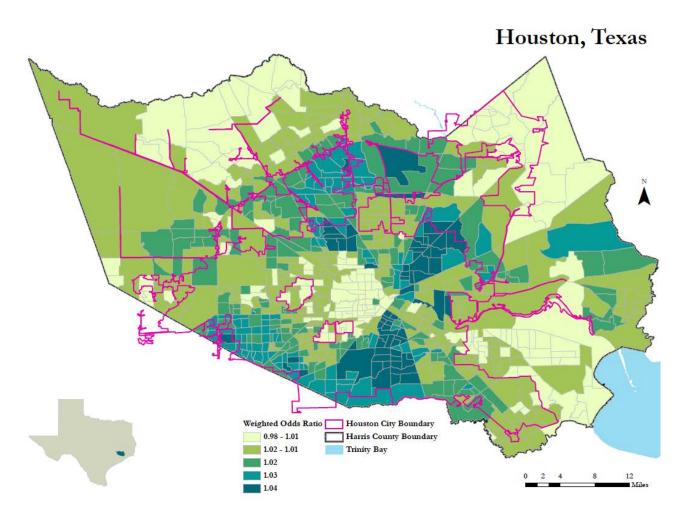


Figure 2.3.d of San Francisco's mapped weighted odds ratios shows a clear pattern with increasing odds observed in the north central portion of the study area, extending to the southeast along the coast. Overall, the map presents what appear to be mostly low odds ratios, with spatially extensive census tracts assuming lower values. However, the scale of the map indicates that the magnitude of effect in the north central and eastern coastal areas was quite high in comparison to the lower estimates that appear to dominate the spatial extent of the map.

Figure 2.3.e. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Houston, TX



In Figure 2.3.e we observed, compared to the other four cities, a less consistent pattern of the weighted odds ratios across the Houston study area. Although the central portion of Houston seemed to have the smallest magnitude associations

between extreme heat and cardiorespiratory death, the outermost northwest, northeast and southeast areas also had lower odds ratios. Dense pockets of census tracts in the south and extending slightly north within the Houston city boundary had the highest weighted odds ratios.

2.7 Discussion

The goal of this analysis was to demonstrate a method for downscaling, to the census tract level, effect estimates of the association between extreme heat and cardiorespiratory death among vulnerable populations, and to map the results for evaluation of spatial patterns. To our knowledge, this is the first attempt to estimate the effect of extreme heat at a neighborhood level, accounting for the spatial distribution of population characteristics at that spatial scale that contribute to heat vulnerability for 20 large US cities.

This approach and the resultant information is valuable because many administrative records of mortality only provide information about the decedents' residence locations at a larger geographic scale, such as the county, and decision-makers and health departments may wish to apply preventive interventions at a smaller spatial scale. The U.S. Centers for Disease Control and Prevention (CDC) Climate-Ready States and Cities Initiative specifically calls for, as part of a framework for preparedness, approaches that address "projecting the disease burden where a health department, as best as possible, estimates or quantifies the additional burden of health outcomes due to climate change to support prioritization and decision-making". [CDC, 44]. Maps are increasingly being used to communicate empirical risk to heat exposure [35, 36, 45]. By identifying spatial patterns that elucidate heat-related vulnerability, this method moves in the direction of combining data on adverse health outcomes with projected ambient temperature or environmental characteristics to better communicate the risks associated with extreme heat exposure.

2.7.1. Spatial heterogeneity

Spatial heterogeneity has been documented in many studies that investigate the effect of extreme heat on human health in US populations. Most of these studies consider the

regional differences across study sites and populations [3, 4, 7, 8, 28-32] and utilize meta-analyses to evaluate modification of heat-health associations by city characteristics (e.g., prevalence of central air conditioning) that vary within and between cities [29]. While such analyses can inform us about potential reasons for heterogeneity of the heat effect between cities, the results are commonly derived from county-level effect estimates. In Philadelphia County, Pennsylvania, a multi-stage analysis concluded that the heat-related mortality distribution associated with high apparent temperatures was not random, but instead was elevated in select ZIP code tabulation areas characterized with low socioeconomic status, high surface temperature and elderly populations [45].

However, rarely are such within-city variations in estimates of the heat effect considered, in part because of limitations in the spatial resolution of health data. We considered neighborhood level spatial extents designated as census tracts, which represent permanent statistical subdivisions of a metropolitan area that contain between 2,500 and 8,000 inhabitants

(http://www.census.gov/geo/www/cen_tract.html). Spatial patterns of the risk of death associated with extreme heat exposure were city-specific, with no distinct pattern observed for cities in the same climate zone (results shown in Appendix A). Chicago, where we observed the most subpopulations experiencing significantly elevated risks of death associated with extreme heat, displayed the highest risks outside western border of the city boundary and in small pockets within the southern portion of the city. The results reflect what was observed during the extensively documented 1995 Chicago heatwave that claimed over 700 excess deaths [19]. Elderly and socially isolated individuals were considered most vulnerable during that heatwave. While the analysis presented here does not account for social isolation, older individuals, particularly in nonwhite populations, were observed to have significantly higher odds of death during extreme heat, suggesting that areas dominated by these subpopulations could benefit from targeted programs to reduce risk during extreme heat.

Previous research has shown a negligible heat effect in southern US cities, where average ambient temperatures are high. In the southern cities presented here, we observed comparably negligible heat risks, but the mapped downscaled odds ratios

illustrated differential spatial patterns of risk, elucidating city neighborhoods where the effect of heat could be more prominent. Inconsistent spatial risk patterns were observed in cities sharing the same climate zone designation, indicating that place-based risk of death due to extreme heat was not explained entirely by climate similarities.

2.7.2. Vulnerable subpopulations

Specific demographic subgroups, particularly elderly and minority populations [28, 42], are known to be experience higher heat-related mortality. Additionally, individual-level characteristics such as education and pre-existing health conditions and community-level characteristics (e.g., high population density, low median income, little green space) confer vulnerability to extreme heat. Because urban neighborhood demographic composition is spatially dynamic, one would expect that fine-scale characterization of the response to extreme heat would be reflected across a cityscape. Individual- and neighborhood-level age, race and gender characteristics were included in this analysis as they were available at the individual level for all 20 study cities. As more of the US population is expected to live in large metropolitan areas, in addition to a growing aging population, the age, race, and gender-specific subpopulations presented here capture a significant portion of populations most vulnerable to extreme heat. Analyses that consider additional individual level characteristics of vulnerability, such as education and health status, would further inform knowledge about city-specific spatial distributions of heat-related risk of death.

2.7.3. Downscaling effect estimates

The time-stratified case-crossover study design permitted the use of higher order interactions in this analysis. The model is flexible, accommodating the complex association between variables that are known to contribute differentially to the heathealth association. Statistically, the three-way interaction terms are a form of standardizing the data so that we could calculate weighted averages of the subpopulation specific odds ratios. Not all subpopulations observed statistically significant odds ratios. In an effort to demonstrate a method for downscaling effect estimates to reflect census tract-level population composition, non-significant effect estimates were included in the calculation of city-specific downscaled odds ratios, which

made use only of the point estimates and not the confidence intervals. Future applications of this method could evaluate the precision of the effect estimates and incorporate those into the calculation of city-specific weighted odds ratios.

2.7.4. Extreme heat

Models of the extreme heat-mortality relationship can be complex, with the definition of extreme heat exposure dependent on the structure of the temperature effect. However, it is well-established that the heat effect increases significantly at higher temperatures [7] and that apparent temperature on the day prior to and the day of death (ATO1) captures the acute effect of heat [46]. By defining extreme heat exposure as being above the month-specific 95th percentile of ATO1, our model accounted for deaths that occurred in earlier and later summer months that might otherwise be excluded an analyses where extreme heat exposure is defined as above a specified percentile of ATO1 across all summer months. Recent analyses have indicated that early summer heat events are often the most deadly [47]. With increasing global average temperatures observed in the last two decades, month-specific exceedances of ATO1 appropriately capture the effect of the increasing trend of temperature.

2.7.5. Limitations

Chloropleth maps that display continuous values, such as the downscaled odds ratios presented here, assume a smoothly varying surface. Because subpopulation demographic information was derived from aggregate census tract measures, we can only, in effect, display semi-discontinuous measures of the heat-health association. By mapping the data based on their natural breaks, we could roughly evaluate the spatial clusters of weighted odds ratios amongst census tracts. Despite the somewhat crude approach to presenting the fine-scale spatial relationships, chloropleth maps are reportedly the map types that are both preferred and more accurately used by epidemiologists than other visualization approaches [48].

Concentrations of air pollutant and ozone were not included in this analysis. Although air pollution does modify the risk of heat-related mortality among vulnerable populations, ambient concentrations at geographic scales consistent with the study

design were not available in the 20 study cities. Ozone's contribution to mortality during heat events is neither well-understood [11] nor spatially consistent [49] and, thus, was not deemed appropriate for inclusion in this analysis [50].

2.7.6. Adaptation planning for heat related vulnerability

The method presented here could allow a practitioner to visually present the public health burden of extreme heat on vulnerable populations in a manner that can be utilized to develop targeted public health initiatives. The US National Climate Assessment defines adaptation as "changes made to better respond to new conditions thereby reducing harm or taking advantage or opportunity" [51]. In financially strapped cities across the US, sweeping adaptation measures are neither likely nor prudent approaches to prepare for adverse health outcomes resulting from a changing climate. By identifying specific locations within an urban area where the impact of temperature could be greatest in terms of human health, local decision-makers and planners can focus and implement limited resources where their impact will be greatest.

2.8 Conclusion

Heat-related mortality is preventable, and targeted interventions can be more effective when officials and community leaders are armed with the knowledge of where specific vulnerable populations are located. Maps, increasingly used to communicate empirical characterizations of public health risks, can provide visual displays of fine-scale estimates of the risk of death associated with extreme heat. Downscaled estimates of the effect of extreme heat on cardiorespiratory mortality displayed spatial heterogeneity across the 20 study cities. While the tract-specific odds ratios are only estimates of how heat may affect mortality differentially according to population vulnerability, they are based on epidemiologic effect estimates from location-specific data. Evaluation of how these estimates correspond with odds ratios computed using health outcome data that identifies the residential location is needed, and we plan to conduct such a validation study using Detroit-based data as a next step. Overall, we feel these analyses contribute to place-based assessments of heat-related mortality among vulnerable subpopulations,

and have the potential to inform targeted climate adaptation plans and public health
interventions.

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Chapter 3.

Fine-scale heat vulnerability mapping for Detroit, Michigan, USA

3.1 Abstract

BACKGROUND: Heat-related deaths are preventable and maps showing areas where people may be more vulnerable to heat have received attention as potential planning tools for states and municipalities in preparing for heat events. Assessments of heat vulnerability are often done by combining data on various components of vulnerability (e.g., demographic, socioeconomic and health characteristics of the population, natural and built environmental conditions) into a composite index that can be assigned to a geographic unit (e.g., neighborhood) and visually displayed for research and application. Thus, it is crucial to evaluate the commonly used methodology and provide guidance on how to interpret such composite heat vulnerability indices.

OBJECTIVE: The goal of this analysis was to create a fine-scale composite heat vulnerability index for Detroit, Michigan, USA and critically evaluate the process, focusing particularly on the behavior of the index when differing combinations of variables are used in its construction.

METHODS: Fine-scale land cover classification, ZIP-code level disease prevalence data, and census variables were used in a principal component analysis (PCA) to create 21 heat vulnerability indices (HVIs) for census block groups in Detroit, Michigan. Spearman correlations were used to evaluate similarities across HVIs. Geographic information systems (GIS) were used to map and assess spatial patterns of vulnerability. Block groups that most frequently were identified as having a top 5% HVI score were mapped.

RESULTS: HVIs for the 913 census block groups showed general patterns of vulnerability, which differed depending on the combinations of input variables used in the PCA. Spatial patterns of vulnerability were not explicitly consistent across HVIs. However, among the 5% of block groups identified as having the highest HVI scores, the downtown areas of Detroit were ranked as most vulnerable across multiple HVIs. Spatial patterns of vulnerability were dependent on the combination of input variables including in the PCA.

CONCLUSIONS: Overall inconsistent spatial patterns suggest that the HVI is sensitive to the input variables and must be interpreted with caution. Such maps can serve as preliminary screening tools for identifying potential spatial pockets of heat vulnerability, but local knowledge about environmental characteristics, population vulnerabilities and intervention points, and validation of the different HVIs with health outcome data that can choose the most predictive indices are important next steps to optimize the impact of targeted intervention.

3.2 Keywords

Heat vulnerability index, principal component analysis, vulnerability

3.3 Abbreviations

ACS: American Community Survey

CMS: Center for Medicare and Medicaid Services

GIS: Geographic information system

HVI: Heat vulnerability index

NLCD: National Land Cover Database

PCA: Principal component analysis

3.4. Background

As regions, states and municipalities are faced with responding to and preparing for the impact of a changing climate on human health, research that contributes to decision-making and policy development for climate change adaptation at a scale relevant to communities is needed. Such decisions and policies rely heavily on vulnerability assessments, many of which hinge on public health research. The United States (U.S.) National Research Council (NRC) defines vulnerability to climate change as "the capacity to be harmed" via the magnitude of changes, underlying factors that contribute to sensitivity (e.g., social, cultural, economic, geographic, ecologic) and the ability to avoid, prepare for and respond to impacts at various levels [1]. Numerous studies have demonstrated the adverse impact of weather on human health. Heat, in particular, was responsible for more weather-related deaths in the U.S. from 2002 to 2011, on average, than floods, tornadoes, hurricanes, earthquakes and lightning [2, 3]. Research suggests that heat-related morbidity and mortality, and vulnerability to extreme heat, are dependent on characteristics of the heat event, and the health status and geography of the affected population [2, 4-10].

The relationship between heat and health has been extensively studied and documented in environmental epidemiological literature. Studies consistently show that individual and community-level characteristics influence one's vulnerability to heat. Whether individuals have a pre-existing health condition such as cardiovascular disease, respiratory disease, diabetes, cerebrovascular disease, renal disorder, nervous disorder and/or mental health condition can enhance risk [8, 11-13]. Demographic and socioeconomic characteristics (e.g., older populations [12, 14-16]; minority populations [8, 17]; low-income [12]; those who are high school educated or less [16, 17]; the unmarried [15, 18]), as well as social factors (e.g., living alone, having limited access to transportation [19]), have been associated with increased risk of death during heat events. Housing characteristics, including central air conditioning in the home [20], can protect individuals from heat.

Moving beyond individual-level characteristics, research suggests that community-level variables can reliably predict risk to heat-related health effects. The area of residence is among these variables. People living in urban versus rural or suburban areas are more at risk for heat-

related mortality [21], in part due to the urban heat island (UHI) effect. The UHI effect occurs when higher ambient temperatures are observed in urban areas, compared to surrounding suburban and rural areas [22, 23] and is also characterized by increased nighttime ambient temperatures resulting from the slow release of energy retained by impervious materials [24]. The effect is due to a confluence of urban characteristics including a higher percentage of impervious, heat-absorptive surfaces; less vegetation (or green space); waste heat from buildings and vehicles; and overall more energy consumption per capita [25, 26] that alter the surface energy balance [23]. Plants and proximity to water may reduce heat exposure in microclimates within urban areas, however, even when an UHI exists. Urban vegetation, including tree canopy cover, can reduce ambient temperatures via increased shading and evapotranspiration [27], indirectly reduce greenhouse gas emissions [28] and may also play a role in protecting against heat-related mortality [29]. Measures of distance to large bodies of water (e.g., rivers, lakes), in cities like Detroit and Chicago, provide improved estimates of the intra-urban heat island by accounting for reduced temperature from nearby water sources [30-32]. The various contextual characteristics that contribute to changes or increases in the UHI can make it challenging to identify one or two characteristics that could serve as intervention points. However, recent research found that urban poor populations and surface UHI measurements were significant indicators of risk during extreme heat events [33] highlighting that there is much to be explored about individual level and community level characteristics of heat-related vulnerability.

Heat-related deaths are preventable [34, 35] and local governments are being called upon to implement or strengthen programs and systems that identify and respond to heat-related risks in a changing climate [36]. Vulnerability maps have received attention as potential planning tools for states and municipalities in preparing for heat events. Reid and colleagues [37] created a composite heat vulnerability index comprised of individual- and community-level characteristics representing factors that contribute to heat vulnerability. The index was calculated at the census tract level (approximately 4,000 inhabitants) in large cities and mapped to illustrate variability across the U.S. Using data at the census tract spatial scale can mask intra-urban variation of factors contributing to vulnerability, including vegetation, population distribution, and disease prevalence.

Composite indices, such as the Reid at al index, are products that combine multidimensional data and create scores that reflect the relative relationships in the data. Indices are useful in identifying spatial patterns of relative vulnerability. For instance, the Carstairs [38] and Townsend [39] indices were among the first used to measure and identify areas of relative deprivation in the United Kindgom. Indicators on social class, car ownership, male unemployment, overcrowding (Carstairs), and home ownership (Townsend) comprise the indices. The British Department for Communities and Local Government has been creating a similar deprivation index since 1999 to rank small areas across the country [40]. Cutter and colleagues created the Social Vulnerability Index (SoVI) which included myriad of variables reflecting comunity-level demographics, socioeconomic status, population structure, and the built environment to identify social vulnerability to environmental hazards for counties across the U.S. [41]. The results demonstrated spatial patterns of vulnerability in eastern cities, the Mississippi delta, and southern Texas. The SoVI captures the social components that contribute to vulnerability to heat, and has provided a framework for considering social components that may contribute to heat vulnerability.

Much of what is known about heat-related vulnerability stems from retrospective analyses in which data were extracted from death certificates and hospital admissions records, archived temperature data, census and other broad population-based surveys (e.g., the Behavioral Risk Factor Surveillance Survey (BRFSS) http://www.cdc.gov/brfss/; American Housing Survey (AHS) http://www.census.gov/housing/ahs/). Analyses using these data sources must often limit the spatial and temporal resolutions to the coarsest spatial scale across variables, resulting in estimates reflecting the impacts of heat on populations of thousands to millions; for example, at the county level.

A vulnerability index can be created using varying levels of spatial-scale information on demographic and environmental characteristics. Population characteristics on human health status are one variable clearly linked to vulnerability, as is local vegetation and green space. However, health data is difficult to acquire, making it challenging to adequately account for population vulnerability to heat in the absence of information on the underlying health status of a population, especially at finer spatial scales. Green space, a commonly used indicator assumed to reduce heat vulnerability, has been calculated from satellite imagery, vegetation

indices, and measures of impervious surface [42]. City-specific indices of heat vulnerability that incorporate finer-scale (census block group, 600-3,000d inhabitants) environmental, demographic and health information can communicate the spatial distribution of heat vulnerability and have the potential to aid municipal heat-health prevention and planning.

A composite index can be calculated, interpreted and applied at the creator and/or user's discretion, typically for detailed, place-specific applications. Because the construction and interpretation of composite indices can vary, in the interest of communicating spatial vulnerability for both research and application it is critical to evaluate the commonly used methodology and provide guidance on how to interpret composite heat vulnerability indices.

3.5 Objectives

In this paper, we assess whether the creation of a heat vulnerability index (HVI), comprised of fine-scale demographic, environmental, health and socioeconomic variables, depicts explicit spatial patterns of heat-related vulnerability in Detroit, Michigan, USA. We further evaluate if the spatial patterns hold when different environmental (e.g., green space) and health data are used in the creation of the index, to investigate how robust the PCA method is in determining heat vulnerability within a metropolitan area.

3.6 Methods

3.6.1. Study location

The City of Detroit and neighboring southeast Michigan cities have been planning for heat events since 2007 under the Office of Homeland Security's all-hazard plan. The plan is led by the local health department and includes input from a heat committee comprised of citywide partners. Heat preparedness programs in place include an established network of cooling centers, outreach and education via community-based organizations, utility assistance programs, and a community emergency response team [43]. Although Southeastern Michigan is a northern U.S. area known for harsh winters, the area does experience periods of prolonged heat. The demographic and socioeconomic profiles of the resident population reflect known heat vulnerabilities. Detroit represents a city in the beginning stages of incorporating adaptation programming and planning, and has expressed interest in calculating a heat

vulnerability index. Thus, using data from this area is useful to illustrate a methodology and may also have practical application. Two cities, Highland Park and Hamtramck, are located within the boundary of Detroit, and these cities were included in the present analysis to maintain continuity across the city.

