AMBIENT OUTDOOR HEAT AND HEAT-RELATED ILLNESS IN FLORIDA

Laurel Harduar Morano

A dissertation submitted to the faculty at the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Epidemiology in the Gillings School of Global Public Health.

Chapel Hill
2016

Approved by:

Steve Wing

David Richardson

Eric Whitsel

Charles Konrad

Sharon Watkins

ABSTRACT

Laurel Harduar Morano: Ambient Outdoor Heat and Heat-related Illness in Florida
(Under the direction of Steve Wing)

Environmental heat stress results in adverse health outcomes and lasting physiological damage. These outcomes are highly preventable via behavioral modification and community-level adaption. For prevention, a full understanding of the relationship between heat and heat-related outcomes is necessary. The study goals were to highlight the burden of heat-related illness (HRI) within Florida, model the relationship between outdoor heat and HRI morbidity/mortality, and to identify community-level factors which may increase a population's vulnerability to increasing heat. The heat-HRI relationship was examined from three perspectives: daily outdoor heat, heat waves, and assessment of the additional impact of heat waves after accounting for daily outdoor heat. The study was conducted among all Florida residents for May-October, 2005–2012. The exposures of interest were maximum daily heat index and temperature from Florida weather stations. The outcome was work-related and non-work-related HRI emergency department visits, hospitalizations, and deaths. A generalized linear model (GLM) with an overdispersed Poisson distribution was used. GLMs were run for each sub-region within Florida and statewide estimates were obtained via random effects meta-regression. The results of the burden analysis indicated that the rates of HRI varied by geography, data source, and work-related status. The sub-groups with the highest relative rates were for males and rural residents. HRI rates increased with increasing heat index/temperature. The strongest effect was associated with the current day's heat index/temperature. As heat index/temperature increased, at higher heat index/temperature values, there appeared to be some heat adaptation. For a Florida specific heat wave definition, duration should be two days or more above an intensity threshold which is defined by a constant value for heat index or an area varying relative value. Focus on heat waves is not appropriate for Florida. Community-level factors which may

identify vulnerable Florida populations include populations with a high proportion of: impervious surfaces, renters, or individuals reporting Black race alone. This is the first study to explore the heat-HRI relationship stratified by work-related status and is the first to fully evaluate the heat-HRI relationship in Florida. This study highlights the importance of studying and mitigating the effects of heat in a humid sub-tropical climate.

To all the pray warriors in my life whose prayers have protected me, sustained me, and guided my path. And to my grandmothers, Leceta Pryce, Octavia Richardson, and Muriel Carpenter; your prayers, hard work, sacrifice, and unconditional love are the foundations of my success.

ACKNOWLEDGEMENTS

This work would not have been possible without the collaboration of my many colleagues at the Florida Department of Health, especially Meredith Jagger and Kristina Kintziger. I would like to thank Melissa Griffin who provided meteorological data and answered all of my Florida specific metrological questions. I would like to thank my committee members for their guidance and all their wonderful feedback. I would especially like to thank Steve Wing who has been my advisor throughout my doctoral program. He has always asked the right question at the right time and his philosophy of epidemiology, research, and the spoken word has been has been invaluable to me. I feel blessed to have had the chance to work with him. I am ever grateful to Sharon Watkins who has been my mentor for almost a decade, who encouraged me to start this process, and who has provided me much needed prospective throughout the program. To Jessica Rinsky who was my dissertation partner even after she graduated, I am grateful for her support, wordsmithing, and being an amazing sounding board. To all my friends and classmates who read, edited, and provided feedback on this document – thank you. I would like to acknowledge my family who has always supported me and expected the best of me. Finally, I am forever grateful to my husband, Aaron Morano, who joined me on this roller coaster of ups and downs that is a PhD program. His support and unfailing confidence in me is amazing.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF EQUATIONS

LIST OF ABBREVIATIONS

ACS	American Community Survey
AHCA	Agency for Health Care Administration
AIC	Akaike information criterion
AWOS	Automated Weather Observation System
ASOS	Automated Surface Observing System
BRFSS	Behavioral Risk Factor Surveillance
C	Celsius
Cal/OSHA	California Occupational Safety and Health Administration
CDC	Centers for Disease Control and Prevention
CFOI	Census of Fatal Occupational Injuries
CI	Confidence Interval
CO	Carbon monoxide
COOP	Cooperative Observer Program
CPS	Current Population Survey
DF	Degrees of freedom
Ecode	External cause of injury code
ED	Emergency department
F	Fahrenheit

FAWN	Florida agricultural weather network
FDOH	Florida Department of Health
FTE	Full time equivalent
HRI	Heat-related illness
HHWS	Heat health warning systems
HVI	Heat Vulnerability Index
HW	Heat wave
ICD-9-CM	International Classification of Diseases, Ninth Revision, Clinical Modification
ICD-10	International Classification of Diseases, Tenth Edition
IQR	Interquartile range
IRR	Incidence rate ratio
IPCC	Intergovernmental Panel on Climate Change
L	Liter
LRT	Likelihood ratio test
mL	Milliliter
MRLC	Multi-Resolution Land Characteristics Consortium
NLCD	National Land Cover Database
NO2	Nitrogen dioxide
NWS	National Weather Service
OR	Odds ratio

OSHA	Occupational safety and health administration
PM10	Particulate matter less than 10 micrometers
PM2.5	Particulate matter less than 2.5 micrometers
PM10	Particulate matter less than 10 micrometers
RHRC	Rural Health Research Center
RR	Rate ratio
SD	Standard deviation
SE	Standard error
SES	Socio-economic status
SOC	Standard Occupational Classification
SOII	Survey of Occupational Injuries and Illnesses
SSC	Spatial synoptic classification
U.S.	United States
WBGT	Wet bulb globe temperature
WFO	Weather forecasting office
WWAMI	Washington, Wyoming, Alaska, Montana, and Idaho
ZCTA	Zip code tabulation area

CHAPTER 1. INTRODUCTION

Environmental heat stress is associated with increased morbidity and mortality among humans. The general physiological mechanism is clear; when exposed to prolonged heat the body loses the ability to maintain thermo-equilibrium resulting in illness and death. However, humans have an innate ability to adapt to their environment. As such, behavioral modifications, when implemented, have a direct impact on the prevention of heat-related outcomes. Regardless, heat can be a hazardous to humans, even in tropical and sub-tropical climates where individuals and communities are adapted to their environment [1-4].

The National Weather Service (NWS) estimates that between 1986 and 2015 the average number of heat-related fatalities per year in the United States (U.S.) was higher than any other weather-related occurrence [5]. Over the last 30 years, numerous heat-waves across the globe have resulted in an awareness of environmental heat as a public health threat [2, 6-8]. This has led to increased surveillance of heat-related outcomes, identification of individual- and community-level risk-factors, and research into the relationship between heat and heat-related outcomes both during and outside a heat wave [2, 9-12].

It has been identified that the burden of heat-related outcomes varies from population to population depending on the availability of resources to implement adaption strategies (e.g., land-use/urban planning, development of heat plan, or public health education) and to mitigate health effects (e.g., provision of cooling centers or access to medical care) [13-15]. Further, within a population there will be sub-populations (e.g., elderly, socially disadvantaged, disabled) who are at higher risk of a heat-related outcome [15, 16]. Individuals not traditionally viewed as vulnerable may also be at increased risk of heat-related outcomes due to their activities, such as outdoor workers or athletes [16]. The proportion of the types of susceptible groups (e.g., elderly or athletes) within a

population may vary, impacting the overall population's ability to adapt to increasing heat [14, 16]. Additionally, studies of the relationship between heat and heat-related outcomes have resulted in the development of local strategies, triggered by weather forecasts, for mitigating the effects of heat and protecting the population.

However, much of this work globally, has been conducted in higher latitudes and the impact of heat on populations in sub-tropical and tropical climates has been assumed to be minimal [17-19]}. As a result there is a large gap in the literature about the burden of heat-related outcomes, the identification of risk factors, and the relationship between heat and heat-related outcomes in lower latitudes.

Environmental heat is increasing as a result of global climate change. The mean global temperatures between 1906 and 2005 increased by 0.7°Celsius (C) with the majority of the increase occurring in the latter time period – an increase of 0.13°C per decade between 1956 and 2006 [20]. The Intergovernmental Panel on Climate Change (IPCC) reports that since the 1950's the annual number of extreme heat events (heat waves) as well as the annual number of warm nights have increased globally. To a lesser degree, the annual number of hot days has also increased [21]. The IPCC defined a warm night or day as a day where the minimum or maximum temperature, respectively, exceeds the 90^{th} percentile based on the historical distribution of daily temperature [21]. An extreme heat event is an episode of several consecutive warm days and nights [21].

With the changing of the global climate, morbidity and mortality as a result of exposure to heat is projected to increase [15]. The magnitude of the impact is expected to vary based on latitude and location [20]. Therefore, in order to reduce the current and future morbidity and mortality, a clear picture of the current burden of heat-related outcomes in <u>all</u> latitudes is required, as well as an understanding of the relationship between heat and mortality/morbidity at the population and sub-population level [2, 14, 16].

CHAPTER 2. REVIEW OF THE LITERATURE

HISTORICAL BACKGROUND

Throughout recorded history there has been an observable association between high temperatures (or hot days) and morbidity/mortality. Early records of heat-related illness (HRI) are mostly from military campaigns. Both the Bible[1] and Homer's Iliad spoke of HRI during military operations [3]. In 24 BCE, a roman military campaign to Arabia was unsuccessful due to HRI among the soldiers [22]. In 400 BCE, Herodotus recorded the relationship between work, heavy clothing, and heat stress in a battle between the Athenians and the Spartans [3]. According to Plutarch (50-120 CE), at the end of Alexander the Great's campaign he lost approximately 75 percent of his soldiers and entourage to dehydration during the two month (September/October) march from the Hyphasis River[2] to the Persian palace at Pura [3, 23]. During the crusades, King Edward's heavily armor-clad soldiers lost the final battle with the Saracens, who wore light armor, in large part due to HRI [24]. Additionally, the English soldiers did preparations for battle during the heat of the day, whereas, the Saracens completed their preparations in the cool of the evenings; an early historical reference to a behavioral heat adaption strategy [24]. Further behavioral modification was reported in 1774 when De Meyserey, a French army surgeon, proposed the use of white leather helmets by soldiers to prevent the effects of the sun's rays [25].

Among the civilian population, HRIs were also alluded to in the Bible[3] [26]. In ancient Greece, heat stroke was referred to as siriasis after the dog-star Sirius [27]. In ancient times this star was

---

[1]Joshua 10:12-13

[2]Modern name = Beas River, in northern India

[3]Judith 8:3 and 2 King 4:18-20

associated with the hottest months of summer. In 1902, in his treatise to the American Medical Journal of Sciences, Dr. Joseph Spellissy laments that there is very little information available between Antiquity and the writings of Domini Petri Foresti who spoke of an epidemic of apoplexy[4] during hot and humid conditions in Italy in 1562 [30]. Until about 1850, HRI was usually confused with apoplexy [31]. The identification of historical instances of HRI is often due to the symptoms recorded and the time of year or temperature reported. For instance, Gjuro Baglivi observed an epidemic of apoplexy during the unusually hot summers of 1694 and 1695 in Rome [30, 31]. In 1789, Dr. Benjamin Rush described the symptoms of HRI but suggested the disorder was caused by drinking cold water in warm weather [30]. In 1818, citations were posted at water pumps in Philadelphia warning of sudden death from drinking cold water [31].

Within the 19th century, cases of suspected HRIs were medically observed, treated, and autopsied. At the beginning of the 19th century the common treatment for HRI was blood-letting. Towards the end of the 19th century, the medical community shifted their treatment methods to the more modern day treatment of cooling the individual [30]. This was in part spurred on by mid-19th century findings identifying HRI as a separate disease from apoplexy and the findings that the disorder *caused by drinking cold water* originated from the body overheating [30]. The idea that heat was responsible for the illnesses and not the sun alone was also observed by medical experts of the time, mainly from observations in an occupational setting such as deaths in the military, in sugar refineries, in laundries, and the illness of a Black preacher giving a sermon in a crowed and overheated church [25, 26, 30, 31]. At this time, the leading medical experts began to suspect that high humidity was also a factor in the afore mentioned occupational cases, as well as for cases seen in the humid climates of India and the West Indies (e.g., Barbados)[5] [25, 26, 30].

---

[4]Prior to the middle of the 19th century, apoplexy was used to describe conditions where an individual fell unconscious for no apparent reason [28]. Over the centuries it has also been associated with stroke and convulsions [28, 29].

[5]An experiment done by Blagden and Fordyce in 1775 indicated that men “of good health” suffered no ill effects when standing in dry heat at 250°F for 15 minutes. When placed in humid heat 54.4°C (129.9°F) for the same time period, the men’s temperatures rose to 37.8°C (100°F). Regardless of

In the early 20th century, the burden of heat-related disease started to be quantified including trends over time and geographical and demographic variations. Shattuck and Hilferty noted that in the United States Registration Area[6], the "Heat and Sunstroke" death rate for 1901, a particularly warm year with a hot summer, was 12.8 per 100,000 population [33]. Comparatively, the 29 year (1900-1928) average "Heat and Sunstroke" death rate was 1.9 per 100,000 population. Shattuck and Hilferty also conducted a detailed analysis of Massachusetts and observed that during years with heat waves, urban districts had a higher mortality rate than rural districts [34]. In 1938, Grover presented an analysis of excess mortality associated with elevated temperatures in 86 U.S. cities from 1925-1937 [35]. She noted the magnitude of effect varied by latitude with the greatest effect seen in the northeast. Grover also noted in her 1938 analysis that the majority of excess deaths due to heat were not coded as heat-related deaths on the death certificate. Considering her findings, Grover suggested that all-cause mortality would be a better measure than heat-related deaths [35]. In his discussion of the 1966 heat wave in the U.S., Schuman noted that "…poverty, crowding, poor housing, and age are critical factors" in heat-related mortality [36]. He also commented that planning and preparation by city officials, urban planners, and medical personnel for heat events could reduce heat-related mortality.

During the first half of the 20th century both the British and U.S. military worked extensively to prevent adverse heat-related outcomes and to understand the susceptibility factors. For instance, by 1917 in Mesopotamia the British provided heat stroke stations which were supplied daily with ice. Where possible, troops did not work between 10am and 2pm and were provided education on how to mitigate the effects of heat. During their time in Mesopotamia, risks factors for heat exhaustion and heat stroke, such as increasing age and concurrent infections, were identified. A 1927 study of U.S. Navy casualties from 1861-1926 observed that acclimatization was a prevention factor for casualties due to heat [31]. During World War II, the U.S. Army observed that the majority of heat casualties

---

this study, the general belief till the latter half of the 19th century was that the sun's rays alone cause heat illness [25].

[6]"… all states and cities having at least 8,000 inhabitants for which the registration of deaths under local laws and ordinances was found to be sufficiently accurate for use by the Census Office."[32]

were occurring during training, as such, guidelines for work-rest cycles based on wet bulb temperature were established [25]. These guidelines were based on military studies of the meteorological conditions at the time of a heat-related death [25, 37].

In the latter quarter of the 20th century, a clear association between high temperatures and heat-related morbidity and mortality became apparent. However, as was underscored by Klinenberg's social autopsy of the 1995 Chicago heat wave, behavioral responses (individual and population level), community cohesion, and socio-economic factors are just as (or possibly more) important as the meteorological factors in understanding the heat-health relationship [7]. Note that within this paper the heat-health relationship refers to the relationship between heat and heat-related morbidity or morality. When individuals are not able to employ adaption strategies (e.g., change their behavior) they are more likely to succumb to heat-exposure regardless of the temperature. For instance, a low income senior without an air-conditioned home who feels safe leaving their home, frequenting businesses, and visiting neighbors will have a lower risk of a HRI than a similar senior who does not feel comfortable leaving their home. During the Chicago heat wave, seniors who left their home had access to individuals who could provide help if needed and access to cooling by way of commercial centers, all of which reduced their risk of heat illness [7]. Explicit assessments of many of the non-meteorological factors are not always practical. Still, a solid foundation for policy interventions and future work should be made by thoroughly characterizing the relationship between HRI, environmental heat, and, where explicitly possible, non-metrological factors [2, 14, 38-40].

PHYSIOLOGY

Through physiological and behavioral modifications, the human body is able to maintain temperature equilibrium of 37°C ± 0.5 [41, 42]. Heat produced internally, through metabolic processes, or absorbed from the external environment, can raise body temperature if not dispersed properly. If temperature equilibrium is not maintained the body quickly deteriorates leading to illness and death. Within this section the physiological mechanisms of this balance and mechanisms of heat loss will be discussed.

The maintenance of temperature in the human body can be conceptualized in two parts, the core and the shell (Figure 2-1). The core maintains a temperature of 36.6-36.8°C[7] (97.9-98.2°F) and roughly includes the brain, parts of the skeletal muscles, and vital organs [44-46]. The core can tolerate very little variation in temperature and over the course of the day varies by only 0.6°C (1.1°F) [47]. On the other hand, the shell, which is comprised of the outer tissues of the body (e.g., skin) varies as external environmental conditions change as well as with the thermoregulatory state of the body (i.e., core temperature lower or higher than normal) [45]. The mean temperature of the shell is approximately 33.7-34°C (92.7-93.2°F) [45]. The physical distinction between the two parts is not exact and varies dependent on the external temperature. In a warm or hot environment the shell may comprise 10-20 percent of the human body mass while in cold conditions the shell may comprise 30-40 percent of the human body mass [41]. The temperature gradient between the core and the shell is important in passive thermoregulation and will be discussed later.

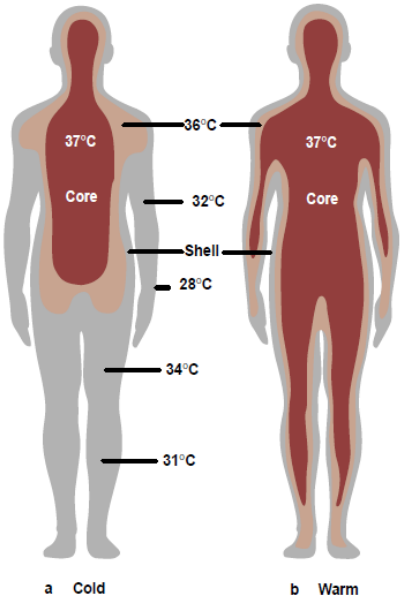


Figure 2-1. Human body temperature maintenance conceptualized in two parts. For cold periods (a) the core is reduced in order to conserve body heat for crucial areas. For warm periods (b) the core is increased to facilitate whole body cooling. (Reproduced from Wenger 2001[46])

MECHANISMS OF HEAT DISSIPATION

Internally, heat is produced as a by-product of cellular metabolism and the mechanical work of the skeletal muscles [48]. These processes are highly inefficient. Only 27 percent of the energy obtained from food is used by the body's functional systems, the rest becomes heat and must be removed from the body [47]. Heat is removed from the human body through any of the following heat

[7]This temperature value is the oesophageal temperature and is an approximate value of core. The value of the core temperature depends on where the measurement is obtained (e.g., oral, tympanic, rectal, or oesophageal). For instance rectal temperature is usually about 0.6°C higher than oral temperature [43].

transfer mechanisms: radiation, conduction, convection, and evaporation [47, 48]. Radiation is the transfer of heat through infrared heat rays (wavelengths of 5-20 micrometers). All objects not at absolute zero temperatures radiate heat. When the external temperature is less than the body temperature then the body will radiate a greater amount of heat than it absorbs. Conduction is the transfer of heat from one object to another through kinetic energy. Molecules in the warmer object vibrate and move more than molecules in the cooler object. As the faster moving molecules collide with the slower moving molecules, energy is transferred. In this manner heat is transferred down a temperature gradient. The human body, especially the tissue and fat, is a poor conductor, limiting the amount of heat transferred from the core tissue to the shell through conduction. Convection is the transfer of heat though liquids or gas. The transfer of heat from the core to the shell through the blood is a very effective mechanism of heat transfer. Evaporation occurs when water is converted from liquid to gas. For homeotherms (i.e., organisms with a constant body temperature), water is continually lost through diffusion of water molecules to the skin and respiratory surfaces, leading to insensible evaporation [45, 47]. For humans, insensible water loss occurs at a rate of 450-600 mL/day of water [47]. Heat loss through insensible evaporation is not part of the mechanisms of heat dissipation, however, evaporation of sweat is.

When the skin temperature of the human body is greater than the external temperature, the resulting temperature gradient allows for heat transfer into the external environment through conduction, convection, and radiation. For instance, a nude individual sitting in a room at normal temperature will lose approximately 60 percent of total heat loss though radiation, 3 percent through conduction to an object (i.e., the chair), 15 percent through convection to the air, and 22 percent of total heat loss to evaporation [47]. When the external temperature is greater than the skin temperature, evaporation is the body's main mechanism of heat transfer [49] and heat is gained through the other mechanisms. However, as the water content of the air (humidity) increases, the rate of evaporation decreases [50].

BODY TEMPERATURE REGULATION

Regulation of body temperature is controlled though the temperature-regulating centers in the preoptic area of the anterior hypothalamus. In addition to the brain, there are internal receptors located mainly in the spinal cord, the abdominal vicera, and in/around the great veins of the upper abdomen and thorax and peripheral receptors located in the skin [44, 47]. As the core body temperature increases beyond a set point the heat sensitive neurons in the hypothalamus begin firing invoking heat loss mechanisms [41, 47, 50]. When the peripheral receptors sense an increase in the external temperature, heat loss mechanisms are invoked before the core temperature increases [41, 47, 50]. Additionally, the peripheral receptors allow for a localized heat loss response [51]. The main responses are reduction of the basal metabolic rate, peripheral venous vasodilation of the blood vessels, and sweating [47]. With vasodilation, heat is transferred in the blood through convection from the core to the periphery.

During peripheral venous vasodilation the rate of blood flow can increase from 250 mL/minute to 6-8 L/minute [48]. The increase in blood flow results in increased cardiac output. To compensate for the increase in blood flow to the skin there is a corresponding reduction of blood flow to the renal and splanchnic bed [41, 52]. Additionally, sweating secretion results in loss of water and sodium which leads to decreased blood volume further taxing the cardiovascular system. During exercise or work, the body is also prioritizing blood flow to the muscles to support metabolism [41, 52].

The body's responses are proportional to the temperature degree above the set point. The thermoregulatory set point varies throughout the day and throughout an individual's life stages (e.g., circadian rhythms, menstrual cycle, fever, heat acclimatization) [44, 47]. Additional behavioral responses also occur as the body temperature increases such as increasing the body's surface area by changing body position (e.g., curling into the fetal position when cold), removing/changing clothing, restricting activity, and moving to a cooler environment [44, 53]. However, an individual's behavioral response is not solely dependent on physiological changes but on the perception of their thermal environment, prior experience, and the needs of the situation. [44, 53].

ACCLIMATIZATION

Acclimatization is the combined physiological changes that occur in the body to more efficiently maintain the thermo-equilibrium in a hot environment while at the same time decreasing strain on the body's systems. The following physiological changes occur with acclimatization: increased cardiac performance, expanded plasma volume, sodium-conservation in the sweat glands and the kidneys, increased ability of the sweat glands to secrete sweat, increased efficiency in the shunting of blood away from non-critical circulatory systems (e.g., splanchnic bed), increased filtration rate of the kidney, and increased resistance to exertional rhabdomyolysis [41, 48, 52]. Finally, with acclimatization the thermoregulatory set point is lowered allowing the initiation of vasodilation and sweating to occur at a lower temperature [41, 48]. This process of complete acclimatization takes approximately 2–3 weeks but starts occurring within days of exposure to heat [41, 49].

BIOLOGICAL FACTORS AFFECTING THERMOREGULATION

There are clinical and biological factors that may limit the body's ability to thermoregulate properly. Individuals with chronic medical conditions may be less able to cope with the biological strain of the heat loss mechanisms especially those with circulatory or renal problems. As individuals age there is a decreased circulatory response to heat, impairing heat loss through convection [53]. Also the peripheral sensors become less sensitive meaning that individuals are not able to react as well physiologically or behaviorally to the warming external environment [53]. On the reverse side, children are at a special disadvantage when regulating their body temperature [41, 54, 55]. They have a higher metabolic rate, lower blood volume, and a lower cardiac rate than adults. This means that children produce more internal heat than adults and may have a harder time dispersing that heat though peripheral blood flow. Further, the sweat glands in children produce sweat at a lower rate per gland than adults resulting in less sweat. The reduction in sweat is compensated by a larger surface area-to-mass ratio which allows for greater heat dispersion through radiation as opposed to evaporation. However, in environments where the external temperature is warmer than skin temperature, their larger surface area-to-mass ratio results in greater heat absorption [41, 54, 55].

As previously mentioned, peripheral warmth sensors are located in the skin. Damage to the skin (e.g., sunburn or burns) can prevent these sensors from detecting increases in external temperature. Skin damage may also occlude the sweat glands preventing sweat from being delivered to the skin surface. Sweat glands can also be destroyed when skin is injured and will not regenerate when scar tissue is present [53]. Individuals with spinal injuries have varying degrees of thermoregulation impairment dependent on the level and location of injury. In these individuals the peripheral warm sensors are not able to transmit information to the brain, delaying activation of heat loss mechanisms [41]. Additionally, below the lesion, the body may not have the ability to dilate the peripheral blood vessels and the sweating response is severely compromised [41].

Finally, medication usage can modify an individual's ability to tolerate heat [56, 57]. Certain medications can increase internal production of heat by increasing the metabolic rate (e.g., stimulants) or by producing muscle hyperactivity (e.g., typical antipsychotics). Medications may disrupt the signaling mechanisms for sweat activation by blocking neurotransmitters required for sweat activation (e.g., anticholinergic medications such as tricyclic antidepressants and antihistamines) [56-58]. Cardiovascular medications that lower blood pressure (e.g., antihypertensives) and medications that increase removal of water from the body (e.g., diuretics) may also inhibit the body's heat response [58].

DISEASE: HEAT-RELATED ILLNESS

PATHOPHYSIOLOGY

As mentioned previously, heat is created through the body's metabolic process or absorbed from the environment. The thermoregulatory system maintains temperature equilibrium through heat dissipation to the external environment. Failure of the thermoregulatory system occurs in increments as the body absorbs or creates heat faster than it can be dissipated. The general physiological changes that occur can be summarized as follows: excessive heat is deadly to cells and results in the denaturing of proteins, apoptosis and cellular necrosis [41, 48, 52]. The vasoconstriction of the renal and splanchnic bed, which occurs in response to peripheral venous vasodilation, results in a

reduction of tissue oxygenation leading to hypoxia which in turn causes the generation of highly reactive oxygen and nitrogen species that damage tissue. During the initial failure of the thermoregulatory system there is an immune response which protects the cells from heat damage. However, as the core continues to heat, this response overwhelms the body leading to inflammation associated injury including injury to the brain and neurons. Elevated temperatures also result in vessel damage resulting in activation of the coagulation pathway which may cause small clots in the blood vessels disrupting blood flow to the organs. If left untreated, the above physiological changes will result in renal failure, coagulopathy, hepatic dysfunction, and finally multi-organ failure [41, 48, 52].

DIAGNOSIS

As the thermoregulatory system fails, the resulting medical problems associated with failure are classified by medical practitioners as a continuum of disorders under the heading of heat-related illness (HRI) [48, 49, 59]. Timely medical intervention can prevent mild cases of HRI from becoming severe, potentially preventing death. A summary of the disorders associated with heat-related illness are listed below including the characteristics and pathophysiology.

Heat Edema (ICD-9-CM 992.7): The mildest form of HRI is heat edema which is characterized by swelling in the hands and feet due to accumulation of interstitial fluid. It is caused by the pooling of blood in the skin and lower extremities during peripheral venous vasodilation and venous stasis (i.e., slow blood flow). Heat edema usually occurs in individuals who are not acclimatized to the heat and individuals who have been sitting for long periods of time, particularly the elderly.

Heat cramps (ICD-9-CM 992.2): Heat cramps are characterized by painful muscle spasms in the arms, legs or abdomen. Heat cramps usually occur after vigorous exertion (e.g., work or exercise). The condition is due to low sodium concentration in the plasma. The decreased sodium in the extracellular fluid leads to water entering intravascular space (e.g., plasma/blood vessels) to restore the sodium concentration balance between the intravascular and interstitial fluid (i.e., fluid

around the cells). The decrease in water in the interstitial fluid causes deformation of the matrix around the cells leading to mechanical deformation causing the skeletal muscle cells to contract resulting in muscle cramps. Acclimatization reduces the risk of heat cramps by decreasing the amount of electrolytes (e.g., sodium or potassium) lost in sweat.

Heat Syncope (ICD-9-CM 992.1): Reduced blood flow to the central nervous system due to increased blood flow to the periphery as well as reduced central venous return results in heat syncope [55]. The reduction in central venous return is caused by depletion of extracellular fluid volume, peripheral vasodilation, and poor vasomotor tone [55]. Vasomotor tone is required for modulation of vasodilation and vasoconstriction of blood vessels [55]. Heat syncope usually occurs in the elderly or non-acclimatized individuals. It is characterized by fainting or dizziness.

Heat Exhaustion (ICD-9-CM 992.3 and 992.4) and Heat Stroke (ICD-9-CM 992.0): Heat stroke, a medical emergency, is the most severe form of heat-related illness. Heat exhaustion is often considered a warning of impending heat stroke and untreated heat exhaustion will result in heat stroke [55]. There is a great deal of variation in the symptoms between mild heat exhaustion and heat stroke. The medical recommendation is that if there are any doubts regarding the diagnosis, it is prudent to treat the patient for heat stroke [48]. A comparison of the symptoms of heat exhaustion and heat stroke can be found in Table 2-1. Heat exhaustion is due to volume depletion from sweating and occurs in individuals working/exercising in hot conditions. It can be generally classified as either heat exhaustion due to water depletion or heat exhaustion due to salt depletion [48, 55]. Heat exhaustion due to water depletion occurs with inadequate fluid intake. Heat exhaustion due to salt

Table 2-1. Comparison of symptoms related to Heat Exhaustion and Heat Stroke* [48, 55, 59]

Heat Exhaustion	Heat Stroke
	Seizures/coma
Irritability	Delirium/hallucinations
Fatigue/weakness	Respiratory alkalosis
Light-headedness	Cerebellar dysfunction (e.g., Ataxia)
Sweating	Maybe sweating, but hot skin
Headache	Headache
Nausea/vomiting	Nausea/vomiting
Orthostatic hypotension	Hypotension
Tachycardia	Tachycardia
Increased thirst (dehydration)	Diarrhea
Muscle cramps	Muscle spasm
Core Temperature <40°C (104°F)	Core Temperature ≥40°C (104°F)

*Not all symptoms are present in all patients

depletion is similar, however, water replenishment occurs but with inadequate sodium intake [48, 55]. The latter condition takes longer to develop.

The main difference between heat stroke and heat exhaustion is that with heat stroke there is a core body temperature of 40°C (104°F) or higher and it includes dysfunction of the central nervous system. In the aforementioned forms of heat illness, the thermoregulatory mechanisms continued to function however, with heat stroke these mechanisms no longer function. Heat stroke can be divided into two categories; classical and exertional. Classical heat stroke occurs in hot environments and usually affects those whose thermoregulatory system is compromised due to medication usage, injury, chronic illness, underdevelopment (i.e., children) or individuals who are not able to employ behavioral modifications such as the very young or very old [48, 55, 59]. Exertional heat stroke occurs in young healthy individuals under conditions of strenuous activity, usually in hot humid weather [48, 59]. In exertional heat stroke, the body's endogenous heat production overwhelms the thermoregulatory system. Profuse sweating is common in exertional heat stroke whereas in classical heat stroke patients show signs of anhidrosis (inability to sweat) [48, 55]. As heat stroke progresses it is common to see moderate hyperurcemia in classical heat stroke and severe hyperurcemia in exertional heat stroke [56, 60]. Acute renal failure, lactic acidosis, hypokalemia, disseminated intravascular coagulation, and rhabdomyolysis are all common in exertional heat stroke but not classical heat stroke [48, 52, 55, 59]. In the most severe cases of heat stroke, multi-organ dysfunction syndrome occurs [52].

OTHER POTENTIAL HEAT-OUTCOMES

In practice, the continuum of disorders known as HRI is difficult to diagnose and the diagnostic definition varies by geographic location and medical facility [2, 13, 61]. Additionally, prolonged exposure to high heat can lead to a cascade of body system failures [52]. As such, HRI are often recorded in the medical or death record as the condition that arises from response to the stress of the thermoregulatory process (e.g., acute renal failure, acute respiratory distress syndrome, or myocardial injury) or the pre-existing condition that may have been exacerbated by the stress of the thermoregulation process (e.g., cardiovascular disease, renal failure, or diabetes) [2, 52, 62-64].

Recording of the signs and symptoms of HRI without a corresponding HRI diagnosis leads to an underestimation of HRI in the administrative records [13]. Further, as heat exposure may not be critical to the treatment needs of the patient, heat exposure may not be acknowledged at all in the record. Some systematic bias in diagnosis of HRI may also exist. For example, if a doctor or medical examiner is aware of high ambient temperatures they may be more likely to code the illness or death as heat-related leading to differential information bias [2, 65]. In order to avoid the potential biases incurred when only HRI-specific codes in the medical/death records are considered, researchers will often use other physiologically plausible endpoints when examining the heat-health relationship.

As the thermoregulatory system fails it affects multiple organs and systems throughout the body. As such, all cause morbidity and mortality is a common endpoint used to capture all potential cases of HRI when analyzing the heat–morbidity/mortality relationship [1, 2, 66]. Often this endpoint is adjusted and excludes injuries/poisonings and external causes of injuries[8] as these causes of morbidity and mortality are not direct biological symptoms of the failure of the thermoregulatory process but may occur as body systems start to fail (e.g., neurological impairment such as dizziness may cause a worker to fall from scaffolding) [1, 18]. A limitation of all-cause morbidity and mortality for analyses is the potentially low specificity of this outcome in identifying HRI. As such, cause-specific endpoints are also used. Many studies have used very broad groupings when studying HRI, such as, total diseases of the circulatory system or total respiratory disease [1, 66-68]. However, these broad groupings contain many different subtypes of diseases (e.g., broad group = cardiovascular disease; sub-groups = ischemic heart disease, hypertension, cardiac dysrhythmia, and heart failure) which may have different causal associations with heat exposure [66]. As such, researchers have also examined subtypes of diseases in association with heat [1, 66].

The observed magnitude of the heat-health relationship for each cause-specific endpoint (e.g., cardiovascular or renal outcome) is different dependent on the data source (i.e., mortality versus morbidity data) [69, 70]. The reasons for the differences in magnitude are unclear but may be

[8]Example: Injuries/poisonings = ICD-9 codes 800-999 or ICD-10 codes S and T; External Causes = IC-9-CM E-codes or ICD-10 codes V-Y. The inclusion or exclusion of these two groupings depends on the study (e.g., include injuries/poisonings but exclude external causes).

due to the rapid onset of the diseases such as cardiovascular event where the individual die before reaching the hospital and therefore would not be counted in the morbidity data [69, 71, 72]. Additionally, the majority of cause-specific morbidity studies only use the primary diagnosis code when using administrative data while mortality studies may only use the underlying cause of death. This may result in an underestimation of the cause-specific endpoint and a small (or no) heat-health association [66, 73, 74]. For instance, an excess in cardiovascular[9] mortality has been observed during heat-waves [2] but not as consistently for cardiovascular morbidity [66]. However, during the 1995 Chicago heat wave, Semenza et al. observed an excess of 89 primary diagnosed cardiovascular hospitalizations ($p = 0.2$) and an excess of 461 cardiovascular hospitalizations ($p = 0.02$) when all diagnosis codes were used [75]. For the remainder of this document heat-related outcomes will be defined as all HRI outcomes and any heat-related endpoints associated with thermoregulatory system failure.

Endpoints that have been shown to have an association with outdoor heat in multiple studies are summarized below.