3.6.2. Heat Vulnerability Index (HVI) Data

Population health status

Because of the dearth of reliable population disease prevalence statistics at spatial scales finer than the state or county, we decided to use hospital admissions data as a proxy for ZIP-code level disease prevalence among older persons in Detroit. Hospital admissions were acquired from the Center for Medicare and Medicaid Services (CMS) for Detroit, Michigan. Hospital admissions from 2006 were used in this analysis to maintain temporal consistency with the other data. Medicare covers nearly 98% of all individuals over the age of 65, providing comprehensive health data on the elderly population [44]. The University of Michigan Institutional Review Board approved use of these data. ZIP codes in which patients lived were extracted from the admissions records. Census block groups were assigned a ZIP code where at least 90% of the block group was contained within the 2006 ZCTA boundaries (http://mcdc.missouri.edu/websas/geocorr12.html.

International Classification of Disease codes, version 9 (ICD-9), were used to define specific causes of admissions if they were listed as the primary admission cause on the Medicare billing record, as follows: All-cause (all admissions), cardiovascular (390-429); diabetes (250); renal (580-589); respiratory (480-492,494-496); that is, all respiratory diseases except asthma, 493).

Vegetation, green space, and water proximity

Land cover data was derived from three sources. The first source was an aerial photograph of the metropolitan Detroit area. The 2005 one-meter resolution aerial photograph was acquired from the 2005 Southeast Michigan Council of Governments (SEMCOG) Imagery product (http://www.semcog.org/Aerials_2005_Imagery_Project.aspx). Land cover classifications included proportion of impervious surface, bare earth, open space, trees, and water. To create vegetation variables consistent with increased hypothesized vulnerability to heat, we calculated

"non vegetation" as $[1 - \Sigma(\text{open space} + \text{trees} + \text{water})]$ within a block group. As many cities are adapting tree planting programs as a strategy for increasing green space, we wanted to include a measure of vegetation that was only tree-specific. A second vegetation variable was constructed from the aerial photograph-derived land cover classification, "non-trees", and was calculated as [1 - (proportion of trees)]. The second source is the 2006 National Land Cover Database (NLCD) impervious surface layer. The 30-meter resolution product has been used in heat-health studies to characterize land cover that is not vegetation. In this analysis, the impervious surface layer served to represent the common non-green space characterization. Impervious surface was extracted and recorded as a proportion across a block group. (http://www.mrlc.gov/nlcd06_leg.php). Lastly, we used the 30-meter 2001 NLCD tree canopy layer to calculate the proportion of a block group that is covered by tree canopy. The 2001 dataset is the only publicly-available tree canopy assessment for the City of Detroit [45].

Distance to water was calculated as a straight-line distance from the Detroit River (ESRI, 10.41) to the centroid of each census block group. The measurements were scaled to have a value between 0 and 1, so that 1 indicated the furthest distance from the river, with further distances hypothesized to confer higher vulnerability to heat exposure.

Demographic variables

Demographic variables were extracted from the American Community Survey (ACS) 5-year estimates for 2006-2010 for the block groups in Detroit, Michigan (www.census.gov/acs). Variables (again defined so that higher values would be related to the hypothesized greater heat vulnerability) included proportions of the following groups of individuals living in a census block group: over the age of 65; living alone; individuals over the age of 65 and living alone; with less than a high school education; living at or below the poverty level; and minority status. Minority status was based on the U.S. EPA Office of Environmental Justice's definition of minority that includes Hispanics, Asian-Americans and Pacific Islanders, African Americans, and American Indians and Alaskan Natives (http://www.epa.gov/region2/ej/guidelines.htm). We calculated the proportion of the population who met the EPA definition of minority status for each census block group as $[1-\Sigma(\text{proportion all white, non-Hispanic population)}]$.

3.6.3. Calculation and mapping of heat vulnerability index

One of our goals was to utilize principal component analysis (PCA), a method that has been used in other heat vulnerability index projects, to apply to a finer spatial scale data for Detroit, and assess how robust the index rankings were to inclusion of different variables. The purpose of conducting a PCA is for data reduction, allowing one to identify underlying factors in a dataset that explain most of the variation within the dataset; in this case, variation in heat vulnerability. Specifically, we were interested in how various measures of green space (or, conversely lack of green space) and health status contributed to differences in the index. In total, 21 indices were produced with variation on the health and non-vegetation variables, with all other variables included in the index remaining constant. Using the methodology established in Reid, et al [37], we created the 21 indices from the pre-specified variable combinations. Based on previous literature, we calculated the variables in a form for which a higher value would be related to higher hypothesized vulnerability (e.g., higher elderly population, higher non-green space, etc.), so the interpretation would be consistent.

First, Spearman correlations at the census block group level were run for all variables that would be used in calculating the indices. We then performed a PCA on the correlation matrix using varimax rotation (varimax does not allow the factors to be correlated) to create independent factors. The number of factors retained were determined via a visual examination of the scree plot [46] and using Kaiser criteria where eigenvalues > 1 [47]. Although variance explained by the factors can be included in determining the number of factors retained, we refrained from deciding on a cutoff point, as we assumed most users of the PCA method for creating an HVI would follow the suggestions of the statistical software (i.e., eigenvalues > 1). Factor scores were calculated and normalized to have a mean of 0 and a standard deviation of 1, and then divided into six categories based on standard deviations. Factor scores were summed across factors for each census block group and mapped. Spatial patterns were visually assessed and reported. SAS version 9.3 (Cary, NC) and ESRI 10.1 (Redlands, CA) were used for the statistical analysis and spatial mapping.

3.6.4. Vulnerability index comparisons

Since no standard method for comparing HVIs exists, we evaluated how the indices changed as a result of differing input variables in four ways. First, we conducted Spearman rank

correlations of each HVI compilation using the calculated HVI scores for the census block groups. We hypothesized that the indices where the non-green variable came from different sources (e.g., aerial photograph, NLCD) would have low correlations. Second, we visually assessed the spatial patterns of the mapped HVI scores. We hypothesized that the spatial patterns of vulnerability would remain consistent regardless of differing health and/or nongreen measures used in the index calculation. Third, to explore correlation and spatial patterns, we selected four of the most distinctly different HVIs and compared the number of factors retained in the PCA and the variance explained by those factors, and identified ways to characterize the factors. We hypothesized that the computed factors in each index would be comprised of the same variables, would explain the same amount of variance, and could be characterized the same way. Lastly, to evaluate whether specific block groups were consistently scored with higher vulnerability index values, we identified which block groups across the 21 HVIs were in the top 5% of the index scores. The frequency of ranking in the top 5% was summed and mapped to identify block groups that ranked highly on all index calculations. We hypothesized that the same block groups would consistently be identified in the top 5% for the HVIs calculated with differing non-vegetation variables (e.g., aerial photograph-derived, impervious surface, tree canopy).

3.6.5. Vulnerability index value comparisons with downscaled effect estimates

The previous chapter of this dissertation presented a method to downscale county-level effect estimates of the association between extreme heat and cardiorespiratory-cause deaths to the census tract level. By identifying vulnerable populations defined by their age, race, and gender as provided on death certificates and using census tract-level demographic information, I computed city-specific weighted odds ratios. The weighted odds ratios were mapped and presented at the tract level. To evaluate the performance of the downscaling methodology presented in Chapter 2 and the fine-scale HVIs presented in this chapter, Spearman correlations were used to test the association between the Detroit specific tract-level downscaled weighted odds ratio of the odds of cardiorespiratory death and the four HVIs presented here. We hypothesized that there would be a positive association between the index values for all of the HVIs and the tract-level weighted odds ratios.

3.7 Results

In the City of Detroit, 918 census block groups were present; 5 did not have a population reported in the census and were therefore were not included in the HVI calculation. The sample size for computing the indices was thus 913. For the disease prevalence, Medicare data were available at ZIP code level (n = 31). Descriptive statistics for the variables used in computing HVIs are found in Table 3.1. On average, 91% of the residents of Detroit block groups were members of minority populations, and 35% were living at or below the poverty level. Smaller proportions of the Detroit population were of older ages, lived alone, and had less than a high school education. Mean values of land cover in the block groups differed by data source. The aerial photograph-derived 'non-trees' was comparable to the 2006 NLCD impervious surface measurement, with the mean non-tree and impervious surface coverage being 67% and 60%, respectively. The 2001 NLCD tree canopy data showed an average of 95% of non-trees in the City of Detroit. For the 31 zip codes for which health status information was available, hospital admissions rates (our proxy for prevalence among people over 65) were highest for all-cause diseases, followed by cardiovascular, respiratory, renal diseases, and diabetes.

Table 3.1. Descriptive statistics for variables used in computing census block group level (n=913) heat vulnerability indices (HVIs) for Detroit, MI, USA.

Category	Data Source	Variable	Minimum	Mean	Maximum	Std Dev
Socioeconomic	American Community Survey ^a	Over 65	0.00	0.12	0.51	0.08
	5-year estimates (2006 - 2010)	Over 65, living alone	0.00	0.04	0.43	0.05
		Living alone	0.00	0.14	1.00	O.11
		Minority	0.03	0.91	1.00	0.14
		Less than HS education	0.00	0.14	0.49	0.09
		Below poverty line	0.00	0.35	0.88	0.19
Health status	Medicare, Hospital admissions b	All-cause	22.71	37.26	53.52	5.46
	ZIP-code, $n=31(2006)$	CVD	5.43	9.62	13.04	1.40
		Respiratory	1.72	3.48	4.82	0.63
		Renal	0.61	1.89	3.92	0.55
		Diabetes	0.00	0.36	0.86	0.14
Land cover	ESRI 10.4 River Shapefile (2010) °	Distance from water	0.00	0.41	1.00	0.23
	Aerial photograph, 1-meter ⁴	Non-vegetation	0.10	0.48	1.00	0.14
	SEMCOG (2005)	Non-trees	0.19	0.67	1.00	0.15
	Impervious layer, 30-meter ^e NLCD (2006)	Impervious surface	0.00	0.60	0.92	0.11
	Tree canopy layer, 30-meter ^t NLCD (2001)	Non-trees (tree canopy)	0.00	0.95	0.99	0.07

^a Proportion of population for each census block group

b Disease-specific prevalence calculated using CMS hospital billing records for cause-specific primary admission divided by CMS Old-age, Survivors, and Disability Insurance (OASDI) ZIP code populations http://www.ssa.gov/policy/docs/statcomps/oasdi_zip/2010/index.html

[°] Relative measure of straight-line distance (miles) from centroid of census block group to west Detroit River boundary

^d Proportion of 1-meter area classified as non-vegetation = $[1 - \Sigma(\text{open space} + \text{trees} + \text{water})]$, or non-trees = [1 - (trees)]

[°] Proportion of 30-meter area classified as impervious http://www.mrlc.gov/nlcdo6_leg.php

 $^{^{}t}\ \ Proportion\ of\ 3o\text{-meter}\ area\ classified\ as\ having\ tree\ canopy\ coverage\ http://www.mrlc.gov/nlcdo1_data.php$

Table 3.2 presents a matrix of the variable combinations used to create the 21 indices. Spearman correlations among variables used in the PCA analyses are shown in Table 3. 3. We observed significant and moderate correlations between most of the variables, most notably between non-vegetation variables from the aerial photograph, NLCD impervious surface and tree canopy sources. We observed significant correlations between demographic variables, but the magnitudes of the correlations were relatively low, except for populations over 65 and over 65 and living alone (0.62).

Table 3.2. Matrix of variables available used for heat vulnerability index (HVI) creation for Detroit, MI, USA, by combination

HVI Name	Land Cover Classification Aerial Impervious Surface Tree Canopy		Tree Canopy		Socioecon	omic V	ariable $s_{ m AC}$	S 5 yr est. 2006-20	010	Health Variables _{Medicare Prev 2006}						
	Distance to water	Non- Green	Non- Vegetation	Non- Trees	% I mpervious	Non Trees	Over 65	Over 65, Living Alone	Living Alone	Minority	Less than HS Diploma	Below Poverty Line	All-cause (CVD Respir	tory Re	nal Diabete
IIII Dat	Water		regettation	11003			37	37		v	TIO DIPIONIA					v
HVI_Det	37	X	37				X	X 37	X	X	X X	X	37			X
HVI_Det_1	X		X	37			X	X	X	X	X	X	X			
HVI_Det_2	X		77	X	I		X	X	X	X	X	X	X	77		
HVI_Det_3	X		X	**			X	X	X	X	X	X		X		
HVI_Det_4	X			X	l		X	X	X	X	X	X		X		
HVI_Det_5	X		X				X	X	X	X	X	X		X		
HVI_Det_6	X			X	l		X	X	X	X	X	X		X		
HVI_Det_7	X		X		_		X	X	X	X	X	X				X
HVI_Det_8	X			X			X	X	X	X	X	X				X
HVI_Det_9	X		X				X	X	X	X	X	X				X
HVI_Det_10	X			X			X	X	X	X	X	X				\mathbf{x}
HVI_1N					X		X	X	X	X	X	X	X			
HVI_2N					X		X	X	X	X	X	X		\mathbf{X}		
HVI_3N					X		X	X	X	X	X	X		X		
HVI_4N					X		X	X	X	X	X	X				X
HVI_N					X		X	X	X	X	X	X				X
HVI_1TC						X	X	X	X	X	X	X	X			
HVI_2TC						X	X	X	X	X	X	X		X		
HVI_3TC						x	x	x	x	X	X	x	_	X		
HVI_4TC						x	X	X	X	X	X	x				X
HVI_TC						X	X	X	X	X	X	X				X
Land Cover (A	verial)				Land Cover (NLCD)											
	- (Σ Imperviou	s. Bare)			%Impervious = propor	tion of imperviou	ıs cover									

NonVegetation=1-(\sum Trees, Open Space, Water) NonTrees = 1 - (Trees_proportion)

NonTrees = 1 - % Tree canopy

Table 3.3. Spearman correlations between scored heat vulnerability index (HVI) values, by census block group (n = 913) across HVIs referenced in Table 2.2 matrix, for Detroit, MI, USA.

					_								D	isease Prevale	nce	
	Over 65 yrs	Over 65, living alone	Living alone	Minority	Less than HS education	Below poverty line	Distance from water	Non- vegetation	Non- trees	Impervious surface	Non-trees (tree canopy)	All-cause	CVD	Respiratory	Renal	Diabetes
Over 65 yrs	1.00															
Over 65, living alone	0.62 **	1.00														
Living alone	0.38 **	0.60 **	1.00													
Minority	0.20 **	0.09 *	0.08 *	1.00												
Less than HS																
education	0.19 **	0.10 *	0.03	-0.08 *	1.00											
Below poverty line	-0.18 *	-0.07*	-0.03	0.03	o.30 **	1.00										
Distance from water	0.03	-0.04	-0.06	0.14 **	-o.3o **	-O.21 **	1.00									
Non-vegetation	-0.02	0.06	0.07 *	- 0.14 **	0.23 **	0.112 **	-o.38 **	1.00								
Non-trees	-0.04	0.06	0.12 **	-0.16 **	0.29 **	0.213 **	-0.61 **	0.81 **	1.00							
Impervious surface	0.04	0.10 *	0.08 *	-0.06	0.21 **	0.101 *	-0.24 **	0.92 **	0.65 **	1.00						
Non-trees (tree																
canopy)	-O.O1	0.09 *	0.07 *	-O.11 **	0.20 **	0.16 **	-0.44 **	0.72 **	0.79 **	o.66 **	1.00					
All-cause	-0.22 **	-0.10 *	-0.03	- 0.13 **	0.03	0.21 **	-o.38 **	0.04	0.20 **	-0.02	0.04	1.00				
CVD	-0.16 **	-0.06	-0.04	-0.04	0.05	0.23 **	-0.47 **	0.09 *	0.28 **	0.03	0.19 **	0.87 **	1.00			
Respiratory	-0.32 **	-0.17 **	-0.08 **	-0.35 **	0.12 **	0.19 **	-0.46 **	0.03	0.25 **	-0.08 *	0.07 *	0.74 **	0.65 **	1.00		
Renal	0.04	0.09 *	0.14 **	-0.07 *	0.28 **	0.29 **	-0.51 **	0.37**	0.50 **	0.34 **	0.45 **	0.34 **	0.38 **	0.31 **	1.00	
Diabetes	-0.07 *	-0.07 *	-0.07 *	0.16 **	-0.04	0.09 *	-o.17 **	-0.05	0.02	-0.06	-0.06	0.57 **	o.58 **	0.14 **	0.05 **	1.00

Statistically significant

^{*} $p \le 0.05$

^{**}p ≤ 0.01

PCAs were run for all 21 combinations of variables displayed in Table 2.2. The HVI value assigned to each block group was calculated by summing standardized factor scores. The scale of the index is dependent on the number of factors retained from the PCA (based on the Kaiser criterion and scree plots). For instance, a PCA result that retained three factors would have a potential scale of 3-18, whereas an index with four retained factors would have a potential scale of 4-24. Standardized factor scores were then computed and mapped (Appendix B) for the 913 census block groups in Detroit. Because of the large number of block groups, visual discernment of differences between the computed indices among the 21 maps was difficult.

However, the Spearman correlations calculated between each HVI (Tables 3.4.a-c) allowed us to identify which indices were statistically similar to each other without initially relying on visual interpretation. Correlations over 0.80 were considered 'high', suggesting similarities between the HVI values calculated for the 913 Detroit census block groups. Conversely, we considered correlations below 0.40 to be 'low', suggesting differences between HVI values calculated for the 913 Detroit census block groups. Table 3.4.a shows that about half of the HVIs computed using aerial photograph-based land cover classifications were highly correlated, suggesting that these HVIs were relatively similar. Table 3.4.b. shows both high and low correlations between the HVIs computed using NLCD impervious surface and the HVIs computed with the aerial photograph-based non-vegetation. HVI_3N and HVI_Det_6 had the lowest correlation across all HVIs; they both included respiratory disease as the health variable, but differed in non-vegetation in that HVI_3N included percent impervious surface and HVI_Det_6 included the proportion of aerial photograph-derived non-tree classification. HVI_3N also had the most 'low' correlations of all the 21 HVIs, as well as no 'high' correlations. Additionally, in Table 3.4.c., HVI_2TC, calculated using the NLCD tree canopy land cover classifications, had 'low' correlations with both HVI_Det_6 and HVI_3N.

Tables 3.4(a-c). Spearman correlations between all heat vulnerability indices (HVIs) calculated using aerial-based land cover classifications, the National Land Cover Dataset (NLCD) 2006 percent impervious surface, and the 2001 NLCD tree canopy coverage for Detroit, MI, USA.

Table 3.4.a. Spearman correlations between heat vulnerability indices (HVIs) calculated using aerial-based land cover classifications, Detroit, MI, USA.

				ŀ	lealth Variabl	es Included in	HVI Calculati	on			
	Diabetes	All-o	cause	Cardiovas	cular-cause	Resiprat	ory-cause	Renal	-cause	Dia	betes
	HVI_Det	HVI_Det_1	HVI_Det_2	HVI_Det_3	HVI_Det_4	HVI_Det_5	HVI_Det_6	HVI_Det_7	HVI_Det_8	HVI_Det_9	HVI_Det_10
HVI_Det	1.00	0.80 *	0.77	0.76	0.77	o.83 *	0.73	0.82*	0.65	0.86 *	0.80 *
HVI_Det_1		1.00	0.90 *	0.86 *	o.88*	0.81 *	0.57	0.82 *	0.71	0.81 *	0.77
HVI_Det_2	:		1.00	0. <i>7</i> 6	0.93 *	0.77	0.56	0.79	o.83 *	0.84 *	0.82 *
HVI_Det_3	}			1.00	0.79	0.76	0.55	0.77	0.57	0.74	0.72
HVI_Det_4	ļ				1.00	O. <i>77</i>	0.56	0.78	0.82*	o.86 *	0.84 *
HVI_Det_5	i					1.00	0.81*	0.91 *	0.66	0. <i>7</i> 5	0.66
HVI_Det_6	•						1.00	0.80*	0.50	0.58	0.50
HVI_Det_7	,							1.00	0.70	0.74	0.68
HVI_Det_8	}								1.00	0.78	O. <i>77</i>
HVI_Det_9	•									1.00	0.94 *
HVI_Det_1	o										1.00

^{*} denotes 'high' correlation, > 0.80

All correlations have $p \le 0.05$

Even value HVIs calculated with non-vegetation variable

Odd value HVIs calculated with no-tree variable

Table 3.4.b. Spearman correlations between heat vulnerability indices (HVIs) calculated

using NLCD (2006) impervious layer, Detroit, MI USA

0 \		., ,	,	
	HVI_1N	HVI_2N	HVI_3N	HVI_4N
HVI_Det	0.79	0.66	0.36 ±	0.82 *
HVI_Det_1	0.97*	0.79	0.47	0.79
HVI_Det_2	0.91 *	0.70	0.63	0.74
HVI_Det_3	0.85 *	0.92 *	$0.31 \pm$	0.75
HVI_Det_4	0.89*	0.74	0.61	0.73
HVI_Det_5	0.80 *	0.63	0.34 ±	0.91 *
HVI_Det_6	0.57	0.41	$0.13 \pm$	0.84 *
HVI_Det_7	0.81 *	0.65	$0.35 \pm$	0.94 *
HVI_Det_8	0.72	0.50	0.78	0.62
HVI_Det_9	0.82*	0.70	0.57	0.70
HVI_Det_10	0.79	0.71	0.59	0.62
HVI_1N	1.00	0.80 *	0.48	0.78
HVI_2N		1.00	$0.29 \pm$	0.62
HVI_3N			1.00	$0.27 \pm$
HVI_4N				1.00
		_		

^{*} denotes 'high' correlation, > 0.80

All correlations have $p \le 0.05$

HVI 1N: Calculated with all-cause disease

HVI 2N: Calculated with cardiovascular disease

HVI 3N: Calculated with renal disease

HVI 4N: Calculated with diabetes

[±] denotes 'low' correlation, < 0.40

Table 3.4.c. Spearman correlations between heat vulnerability indices (HVIs) calculated using NLCD (2001) tree canopy layer, Detroit, MI, USA.

	HVI_1TC	HVI_2TC	HVI_3TC	HVI_4TC
HVI_Det	0.61	0.56	0.61	0.66
HVI_Det_1	0.74	0.62	0.51	0.68
HVI_Det_2	0.80 *	0.63	0.59	0.75
HVI_Det_3	0.69	o . 73	0.47	0.65
HVI_Det_4	$0.82 \pm$	0.69	0.62	0.78
HVI_Det_5	0.52	0.47	0.62	0.64
HVI_Det_6	0.33 ±	$0.37 \pm$	0.69	0.57
HVI_Det_7	o . 56	0.52	0.64	0.70
HVI_Det_8	0.75	0.53	9.68	0.78
HVI_Det_9	0.78	0.68	0.65	0.76
HVI_Det_10	o . 83 *	o . 73	0.66	0.79
HVI_1N	0.76	0.64	0.52	0.69
HVI_2N	0.71	o . 8o *	0.41	0.62
HVI_3N	0.64	0.34 ±	0.42	0.53
HVI_4N	0.49	0.46	0.62	0.62
HVI_N	0.80 *	0.69	0.65	0.77
HVI_1TC	1.00	o . 8o *	0.62	0.84 *
HVI_2TC		1.00	0.59	0.78
HVI_3TC			1.00	o . 83 *
HVI_4TC				1.00

^{*} denotes 'high' correlation, > 0.80

All correlations have $p \le 0.05$

HVI 1TC: Calculated with all-cause disease

HVI 2TC: Calculated with cardiovascular disease

HVI 3TC: Calculated with renal disease

HVI 4TC: Calculated with diabetes

Based on the Spearman correlation and mapped spatial analysis, in Table 3.5 we provide a detailed comparison of the PCA results for the four most distinct HVIs. Although HVI_Det_8 did not have remarkably high or low correlations with the other HVIs, we include it in the comparison because it includes the proxy of renal disease prevalence in the population, which is increasingly being identified as a strong predictor for vulnerability during heat events [48]. Except for HVI_3N, which had four factors, the other selected indices had three factors retained from the PCA. The percent variance explained when three or four factors are retained is included to illustrate how the inclusion (or exclusion) of an additional factor can affect how much of the dataset's

[±] denotes 'low' correlation, < 0.40

variance is explained by the factors. The main difference between HVI_3N and the other HVIs in Table 3.5 is that the non-vegetation measure is based on the NLCD calculation of percent impervious. The only other PCA that retained four factors was for HVI_1N where all-cause disease prevalence was the health data variable (results not shown). In three of the four HVIs presented in Table 3.5, the first factor, which explains most of the variation in the dataset, is comprised of the elderly and those who live alone. Except for HVI_Det_8, the first factor in the other 20 HVIs was elderly/isolation which generally accounted for somewhere between 24 and 26% of the variance of the dataset (results not shown). Second and third factors did not remain consistent across the HVIs. Health variables and location (e.g., distance to water) were grouped to the same factor and were interchangeably included with socioeconomic variables and minority status. Nonvegetation variables also did not follow a factor-loading pattern. Overall, the factors explained between 60 and 73 percent of the variance in the data.

Table 3.5. Comparison of principal component analysis (PCA) results for four selected heat vulnerability indices (HVIs) for Detroit, MI, USA.

	HVI Detroit 6	HVI Detroit 8
Number of components selected	3	3
% variance explained (3 components)	61.45	62.80
% variance explained (4 components)	71.88	73.41
Component interpretation	Factor 1. Elderly/isolation (25.8)	Factor 1. Location, green space, health (29.2)
(% variance explained)	Factor 2. Socioeconomic, location and health (23.6)	Factor 2. Elderly/isolation (22.2)
	Factor 3. Minority and green space (12.1)	Factor 3. Socioeconomic (11.4)
	HVI Detroit 3N	HVI Detroit 2TC
Number of components selected	4	3
% variance explained (3 components)	59.55	60.17
% variance explained (4 components)	70.79	70.87
Component interpretation	Factor 1. Elderly/isolation (25.6)	Factor 1. Elderly/isolation (25.8)
(% variance explained)	Factor 2. Minority, location, health (22.5)	Factor 2. Location, green space, health, poverty (22.4)
	Factor 3. Socioeconomic (11.5) Factor 4. Green space (11.2)	Factor 3. Minority status, education (12.0)

3.7.1. Spatial patterns of vulnerability

Figures 3.1. (a-d) are the heat vulnerability maps for the four HVIs listed in Table 3.5. Index values are displayed as quintiles so that patterns of vulnerability are more easily discernible amongst the four maps. Data sources and factor loadings are also presented in each figure. The top 5% census block groups for each HVI map are displayed with cross hatch patterns. Maps for all the 21 vulnerability indices are available in Appendix B.

Spatial patterns of vulnerability were not consistent across the four mapped HVIs. In Figure 3.1a, the mapped heat vulnerability index for HVI Det 6, the west-northwest portions of the city were assigned high vulnerability index values, with pockets of high vulnerability observed in southwest Detroit and in some portions of central Detroit. The least vulnerable areas were identified in the west and east-northeast areas of the City. Block groups in the top 5% of the index were primarily located in the northwestern portion of the City, with a few also identified on the east side. The pattern of heat vulnerability was much different for the maps of HVI_Det_8 (Figure 3.1.b) and HVI_3N (Figure 3.1.c), where high vulnerability index values, as well as the top 5% of the index values, were located mostly in central, or downtown, Detroit. Figures 3.1.b and 3.1.c indicate that the highest vulnerability is generally located along the riverfront, which contrasts with the pattern seen with HVI 6 in Figure 3.1.a. Further, in Figure 3.1.d we see another specific vulnerability pattern that has the block groups assigned the highest vulnerability values located along the riverfront, but distributed mostly towards the northeastern portion of the city. Despite the variability in the spatial patterns of the indices presented here, one can conclude that across three of the four figures and indices (HVI 8, 3N and 2TC) there is a pattern of highest vulnerability in the central portions of the city, near the riverfront. An additional consistency observed in these analysis is that two small block groups in the southwestern-most region of the City were identified among those with the highest vulnerability. Block groups in the cities of Highland Park and Hamtramck were consistently ranked as highly vulnerable to heat.

Figure 3.1.a. Heat vulnerability index (HVI) map for census block groups (n = 913) in Detroit, MI, USA. The HVI incorporates respiratory disease prevalence and aerial photograph-derived calculations of non-trees (referenced in Table 3.2 matrix).

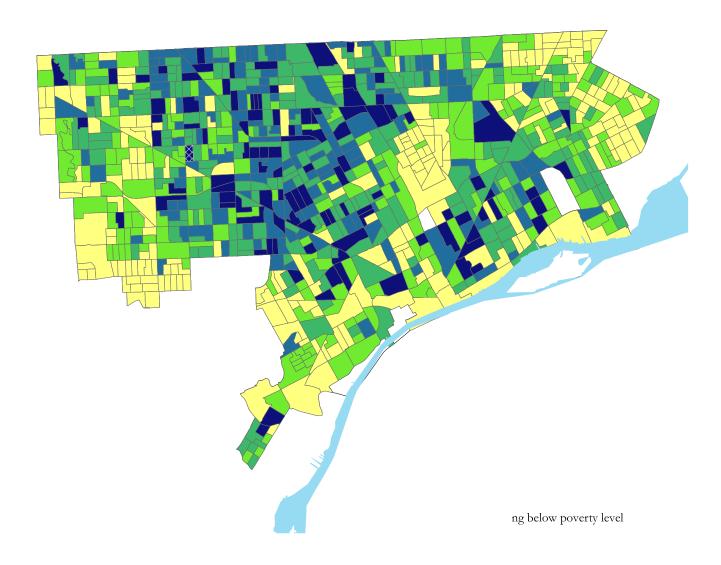


Figure 3.1.b. Heat vulnerability map for census block groups (n = 913) in Detroit, MI. Incorporates renal disease prevalence and aerial photograph-derived calculations of non-trees (referenced in Table 3.2 matrix).

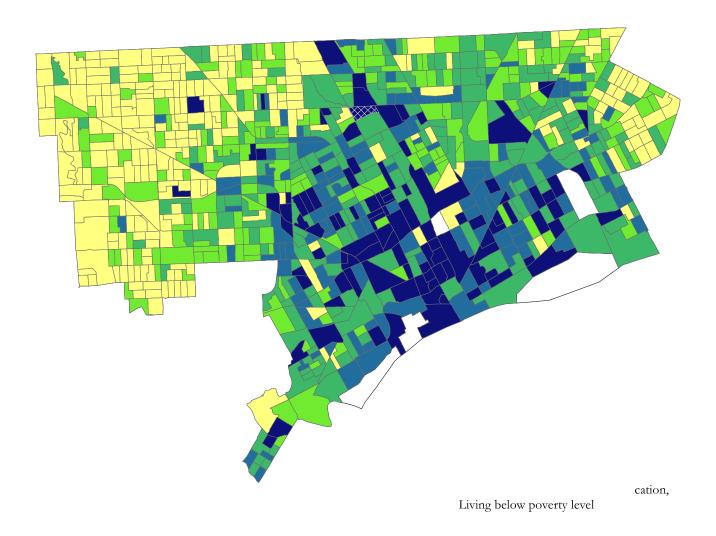
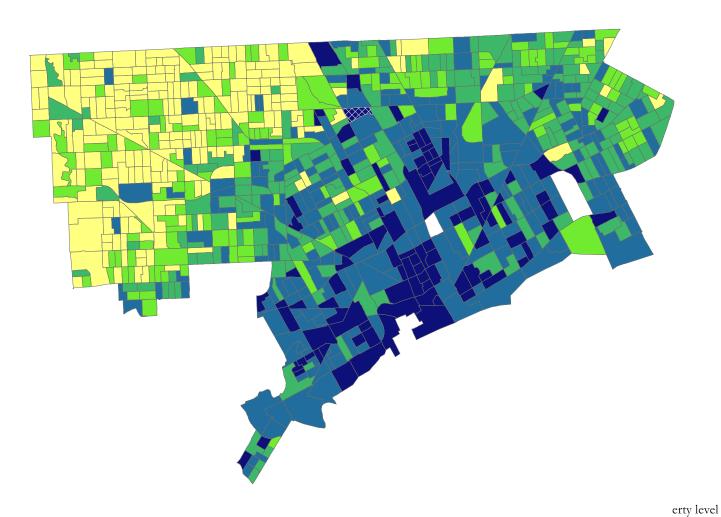
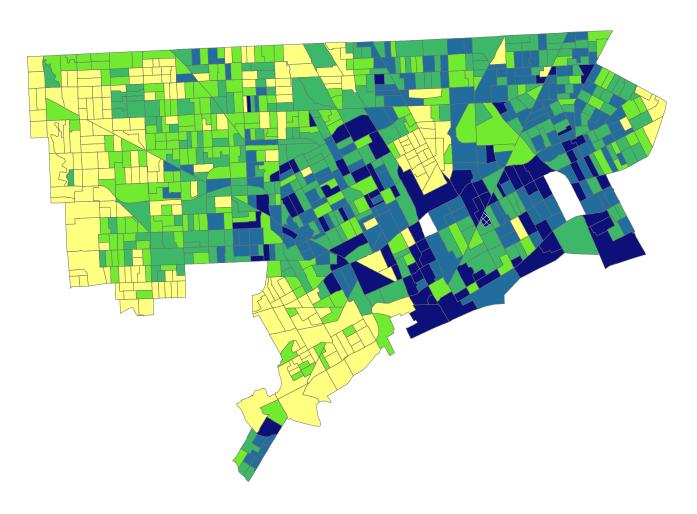


Figure 3.1.c. Heat vulnerability map for census block groups (n = 913) in Detroit, MI. Incorporates respiratory disease prevalence and NLCD-derived calculations of impervious surface (referenced in Table 3.2 matrix).



Factor 4 Impervious surface

Figure 3.1.d. Heat vulnerability map for census block groups (n = 913) in Detroit, MI. Incorporates cardiovascular disease prevalence and NLCD-derived calculations of non-tree canopy (referenced in Table 3.2 matrix).



status

Figures 3.2.s-c display maps of the Detroit, MI census block groups that were most frequently identified in the top 5% of all heat vulnerability indices (referenced in Table 3.2 matrix). These are grouped by land cover source type with aerial photograph, NLCD impervious or tree canopy; respectively, labeled in Figures 3.2.a, 3.2.b, 3.2.c. Visual comparison across the three maps indicates consistency in block groups with the highest vulnerability measures. In general, the most vulnerable block groups are located in the downtown, or central area near the Detroit River, and to the slight east and west of the downtown. Only a few block groups in the peripheral parts of the City were assigned high vulnerability scores. These are found in the northern and southwestern areas.

Figure~3.2.a.~Top~5%~Detroit,~MI~census~block~groups~identified~in~heat~vulnerability~indices~calculated~with~aerial~photograph-based~land~cover~classifications

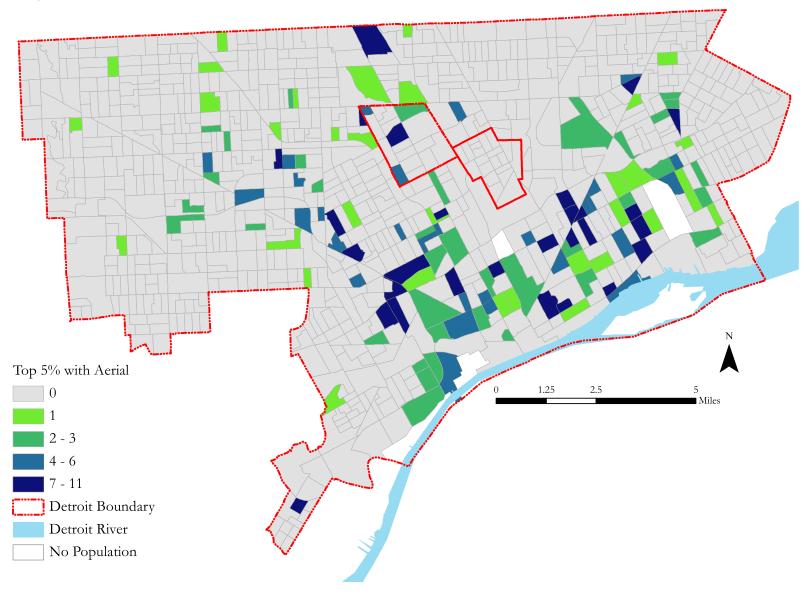


Figure 3.2.b. Top 5% Detroit, MI census block groups identified in heat vulnerability indices calculated with 2006 NLCD impervious surface layer

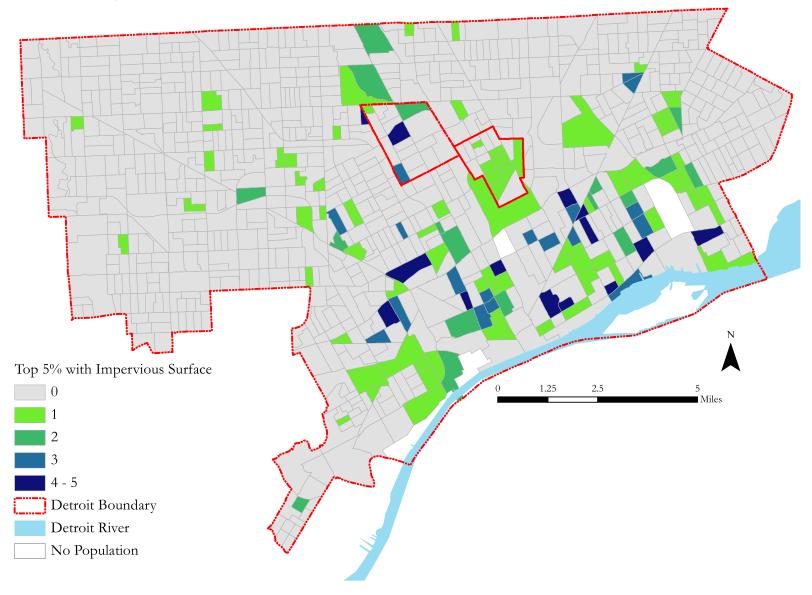


Figure 3.2.c. Top 5% Detroit, MI census block groups identified in heat vulnerability indices calculated with 2001 NLCD tree canopy layer

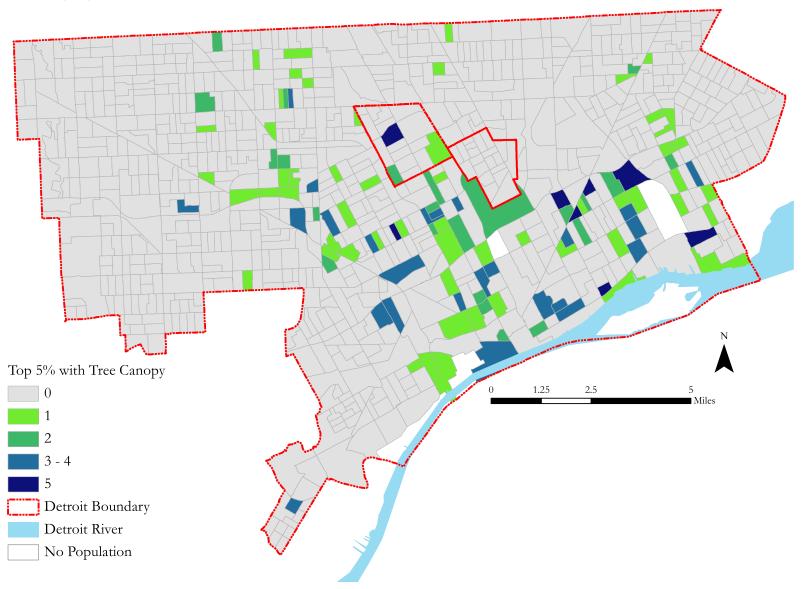


Table 3.6. Spearman correlation (p-value) between census tract-level weighted odds ratios of cardiorespiratory death and HVI 6, 8, 3N and 2TC values for Detroit, MI.

Heat Vulnerability Index	Weighted odds ratios of cardiorespiratory death
Detroit 6	0.10 (<0.001)
Detroit 8	0.40 (<0.001)
Detroit 3N	-0.02 (0.01)
Detroit 2TC	0.44 (<0.001)

The block group-level HVI values were significantly correlated with the weighted odds ratios of cardiorespiratory death, as presented in Table 3.6. The correlations were relatively low, however, across all four HVIs. Notably, we observed a negative correlation between HVI 3N values and the tract-level weighted odds ratios of cardiorespiratory death, which was contrary to our hypothesis that the HVI values would be positively associated with the downscaled effect estimates.