Diseases of the Circulatory System[9]

In order to regulate body temperature, excess heat is removed through increased blood flow to the skin. Individuals with pre-existing conditions may not be able to meet the requirements placed on the circulatory system for maintaining thermo-equilibrium (e.g., increased cardiac output). Even healthy individuals may succumb to the stress of thermoregulation placed on the circulatory system. For instance in healthy individuals, haemoconcentration (an increase in the concentration of red blood cells), caused by salt and water loss in sweat, can lead to coronary and cerebral thrombosis [76]. Prior work has demonstrated that mortality related to the circulatory system increases with increasing ambient heat [1, 2, 77]. This relationship is not as clear with circulatory morbidity [66] where the

[9]Previous authors have used the broad grouping labeled cardiovascular disease in the text however the codes (e.g., ICD-9-CM/ICD-10) listed in their papers usually refer to diseases of the circulatory system and include cerebrovascular disease [1].

majority of the literature has observed a non-statistically significant increase [69, 70, 78-81], although a few studies have observed a decrease in admissions with increasing temperature [71, 79, 82][10].

When examining the cause-specific endpoints within the circulatory system, there does appear to be a positive relationship between ambient heat and ischemic heart disease for both morbidity [75, 78, 81-83] and mortality [67, 83-86]. For example, for every 1°C increase in maximum temperature, a 1.7 percent (95% CI = 1.2, 2.2) increase in mortality for ischemic heart disease was seen during the summer months in England and Wales (1993–2006) [85]. For morbidity, between 1991 and 2004 in New York City a 2.54 percent (95% CI = 1, 4[11]) increase in ischemic heart disease hospitalizations was observed for every 1°C increase in mean apparent temperature [81]. A California study of all ED visits for the summer months of 2005–2008 observed a 1.7 percent (95% CI = 0.2 to 3.3) increase in ischemic heart disease for every 5.5°C (10°F) increase in mean apparent temperature [78].

For myocardial infarction the magnitude of the positive association is weaker but still generally present for both morbidity [75, 78, 80, 82, 87-89] and mortality [67, 85, 90, 91]. During the summer months in England and Wales (1993–2006), Gasparrini et al. observed a 1.1 percent (95% CI = 0.7, 1.5) increase in myocardial infarction mortality for every 1°C increase in maximum temperature [85]. The California study of all ED visits for the summer months of 2005–2008 observed a 1.7 percent (95% CI = −0.5 to 4.0) increase in myocardial infractions risk for every 5.5°C (10°F) increase in mean apparent temperature [78]. Studies have also shown increased hospital admissions related to cardiac dysrhythmia [75, 78, 81, 89]. However, the results have been inconsistent when examining the relationship between ambient heat and heart failure [75, 78, 81, 82, 84, 88, 89] or hypertension [75, 78, 81, 88]. Positive associations have been seen for outdoor heat and death due to heart failure [84, 85] and hypertension [84].

---

[10]For a summary of the results for the referenced studies please see within this document sub-section titled *Epidemiology of HRI in the General Population* within the *Epidemiology* section.

[11]The actual confidence intervals were not provided in the text by the authors. Instead the authors provided a figure with the confidence interval. The values presented here are estimated from the author's figure.

Cerebrovascular endpoints have been examined in relation to ambient heat and a negative relationship has been seen for morbidity [69, 81-83, 87], while a potentially positive relationship is seen for mortality [83-85]. However, when examining the more specific cerebrovascular sub-types, a strong negative association has been observed with hemorrhagic stroke morbidity [78, 82] and a modest increase has been observed for ischemic stroke morbidity [70, 75, 78, 82]. One study examined ambient heat in relation to ischemic stroke mortality but found no association [92].

Diseases of the Respiratory System

Numerous studies have been conducted which indicate that respiratory morbidity [66, 70, 86] and mortality [1, 2, 84] increase with increasing ambient heat[10]. The mechanisms for this association are unclear. It may be due to poor air quality that is often associated with increased outdoor temperatures and exacerbation of pre-existing conditions due to the stress of the thermoregulatory process [93]. Additionally, respiratory alkalosis and adult respiratory distress are both symptoms of HRI [52, 94]. An increase in pneumonia morbidity [75, 78, 82, 88] and mortality [84, 90] has been observed. The literature suggests a decrease in asthma morbidity [75, 78, 82] and an increase in asthma mortality [85] with increasing heat exposure. There is also the suggestion of an increase in chronic bronchitis and emphysema morbidity [75, 78, 81, 82] and mortality [84, 85, 90] with increasing ambient heat.

Other Diseases

A positive association has been observed when looking at total renal morbidity [65, 69, 70, 78, 86, 87, 95] and mortality [84-86]. Acute renal failure and rhabdomyolysis are both symptoms often seen in exertional heat stroke [48, 96]. This is observed in the literature for morbidity where there is a clear positive association between ambient heat and acute renal failure [65, 69, 75, 78, 82, 95]. Interestingly, there does not appear to be an association between morbidity due to chronic renal failure and ambient heat [65, 75, 95]. This may be due to external protective factors (e.g., regular dialysis treatment in air conditioned hospitals) as opposed to biological mechanisms [75]. In mortality studies, researchers have not looked at chronic and acute renal failure separately.

Dehydration is a common symptom of HRI as the body loses water and electrolytes through sweat. This symptom has also been used as an endpoint for heat-related morbidity and shows a strong positive association with increasing ambient heat [75, 78, 82, 87].

Both type I and type II diabetes are chronic conditions which impair an individual's ability to properly thermoregulate [93, 97]. The ability of the blood vessels in the skin to dilate may be impaired in individuals with diabetes [93, 97]. Further, with type II diabetes, vasodilation occurs at a higher temperature than individuals without diabetes [51]. Additionally, individuals with type II diabetes have decreased sweating response [93]. When used as an endpoint, diabetes mortality [67, 84, 85] and morbidity [70, 75, 78, 82, 87] increase with increasing ambient heat.

It is important to note that while a number of cause-specific endpoints have demonstrated biological plausibility in relation to failure of the thermoregulatory system and appear to be associated with increasing heat [38, 62, 98] there is no information in the literature on the sensitivity and specificity of these endpoints for identifying outcomes associated with failure of the thermoregulatory system in administrative datasets. It is unclear which outcome, all-cause, cause-specific, or diagnosed HRI provides the least biased approximation of HRI (i.e., failure of the thermoregulatory system). Further research in this area is required.

ENVIRONMENTAL PARAMETERS

PARAMETERS AFFECTING RESPONSE TO HEAT

How an individual responds (physiologically and behaviorally) to heat varies from person to person. As such, there is no uniform definition of what constitutes too warm or too hot of a thermal environment. However, there are six basic parameters that, in combination, largely determine how an individual reacts, physiologically and behaviorally, to their thermal environment [3, 44]. Two of the parameters, clothing insulation and metabolic heat production, are specific to the individual. The type and amount of clothing that an individual wears will affect the body's ability to dissipate heat. The amount of metabolic heat produced will depend on the level of exertion and the physical fitness of the

individual. The other four factors are environmental: ambient air temperature, wind velocity, mean radiant temperature, and humidity.

- Ambient air temperature is the temperature of the air surrounding the body. It is usually measured in Celsius or Fahrenheit.
- Air velocity describes the speed of air moving across an individual. Moving air increases heat loss; heat is dissipated from the body through the air, the now warm air is replaced with cool air, and the process starts over.
- Mean radiant temperature is the average of the combined heat emitted from all heat sources in the environment. All objects in an environment radiate some heat with heat sources (e.g., sun, fire, ovens, dryers, lights, etc.) radiating a greater proportion of the heat.
- Humidity is the amount of water vapor in the air. It is often measured as absolute humidity, relative humidity, or dew point. Absolute humidity is the actual amount of water in the air (conveyed as grams of water vapor per cubic meter volume of air) while relative humidity is the amount of water in the air relative to the amount of water that the air can hold at the current air temperature (conveyed as a percentage). The lower the temperature the less water vapor the air can hold. Dew point is the temperature to which the air must be cooled to reach 100 percent relative humidity and is measured in Celsius, Fahrenheit, or Kelvin. The higher the dew point the greater the amount of water vapor.

An individual's physiological responses are directly impacted as the levels of these six parameters vary. If the air temperature and wind velocity are high and the humidity is low the body can remove heat though all heat loss mechanisms. Depending on the amount and type of clothing that an individual is wearing at the time of exposure the body may not be able to disperse heat to the environment; in fact, excess heat may be trapped between the skin and the clothing layer further heating the body [44]. For example, fire fighters create excess metabolic heat though exertion, additionally, they must wear heavy protective clothing while entering a high-heat environment and as a result they are at higher risk of heat-related illness [99].

PROXIES FOR HEAT EXPOSURE

The perception of what constitutes a hot thermal environment, and in turn the resulting behavioral response varies from person to person but we can approximate the exposure a person receives using the six basic parameters mentioned above (i.e., clothing insulation, metabolic health production, ambient air temperature, wind velocity, mean radiant temperature, and humidity). Ideally, the best proxy will incorporate all six of the basic parameters; however, realistically this is not possible in large populations. There are a number of different measurements and metrics which have been used as proxies when characterizing the heat-health relationship. These proxies have also been used to alert populations to dangerous heat situations in order to prevent heat-related morbidity and mortality.

The wet bulb globe temperature[12] (WBGT) incorporates all four of the environmental parameters into the measurement. Predetermined WBGT limits have been used in small populations (e.g., a work-site or sporting event) to effectively induce behavioral response which prevent heat-related morbidity and mortality [44, 96]. Heat exposure limits for work and rest schedules have also been determined based on the WBGT in combination with calculations which incorporate the other two basic parameters[13] [100, 101]. Exertional heat-related morbidity/mortality has been prevented (and reduced) by use of this combined method [44, 100]. While the WBGT measurement system is effective in eliciting behavioral responses to prevent HRI in real time application for local environments, it is labor intensive, requires precise setup/monitoring and is not applicable for monitoring large geographical areas. Additionally, the incorporation of the individual parameters with WBGT requires complicated calculations. Previously created tables specific to work-sites/situations

[12]The wet bulb globe temperature (WBGT) is a measurement of heat that incorporates air temperature, humidity, air velocity, and solar radiation. The WBGT requires three separate thermometers to obtain the reading; a black globe thermometer (solar radiation), a wet-globe thermometer (humidity), and a dry bulb thermometer (air temperature).

[13]For instance, OSHA guidelines recommend 75% work and 25% rest per hour when the WBGT is: 30.6°C (87°F) for light work, 28.0°C (82°F) for moderate work, and 25.9°C (78°F) for heavy work. OSHA's Technical Manual also provides adjustments to the work/rest schedule dependent on the amount of clothing worn. The military has a flag system based on the WBGT and assumptions of clothing worn which indicates what type of training can and cannot be done [100]. Organizers of athletic events are encouraged to cancel events when the WBGT is too high [96].

allow for real time application in local work environments; however, the use of these tables is not practical when working with large populations where information about the work situation and amount/type of clothing for the individuals is not available.

The spatial synoptic classification (SSC) system is an exposure metric which combines measurements of all four of the environmental parameters. It is sometimes used when examining the heat-health relationship in populations [102-105] and is often used in the development of city-specific heat health warning systems (HHWS) [106-108]. The system classifies weather conditions and patterns for each day into six main types and two subtypes: dry polar (associated with lowest temperatures in a region with clear dry conditions), dry moderate (mild and dry air), dry tropical (hottest and driest conditions at any location), moist polar (typically cloudy, humid, and cool), moist moderate (warmer and more humid than moist polar), moist tropical (warm and very humid) , moist tropical plus (the hottest and most humid days within the moist tropical classification) and transitional (occurs when one weather type yields to another) [109]. To calculate daily SSC for use in characterizing the heat-health relationship the following measurements obtained four times daily are required: temperature, dew point, wind direction, cloud cover, and sea-level pressure [109]. After the heat-health relationship has been described, forecasting data is used to calculate SSC for the HHWS [106]. The SSC has not been used very frequently in the heat-health literature and results using this metric as an exposure will not be comparable to the majority of prior work. Further, a comparison of the predictive capacity of the heat exposure metric used in HHWS across the globe found that synoptic classification was not more effective at predicting days with the highest excess mortality than other methods and the effectiveness (compared to the other methods) varied by locale [110].

One of the most consistently collected and widely available parameter is dry bulb temperature [111]. In many time-series analysis studies the daily average, maximum, or minimum temperature is used [2, 66]. Temperature is diurnal with the maximum usually occurring during the peak of the day and the minimum usually occurring at night. Continuous days of heat with very little night time cooling may increase the likelihood of heat-related morbidity and mortality [112-114]. For instance, fewer cases of heat-collapse were observed in workers constructing the Hoover Dam when

they were allowed to sleep at night in air-conditioned rooms [94]. Minimum temperature may capture the impact of night time cooling [112, 113]. Some studies have also included the difference between average temperature and minimum temperature to try and capture minimal night time cooling [115, 116]. Brief periods of intense heat will also increase the likelihood of HRI which would be captured by using maximum temperature. It is very rare that studies have information regarding the time of day that the exposure or event occurred and average temperature may provide a more reliable exposure measure than the other two measures, especially since it includes multiple readings during the day, potentially reducing measurement error.

Dew point or relative humidity is often used as a separate variable in conjunction with air temperature when modeling the relationship between heat and heat-related morbidity and mortality [1, 2, 113]. This is because the temperature feels warmer to an individual as the water content of the air increases and the ability to cool by evaporation decreases [50]. As previously mentioned, how an individual perceives their environment may determine their behavioral modifications, in turn, providing a more accurate assessment of the relationship between heat and heat-related morbidity and mortality. The literature has used a number of different indexes in environmental research which incorporate both humidity and air temperature; three examples are the humidex, the heat index, and apparent temperature[14] [120]. These indexes provide a general idea of how hot it feels and are used widely in the literature when describing the heat-health relationship. Additionally, the heat index (U.S. NWS) and the humidex (Environment Canada) are used, both actual and forecasted values, when communicating summer weather information to the public including heat warnings and advisories. Each of the three metrics uses air temperature but humidity is incorporated differently; heat index uses relative humidity, humidex is calculated using dew point (which is used to calculate vapor pressure), and the apparent temperature uses dew point [118, 119, 121]. The heat index assumes a

[14]The heat index is a regression equation based on the work by R.G. Steadman[117]. Steadman originally created a table that accounted for a number of parameters including human ventilation rate, surface radiation/convection, clothing resistance to heat, and moisture transfer. The equation is Heat Index = $-42.379 + 2.04901523*T + 10.14333127*R - 0.22475541*T*R - (6.83783x10^{-3})*T^2 - (5.481717x10^{-2})*R^2 + (1.22874x10^{-3})*T^2*R + (8.5282x10^{-4})*T*R^2 - (1.99x10^{-6})*T^2*R^2$ where T = ambient dry bulb temperature (F) and R = relative humidity (percentage) [118]. A simplified version of this equation was created and is often referred to in the heat literature as apparent temperature ($AT = -2.653 + 0.994*T + 0.0153*(DT)^2$) where T = air temperature and D = dewpoint [119].

wind speed of 5 knots (~6 miles/hour) while apparent temperature assumes a wind speed of zero [98, 118, 119]. Both heat index and apparent temperature assume that the measurements were taken in the shade [119]. For instance, exposure to direct sun light can increase the heat index by up to 15°F. The humidex only incorporates temperature and humidity, without any further assumptions [121]. The equation for calculating the heat index was based on R.G. Steadman's 1979 work relating dry bulb temperature (at various humidity levels) and the skin's resistance to heat and moisture transfer while accounting for a number of biometeorological parameters [118]. As a result, a limitation of the heat index is that it produces results that deviate from Steadman's original tables when the air temperature is <26.67°C (80°F) or the relative humidity is <40 percent [122, 123]. The U.S. NWS has created (and uses) an algorithm to account for the limitations of the heat index calculation [120, 124].

In practice, because of its availability, temperature (e.g., minimum, maximum, or average) or a measure that combines both temperature and humidity (e.g., heat index, humidex, apparent temperature) is often used as a proxy for heat exposure. It is unclear which measure best represents heat exposure in population studies [112, 113, 123]. When examining the heat-mortality relationship among 107 U.S. cities (1987-2000), Barent et al. concluded that the best measure of ambient outdoor heat[15] varied by location and region under study [113]. In the north-east, maximum temperature fit the heat-health model best while in the south-east and north-west the best exposure measure/metric was the humidex. However, the authors mention that there was temporal, within city variability in relation to which exposure measure/metric provided the best fit [113]. Hajat et al. also noted, in a study that looked at data from three European cites, that the effectiveness of apparent temperature as a predictor of mortality depended on the location with apparent temperature being a good predictor for Budapest but not in London or Milan [112]. They also found that mean temperature was a better predictor of the heat-mortality relationship than minimum or maximum temperature [112]. A study in

[15]The paper examined 10 measures of heat exposure: minimum, maximum, and average temperature, humidex, minimum, maximum, and average apparent temperature, and minimum, maximum, and average temperature plus humidity.

New York City concluded that all the measures of heat-exposure[16] examined produced similar results when looking at the heat-mortality relationship, however, the heat index produced the best fitting model [123]. The measures of heat, especially temperature, are also highly correlated indicating similar predictive ability. Although performance of particular measures/metrics varies among previous studies, this work agrees that when deciding which measure/metric to use as a proxy for population heat exposure it is important to consider which data source has the largest spatial coverage for the study area; the least amount of missing data for the time period; and the highest quality data (i.e., traditional characteristics for choosing an exposure metric).

COLLECTION OF THE BASIC ENVIRONMENTAL PARAMETERS

In the U.S., surface weather data is collected by monitoring stations throughout the country. The NWS collects data from two types of monitoring stations, first-order stations and Cooperative Observer Program (COOP) stations which are part of the U.S. Cooperative Observing Network [125, 126]. First order stations are automated and collect hourly data on a number of weather parameters including but not limited to temperature, dew point, and wind speed [126]. These monitoring stations are professionally maintained by the NWS, the Federal Aviation Administration and the Department of Defense and are often located at airports. The COOP stations are maintained by volunteers across the country and collect minimum/maximum air temperature and precipitation [125]. Many of the COOP stations also collect metrological and hydrological data such as soil temperature or evaporation. Additional automated weather networks (Mesonets) are maintained by universities and state agencies across the country[17]. These networks collect approximately the same data as the first-order NWS stations. However, the Mesonet sites are recent additions and typically do not have the amount of historical data that the other monitoring stations have.

---

[16]The paper examined 5 measures of heat exposure: minimum, maximum, and average temperature, heat index, and spatial synoptic classification.

[17]Examples include: the Florida Automated Weather Network (http://fawn.ifas.ufl.edu/), the Georgia Automated Weather Network (http://www.georgiaweather.net/), the NC ECONET (http://www.nc-climate.ncsu.edu/econet), the High Plains Regional Climate Center Automated Weather Network (http://www.hprcc.unl.edu/awdn/) and the Michigan Automated Weather Network (http://www.agweather.geo.msu.edu/mawn/).

AIR POLLUTION

Air pollution is an additional environmental variable that may affect the heat-health relationship. There are an increasing number of papers published in the current literature suggesting that air pollution may modify or confound the heat-health relationship [1]. Sunlight is a key component of the chemical formation of many air pollutants and consequently there is a positive correlation between hot days and high air pollution. In the air pollution literature, there is a clear relationship between increased levels of air pollution and increased morbidity and mortality [127, 128]. Temperature has a clear effect on air pollution and both temperature and air pollution effect morbidity and mortality. Therefore, air pollution is on the causal pathway (Figure 2-2) and should not be treated as a confounder in studies examining the heat-health relationship where temperature is used as a proxy for heat [129].

However, it is possible that air pollution may modify the heat-health relationship. Only a few studies have been conducted examining air pollution as an effect measure modifier. Modification of the heat-health relationship by variables for air pollution were found in most [130-134] but not all [92, 135] of those studies. When modification was present it appeared to vary in magnitude by locale and outcome [130, 133, 134]. A study examining the joint effect of temperature and ozone on mortality in nine French cities during the August 2003 heat wave found that the contribution of ozone to the overall excess risk of death varied by city [130]. The contribution of ozone[18] to the overall excess mortality was lowest in Bordeaux (due to ozone = 2.46%; due to temperature 97.54%) and highest in Toulouse (due to ozone =85.34%; temperature = 14.66%) [130]. A study of 95 U.S. cities found that, during June–September 1987–

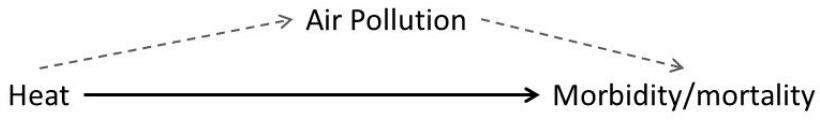


Figure 2-2. A simplified Directed Acyclic Graph depicting the effects of heat on morbidity and mortality. The assumption of this DAG is that temperature is a proxy for heat exposure. The solid black line represents the direct effect while the dotted line represents the indirect effect. The total effect is represented by both the solid and dotted line.

[18]The contribution of ozone was calculated as the logarithm of ozone divided by the joint effect of temperature and ozone.

2000, ozone modified the relationship between maximum temperature and mortality related to diseases of the circulatory system [133]. As with the French study, the results varied by city and region, the largest modification by ozone of the temperature-mortality effect was seen in the North. The magnitude and statistical significance of effect modification in the South and Midwest depended on how the cities were grouped into regions [133]. In Brisbane, Australia, a study found that PM10 modified the relationship between temperature and respiratory mortality, Emergency Department (ED) visits and hospitalizations, circulatory system hospitalizations and mortality, and all non-accidental mortality, but did not modify the relationship for circulatory system ED visits [134]. A study of nine U.S. cities found no modification by PM2.5 of the relationship between apparent temperature and non-accidental mortality [135]. A limitation of the aforementioned studies is that modification by air pollution was examined only in urban areas [130-135]. Since the magnitude of the modification varies, it is feasible that modification may not be present in some areas, especially rural areas. For instance, a study of nine California counties conducted during May–September 1999–2003 reported that modification by air pollution (i.e., ozone, PM10, PM2.5, NO2, CO) on the relationship between temperature and non-accidental mortality was not seen [92]. Ultimately, the decision to include modification by air pollution, even if it is present, is determined by the type of effect that the researcher is interested in studying; the direct effect of heat on morbidity and mortality, the indirect effect of heat on morbidity and mortality, or the total effect of heat on morbidity and mortality (Figure 2-2) [129].

EPIDEMIOLOGY

According to the Centers for Disease Control and Prevention, there was an average of 688 U.S. deaths per year between 1999 and 2003 attributable to exposure to extreme heat, hyperthermia, or both [136]. Nationally, in 2009, an estimated 7,151 individuals were hospitalized and an additional 48,876 individuals were treated in the ED for HRI (HRI) [137]. Between 2005–2009 an estimated 9,237[19] high school athletes lost one or more days of activity due to dehydration, heat exhaustion, or

[19]Weighted average of sample; sample = 118 cases with a rate of 1.6 per 100,000 athlete-exposures.

heat stroke [138]. Additionally, in 2009, the Bureau of Labor and Statistics (BLS) reported that exposure to environmental heat resulted in 35 occupational deaths (rate = 0.015/100,000 workers[20]) and 2,170 injuries to workers requiring days away from work (rate = 2/100,000 full-time workers)[21] [140].

Heat waves rather than daily exposure to heat (e.g., high ambient temperature) appear to be a large part of the current global focus of response/interventions for heat-associated morbidity and mortality [6, 13, 38, 98, 141]. This may be due in part to the large number of deaths (and excess morbidity) that occur within a short time period, similar to other natural disasters (e.g., floods or tornadoes) [13, 142-144]. For example, during the 1995 Chicago heat wave (July 14–20) there were an estimated 739 deaths (485 recorded as HRI by the medical examiner) and 1072 hospitalizations (731 recorded as HRI in the medical record). [75, 145]. During the August 2003 heat wave in Europe, it is estimated there were between 27,000 and 44,800 deaths (dependent on the countries included) [8, 146]. However, heat-related outcomes do occur outside of these short intense heat episodes and understanding the relationship between ambient temperature (or daily heat) and heat-related outcomes is essential to characterizing the heat-health relationship. Understanding this relationship will also guide response and interventions related to prevention of HRI morbidity and mortality.

In the northern hemisphere, HRI is typically seen during the summer months (e.g., May–September), although a few cases are observed outside this time period [147, 148]. When examining the heat-health relationship outside of a heat wave, restricting the analysis to the summer months is common in the literature in order to reduce bias due to seasonal trends [1]. Studies of heat-related ambulance call-outs and emergency dispatches indicate that the number of heat-related events does not vary greatly by day of the week, although some studies have suggested a slightly higher number

[20]Number of employed civilian non-institutional population in 2009 = 139,877,000 [139]

[21]Note that the number of HRI hospitalizations/emergency departments are obtained from administrative data used for medical billing purposes. These cases are identified by an ICD-9-CM code of 992.0–992.9. The numbers reported by the Bureau of Labor Statistics (BLS) are from the annual Survey of Occupational Injuries and Illnesses (SOII) and are obtained from Occupational Safety and Health Administration (OSHA) logs of workplace injuries and illnesses maintained by employers. As such there may be some overlap between the hospitalization/ED numbers and the BLS numbers dependent on where the individual sought treatment.

of events occurring on Saturdays and holidays [149-152]. It has also been indicated that the afternoon to early evening is the peak time for HRI call-outs and ED admissions [150, 151, 153, 154].

EPIDEMIOLOGY OF HRI IN THE GENERAL POPULATION[22]

There is a strong positive relationship between HRI morbidity and mortality and increasing outdoor temperature. For instance, a study of hospitalizations among California residents (1999-2005) saw a 404 percent increase in HRI (ICD-9-CM = 992) for every 5.6°C (10°F) increase in mean apparent temperature (95% CI = 309.2, 520.8), while a similar study of ED visits (2005–2008) observed a 393.3 percent increase in HRI for every 5.6°C (10°F) increase in mean apparent temperature (95% CI =331.2 to 464.5) [78, 82]. In Toronto, Canada there was a 33 percent (SE = 0.0303, p< 0.0001) increase in the rate of ambulance call-outs for every 1°C increase in mean temperature. During heat waves, the magnitude of the effect varies. For instance, in Adelaide Australia the rate of HRI[23] treated in the ED and hospital during three heat waves ranged from 2.68-12.01 and 3.12-13.66 times as high as the rate during non-heat wave periods, respectively. [155]. During the 2006 California heat wave, the rate of HRI (ICD-9-CM = 992) ED visits and HRI hospitalizations were 6.30 (95%CI = 5.67, 7.01) and 10.15 (95% CI = 7.79, 13.43) times, respectively, the rate during non-heat wave periods [87]. During a hotter than average year (2005) in Maricopa County, Arizona, a 182 percent increase in mean HRI-deaths (ICD-10 = X30) compared with prior years was observed [156]. In a study of mortality during six heat waves in France (1973–2003) the ratio of observed numbers of heat-deaths[24] compared to the expected, ranged from 3.37 to 18.15 [68].

---

[22]Only papers which report a simple summary statistic (e.g., percent change, odds ratio, rate ratio) for all age groups are reported here. The methodology to estimate the simple summary statistic varies from paper to paper.

[23]Defined as: effects of heat and light [ICD-9-CM 992; ICD-10 = T67], heat stroke [ICD-9-CM = E900], dehydration [ICD-9 = 276.5; ICD-10 = E86], heat stroke [ICD-9 = E900; ICD-10 = X30]

[24]Defined as: dehydration [ICD-9 = 276.5; ICD-10 = E86], syncope/collapse [ICD-9 = 780.6; ICD-10 = R50.9] and heat stroke [ICD-9 = E900; ICD-10 = X30]

The most often studied outcome when examining the heat-health relationship is all cause morbidity or mortality. For all-cause mortality, globally, the percent change in mortality for a 1°C change in temperature ranges from 0.4-18.8 percent, with warmer climates having a potentially larger effect estimate than cooler climates [1, 4, 74, 85, 86, 112, 157-168]. For studies comparing extreme heat days to non-extreme heat days, the percent increase for mortality ranges from 1.26-43 percent depending on how a heat wave or extreme heat day is defined (see section on Heat Waves) [18, 84, 143, 169-176]. Among heat wave studies, a larger relative effect of heat waves on mortality is seen in cooler climates than in warmer climates [91, 143]. In the U.S., for a 5.5°C (10°F) change in temperature, the percent change in mortality ranges from 2-14.9 percent [1, 177]. The heat-health literature on mortality is much more extensive than for morbidity. However, heat wave studies have observed an increase in all-cause morbidity ranging from 1-11 percent [70, 75, 83, 87, 155].

Diseases of the circulatory system and respiratory diseases are the most commonly examined heat-related cause-specific outcomes. For a 1°C change in temperature, the literature reports a 0.7-7.7 percent increase in circulatory system mortality[25] [4, 74, 85, 158, 159, 162, 164, 166, 178] and a 0.8-12.5 percent increase in respiratory mortality [4, 74, 85, 158-160, 162, 164, 168]. Studies of heat waves also note an increase in circulatory (2.1-139% increase) and respiratory mortality (2.4-82% increase[26]) [18, 84, 170, 172, 173, 176, 179]; however, not all results are statistically significant[27] [18, 170]. A few studies have shown either no association or a non-statistically significant association for circulatory system mortality [162, 164, 166, 168, 180] and respiratory mortality [4, 166].

The findings for morbidity due to diseases of the circulatory system and heat are inconsistent. For every 5.5°C (10°F) increase in temperature, Basu et al. reported a 0.2 percent increase (95% CI

[25]One possible outlier – in rural Bangladesh a 62.9 percent increase (95% CI 23.2, 115.2) in circulatory system mortality was observed between 1996 and 2002 [165].

[26]One possible outlier – during the 1994 heat wave in the Netherlands, a 120 percent increase in respiratory mortality was observed compared to a 31 day moving average of the prior 2 years. For the other five heat waves observed the excess respiratory mortality ranged from 9.4–47.1 percent [176].

[27]One possible outlier – Bell et al. observed a -7.15 percent change (95% CI = -15.43, 1.95) in respiratory mortality in Mexico City between 1998 and 2002 [170].

= -0.9, 1.3) in ED visits for diseases of the circulatory system while Green et al. reported a 0.2 percent decrease (-1.2, 1.0) in hospitalizations due to diseases of the circulatory system [78, 82]. In Europe, a decrease in circulatory hospitalizations was also observed (North Continental cities: % change = -0.6; 95% CI = -1.2, 0.1, Mediterranean cities: % change = -0.6; 95% CI = -1.8, 0.5) while in New York City (% change = 3.6, 95% CI = 0.3, 6.9) and London (% change = 1.71; 95% CI = -2.70, 6.33) increases were observed [69, 71, 81]. One study of heat waves in Brisbane, Australia noted a 0.4 percent increase (95% CI = 0.87, 1.24) in circulatory morbidity, all other studies showed a negative association, 0.1-0.2 percent decrease [70, 79, 83, 181].

The evidence for an association between respiratory morbidity and heat is stronger than for circulatory morbidity. For a 1°C increase in temperature a 0.3-5.44 percent increase in respiratory hospitalizations has been observed [69, 71, 81, 86]. However, Basu et al. reported a 0.7 percent decrease in respiratory ED visits for 5.5°C (10°F) increase in temperature [78]. The differences in circulatory morbidity and mortality results may be due to the rapid onset of the outcome and the short period between heat exposure and death [69, 71, 72]. In other words, there may not be enough time for individuals with a circulatory endpoint to present to a hospital or emergency department. However, the onset of a respiratory outcome is slower, allowing time for individuals to be treated in a hospital or ED [71, 72].

A third endpoint of particular importance is acute renal failure, as this is a symptom of exertional HRI. However, very few morbidity studies and no mortality studies have examined this cause-specific outcome. In two California studies, a 5.5°C (10°F) increase in temperature was associated with a 7.4 (95% CI = 4.0, 10.9) and 15.9 (95% CI = 12.7, 19.3) percent increase in hospitalizations and ED, respectively, due to acute renal failure [78, 82]. In New York State a 3.5-6.4 percent increase in hospitalizations for acute renal failure for every 2.78°C (5°F) increase in the temperature exposure metric (e.g., mean, min, max temperature/apparent temperature) was observed [95]. The only non-significant increase was in London (1994–2000) where for every 1°C increase in the mean temperature there was a 2.58 percent increase in ED visits for acute renal failure (95% CI = -0.10, 5.32) [69]. During the 2006 California heat wave, an 11 (95% CI = 8, 15) and

15 (95% CI = 11, 19) percent increase in hospitalizations and ED visits for acute renal failure, respectively, was observed [87]. Additionally, a 12-year study of heat-waves in Australia observed a 25.5 (95% CI = 3.7, 51.9) percent increase in acute renal failure hospitalizations [65]. Finally, the largest percent increase (131%) reported in the literature for hospitalizations due to acute renal failure was during the 1995 Chicago heat wave [75].

OCCUPATIONAL HRI EPIDEMIOLOGY

Exposure to environmental heat is a clear recognized hazard for many occupations where individuals are not able to maintain thermal equilibrium due to their work environment (e.g., hot and humid), clothing type, protective equipment, and the inability to self-regulate their work-rate (i.e., individual behavioral modification). Examples of such occupations include outdoor workers (e.g., construction workers, agricultural workers, or landscapers), firefighters, kitchen workers (including bakers), miners, factory workers, soldiers, and metal smelters [42, 182]. As with most environmental exposures, studies of occupational exposure to heat provides a great deal of information on the diagnosis, treatment, and prevention of HRI [183-186]. However, the epidemiology of occupational HRI is not well characterized. From the information that is available there is an indication that some occupations have a higher burden of heat-related morbidity and mortality than others, especially those which require high exertion and outdoor activity.

Soldiers are required to perform strenuous physical activity for extended periods of time, wear protective clothing which may decrease sweating evaporation, and work in extremely hot environments or spaces[28] [3, 187]. Additionally, highly motivated individuals may exert themselves beyond their physiological capacity in order to exceed individual and group expectations [188]. Among U.S. Army personnel, the rate of heat-related hospitalizations between 1998 and 2002 was 20/100,000 soldiers while the death rate between 1980 and 2002 was 0.3/100,000 soldiers [187]. Over an 11-year period (1981-1991), the British Armed Forces identified an incidence rate for HRI of 70.63/100,000 soldiers in the Army, 13.54/100,000 soldiers in the Royal Navy, and 5.05/100,000

[28]For instance, the interior temperature in an armored fighting vehicle is approximately 13.1°F above ambient temperature which in a hot-dry environment can be 90°F-114°F [3].

soldiers in the Royal Air Force [189]. The Israeli Defense Force found that between 1988 and 1996 the majority of their heat stroke cases occurred during basic training (57%) and screening for special forces (21%) [188].

While information on the epidemiology of occupational HRI is scarce the results from the military may be similar for other occupations where individuals may be required to perform past physiological capacity, in extreme environments, while wearing impermeable protective equipment, such as firefighters. A report from Washington State (2000–2009) found a workers' compensation HRI claim rate of 108.1/100,000 full-time equivalent (FTE) for individuals in the fire protection industry [147]. The military experience may also shed light on HRI in civilian disaster workers. During the Midwest flooding of 1993, the Army National Guard was deployed to Illinois [190]. An HRI incidence rate of 136/100,000 person-days was seen in July – the period of most intense sandbagging and flooding when Guardsmen were reported to have worked 12 hours a day, 7 days a week [190].