3.8 Discussion

The goals of these analyses were 1) to evaluate whether a HVI developed using finer scale data than used previously would show spatial patterns of heat vulnerability within the City of Detroit, Michigan, 2) to illustrate the sensitivity of PCA to different variable combinations in fine-scale heat vulnerability mapping and 3) to evaluate the correlation between downscaled effect estimates of heat-related cardiorespiratory death and HVI values. While certain areas of Detroit were consistently ranked as vulnerable across multiple HVIs, we saw widely differing spatial patterns throughout the study area depending on the combinations of input variables used in the PCAs. Further, PCAs for the HVI combinations produced factors that were neither easy to categorize nor had variables load reliably onto specific factors. The only predictable factor was factor 1, comprised of elderly/isolation variables, for 20 of the 21 PCA results. The anomaly was in HVI_Det_8 where factor 1 was comprised of variables for location, green space and health.

A limited number of other studies have constructed HVIs for urban areas. The census tract-level national HVI presented by Reid et al [37] elucidated heat vulnerability across the US, and within some urban areas, yet relied on relatively coarse scale environmental and health data. Finer scale indices, such as those done for Chicago [49] and Phoenix

[29], demonstrated finer variation in intra-urban heat vulnerability. While all these analyses used similar data, the resulting factors were sometimes quite different. For example, in Phoenix, the factors that comprised the index were first: socioeconomic vulnerability; second: elderly/isolation; third: non-vegetated areas. In Chicago, the factors could not be easily grouped, but generally separated as first: economic status and age; second: lower education, Hispanic ethnicity and 'other' races; third: the built environment and vegetation; and fourth: Black population and land surface temperature.

Although the Chicago and Phoenix analyses included heat-related mortality to evaluate the performance of their respective indices, neither included health data in the index creation. In the absence of finer-scale measures of disease prevalence, the ZIP-code level prevalence estimates used in this study enabled us to estimate disease prevalence heterogeneity across the Detroit study area that would not be captured if we had used estimates from the Behavioral Risk Factor Surveillance System (BRFSS). BRFSS estimates were used in the Reid, et al [37] HVI, and were calculated by downscaling state-level diabetes prevalence to county-level estimates. In using the 31 ZIP-code specific disease prevalence measures presented here, we could better characterize the health status of the Detroit population most vulnerable to heat. To our knowledge, this is the first application of Medicare hospitalization data as a proxy for population disease prevalence for heatrelated vulnerability. Medicare data has been used extensively to document chronic disease in the Medicare population [50]. Although this data source only includes individuals over the age of 65, most heat-related morbidity and mortality occurs among people over the age of 65. Further, with the number of older individuals in the U.S. expected to double in the next 25 years [51] coinciding with expected increasing ambient temperatures, there is reason to anticipate adverse heat-health outcomes for people over the age of 65.

Neighborhood variations in temperature and land cover have been identified in urban areas [27, 30, 52]. The role of green space in the heat-health literature is not well established, but is increasingly being considered as protective for vulnerable populations. In the previously mentioned vulnerability index calculations, characterization of the green space, or lack of green space, is from one source, the NLCD, which is limited to identifying

land cover over 30 meters. We utilized three very different sources for land cover classifications. The aerial photograph-derived land cover classifications allowed for a more accurate representation of vegetation, and consequently the microclimates, across the study area [27]. The NLCD impervious surface layer was used as a proxy for non-green space and the NLCD tree canopy layer was used to indicate tree coverage. We hypothesized that the inclusion of the finer-scale estimate of green space would not impact the spatial patterns of vulnerability due to the eventual aggregation up to the block group level of the land cover measures. Indeed, we observed moderate to high correlations between aerial- and impervious-based HVIs. Tree canopy-based indices were not as consistently correlated with the other two index types. The mean block group tree canopy coverage in Detroit, from the NLCD, was 5%, considerably lower than the aerial photograph-based tree coverage of about 33%. While the two are not easily comparable because they have different health variables in their indices, a distinct and opposite pattern of vulnerability was seen in HVI_6 and HVI_2TC. This may indicate that the contribution of non green variables influences the spatial distribution of vulnerability in the mapped index.

The indices presented here did not identify measures of poverty, education or minority status to be stand-alone indicators of heat vulnerability; these variables were interchangeably coupled throughout the indices. Poverty has been shown to be a primary contributor to social vulnerability [41]. The poverty status of a neighborhood may reflect the limited resources that could be directed towards adaptive measures. Yet, other indicators of socioeconomic status, such as education level, contribute to heat vulnerability but were not identified as primary indicators of vulnerability in these analyses. In this study, 91% of the Detroit population was identified as minorities. Little variation in this characteristic may explain why minority status was not identified as a primary factor in the HVIs.

3.8.1. Index interpretation, application and future directions

A challenge, once an HVI has been created, is interpreting the product and determining how to use the information. In the HVIs produced for Detroit, spatial distribution of vulnerability was variable depending on index composition. Most notably, two indices

computed using the same measure of non-vegetation (HVI_Det_6, HVI_Det_8), but differing health conditions, respiratory- and renal-disease, displayed nearly opposite patterns of high vulnerability. In determining areas of vulnerability based on these two maps, practitioners are faced with evaluating whether one product is 'better' than another.

Assessing whether the indices are identify the most vulnerable areas of an area requires some measure of performance for the PCA/vulnerability method. In our analysis, perhaps the most robust comparison for the HVIs was the maps indicating the top 5% most vulnerable census block groups across vegetation-specific HVIs. We observed consistent patterns of block groups identified as being vulnerable across aerial-, impervious surface, and tree canopy-based indices. These results could be interpreted as suggesting that different ways of characterizing land cover may have little influence on the identification of vulnerable areas when computing HVIs. Yet, such an interpretation should be avoided as it fails to consider, for instance, how the pattern may change if one holds everything constant except disease-specific prevalence.

Sensitivity to changes in the HVI based on the number and combination of input variables used for its construction are crucial in evaluating the final product. Sensitivity analyses of the SoVI [41], similar to the ones we conducted here, informed the authors' confidence in the performance of the index. Composite indices of comparable environmental hazards considered larger geographic extents than what we presented here. The total amount of variation explained by the retained factors for such analyses was larger, usually around 80% [29, 49], than what we observed here. Our study was limited to the geographic extent of a city, whereas most city-specific vulnerability indices are based on larger areas such as counties. Statistically speaking, larger datasets will have more power to explain the variance within observations, which is why we would expect to see both smaller variance explained for our analyses, as well as more sensitivity in the index.

A validation study of how the various indices presented here predict higher heat-specific morbidity and mortality for the most vulnerable block groups could be used to evaluate the performance of the index. In a larger validation analysis of a heat vulnerability index,

Reid et al [37] concluded that areas with high index values generally reflected the overall health vulnerability of that population [53]. We would expect that the contribution of finer scale environmental and health data to the indices created here would provide adequate predictive power for the HVI to identify areas specific heat vulnerability.

Studies have found that respiratory hospitalization rates increased during heat events in California [7, 54], New York [55-57], and in US counties with cool average summer temperatures [58], indicating that respiratory conditions are exacerbated during heat events and identify a population vulnerable to heat exposure. Additionally, renal disease, which is related to fluid imbalance, is a more recently studied morbidity associated with increased ambient temperatures [48]. In the HVIs presented here where both respiratory and renal disease are included, we do not see consistent or similar vulnerability patterns, though the respiratory and renal variables load on higher factors indicating they account for more variance in the data. The inclusion of respiratory and renal disease prevalence in identifying spatial distributions of vulnerability may be important for emergency response and planning purposes. Because the spatial patterns were not clearly discernible in our analysis, a validation analysis of the heat vulnerability indices with respiratory- and renal-cause morbidity or mortality would further inform the performance of the index in identifying heat vulnerability.

Limitations

Prevalence of air conditioning was not included in these analyses due to the lack of fine-scale information of air conditioning for the Detroit study area. Air conditioning has been identified as a protective characteristic during heat events [20]. Having data on air conditioning prevalence, however, even at a finer scale, has its limitations since simply having air conditioning does not ensure that residents use the amenity, particularly in economically deprived areas where energy costs deter use [43].

3.9 Conclusion

We identified and evaluated patterns and inconsistencies that occurred among fine-scale HVIs when created using differing measures of non-vegetation and health status in the for the City of Detroit using the commonly employed PCA method. Overall, inconsistent spatial patterns suggest that the HVI was sensitive to the input variables and must be interpreted with caution. In producing 21 indices and identifying the census block groups that were most frequently assigned a high vulnerability index value, we were able to identify a consistent pattern of vulnerability in downtown Detroit. However, despite the numerous HVI iterations, we conclude that the maps would better serve at identifying spatial pockets of vulnerability in conjunction with local knowledge and validation with health outcomes.

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Chapter 4.

Evaluating the performance of heat vulnerability indices using fine-scale estimates of heat-related mortality in Detroit, MI USA

4.1. Abstract

BACKGROUND: Expected increases in extreme heat pose a risk to human health, particularly to populations vulnerable to heat who live in urban areas. The spatial distribution of heat-related vulnerability has been of particular interest to researchers and policy-makers alike, as understanding this can aid targeting of interventions.

OBJECTIVES: We aimed to provide a better understanding of the spatial dimension of heat-related vulnerability by evaluating correlations of two heat vulnerability indices (HVIs) with excess heat-related mortality at the census block group scale using geocoded mortality data for the city of Detroit, Michigan.

METHODS: We used a case-crossover study design to evaluate the association between geocoded cardiorespiratory mortality in Detroit, MI USA (May – September, 2000 – 2009) and extreme heat days (defined as days when the mean apparent temperature averaged from that day ant the day prior were above the month-specific 95th percentile of apparent temperature). To assess whether residents of block groups having higher HVIs were at greater risk from heat-related mortality, we included interaction terms between extreme heat and an indicator for whether the census block group was greater than or at or below the median, quartile and 95th percentile scores for two different HVIs. A case-only sensitivity analysis and a comparison of the HVI values at the census tract level to the proportion of those who died on an extreme heat day were also conducted.

RESULTS: The association between extreme heat days and cardiorespiratory mortality was not significantly modified by the decedents' census block-specific HVI values. The two HVIs used in this analysis produced similar results, with neither reliably indicating a better performance in predicting heat-related mortality, at either the block group or Census tract scale. Census block group measurements of community-level variables were not predictive of cardiorespiratory mortality at the block group scale.

CONCLUSIONS: The HVIs presented in this analysis did not demonstrate a strong ability to identify whether Detroit residents living in census block groups assigned high values of heat-related vulnerability had higher odds of cardiorespiratory death during extreme heat. Future analyses should consider comparing different modeling approaches for evaluating the predictive power of HVIs. The spatial distribution of heat-related vulnerability may rely heavily on the explicitly specific distributions both individual and community level characteristics that may be lost when aggregated.

4.2. Keywords

Heat vulnerability, heat vulnerability index, evaluation, adaptation

4.3. Abbreviations

HVI Heat vulnerability index

MDCH Michigan Department of Community Health

NCDC National Climatic Data Center

PCA Principal component analysis

UHI Urban heat island

4.4. Introduction and background

The role of very high, or extreme, temperatures on human mortality has been well researched [1]. Urban areas are considered important for the study of the heat-health association in part due to the urban heat island (UHI) effect, where higher observed ambient temperatures occur in urban areas compared to surrounding suburban and rural areas, but also due to population characteristics, such as old age, socioeconomic status, and environmental characteristics like green space, that have been shown to contribute to heat-related vulnerability [2-6]. The non-uniform distribution of these characteristics [7, 8] poses a unique opportunity to potentially identify pockets of heat-related vulnerability within urban areas. However, the knowledge gained from such studies of the heat-health relationship is limited in scope and evaluation.

The heat vulnerability indices (HVIs) presented in Chapter 3 are iterations of the recently-developed applied method for grouping environmental, demographic, socioeconomic and human health variables that can then be used to calculate a proxy for heat-related vulnerability [8-10]. Components comprising an HVI differ, depending on the variables included in its construction, typically selected because they are believed to contribute to heat-related vulnerability. One interpretation is that it represents locations across a spatial extent where populations most vulnerable to heat reside. Further, an HVI allows evaluation of the distribution of heat-related vulnerability by considering the assigned index values relative to surrounding index values. Theoretically, the HVI would be able to predict, within a geographic extent, where heat-related morbidity or mortality would be greater among the population on days with extreme or high heat. Such information could aid in preparing for and responding to impending increases in ambient temperature, and provide a quantitative approach for selective intervention or targeted adaptation measures. While the HVI is a relatively recent application of the understanding of the heat-health relationship, understanding of how accurate the HVI is in its performance on identifying where heat-related vulnerability occurs is limited. At the date of this writing, three evaluations of HVI performance in identifying specific locations of acute heat-related morbidity and mortality have been published [8, 10, 11]. Despite their different index constituents, the three studies reported moderate- to

strong predictive capabilities for heat related mortality, suggesting that those HVIs may be of use in guiding preventive interventions during hot weather.

As heat vulnerability research continues to be relevant and critical to the field of public health and disease prevention, the application of knowledge gained from robust epidemiologic, climatological, and social science research findings that inform the development of climate adaptation plans will experience increasing scrutiny. Therefore, contribution to the understanding of how fine-scale or city-specific applications of heat vulnerability research, such as the predictive capacity of HVIs in identifying where those vulnerable during heat events live, is crucial. The HVIs presented in Chapter 3 are specific to Detroit, Michigan, a U.S. city that has been studied for its heat-health response particularly due to its unique geographic location and its distinctly segregated — in terms of race and socioeconomic status — population [2, 12, 13]. In the analysis presented here, we used numerous methods to evaluate whether within-city areas that were ranked higher by two heat vulnerability indices, created from fine-scale, community-level aggregated demographic, environmental, and health characteristics, had higher associations between cardiorespiratory-cause death and extreme heat in Detroit, MI.

4.5. Methods

4.5.1. Heat vulnerability index

Two heat vulnerability indices were assessed in this study. Both indices, which are previously reported in Chapter 3 of this dissertation, contained the following variables calculated as the proportion per census block group: population over the age of 65; population living alone; population over the age of 65 and living alone; population characterized as having minority status — which is defined by the US EPA Office of Environmental Justice as being Hispanic, Asian-American and Pacific Islander, African American, and American Indians and Alaskan Natives

(http://www.epa.gov/region2/ej/guidelines.htm); population with less than a high school education; and population living below the poverty line. The indices also included ZIP code-level estimates of respiratory disease prevalence, calculated from

2006 hospital admissions data obtained from Centers for Medicare and Medicaid Services. The indices differed in their measurement for non-green space. HVI 6 (Table 3.1., Chapter 3) characterized non green space from a 1-meter aerial photograph image (SEMCOG, 2006) as being the proportion of a census block group that was not identified as containing trees. HVI 3N characterized non green space as 1 – the percent impervious surface as provided by the impervious surface layer from the 2006 National Land Cover Database (NLCD). The two measures of non-green space were relatively similar, with the aerial photograph-derived non-trees, on average, covering 67% of the Detroit census block groups, and the NLCD percent impervious, on average, covering 60% of the census block groups in Detroit. Despite the similarities in the data used to calculate the HVIs, the HVI-specific principal component analysis (PCA) produced principal components comprised of different combinations of variables, as well as maps with distinct spatial patterns. For these reasons, we chose to further investigate and compare the HVI 6 and HVI 3N with respect to heat-related mortality in Detroit.

4.5.2. Mortality data

Daily, geocoded mortality data were obtained from the Michigan Department of Community Health (MDCH) for the years 2000 – 2009. Institutional Review Boards for the University of Michigan and the MDCH approved this study (UM IRB: HUM00067448). Daily deaths with primary causes being cardiovascular (International Classification of Diseases 10th revision (ICD10): I01 – I59) or respiratory (ICD10: J00 – J99) diseases were aggregated to create cardiorespiratory causes. Decedent information on age, race and gender were available and extracted from the death certificates. We limited the analysis dataset to May through September months over the 10 year time period because our primary interest was in extreme heat occurring during the warm season.

The City of Detroit, contained within Wayne County, was the study area of interest for this analysis. Death records were geocoded by the Michigan Center for Geographic Information (CGI) (www.michigan.gov/cgi), and had a 98% match rate. Death records that we were unable to geocode were removed from this analysis. The total number of geocoded deaths within the city boundary of Detroit was 14,103 for the May through

September months of the study period. Each death was assigned a census block group identifier using the spatial join function available in ArcGIS ArcMap 10.0.

4.5.3. Weather data

Hourly mean temperature and daily dew point data from two weather stations near the Detroit city boundary were obtained from the National Climatic Data Center (NCDC). Apparent temperature (AT °C) (EQ.1), a measure similar to a heat index that incorporates dew point and reflects the temperatures felt by individuals [14] was calculated from these data.

EQ 1. AT = -2.653 + (0.994 x ambient temperature) + (0.0153 x dew point temperature)

Because exposure to heat does not immediately impact human health, we calculated the AT to include the mean apparent temperature occurring on the date of death and the day prior, or lag01. Studies have indicated that two day moving averages of mean apparent temperature of lag01 (AT01) adequately captures the acute effect of heat [15]. To account for deaths that occurred earlier in the summer seasons, we defined extreme heat as being above the month-specific 95th percentile of AT01 for the years 2000 – 2009.

4.5.4. Case crossover modeling and statistical analysis

The time stratified case crossover design was used to estimate the association between extreme heat and cardiorespiratory death in the Detroit, MI population. The case crossover analysis is, essentially, a matched case control design that allowed us to control for time-invariant characteristics, match on geographic identifiers, as well as incorporate interaction terms for the census block-group specific HVI values. To evaluate whether certain higher scores of vulnerability were better at predicting cardiorespiratory death, we looked at the association when treating HVI as a continuous variable and as indicator variables for the median, quartiles and the top 5%.

The models used to evaluate HVIs 6 and 3N were:

logit(mortality_{CVDRESP}) = β_1 EH01₉₅ + β_2 (EH01₉₅ * HVI)

```
\begin{split} &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Median}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Quartile}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{95th \; percentile}) \end{split}
```

To consider the contribution of the variables used in the calculation of the HVIs, we also looked at whether block group-specific proportions of individual variables (e.g., proportion of people over 65 living alone) modified the effect of extreme heat on cardiorespiratory mortality. The models used to calculate the association between extreme heat and cardiorespiratory death, and effect modification by the block group variables were:

```
\begin{split} &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Over~65}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Living~alone}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Over~65,~Living~Alone}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Less~HS~education}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Minority~status}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Below~Poverty~level}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Impervious~surface}) \\ &logit(mortality_{CVDRESP}) = \beta_1 EHO1_{95} + \beta_2 (EHO1_{95} * HVI_{Non~trees}) \\ \end{split}
```

We hypothesized that the fine-scale vulnerability indices would be moderately predictive of heat-related cardiorespiratory mortality in Detroit MI for the 2000-2009 study period. Due to the sensitivity of the indices to the variables included in their creation, as shown in Chapter 3, we did not expect either index being evaluated to be significantly better at predicting heat-related cardiorespiratory death.

4.5.5. Sensitivity analysis

One limitation of heat vulnerability analyses relates to the lack of publicly available fine-geographic scale health, demographic and environmental data. To determine whether the associations found in the case-crossover analysis presented here, which were computed with the block group-level HVIs, remained when using a tract-level HVI, we recalculated the block group level HVIs at the tract-level. Following the same logic tested in the case-crossover analysis, one would expect to observe a positive association between higher HVI values and a higher number of deaths on an extreme heat day if the heat vulnerability index is a valid proxy for the risk of adverse health outcomes when

high ambient temperatures are observed. Rather than creating controls for the analysis, we conducted a logistic regression on those individuals who died on an extreme heat day (EH01₉₅) and the tract-level HVI values [16]. Extreme heat days were defined as days where the AT01 was above the month-specific 95th percentile during the May — September 2000 — 2009 study period. Case-only analyses have been used to evaluate individual-level effect modifiers of temperature and mortality in a Detroit, MI population [16]. By evaluating only those individuals who died on days when extreme heat occurred, we were still able to assess the performance of the HVIs in their ability to predict areas of high vulnerability to heat, or, where deaths would be expected to occur. We estimated the relative odds of cardiorespiratory death on an extreme heat day using a logistic regression model that tested whether a one unit increase in a tract-specific HVI (HVI 6 and HVI 3N) predicted cardiorespiratory deaths that occurred on extreme heat days. An example of the model follows:

$$logit(mortality_{CVDRESP \mid EH0195}) = \beta_1 HVI_6$$

Because individual-level characteristics such as age and race have been shown to modify the heat-mortality association, we included them as potential modifiers in the sensitivity analysis.

In an additional analysis, Spearman correlations were computed to evaluate the association between the tract-level HVIs and the proportion of individuals, or cases, who died on an extreme heat day compared to the entire population of individuals who died of cardiorespiratory-related causes on any days during the May – September, 2000 – 2009 study period. We expected that increased HVI values would be associated with an increased proportion of cardiorespiratory deaths that occurred on extreme heat days.

4.6. Results

Population-specific characteristics are presented in Table 1. Compared to the Detroit population studied in Chapter 1, which was for the time period 1990-2006, we saw a higher fraction of people at the extremes of the age range, with individuals who are very old (over the age of 75) making up 49% of the decedents and those who are young below

65 years), 32.9%. Additionally, we observed a much larger proportion of nonwhite individuals (79.1%). Gender distributions were about equal in both studies.

Table 4.1. Summary statistics of cardiorespiratory cause deaths and apparent temperature, Detroit MI, US (2000 - 2009).

Total cardiorespiratory mortality (n)	14.010
	14,013
Age in years, (%)	
< 65	32.9
65 - 75	18.2
≥ 75	49.0
Race, (%)	
White	20.9
Nonwhite	79.1
Gender, (%)	
Male	50.2
Female	49.8
Mean daily number of cardiorespiratory deaths	
by month (2000 - 2009)	
May	9.36
m June	9.30
July	9.27
August	9.06
September	8.79
Apparent Temperature (°C), 95th percentile	
May	21.91
June	29.09
July	30.48
August	29.34
September	26.29
r	

The results from the case crossover model that incorporated the various HVI measurements are presented in Table 4.2. We observed a statistically significant overall effect of extreme heat (ATO1) and cardiorespiratory mortality in the Detroit study population where the odds of cardiorespiratory mortality on an extreme heat day were 1.11 the odds of cardiorespiratory mortality on a non-extreme heat day. In assessing the predictive power of the HVI, with the HVI values treated as continuous, we observed no association of cardiorespiratory mortality risk with increased HVI values for both HVIs 6 and 3N, with odds ratios of 0.99 (95% confidence: 0.95, 1.04) and 0.97 (95% confidence: 0.93, 1.01), respectively.

Table 4.2. Results for the case-crossover analysis between extreme heat and cardiorespiratory-cause deaths in Detroit, MI May - September, 2000 - 2009, including HVI 6 and 3N values as effect modifiers.

		and on values as effect mounters.	
Model	Odds Ratio	95% Confidence Interval	
EH > 95th	1.11	(1.02, 1.20)	
	HVI 6		
EH > 95th	1.18	(0.71, 1.98)	
95th, HVI 6	0.99	(0.95, 1.04)	
EH > 95th	1.13	(0.95, 1.35)	
95th, HVI 6 Quartile 2 (25th)	1.11	(0.93, 1.33)	
95th, HVI 6 Quartile 3 (50th)	1.03	(0.88, 1.22)	
95th, HVI 6 Quartile 4 (75th)	1.15	(1.00, 1.33)	
EH > 95th	1.03	(0.66, 1.59)	
95th, HVI 6 Top 5%	0.83	(0.60, 1.22)	
HVI 3N			
EH > 95th	1.67	(0.94, 2.96)	
95th, HVI 3N	0.97	(0.93, 1.01)	
EH > 95th	1.12	(0.94, 1.33)	
95th, HVI 3N Quartile 2 (25th)	1.23	(1.01, 1.51)	
95th, HVI 3N Quartile 3 (50th)	1.13	(0.99, 1.29)	
95th, HVI 3N Quartile 4 (75th)	1.00	(0.84, 1.18)	
EH > 95th	1.36	(0.79, 2.35)	
95th, HVI 3N Top 5%	0.82	(0.58, 1.19)	

In treating the HVI values according to their quartile ranks, with the lower 25th percentile serving as the reference category, we observed in both HVIs 6 and 3N no associations between the HVI values and cardiorespiratory mortality. HVI 6 values in the 75th percentile had a 1.15 increased odds of cardiorespiratory mortality (95% CI: 1.00, 1.33) when compared to the lowest quartile, but was marginally statistically significant.

Similarly, HVI 3N values in the 25th percentile were positively associated with 1.23 increased odds of cardiorespiratory mortality compared to those in the lowest quartile but were also marginally statistically significant. HVI3N values above the median observed a positive association (odds ratio=1.13), was statistically insignificant. When assessing the HVI's performance with only the top 5% of HVI values, we observed no associations for both HVIs 6 and 3N between the block group assigned HVI values and the odds of cardiorespiratory mortality.

Overall, the associations between the HVI values and the risk of cardiorespiratory mortality were consistently insignificant. Analyses that compared quartiles of HVI values for both HVI 6 and 3N indicated an increase in cardiorespiratory death risk, whereas the analyses where HVI was treated as a continuous predictor or when using the index values in the top 5% produced odds ratios suggesting a decrease in heat-related cardiorespiratory death risk. There is no clear pattern on the risk of cardiorespiratory death and living in a census block group with a high HVI value. The odds ratios for these analyses are not very stable and are accompanied by large confidence intervals.

To investigate the community-level characteristics used in the HVI creation, we evaluated those characteristics' ability to predict heat-related cardiorespiratory mortality in this study population. Table 4.3 presents the distribution of the community-level characteristics for the block groups in Detroit, MI. There was not an even distribution of the characteristics. Minority populations, on average, comprised nearly all of the study census block groups. Individuals over the age of 65 were not a large proportion of the population, but in some block groups they did account for half of the population. Additionally, while the average percent of people living below the poverty level was 35%, at least one block group was identified as having almost 90% of the population living below the poverty level. Lack of tree coverage and the distribution of impervious surface, both characteristics that can contribute to an increase in ambient temperatures, were comparable and some block groups were characterized as completely non-vegetated.

Table 4.3. Distribution of the proportion of block group population characteristics used in the creation of heat vulnerability indices (HVIs) 6 and 3N.

Variable (proportion)	Mean	Std Dev	Minimum	Maximum
Over 65	0.12	0.08	0.00	0.51
Over 65, Living alone	0.04	0.05	0.00	0.43
Living alone	0.14	0.11	0.00	1.00
Minority	0.91	0.14	0.03	1.00
Less than HS education	0.14	0.09	0.00	0.49
Living below poverty line	0.35	0.19	0.00	0.88
Non trees	0.67	0.15	0.19	1.00
Impervious surface	0.60	0.11	0.00	0.92

The associations between the continuous predictors of community-level characteristics and the risk of heat-related deaths are shown in Table 4.4. We observed no associations between any community-level characteristics and the risk of heat-related cardiorespiratory-cause death. Although it was not statistically significant, the block group-level measure of minority status was the only community-level characteristic that indicated a positive relationship with cardiorespiratory-cause death and extreme heat.

Table 4.4. Case-crossover model results of the association between extreme heat and cardiorespiratory-cause deaths in Detroit, MI May - September, 2000 - 2009, as modified by community-level characteristics used to construct HVIs 6 and 3N.

Model	Odds Ratio	95% Confidence Interval
Over 65		
Extreme Heat (> month-specific 95th percentile)	1.11	(1.02, 1.20)
Extreme Heat*Over 65	0.52	(0.20, 1.33)
Living alone		
Extreme Heat (> month-specific 95th percentile)	1.11	(1.02, 1.20)
Extreme Heat*Living alone	0.50	(0.25, 0.98)
Over 65, Living alone		
Extreme Heat (> month-specific 95th percentile)	1.11	(1.02, 1.20)
Extreme Heat*Over 65, living alone	0.28	(0.07, 1.17)
Less High school education		
Extreme Heat (> month-specific 95th percentile)	1.11	(1.02, 1.20)
Extreme Heat*Less than HS education	0.72	(0.27, 1.89)
Minority status		
Extreme Heat (> month-specific 95th percentile)	1.11	(1.02, 1.20)
Extreme Heat*Minority status	1.45	(0.77, 2.75)
Living below poverty level		
Extreme Heat (> month-specific 95th percentile)	1.11	(1.02, 1.20)
Extreme Heat*Living below poverty level	0.89	(0.57, 1.39)
Non trees (aerial)		
Extreme Heat (> month-specific 95th percentile)	1.11	(1.02, 1.20)
Extreme Heat*Percent nontrees	0.88	(0.49, 1.61)
Impervious surface		
Extreme Heat (> month-specific 95th percentile)	1.11	(1.02, 1.20)
Extreme Heat*Percent impervious surface	0.78	(0.36, 1.69)

Table 4.5 Relative odds (95% CI) of cardiorespiratory death occurring on an extreme heat day per one unit increase in HVI 6 and 3N values, among people living in census tracts (n = 308), including individual-level characteristic, Detroit MI, May-September 2000 – 2009.

	Relative Odds [±] (95% CI)		
	HVI 6	HVI 3N	
Crude	0.98 (0.94, 1.02)	0.97 (0.93, 1.01)	
Individual-level characteristic			
65 - 75 years	1.01 (0.91, 1.13)	1.02 (0.91, 1.14)	
≥ 75 years	0.97 (0.89, 1.06)	0.97 (0.89, 1.05)	
Nonwhite	0.99 (0.90, 1.10)	0.98 (0.89, 1.09)	

[±] Relative odds of cardiorespiratory death occurring on an extreme heat day compared to a non-extreme heat day

The sensitivity analysis provided additional insight to the performance of the HVI pertaining to cardiorespiratory-cause death. We did not observe an association between tract-level HVI values treated as continuous variables and the odds of death on an extreme heat day (Table 4.5). Individual-level age and race characteristics were not statistically significant effect modifiers of the probability of cardiorespiratory death occurring on an extreme heat day and the tract-level values for HVIs 6 and 3N (Table 4.7). These results cannot be compared to the block group-level HVI analyses, as we did not assess the relationship between cardiorespiratory-cause death, HVI values and whether age or race modified the effect of that association.

We did observe significant negative correlations between the tract-level HVI values (also treated continuously) and the proportion of all summertime cardiorespiratory deaths occurring on extreme heat days in a census tract (Table 4.6). The correlations suggested that an increase in the HVI value was associated with a decrease in the proportion of cardiorespiratory deaths that occurred on extreme heat days.

Table 4.6. Spearman correlations (p-value) of tract-level heat vulnerability indices (n = 308) and the proportion of summertime cardiorespiratory deaths that occurred on an extreme heat day*, Detroit, MI, May-September 2000 - 2009.

extreme heat day, Detroit, Mil, M	iay-beptember 2000 – 200	J.	
	HVI 6	HVI 3N	
Proportion of cardiorespiratory deaths on extreme heat day	-0.09 (<0.001)	-0.12 (<0.001)	

^{*} Extreme heat day is a day that is above the month-specific 95th percentile of mean apparent temperature for lag days 0,1

^{*} Extreme heat day is a day above the month-specific 95th percentile of mean apparent temperature for lag days 0,1

4.7. Discussion

Evaluating the indices

The aim of this analysis was to evaluate previously constructed heat vulnerability indices using fine-scale mortality data for the 2000 – 2009 Detroit, MI population. We used three different analytic methods to evaluate the hypothesis that HVIs would reliably predict the risk of heat-related cardiorespiratory mortality among the Detroit population at the census block group and tract levels. The results of the time stratified case crossover analysis indicate that neither HVI 6 nor 3N are able to reliably predict the risk of heat-related cardiorespiratory mortality among the Detroit population at the block group level. These results are contrary to our hypotheses that the indices would be able to moderately detect fine-scale heat-related risk. In treating the block group level HVIs as quartiles and as indicators for the highest HVI values, we did not observe an association between heat-related cardiorespiratory death and the block groups assigned the most vulnerable HVI values. The block group level HVIs were consistently unable to precisely identify block groups where heat-related cardiorespiratory deaths occurred. The block group level measures of community characteristics shown in previous research to be associated with extreme heat [1, 17] were not predictive of cardiorespiratory mortality at the block group. When we considered the heat vulnerability index values at the census tract level and assessed their relationship with the relative risk of cardiorespiratory death on extreme heat days, we did not observe any association. The association, however, between the proportion of cardiorespiratory deaths that occurred on extreme heat days and tract-level HVI values was statistically significant, but in the opposite direction of our hypothesis.

The counterintuitive results of the analyses presented here require thorough interpretation of the presented effect estimates and the suggested associations. For instance, in estimating whether the top 75% of block group index values from HVI 3N are associated with higher odds of cardiorespiratory death among decedents in those block groups, the parameter estimate of -0.11 indicates a protective effect of block groups that are assigned higher HVI values. The interpretation that the higher index values are protective of extreme heat compared to the lower index values may not

consider the role of the variables comprising the index. It could be the case that the -0.11 parameter estimate explains that the first factor (e.g., explaining the most variance in the dataset) which was comprised of individuals who were over the age of 65, living alone, and people over the age of 65 living alone contributes most to the negative association between index value and cardiorespiratory mortality. Further, one may conjecture that a census block group that was assigned a high vulnerability value, from HVI 3N, contained a high proportion of people who could be characterized as being over the age of 65, living alone, and over the age of 65 and living alone. Thus, one explanation for the negative association could be that these individuals may experience a protective due to some unexplained characteristic. The census data that was used to create the indices may include individuals living in senior centers to be older and living alone. During an extreme heat event, these individuals would likely be protected as result of the social network provided by the center. Contrarily, if you were to then take a person of the same characteristics and consider them living in a block group where a high proportion of residents are young families, that older individual may be more likely to die as a result of extreme heat if the protective effect of the like population is lost, which would be supported by the associations observed here. This explanation, of course, is speculative and requires a refined analysis of the distribution of the demographics and social networking to evaluate its credence.

Few studies have evaluated the use of the HVI in predicting heat-related morbidity and mortality. One study concluded that the HVI was unable to reliably distinguish between days of extreme heat and days of non-extreme heat and the association between general indicators of ZIP-code level morbidity and mortality [11]. Harlan et al [10] used binary logistic regression to estimate the odds of at least one heat-associated death (geocoded) and an HVI, similar to those presented here, to evaluate the predictive power of the index in Phoenix, AZ and concluded that the index including surface temperature measurements was associated with heat-associated death. The ability of HVIs to discriminate between high temperatures is especially relevant for climate adaptation plans that discriminately focus resources to the most vulnerable areas.

Studies on the temperature profiles of urban areas strongly support the need for considering spatial variability in assigning exposure at the individual level [18]. In this analysis, two temperature stations were used to characterize the heat exposure as being above a month-specific 95th percentile of AT01. It is common that area-level measurements of temperature are used to investigate the association between extreme heat and mortality. This study was limited as it used temperature and dew point measurements from two area monitors, which may have introduced exposure misclassification in this analysis.

An important caveat that has yet to be discussed in detail is that the commonly-used method to construct an HVI assumes equal contribution of the variables of which it is comprised. The extensive heat-health literature does not support this assumption; the impacts of extreme heat can differ depending on population of interest, the regional location of the population, access to protective services and utilities (e.g., cooling centers, having central air conditioning). There is some evidence that suggests that individual-level and area-level characteristics commonly used in heat-health analyses may confound each other [19, 20], suggesting that future analyses should consider the contribution of both individual level and community level characteristics beyond simply including them in the creation of an HVI.

As demonstrated here, the interpretability of the HVI is limited to explicit knowledge of the population whose vulnerability is being characterized. Despite the incorporation of fine-scale data, which can be considered to provide more accurate representations of the population of interest, this analysis suggests that additional information is necessary to critically evaluate the spatial distribution of heat-related risks. In Chapter 3, we saw that varying distribution of vulnerability index values, where, in some cases a census block group assigned to the highest interval of index values is adjacent to block groups assigned to the lowest interval of index values.

Extensive epidemiological research suggests that, depending on a myriad of characteristics, there are varying associations between heat and health, with particular differences in regards to location. These varying associations may explain why the HVIs

presented here are unable to capture a spatial distribution of vulnerability that is representative of the heat-health relationship in Detroit, MI. In the analyses presented here, we saw that the community-level characteristics that comprised the HVIs, were not significantly associated with the risk of cardiorespiratory death when exposed to extreme heat. While the variables that comprised the HVIs presented here may be appropriate and predictive in other cities, we were not able to establish a significant association. One possibility for this is that the limited variation of these characteristics across the general population of the City of Detroit may confer an inability to distinguish the individuals who are even more vulnerable. Further, this analysis did not include all heat vulnerability-related variables, such as median income [7, 21], which may serve as the main explanatory characteristic for heat-related cardiorespiratory mortality in Detroit, MI. Lastly there is the explicit limitation in how to appropriately interpret a one-unity increase of heat-related vulnerability, as depicted in an HVI. Future research should consider the relative predictive power of individual area-level characteristics compared to HVI calculations in estimating adverse health responses. The HVI method used here, and in other HVI applications, assumes that characteristics that make a person vulnerable in one location are the same in all locations. The results presented here may be due to incorrect assumptions of the population characteristics of heatrelated vulnerability for the Detroit, MI population.

Although this analysis does not provide substantial support for the use of HVIs as a proxy for heat-related mortality, the value of the index is not entirely lost as it can act as springboard to conducting place-based assessments. The development and use of environmental health indicators that are related to climate change is considered an imperative step in evaluating the relative impacts of climate change on human health. English, et al [22] calls for the specific development of fine-scale indicators of the impacts of climate change on environmental health. In many disciplines, the HVI may be considered a comprehensive indicator of heat-related vulnerability across a spatial extent. Although the HVI can provide a visual display of possible vulnerable areas in a city, there is no conclusive evidence that an HVI can reliably identify very specific locations where residents are likely to succumb to extreme heat.

4.8. Conclusions

The analysis presented here suggests that heat vulnerability indices are not a powerful enough tool that can be used to identify block group level heat-related mortality in the Detroit MI population. Statistical associations indicating protective effects of high census block group-specific HVI values were not statistically significant yet were consistently indicating opposite effects of index values on cardiorespiratory mortality. Due to the increased interest in the application of HVIs as tool to inform climate change adaptation, it is imperative that further evaluations of the HVI and heat-related health outcomes are conducted, which consider the relative contribution from the factors that comprise the index, as well varying study locations to account for regional adaptations that are not captured in the HVI, and study design to determine whether results are robust and reliable.

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Chapter 5.

Conclusion

5.1. Overview

The overall goal of this dissertation was to implement novel approaches for identifying, characterizing and evaluating estimates and proxies of fine-scale heat-related vulnerability. Within that framework, this dissertation also aimed to incorporate knowledge of the complex and dynamic relationship between human health and the surrounding environment with a specific goal of providing research products that can be used to eventually inform programs and policies to protect vulnerable populations. The results of this dissertation provide insight into two methodologies for identifying and displaying population-specific heat-related vulnerability at the fine scale. Although the importance of considering vulnerability at fine scales seems evident, few studies have demonstrated methods in which fine scale estimates or measures for heat-related risks are presented.

5.2. Chapter 1: Downscaling epidemiologic effect estimates for heatrelated mortality

The majority of epidemiologic research pertaining to the relationship between extreme heat and mortality calculates the risk of an adverse health outcome that reflect populations across large geographic extents, such as the county level. While those estimates can be useful in characterizing the general form of the relationship between heat and health, they are less informative of the spatial distribution of heat-related vulnerability within geographic zone in which interventions could be effectively targeted and implemented.

The concept of downscaling is regularly used in climatological sciences, as one can extract from large-scale global climate models projections on how the climate will change, and, using supplemental information that characterizes the finer geographic area, downscale the projections. In borrowing from the climatology field, the first chapter of this dissertation presents a method for downscaling epidemiologic effect estimates to the census tract scale. To the best of my knowledge, this is the only documented exercise in which fine-scale epidemiologic estimates are generated based on population-level demographic characteristics.

The results of this aim indicate that population characteristics that are known to be associated with increased risks of heat-related mortality can be used to downscale the effect estimates derived at the county level, thus distinguishing the visual patterns of lower versus higher heat-related mortality risk within and across study cities. The patterns, while not confirmed with spatial statistics, do suggest that within our 20 study cities the association between heat and mortality is variable, with some cities showing consistent patterns of vulnerability, where other cities have what appear to be random patterns of vulnerability. Maps showing the fine-scale estimates could serve as useful communication tools for public health officials working on climate and health-related projects.