Within the civilian population, individuals working in the mining industry are at risk of HRI. This is especially the case for underground mining which is hot and humid due to the geothermal gradient, auto-compression (i.e., increasing air temperature with decreasing depth due to increasing air pressure), and transfer of heat from groundwater and mining water to the air (increased humidity) [191]. In Australia an HRI rate of 43.0/million man-hours for underground metal mining was observed while in South Africa an HRI rate of 46.8/million man-hours for underground gold mining was seen [191, 192]. There is very little information on the incidence of HRI in other sectors of the mining industry; however, a study in the U.S. using reports of injuries and accidents to the Mine Safety and Health Administration found that the rates of HRI varied greatly by type of mining operation, most likely due to the type of material and the methods of extraction [192]. For instance, in the U.S., underground coal mining had a lower rate (0.00275/million man-hours) than underground metal mining (rate = 0.168/million man-hours) or work at a stone mill/preparation plant (rate = 0.417/million person hours). Overall, employees working in mills/preparation plants (0.0629/million person hours) and those in surface mine operations (0.0424/million person hours) had a higher rate of HRI than employees working in the underground mines (0.0184/million person hours). This study suffered from

substantial under-reporting of HRI occurring in U.S. mine operations[29] [192]. Regardless of the rates in the U.S. study, the distribution provides information on how the occurrence of HRI is affected by work-environment even in a single industry. Additionally, in the U.S. study and Australian studies, the majority of HRI cases occurred during the summer months when the ambient temperature was high, even in the underground mines [191-194].

Individuals working outdoors in hot weather in jobs which may require heavy labor and limited ability to self-regulate their work-rate are also at risk of HRI. A study in Washington State (1995-2005) using workers' compensation claims identified the following industries as having the highest HRI claim rate: fire protection (rate = 80.9/100,000 FTE), roofing construction (rate = 59.0/100,000 FTE), and highway, street and bridge construction (rate = 44.8/100,000 FTE), site preparation construction (rate = 35.9/100,000 FTE) and poured concrete foundation and structural construction (rate = 35.9/100,000 FTE) [195]. The California Occupational Safety and Health Administration (Cal/OSHA) found that the majority of their HRI investigations in 2005 and 2006 were among construction workers (2005 = 29%; 2006 = 21%) and agricultural workers (2005 = 38%; 2006 = 29%) [196, 197].

In North Carolina, a study examining death certificates from 1977–2001, found that occupational heat-related deaths most often occurred at a farm/agricultural property (40%) or at a construction site (25%) [198]. The study also reported a heat-related fatality rate among farm workers of 1.52/100,000 workers/year [198]. Nationwide, between 1992 and 2006, the heat-related death rate for crop workers was 0.39/100,000 workers compared to a rate of 0.02/100,000 for all U.S. civilian workers [199]. The majority of deaths among crop workers nationwide (57%) occurred in California, Florida, and North Carolina [199]. Additionally, a national analysis of the Census of Fatal Occupational Injuries (CFOI) database from 2000–2010 found that six of the ten states with the highest rates of occupational heat-related deaths were located in the southeastern U.S. [10]. In order to assess the impact of HRI prevention measures in the working population or to have a clear picture

[29]For instance, over a 19 year period, only 2.5% of U.S. mining operations reported any cases of HRI [192].

of which industries and occupations to target, further information on the incidence of occupational HRI is required.

POTENTIAL SUSCEPTIBILITY FACTORS

There are a number of vulnerability factors that have been demonstrated in the literature to increase the risk of heat-related outcomes, for both the general population and the working population. Many of the factors are inter-related and vary dependent on another factor. For instance, the impact of poverty on the heat-health relationship may be greatest among the elderly [200, 201]. The identification of factors associated with heat-related outcomes can be used to target interventions of high risk areas, defined as the geographical cluster of vulnerable sub-groups (e.g., elderly, Black men residing in the inner city) [129, 200, 202, 203].

AGE

Age modifies the heat-health relationship. Within the mortality literature the elderly have been shown to have the highest risk of heat-related outcomes [1, 2]. This may be due to the biological effects of aging (e.g., decreased ability to sense temperature changes), the increase in chronic diseases, or the increased use of medications which may impede the thermoregulatory system. Additionally, older individuals may be more apt to suffer from social isolation or the inability to employ behavioral modifications due to immobility. The definition of elderly varies by study (e.g., 65+ or 65+ in 10 year bins) with the largest impact seen among those 75 or older [204]. The very young (<5 years) have also been shown to have a higher risk of heat-related outcomes which may be in part due to their underdeveloped thermoregulatory system or their inability to employ individual behavioral modifications [1, 2]. Similar results for age are seen within the morbidity data. This may a true association or it may be a factor of how the majority of morbidity studies categorize age, which are done in a similar manner to the mortality studies.

Typically studies examine the young (e.g., <1, <5, occasionally 5-14), the elderly (e.g., 65-75, 75-84, 85+), and all other ages combined (e.g., 15-64). As discussed previously there are two types of HRI –exertional and classical, with exertional HRI most often affecting young and healthy

individuals. Fatal exertional HRI cases are rare and therefore not typically observed in the mortality data [96]. A recent study using data from the National Electronic Injury Surveillance System of the U.S. Consumer Product Safety Commission specifically looked at exertional HRI in the general population and observed that only 9.6 percent of HRI cases were transferred to the hospital while the majority were treated in the emergency department and released [205]. Further, a study of individuals hospitalized for acute renal failure during the summer in New York (1991–2004) found that for every 2.8°C (5°F) increase in temperature individuals age 25-44 had the highest estimate of hospitalization (OR = 1.18 95% CI = 1.08, 1.27), twice that of the reference group age 45-64 (OR = 1.09; 95%CI = 1.03, 1.14; p= 0.09) [95]. Very few studies have included age at a finer resolution than the broad grouping of ages 15-64 years (morbidity [72, 205, 206]; mortality [84, 136, 207]). In order to have a clearer and more complete picture of the relationship between HRI and heat, categorization of age needs to be analyzed at a finer resolution than has been done in prior studies.

GENDER

Biologically speaking there is no difference between men and women in their susceptibility or thermoregulatory response to heat [41], although it has been suggested that women tolerate humid heat better than men and men tolerate dry heat better than women [208]. In previous work, gender appears to modify the heat-health relationship [1, 161, 169, 209], although a few studies have not found modification [67, 82, 95, 210, 211]. For instance, the majority (66%) of heat-related deaths in all age groups in the U.S. between 1999 and 2003 occurred in men [136]. Conversely, more often within the mortality literature, heat-related mortality is greater for women than for men [161, 169, 212]. This observation may be due to other factors such as the gender age-distribution in the population (e.g., greater number of elderly women) and social factors (e.g., married versus unmarried) [65, 213]. Gender has not been examined often within the morbidity literature, but heat-related morbidity has been seen to be greater for men than for women [88, 89, 205, 214]. This may be more a factor of exertional HRI and related to the activities that men versus women are involved in. For instance, Kerr and colleagues used the National High School Sports–Related Injury Surveillance System data (2005/2006–2010/2011) to examine HRI among high school athletes [215]. They found that the

majority of heat-events (87.7%) were among boys, however, when football players were removed, boys accounted for 50.9 percent of events [215]. A study of Marine recruits on Parris Island between 1982 and 1991 saw no difference in the rate of HRI among male and female recruits but, potentially due to a more rigorous training regime, the type of HRI was more severe among male recruits than females [148]. Increased severity in males compared to females has been reported in other studies [216]. Additionally, males are also more likely to work in areas with higher risk of heat-related injury such as construction or fire protection [147, 217].

RACE/ETHNICITY

Race and ethnicity appear to modify the heat-health relationship although the results are somewhat inconsistent [64, 81, 87, 145, 218]. A case-control study of Marine recruits, matched on initial training platoon, found that Black recruits and recruits of other races had 1.6 (95% CI = 1.2, 2.1) and 1.3 (95% CI = 0.7, 2.3) times the odds, respectively, of HRI compared with White recruits [219]. In comparison, a study of HRI in the U.S. Army found that all races, except Asian/Pacific Islander males and males of other races, had a lower rate of HRI compared to Whites[30] [187]. Within the U.S. population, the rate of HRI death among Blacks is 4.9 times that among Whites, while the HRI death rate among other minorities is 0.8 times that among Whites [220]. Modification by race/ethnicity also appears to vary by data source (e.g., hospital, ED, death certificates) and type of outcome (e.g., ischemic heart disease, acute renal failure, respiratory) [87]. In California, a study of cause-specific hospital admissions and daily temperature during the summer months found no modification by race or ethnicity [82], while a similar mortality study saw a varying increase in non-accidental mortality by race/ethnicity for every 5.6°C (10°F) increase in mean daily apparent temperature (estimated percent increase: Whites = 2.5%, Blacks = 4.9%, Hispanics = 1.8%)[31] [67]. Finally, a third California study,

---

[30]The racial groups were White, Black, Hispanic, Asian/Pacific Islander, Native American/Alaska Native and other. Only the comparison for Blacks compared to Whites (both male and female) and Hispanics females compared to White females were statistically significant.

[31]The numerical confidence interval values for each percent increase were not provided in the paper. Instead, the authors presented a figure with these results. Examination of the figure indicates non-statistical significance for all comparisons. However, it appears even when the variance of the effect estimates are accounted for, there is a larger difference between the results for Blacks versus Whites than for Hispanics versus Whites. The confidence interval for Whites appears to be completely within

with similar methodology, examined ED data and found that for every 5.6°C (10°F) increase in mean daily apparent temperature compared to Whites: Hispanics had a higher risk of ischemic heart disease (% excess risk = 7.2%; 95% CI = 2.7%, 11.9%), ischemic stroke (% excess risk = 5.2%; 95% CI = -0.1%, 10.7%) and acute renal failure (% excess risk = 21.8%; 95% CI = 14.6%, 29.5%); Asians had a higher risk of dehydration (% excess risk = 37.4% ; 95%CI = 24.9%, 51.1%) and primary diabetes (% excess risk = 7.6%; 95% CI = -0.1%, 17%) [78]. Blacks had a lower or similar excess cause-specific ED morbidity as compared with Whites [78]. In another area of the country, a case-cross over study in New York city among individuals hospitalized for acute renal failure during July and August 1991–2004, found that compared with Whites (OR = 1.08; 95% CI = 1.05, 1.11), Blacks had a higher odds of acute renal failure (OR = 1.14; 95% CI 1.08, 1.20; p =0.06) while Asians had a lower odds (OR = 0.88; 95% CI= 0.74, 1.05; p= 0.03). Hispanics had a higher odds of acute renal failure (OR = 1.20; 95% CI = 1.09, 1.32; p = 0.05) compared with non-Hispanics (OR =1.09; 95% CI = 1.06, 1.12) [95].

Inconsistencies between the data sources may be due to the differences in medical care/facility usage among minority populations [87]. Additionally, the modification by race/ethnicity demonstrated in the literature may also be a proxy for other factors such as socio-economic status (including access to external cooling mechanisms), community concession, and occupation. O'Neill and colleagues studied the association between temperature and mortality in four U.S. cities (1986–1993) and found that the percent change in mortality for temperatures at 29°C (84.2°F) compared to 15°C (59°F) were higher for Blacks (% change = 9.0; 95%CI = 5.3, 12.8) than for Whites (% change = 3.7; 95% CI = 1.9, 5.4) [171]. The authors also noted the prevalence of central air conditioning usage in the four cites was double the usage for Whites as compared to Blacks explaining approximately 64 percent of the disparity in heat-related mortality [171]. In Klineberg's social autopsy of the 1995 Chicago heat waves he postulates that the reason Blacks had the highest death rate during the heat wave was that they were the only group who lived in communities with "… high levels of abandoned housing stock, empty lots, depleted commercial infrastructure, population decline, degraded

---

the confidence interval for Hispanics while the upper confidence limit for Whites is smaller than the effect estimate for Blacks.

sidewalks, parks and streets, and impoverished institutions" [7]. The contribution of all of these factors, during the 1995 Chicago heat wave, made it difficult to use public spaces (e.g., spending time in cooler outdoor locations like parks) and organize effective social networks, which in other communities may have lowered the risk of death [7]. Finally, there is a suggestion that among the working population there may be a higher proportion of minorities with HRI as compared to Whites which may be due to the proportion of minorities employed in high risk occupations [197-199, 221].

SOCIO-ECONOMIC STATUS

Indicators of socio-economic status (SES) have been examined in a number of studies. These indicators include education status/attainment, income level, percent poverty, and various social deprivation indexes. The heat-health relationship has been shown to vary by individual socio-economic indicators [210, 218, 222, 223], although no association, or association with a large variance, has also been observed with educational attainment [67, 170, 224]. Further, when employing socio-economic indicators at a community level (e.g., SES at the census tract level) the modification is not quite as clear [17, 90, 95, 201, 225, 226]. A study of four Italian cities (1997–2003) found that median population income level within the residential census block did not modify the mortality-mean apparent temperature relationship [212]. However, another study of the same four Italian cities, limited to heat waves in 2003, observed the greatest excess mortality within the cities were for sub-groups with a low SES indicator in Turin (low education attainment = 43% increase) and Rome (low SES in census tract of residence = 17.8% increase) [179]. In their study of respiratory and cardiovascular deaths in 12 U.S. cities, Braga et al. found no association between death on hot days (24 hour mean of 30°C [86°F]) and percent of population in poverty, with a college degree, or unemployed [90]. However, in Curriero et al.'s analysis of 11 Eastern U.S. cities (1973–1994) the authors found that a 10 percent increase in the percentage of persons living in poverty within a city and an increase in the percentage of persons 25 years or older who did not complete high school increased the mortality risk by 4.3 (SE = 0.42) and 2.8 (SE =1.01) percent, respectively, per 5.6°C (10°F) increase in temperature after adjustment for latitude [17]. A study in Brisbane, Australia (1996–2004) observed a higher percent increase in mortality for every 1°C increase in average temperature

for men residing in census areas with a high SES (% change = 4.34; 95% CI = -6.39, 15.06) compared to men residing in areas with a low SES (% change 1.13; 95% CI = 0.42, 11.13), for women there was no difference (% change: high SES = 11.50, low SES = 12.69) [161]. Part of the discrepancy observed with socio-economic indicators may be the size and potentially heterogeneous nature of the geographic areas used in analyses, which may obscure any modification [161, 201]. Additionally, when modification is observed it may be a proxy for other factors which may play a larger role in the relationship between heat-related outcomes and health, such as access to medical care, cooling mechanisms, or housing condition [141, 200].

Medical Factors

As has been discussed previously, individuals with chronic diseases are at higher risk of heat-related outcomes. In addition to cardiovascular diseases, respiratory diseases, and diabetes, individuals with mental illness (or cognitive impairment), cancer, and obesity have also been shown to be susceptible [181, 207, 218]. For individuals with mental illness, their susceptibility may be related to their inability to employ behavioral modification due to cognitive impairment [227]. The use of medications and illicit drugs which impede the body's ability to thermoregulate also increase an individual's risk of a heat-related outcome [57]. Thermoregulation impairment through medication usage can potentially be a factor for a number of chronic conditions. Individuals who have previously had a HRI episode may be at high risk of another episode due to innate heat intolerance [228-230]. In individuals with sickle cell anemia, excertional heat stress may lead to increased sickling of red blood cells and the rapid onset of severe, potentially fatal rhabdomyolysis [231]. Finally, individuals who lack acclimatization or who are physical unfit are more likely to have a heat-related outcome [93, 96].

Air-Conditioning

Reducing exposure to hot environments provides protection against HRI. As such, the use of air-conditioning is considered behavioral modification (a type of adaptation). Access to air-conditioning in the U.S. has been shown to be a protective factor against the development of heat-related outcomes [1, 17, 141, 171, 202, 232]. However, access and usage of air-conditioning is

dependent on a number of factors including social economic status (e.g., inability to afford air-conditioning unit or pay for electricity), geographic location (e.g., higher prevalence in warmer climates), personal feelings about usage (e.g., dislike of air-conditioning) [171, 233, 234]. A limitation of these studies is the limited availability of data on the presence and use of air-conditioning; as such studies have only been conducted in urban areas, where most information is available. Additionally, some authors have hypothesized that widespread usage of air-conditioning may make individuals more susceptible to the effects of heat due to a lack of physiological acclimatization [235].

Other Factors

Housing characteristics which increase the indoor temperature such as residing in a home with a high thermal mass (e.g., brick house) or living on the top floor of an apartment building, are factors which increase susceptibility [39, 236]. As heat rises, the top floor of an apartment building is usually the warmest. Additionally, traditional roofs absorb solar energy and can reach between 150 to 185°F (66-85°C) during the day [236]. Residents closest to the roof will be more affected by this increase in temperature than residents on lower floors. Homes with a high thermal mass are slow to absorb heat but retain the heat longer than homes with a lower thermal mass [237]. Without proper internal cooling (e.g., air conditioning) the home can become very warm.

Individuals who reside in an institution (e.g., nursing home, or long-term care facility) have increased susceptibility for developing HRI during an extreme heat event or as temperatures increase [212]. This may be due in part to immobility, the inability to employ individual behavioral modification, or building characteristics (e.g., lack of air-conditioning). Finally, social isolation, the inability to care for oneself, and being a non-native speaker all put an individual at higher risk for a heat-related outcome [7, 202, 204, 238, 239].

Urban Heat Island Effect [236]

Residing or working in an urban area is considered a risk factor for heat-related outcomes due in large part to the urban heat island effect [6, 39, 77, 204, 208, 236, 240]. Urban heat islands refer to pockets of the landscape that have a higher temperature than the surrounding area. Urban

heat islands occur in metropolitan and sub-metropolitan areas where dry, impermeable, and reflective surfaces cause the temperature in those areas to be higher than the temperature in the surrounding rural areas where surfaces are permeable and moist[32]. There are two types of urban heat islands: surface and atmospheric (Figure 2-3).

Surface heat islands are due to the sun heating dry exposed areas (e.g., roofs of buildings or pavement). The surface effects are present throughout the day and night but are the most intense during the day when the sun is shining directly on the exposed surfaces. The temperature due to the surface effects during the day can be between 10-15°C (18-27°F) higher than in the surrounding rural area and at night between 5-10°C (9-18°F) higher than the surrounding rural areas. Micro-heat Islands within urban areas can also occur in areas without vegetation. Areas with vegetation provide shade and moisture which, respectively, lowers surface temperatures and releases water into the air cooling the surrounding area.

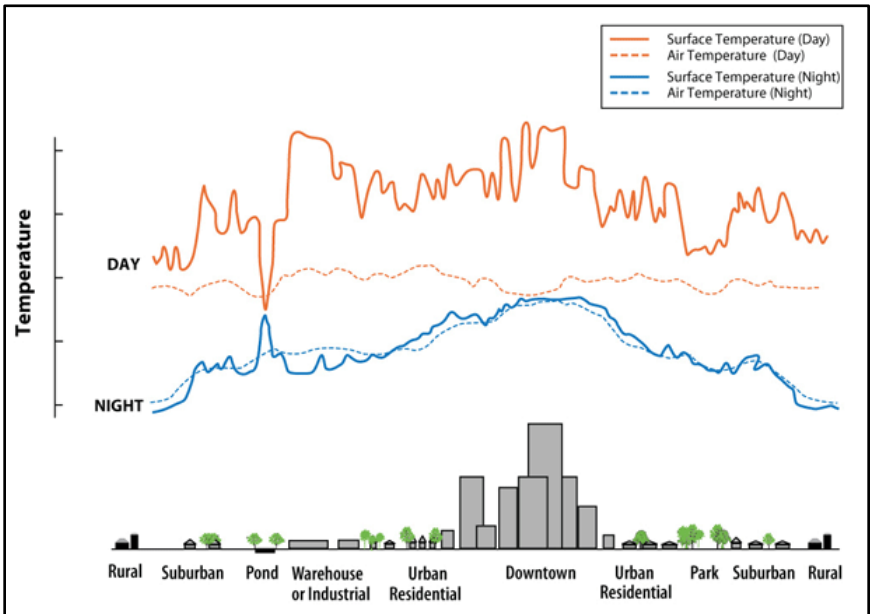


Figure 2-3. Example of variations of surface and atmospheric temperatures in relation to various levels of urbanization (Reproduced from EPA 2008 [236])

Atmospheric heat islands are divided into the canopy layer (i.e., ground level to the tops of trees/buildings) and the boundary layer (i.e., top of trees/buildings to approximately one mile up). The canopy layer is most often observed and relevant to human exposure. As such, the boundary heat islands will not be discussed further. Canopy heat

[32]Heat island effects can also be seen in desert regions with dry impermeable surfaces as compared with surrounding regions whose surfaces are moist and permeable.

islands are most notable at night where the temperature can be 7-12°C (12.6-21.6°F) higher than surrounding rural areas. During the day urban structures absorb heat from the sun and heat reflected off other surfaces (e.g., glass or pavement). At night the absorbed heat is released creating heat islands. Additionally, the make-up of cities (e.g., tall buildings situated close together) traps the released heat within the canopy layer preventing it from dissipating into the upper atmosphere.

RURAL AREAS

The majority of the research examining the heat-health relationship has been conducted in urban areas [1, 66, 157]. There is very little research examining this relationship in rural areas and therefore it is difficult to say if risk of HRI differs for individuals residing or working in rural versus urban areas. The studies that have been conducted indicate that even in rural areas heat-related outcomes increase with increasing heat (e.g., temperature or heat wave versus no heat wave). For instance, in townships located in rural Victoria, Australia there was an 8-65 percent increase (dependent on location) in total mortality among the elderly population for days where the temperature was above the pre-defined threshold [241]. The literature suggests that urban areas have a higher burden of heat-related outcomes than rural areas; however, only a handful of studies have been conducted that have mentioned both rural and urban areas. A study conducted during the summer months in Ohio for the period 1975–1998, found that rural counties had a higher rate of total mortality than urban counties (27 versus 21/million population/year, respectively) [105]. A study of HRI deaths among elderly U.S. residents (1979–1985) found that the highest HRI death rates among non-White residents were in poor urban counties, while the highest HRI death rates for Whites were in rural/non-urban counties [225]. These studies are obviously limited by the large geographical scale used to classify urban and rural areas. Hajat and colleagues classified rural/urban areas in England and Wales by a smaller geographical unit, census output areas[33] and found that the heat effect for total mortality was greater for urban areas than for rural areas [226]. However, when rural/urban areas were further stratified by a social deprivation index (e.g., SES), rural areas with the highest

[33]The census output areas have an average population size of 300 and are the smallest geographical unit used in the 2001 United Kingdom census.

level of social deprivation (i.e., lowest SES) had the highest relative risk of death[34] [226]. During the 2003 heat wave in Europe, a 40 percent increase in mortality was estimated in rural Spanish villages compared with a 10 percent increase mortality in Spanish urban areas [242]. In Germany, during two heat waves, total mortality in the urban area of Berlin was examined in conjunction with the surrounding rural area of Brandenburg. For both heat waves, the percent increase in mortality compared to baseline was higher in Berlin (1994: Berlin = 44.7%, Brandenburg = 32.3%; 2006: Berlin = 19.2%, Brandenburg = 16%) [243]. A heat wave in the Latium Region of Italy during July of 1983, observed a 23.5 percent increase in total mortality in Rome and 48.5 percent increase in the rural areas surrounding Rome [244]. Finally, a study looking at the July 1980 heat wave in Missouri, found that increase in total mortality in rural areas (9.5%) was less than the increase in total mortality in urban areas (St Louis = 56.8% and Kansas City 645.2%) [245]. Regardless of a potentially lower burden of injury in rural areas, rural areas may have a different set of associated factors related to the heat-health relationship than urban areas, such as disparities in socio-economic status, disparities in access to health care, increased humidity (due to vegetation), and greater (or different) occupational risks (e.g., farmers/agricultural workers) [196, 197, 200, 202, 246].

KEY ASPECTS OF MODELING THE HEAT-HEALTH RELATIONSHIP

EXPOSURE-RESPONSE CURVE

Current literature has also explored the shape of the association between heat and morbidity and mortality as well as the relevant time windows. Specific aspects of the shape and timing of exposure will be discussed here.The relationship between temperature and morbidity/mortality is non-linear with short lagged effects. The shape of the exposure-response curve has been shown, in the literature, to be linear (above a threshold) or J-shaped [2, 17, 18, 88, 162, 232]. When the entire temperature spectrum (cold to hot) is considered the relationship between morbidity/mortality and temperature is U-shaped [2, 17, 88]. The shape of the exposure-response relationship may vary by location. For instance, when examining total mortality in 11 cities in the Eastern U.S. (1973–1994) the

[34]The variability in the rural results is greater than in the urban results (tight confidence intervals) according to the figures presented in the paper.

hot/cold extremes exposure-response curve for southern cities could be described more as a hockey stick[35] than a U-shape [17]. A hockey stick exposure-response curve has the highest response at the lowest temperature (e.g., -20°F), the second highest response at the highest temperature (e.g., 95°F) and the smallest response at a threshold or change point (e.g., 70°F). The exposure-response relationship from the change point to the lowest temperature and the exposure-response relationship from the change point to the highest temperature are usually linear. In a study of 15 European cities (1990–2000), Mediterranean cities were found to have a steeper slope above the heat threshold as compared with Northern-continental cities [162]. Based on the available information it appears that it is important to model a non-linear, region (or area) specific exposure-response curve to prevent erroneous effect estimates [18, 247].

THRESHOLDS

The temperature above which minimum heat-related outcomes are observed is the threshold temperature. The temperature threshold may be a single value or a range or values. Within the literature three general methods have been used to identify threshold temperatures; 1) visual inspection of the exposure-response curve, 2) statistical (i.e., maximum likelihood) or mathematical methods (i.e., derivatives) applied to exposure-response curve, or 3) percentiles of the exposure data (e.g., 95th or 99th) [1]. Note that identification of threshold points for the latter method is based entirely on the exposure data. When the method used to estimate the threshold cut-point varied the resulting effect estimate for the heat-health relationship also varied. For instance, Hajat et al. examined four threshold cut points in their analysis of daily mortality in the greater London area (1976–1996) – 18.9°C (90th percentile), 20.6°C (95th percentile), 21.5°C (97th percentile), and 23.3°C (99th percentile) – and observed a 2.5 (95% CI =2.35, 2.9), 3.2 (95% CI =2.56, 3.8)[36], 3.4 (95% CI =2.47, 4.23), and 5.71 (95% CI = 4.3, 7.15) percent increase in average deaths per one degree increase in temperature above the threshold, respectively [163]. As previously mentioned, average daily temperature varies by geographic location and individuals become acclimatized to their average

[35]Hockey stick = 

[36]The effect estimates and 95% CIs for the 90th and 95th percentile are approximations based off the graph provided in the paper.

temperature. As such, the threshold value varies by location, with warmer climates having a higher threshold than cooler climates, potentially indicating, in the warmer climates, a more acclimatized population (both physiological and behavioral) (Figure 2-4). As such, within the literature, city or region specific thresholds are typically used.

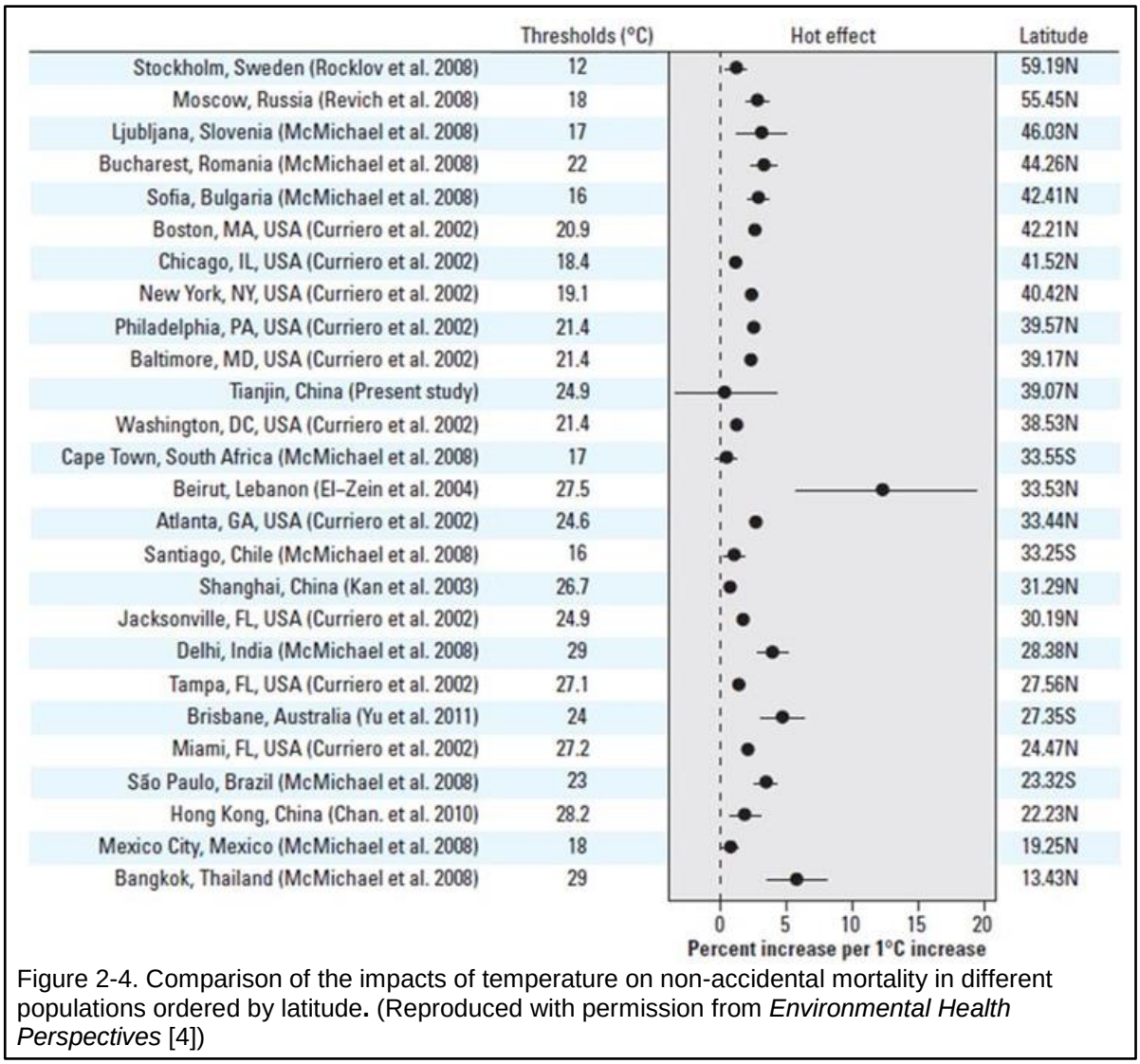


Figure 2-4. Comparison of the impacts of temperature on non-accidental mortality in different populations ordered by latitude**.** (Reproduced with permission from *Environmental Health Perspectives* [4])

LAGGED EFFECTS

Within the literature, it has been observed that temperature generally has an immediate effect on the heat-related outcome (lag 0) but potentially related outcomes may be observed up to three days later (lag 1-3) [17, 66, 82, 158, 162, 232]. Additionally, some studies suggest that the largest

effect (lag 0-3) may vary by sub-group and cause-specific outcome [70]. For instance, a study of hospital admissions for acute renal failure in New York state (1991–2004) reported for a 2.78°C (5°F) change in maximum daily temperature an odds ratio of 1.059 (95% CI = 1.045, 1.086) at lag 1 (i.e., the prior day) and 1.043 (95% CI = 1.024, 1.063) at lag 0 (i.e., the current day) [95]. Among low-income elderly Koreans, the largest effect estimate for the mortality-maximum temperature relationship was at lag 2 while for the general population it was at lag 0 [223]. The shape of the exposure-response curve may be different dependent on the lag day. A study of daily mortality in New York City (1997–2003) observed a linear temperature-mortality relationship for the current day and a non-linear relationship for lags 1, 2, and 3 [123].

Some authors have commented on the contribution of mortality displacement (i.e., harvesting) to the mortality-heat relationship. Mortality displacement occurs when, due to the exposure (e.g., temperature), the deaths of susceptible individuals are accelerated by a few days. They noted that mortality displacement may explain some of the increased mortality observed with increasing temperature [1, 66, 164]. However, this displacement has not been observed in all studies and further investigation is necessary [141, 248]. Additionally, the proportion of heat-related outcomes attributed to mortality displacement may depend on the health status of a population [164].

HEAT WAVES

Heat waves are considered to be periods (i.e., contiguous days) of intense heat, there is currently no uniform definition of the duration or intensity of a heat wave and as a result the definition of heat wave and magnitude of effect (e.g., excess deaths) varies by study making conclusions that would inform policy difficult [16, 38, 52, 249]. As with other heat-health studies, the exposure metric (e.g., maximum temperature or apparent temperature) varies from study to study, with single or multiple exposure metrics (e.g., minimum and maximum temperature) used to define a heat wave [69, 84, 112, 181, 250]. Thresholds are often used to define the intensity of a heat wave. Thresholds are based on the historical summer temperature distribution with the 95^{th}, 97^{th}, 98^{th}, or 99^{th} percentile of that distribution used to define the threshold [65, 84, 98, 155, 169, 181]. For duration, a period of two, three, or four consecutive days above a particular threshold is typically used [18, 65, 79, 83, 85, 112,

169, 173] . However, Gasparrini and Armstrong observed that between 1987 and 2000, 45.4 percent of the 108 U.S. communities within their study had a heat wave of at least 10 days and 81.5 percent of the 108 communities had a heat wave of at least 7 days [251]. Other definitions of a heat wave have also been used. For instance, Bobb et al. defined a heat wave as at least three consecutive days where the maximum daily temperature is above the 97.5th percentile and where the temperature does not fall below the 81st percentile and the average of the entire period is greater than the 97.5th percentile [144]. Gabriel and Endlicher combined three different methods (95th percentile thresholds, thresholds identified by regression model, and days exceeding perceived temperature [32°C during day and 20°C at night]) to identify 3-week high-heat load periods for each year (1990–2006) [243]. Finally, a number of studies have looked at specific heat waves identified by a local/regional heat wave definition[37] [70, 87, 172, 252, 253].

Studies that have examined the varying definitions of intensity and duration of heat have demonstrated that the health effects do vary depending on the definition used [18, 79, 112, 143, 163, 169, 249, 254]. In general, greater effects on morbidity and mortality were seen during longer and more intense heat waves, although intensity appears to have a larger impact. In Brisbane, Australia (1996-2005), a short high intense heat wave defined as at least 2 consecutive days at or above the 99.5th percentile (morbidity [ED] OR = 1.20 [95% CI = 1.13, 1.27]; mortality OR = 1.60 [95% CI =1.33, 1.91]) had a larger effect than a longer less intense heat wave which was defined as four consecutive days at or above the 95th percentile (morbidity [ED] OR = 1.06 [95% CI = 1.03, 1.08]; mortality OR = 1.20 [95% CI =1.15, 1.33]) [249]. In a study of 107 U.S. communities (1987-200), when the intensity of a heat wave was defined as the temperature at or above the 98th percentile, the percent increase in mortality was 3.2 (95% posterior interval = 2.1, 4.3) for a 2 day or greater heat wave and 3.9 (95% CI =2.1, 6.0) for a 4 day or greater heat wave. When the intensity definition increased to at or above the 99.5th percentile, the percent increase in mortality also increased (≥ 2 days: % increase = 6.1 [95% PI =4.0, 8.2]; ≥4 days: % increase 10.6 [95% PI =6.1, 15.3]). A study of seven Korean cities (2000–

[37]For instance: Knowlton et al. referenced a meteorological analysis of the 2006 California heat wave to define the heat wave period used in their analysis while Weisskopf et al. used the regional 1999 National Weather Service heat advisory criteria [87, 252].

2007) produced similar results. When the intensity of a heat wave was defined as the temperature at or above the 97th percentile, the percent increase in mortality was 1.8 (95% CI = -7.2, 11.7) for a 2 day or greater heat wave and 3.8 (95% CI =-27.1, 47.8) for a 3 day or greater heat wave [169]. Again, when the intensity definition increased to at or above the 98th percentile the percent increase in mortality also increased (≥ 2 days: % increase = 8.4 [95% CI =0.1, 17.3]; ≥3 days: % increase 13.5 [95% CI = -0.1, 28.9]) [169].

Studies of the heat-health relationship have also noted that heat waves earlier in the season have a greater impact on health compared to those later in the season [163, 169, 255], although there is a suggestion that this may vary by location [143]. Increased morbidity/mortality earlier in the season may be due to less acclimatization of the population at the beginning of the warm season and/or a harvesting effect [141, 162].

SELECTED RESEARCH GAPS

WARM AND HUMID CLIMATES

The physiological and behavioral responses of an individual or community to hot weather vary by latitude, with populations in lower latitude areas being better adapted to a warm climate. In warm climates physiological changes (e.g., acclimatization) and behavioral responses (e.g., use of air conditioning and lighter weight clothing) allow individuals to adapt to their climate by mitigating their heat exposure. Communities in warm climates may further mitigate heat exposure in other ways such as type of housing (e.g., single level), building material, or increased green space. However, even in areas well adapted to warm weather individuals may still succumb to HRI and other heat-related outcomes, especially individuals physically exerting themselves in the heat [154, 256-258]. Additionally, even within the same latitude the climate can vary by location (Figure 2-5) [259]. Because the thermoregulatory system responds differently dependent on the type of heat (dry versus humid), the at-risk groups may also vary by climate [59]. For instance, individuals exerting themselves in the heat produce sweat to dissipate externally absorbed and internally produce heat. In dry areas this is an effective cooling mechanism, however, it is ineffective in a humid environment,

potentially leading to more cases of exertional HRI [208]. It is difficult to determine if and how the magnitude of heat-related outcomes and risk factors vary by latitude and climate as the majority of studies examining the heat-health relationship have been conducted in the northern U.S., Europe, and Australia [1, 2, 66]. Only a handful of studies globally have been conducted in areas with climates similar to the southeastern U.S., subtropical or tropical.

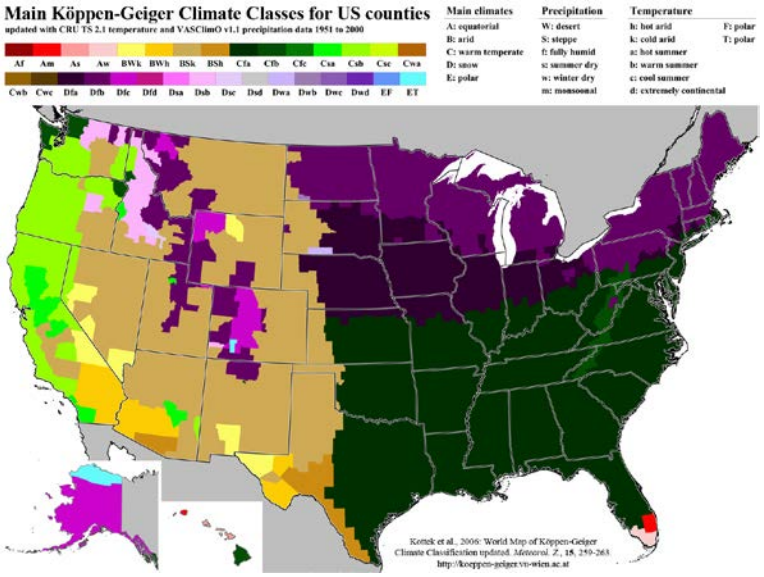


Figure 2-5. U.S. climate classifications: The majority of the southern U.S. is classified as having a warm climate with fully humid precipitation and hot summers (Cfa), although, there is a swath with warm summers (Cfb). Note that a hot summer is defined as a maximum mean monthly temperature ≥22°C (71.6°F). (Reproduced from Kottek et al. 2006 [259])

Within the U.S., heat wave studies have been conducted in four southeastern areas; Birmingham, Alabama (1976 heat wave) [260], St. Louis and Kansas City, Missouri (1980 [245, 261] and 1995 [254] heat wave), Memphis, Tennessee (1980 heat wave [262]), and Texas (1980 heat wave [221]). The percent increase in total mortality compared to prior years for these studies ranged between 30 percent and 64 percent [245, 260][38]. Increase in morbidity[39] was also observed with the excess occurring (temporally) prior to the excess in deaths [245, 260, 262]. Individuals with higher number/rates of morbidity and mortality were nonwhite, of a low socio-economic status, and individuals engaged in physical activity outdoors [221, 260-262]. Non-heat wave studies of HRI deaths [198] and ED visits [206, 263] have been conducted in North Carolina. The North Carolina ED

[38]Increased mortality was observed in all of the aforementioned heat wave studies but a simple summary number was not provided. Additionally, only heat-related deaths were examined in Texas and Tennessee.

[39]A simple summary number (e.g., percent increase) was not provided in the papers.

studies observed a linear increase in HRI ED visits with increasing mean temperature and a potential[40] threshold at 36.7°C (98°F) [206]. Further, a 22 percent (95%CI: 1.21, 1.23) increase in the rate of HRI ED visits was observed for every 0.6°C (1°F) increase in temperature over 15.6°C (60°F) [263].

Internationally studies of heat-related outcomes in subtropical and tropical climates have been done in Taiwan [200, 264], South Korea[41] [169, 223], Sao Paulo, Brazil [201], Tokyo, Japan [88, 214], Shanghai, China [167] and Brisbane, Australia [70, 249, 265]. The results from these studies are similar to results seen in other areas. In Taiwan, Brazil[42], and Australia when the entire temperature spectrum was examined in relation to mortality, a U-shaped curve was observed [201, 249, 264]. During a hot period in Sao Paulo, Brazil, researchers also observed a 2.6 percent (95% CI = 1.021, 1.028) increase in the relative risk of total mortality for every 1°C increase in mean temperature above 20°C [201]. While in Seoul, South Korea, a 2.7 percent (95% CI = 1.005, 1.051) increase in the relative risk of total mortality for every 1°C increase in mean temperature above 30°C was observed [223]. When comparing heat waves in Brisbane, Australia to non-heat wave periods, increases in total mortality (OR =1.48; 95% CI =1.23, 1.79) and cardiovascular mortality (OR=2.01; 95% CI =1.53, 2.64) were observed; in addition increases in total ED visits (OR = 1.16; 95% CI =1.08, 1.24) and renal ED visits (OR = 1.45; 95% CI = 1.12, 1.88) were also observed [70]. The number of heat stroke emergency transport cases within the Tokyo population was observed to increase with increasing temperature; although, the rate was higher among males than among females [214]. Within these international studies a lower socio-economic status and increasing age (e.g., >65) was associated with higher heat-related outcomes [70, 169, 200, 214, 223].

A few multi-city mortality [17-19, 90, 91, 135, 143] studies and one morbidity study [80] have also been conducted which also included southeastern U.S. cities. These studies found a marginal effect for heat-associated mortality in the southeastern region. Three international multi-city studies

---

[40]This is based on a graph of maximum temperature against the mean number of ED visits per day.
[41]Korea's climate has dry winters but hot and humid summers.

[42] The Brazil study used total mortality while the Taiwan study saw effects among the elderly for ischemic heart disease and cerebral infarction.

have included subtropical/tropical cities. Within the first study, 7 of 12 cities used to examine the heat-health relationship in low and middle income countries were located below a latitude of 30° N (or S) and had a subtropical or tropical climate. The study looked at both cold and hot periods and observed a U-shaped relationship between temperature and total mortality. The increase in total mortality per 1°C increase above the city specific threshold was largest in the following cities: Monterrey (Threshold = 31°C; % increase=18.8; 95%CI =13.0, 25.0), Bangkok (Threshold = 29°C; % increase=5.78; 95%CI =3.52, 8.09), Delhi (Threshold = 29°C; % increase=3.94; 95%CI =2.80, 5.08) and Sao Paulo (Threshold = 23°C; % increase=3.46; 95%CI =2.62, 4.31) [257]. A second international study examined the heat-health relationship in three Latin American cities, two of which had subtropical climates [170]. The study found that the temperature-mortality curve above the minimum mortality temperature threshold varied by location; for Sao Paulo, Brazil there was a steep linear increase above 20.5°C, for Mexico City, Mexico mortality increased but nearly plateaus as temperature increased above 12.3°C, while in Santiago, Chili[43] there was a shallow linear increase in mortality as temperature increased above 11°C [170]. Further, when comparing the 95 percentile mean temperature to the 75 percentile mean temperature the risk of mortality was highest in Sao Paulo, Brazil (% increase = 4.43; 95% CI =2.36, 6.54) and lowest in Mexico City, Mexico (% increase = 1.26; 95% CI = -0.39, 2.93) [170]. The third study observed that for three subtropical/tropical cities in East Asia, mortality increased with increasing temperature, although the shape of the curve was different for each city [159]. The largest percent increase in mortality for a 1°C increase in apparent temperature was in Taipei (% increase = 12.5; 95% CI = 6.3, 19.1) followed by Seoul (% increase = 3.4; 95% CI = 2.1, 4.6) and Tokyo (% increase = 2.0; 95% CI = 1.7, 2.4) [159].

Heat-related outcomes do occur in warm humid climates, in spite of individuals and communities being adapted to their climate. These areas also experience an increased burden of occupational HRI, increase in renal outcomes during heat waves, and increase in heat-related outcomes among males [70, 198, 214, 221, 260-262]. Further as shown above, even within warm areas, regional differences are observed. Part of this may be due to differing physiological

[43]Santiago, Chili has a Mediterranean climate to semi-arid climate with dry hot summers

adaptations (e.g., acclimatization) and behavioral modifications (i.e., adaption) employed by individuals and populations. As such, research needs to be conducted to characterize the heat-health relationship in multiple subtropical and tropical regions, including defining the exposure-response function, magnitude effect, and identification of associated risk factors.

HEAT WAVE EFFECT MODIFICATION

As previously discussed, the literature can be divided into heat wave (acute episode) analysis and continuous-temperature time-series analysis. There is very little research characterizing, simultaneously, the heat-mortality/morbidity burden observed during single days over the summer and the (potential) additional burden observed during heat waves [18, 112, 143, 166, 251, 266]. Examining these two exposures (i.e., daily temperature over time and heat waves) in conjunction may present a different picture of the heat-health relationship. For instance, Hajat et al. saw in their study of three European cities that the total burden attributable to heat waves was small (0.15-0.19% of year-long deaths) in comparison to the overall summer heat-mortality burden (0.39%-1.58% of year-long deaths) [112]. A study of 108 U.S. communities (1987-2000) also saw a small increase in mortality (2.8%) attributable to heat waves but only when a heat wave was defined as four continuous days of high temperatures [251]. Rockov et al. observed an increase in deaths associated with exposure to exposure to persistent extreme heat (i.e., heat waves) in Stockholm County, Sweden for those 45-79 years but did not observed the same association for exposure to daily high temperatures [166]. For the elderly (80+), an association was observed for heat waves and for daily high temperature [166]. In their study of 107 U.S. communities, Anderson et al. noted regional variation, with the added effect of heat waves having the smallest impact in the Southern regions [143].

Models that incorporate the joint effects of daily temperature and heat waves will assist in the understanding of the heat-mortality/morbidity relationship and further support the timing of prevention messages. For instance, the current information for heat provided by the Centers for Disease Control and Prevention (CDC) is in relation to extreme heat events only and includes checking local news stations for extreme heat alerts [267]. Additionally, many cities are implementing Heat-Health Watch Systems (HHWS) to identify when a heat wave with potentially high morbidity/mortality may occur

[107, 110]. When an *a-priori* temperature (or ambient outdoor heat metric) threshold is reached the HHWS triggers a response which varies by locale but may include activation of the Incidence Command System (i.e., emergency management response) and a coordinated response between government, business (e.g., power companies), and community-level organizations in order to prevent heat-related morbidity and mortality. Responses may include but are not limited to suspension of utility shut-offs, designated cooling centers, and public messaging. However, if the burden of disease attributed to heat waves is small than HRI prevention should occur throughout the summer and not only (or mainly) during heat waves. Furthermore, the public may need to be made aware that HRI is a possibility outside extreme heat situations and encouraged to employ mitigation strategies even when they may not traditionally perceive a danger.

Data Sources for Morbidity and Mortality

The use of multiple data sources to identify heat-related morbidity and mortality within the same time period can provide valuable information. However, within the last 30 years, the majority of studies that use multiple data sources (e.g., death certificates, hospitalizations, or ED visits) have been conducted in Australia[44] [70, 83, 134, 152, 155, 265]. To our knowledge, five sets of studies outside of Australia have used multiple sources. The first study examined ambulance call-outs and mortality during the 2003 heat wave in in Switzerland [268], while a second study looked at ED visits and hospitalizations during the 2006 California heat wave [87]. A third set of studies have examined mortality, hospitalizations, and ED visits during the 1995 Chicago heat wave [75, 153, 172]. Finally, Weisskopf et al. used mortality data and ambulance call-outs when comparing the 1999 and 1995 Milwaukee heat waves [252] while Petti et al. used mortality, hospitalizations and ED visits when identifying temperature/heat index trigger points for public health action [269].

[44]Nitschke et al.2011 = ambulance call-out/ED visits/hospitalizations/mortality [155]; Nitschke et al.2007 = ambulance call-out/hospitalizations/mortality [83]; Wang et al.2012 = Mortality and ED visits [70]; Wang et al.2009 = hospitalizations/ED visits [265]; Schaffer et al.2012 = ambulance call-outs/ED visits [152]; Ren et al.2009 = ED visits/hospitalizations/mortality [134]

Assessment of disease severity will be dependent on the data source. For instance, acute cases will be treated in the ED while the severest cases will result in death. The characteristics (e.g., age or gender) of individuals suffering from heat-related outcomes may also vary based on severity. Knowlton et al. observed that among children 0-4 years there was statistically significant increase in ED visits (RR = 1.19; 95% CI = 1.10, 1.30) when comparing the 2006 California heat wave to a reference period[45], while a similar finding for hospitalizations was not observed (RR = 1.06; 95% CI = 0.97, 1.16) [87]. The data source may also affect the distribution or type of cause-specific disease observed. As previously mentioned, the observed directional difference between circulatory morbidity and mortality may be due to individuals dying before receiving care [270]. Further, dependent on the data source, the temporal observance of disease severity (or cause-specific diseases) may vary. A rise in ED visits was observed five days prior to the rise in mortality during the 1995 Chicago heat wave [153]. A similar increase in ED visits prior to mortality was seen in the 1995 and 1999 heat wave in Milwaukee [252]. Finally, observing the differences in excess morbidity and mortality between the data sources may provide a suggestion of the effectiveness of adaption strategies implemented by a population. During the 2003 European heat wave, Cerutti et al. observed no correlation between ambulance call-outs and mortality in Switzerland, and the others theorized that ambulance service interventions prevented mortality [268]. In Adelaide, Australia, an analysis of the 2009 heat wave indicated that the greatest excess mortality was seen among those 15-64 years and the greatest morbidity (i.e., ambulance call-outs, hospitalizations, and ED visits) was among those 65 years or older [155]. The reasoning, as the authors suggest, for lower mortality among the elderly is the high level of care the elderly receive in Adelaide and the awareness of the increased risk of heat-related outcomes within this group [155]. In order to have a complete picture of the heat-health relationship and inform intervention and mitigation efforts, it is essential to define the outcome using multiple data sources and examining them within the same time frame.

[45]Heat wave = 15 July to 1 August 2006; Reference period = 8–14 July and 12–22 August 2006

CHAPTER 3. SPECIFIC AIMS

Heat-related illness (HRI) is preventable and the burden of disease will vary by geography and population according to the population's ability and capacity to adapt to heat, both physiologically and behaviorally. However, the current adaption and mitigation strategies employed in the U.S. related to HRI are based on research in northern climates. The identification of regional specific target populations, individuals most at risk of HRI morbidity and mortality, should direct the type and direction of regional prevention methods. For regional prevention, it is important to know if increasing heat is associated with increasing HRI morbidity and mortality in the specific region; and, if an association is present, are there particular groups who are more (or less) susceptible to the effects of heat? As there is currently a lack of studies examining the heat-morbidity/mortality relationship in the southeastern U.S. region, it is unclear if the answers to the above questions would be the same as that currently seen in the literature for other U.S. regions. Further, models that incorporate the effects of heat waves (i.e., intensity, duration, and seasonal timing) into heat-health models will strengthen the overall response to the afore mentioned questions by providing a more comprehensive picture of the exposure. Additionally, exploration of effect of heat waves will help guide public policy related to public health interventions in the southeast.

To address the limitations of previous work, this study will examine HRI morbidity and mortality outcomes in Florida using data from emergency department visits, hospital discharge data, and for 2005–2012. Ambient outdoor temperature and heat-index obtained from Florida weather stations will be used as the heat metric. The primary aims of the study are:

Aim 1. Model the relationship during the warm season between ambient outdoor heat and HRI morbidity and mortality in Florida.

- Determine if there is a relationship between increasing maximum daily ambient outdoor heat and HRI morbidity and mortality.
- Determine if HRI morbidity and mortality increase during a heat wave, defined as duration (contiguous days) and intensity of heat above a particular temperature threshold.
- Determine if the relationship between increasing daily ambient outdoor heat and HRI morbidity and mortality is modified by a heat wave which is defined as duration (contiguous days) and intensity of heat above a particular temperature threshold.

Hypothesis: In a hot and humid environment where individuals are well adapted to heat, both physiologically and behaviorally, there is still a positive relationship between daily ambient outdoor heat and daily HRI. Within a hot and humid environment a heat wave effect (contiguous days of high heat) is not present and the heat-health relationship is explained by modeling the relationship between average daily ambient summer heat and daily HRI morbidity and mortality within Florida zip codes.

Aim 2: Identify physical, social, adaptive, and environmental factors that may modify the relationship between daily ambient outdoor heat and HRI morbidity and mortality across Florida.

- Determine which of the variables listed below modifies the relationship between daily summer ambient heat and HRI morbidity and mortality (as defined in Aim 1): Age, gender, race, ethnicity, employment status, proportion of zip code population living alone, proportion of zip code population living in poverty, proportion of non-English speakers within a zip code, proportion of renters in community, rural/urban status of zip code, proportion of impervious surfaces within a zip code.

Hypothesis: The mechanism of an individual developing the HRI morbidity and mortality is not only determined by outdoor heat but also by how the individual and their community responds and adapts to heat (both physiologically and behaviorally). There are a combination of factors that should modify the relationship between daily summer ambient heat and HRI morbidity and mortality.

CHAPTER 4. MATERIALS

STUDY POPULATION

The population for the study is all Florida residents living in Florida within the summer months (May-October) for the years 2005 to 2012. The Florida population was estimated using data from three data sources dependent on the geographic level of analysis. For state wide analysis and county level analysis the data source was the Florida Legislature's Office of Economic and Demographic Research. For this data source, the Florida population is estimated annually using the housing unit method which is: the number of occupied housing units multiplied by the average number of persons per household plus the number of

Table 4-1. Selected characteristics of the Florida population from the American Community Survey average 5-year estimate (2007–2011)

	Population	Percent
Total Florida population	*18,688,787*	
Male	9,138,819	48.9%
Female	9,549,968	51.1%
Under 5 years	1,079,187	5.8%
5 to 9 years	1,076,647	5.8%
10 to 14 years	1,126,827	6.0%
15 to 19 years	1,228,375	6.6%
20 to 24 years	1,228,535	6.6%
25 to 34 years	2,275,039	12.2%
35 to 44 years	2,474,758	13.2%
45 to 54 years	2,702,167	14.5%
55 to 59 years	1,185,710	6.3%
60 to 64 years	1,105,089	5.9%
65 to 74 years	1,682,030	9.0%
75 to 84 years	1,095,375	5.9%
85 years and over	429,048	2.3%
White	14,270,053	76.4%
Black or African American	2,946,899	15.8%
Other	1,088,951	5.8%
Hispanic	4,122,759	22.1%
Non-Hispanic	14,566,028	77.9%
Employed (civilian labor force)*	8,258,511	89.7%
Urban area	17,139,844	91.2%
Rural area**	1,661,466	8.8%

* Number/proportion of all individuals over the age of 16 employed in the civilian labor force. Where the denominator is 15,169,949, all individuals over the age of 16.
**Data for rural/urban status was obtained from the 2010 U.S. census summary file 1. The total Florida population in 2010 was 18,801,310.

persons living in group quarters (e.g., barracks, dormitories, nursing homes, transient population). These components are estimated from various data sources including but not limited to census data, births, deaths, Medicare enrollment, residential building permits, and information from the Florida Department of Corrections and the Florida Department of Children and Families [271]. State wide estimates of the Florida work-force were obtained from the Current Population Survey (CPS). The CPS is a monthly household survey run by the U.S. Bureau of Labor and U.S. Census Bureau which collects labor force and demographic statistics [272]. The work-force will be defined as number of civilian workers age 16 years or older categorized as *employed at work* or *employed.* The final data source is the American Community Survey (ACS). The ACS is an annual survey of the U.S. population conducted by the U.S. Census Bureau [273]. The survey collects demographic, social, and economic information as well as information on housing characteristics of the population. The first year of available data is 2005. Aggregate geographic level data published by the ACS is available in groupings ranging from census blocks to the national level. However, due to data collection methodology, the type of data available depends on population size; annual estimates are available for populations of 65,000 or greater, 3-year estimates are available for populations of 20,000 or greater, and 5-year estimates are available for smaller populations. As such, the data at the census block group, census tract, or Zip Code Tabulation Areas (ZCTAs) are only available when using 5-year estimates. ZCTAs are geographic representations of zip codes. County level work-force population estimates for all Florida counties were only available from the 5-year estimates. When the geographic area of analysis was the zip code, estimates from the 2007–2011 ACS were used to represent the Florida population, as ZCTA data was only available from 2007 onwards [273]. A description of the Florida population for 2007–2011 is presented in Table 4-1.

OUTCOME ASSESSMENT

HRI events were identified from ED visits, hospitalizations, and deaths. The data for ED visits and hospitalizations was obtained from confidential administrative datasets managed by the Florida Agency for Health Care Administration (AHCA). The *Emergency Department Patient Data*

includes all ED visits in which ED registration occurs and the patient is not admitted for inpatient care at the reporting entity.

The *Hospital Inpatient Discharge Data* contains information on all individuals hospitalized in all non-federal Florida hospitals, except those individuals hospitalized in state-run facilities[46]. Florida requires by statute that data for both data sets are submitted quarterly by the required health care facilities [Florida Statute 408.061 and Chapters 59-E7 and 59-B9 of the Florida Administrative Code]. The smallest geographical level available in the confidential morbidity data is residential zip code. Due to the nature of these datasets, it is impossible to remove individuals who were treated in the ED, released, and then returned to the ED or Hospital. As such, these individuals may have multiple records for the same event.

The mortality data was obtained from death certificates maintained by the Florida Department of Health (FDOH) Office of Vital Statistics. By Florida statute, all deaths occurring within the state of Florida are registered with the department within five days of the death [Florida Statute 382.008]. The smallest geographical level available is the zip code. For individuals whose death was caused by an injury or poisoning, the zip code where the injury occurred is available in addition to the decedent's residential zip code.

All datasets were restricted to Florida residents. Within the morbidity data, residence is determined by the billing address of the individual. For the mortality data, the decedent's recorded address of residence was used. The date of HRI was defined as date of visit for the ED data, date of admission for the hospitalization data, and date of death for the mortality data. For those hospitalizations admitted through the ED the date of ED visit will be used instead of the data of hospitalization.

The main outcome for this analysis was a diagnosis indicating failure of the thermoregulatory system (i.e., HRI). Within the ED and hospitalization data, codes from the International Classification

[46]For the study period there were seven state-run hospitals that did not report to AHCA: the state Tuberculosis hospital (AG Holley), a hospital run by the Florida Department of Corrections for inmates, and five mental-health treatment facilities run by the Florida Department of Children and Families.

of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) were used to define cases (Table 4-2). All diagnosis fields (primary and secondary) and Ecode fields were used [267]. As mentioned, the literature currently does not have a consistent definition of HRI [68, 78, 82, 86, 155]. For instance, some studies use only the disease specific definition (ICD-9-CM = 992) [78, 82] while others use a combination of the disease specific definition with external cause of injury codes (Ecodes) for accidents caused by excessive heat and/or codes for dehydration [68, 86, 155]. Further, HRI is often coded in the administrative database as the condition that arises from response to the stress of the thermoregulatory process [2, 52]. For this analysis, in addition to the disease-specific definition of HRI (ICD-9-CM = 992), Ecodes were used to help identify cases that are not coded specifically with a heat disease diagnosis but the resulting outcome was due to heat as noted by the presence of E900.0 (excessive heat due to weather) or E900.9 (excessive heat of unspecified origin) (Table 4-2). Ecode 900.1, excessive heat due to man-made conditions, was also part of the definition as this code is used to identify individuals whose condition was caused by excessive heat due to being trapped in a hot vehicle [267]. Additionally, outdoor temperature is positively correlated with indoor temperature and depending on the building condition, may raise the indoor temperature of facilities with man-made heat (e.g., steel mills) further supporting the use of case identification through the code E900.1 [274].

Table 4-2. Case definitions for HRI morbidity and mortality

Description	ICD-9-CM	ICD-10
Effects of heat and light	992.0–992.9	T67.0-T67.9
Excessive heat due to weather conditions	E900.0	X30
Excessive heat due to man-made conditions	E900.1	W92
Excessive heat of unspecified origin	E900.9	---

For mortality data, codes from the International Classification of Diseases, Tenth Revision (ICD-10) were used to define cases [275]. Both the underlying and all contributing causes of death fields were used. HRI-death was defined in a similar manner to HRI-morbidity (Table 4-2).

As previously mentioned, exposure to environmental heat is a recognized occupational hazard [182]. However, there is currently very little research related to the epidemiology of

occupational heat-related outcomes. The heat-health relationship and associated risk factors may vary by occupational status. To provide some elucidation into this area, this study examined separately work-related and non-work-related HRI. Work-related morbidity cases were restricted to individuals age 16 years or older where the expected principal payer was workers' compensation or a work-related ICD-9-CM Ecode was present (Table 4-3) [276, 277]. A work-related death was restricted to individuals' age 16 years or older where the death certificate indicated the injury occurred at work.

Table 4-3. Codes used to define a work-relatedness within the morbidity data

ICD-9-CM	Definition
---	Principal payer is workers' compensation
E000.0	Civilian activity done for income or pay
E000.1	Military activity
E800-E807	Railway accident among railway employee (4th digit = 0)
E830-E838	Water transport accident among crew, Dockers and stevedores (4th digit = 2 or 6)
E840-E845	Air and space transport accidents among crew and ground crew (4th digit = 2 or 8)
E846	Accidents involving powered vehicles used solely within the buildings and premises of industrial or commercial establishment
E849.1	Place of occurrence: farm building/land under cultivation
E849.2	Place of occurrence: mine or quarry
E849.3	Place of occurrence: industrial place and premises

DISCLAIMER

Note that the conclusions presented in dissertation are the authors own and do not necessarily reflect the opinions of the FDOH or the Florida AHCA.

EXPOSURE ASSESSMENT

All exposure metrics for the analysis are based on daily ambient outdoor temperature (i.e., dry bulb temperature) and daily heat index. Ambient outdoor temperature or a calculated measure of temperature and humidity (i.e., heat index) were used to estimate outdoor heat. Within Florida, the heat index is currently used by Florida Weather Forecasting Offices to alert the Florida population to dangerous heat conditions [278, 279].

The state of Florida is divided into seven National Weather Service (NWS) regions, one for each of the weather forecasting offices (WFOs). Each WFO issues weather related warnings and alerts to citizens within their jurisdiction (Figure 4-1).

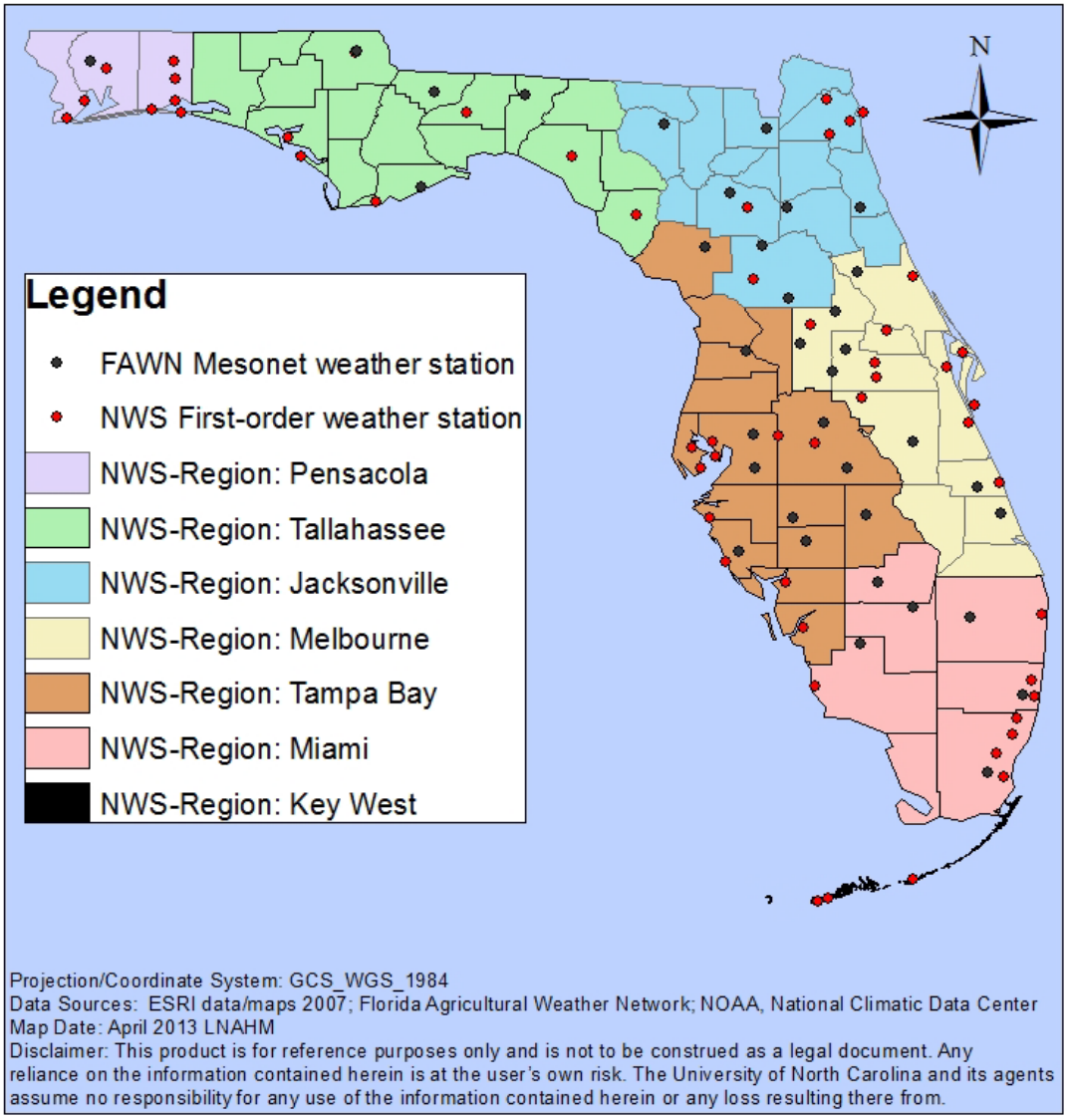


Figure 4-1. National Weather Service regions and weather station locations**.** Florida is divided into seven National Weather Service regions with the Key West region only covering a small land area.

Weather data were obtained from the 56 National Center for Environmental Services Land-Based first-order stations and the 36 Florida Agricultural Weather Network stations (i.e., Mesonet sites [FAWN]) (Figure 4-1). The first-order stations include the Automated Weather Observation System (AWOS) and the Automated Surface Observing System (ASOS), which are maintained by the

Federal Aviation Administration, the NWS, and the U.S. Department of Defense. The exposure variables were linked to the morbidity data by assigning a weather station to each zip code within Florida. Using ArcGIS 10.3.1, the coordinates for each weather station were mapped and the Euclidian distance between each weather station and each zip code centroid was calculated. The zip code was attached to the nearest weather station (i.e., smallest Euclidian distance). This was an iterative process; as weather stations became available (e.g., come on-line) or unavailable during the study period, the nearest station to each zip code was recalculated.

Equation 4-1. Water Vapor Content

$$E = (6.11) \times \left(10^{\left(7.5 \times dewpoint / 237.7 + dewpoint\right)}\right)$$

where dewpoint is in Celsius

Equation 4-2. Water Vapor Capacity

$$ES = (6.11) \times \left(10^{\left(7.5 \times temp\_C / 237.7 + temp\_C\right)}\right)$$

where temp_C = dry bulb temperature in Celsius

Equation 4-3. Relative Humidity

$$RH = \frac{\text{water vapor content}}{\text{water vapor capacity}} = \frac{E}{ES} \times 100\%$$

Equation 4-4. Heat Index

Where T = temperature in Fahrenheit and T ≥ 80°F:

$$HI = -42.379 + 2.04901523(T) + 10.14333127(RH) - 0.22475541(T)(RH) - \left(6.83783 \times (10^{-3})\right)(T^2) - \left(5.481717 \times (10^{-2})\right)(RH^2) + \left(1.22874 \times (10^{-3})\right)(T^2)(RH) + \left(8.5282 \times (10^{-4})\right)(T)(RH^2) - \left(1.99 \times (10^{-6})\right)(T^2)(RH^2) - K$$

where K

$$= \begin{cases} \left(\frac{13 - RH}{4}\right) \times \sqrt{\frac{17 - |T - 95|}{17}}, & RH \leq 13\% \; and \; 80 \leq T \leq 112 \\ \left(\frac{RH - 85}{10}\right) \times \left(\frac{87 - T}{5}\right), & RH > 85\% \; and \; 80 \leq T \leq 87 \\ 0, & 13\% < RH \leq 85\% \; and \; T \geq 80 \\ 0, & RH \geq 85\% \; and \; T \geq 87 \end{cases}$$

Where $T < 80$°F: $HI = -10.3 + 1.1(T) + 0.047(RH)$

Hourly weather data were obtained from each of the stations for 2005–2012. For the NWS first order stations, standard observations are taken once per hour. For some stations, observations are only recorded when an individual is

present, resulting in 8 readings per day (standard work shift) instead of 24. The FAWN stations provide readings every 15 minutes. The hourly data provided by FAWN is the hourly average of the 15 minute data. Relative humidity is required to calculate heat index; however, this variable is not collected by the NWS first order stations. Instead, hourly dew point and temperature (in Celsius) data were collected from the NWS first-order weather stations and were used to calculate relative humidity (Equation 4-1, Equation 4-2, and Equation 4-3). Hourly data for relative humidity and temperature (in Celsius) were obtained for each of the FAWN stations. For analysis, temperature data was converted to Fahrenheit. Heat index was calculated using relative humidity and temperature in accordance with NWS methodology (Equation 4-4) [118, 120, 124]. As mentioned, some of the stations included in the analysis only recorded readings during working hours (approximately 8:00-17:00) and therefore, the daily average or minimum temperature readings from these stations were overestimated. In order to decrease potential exposure misclassification maximum daily temperature and maximum daily heat index were used in order to increase the number of stations included in the analysis. The highest hourly value of temperature or heat index by station within a 24-hour period (00:00–23:59) was identified as the maximum daily temperature or heat index value used for this analysis.

The 95th and 99th percentiles for maximum daily temperature across the state for May–September were obtained from the Florida Climate Center. The Florida Climate Center[47] created contour maps for each percentile based on the NWS Cooperative Observer Program (COOP) using all available data years (Figure 4-2). Each zip code within Florida was linked using ArcGIS 10.3.1 to a temperature contour indicating the value of the 95th or the 99th percentile. For zip codes which had a geographic area in multiple contours, the contour with the largest portion of the geographic area was used. Humidity information is not collected at the COOP stations; therefore, the 95th and 99th percentile for maximum daily heat index for May–September was calculated, by the Florida Climate Center[47], for each NWS first order station and FAWN station, dependent of the data years available for each station. The range of values for the 95th percentile and 99th percentile of maximum daily heat

[47]The temperature contour maps and 95th/99th percentile for heat index were created/calculated by the assistant state climatologist, Melissa Griffin.

index was 101.0°F – 109.0°F (mean = 105.3°F; standard deviation [sd] = 1.80°F) and 101.3°F – 114.7°F (mean = 109.5°F; sd = 2.99°F), respectively.