5.3. Chapter 3: Fine-scale heat vulnerability mapping

Increased interest in the creation of heat vulnerability maps reflects the growing need for advancing methods that identify heat and health relationships at scales relevant for local intervention. Previously calculated indices were executed at the census tract level and included non-specific place-based measures of green space and population health status. In all of the other extant publication on heat-related vulnerability indices, only one computed index was presented. This implicitly suggests that the index presented is correct, or stable.

The second dissertation chapter considers the possibility that differing measures of like variables (e.g., non green space measured by aerial photograph-derived land cover classifications and percent impervious surface), and differing combinations of variables

known to be related to heat vulnerability, will yield different statistical and spatial results in the creation of a heat vulnerability index. The 21 indices computed in this chapter utilized population health data that reflects the general health of elderly individuals as well as fine-scale estimates for green space that were not included in previous heat vulnerability mapping exercises. We hypothesized that some indices would identify different block groups as highly vulnerable, but that certain block groups would be consistently allotted a high vulnerability index value. We also hypothesized that there would be consistent spatial patterns across the 21 indices, despite the differing combinations of variables used in the index creation.

We observed both different statistical factor loadings and spatial patterns across the 21 indices, and four could be considered different based on their Spearman correlations and the display of spatial patterns. In creating the numerous HVIs using marginally different variables, we conclude that HVIs at the census block group level are sensitive to the data from which they are being created. Moreover, no one index could be considered clearly 'better' than another. The unstable performance of the indices suggests that users must be cautious in the use of HVIs. Yet, there does appear to be value in identifying the block groups most frequently assigned a high HVI value, as these block groups were identified as stable when comparing indices across their 'non green space' variables. Although the indices are visually appealing and serve as communication conduits for audiences not familiar with the complexities of heat-relate vulnerability, they must be interpreted and used with caution.

5.4. Chapter 4: Evaluating fine-scale estimates of heat-related vulnerability

The development of heat vulnerability indices presented in Chapter 3 reflect similar indices that have been calculated for other major urban areas in the US. Evaluation of the indices' performance in predicting differential relationships between extreme heat and a deleterious health outcome, such as death, is presented in Chapter 3.

Considering that little to no documentation of the use or application of a heat vulnerability index outside of general research exists, identifying whether the index is

able to convincingly attribute higher vulnerability index values to block groups where more people die during extreme heat events is critical to establishing the added value that constructing an index can provide. Contrary to our modest hypothesis that higher index values would be associated with higher risk of heat-related death in our Detroit, MI population, we observed no statistically significant associations between HVI measures and heat-related death. When investigating the how different levels of the HVI could be partitioned (e.g., above the median, at the 95th percentile), point estimates of the associations, though statistically non-significant, were in the opposite direction as expected, suggesting that higher values conferred protective effects of heat. While identifying spatial characteristics of heat-related vulnerability can be useful in displaying basic relationships mapped indices may not be useful for giving insights into probable high-risk areas for heat-related health problems when conducted at a fine scale.

5.5. Public health implications and future research needs

From a public health perspective, it is imperative to identify vulnerable populations and target interventions that can reduce an at-risk population's risk of morbidity or mortality to a particular exposure. The research presented here indicates that targeted interventions to reduce vulnerability may be difficult to develop solely based on information provided from a city-specific heat vulnerability index.

A working hypothesis posits that increased population density within an urban setting may contribute to increased risk of heat-related morbidity and mortality [1], although results are not consistent across all urban areas [2]. In considering the likelihood that the spatial distribution of population density is not uniform, as is assumed with the current HVI, one possible approach to better identify the spatial patterns of heat vulnerability is to use dasymetric data, which can account for variations in the distribution of population density and, thus, possibly provide a better characterization of the population characteristics associated with increased risk to heat exposure. It is currently estimated that nearly 80% of the US population lives in urban areas. This number is expected to increase with burgeoning population growth. Adequate response

to the growing population of potentially heat vulnerable individuals must consider these projections and further elucidate factors that can reduce risk.

The pressing need to continually update and refine methodologies to identify populations most vulnerable to heat requires critical evaluation of methods and data used to estimate the heat-health association, especially within known vulnerable subpopulations. Climate change adaptation programs and policies rely heavily on integrated assessments, many of which hinge on public health research such as that which is presented in this dissertation. It will be imperative that future research of the heat-health relationship focuses on precise methodologies that can accommodate the complex individual- and community-level characteristics that contribute to heat vulnerability.

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Appendix A.

A2.1 – A2.20. City-specific, census tract-level maps of subpopulation-weighted odds ratios of the association between cardiorespiratory mortality and extreme heat

Figure. A2.1. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Austin, TX

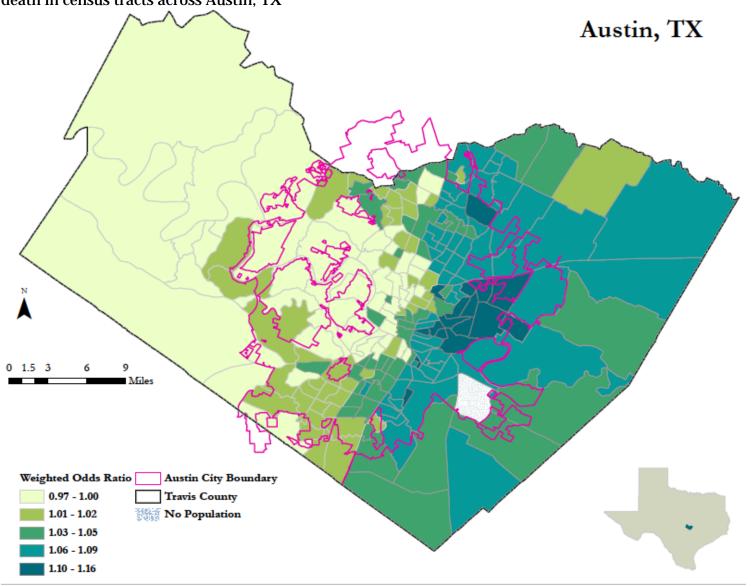


Figure. A2.2. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Baltimore, MD

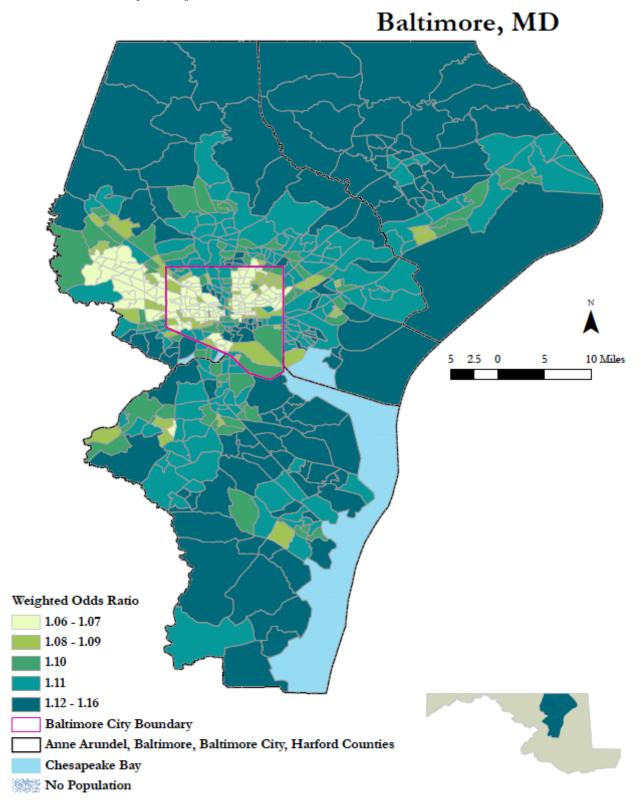


Figure A2.3. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Boston, MA

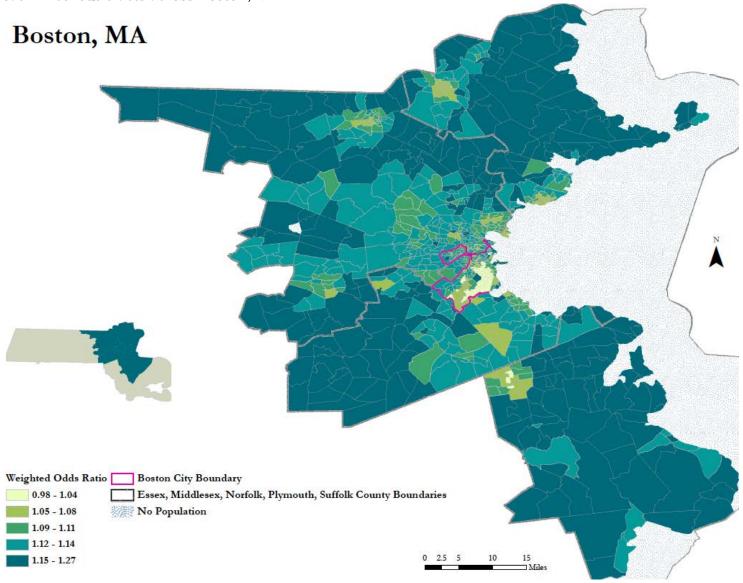


Figure. A2.4. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Chicago, IL

Chicago, IL

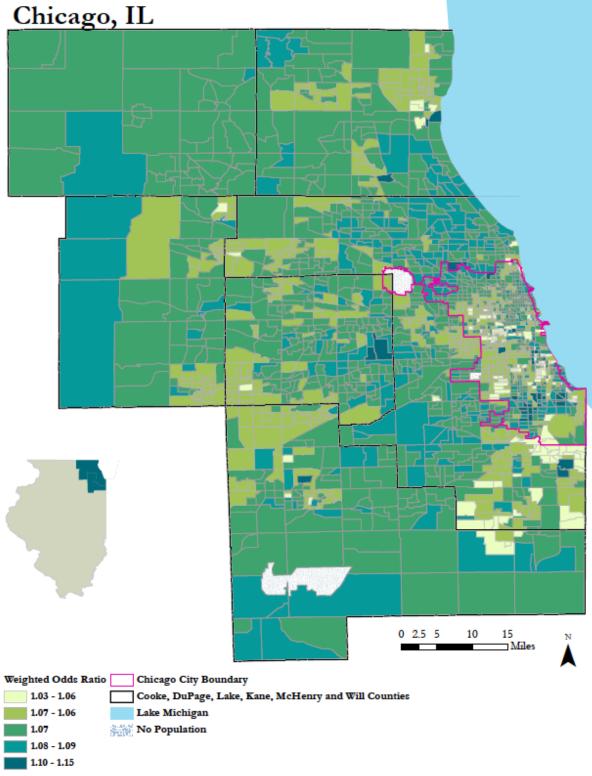


Figure. A2.5. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Columbus, OH

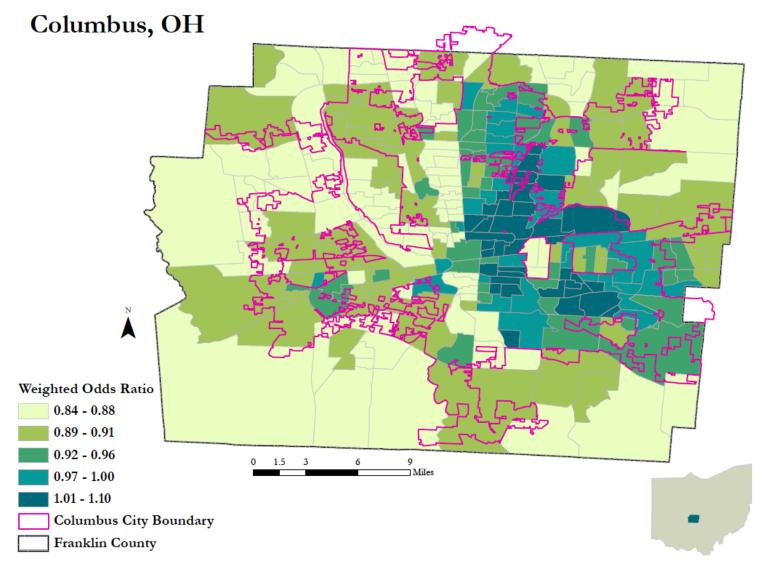


Figure. A2.6. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Dallas, TX

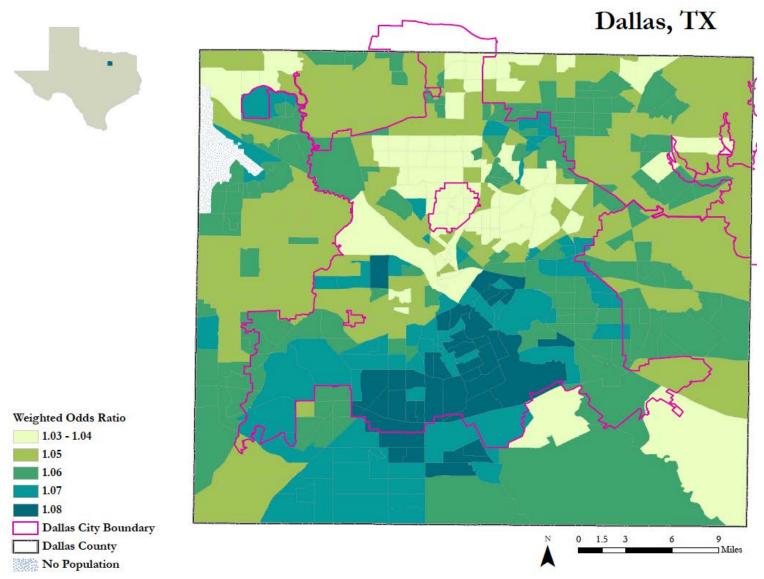


Figure. A2.7. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Detroit, MI

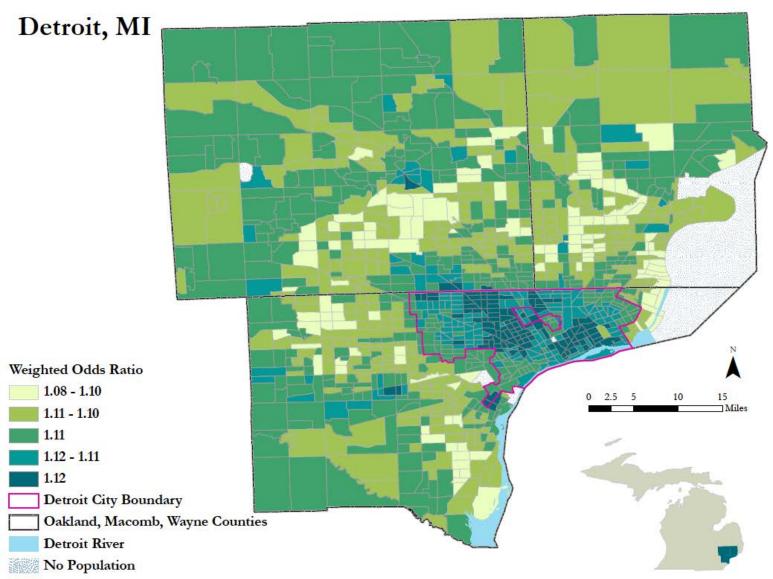


Figure. A2.8. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Houston, TX

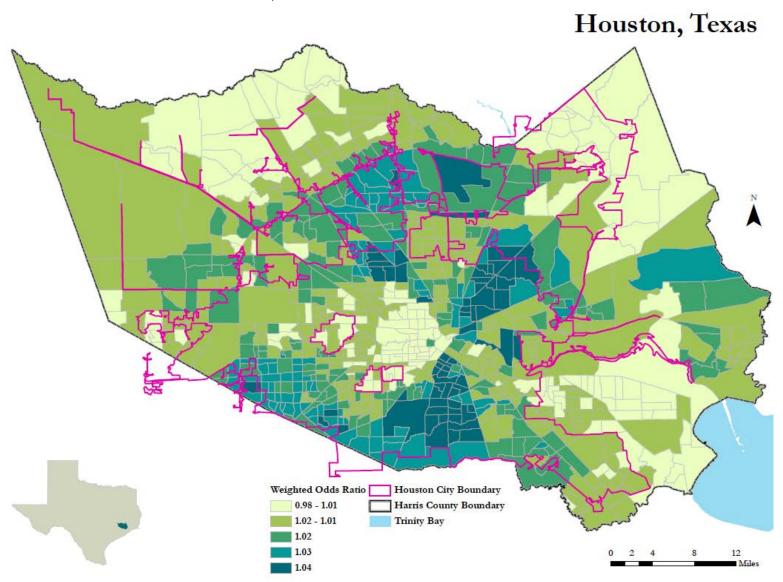


Figure. A2.9. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Indianapolis, IN

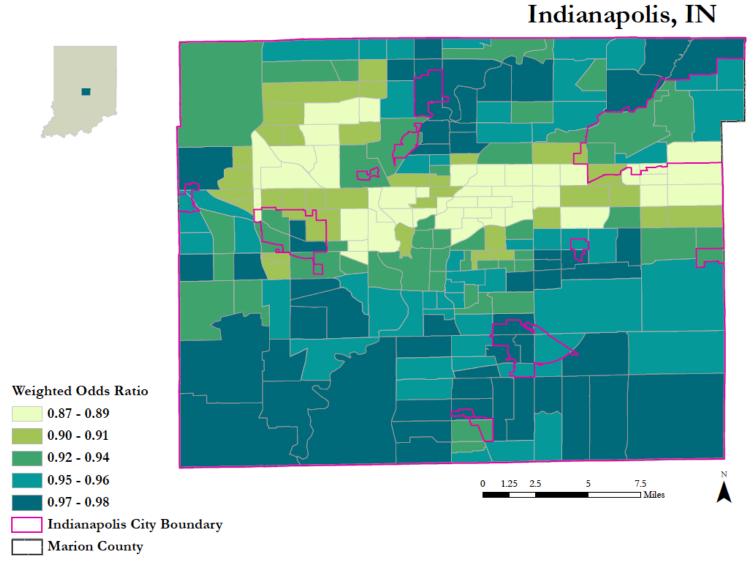


Figure. A2.10. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Jacksonville, FL

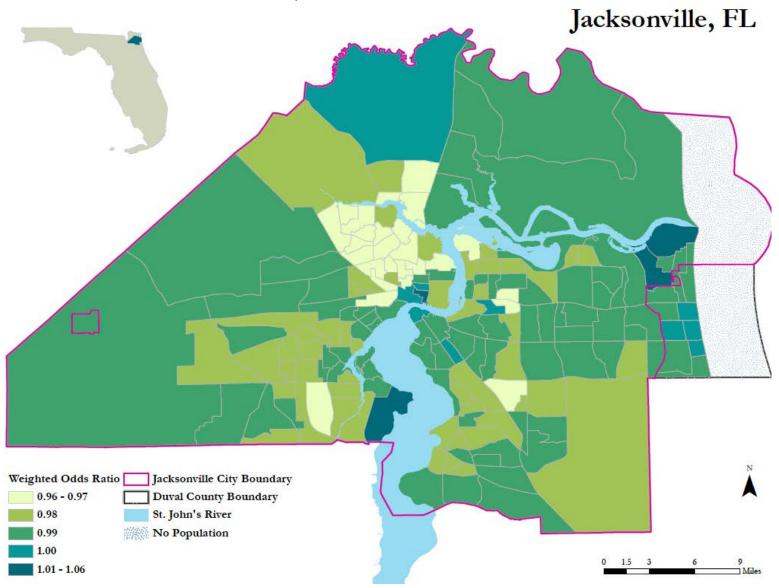


Figure. A2.11. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Los Angeles, CA