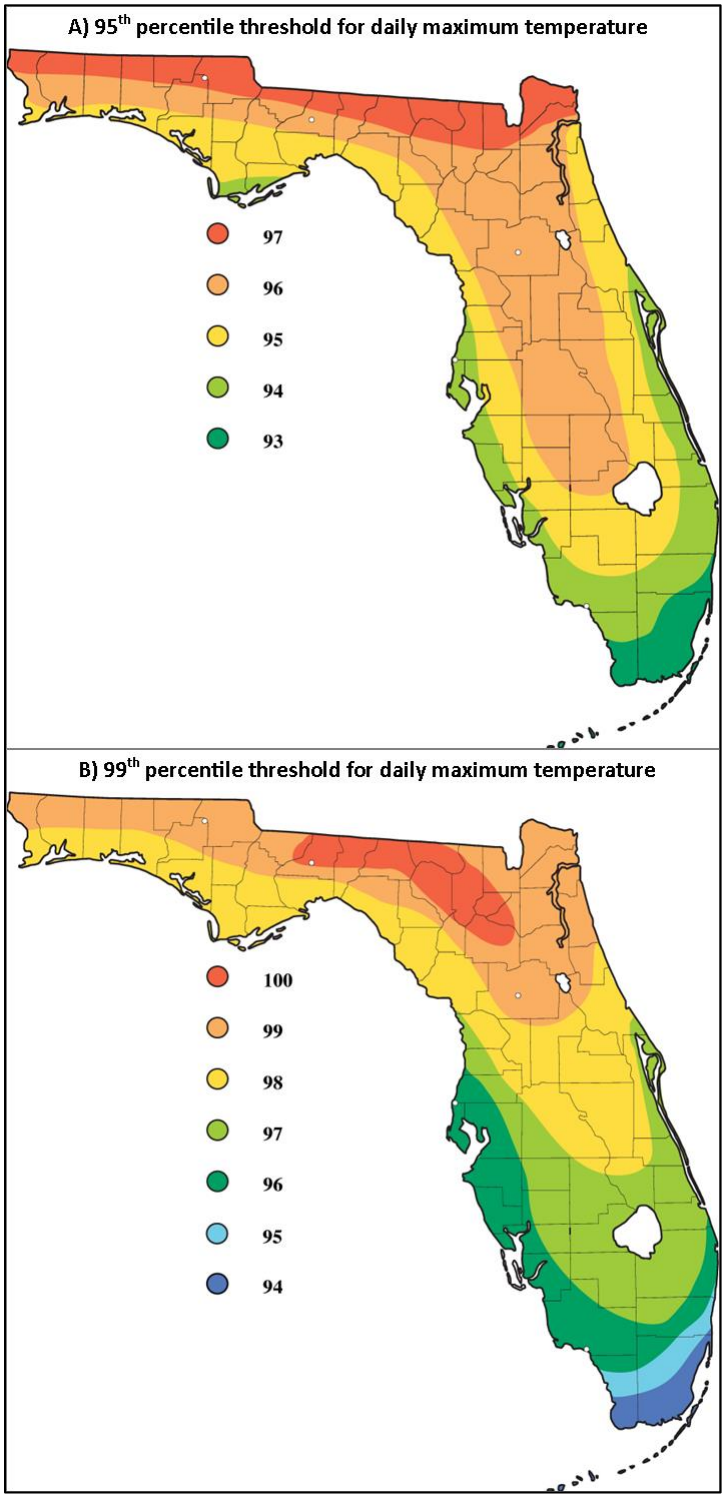


Figure 4-2. Maximum daily temperature threshold value contour maps for Florida. The colors on the map represent the threshold temperature in Fahrenheit within a contour area.

CHAPTER 5. A COMPREHENSIVE EVALUATION OF THE BURDEN OF HEAT-RELATED ILLNESS WITHIN THE FLORIDA POPULATION

INTRODUCTION

The term heat-related illness (HRI) captures a continuum of disorders that occur as the human body absorbs and creates more heat than can be dissipated [48, 49, 59]. As HRI progresses, single and multi-system failure occurs [52]. Timely medical intervention can prevent mild cases of HRI, such as heat edema, from becoming severe (i.e., heat stroke) and potentially resulting in death. However, even with medical intervention, severe HRI may have lasting effects, including neurological and organ damage and decreased heat tolerance, making an individual more susceptible to another HRI event [228-230]. Therefore, prevention of HRI is essential. Fortunately, HRI is highly preventable through individual- and community-level behavioral and structural modifications. To understand where and potentially which type of behavioral and structural modifications may have the largest effects on HRI prevention, characterization of HRI within the population and identification of groups with the highest (and lowest) burden is necessary.

The heat-health relationship (i.e., heat-related outcomes and ambient outdoor heat) varies by latitude with those in the lower latitudes being better adapted to heat through behavioral and structural modification than those in higher latitudes. Even in areas well adapted to warm weather, individuals still succumb to HRI, especially individuals physically exerting themselves in the heat [154, 256-258]. It is difficult to determine how HRI susceptibility factors previously identified in the literature vary by latitude and climate, as the majority of studies examining the heat-health relationship have been conducted in the northern U.S., Europe, and Australia [1, 2, 7, 9, 38, 42, 66, 203, 215]. Only a handful of studies globally have been conducted in tropical or humid subtropical areas such as the southeastern U.S. The tropical or humid subtropical climate may have an impact on the distribution of risk factors within the population. For instance, in a tropical or humid subtropical climate, sweating is

an ineffective cooling mechanism potentially leading to an increased number of individuals working or playing outdoors succumbing to HRI compared with a dry or temperate climate [59, 208].

The purpose of this paper is to describe the burden of severe HRI morbidity and mortality among Florida residents during the warm season. The results of these analyses identify populations appropriate for future investigations into and targeted interventions. In addition, this analysis provides a baseline for future evaluations of interventions and for evaluating changes in the HRI burden over time. Finally, the results may be applicable to other areas of the southeastern U.S, and may provide a framework for exploring the HRI burden within other jurisdictions.

METHODS

DATA SOURCES

The analysis was restricted, unless otherwise mentioned, to Florida residents between May-October (2005–2012) [280]. Emergency department (ED) and hospital discharge data were obtained from the Florida Agency for Health Care Administration. The ED dataset contained those treated and released, while the hospital discharge dataset contained all admissions regardless of source. In order to estimate HRI onset, where available, the ED visit date was used [281]. Death certificate data were obtained from the Florida Department of Health (FDOH), Office of Vital Statistics. Again, in order to estimate the HRI onset, where available, both the date of injury and the date of death were analyzed [281].

The three datasets were not able to be linked and therefore had potentially overlapping cases. The overlap between the morbidity and mortality data was estimated via a variable in the morbidity data indicating if the patient died and a variable on the death certificated indicating if the death occurred in a hospital. Within the morbidity data, patients were included in both the ED and hospital discharge datasets if a patient was seen in the ED and transferred to another hospital (as opposed to the hospital connected to the ED). These transfer patients were identified via an ED discharge status indicating transfer to one of the following facilities: a short term general hospital for

inpatient care, children's hospital, cancer center, Medicare certified long-term care hospital, psychiatric hospital or distinct unit, or critical access hospital.

In this analysis, individuals who sought treatment, were released, and subsequently again sought treatment for the same event or who died would be counted twice within a single dataset (e.g., ED) or counted in multiple datasets. Unfortunately, it was not possible to identify these individuals. However, due to the disease course for HRI and effectiveness of treatment, it is assumed that these duplicate cases will be negligible.

DEFINING HRI

HRI was defined as the presence of an International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM, morbidity) or Tenth Revision (ICD-10, mortality) code for the effects of heat and light (992.0–992.9/T67.0–T67.9) or an excessive heat external cause of injury (Ecode) (E900.0/X30, E900.1/W92, E900.9) [156, 277, 282]. All diagnosis/Ecode fields and underlying/contributing cause of death fields were used.

HRI cases were also stratified by work-relatedness. Workers may be at high risk for HRI, and susceptibility factors within this group may differ from the general population; however, characterization of HRI in this population is sparse [10]. In the morbidity datasets, work-relatedness was defined as expected payer equals workers' compensation or the presence of the following ICD-9-CM Ecodes indicating that the injury occurred at work or a probable work-location: E000.0, E000.1, E800–E807 (4th digit = 0), E830–E838 (4th digit = 2 or 6), E840–E845 (4th digit = 2 or 8), E846, E849.1–E849.3 (Table 4-3) [276, 277]. For mortality, if the death certificate variable *injury at work* was marked yes, than the death was considered work-related. Work-relatedness was restricted to those aged 16 years or older. All HRI cases not classified as work-related were classified as non-work-related.

VARIABLE SELECTION

The following presents the parameterization used in this analysis for the individual-level factors identified based on a literature search.

Gender is associated with HRI morbidity and mortality, with men having a higher morbidity than women and the converse for mortality [1, 88, 89, 161, 169, 205, 212, 214]. Gender was available in all datasets and modeled as binary (male/female).

Race and ethnicity have been observed to modify HRI rates. The differences are non-biological and suspected to be related to other factors [7, 9, 41, 87, 171, 198, 283]. In this study, race and Hispanic ethnicity will be examined in part to inform future analyses which may include access to additional information related to factors such as socio-economic status (SES), access to resources, or racial/ethnic distribution within outdoor occupations. Race was categorized as White, Black or African American, and other, while ethnicity was categorized as Hispanic or non-Hispanic.

The elderly [53, 160, 238, 284] and very young (< 5 years) [1, 2] have been shown to have a high risk of HRI. Teenagers and young adults may also be at increased risk of HRI due to participation in athletics or high-risk occupational activities [147, 205, 215]. Age at time of visit, admission, or death was calculated by the data custodians and included in each dataset. For this analysis, age was grouped into 5-year age categories (0-4, 5-9, … 30-34, 35-44, … 75-79, 80-84, 85+).

Rural and urban populations may have different heat exposure and may react or adapt to heat differently because of differences in available resources (e.g., medical care, air conditioning usage, or outdoor activities) [196, 200, 202, 246]. For each dataset, county of residence was used to determine rural or urban status. Rural and urban counties were defined in accordance with the definition provided by the FDOH Office of Rural Health based on the 2000 census [285].

An individual's SES may affect their ability to prevent an HRI outcome (e.g., lack of air conditioning or poor housing conditions) or reduce the severity of the outcome (e.g., access to

medical care). Within the morbidity data, low SES was assessed using the potential payer field. Those records which had Medicaid, Kidcare[48], or self pay/charity listed as the payer were classified as low SES (i.e., socially disadvantaged). A variable for assessing SES was not available in the mortality data. Note that the SES analysis was not stratified by work-related status, as the morbidity data only has a single payer code and part of the work-related definition is payer equal to worker's compensation. Instead, all analysis related to SES was based on all HRI ED visits or hospitalizations.

As the thermoregulatory system fails, it affects multiple organs and systems throughout the body [52, 63, 64]. Further, individuals with chronic diseases or prior conditions are at higher risk of adverse HRI effects due to the increased stress caused by the thermoregulation process on already impaired organs and systems. For this analysis, among HRI cases, additional outcomes with consistent evidence of a positive relationship with heat and a hypothesized biological mechanism [1, 48, 52, 63, 66-68, 76, 93, 94, 96, 97] were examined: cardiovascular disease (ICD-9-CM codes: 390–429, 440–448; ICD-10 codes: I00–I51, I70–I79), cerebrovascular disease (ICD-9-CM codes: 430–438; ICD-10 codes: I60-I69), respiratory disease (ICD-9-CM codes: 460–519; ICD-10 codes: J00–J99), renal disease (ICD-9-CM codes: 580-589; ICD-10 codes: N00–N07, N17–N19, N25–N27), diabetes mellitus (ICD-9-CM code: 250; ICD-10 codes: E10–E11, E13–E14) and injuries (ICD-9-CM codes: 800–904 and 910–950; ICD-10 codes: S00–T35). A direct biological mechanism is not present for injuries, however, the symptoms of HRI (e.g., neurological impairment such as dizziness) may lead to injury (e.g., a worker falls from scaffolding) [1, 18, 182, 274, 277, 286].

Part of the HRI prevention message is an awareness of the signs and symptoms of HRI so that individuals may seek immediate treatment to prevent more severe outcomes [287, 288]. Length of stay (LOS) within the hospital is a simple and easily calculable measurement that can, to some extent, represent severity of outcome and effectiveness of treatment (i.e., shorter stay equals less severe outcome or more effective treatment). LOS was calculated in this study to provide a baseline estimate of HRI to aid in the understanding of the effectiveness of HRI awareness messaging. LOS was the difference between the date of hospital discharge and the date of hospital admission or the

[48]Health insurance for low-income children age 0-18 provided by the state of Florida

date of ED visit for those admitted through the ED. For decedents that were admitted to the hospital prior to death, LOS was calculated as the date of death minus the date of injury.

ANALYSIS

Crude and, where appropriate, age-adjusted rates and standard errors (SE) were calculated for Florida overall and stratified by county or by the susceptibility factors of interest. The numerator was HRI ED visits, hospitalizations, or deaths. The denominator for the Florida population and civilian workforce was obtained from the Florida Legislature's Office of Economic and Demographic Research and the Current Population Survey (CPS), respectively. The civilian workforce was enumerated as the number of civilian workers aged 16 years or older categorized as *employed at work* or *employed absent.* CPS county-specific estimates of the workforce were not available; therefore, for work-related county-specific analyses, the American Community Survey (ACS) 5-year estimates (2005-2009 and 2008-2012) were used. A limitation of using the 5-year ACS estimates is the assumption of a stable population. As the Florida population has increased over time, the 2008-2012 ACS was used for the years 2010-2012. Each yearly estimate was divided by two to produce the person-time at risk for May-October. Age-adjusted rates were standardized to the 2000 U.S. standard population using direct standardization. The crude rate ratios (RR) and corresponding 95 percent confidence intervals (CI) were calculated to compare rates of HRI by the aforementioned susceptibility factors (e.g., crude HRI rate in males divided by crude HRI rate in the referent group, females). Calculated measures of variance (e.g., 95% CI or standard error [SE]) were used to indicate the stability of the rate or RR estimate.

The death certificate contains text fields with information on how the death occurred and the usual occupation of the decedent. The former is completed by the medical certifier and required for all deaths with an injury (e.g., HRI) or poisoning, while the latter is completed by the funeral director. These fields provided information related to the specific activity or situation of the decedent that contributed to the HRI death. These fields were reviewed and manually summarized (i.e., codes created) into categories.

RESULTS

HRI SEASONALITY

Using annual data from the entire study period (2005–2012), among Florida residents, the majority of non-work-related HRI ED visits (83.9%; n = 22,669), hospitalizations (86.1%; n = 4582), and deaths (85.4%; n = 135) were observed between May and September (Figure A-1). The largest proportion of non-work-related ED visits (23.6%; n = 6377) and hospitalizations (25.1%; n = 1338) occurred in August while the largest proportion of non-work-related deaths, 26.6 percent (n = 42), occurred in July. A similar, although slightly higher, distribution was observed for work-related HRI with 87.8 (n= 2,838), 91.2 (n = 394), and 92.0 (n = 23) percent of ED visits, hospitalizations, and deaths occurring between May and September (Figure A-2). The largest proportion of work-related HRI morbidity and mortality occurred in August (ED visits: n = 896, 27.7%; hospitalizations: n = 130, 30.1%; deaths: n = 7, 28.0%).

HRI OCCURRENCE

Among Florida residents, during the Florida warm season (May-October) for 2005–2012, there were 23,981 non-work-related HRI cases treated in the ED, 4816 HRI hospitalizations, and 139 HRI deaths. These cases accounted for 0.10 percent of all-cause warm season non-work-related ED visits, 0.05 percent of non-work-related hospitalizations, and 0.02 percent of non-work-related deaths. Among work-related HRI cases, there were 2979 cases treated in the ED, 415 hospitalizations, and 23 deaths. The work-related HRI cases accounted for 0.66, 0.98, and 2.3 percent of all-cause work-related ED visits, hospitalizations, and deaths during the warm season.

EMERGENCY DEPARTMENT VISITS

During the warm season of eight-year study period, the age-adjusted rate for non-work related HRI ED visits among Florida residents was 33.11 visits per 100,000 person-years (SE = 2.17), while the age-adjusted rate for work-related HRI ED visits was 8.46 visits per 100,000 worker-years (SE = 1.40). Among those treated in the ED but not admitted to the hospital, there were 15 non-work

related HRI deaths and 1 work-related HRI death. The highest rates of HRI ED visits were found in the Florida panhandle (Figure 5-1).

Demographic non-work-related and work-related HRI ED visit characteristics can be found in Table 5-1 and Table 5-2, respectively. The rate of HRI ED visits was higher among males than females (non-work-related: RR= 2.77; work-related: RR =5.91), for minorities (Blacks and others) compared to Whites (non-work-related: Blacks RR = 1.43, others RR = 1.14; work-related: Blacks RR = 1.18, others RR = 1.27), and for rural areas versus non-rural areas (non-work-related: RR = 1.62; work-related: RR = 2.51). Rates were lower for Hispanics compared with non-Hispanics (non-work-related: RR = 0.46; work-related: RR = 0.56). The rate of non-work-related HRI ED visits by age group was highest for those aged 15–19 years (60.41 per 100,000 person-years) and decreased as age increased (Figure 5-2a). The lowest non-work-related HRI ED rate is among children less than 10 years of age (9.83 per 100,000 person-years). Work-related HRI ED rates were highest among those under the age of 35 years (12.46 per 100,000 worker-years) and lowest for those aged 70 years or older (2.69 per 100,000 worker-years) (Figure 5-2b).

Among non-work-related HRI ED visits, 28.3% (n=6789) had a code for one or more of the selected co-morbid conditions; while for work-related cases, 24.8% had a co-morbid code (n = 740) (Figure A-3). The most frequent co-morbid codes for non-work-related HRI cases were cardiovascular disease (67.2%; n = 4565), diabetes (19.4%; n = 1319), and respiratory disease (19.2%; n = 1302). While for work-related HRI, the most frequent co-morbid codes were cardiovascular disease (64.7; n = 479), renal outcomes (16.5; n=122), and injuries (14.9%; n= 110).

Forty-four percent (n = 11,981) of all HRI ED cases were identified as low SES according to the payer codes, while 54.4 percent (n= 12,847,894) of total all-cause ED visits (n = 23,601,926) were identified as low SES.

HOSPITALIZATIONS

During the study period, the age-adjusted rates for non-work-related and work-related HRI hospitalizations were 5.88 hospitalizations per 100,000 person-years (SE = 0.87) and 1.12 hospitalizations per 100,000 worker-years (SE = 0.51), respectively. During the study period, there was an average of 14.6 non-work-related HRI ED visits per year (n =117) where the patient was transferred to a hospital not connected to the visited ED and potentially counted in both morbidity datasets. Additionally, there were less than five work-related HRI cases where the patient was transferred to a hospital not connected to the visited ED. Fifty-six non-work-related deaths and 3 work-related deaths were identified among those hospitalized with HRI. The counties with the highest rates of non-work-related and work-related HRI hospitalizations were scattered across the northern and middle portion of the state (Figure 5-1).The mean LOS for non-work-related hospitalizations was 3.1 days (SE = 0.07; Median = 2; Range = 0–116), while the mean LOS for work-related hospitalizations was slightly lower at 2.5 days (SE = 0.19; Median = 2; Range = 0–53).

The demographic patterns for hospitalizations were the same as ED visits except for the age-group distribution (Table 5-1 and Table 5-2). Hospitalizations for non-work-related HRI increased as age increased, with the lowest rate for ages 0–14 (0.65 per 100,000 person-years) and the highest rate for those 75 years or older (14.17 per 100,000 person-years) (Figure 5-2a). The highest rates of work-related HRI were for those aged 45 to 54 years (1.46 per 100,000 worker-years) and lowest for those 70 years of age or older (0.38 per 100,000 worker-years) (Figure 5-2b).

The proportion of HRI hospitalizations with a code for one or more of the selected co-morbid conditions was slightly higher for non-work-related (80.8%; n=3893) and work-related cases (72.2%; n = 302) (Figure A-4). For both non-work-related and work-related HRI hospitalizations, the most frequent co-morbid codes were for cardiovascular disease (non-work-related: 69.1%, n= 2691; work-related: 53.5%, n = 160) and renal outcomes (non-work-related: 44.5%, n = 1693; work-related: 64.9% n=196).

The indicator of low SES was recorded in 34 percent (n = 1754) of all HRI hospitalizations and 28.6 percent (n = 2,836,156) of total all-cause hospitalizations (n = 9,907,258).

DEATHS

The age-adjusted rate of HRI death during the warm season of the eight-year study period was 0.17 per 100,000 person-years (SE = 0.15) for non-work-related HRI and 0.06 per 100,000 worker-years (SE = 0.11) for work-related HRI. According to the death certificate, 60 percent (n: non-work-related = 80; work-related = 17) of all individuals who died of (or with) HRI were taken to the hospital or ED prior to death. Among the 36 decedents admitted to the hospital, as recorded in the death certificate, prior to death with an available date of HRI onset, the average LOS was 3.6 days for non-work-related HRI deaths (n = 29; SE = 0.99, Median= 1, Range = 0–22) and 9.3 for work-related HRI deaths (n = 6; SE = 6.08; Median = 3.5; Range = 0–39). The number of deaths per county was too small to provide stable county-specific rates.

Table 5-1 contains demographic characteristics for non-work-related HRI deaths. The results were similar to the non-work-related morbidity results except for age. The rate of non-work-related deaths was highest for the very young (age <5: Rate = 0.55/100,000 person-years) and the elderly (age 75+: Rate = 0.39/100,000 person-years), followed by those aged 45–54 (Rate = 0.26/100,000 person-years) (Figure 5-2a).

The demographic characteristics for work-related HRI deaths can be found in Table 5-2. Unlike work-related HRI morbidity, the rate of work-related HRI deaths was higher among Hispanics than among non-Hispanics (RR = 2.35; 95% CI = 1.02, 5.43). Age-specific rates were highest for workers aged 30-34 years (0.18 per 100,000 worker-years) and 55-59 years (0.15 per 100,000 worker-years) (Figure 5-2b). Among decedents with work-related HRI, only half (n = 11) of the death certificates had the usual industry/occupation listed, of which all were outdoor workers.

Among non-work-related and work-related HRI deaths, 33.7 (n = 55) and 2.5 (n = 4) percent of deaths, respectively, had a code for one or more of the selected co-morbid conditions. The most

common condition was cardiovascular disease (non-work-related: 81.8%, n = 45; work-related = 100%, n = 4).

There were 137 (84.6% [n: non-work-related = 114; work-related = 23]) death certificate records which contained text information on how the death occurred. Among those HRI deaths with situational information, 20.4 percent (n: non-work-related = 13; work-related = 15) of the notes explicitly mentioned the decedent exerting themselves in hot weather, 10.9 percent (n: non-work-related = 14; work-related = 1) were related to alcohol intoxication or illicit drug use, and 27.7 percent (n: non-work-related = 37; work-related = 1) were due to being trapped in a car. For those died trapped in a car, 57.9 percent (n =22) were under the age of 5 years.

DISCUSSION

This paper provides an overview of the burden of HRI resulting in a death or provision of medical services in an ED or hospital among Florida residents. This is one of a small number of studies that have looked at the burden of HRI and the first study within the southeastern U.S. to use multiple data sources (i.e., ED, hospitalization, and death certificates) for this evaluation [10, 12, 148, 187, 195, 198, 205, 206, 277, 282, 289-291]. Most studies have examined risk factors for heat-related outcomes (which include but are not limited to HRI, all-cause morbidity/mortality, cardiovascular disease, or respiratory disease) in relation to high temperatures or heat-waves [1, 2, 9, 66]. However, HRI can occur outside heat waves and extreme temperatures. Using multiple data sources provides a more complete understanding of the HRI burden, as well as, a better baseline for assessing changes in the HRI distribution within the population.

HRI burden differed geographically throughout the state. The lowest rates of HRI morbidity were observed in southern Florida counties, while the highest rates of HRI morbidity were observed in counties in the panhandle, or in the northern part of the state. Average summer temperatures are higher and more variable in the northern part of the state compared to average summer temperatures in the southern part of the state [292]. High rates of work-related HRI were also observed in south central Florida, an area with a large proportion of citrus agricultural, which is labor intensive [293,

294]. The highest HRI morbidity counts were found in counties with urban centers: Broward, Hillsborough, Orange, Palm Beach, Pinellas, and Miami-Dade (Figure A-5). Counties with the highest rates of HRI morbidity differed by data source, while counties with the highest counts did not.

Within this study, higher HRI morbidity and mortality rates were also observed for rural compared to urban counties. This result has been observed in prior studies [105, 282] . Rural-urban differences in the burden of HRI may be explained by differences in the distribution of factors such as disparities in socio-economic status or access to health care, greater (or different) occupational risk factors (e.g., agricultural work), or differences in the proportion and type of impervious surfaces (e.g., higher humidity due to more vegetation in rural agricultural areas) [105, 197, 200, 202, 246]. For instance, a study of HRI ED visits in North Carolina (2007-2012) found that for rural zip codes, HRI decreased as the percentage of impervious surfaces and developed land increased [295]. However, in urban areas, impervious surfaces absorb heat contributing to an urban heat island effect and were associated with increased morbidity and mortality [236]. Further, a study by Hajat et al. classified rural/urban census output areas[49] in England and Wales and found that rural areas with the highest level of social deprivation (i.e., lowest SES) had the highest relative risk of death[50] [226].

The distribution of rates of HRI by age group differed for ED visits (treated and released), hospitalizations, and deaths. Within the ED data, the highest rates of HRI were among teenagers and young adults, regardless of work-related status. The highest hospitalization rates for non-work-related HRI were among the elderly. The highest HRI death rates were for the elderly and those under age five. This non-work-related HRI age-group distribution is similar to what has been shown in studies that have included age at a finer resolution than 15–64 years (morbidity [205, 206, 282, 290, 291]; mortality [12, 84, 136, 207]). Both the elderly and very young children have a decreased ability to thermoregulate due to biologic effects (e.g., aging or underdevelopment) and decreased ability (or inability) to employ behavioral modifications [1, 2, 53, 160, 238, 284]. Further, the prevalence of co-

[49]The census output areas have an average population size of 300 and are the smallest geographical unit used in the 2001 United Kingdom census.

[50]The variability in the rural results is greater than in the urban results (tight confidence intervals) according to the figures presented in the paper.

morbid conditions, which put individuals at increased risk of HRI, increases with age. In this analysis, comparing HRI ED visits and hospitalizations, the proportion of selected co-morbid conditions among HRI hospitalizations was greater than among HRI ED visits.

Heat stroke can be divided into two categories: classical and exertional. Classical heat stroke typically develops over days and occurs in hot environments among those whose thermoregulatory system is compromised [48, 55, 59]. Exertional heat stroke, on the other hand, can develop within hours often in young, healthy individuals under conditions of strenuous activity, usually in hot humid weather [48, 59]. Those with compromised thermoregulatory systems are also susceptible when involved in strenuous activities. The data used for the present analysis do not include variables to identify classical and exertional heat stroke. Although it was not possible to distinguish between the two heat stroke subtypes, 43.5 and 64.9 percent of all non-work-related and work-related HRI hospitalizations, respectively, had a renal diagnosis. Acute renal failure is a common symptom of exertional heat stroke [48, 52, 55, 59]. The higher proportion of cases with a renal outcome among work-related HRI hospitalizations may be related to individuals exerting themselves in the heat as part of their job duties. Further, of those HRI deaths with a situational text note on the death certificate, 11.4 and 65.2 percent of non-work-related and work-related deaths, respectively, explicitly indicated exertion during hot weather. A potentially higher proportion of exertional heat stroke in the Florida population (versus classical heat stroke) may have implications for how HRI prevention is implemented, as well as how HRI is modeled when examining the heat-health relationship or estimating projections of future burden. For instance, it may be more effective to target schools, athletic events, or outdoor workers for prevention programs as opposed to targeting the elderly or isolated individuals. It may also be more appropriate to model heat as a continuous exposure over time as opposed to binary, heat wave versus non-heat waves.

Among Florida residents, men have higher rates of both HRI morbidity and mortality than women, regardless of work-related status. For morbidity, this result reflects what has been observed in the literature [88, 89, 205, 214]. Researchers have suggested that this difference may reflect the activities men (versus women) are involved in [205]. For instance, Kerr and colleagues used the

National High School Sports–Related Injury Surveillance System data (2005/2006–2010/2011) to examine HRI among high school athletes [215]. They found that the majority of HRI (87.7%) occurred among boys; however, when football players were removed, boys accounted for 50.9% of events [215]. Occupationally speaking, males are more likely to work in jobs with greater risk of HRI, such as construction or fire protection [147, 217]. This may also explain the larger gender-specific relative difference observed for work-related compared to non-work-related HRI. The majority of the literature examining heat-related mortality and gender found that either mortality was greater for women than for men [1, 161, 169, 212] or no association [67, 218]. Men and women have different underlying HRI risk profiles. For instance, unmarried elderly men tend to be at higher risk of HRI due to social isolation [7, 238]. Women have a higher proportion of chronic conditions than men due to living longer, and this places women at higher risk of HRI than men. The distribution of these underlying HRI risk profiles within each population will impact whether gender differences in HRI rates are observed or not observed [7, 9]. Additionally, the gender differences observed in the literature may be a reflection of the outcome used [218, 296]. Many studies use all-cause mortality or a cause-specific outcome (e.g., all cardiovascular or all respiratory disease) when examining the heat-health relationship [1]. However, in the few studies that looked specifically at HRI mortality, men had a higher risk of HRI than women [136, 198, 289, 297]. Further, an analysis of heat-related mortality among U.S. workers (2000-2010) also saw much higher rates of HRI among male workers than among female workers [10].

Full understanding of the racial and ethnic differences observed within Florida requires further exploration. The lower rate of non-work-related HRI morbidity and mortality among Hispanics compared with non-Hispanic Whites may be related to the unique distribution of Hispanic origin groups within the state. As such, this lower rate observed in this study may not be reflective of the HRI burden among all Hispanic origin groups within Florida. Further, the similar and higher work-related HRI hospitalization and death rates, respectively, among Hispanics compared to non-Hispanics may be due to susceptibility factors unique to the sub-population employed in high HRI risk occupations. These susceptibility factors may include language barriers, management style, and cultural identity [298, 299]. South Florida has a large Hispanic population; however, this region also

has some of the lowest rates of HRI. South Florida is predominantly Hispanic (64.5% of population in Miami-Dade County) with the plurality being of Cuban origin (34.3%) [300, 301]. With such a majority of Hispanics in South Florida, Hispanics may represent a large percentage of nearly every industry, occupational, and job class. Further, as Klinenberg noted in relation to the 1995 Chicago heat-wave, the social and spatial composition of Hispanic communities including high population density and vibrant/active public and retail spaces may have contributed to the lower mortality rates among Hispanics [7]. In North Florida, Hispanics comprise a small portion of the population (5% of population in the panhandle[51]) and are primarily of non-Cuban descent (e.g., 39% of panhandle Hispanics are of Mexican origin), and the vast majority of these individuals are employed in the service or construction/extraction occupations, which are major occupation groups at high risk for HRI [147, 301, 302] [CPS 2005-2011]. In addition, there may be other (protective) factors involved in residing in South Florida, such as cooling ocean breezes or access to medical care [303].

Few researchers have considered the relationship between heat and injuries when evaluating heat-related outcomes [84, 85, 286, 304] and injuries are often excluded when the relationship between heat and all-cause mortality or morbidity is analyzed [1, 11, 18, 92]. As HRI progresses, it affects the neurological system, potentially resulting in decreased cognitive function (including short-term memory) and motor skills [42, 305]. Within this analysis, injuries were identified as a potentially important co-morbid condition for HRI, especially for work-related cases. Fifteen percent of work-related and 13 percent of non-work-related HRI ED visits (treated and released) with a selected co-morbid condition had a diagnosis code indicating an injury. Injury diagnoses were also present among HRI hospitalizations and non-work-related HRI deaths, indicating the potential need for inclusion of injuries in further research and interventions related to the prevention of HRI. This may be especially true for workers at risk of HRI who work in potentially precarious situations (e.g., on scaffolding) or with potentially dangerous equipment (e.g., nail gun).

---

[51]Panhandle = Bay County, Calhoun County, Escambia County, Franklin County, Gadsden County, Gulf County, Holmes County, Jackson County, Jefferson County, Leon County, Liberty County, Madison County, Okaloosa County, Santa Rosa County, Taylor County, Wakulla County, Walton County, Washington County

LIMITATIONS

The present analysis does not capture the full burden of HRI in Florida. Individuals seeking treatment at medical facilities outside of an ED or hospital (e.g., doctor's office or urgent care) or individuals not seeking care but remaining alive are not included in this analysis. Future research, with additional data sources, will be required to estimate the full burden of HRI in Florida and to characterize the distribution of susceptibility factors among these additional cases. Although this limitation must frame the results presented here, the present analysis characterizes the burden of HRI severe enough to warrant medical attention in the ED or hospital or that results in death. Prevention and interventions targeted to those groups (e.g., males) with the largest burden of severe HRI will reduce the overall burden of HRI, as well as reduce overall costs to the health care system. Additionally, this analysis was limited to Florida residents because denominator data were not available to estimate the non-resident population. However, Florida industries employ thousands of out-of-state workers and the large tourism industry brings millions of non-residents (tourists) to the state [306, 307]. These individuals may not be acclimatized to the Florida climate and may be at increased risk of HRI. Although not part of the analyses, during the study period there were 2493 non-work-related HRI ED visits, 308 HRI hospitalizations, and 10 deaths among non-residents as well as an additional 128 work-related ED visits, 25 hospitalizations, and 4 deaths among non-residents. Future research will also be required to characterize the distribution of susceptibility factors among non-residents.

The majority of work-related HRI cases were identified by workers' compensation as the expected payer source (ED visits =78.1%; Hospitalizations =69.2%). However, there are many barriers to a worker's ability to access workers' compensation, and many of those at high risk for work-related HRI may not qualify for workers' compensation or may be unaware of its availability [299]. While the use of Ecodes for classifying work-relatedness helps identify those individuals not captured by workers' compensation, the Ecodes, especially the location codes, may incorrectly classify non-work-related cases as work-related. As work-relatedness is highly under-identified [308-

310], classifying non-work-related cases as work-related via Ecodes is assumed to result in a smaller amount of bias then incorrectly classifying work-related cases as non-work-related.

A further limitation of these analyses is the reliance on diagnosis codes. Assignment of ICD-9-CM/ICD-10 codes and their usage (or non-usage) varies by facility and geographic location [2, 13, 61]. No uniform guidelines exist to inform the assignment of a HRI. As a result, HRIs are often recorded in the medical or death record as the condition that arises from response to the stress of the thermoregulatory process (e.g., acute renal failure, acute respiratory distress syndrome, or myocardial injury) or the pre-existing condition that may have been exacerbated by the stress of the thermoregulatory process (e.g., cardiovascular disease, renal failure, or diabetes) [2, 52, 62-64]. This leads to a potential under-reporting of HRI. It is unclear if the potential under-reporting of HRI is differential with respect to the susceptibility factors examined here. Non-differential under-reporting of HRI within susceptibility factors would increase the rates but would not change the ratio of observed differences.

The unit of analysis for this study was the county of residence, the smallest geographic unit available for which Florida population estimates could be obtained from the Florida Legislature's Office of Economic and Demographic Research. These population estimates provide a reasonably accurate estimation of person-time which do not require the assumption of a constant population during the study time period or the interpolation of decennial census data. The data from the Florida Legislature's Office of Economic and Demographic Research combines multiple data sources to estimate how the Florida population demographics changes from year to year [271] . Unfortunately, summarizing the data via county can obscure within county differences, which may be large. For instance, Orange County includes both a large urban area (i.e., Orlando) and rural agricultural areas; for this analysis Orange County was classified as urban. In this situation, if the majority of the HRI cases in an urban classified county were in the rural area (i.e., misclassified as urban), then the rural/urban differences would be larger than originally estimated. If the reverse were true, where the majority of HRI cases were in the urban area of a urban classified county (i.e., correctly classified as urban), then the rural/urban results would not change. However, when targeting HRI prevention to

rural areas, it is important to know if rural areas within urban counties would benefit from prevention programs or if it is only rural areas within rural counties that would benefit. Analysis of HRI and various susceptibility factors within smaller geographic areas could provide further information useful in guiding the use of limited resources to prevent and reduce HRI. However, county-specific rates can be useful for evaluating reductions in the overall burden of HRI.