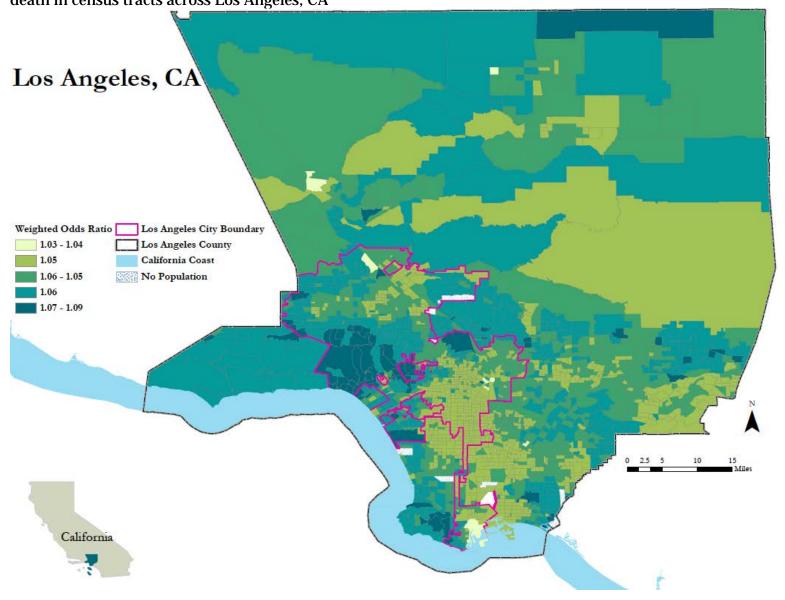


Figure. A2.12. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Memphis, TN

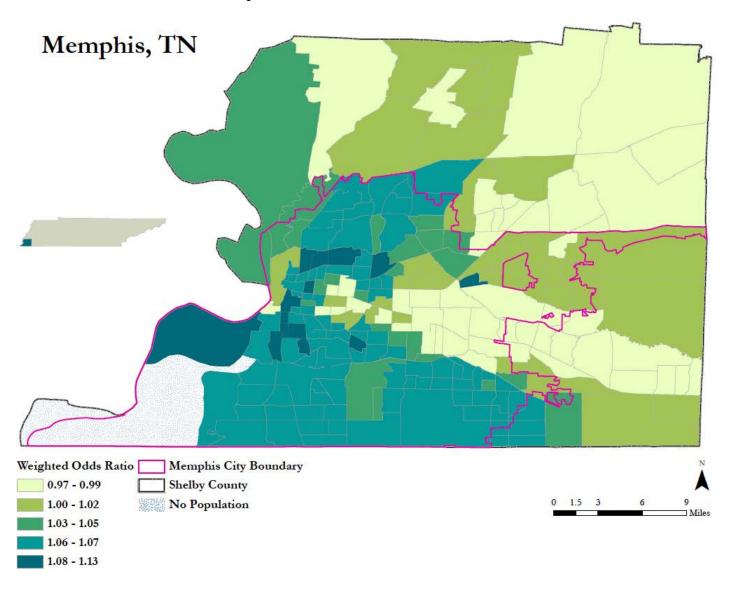


Figure. A2.13. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Milwaukee, WI

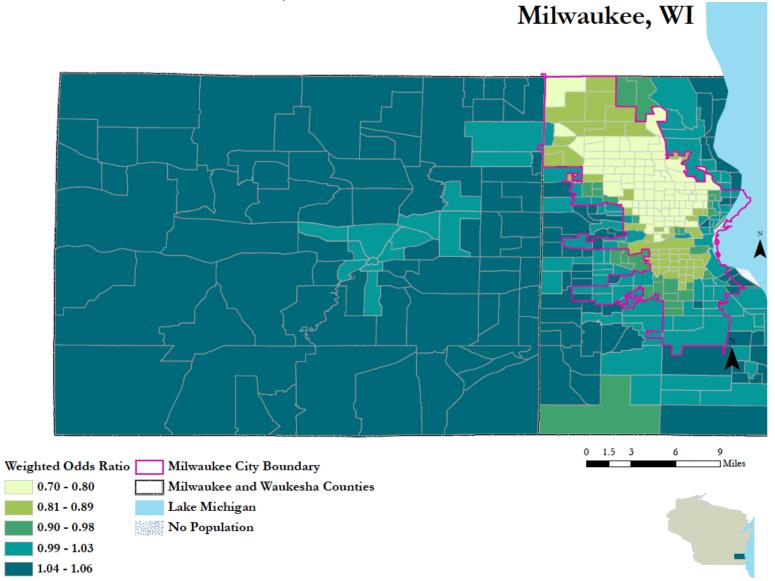


Figure. A2.14. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across New York City, NY

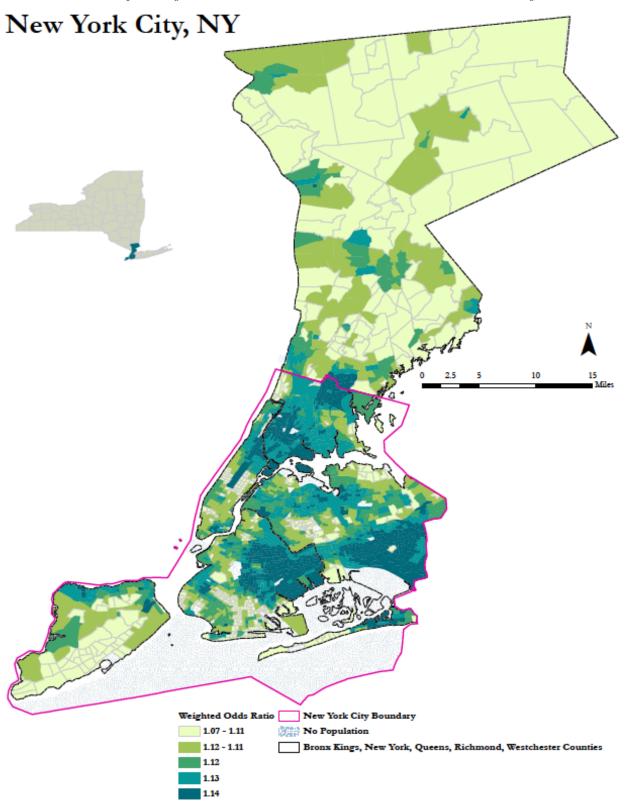


Figure. A2.15. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Southern New York City, NY

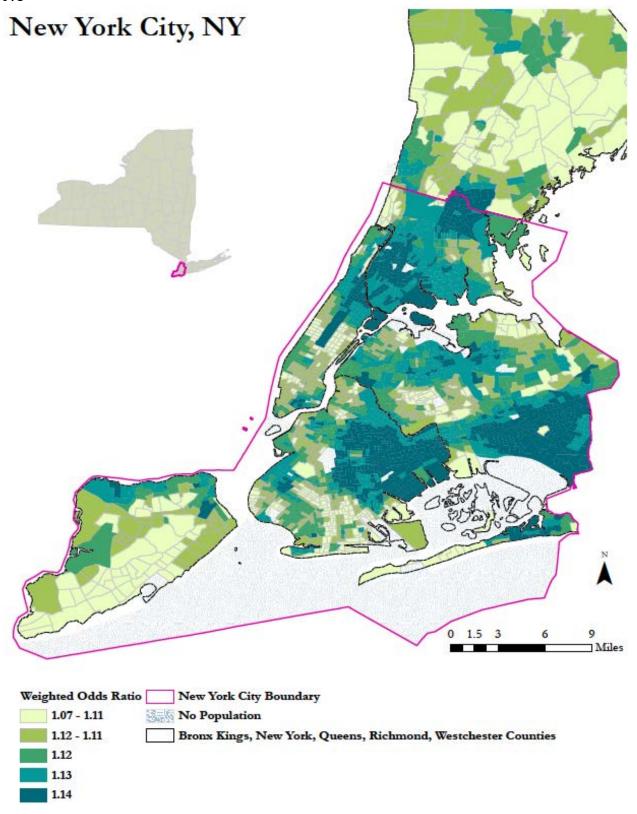


Figure. A2.16. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Philadelphia, PA

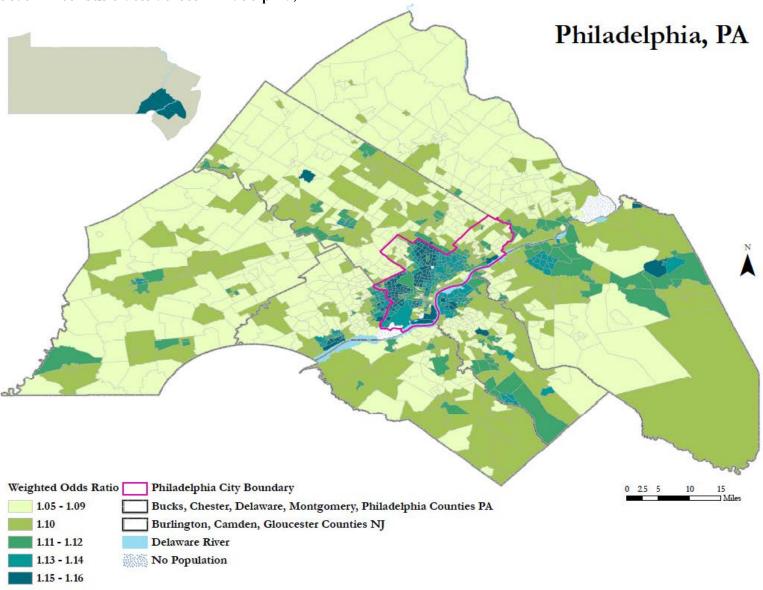


Figure. A2.17. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across Phoenix, AZ

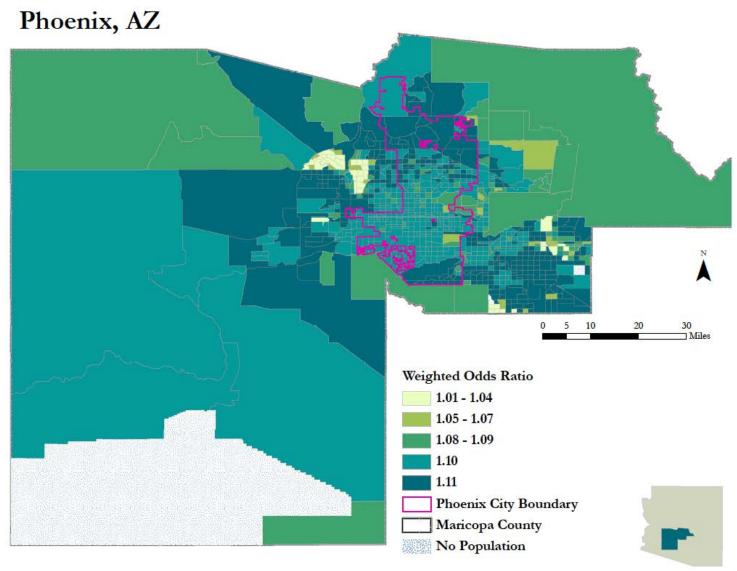


Figure. A2.18. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across San Antonio, TX

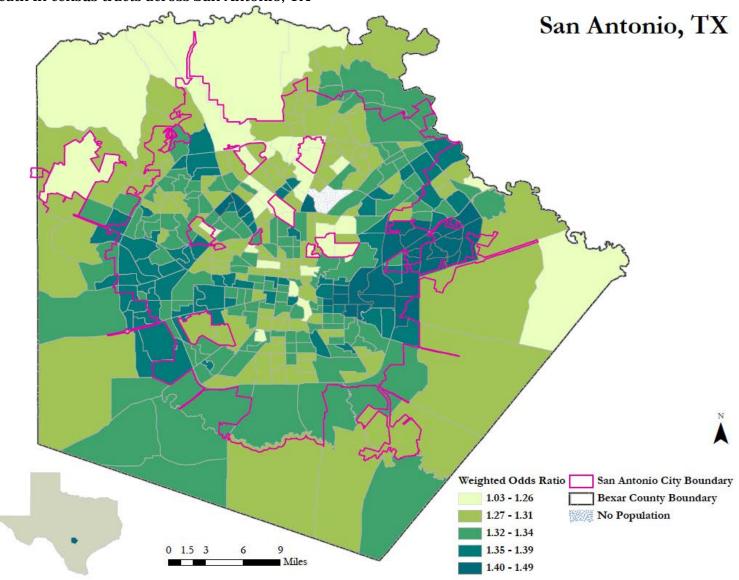


Figure. A2.19. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across San Diego, CA

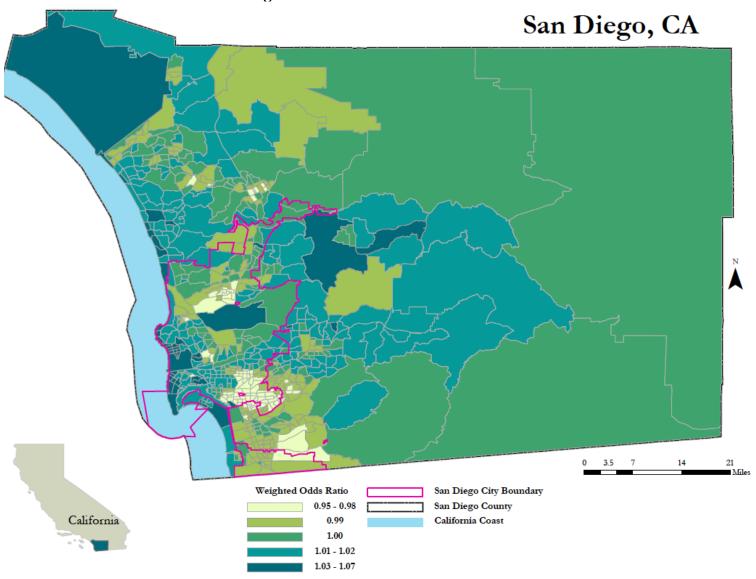


Figure. A2.20. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across San Francisco, CA

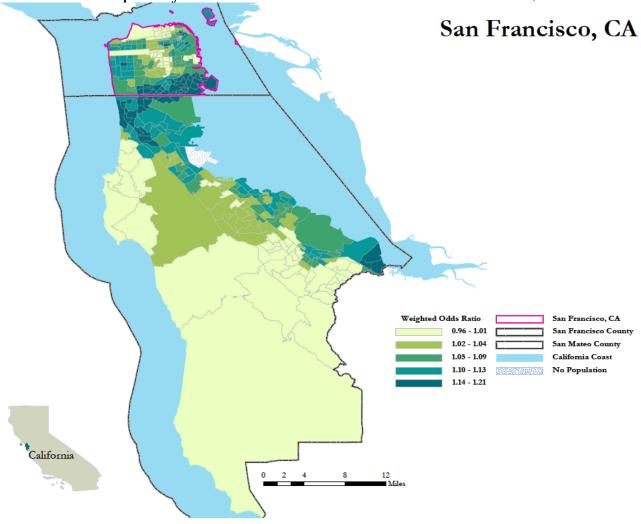
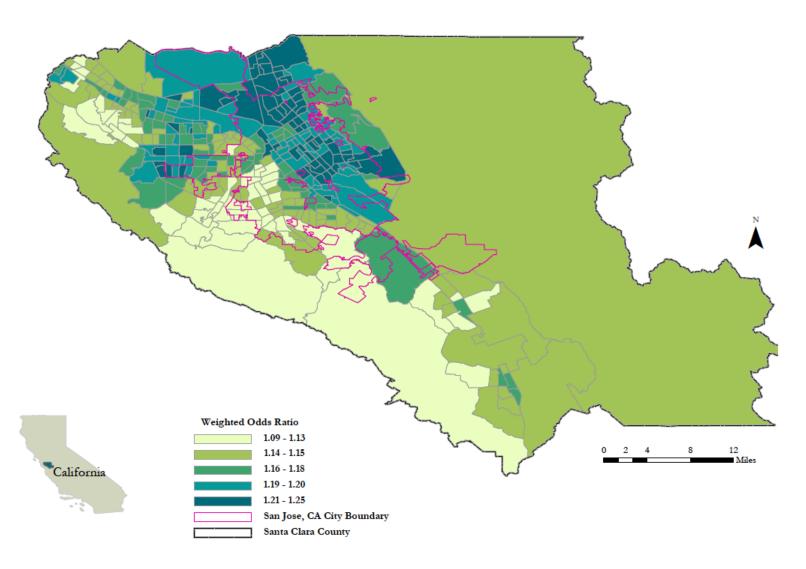


Figure. A2.21. Subpopulation weighted odds ratios of the association between extreme heat and cardiorespiratory-cause death in census tracts across San Jose, CA

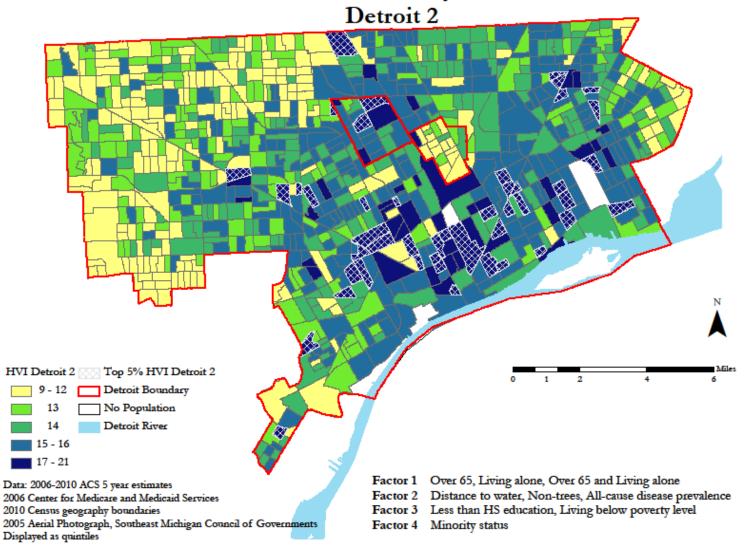
San Jose, CA

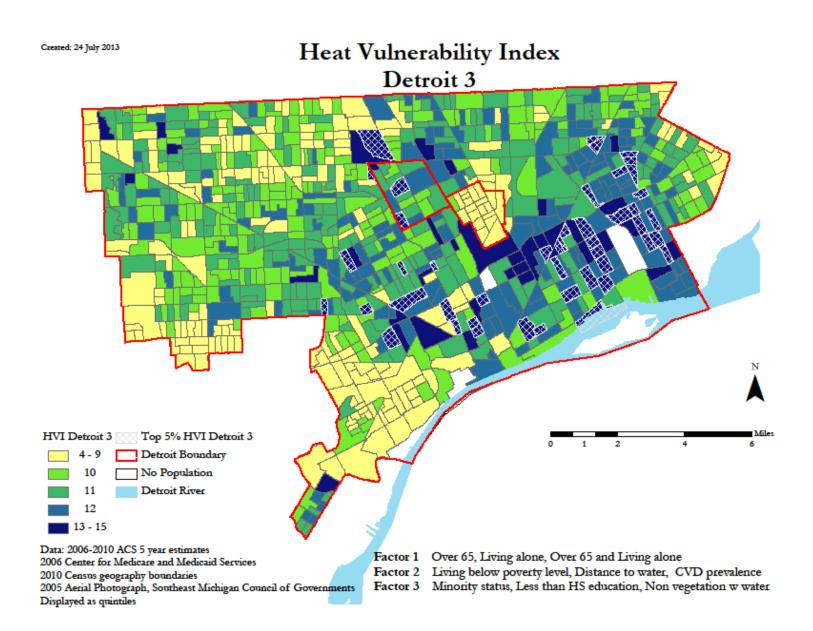


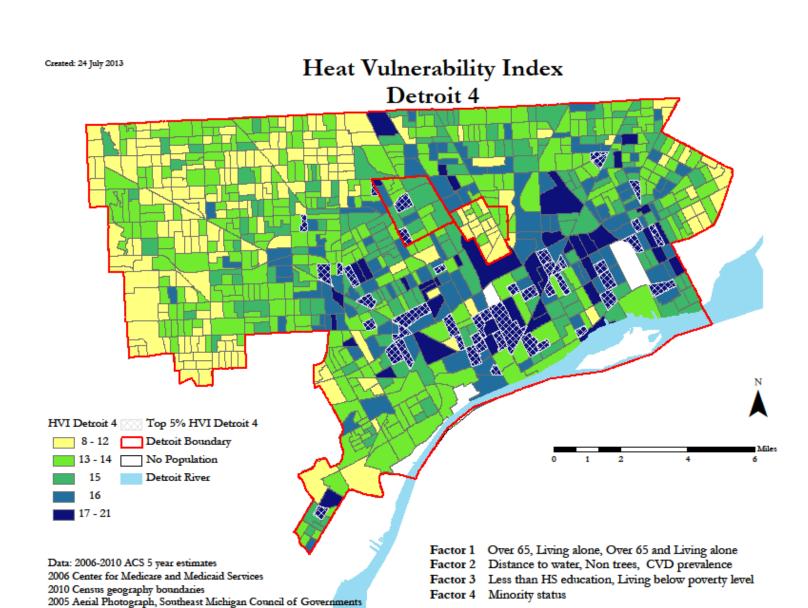
Appendix B.

Mapped heat vulnerability indices for all combinations described in matrix of Table 3.2, for Detroit, MI USA.



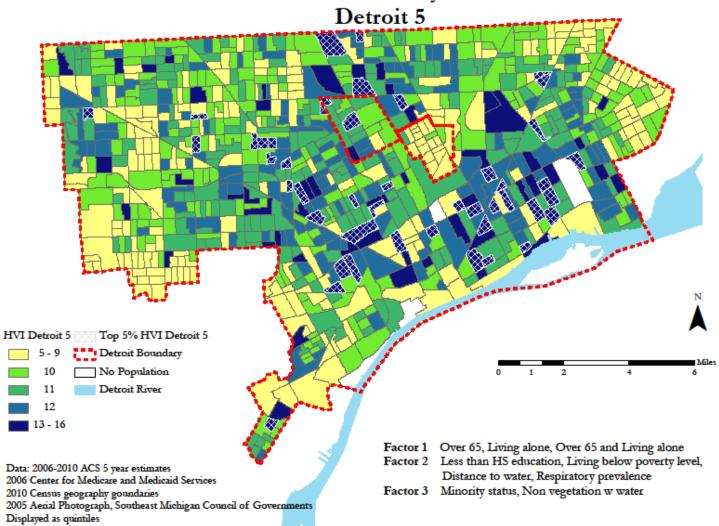




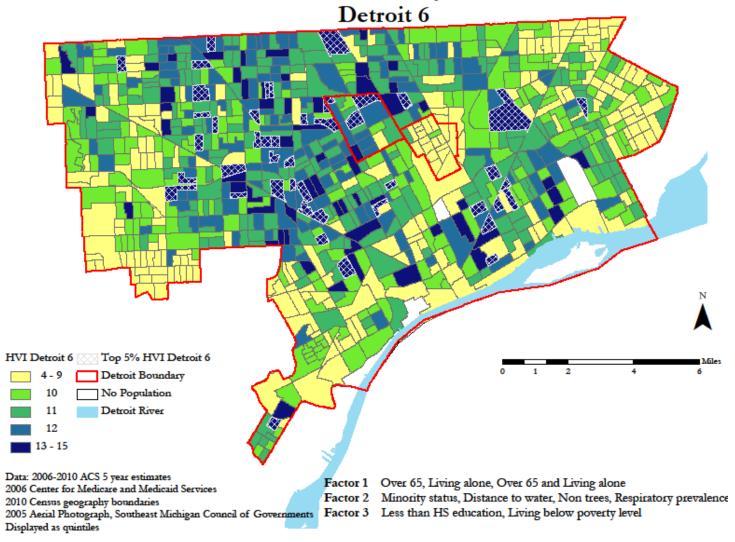


Displayed as quintiles

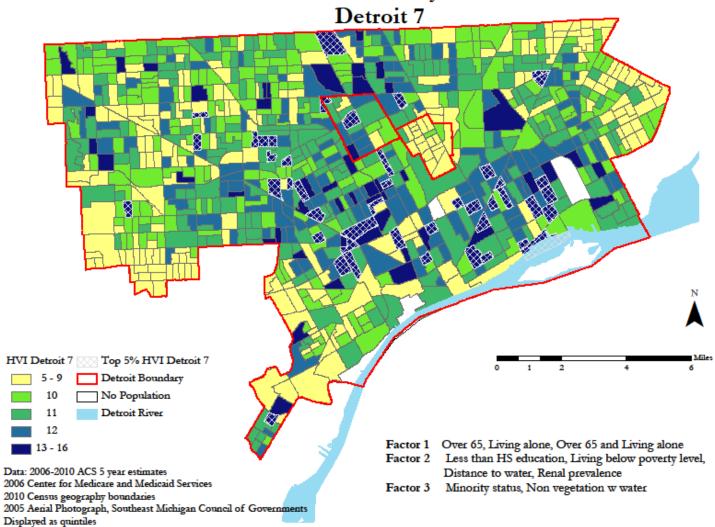




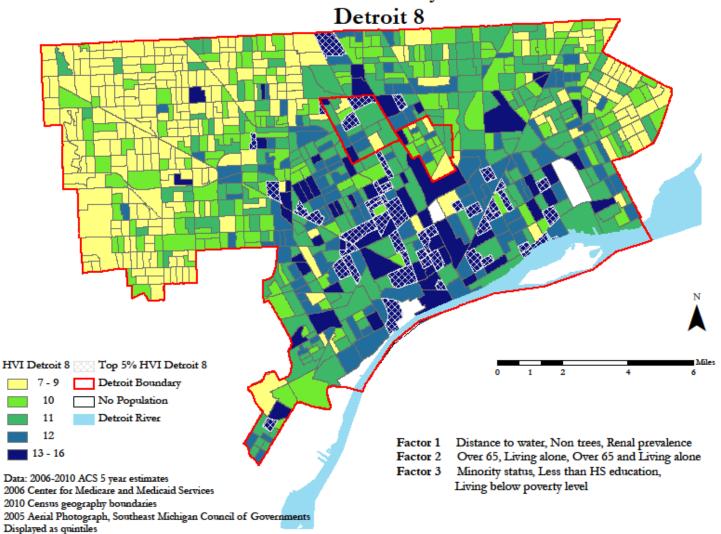




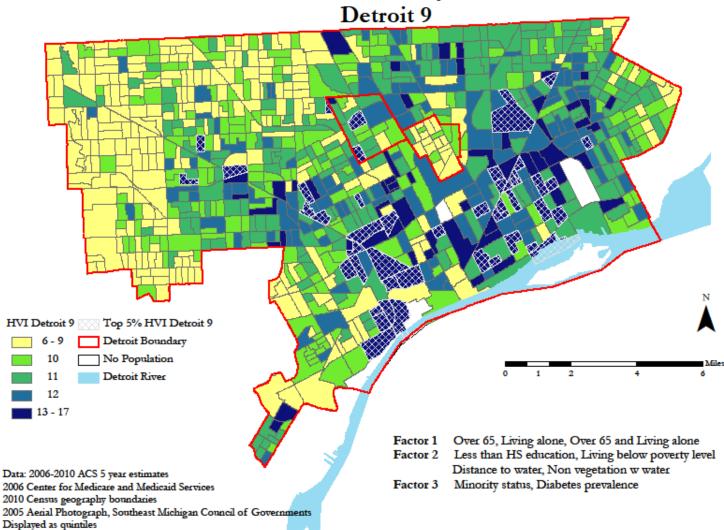




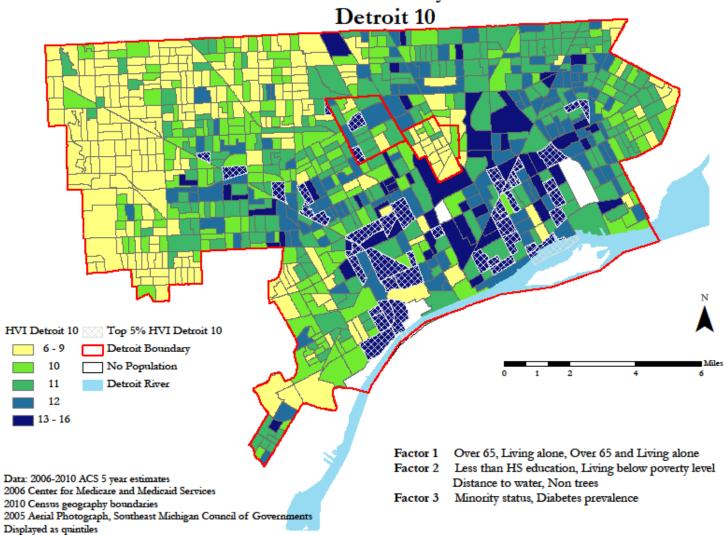




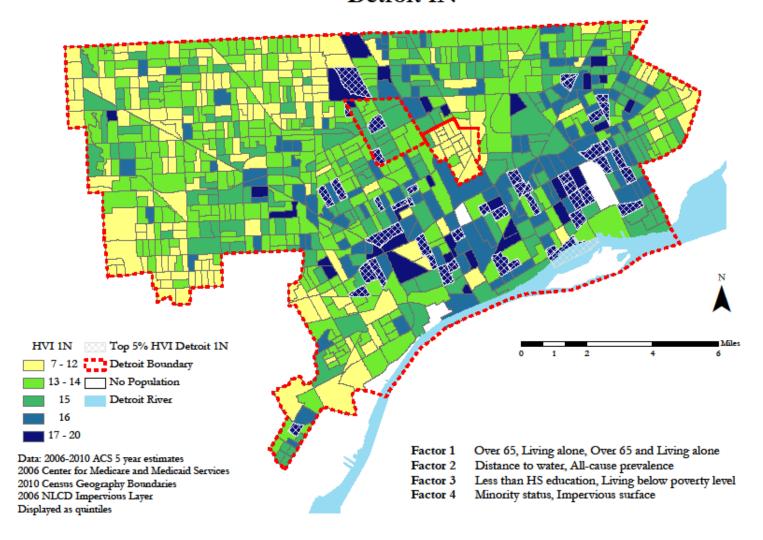


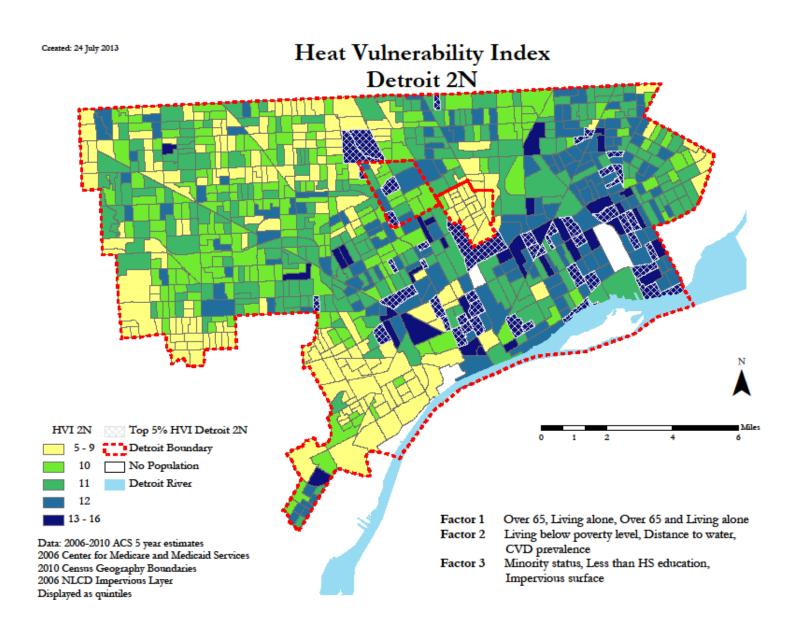






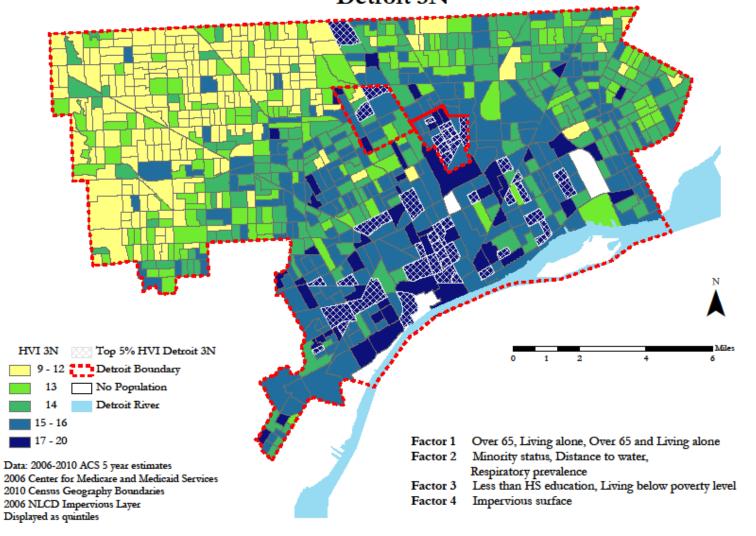
Heat Vulnerability Index Detroit 1N



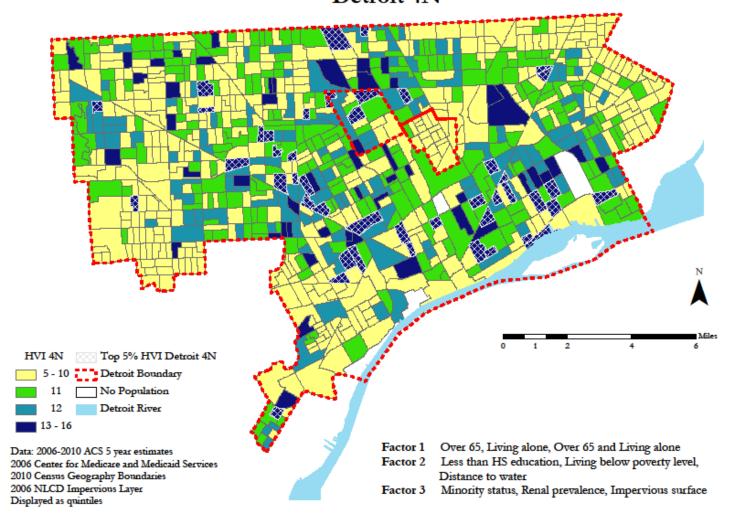


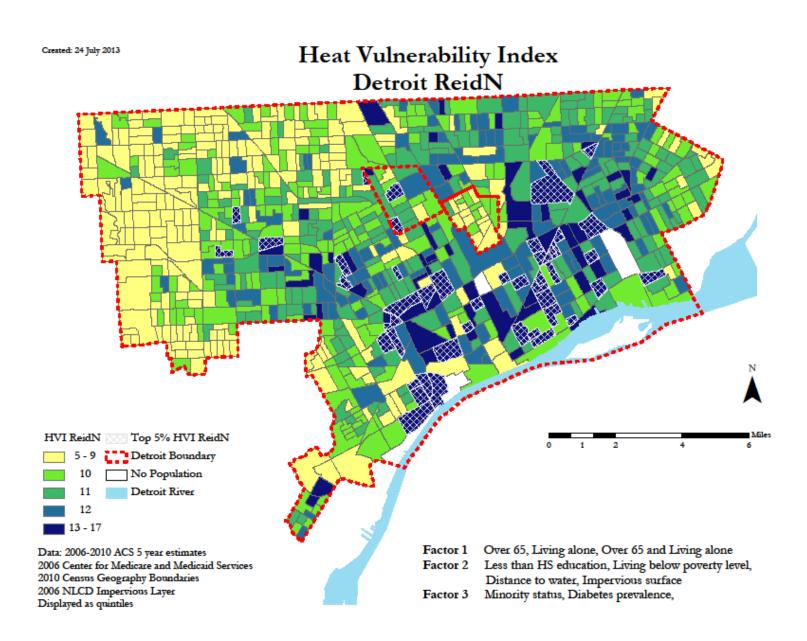


Heat Vulnerability Index Detroit 3N

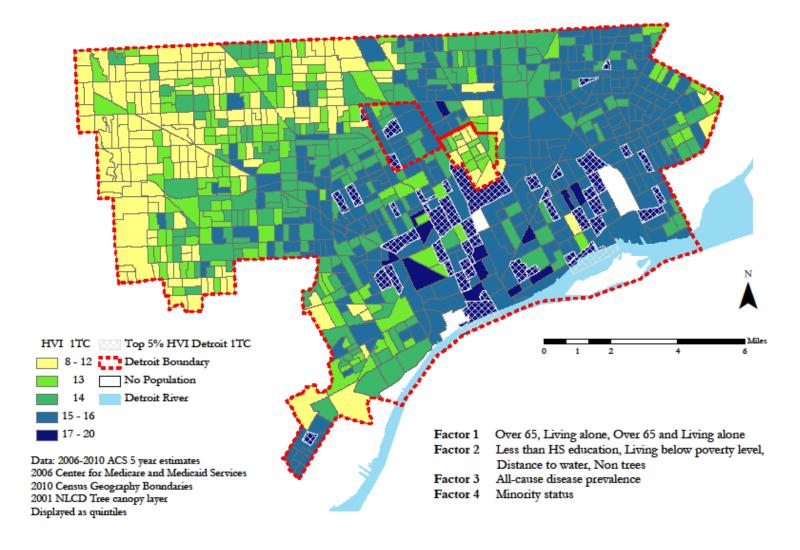


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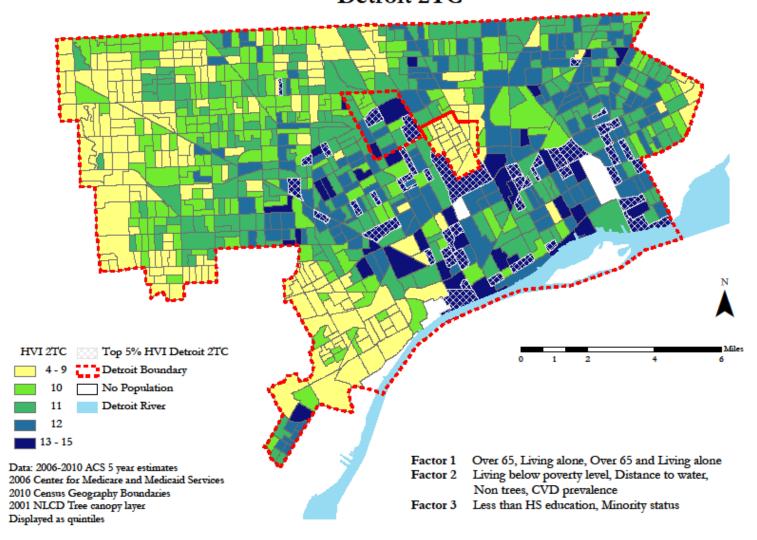




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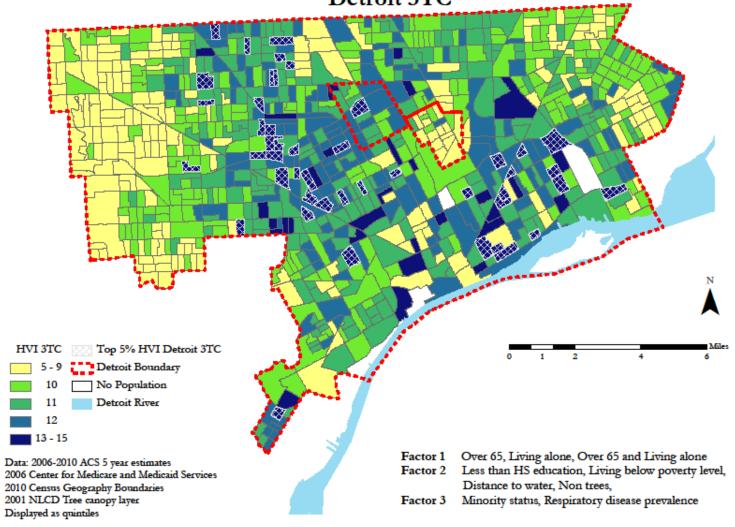


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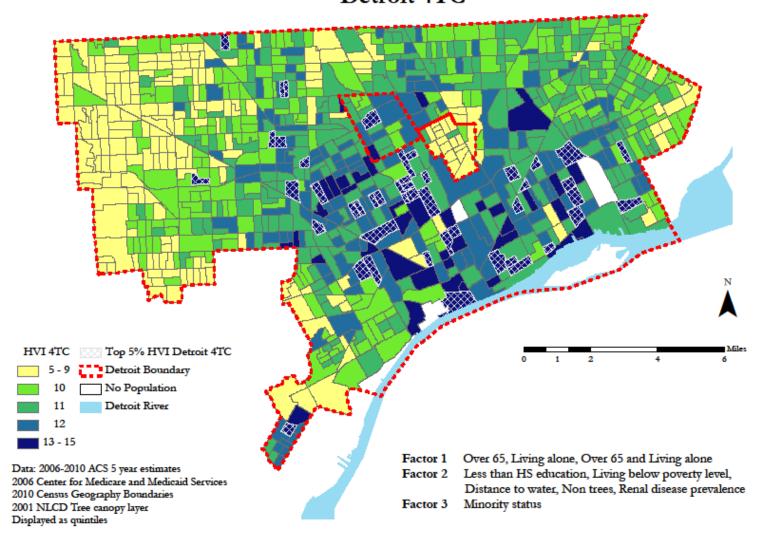




Heat Vulnerability Index Detroit 3TC



Heat Vulnerability Index Detroit 4TC





Heat Vulnerability Index Detroit ReidTC