CONCLUSION

This study describes the burden of HRI among Florida residents using a combination of three data sources: ED, hospital discharge, and death certificates. These three datasets are not often used collectively within the heat-health literature. However, using this information in aggregate provides a more complete picture of the burden of HRI requiring medical attention. Regional and sub-population differences were observed, reflective of Florida's diverse climate and population. However, the highest rates and counts of HRI were observed in males, those living in rural counties, and residents' aged 15-35 years. HRI is highly preventable with behavioral modifications and community-level interventions. Therefore, interventions instituted among sub-populations identified here as having the highest HRI burden may result in the greatest reduction in HRI (and other heat-related outcomes). However, further analysis will be required to: clarify the relationship between HRI, race/ethnicity, and SES among Florida residents; identify additional susceptibility factors using or creating other data sources; and understand how all the susceptibility factors may work in conjunction with each other to increase an individual's or community's HRI risk.

Table 5-1. Distribution of *non-work-related* HRI among Florida residents by selected characteristics (2005–2012)

Characteristic	Florida Residents	HRI ED visits			HRI Hospitalizations			HRI Deaths		
	N (%)	N (%)	Rate*	Rate Ratio (95% CI)	N (%)	Rate*	Rate Ratio (95% CI)	N (%)	Rate*	Rate Ratio (95% CI)
Gender										
Male	72,742,430 (48.9)	17,405 (72.6)	47.85	2.77 (2.69, 2.85)	3,807 (79)	10.47	3.94 (3.68, 4.23)	94 (67.6)	0.26	2.18 (1.53, 3.11)
Female	76,018,379 (51.1)	6,576 (27.4)	17.30	Reference	1,009 (21)	2.65	Reference	45 (32.4)	0.12	Reference
Race*										
White	117,671,057 (79.1)	17,511 (73.5)	29.76	Reference	3,649 (76.5)	6.20	Reference	101 (72.7)	0.17	Reference
Black	24,114,183 (16.2)	5,129 (21.5)	42.54	1.43 (1.39, 1.47)	870 (18.2)	7.22	1.16 (1.08, 1.25)	36 (25.9)	0.30	1.74 (1.19, 2.54)
Other	6,975,569 (4.7)	1,182 (5)	33.89	1.14 (1.07, 1.21)	253 (5.3)	7.25	1.17 (1.03, 1.33)	2 (1.4)	0.06	0.33 (0.08, 1.35)
Ethnicity†										
Non-Hispanic	116,393,541 (78.2)	21,033 (88.6)	36.14	Reference	4,194 (88.3)	7.21	Reference	124 (89.2)	0.21	Reference
Hispanic	32,367,268 (21.8)	2,716 (11.4)	16.78	0.46 (0.45, 0.48)	557 (11.7)	3.44	0.48 (0.44, 0.52)	15 (10.8)	0.09	0.44 (0.25, 0.74)
Rural/Urban										
Urban	139,458,520 (93.7)	21,636 (90.2)	31.03	Reference	4,379 (90.9)	6.28	Reference	127 (91.4)	0.18	Reference
Rural	9,302,289 (6.3)	2,345 (9.8)	50.42	1.62 (1.56, 1.70)	437 (9.1)	9.40	1.50 (1.36, 1.65)	12 (8.6)	0.26	1.42 (0.78, 2.56)

*Crude rate per 100,000 person-years
†Missing race: ED = 159, Hospital = 44
‡Missing Ethnicity: ED = 232, Hospital = 65

Table 5-2. Distribution of *work-related* HRI among Florida resident by selected characteristics (2005–2012)

Characteristic	Florida Residents N (%)	HRI ED visits N (%)	HRI ED visits Rate*	HRI ED visits Rate Ratio (95% CI)	HRI Hospitalizations N (%)	HRI Hospitalizations Rate*	HRI Hospitalizations Rate Ratio (95% CI)	HRI Deaths N (%)	HRI Deaths Rate*	HRI Deaths Rate Ratio (95% CI)
Gender										
Male	35,690,570 (52.7)	2,586 (86.8)	14.49	5.91 (5.32, 6.58)	396 (95.4)	2.22	18.73 (11.82, 29.69)	22 (95.7)	0.12	19.77 (2.67, 146.71)
Female	32,080,314 (47.3)	393 (13.2)	2.45	Reference	19 (4.6)	0.12	Reference	1 (4.3)	0.01	Reference
Race*										
White	55,638,157 (82.1)	2,348 (79.3)	8.44	Reference	298 (72)	1.07	Reference	16 (69.6)	0.06	Reference
Black	9,547,987 (14.1)	474 (16)	9.93	1.18 (1.07, 1.3)	88 (21.3)	1.84	1.72 (1.36, 2.18)	7 (30.4)	0.15	2.55 (1.05, 6.2)
Other	2,584,739 (3.8)	138 (4.7)	10.68	1.27 (1.07, 1.5)	28 (6.8)	2.17	2.02 (1.37, 2.98)	(0)	0.00	---
Ethnicity†										
Non-Hispanic	53,220,195 (78.5)	2,560 (86.8)	9.62	Reference	324 (78.5)	1.22	Reference	14 (60.9)	0.05	Reference
Hispanic	14,550,689 (21.5)	389 (13.2)	5.35	0.56 (0.5, 0.62)	89 (21.5)	1.22	1.00 (0.79, 1.27)	9 (39.1)	0.12	2.35 (1.02, 5.43)
Rural/Urban										
Urban	62,309,645 (93.7)	2,613 (87.7)	8.39	Reference	366 (88.2)	1.17	Reference	21 (91.3)	0.07	Reference
Rural	3,479,229 (6.3)	366 (12.3)	21.04	2.51 (2.25, 2.8)	49 (11.8)	2.82	2.4 (1.78, 3.23)	2 (8.7)	0.11	1.71 (0.40, 7.27)

*Crude rate per 100,000 person-years
†Missing race: ED = 19, Hospital = 1
‡Missing Ethnicity: ED =30, Hospital = 2

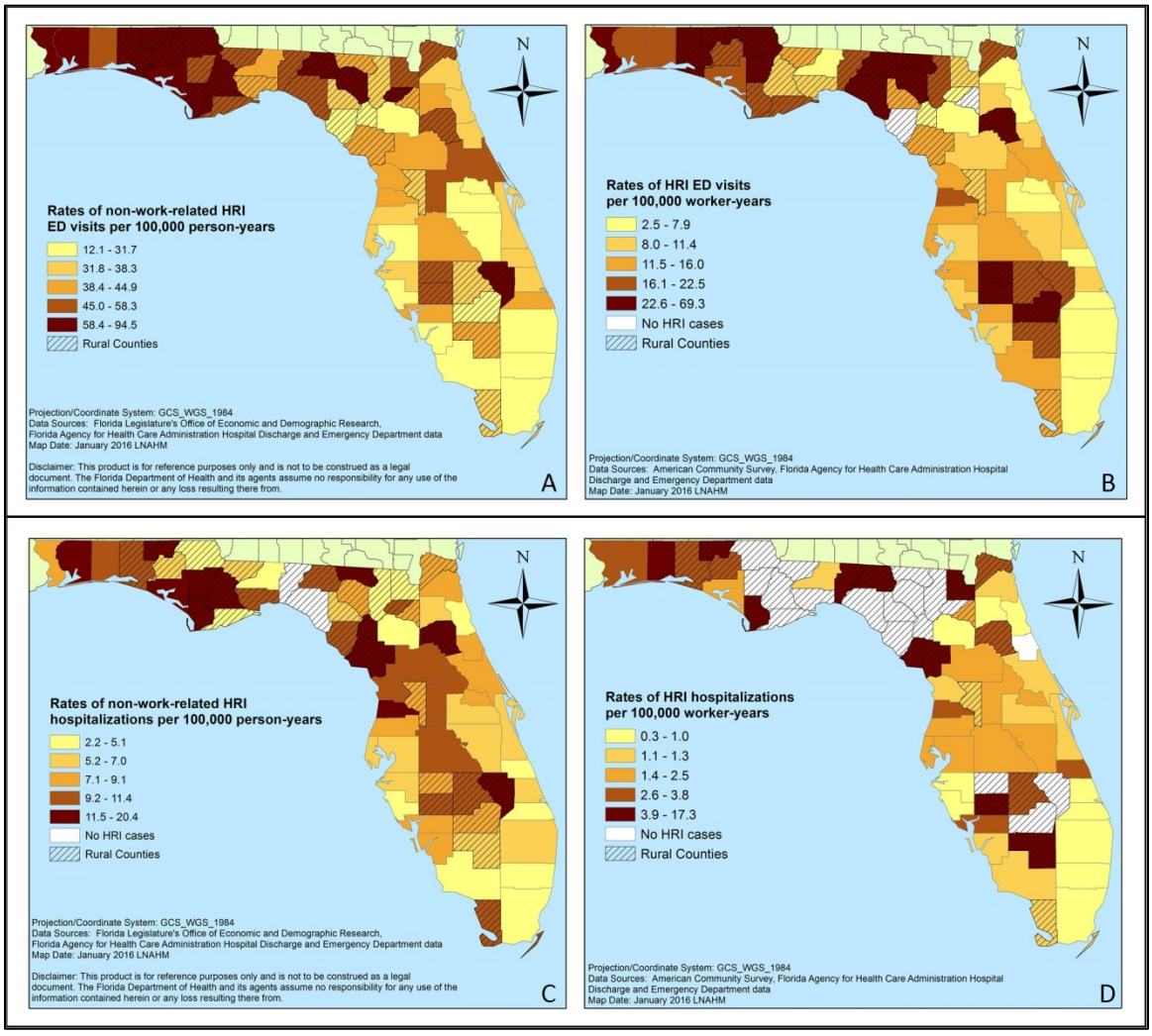


Figure 5-1. County-specific rates of HRI ED visits and hospitalizations among Florida residents during the warm season (2005–2012): (A) Non-work-related HRI ED visits rates per 100,000 person-years; (B) work-related HRI ED visits rates per 100,000 worker-years; (C) Non-work-related HRI hospitalizations rates per 100,000 person-years; (D) work-related HRI hospitalizations rates per 100,000 worker-years

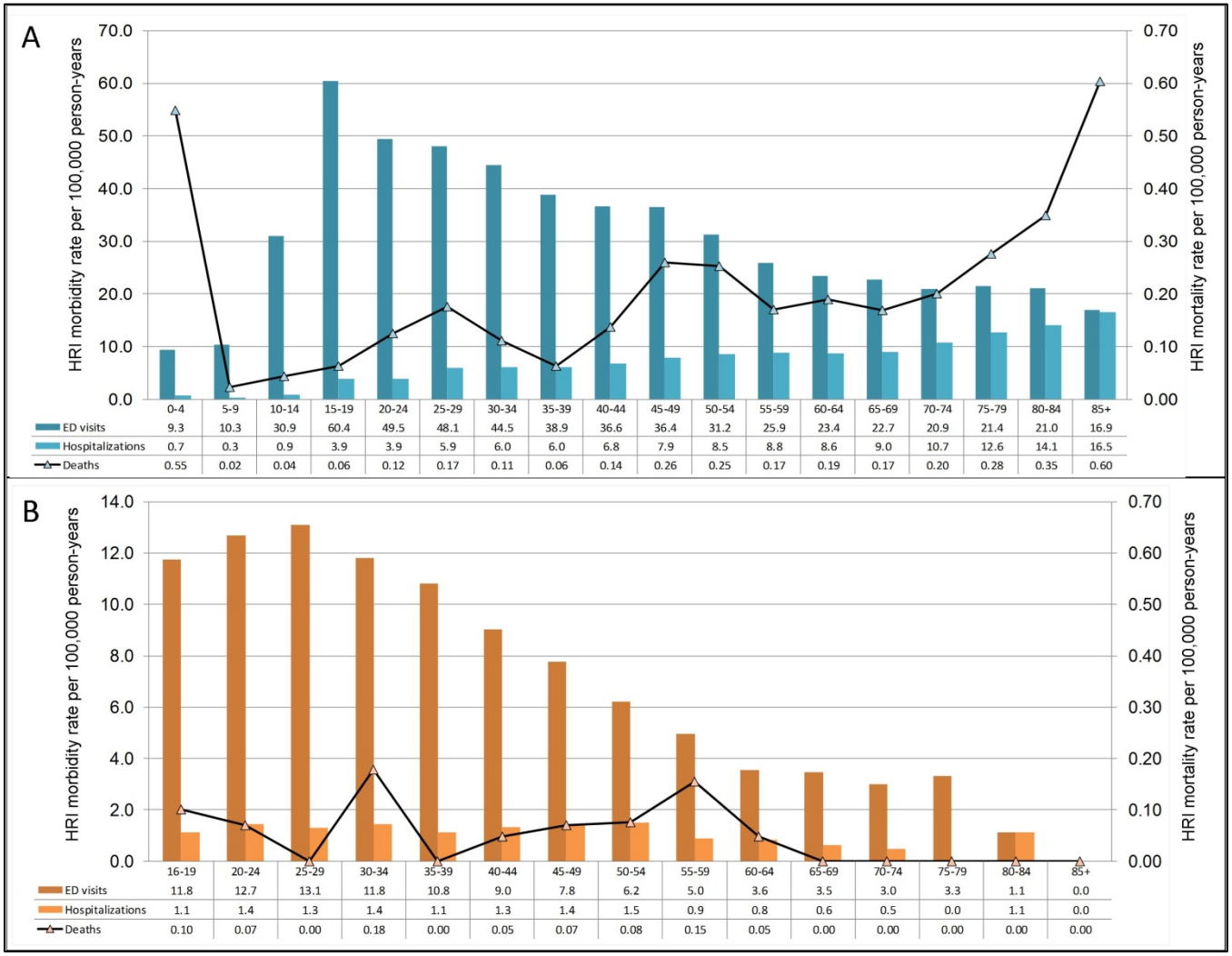


Figure 5-2. Age-specific rates of *non-work-related* (A) and *work-related* (B) HRI identified among Florida residents (2005–2012). All rates are per 100,000 person years. In panel (A), the non-work-related morbidity rates (left y-axis) are two orders of magnitude larger than the non-work-related mortality rates (right y-axis). In panel (B), the age distribution for work-related HRI starts at age 16 and the morbidity rates are approximately an order of magnitude larger than the mortality rates.

CHAPTER 6. HEAT-RELATED MORBIDITY AND MORTALITY ASSOCIATED WITH AMBIENT OUTDOOR HEAT AND HEAT WAVES

INTRODUCTION

High outdoor temperatures can result in adverse health outcomes to those exposed to the heat, especially during heat waves defined as short periods (i.e., contiguous days) of intense heat [38, 311]. For example, during the 1995 Chicago heat wave (July 14–20), there were an estimated 739 deaths (485 recorded as heat-related illness [HRI] by the medical examiner) and 1,072 hospitalizations (731 recorded as HRI in the medical record) attributed to the heat wave [75, 145]. During the August 2003 heat wave in Europe, there were between 27,000 and 44,800 deaths (dependent on the countries included) [8, 146]. Adverse heat-related outcomes also occur outside of heat waves and in the literature these cases are included in analyses which examine the relationship between heat-related outcomes and ambient heat over the summer [1, 66, 312]. The magnitude of effect identified in heat-wave or episodic analyses tends to be larger than analyses examining ambient heat over the entire summer as any analyses of total summer days includes days of less intense heat.

The relationship between temperature and heat-related morbidity/mortality is non-linear with short lagged effects. The shape of the exposure-response curve for the entire temperature spectrum has been shown to be linear positive and linear negative above and below a threshold, respectively, U-shaped, or J-shaped [2, 17, 18, 88, 162, 232]. There is very little research, however, which has characterized the heat-mortality/morbidity burden observed over the summer versus the (potential) additional burden observed on contiguous days of high heat [18, 112, 143, 166, 251, 266]. Examining the interactive effect of these two conditions (i.e., daily heat and heat waves) will present a

more complete picture of the heat-health[52] relationship, informing the timing of public health messages about heat-awareness and prevention of adverse health outcomes. If, for instance, the burden of heat-related outcomes is not only attributed to heat waves, then the public may need to be made aware that adverse heat-related outcomes are a possibility outside heat waves and be encouraged to employ mitigation strategies even when they may not traditionally perceive a danger.

The exposure-response curve and the number of individuals affected during a heat wave depend on the physiological and behavioral responses of an individual or the behavioral responses of a community to hot weather [17, 18, 162, 247]. These responses may vary by latitude, with populations in lower latitudes being better adapted to a warm climate. In warm climates, physiological changes (e.g., acclimatization) and behavioral responses (e.g., use of air conditioning and lighter weight clothing) allow individuals to adapt to their climate by mitigating their heat exposure. Communities in warm climates may further mitigate heat exposure in other ways such as type of housing (e.g., single level), building material, or increased green space. However, even in areas well-adapted to warm weather, individuals may still succumb to HRI and other adverse heat-related outcomes, especially individuals physically exerting themselves in the heat [154, 256-258]. In warm humid climates, there may also be an increase in the burden of occupational HRI, incidence of renal outcomes during heat waves, or other heat-related outcomes, especially among males [70, 198, 214, 221, 260-262].

In order to create effective heat adaption and mitigation strategies in subtropical and tropical regions, it is important to conduct region-specific research to characterize the heat-health relationship. It is difficult to determine if and how the magnitude of heat-related outcomes, the shape of the exposure-response curve, and the temperature value of the effect threshold (i.e., intensity of heat wave) vary by latitude and climate. The majority of studies examining the heat-health relationship have been conducted in the northern U.S., Europe, and Australia [1, 2, 66]. Only a

[52]For this paper the phrase heat-health refers to the relationship between heat-related outcomes (e.g., HRI) and a metric for heat exposure (e.g., heat index).

handful of studies globally have been conducted in areas with subtropical or tropical climates similar to the southeastern U.S.

To address some of the limitations of prior work, the objective of this chapter is to examine the heat-health relationship among Florida residents. This includes an analysis to model the shape of the exposure-response curve. Given that there is currently no uniformly accepted definition of a heat-wave. A series of models were run to identify the optimal threshold (i.e., intensity) and duration values for a Florida-specific heat wave definition. In order to explore the relationship between increasing daily outdoor heat and HRI morbidity, an assessment of whether the effect is modified by heat waves was made. In other words, is there an additional duration effect for contiguous days of high heat? Since prior work has noted that occupationally related adverse heat-outcomes are of potential concern in warm and humid environments, this analysis models the heat-health relationship separately for both work-related and non-work-related HRI. Finally, as Florida covers a large geographical area, the assumption was made that the heat-health relationship varied across the state and statistical methods were employed to account for the variation.

MATERIALS AND METHODS

STUDY POPULATION

The analysis was restricted to Florida residents for the years 2005–2012 and during the warm season, 1 May–31 October [280]. The analysis was restricted to the warm season as the general exposure(s)/etiologic pathway(s) for heat-related outcomes may differ by season. The geographical area of analysis was the zip code. For this analysis all zip codes were standardized to the 2007 zip code as provided by the Esri 2008 zip code polygon shapefile [313]. The population data was obtained from the 2007–2011 American Community Survey (ACS) 5-year estimate. The denominator for non-work-related cases was the Florida population while for work-related cases the employed population was defined as the Florida civilian work-force (employed at work and employed absent). For the analysis the populations were assumed to be stable.

OUTCOME: MORBIDITY DATA

Emergency department (ED) and hospital discharge data for Florida residents for the years 2005–2012 were obtained from the Florida Agency for Health Care Administration (AHCA). The two datasets are mutually exclusive with an individual admitted to the hospital through the ED only counted in the hospital discharge data. For this analysis, both data sets were combined to create one morbidity data set. A record was defined as a heat-related illness (HRI) case if one of the diagnosis codes 992.0–992.9 (effects of heat and light) from the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) were found in any of the diagnosis fields. Additionally, a record was also defined as an HRI case if one of the following external cause of injury codes (Ecode) were present in any of the Ecode fields: E900.0 (excessive heat due to weather), E900.1 (excessive heat due to man-made origin), or E900.9 (excessive heat due to unspecified origin)[53].

Timing of the onset of HRI symptoms needed to be estimated from these administrative datasets. For records obtained from the hospital discharge data, the date of ED visit was used for those who were admitted to the hospital via the ED. Otherwise the date of hospital admission was used. This was done in order to more accurately estimate HRI onset [281].

The HRI morbidity data was stratified by work-relatedness. A work-related case was defined as a record where the expected payer was workers' compensation or if one of the following Ecodes was present in any of the ICD-9-CM Ecode fields: E000.0, E000.1, E800-E807 (4th digit = 0), E830–E838 (4th digit = 2 or 6), E840–E845 (4th digit = 2 or 8), E846, E849.1–E849.3 (Table 4-3) [277]. All HRI cases not classified as work-related were classified as non-work-related.

OUTCOME: MORTALITY DATA

Death certificate data was obtained from the Florida Department of Health (FDOH) Office of Vital Statistics for the years 2005–2012 and restricted to Florida residents. A death was classified as heat-related if any of the International Classification of Diseases, Tenth Revision (ICD-10) codes

[53]Note: that sometimes the fourth digit is not used, instead E900 is incorrectly recorded. If a record had E900 present in any of the Ecode fields the record was considered a HRI case.

T67.0–T67.9 (effects of heat and light), X30 (excessive heat due to weather), or W92 (excessive heat due to man-made origin) were found in any of the underlying cause or contributing cause of death fields. Death certificates contain a field indicating the date of injury. In order to more accurately access HRI onset/exposure and to potentially account for misclassification bias the date of injury was assessed in addition to the date of death. If the *injury at work* field on the death certificate was marked yes and the individual was 16 years or older then the death was classified as work-related.

METEOROLOGICAL DATA

The state of Florida is divided into seven National Weather Service (NWS) regions, one for each of the weather forecasting offices (WFOs) (Figure 4-1). Each WFO issues weather related warnings and alerts to citizens within their jurisdiction.

Weather data were obtained from the 56 National Center for Environmental Services Land-Based first-order stations and the 36 Florida Agricultural Weather Network stations (i.e., Mesonet sites [FAWN]) (Figure 4-1). Using ArcGIS 10.3.1, the coordinates for each weather station were mapped and the Euclidian distance between each weather station and each zip code centroid was calculated. The zip code was attached to the nearest weather station (i.e., smallest Euclidian distance). This was an iterative process; as weather stations become available (e.g., come on-line) or unavailable during the study period, the nearest station to each zip code was recalculated.

Hourly meteorological data were obtained from each of the stations for 2005–2012. Heat index was calculated using relative humidity and temperature in accordance with NWS methodology (Equation 4-4) [118, 120, 124]. Some of the stations included in the analysis only recorded readings during working hours (approximately 8:00–17:00) and therefore, the daily average or minimum temperature readings from these stations would be overestimated. In order to decrease potential exposure misclassification maximum daily temperature and maximum daily heat index were used in order to increase the number of stations included in the analysis.

The 95^{th} and 99^{th} percentiles for maximum daily temperature across the state for May–September were obtained from the Florida Climate Center (Figure 4-2) and calculated based on NWS

Cooperative Observer Network (COOP) using all available data years. The 95th and 99th percentile for maximum daily heat index for May–September was also calculated by the Florida Climate Center for each NWS first order station and FAWN station, dependent of the data years available for each station. The range of values for the 95th percentile and 99th percentile of maximum daily heat index was 101.0°F–109.0°F (mean = 105.3°F; standard deviation [sd] = 1.80°F) and 101.3°F–114.7°F (mean = 109.5°F; sd = 2.99°F), respectively.

AMBIENT HEAT

Two metrics of heat were used in this analysis: maximum daily temperature and maximum daily heat index. Temperature is the most consistently and widely available metric for heat exposure and within the obtained weather data had less station-specific missing data than heat index (Figure A-6). However, heat index is the current metric used by the NWS for issuing heat warnings and advisories to the public [278, 314].

One of the *a-priori* assumptions was that the heat-health relationship varied by region. As such, model parameterization was conducted by NWS regions for morbidity data. The following model parameterizations were examined: linear, quadratic, natural cubic spline with five degrees of freedom, and piecewise linear. The exposure was also parameterized as a piecewise constant function with an indicator term for every two degrees of temperature or heat index. This categorical parametrization was done in order to assess the actual shape of the exposure-response relationship. The models were compared using the Akaike information criterion (AIC) where the model with the smallest value was chosen. However, if the difference in AIC between the two models was less than two (i.e., $|\mathrm{AIC}_1 - \mathrm{AIC}_2| < 2$) then the model with the simplest parametrization was chosen [315]. For each combination of work-related status and morbidity, the chosen parameterization was the parametrization that was the best fit for the majority of regions. Piecewise linear parameterization was selected to model the relationship between non-work-related morbidity and temperature or heat index. The temperature–work-related morbidity relationship was modeled as linear while for heat index a piecewise linear parametrization was used. A linear model was used for mortality and both

temperature and heat index. A table containing the AIC values and a plot for the exposure-response curves for each NWS region and model specification can be found in Table A-1.

The breakpoint, for piecewise linear parameterization, was identified by NWS region using an iterative process which constrains the distance between the end of the first segment and beginning of the second segment to be approximately zero [316, 317]. However, for the analysis, all modeling decisions were required to be the same across the regions; a single breakpoint value was needed. Three methods were used to identify the statewide breakpoint value: segmented analysis for the entire state (instead of each region), mean of the region-specific breakpoint values, and visual inspection of the exposure-HRI relationship. Each statewide breakpoint value was assessed when modeling the region-specific exposure-HRI relationship. The statewide breakpoint value to be used in the analysis was identified as the smallest sum of the differences in the AIC values between the region-specific breakpoint models and the statewide breakpoint models.

Lags of 1-3 days were also included when modeling maximum daily temperature or maximum daily heat index. To overcome the problem of high correlation between individual lag days and potentially imprecise results, a lag-stratified approach was used, such that, lag 2 and 3 were constrained to be equal (i.e., lag1, lag2-3) [312]. A number of previous studies have observed only a lag of one day [62, 66]. For this reason the prior day was considered separately from the 2-day constrained term. The likelihood ratio test with an alpha level of 0.05 was used to determine the number of lags to be included in the final model.

HEAT WAVES

Heat waves were defined with duration of two or more days or four or more days above the intensity threshold. Three intensity thresholds were defined: the area-specific intensity threshold for the (1) 95^{th} percentile or (2) 99^{th} percentile value for temperature or heat index or (3) by using a constant statewide threshold of 98°F for temperature and 108°F for heat index. The constant values have been used by the NWS in Florida as criteria thresholds when determining when to issue heat advisories [278, 279]. The combination of intensity and duration definitions resulted in six separate

heat wave definitions. Study days were classified as wither a heat wave day or a non-heat wave day. For a heat wave defined as two or more days above the threshold, the first day above the threshold was not classified as a non-heat wave day while for a heat wave defined as four day or more days above the threshold, the first three days above the threshold were classified as non-heat wave days.

STATISTICAL ANALYSIS

A generalized linear model (GLM) with an overdispersed Poisson distribution was used for all analyses. The unit of analysis was the zip code-day. The algebraic representation of these models can be found in Equation 6-1, Equation 6-2, and Equation 6-3. The equation functions are described below. To account for regional differences in the exposure-response curve, models were run for each NWS region. Random effects meta-regression was used to pool the region-specific results to obtain statewide estimates [318, 319]. Each region-specific model contained the same terms: the offset terms, terms for temporal confounding (i.e., $g(\cdot)$), and the exposure term (i.e., ambient heat, $m(\cdot)$, or heat wave,$f(\cdot)$). The offset term used to estimate non-work-related and work-related HRI rates were the natural log of the population data obtained from the ACS, as mentioned previously.

Equation 6-1. Algebraic representation of the outcome and general ambient exposure

$$log[E(Y_i)] = \log(py) + \alpha + \sum_{j=1}^{k} g_j(x_{ij}) + \sum_{l=0}^{L_W} m(t_{i-l})$$

where $Y_i \sim P(\lambda) =$ the daily outcome,

$m(t_{i-l}) = t_i + (t_i - \psi) \times I(t_i > \psi)$,

$py =$ person years,

$k =$ the number of temporal confounding covariates,

$x_i =$ covariate(s) for temporal confounding,

L_w = maximum lag days,

$t_i =$ the heat metric,

ψ = breakpoint value,

$I(\cdot) = 1$ if true statement, 0 if false statement,

Equation 6-2. Algebraic representation of model of the outcome and episodic exposure (heat waves)

$$log[E(Y_i)] = \log(py) + \alpha + \sum_{j=1}^{p} g_j(x_{ij}) + f(d_i) \text{ where}$$

$$f(d_i) = \sum_{l=1}^{L_w}\left[I(t_{i-l} \geq \tau) \prod_{j=0}^{l} I(t_{i-j} \geq \tau)\right],$$

L_w = maximum lag days, τ = the threshold level, and $I(A)$=1 if A is true

Equation 6-3. Algebraic representation of model for the outcome and daily exposure modified by episodic exposure

$$log[E(Y_i)] = log(py) + \alpha + \sum_{j=1}^{p} g_j(x_{ij}) + \sum_{l=1}^{L_W} m(t_{i-l}) + f(d_i)$$

Temporal confounding, that is changes due to (sub-)seasonal effects or demographic shifts, was addressed with the inclusion of three terms: an indicator term for year, an indicator term for day of week, and a restricted cubic spline function for day of year with 5 knots located at day 151, 181, 212, 243, and 273. The knot locations represent the last day of the month for May, June, July, August, and September. The reference category for the indicator terms were the category with the largest number of HRI cases; year = 2010, month = August, and weekday = Saturday for non-work-related HRI and Wednesday for work-related HRI.

Equation 6-1 represents the ambient heat analysis. Dependent on the exposure-response relationship, piecewise linear parameterization or linear parameterization was used to model the maximum daily temperature or heat index ($m(\cdot)$).

The Equation 6-2 represents the heat wave analysis where the exposure is binary; heat wave days compared to non-heat wave days ($f(\cdot)$). Note that non-heat wave days are dependent on the heat wave definition used and therefore the reference group is not the same across heat wave definitions.

The final equation, Equation 6-3, represents the added impact of heat-waves above the general effect of daily temperature or heat index. This model contains a term for maximum daily temperature *or* heat index ($m(\cdot)$*)* and a term for heat wave ($f(\cdot)$).

SOFTWARE

Data management and linkage was done using SAS 9.3. Statistical analysis and creation of figures (except maps) was done in the statistical package R version 3.1.2 [320-326]. Maps were created using ArcGIS 10.3.1.

RESULTS

During the Florida warm season (May–October) 2005–2012, there were 31,191 HRI cases (ED = 26,960; Hospital = 5,231). Only 10.5 percent of cases were coded as work related (n = 3,394). For both work-related and non-work-related HRI, the majority of cases were male (non-work-related:

n = 24,194 [73.7%]; work-related: n = 2,892 [87.9%]) and White (non-work-related: n = 21,160 [74.0%]; work-related: n = 2,646 [78.4%]). For non-work-related HRI, the highest rates of HRI ED visits and hospitalizations were for those aged 15-19 years (60.4/100,000 person-years) and the elderly, aged 75 years or older (14.2/100,000 person-years), respectively. Workers under the age of the 35 had the highest ED rates (14.2/100,000 person-years) while workers aged 45-54 years had the highest HRI hospitalization rates (1.7/100,000 person-years). The NWS has divided the counties within the state of Florida into seven regions. The crude rate of HRI varied across the regions, with the highest rates occurring in the Pensacola, Tallahassee, and Key West regions (Table 6-1).

A total of 162 heat-related deaths were observed during the study period of which 23 (14.2%) were classified as work-related. Seventy-three percent of heat-related deaths (n= 118) had a recorded date of injury on their death certificate. One additional HRI case, not included in this analysis, died outside of the warm season but was noted to be exposed in July. The majority were male (non-work-related: n = 94 [67.6%]; work-related n = 22 [95.7%]) and White (non-work-related: n = 101 [72.7%]; work-related: n = 16 [69.6%]). Because the total number of work-related HRI deaths was so small, regression modeling was not conducted using work-related HRI death as the outcome. The age-specific death rate was highest for children under the age of 5 (0.6/100,000 person-years) and those aged 85 years or older (0.55/100,000 person-years). The highest age-specific rates of work-related HRI deaths were in the 30-34 (0.20/100,000 person-years) and 55-59 (0.18/100,000 person-years) age groups. The crude rate of non-work-related and work-related HRI mortality was highest in the Pensacola (0.28/100,000 person-years) and Tampa (0.1/100,000 person-years) regions, respectively (Table 6-1).

There was a strong correlation between maximum daily heat index and maximum daily temperature ($\rho=0.89$, $p<0.001$). However, the correlation was weaker above 90°F ($\rho=0.62$, $p<0.001$), where for each temperature degree the heat index could vary by (up to) ± 16°F, dependent on the relative humidity. The results of all the analyses indicated that heat index provided a better

representation of the heat-health relationship in Florida than temperature[54]. This is further expounded upon in the discussion. As such, only the heat index results will be presented below. The results for temperature can be found in Appendix B.

In the following sections the results of the analyses for each of the three statistical equations will be presented separately. Within each section non-work-related morbidity and mortality will be discussed followed by work-related morbidity and mortality.

AMBIENT HEAT

The average maximum daily heat index during the warm months increased as latitude decreased (Figure 6-1). The mean statewide heat index was 94°F.

AMBIENT HEAT: NON-WORK-RELATED

A model with multiple lag days was fit and the best fitting models included lag0, lag1 and lag2-3. The magnitude of effect for lag2-3 was minimal and therefore was not included (Appendix C). The final modeled relationship between maximum daily heat index and non-work-related HRI only included a lag of one day (Figure 6-2). The majority of the effect was observed on the current day with a nine percent relative increase in non-work-related HRI for every 1°F increase in the heat index below the breakpoint (101°F) and a six percent relative increase above the breakpoint (Below: 95% CI = 1.08, 1.09; Above: 95% CI = 1.04, 1.07). For every 1°F increase in the heat index on the prior day there was two percent relative increase in the rate of non-work-related HRI below the breakpoint and a one percent relative increase above the breakpoint (Below: 95% CI = 1.01, 1.02; Above: 95% CI = 1.00, 1.02).

The majority of HRI deaths (n =111, 81%) occurred between a maximum daily heat index of 91°F and 103°F. The relationship between HRI deaths and maximum daily heat index was modeled as linear with a single lag day (Figure 6-3). The relative increase in the death rate for every 1°F

[54]The AIC was used to compare the models with different heat metrics. For non-work-related HRI morbidity: temperature = -128.94, heat index = -145.92. For non-work-related mortality: temperature: -20.82, heat index = -27.23. For work-related HRI: temperature = -53.17, heat index = -95.43.

increase in the heat index was six percent (95% CI = 1.00, 1.13) for the current day and three percent for the prior day (95% CI = 0.99, 1.07).

AMBIENT HEAT: WORK-RELATED

Maximum daily heat index and work-related HRI were modeled with lag0 and lag1 (Figure 6-4). The inclusion of lag2-3 did not substantially change the fit of the model (Appendix C.). As with the prior models, the majority of the effect was observed on the current day. For every 1°F increase in the heat index below the breakpoint (99°F), there was a 13 percent relative increase in the rate of work-related HRI; while above the breakpoint, there was a 6 percent relative increase (Below: 95% CI = 1.11, 1.15; Above: 95% CI = 1.04, 1.09). For the prior day's heat index, the relative increase in the rate of work-related HRI for every 1°F increase in maximum heat index was two percent below the breakpoint and three percent above the breakpoint (Below: 95% CI = 1.00, 1.03; Above: 95% CI = 1.01, 1.05).

Seventy four percent of work-related HRI deaths (n=17) occurred within the heat index range of 91°F to 103°F. The exposure-response curve for work-related HRI deaths and heat index was not estimated as there were too few work-related deaths over the study period.

HEAT WAVES

For all heat-wave intensity definitions, the magnitude of effect was larger and the variance smaller for heat waves with a duration of two or more days versus those with a duration of four or more days (Figure A-7 and Table A-2). As such, only the results from the 2-day heat wave will be presented.

The state wide median heat index during a 2-day heat wave was 107°F, 109°F, and 110°F for the 95th percentile, 99th percentile, and constant threshold value (108°F). Heat wave days defined by two or more days above the 95th percentile, 99th percentile, and 108°F, the constant threshold value, accounted for 2.3, 0.4, and 0.6 percent of study period days, respectively. The Melbourne region had the highest number of distinct heat waves occurring during the study period as well as those with the

longest duration when intensity was defined by the 95th or 99th percentile. When intensity was defined by the constant threshold (108°F), the Tampa region had the highest number of distinct heat waves, and the Tallahassee region had the longest heat wave. For all heat wave definitions, the Key West region had the fewest and shortest distinct heat waves (Table 6-2).

HEAT WAVES: NON-WORK-RELATED

Approximately six, one, and two percent of non-work-related HRI cases occurred during a two-day or longer heat wave with intensity defined at the 95th percentile, 99th percentile, and constant threshold (108°F), respectively (Table 6-3). Greater than 75 percent of non-work-related HRI cases occurred during the first three days of a heat wave defined as two days or longer, with the majority of cases occurring on the first two days (95th percentile: day 1 = 39.9%, day 2 = 23.4%, day 3 = 13.4%; 99th percentile: day 1 = 52.5, day 2 = 21.0%, day 3 = 9.5%; 108°F: day 1 = 42.2%, day 2 = 25.8%, day 3 = 11.6%).

The results of HRI rates during heat waves compared to HRI rates during non-heat waves by heat wave definition can be found in Figure 6-5a. A heat-wave of two days or more defined by a constant intensity (108°F) had a larger magnitude of effect and a slightly smaller variance (RR = 1.93; 95% CI = 1.68, 2.23) than a heat-wave with intensity at the 95th percentile (RR = 1.81; 95% CI = 1.55, 2.12).

There were 8 (5.8%), 4 (2.9%), and 3 (2.2%) non-work-related heat-related deaths that occurred during a two-day or longer heat wave with intensity at the 95th percentile, 99th percentile, and constant threshold (108°F), respectively. According to the date of injury on the death certificate symptom onset occurred prior to the defined heat wave period but the death occurred during the heat wave period for: three, one, and two deaths, respectively.

HEAT WAVES: WORK-RELATED

The distribution of work-related HRI cases during heat waves within the study period was similar to the distribution for non-work-related HRI cases, with the largest proportion of cases, six

percent, occurring during heat waves defined as two or more days with intensity at the 95th percentile (Table 6-3). Additionally, greater than three-quarters of all work-related HRI cases occurred within the first three days of a heat wave, regardless of definition (95th percentile: day 1 = 38.5%, day 2 = 25.6%, day 3 = 13.3%; 99th percentile: day 1 = 65.1%, day 2 = 23.3%, day 3 = 4.7%; 108°F: day 1 = 43.6%, day 2 = 22.5%, day 3 = 11.2%).

HRI rates during heat waves compared to HRI rates during non-heat wave days by heat wave definition can be found in Figure 6-5b. The magnitude of effect and the variance were both larger for a two-day heat wave defined by the constant threshold compared to a two-day heat wave with intensity defined by the 95th percentile (95th percentile: RR= 2.06, 95% CI = 1.56, 2.73; 108°F: RR =2.24, 95% CL = 1.44, 3.49)

There were zero work-related heat deaths that occurred during heat waves defined as four or more days. During the study period, there were 4 (17.4%), 1 (4.3%), and 3 (13%) work-related HRI deaths during heat waves defined by duration of two or more days and intensity at the 95th percentile, 99th percentile, and constant threshold, respectively. According to the death certificates, the injury occurred during a heat wave with intensity defined at the 95th percentile for two of the deaths and intensity defined at the constant threshold for one of the deaths. The other work-related heat deaths were exposed on non-heat wave days.

AMBIENT HEAT AND HEAT WAVES

For both non-work-related and work-related morbidity, the best fitting model did not include a heat wave term. Further, for each of the three heat wave definitions, the null hypothesis that the natural log of the heat wave term was equal to zero could not be rejected (Table 6-4).

DISCUSSION

This paper examines the relationship between HRI and heat (e.g., heat index) from three different angles: the dose-response relationship for the rate of HRI as heat increases, HRI rates and heat waves, and modification of the dose-response relationship by heat waves. To our knowledge,

this is the first study of its kind in Florida and the first study in the southeastern U.S. to look at modification of the heat-health dose-response relationship by heat waves. Further, with the statistical methods employed in this analysis, we were able to efficiently summarize the heat-health relationship over a large geographical area; specifically that there is regional diversity in the HRI rates, the average heat index, and the number of heat waves. The results of this study will guide public health interventions and planning associated with the prevention of heat-related morbidity and mortality in tropical and humid subtropical areas.

This chapter focused on heat index (as opposed to temperature). Although, the goodness of fit statistic indicated the model with heat index provided a better model fit, it is important to mention that the associations of the heat-health relationship identified within this study were similar when using either heat index or temperature as a metric of heat. However, when using the models to predict the number of HRI cases, the temperature model severely under-estimated the number of HRI morbidity cases per warm season (data not shown). Heat index incorporates the amount of humidity in the air, which is important, especially in tropical and humid subtropical areas; because as relative humidity increases, the body's ability to dissipate heat through evaporation decreases. For instance, at a temperature of 94°F, the heat index could range from 87°F to 141°F dependent on the percent relative humidity [327]. Therefore, by not incorporating humidity into the modeling of the heat-health relationship, the variation in humidity and, in turn, the variation in HRI rates that occur for each temperature degree is ignored, underestimating the number of expected cases.

AMBIENT HEAT

Examining the dose-response relationship, HRI was primarily related to the heat index on the day of the event rather than on prior days. Prior studies of HRI morbidity have also observed the strongest association with the same day metric (e.g., temperature) [78, 82, 149, 328]. A positive non-linear exposure-outcome relationship was also observed for HRI morbidity. The percent increase in HRI morbidity for every 1°F increase in exposure was greater for below the breakpoint than for above the breakpoint. This suggests that at higher heat exposure, there may be some implementation of increased adaptation. A similar reduction in slope above a certain temperature was observed in a few

studies: HRI ED visits and maximum daily temperature in Adelaide, Australia; non-accidental mortality and apparent daily temperature in Mexico City, Mexico [170], Seoul, Korea [329] and Detroit, Michigan [135]; and all-cause mortality and maximum daily temperature in Madrid, Spain [157]. However, this result, that is a reduction in the slope, is atypical of the heat exposure-response relationship observed in the literature. To our knowledge, there are five studies available that examined HRI morbidity/mortality and include the exposure-response curves. An exponential association was observed between HRI ED visit rates in North Carolina (2007-2008) and county-level daily mean temperature [263] and between HRI mortality rates in Maricopa County, Arizona (2000-2008) and maximum daily apparent temperature [330]. In Brisbane, Australia (2000-2012), the incidence rate ratio for HRI ED visits increased monotonically with maximum daily temperature [331]. A study of work-related HRI ED visits in Ontario, Canada (2000-2010) and HRI workers' compensation claims in Adelaide, Australia (2001-2010) both reported a monotonic, if not exponential, increase in the outcome as maximum daily temperature increased [304, 332]. The bulk of studies in the literature examine all-cause (or non-accidental) morbidity and mortality, for which the slope of the exposure-response relationship is either close to zero or monotonic as the exposure metric increases [17, 69, 134, 159, 161, 174, 212, 214, 330, 333, 334].

This is the first study that examined work-related and non-work-related HRI separately. In general, the percent increase in non-work-related HRI with increasing heat observed within this study was lower than increases seen in other studies of HRI. Our study reported a 16 and 10 percent increase in non-work-related HRI per 1°C[55] increase in same-day maximum daily heat-index below and above the 38.3°C (101°F) breakpoint, respectively (9% increase below the breakpoint and 6% increase above the breakpoint per 1°F). This is in comparison with a 43 percent increase in HRI ED visits in NC (2007-2009) per 1°C increase in mean daily temperature and a 17 percent increase in Adelaide, Australia (1993-2009) for a 1°C increase in maximum daily temperature above 26°C (78.8°F) [86, 263].

[55]Note that within this paragraph results from this study were converted to Celsius with Fahrenheit equivalents in parentheses. This was done for ease of comparison with other studies from the literature.

Maricopa County, Arizona also observed a 20 percent increase in HRI mortality for every 1°F increase in apparent temperature above 93°F [330]. Whereas within this study, a 15 percent and 6 percent increase in non-work-related HRI mortality was observed per 1°F increase in same-day maximum daily temperature and heat index, respectively.

Work-related HRI rates in Florida increased by 24.2 percent per 1°C increase (13% per 1°F) and 12.6 percent per 1°C (6% per 1°F)[55] in same-day maximum daily heat-index below and above 37.2°C (99°F), respectively. These results were lower than the 75 percent increase in work-related HRI ED visit rate per 1°C increase in maximum daily temperature above 22°C (71.6°F) in Ontario, Canada [332]. However, a closer climatic comparison would be Adelaide, Australia where a 12.7 percent increase in workers' compensation claim rates occurred per 1°C increase in maximum daily temperature above 35.5°C (95.9°F) [304]. Adelaide is characterized by hot dry summers with monthly mean relative humidity ranging from 36-41 percent during the warm season (October-March) [335]. Accounting for the low humidity, the temperature value of 35.5°C (95.9°F) in the Adelaide study is similar to the heat index breakpoint value, 37.2°C (99°F), in our study [327]. As such, same-day maximum daily heat index above for Florida workers was similar to the Adelaide results.

HEAT WAVES

A standard definition of heat waves has not been established in the literature [6, 38, 311]. As result within this paper, six different, but overlapping, definitions of heat waves were assessed to help determine a suitable Florida specific definition (only three presented in text). Duration was assessed as two or more days above an intensity threshold or four or more days above an intensity threshold, with the thought that a longer period of continuous heat leads to a greater rate of HRI than shorter periods [143, 336]. Finally, three intensity thresholds were used. The percentile intensity definitions allowed for variation in potential acclimatization to heat across the state, with northern areas having hypothesized lower heat tolerance than southern areas.

The varying exposure definitions for heat waves and varying outcomes used in the literature make it difficult to compare the results of this study with prior work. However, for those papers that

look specifically at HRI during heat waves, the effect estimates observed on heat wave days compared with non-heat wave days were considerably higher than what was observed in this study [87, 155, 181, 331, 337, 338]. A study in Brisbane, Australia defined heat waves as two or more consecutive days above 98.6°F after controlling for relative humidity, air pollution, and temporal trends, the rate of HRI ED visits (2000–2012) on heat wave days were 18.53 times the rate for non-heat wave days (95% CI = 12.05, 28.49) [331]. However, some researchers have indicated that there may be some variation in the magnitude of effect based on heat wave timing and location, with heat waves occurring earlier in the warm season having a larger impact than those occurring later in the warm season, and heat waves having a smaller impact on warmer locales [143]. In a study in Adelaide, Australia, heat waves were defined as three or more consecutive days above 95°F; between 1993 and 2007, the rate of HRI ED visits and hospitalizations on heat wave days was 2.68 (95% CI: 2.19, 3.28) and 3.12 (95% CI: 2.51, 3.87) times the rate on control days, respectively [155]. In contrast, during the 2009 heat wave in Adelaide and comparing heat wave days to the control period, the rate ratio for HRI ED visits was 12.01 (95% CI: 9.55, 15.12) and for hospitalizations it was 13.66 (95% CI: 8.89, 20.98) [155]. During the 2006 California heat wave, the rate ratio for HRI ED visits during the heat wave compared to the control period ranged from 3.36 to 23.05, with the magnitude of effect decreasing with decreasing latitude [87]. Additionally, it may be that Florida residents have higher rates of HRI on non-heat wave days than other geographical locales, leading to a smaller observed relative effect estimate. For instance, within our study, less than seven percent of deaths occurred on a heat wave day; while in Maricopa County, Arizona, 57% of heat-related deaths in 2005 occurred during a heat wave [156].

In general, the rate ratio comparing HRI rates during a two-day or longer heat wave to non-heat wave days was greater than the rate ratio for four-day or longer heat waves compared to non-(four-day)-heat wave days. This indicates the greatest burden and rate of HRI was observed on the first two or three contiguous days above an intensity threshold. Further, as the majority of HRI cases occur outside of a heat wave, regardless of heat wave definition, adding additional cases to the reference group by increasing the duration of the heat wave captured also increased the variance of the effect estimate. Therefore, as there is no additional benefit (e.g., larger magnitude of effect) of

increasing the definition of a heat wave from duration of two to four days, it is recommended that for public health purposes in Florida, heat wave duration be defined as two days or more above an intensity threshold.

Ideally, the intensity threshold should be defined using the heat index which incorporates both humidity and temperature. As previously mentioned, high humidity can adversely affect the human body's ability to properly thermoregulate, leading to heat-related morbidity and mortality. Florida is the most humid state in the nation with the typical warm season dew point temperature being between 65°F and 75°F [339]. In general, the 99th percentile definition using heat index had a higher station specific threshold value than the other two intensity definitions. As a result, the 99th percentile definition was a less suitable definition since the variance was larger for both non-work-related and work-related HRI morbidity as compared with the other two intensity definitions. The goal for selecting a heat wave definition was to minimize the variance and maximize the effect size. For non-work-related HRI morbidity there was little difference in the magnitude of effect and variance for intensity defined by the 95th percentile or at a constant threshold of 108°F, while for work-related HRI morbidity both the effect size and the variance were larger for the constant threshold than the 95th percentile. Calculating heat waves via a constant threshold is methodologically simpler than via a varying threshold. However, because the heat index varies across the state with fewer days in south Florida (versus north Florida) reaching the constant threshold value, the proportion of summer days classified as a heat wave day decreases with decreasing latitude (data not shown). The use of a constant threshold ignores the potential for acclimatization by the population to an area's average heat index value. As such, the use of a constant threshold may misclassify a heat wave day in south Florida as a non-heat wave day (e.g., higher than average for area heat index but lower than the 108°F). The use of a varying threshold accounts for heat index variation by latitude, with the result being a similar proportion of summer days be classified as heat wave days across Florida. As the difference in effect size and variation between a heat wave defined with the 95th percentile and one defined with the constant threshold were small, when using the heat index to define intensity, the author will leave the decision of relative versus absolute threshold values to the public health practitioner.

Temperature is the most consistently and widely available metric for outdoor heat exposure [111]. The number of weather stations which collect temperature only data is greater than number of weather stations which collect both temperature and humidity data. Due to the larger number of temperature only stations, the geographical areas assumed to have the same exposure will be potentially smaller than the geographical exposure areas created when using heat index, potentially reducing exposure misclassification. If temperature is the metric chosen for defining heat wave intensity for the entire state, then it is recommended, due to the large variation in temperature extremes across the state, that a relative threshold (e.g., 95th percentile) be used instead of a constant threshold value.

AMBIENT HEAT AND HEAT WAVES

This is the first study to examine the additional effect attributed to heat waves using HRI as the outcome. For non-work-related HRI, after adjusting for ambient heat index there was an 11 percent non-statistically significant decrease in the HRI rate for a heat wave defined as two or more days above the constant threshold of 108°F. Prior studies have used all-cause morbidity or mortality as the outcome of interest [112, 143, 166, 251, 340]. These studies have observed small, but positive, heat wave effects. Hajat et al. demonstrated that the total burden attributable to heat waves was small (0.15-0.19% of year-long deaths) in comparison to the overall summer heat-mortality burden (0.39%-1.58% of year-long deaths) in their study of three European cities [112]. In Stockholm, Sweden between 1992 and 2002, an additional 8-11 percent relative increase in total excess mortality was observed per heat wave day, dependent on the heat wave definition and parameterization of the maximum daily temperature and total mortality [340]. A study of 108 U.S. communities (1987–2000), also noted a small increase in mortality (2.8%) attributable to heat waves, but only when a heat wave was defined as four continuous days of high temperatures [251]. However, as noted by Anderson et al. in their study of 107 U.S. communities, regional variation is present, with the added effect of heat waves having the smallest impact in the Southern regions [143].

Overall, the result of this study suggest that during periods of intense heat, Floridians may be implementing behavioral modifications to prevent HRI or are recognizing the symptoms of HRI and

initiating treatment before symptoms become severe enough to require treatment in an ED or hospital. An example in the literature is from Adelaide, South Australia where behavioral modification was suggested to explain the observed concave downward relationship between maximum daily temperature and workers' compensation injury claims. The authors noted workplaces, industries, and trade unions have cessation of work polices for high temperatures [341].

STRENGTH AND LIMITATIONS

The greatest strength of this study was the study population and the geographical location. This is the second study to be completed in Florida and the first to consider general ambient heat exposure [342]. Additionally, to the best of our knowledge, this is the first civilian work-related heat-health analysis conducted in a humid sub-tropical environment. Further, comparatively few studies have examined the heat-health relationship in non-urban communities [343, 344]. Our analysis included all Florida residents and, therefore, includes both rural and urban communities. Finally, the outcome was assessed using three sources (i.e., ED, hospitalizations, and death certificates), allowing for a more complete estimate of the exposure-response relationship than has been provided by prior studies.

The exposure metric used in this analysis was outdoor temperature. However, individuals do not spend all their time outdoors. A handful of prior studies have examined the relationship between indoor and outdoor temperature. A positive correlation has been found between the two, although other factors such as air conditioning usage, building materials, and income level contributed to the variation in indoor heat exposure [345-348].

In this analysis, only a small proportion of all deaths occurred or were exposed, as indicated by date of injury, during a heat wave. This was based on the assumption that the date of death and the date of injury were the same if a date of injury was not recorded on the death certificate. It is possible, if the date of injury had been completed and was different than the date of death, than up to

31 percent[56] of decedents could have been exposed during a heat wave over the eight year study period. However, this estimate is still much smaller than the proportion of all deaths observed during a single heat wave in Arizona [156].

The majority of work-related HRI cases were identified by having the expected payer as workers' compensation. However, there are many barriers to accessing workers' compensation and a large number of individuals who are at high risk for HRI (e.g., agricultural, construction, or landscape workers employed by a small business) may not file claims and would not be classified in this study as a work-related HRI case [299]. The use of additional Ecodes to categorize work-related status helps in the ascertainment of work-related HRI cases; however, we still expect work-related cases to be under-identified [276, 277]. Additionally, the use of Ecodes may incorrectly classify a case as work-related when, in fact, it was not. The latter misclassification is estimated to have a smaller impact than not identifying additional work-related cases through the use of Ecodes.

HRI has been noted in the literature to be underdiagnosed [2, 13, 61]. As such, studies often use other outcomes related to the failure of the thermoregulatory system, such as all-cause or non-accidental mortality/morbidity or cardiovascular disease [1, 2, 66, 204, 311, 349]. However, while these outcomes have a high specificity, due do competing causes, their sensitivity is low. In this study, we chose to have high sensitivity in our outcome definition in order to identify the initial heat-health relationship. One of the key assumptions made when using HRI as the outcome is that classical and exertional HRI are non-differentially diagnosed and coded as HRI (i.e., non-differential specificity). Our results indicated that there was no added impact of a heat wave (i.e., duration effect), suggesting that the majority of HRI in Florida is exertional. However, if classical HRI is being coded as the condition that arises from response to the stress of the thermoregulatory process or the pre-existing condition that may have been exacerbated by the stress of the thermoregulation process, this study would not have captured those cases, and the heat-health relationship presented would be

[56]There were 163 deaths where the date of injury was between May and October (2005–2012). There were 7 deaths where the date of injury was during a heat wave defined as two or more days above the 95 percentile for maximum temperature. There were 44 death certificates that were missing the date of injury. (44+7)/163 =0.312.

incomplete. It is suggested that this study be replicated using other outcomes such as all-cause morbidity/mortality or cardiovascular outcomes.

CONCLUSION

The results presented here indicate that within the humid subtropical and tropical climate of Florida a positive relationship exists between HRI morbidity and mortality among Florida residents and the heat index. The majority of HRI cases, morbidity and mortality, do not occur during a heat wave. It appears that it is the intensity of exposure, and not the duration, that has the largest impact on the relative rate of HRI morbidity and mortality. Examination of the exposure-response curve and comparison of heat wave results with other studies suggest that there may be some form of adaptation occurring among the population. The adaption for higher outdoor heat exposure should be explored in further studies. This study provided summary estimates of the heat-health relationship in Florida, however, this relationship varies across the state and additional sub-area analyses are recommended. Finally, in order to impact the overall burden of HRI morbidity and mortality, the timing of public health prevention and intervention activities should occur during the first days of a heat index range when the highest numbers of cases are expected.

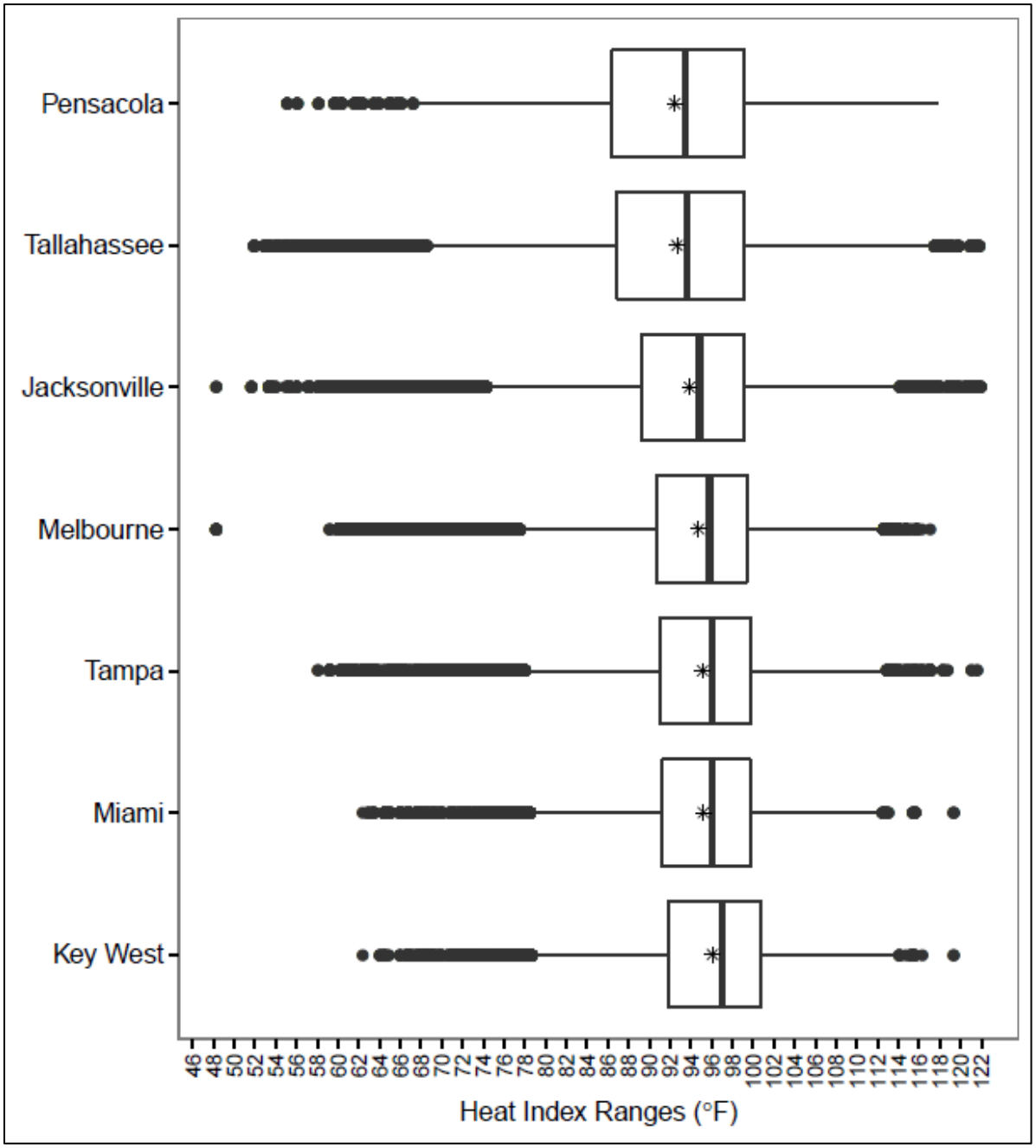


Figure 6-1. Box-and-whisker plot of maximum daily heat index between May-October (2005–2012) stratified by National Weather Service region. The vertical line represents the median heat index value for each region, and the asterisk represents the mean heat index value for each region. The median and mean heat index value for the state is 93.8°F and 94.1°F, respectively.

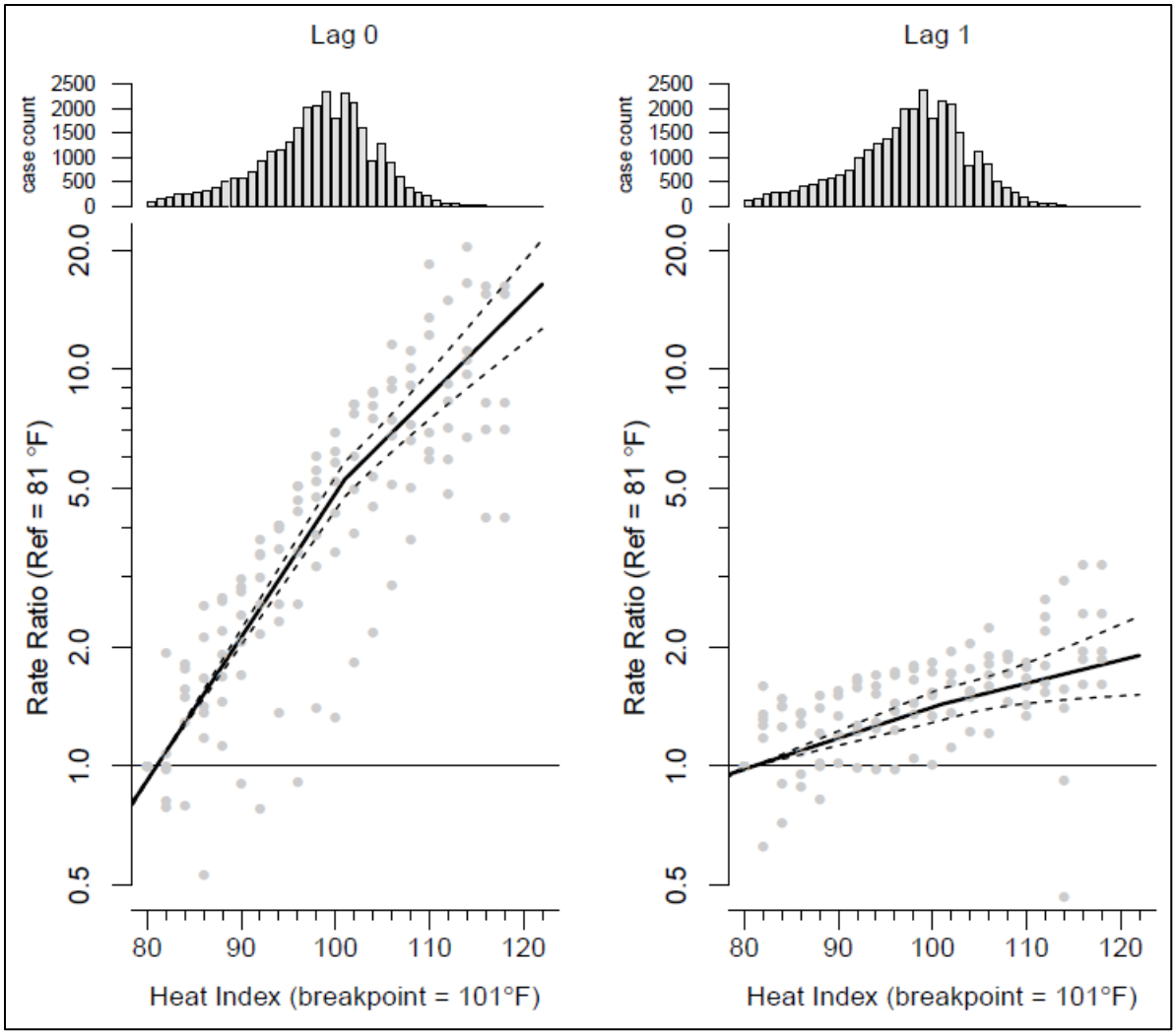


Figure 6-2. Modeled relationship between non-work-related HRI morbidity rates and maximum daily heat index on the current day, lag 0, and the prior day, lag 1, for May-October, 2005–2012 (solid line). The hashed lines are the 95% confidence intervals. The grey dots are the region-specific rate ratios for every two degrees of heat index (e.g., 80-81, 82-83…). The histogram represents the observed number of cases occurring at each heat index degree. All heat index values had at least six cases during the study period except a heat index of 121°F and 122°F which had one and four cases, respectively.

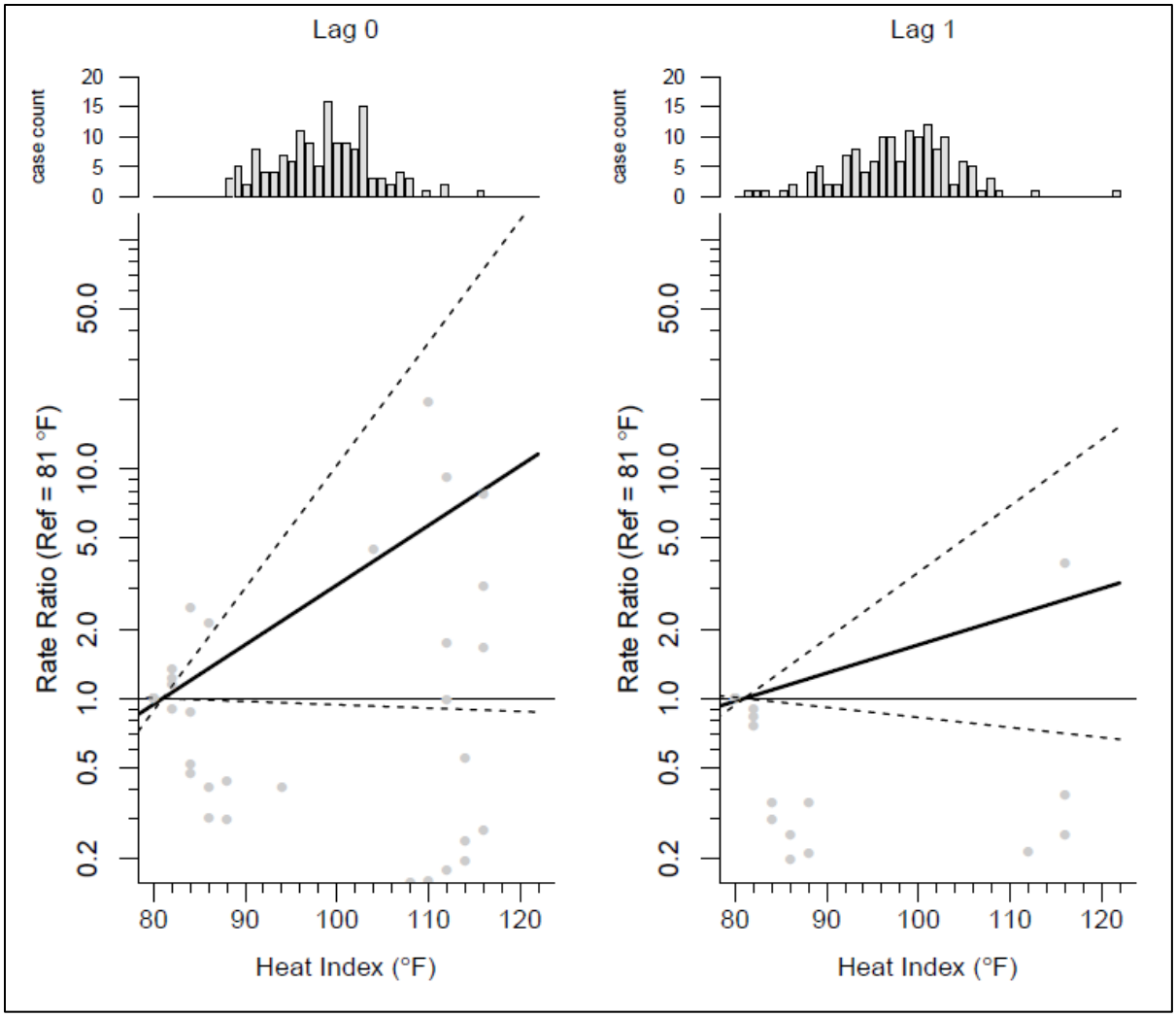


Figure 6-3. Modeled relationship between non-work-related HRI death rates and maximum daily heat index on the current day, lag 0, and prior day, lag 1, for May-October, 2005–2012 (solid line). The grey dots are the region-specific rate ratios for every two degrees of heat index (e.g., 80-81, 82-83…). The hashed lines are the 95% confidence intervals. The histogram represents the observed number of cases occurring at each heat index degree. Note, for instance, that there was one death cases where the current day's heat index was a 115°F.

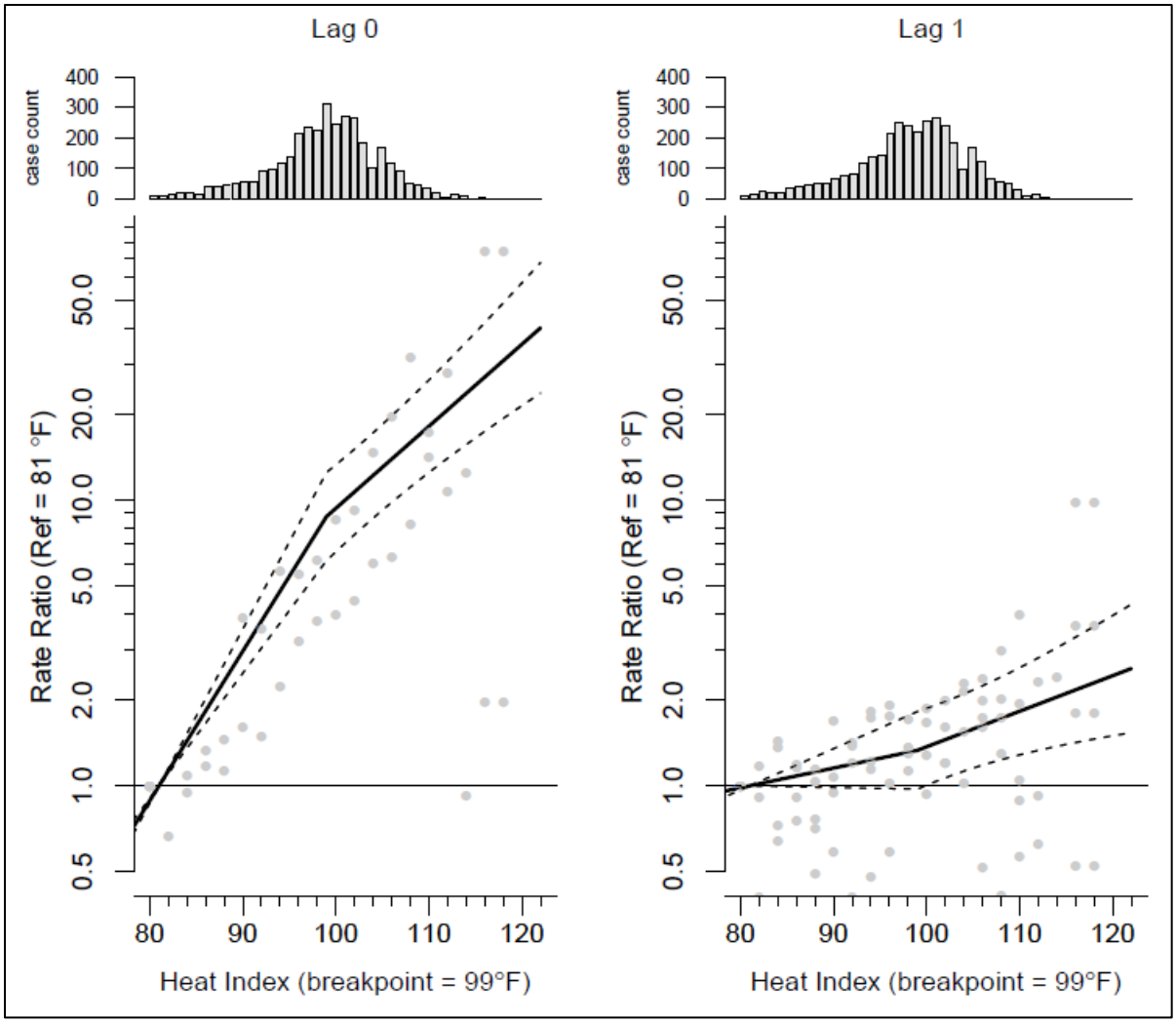


Figure 6-4. Modeled relationship between work-related HRI morbidity rates and maximum daily heat index on the current day, lag 0, and the prior day, lag 1, for May-October, 2005–2012 (solid line). The hashed lines are the 95% confidence intervals. The grey dots are the region-specific rate ratios for every two degrees of heat index (e.g., 80-81, 82-83…). The histogram represents the observed number of cases occurring at each heat index degree. There were zero HRI non-work-related cases at 120°F and 122°F. All other heat index values had at least one case during the study period.

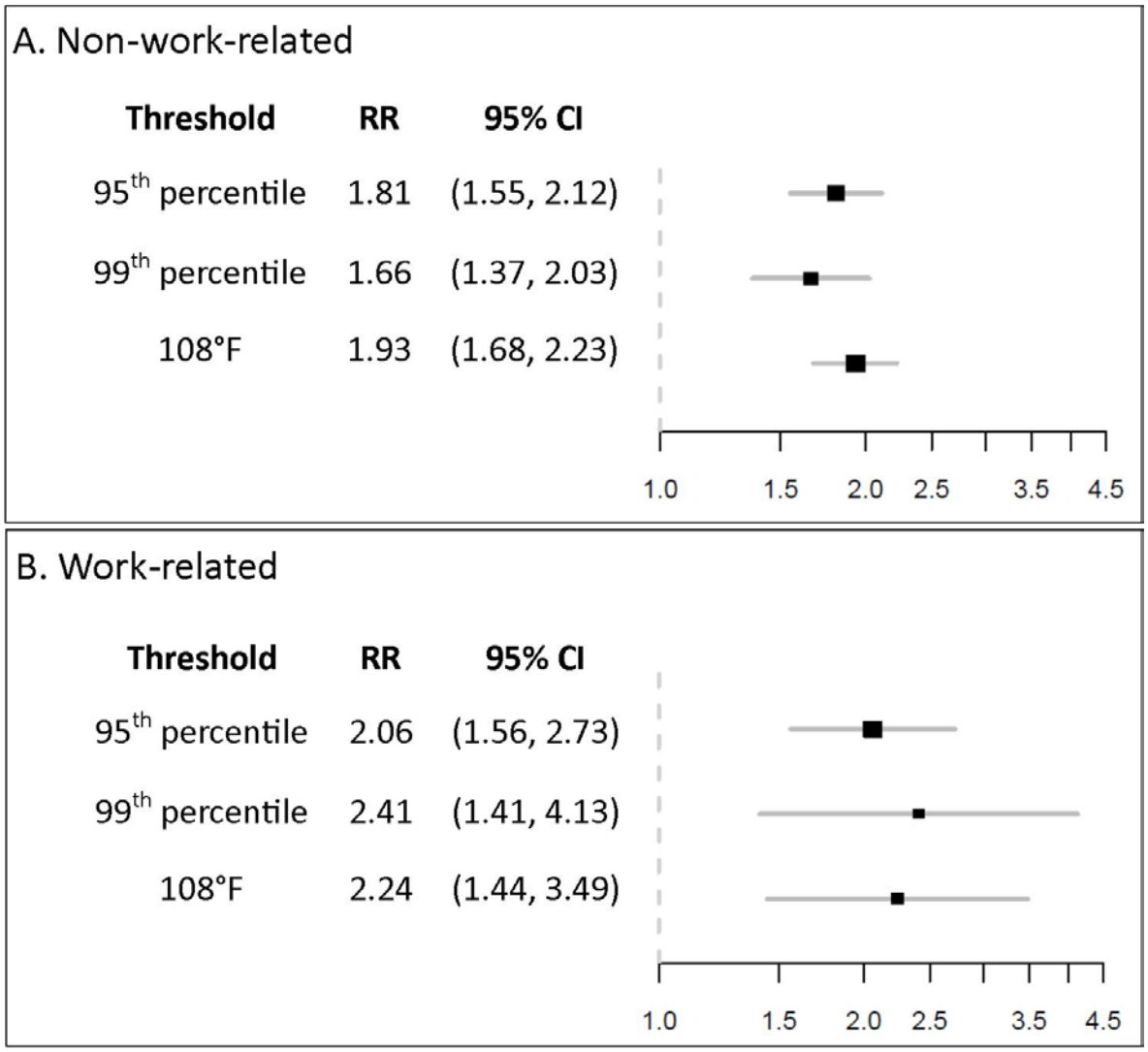


Figure 6-5. Forest plot of the rate ratio (RR) and 95% confidence intervals for non-work-related (A) and work-related (B) HRI morbidity rates occurring during a two-day or longer heat wave compared to rates on non-heat wave days, stratified by varying heat wave definitions. The range of threshold values for the 95th percentile of maximum daily heat index was 101-109°F and for the 99th percentile was 101-115°F.

Table 6-1. Summary of heat related illness (HRI) morbidity and mortality counts and rates among Florida residents by National Weather Service (NWS) region and work-related status (2005–2012)

Type	NWS Region	Non-Work-Related HRI			Work-Related HRI		
		no. HRI cases	*Average annual population*	*Rate per 100,000 person-years (95% CI)*	*no. HRI cases*	*Average annual population**	*Rate per 100,000 worker-years (95% CI)*
Morbidity	Pensacola	1,850	632,505	73.1 (69.9, 76.5)	285	273,650	26.0 (23.2, 29.2)
	Tallahassee	2,058	864,887	59.5 (57.0, 62.1)	251	357,573	17.5 (15.5, 19.9)
	Jacksonville	4,207	2,359,050	44.6 (43.3, 46.0)	437	1,026,905	10.6 (9.7, 11.7)
	Melbourne	6,169	3,750,693	41.1 (40.1, 42.2)	666	1,676,959	9.9 (9.2, 10.7)
	Tampa	8,688	5,244,250	41.4 (40.6, 42.3)	1,157	2,203,323	13.1 (12.4, 13.9)
	Miami	5,705	5,955,060	24.0 (23.3, 24.6)	583	2,731,806	5.3 (4.9, 5.8)
	Key West	120	54,792	54.8 (45.8, 65.5)	15	28,585	13.1 (7.9, 21.8)
	Florida	*28,797*	*18,861,237*	*38.2 (37.7, 38.6)*	*3,394*	*8,298,801*	*10.2 (9.9, 10.6)*
Mortality	Pensacola	7	632,505	0.28 (0.13, 0.58)	-	273,650	---
	Tallahassee	8	864,887	0.23 (0.12, 0.46)	1	357,573	0.07 (0.01, 0.50)
	Jacksonville	21	2,359,050	0.22 (0.15, 0.34)	1	1,026,905	0.02 (0.00, 0.17)
	Melbourne	34	3,750,693	0.23 (0.16, 0.32)	3	1,676,959	0.04 (0.01, 0.14)
	Tampa	37	5,244,250	0.18 (0.13, 0.24)	9	2,203,323	0.10 (0.05, 0.20)
	Miami	32	5,955,060	0.13 (0.09, 0.19)	9	2,731,806	0.08 (0.04, 0.16)
	Key West	-	54,792	---	-	28,585	---
	Florida	139	*18,861,237*	*0.18 (0.16, 0.22)*	23	*8,298,801*	*0.07 (0.05, 0.11)*

*Employed population (defined as employed at work or employed absent from work).

Table 6-2. Number of distinct heat waves with duration of two days or longer in Florida stratified by National Weather Services (NWS) region and maximum daily heat index intensity thresholds (2005–2012)

NWS Region	95th Percentile		99th Percentile		108°F	
	No. distinct Heat Waves	*Max HW days*	*No. distinct Heat Waves*	*Max HW days*	*No. distinct Heat Waves*	*Max HW days*
Pensacola	63	16	6	6	34	8
Tallahassee	96	20	24	6	58	**20**
Jacksonville	94	14	25	7	56	12
Melbourne	**132**	**26**	**53**	**12**	60	18
Tampa	130	18	39	6	**70**	12
Miami	123	19	37	6	41	10
Key West	47	8	4	3	2	3
*Florida**	*291*	*26*	*100*	*12*	*151*	*20*

*The number of distinct Florida heat waves is less than the sum of the regional heat waves. A regional heat wave may have occurred at the same time as a heat wave in another region and, therefore, the heat wave was counted only once when calculating the number of distinct heat waves at the state level. A regional heat wave may not have occurred over the whole region but only for zip codes associated with a few weather stations.

Table 6-3. Proportion of total heat-related illness (HRI) morbidity cases occurring on a two-day or longer heat wave with data stratified by intensity definition* and National Weather Service (NWS) region

NWS Region	Non-work-related			Work-related		
	95th Percentile	99th Percentile	108°F	95th Percentile	99th Percentile	108°F
Pensacola	139 (8.1%)	13 (0.7%)	84 (4.8%)	22 (8.4%)	2 (0.7%)	9 (3.3%)
Tallahassee	221 (12%)	17 (0.8%)	102 (5.2%)	37 (17.3%)	8 (3.3%)	15 (6.4%)
Jacksonville	240 (6%)	30 (0.7%)	107 (2.6%)	38 (9.5%)	5 (1.2%)	19 (4.5%)
Melbourne	379 (6.5%)	127 (2.1%)	80 (1.3%)	44 (7.1%)	14 (2.1%)	3 (0.5%)
Tampa	299 (3.6%)	44 (0.5%)	102 (1.2%)	48 (4.3%)	7 (0.6%)	21 (1.8%)
Miami	399 (7.5%)	64 (1.1%)	18 (0.3%)	37 (6.6%)	7 (1.2%)	4 (0.7%)
Key West	3 (2.6%)	0 (0%)	0 (0%)	---	---	---
Florida	*1680 (5.8%)*	*295 (1%)*	*493 (1.7%)*	226 (6.7%)	43 (1.3%)	71 (2.1%)

*Heat wave intensity defined by maximum daily heat index.

Table 6-4. Assessment of an added heat-wave effect in Florida (2005–2012) via comparison of the model of ambient heat index (i.e., no heat wave term) and models with an additional term for a two-day or longer heat wave stratified by heat wave definition and work-related status .

Model with heat wave term	Non-work-related			Work-related		
	Heat wave effect*	Z Score (p-value)†	AIC‡	Heat wave effect*	Z Score (p-value)†	AIC‡
No heat wave term	---	---	-145.93	---	---	-95.43
95th percentile	1.02 (0.90,1.15)	0.27 (0.8)	-139.3	0.95 (0.75,1.19)	-0.45 (0.7)	-85.72
99th percentile	0.87 (0.70,1.08)	-1.29 (0.2)	-126.17	1.03 (0.68,1.55)	0.13 (0.9)	-76.92
108°F	0.90 (0.77,1.06)	-1.28 (0.2)	-124.08	0.80 (0.50,1.29)	-0.91 (0.4)	-74.19

*The pooled relative effect and 95% confidence interval for HRI morbidity rates during heat waves compared to non-heat wave days after adjusting for ambient heat.
†Z-test H0: the effect estimate is equal to zero
‡Akaike information criterion (AIC) is a test of model fit, the smallest value indicates the best model

CHAPTER 7. COMMUNITY-LEVEL FACTORS AFFECTING THE RELATIONSHIP BETWEEN AMBIENT OUTDOOR HEAT AND HEAT-RELATED ILLNESS

INTRODUCTION

The relationship between heat and adverse health outcomes has been long recognized [1-3, 23, 24, 27]. Adverse health outcomes occur when the human body is no longer able to maintain normal body temperature. At its severest form, these outcomes include neurologic damage, organ or system failure, and even death [48, 49, 59]. Fortunately, heat-related outcomes (e.g., heat-related illness [HRI], cardiovascular disease, respiratory disease, acute renal failure) are highly preventable through individual behavioral and community-level structural modifications. As a result, the rate of adverse heat-related outcomes within a community is determined not only by ambient heat exposure but also by the response and adaptability to heat within each community. An important part of the response and adaptability is identification and prevention of adverse heat-outcomes in vulnerable populations.

In order to identify vulnerable populations, a number of individual- and community-level factors that may increase or reduce the risk of adverse heat-related outcomes have been reported in the literature [7, 9, 38, 42, 203, 215]. These include but are not limited to demographic factors, medical factors, environment (e.g., population density, latitude), availability of community resources, and outdoor occupations or hobbies. The distribution and interaction of these factors within a particular population will impact the burden of heat-related outcomes in that population, and in turn, the most effective use of limited resources for reducing and preventing heat-related outcomes [14, 16]. For instance, if there is a large portion of the population who are elderly and socially disadvantaged, it may be appropriate to concentrate resources on finding ways to reduce social isolation and provide community cooling centers during extreme heat events. However, if there is a large proportion of youth, then it may be more effective to work with schools to make students and

coaches aware of the symptoms of exertional HRI and the importance of acclimatization before participating in outdoor athletics.

Unfortunately, it is unclear which of these individual- and community-level factors are appropriate for identifying vulnerable populations in the humid subtropical area of Florida. Much of the research has been conducted in cooler climates and among urban populations [9, 350]. Therefore, this chapter will focus on modification of the relationship between ambient outdoor heat and HRI by potential susceptibility factors defined at the community level. The susceptibility factors identified in this analysis, in conjunction with prior work (e.g., areas of high HRI burden [see Chapter 5]), will inform decisions on identification of populations vulnerable to heat-related outcomes as ambient heat increases. And by focusing prevention and intervention activities to susceptible populations or areas this may result in a more effective use of limited resources.

MATERIALS AND METHODS

The study population was Florida residents for the years 2005–2012 between 1 May and 31 October (i.e., the warm season [280]). The zip code was the geographical level of analysis. Zip codes were standardized to the 2007 zip code via the Esri 2008 zip code polygon shapefile [313]. Population data were extracted from the 2005-2011 American Community Survey (ACS) 5-year estimate. The population was assumed to be stable during the study period.

Morbidity data at the zip code level was obtained from the Florida Agency for Health Care Administration for the years 2005–2012. HRI was defined as an emergency department (ED) visit or hospitalization with a primary or secondary diagnosis of an International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) code of 992.0–992.9 (effects of heat and light). HRI was also ascertained if one of the following ICD-9-CM external cause of injury codes (Ecode) was listed in the Ecode field: E900.0 (excessive heat due to weather), E900.1 (excessive heat due to man-made origin), or E900.9 (excessive heat due to unspecified origin)[57]. In order to more accurately

[57]Note: that sometimes the fourth digit is not used, instead E900 is incorrectly recorded. If a record had E900 present in any of the Ecode fields the record was considered a HRI case.

establish HRI onset, for those patients who were admitted to the hospital through the ED – the date of ED visit was used instead of the date of hospitalization [281].

The collection and processing of the exposure data has previously been discussed (see Chapter 4). Briefly, maximum daily heat index was obtained from 92 weather stations across the state. All zip code centroids during the study period were linked to their nearest weather station (i.e., smallest Euclidian distance) via an iterative process as stations became (un)available. As presented in the Chapter 6 methods section, maximum daily heat index was parameterized for analysis using a piecewise linear parameterization with the breakpoint at 101°F. The algebraic representation: $t_i + (t_i - \psi) \times I(t_i > \psi)$, where t_i is the maximum daily heat index, ψ is the breakpoint value and $I(\cdot)$ is the indicator function which is equal to one when the statement is true and zero otherwise (Equation 6-1). As the majority of the effect for the heat-health relationship occurs on the same day as exposure, lag periods were not included in the analysis (see Chapter 6). Note that for this paper the term heat-health refers to the relationship between the exposure, maximum daily heat index, and the outcome, daily HRI.

It was assumed that the heat-health relationship varied across the state of Florida, as such, for this analysis the state was broken into 103 zip code-station groups. Each zip code-station group was comprised of all the zip codes linked to a particular weather station. If a zip code linked to multiple weather stations, due to station availability (e.g., station came online), then the zip code was assigned to the group with the majority of study time. Zip code-station groups were created within each of the seven National Weather Service (NWS) regions.

MODIFICATION VARIABLES

A population's heat vulnerability will depend, in part, on the sum of individuals susceptible to a heat-related outcome. As such, there may be differences in the heat-health relationship between populations dependent on the numbers of susceptible individuals. Susceptibility factors were assessed to determine if each factor modified the heat-health relationship. These factors were identified via a literature search and are summarized below.

Age: In addition to the biologic effects of aging (e.g., chronic conditions, reduced ability to thermoregulate), older individuals may be at higher risk of heat-related outcomes due to social isolation or the inability to employ behavioral modification due to immobility [53, 160, 238, 284]. Children less than five years of ager are biologically less able to tolerate the heat than adults or employ individual behavioral modifications [1, 2]. Finally, teenagers and working adults may have increased exertional heat-related outcomes due to their activities. For this analysis, three factors for age were obtained from the 2007–2011 ACS 5-year estimates; the percent of the zip code-station group less than age five years, age 15 to 44 years, and age 65 years or older.

Race/ethnicity: In the literature, race and ethnicity have been identified as modifiers of the heat-health relationship [9, 64, 81, 87, 145, 218]. However, these differences are most likely due to other factors such as socio-economic status, access to resources (e.g., amenities, medical care, housing quality), the racial/ethnic distribution within occupations (e.g., outdoor versus indoor work), and the distribution of chronic conditions that may put an individual at higher risk of HRI such as chronic heart disease or diabetes [7, 171, 197-199, 221]. Race and ethnicity were included in this analysis with the understanding that they represent a complex system of cultural and social factors that are not otherwise captured. Racial and ethnic information was obtained from the 2007–2011 ACS 5-year estimates. Three factors were obtained for race: percent of the zip code-station group who reported their race as White alone, Black alone, or other (i.e., those who were not White alone or Black alone were categorized as other race). The percent of the zip code-station group who reported their ethnicity as Hispanic was also included as a factor.

Living alone: Individuals who are socially isolated have been noted in previous studies to be at higher risk of heat-related outcomes, especially older males [7, 202, 238]. This may be due in part to no one being around who might notice the HRI symptoms. Additionally, what isolates an individual (e.g., limited mobility, cognitive impairment, substance abuse) may also put them at higher risk of HRI or a heat-related outcome. The percent of the zip code-station group identified as male house-holder age 65 years or older living alone was extracted from the 2007–2011 ACS 5-year estimates.

Renter: Renters may represent a transient segment of the population who may not be as socially connected as non-renters [351, 352]. Renter status may also be a proxy for socio-economic status while the type of house (e.g., multi-family apartments or mobile homes) may put renters at higher risk of heat-related outcomes [98, 295, 353, 354]. The proportion of renter occupied houses within a zip code-station group was obtained from the 2007–2011 ACS 5-year estimates.

Non-English speaker: Individuals who do not speak English or do not speak English well may be isolated in their community (depending on the proportion of non-English speakers) leading to a higher risk of a heat-related outcomes due to no one noticing their illness, not being able to effectively access medical care, or not understanding the English-only messaging information about how to prevent HRI [7, 353, 355]. The percent of the population for each zip code-station group age five years or older who speak a language other than English at home and who speak English less than "very well" was pulled from the 2007–2011 ACS 5 year estimates.

Poverty: The socio-economic status of an individual may affect their ability to prevent a heat-related outcome (e.g., lack of air conditioning or poor housing conditions) or reduce the severity of the outcome (e.g., access to medical care). Further, the proportion of the population in poverty may also affect an individual's risk of HRI. A poor person in a rich community may have access to medical care, reduced exposure due to availability of green space, and possibly public transportation to a cooler environment. Conversely, a well-to do person in a poor community may not have access to any of the aforementioned resources. Therefore the heat-health relationship may vary by the average socio-economic status of a population. The percent of the zip code-station group residing below the poverty line was obtained from the 2007–2011 ACS 5-year estimates.

High-risk worker: Dependent on their job, workers may be at a higher risk for a HRI due to their work environment (i.e., hot/humid), clothing type, protective equipment, and an inability to self-regulate their work-rate (i.e., individual behavioral modification). At-risk occupations may include outdoor laborers, emergency responders, or individuals who work in hot environments (e.g., factory or mine). As the proportion of high risk workers within a population increases the magnitude of the heat-health relationship is also hypothesized to increase. The proportion of workers at high-risk of HRI by

zip code station-group was obtained from the 2007–2011 ACS. As very little epidemiologic information is available on which industries and occupations are at high risk for a HRI, this analysis will use broad occupational categories based on Washington State workers' compensation data [195]. This will allow for the inclusion of as many high-risk occupations as possible. High-risk occupations were defined as: Protective services (SOC 33), Food preparation and serving (SOC 35), Building grounds and cleaning maintenance (SOC 37), Natural resource, construction, maintenance (SOC 45, 47, and 49), Production, transportation, material moving (SOC 51 and 53). In addition, in case the high-risk worker factor was too broad, a factor capturing only the proportion of workers in natural resource, construction and maintenance occupations (SOC 45, 47, 49) was also included. This latter factor is based on the occupations with the highest rates of workers' compensation claims in the 2000-2007 study of occupational HRI injuries in Washington state [147]: Farming, fishing, and forestry occupations (SOC 45); Construction and extraction occupations (SOC 47); and Installation, maintenance, and repair occupations (SOC 49).

Latitude: The rate of HRI appears to be highest in the northern part of Florida and decreases with latitude (see Chapter 5) while the heat index is highest in the southern part of the state. As such, populations in higher latitudes may be less adapted to the heat and therefore more susceptible to a heat-related outcome as heat increases than populations in lower latitudes. Additionally, the type of heat may differ by latitude. For instance, the southern part of the state has the benefit of cooling afternoon sea breezes which also increase the humidity [339]. Populations in the different areas of the state may therefore feel the heat differently and react to the heat differently resulting in a differing burden of heat-related outcomes. For this analysis the latitude of the weather station for each zip code-station group was used.

Land cover: The percent of impervious surfaces is an environmental factor that directly impacts the temperature and therefore the heat-index. As the proportion of impervious surfaces increase the surrounding temperature also increases. Within urban areas the magnitude of the heat island effect may be mitigated by an increased proportion of pervious surfaces (e.g., trees and vegetation). However, it is not a simple characterization of urban a hotter and rural areas as cooler.

Crop lands may present a unique micro-climate due to the increased humidity related to irrigation of crops. Further, individuals working on crop lands may have an additional risk of HRI due to their outdoor work environment. There may be a difference in the susceptibility to heat-related outcomes dependent on the proportion of different types of land cover within their community.

Information on the proportion of impervious surfaces, cultivated crop lands, and forested lands were obtained from the 2006 and 2011 National Land Cover Database (NLCD) created by the Multi-Resolution Land Characteristics Consortium (MRLC)[58] [356]. The dataset is primarily based on 2006 and 2011 satellite images. The dataset applies a 16-class land cover classification scheme at a resolution of 30 meters for the entire U.S. Each of the 16 categories is represented by a colored pixel which represents a 30 meter x 30 meter area. Four of the categories relate to imperviousness (<20%, 20-<50%, 50-<80%, >80%), while the rest of the categories are related to type of vegetation (i.e., forest, shrubland, planted/cultivated, and herbaceous) and environmental land cover (i.e., water, wetlands, and barren). The MRLC has also created a raster layer that estimates the percent imperviousness by pixel for the entire U.S. Using ArcGIS 10.3.1, a variable was calculated which contained the sum of each pixel color within a zip code (e.g., 500 pink pixels). The percent of cultivated crop land was calculated for the 2006 and 2011 data as the number of cultivated crop land pixels divided by the total number of pixels for each zip code-station group. Each forest coverage pixel represented an area with greater than 20 percent of total vegetation cover for deciduous, evergreen, or mixed forest. For this analysis, forest coverage was not stratified by type and the percent forest coverage was calculated in the same manner as cultivated crop lands. For impervious surfaces the equation was $\sum_{k=0}^{100} C_k \times k \Big/ \sum_{k=0}^{100} C_k$ where C_k is the number of pixels within each zip code-station group that fell within the k^{th} percent category (e.g., 10% impervious surface). The average of

[58]A consortium of U.S. federal institutions such as U.S. Geological Survey (USGS), Environmental Protection Agency (EPA), National Oceanic and Atmospheric Administration (NOAA), the U.S. Forest Service (USFS), National Atmospheric and Space Administration (NASA) and the Bureau of Land Management (BLM), National Park Service, U.S. Fish and Wildlife Service (USFWS), National Aeronautics and Space Administration (NASA), Office of Surface Mining (OSM), and the Natural Resource Conservation Service (NRCS).

the 2006 and 2011 percent impervious surfaces, forested lands, and cultivated crop lands by zip code-station group was used in the analysis.

Rural/Urban status: There may be a difference in the susceptibility to heat-related outcomes of rural versus urban populations in how they are able to react or adapt to heat due to available resources (e.g., differential distribution of medical care, air conditioning usage, or outdoor activities). For instance, a non-densely populated area connected to an urban center (e.g., via roadways) may have greater access to medical care than a non-densely populated isolated area.

Information on the rural/urban status of zip codes was obtained from a dataset created by the WWAMI[59] Rural Health Research Center (RHRC) at the University of Washington (http://depts.washington.edu/uwruca/). The WWAMI RHRC created a detailed list of 33 codes that can be used individually or in aggregation to define the rural/urban status of an area (e.g., metropolitan area core, micropolitan low commuting, small town high commuting) within the U.S. [357]. The group used the 2000 U.S. Census work commuting information and the U.S. Census Bureau's definition of urbanized areas and urban clusters to create the classification system. The codes were originally assigned at the census tract level and the results were aggregated to the 2006 zip code level. For this project the 33 codes were aggregated into three categories (urban, micropolitan, small/isolated rural town) one of which was assigned to each zip code. The mode of the zip code designations within each zip code-station group was used for the meta-regression analysis.

PARAMETERIZATION OF MODIFICATION VARIABLES

All factors, except for rural/urban status, were originally modeled as both categorical and linear. For each factor four quartile categories were created with the third category, median to <upper quartile, being the reference group. When modeled as linear, the factors were first mean centered. For rural/urban status, there were three categories: urban, micropolitian, and rural with urban status as the reference.

[59]Washington, Wyoming, Alaska, Montana, and Idaho

STATISTICAL ANALYSIS

A generalized linear model (GLM) with an overdispersed Poisson distribution was used for all analyses and is similar to the methodology used in Chapter 6. The unit of analysis was the zip code-day. To allow for variation in the heat–HRI morbidity relationship, the model was run for each zip code-station group. Note that the zip code-station group created for this analysis was the smallest geographic area for which the models would converge. Random effects meta-regression was used to pool the group-level results to obtain statewide estimates [318, 319]. Similar to Equation 6-1, the algebraic representation of the model used for each zip code-station group is: $log[E(Y_i)] = \log(py) + \alpha + \sum_{j=1}^{p} g_j(x_{ij}) + m(\cdot)$. Where Y_i is the daily HRI morbidity, *py* is the person years, function *g* includes the multiple terms (j=1 to p) for temporal confounding (see below), and the function $m(\cdot)$ is the exposure term defined above. The offset term used to estimate the HRI rates are the natural log of the population data obtained from the ACS. Temporal confounding was addressed with the inclusion of three terms: an indicator term for year, an indicator term for day of week, and a restricted cubic spline function for day of year with 5 knots located at day 151, 181, 212, 243, and 273. The knot locations approximated the last day of the month for May, June, July, August, and September. The reference category for the indicator terms were the category with the largest number of HRI cases; year = 2010, month = August, and weekday = Saturday for non-work-related HRI and Wednesday for work-related HRI.

Meta-regression was used to evaluate modification of the heat–HRI morbidity relationship. Within each factor the Akaike information criterion (AIC) was used to determine the parameterization of the factor (e.g., linear or categorical). The model with the smallest value was chosen unless the difference in AIC between the two models was less than two (i.e., $|\mathrm{AIC}_1 - \mathrm{AIC}_2| < 2$) then the model with the simplest parameterization was chosen [315]. If the best fitting model indicated a categorical parameterization the effect estimates within categories were compared to determine if any of the categories could be collapsed. If so, the reduced model was used. Identification of factors which modified the heat–HRI morbidity relationship was done using the likelihood ratio test (LRT). The meta-regression model with each factor as the independent variable (i.e., meta-predictor) was

compared to the intercept only meta-regression model where the *a-priori* criterion for modification was a p-value of 0.05. The I^2 value and the Q-statistic were calculated as a measure of the amount of heterogeneity [318, 358]. The magnitude of modification was assessed for each of the identified factors.

A sensitivity analysis was conducted to determine if the heat–HRI morbidity relationship varied by morbidity data source (i.e., ED visits versus hospitalizations). The results indicated that modification of the heat–HRI morbidity relationship by data source was not present (Appendix D.). As such, the analysis was not stratified by data source.

SOFTWARE

Data management and linkage was done using SAS 9.3. Statistical analysis and creation of figures (except maps) were done in the statistical package R version 3.2.3 [320-326]. Maps were created using ArcGIS 10.3.1.

RESULTS

Among Florida residents during the warm season for 2005–2012 there was a 69.8 percent increase in HRI for every 5°F increase in heat index below the breakpoint (101°F) and 49.1 percent increase above the breakpoint (Below: 95% CI = 1.66, 1.74; Above: 95% CI = 1.4, 1.5). For the rest of the paper the relative increase in HRI for every 5°F increase in heat-index will be referred to as the heat-HRI morbidity relationship.

Prior to assessing modification, the between group variation in the 103 exposure-response curve estimates were explored. There was a small amount of heterogeneity in the exposure-response curve estimates between the 103 zip code-station groups (I^2 = 25.7; Q = 274.6, degrees of freedom [df] = 204, p-value = 0.0007). As expected in accordance with the greater amount of information (e.g., HRI cases, study days), the variance in the distribution of estimates for the natural log of the slope below the breakpoint (mean = 0.1; standard deviation [sd] = 0.03; interquartile range [IQR] = 0.04) was smaller than the variance in the distribution of estimates for the natural log of the slope above the

breakpoint (mean = 0.07; sd =0.06; IQR = 0.05). The inclusion of the intercept of the exposure-response curve explained some of the variation in the heat-HRI morbidity relationship (I^2 = 21.7; Q = 390.07, df = 306, p-value = 0.0008).

Basic statistics describing the distribution of each susceptibility factor (e.g., mean, IQR, minimum, maximum) can be found in Table 7-1. Eight factors met the *a-priori* criterion for modification (Table 7-2). The magnitude of modification for the eight factors which met the modification criterion can be found in Table 7-3 a and b. Within each factor, modification was observed between some but not all categories. Additionally, dependent on the factor, modification of the heat-HRI morbidity relationship was not the same for the entire exposure-response curve. The geographical distribution of the zip code station groups for each factor stratified by the analysis category (e.g., quartile) can be found in Figure 7-1 and Figure 7-2. For instance, in Figure 7-1c the zip-code station groups with the largest proportion of renters is shaded brown while the zip-code station groups with the smallest proportion of renters is shaded yellow.

Modification of the heat-health relationship could have been observed in a number of forms which are described here. Below (or above) the breakpoint the heat-HRI morbidity relationship (i.e., the slope of the exposure-response curve) could vary by factor category. For instance, the average heat-HRI morbidity relationship for the zip code-station groups in quartile one could be stronger than the average heat-HRI morbidity relationship for the zip code-stations groups in quartile three. Both above and below the breakpoint the heat-HRI morbidity relationship could vary by factor category. The heat-HRI morbidity relationship does not vary but the absolute rates (i.e., the intercept of the exposure-response curve) do vary by factor category. The entire exposure-response curve does not vary by factor category. Within this study, the authors did not differentiate between the latter two forms, no modification of the exposure-response curve and no modification of the heat-HRI morbidity relationship. For those factors which met the *a-priori* criterion for modification, the form and magnitude of modification for each factor are summarized below.

In order to provide a comparison of modification across the linear parametrized factors, an estimate of the slope of the heat-HRI morbidity relationship was calculated at three single point

values; the mean and plus/minus one standard deviation. For both of the linear parameterized factors, percent of the population age 15 to 44 years and Black race alone, there was an overlap in confidence intervals (Table 7-3 a). However, there was a 12 percent increase in the heat-HRI morbidity relationship above the breakpoint when the percent of the population reported being of Black race alone was 30.5 percent, that is 2 standard deviations above the mean (RR =1.6; 95% CI= 1.5, 1.7), compared to 4.6 percent of the population reporting Black race alone, that is 1 standard deviation below the mean (RR=1.4; 95% CI=1.4, 1.5).

The percent of the population reporting other race on the census was modeled as binary (above and below the median) with the reference group being 1.8 to < 6.7 percent of the population. Below the breakpoint, there was a six percent increase in the heat-HRI morbidity relationship for areas with 6.7 to 20.7 percent of the population reporting other race compared to the reference group. Above the breakpoint, a statistical but non substantial difference was observed with the non-reference having a one percent decrease in the heat-HRI morbidity relationship compared to the reference group.

The percent of the houses which were renter occupied was modeled as binary (above and below the median) with the larger category, 25.8 to 55.4 percent, being compared to the smaller category, 8.5 to <25.8 percent. Below the breakpoint there was an eight percent increase in the heat-HRI morbidity relationship while above the breakpoint there was a three percent decrease in the heat-HRI morbidity relationship.

The percent of the population age five years or older who spoke a language other than English at home and who spoke English less than “very well” was modeled as categorical. Below the breakpoint there was an 11 percent decrease in the heat-HRI morbidity relationship for the second quartile, 2.7 to <4.7 percent, compared to the third quartile, 4.7 to 10.0 percent, while above the breakpoint no difference was observed.

The percent of the population employed in the high risk natural resources, construction, and maintenance occupations within a zip code-station group was modeled as categorical. The weakest

heat-HR morbidity relationship both below and above the breakpoint was observed for the second quartile, 9.0 to <11.6 percent.

The percent of impervious surfaces within a zip code-station group was modeled as categorical with the first and second quartiles being combined into one category. Below the breakpoint, there was a 10 relative increase in the heat-HRI morbidity relationship for areas in the fourth quartile, 9.7 to 42.8 percent, compared to areas in the third quartile, 3.9 to <9.7 percent. There was a reversal in the relative difference above the breakpoint; areas in the fourth quartile for impervious surfaces had a three percent decrease in the heat-HRI morbidity relationship compared to areas in the third quartile of impervious surfaces.

The percent of an area with 20 percent or more of forested land was modeled as categorical. The reference category was the third quartile, 7.5 to <27.7. Below the breakpoint differences between the other quartile categories and the reference category was observed. Above the breakpoint, there was a nine percent increase in the heat-HRI morbidity relationship for areas with 27.7 to 50.3 percent forested land (i.e., fourth quartile) compared with the third quartile. A seven percent decrease in the heat-HRI morbidity relationship was also observed for areas with 0.01 to <2.01 percent forested lands (i.e., first quartile) compared with the third quartile.

DISCUSSION

The purpose of this study was to identify susceptibility factors which may modify the relationship between ambient outdoor heat and HRI across Florida. The mapping and geographical identification of areas vulnerable to heat-related outcomes as heat increases are being used to target heat-related planning, prevention, and intervention activities [202, 353, 359]. However, it is important to assess if these factors defined at the community-level are actually related to increases in the heat-health relationship. For instance, a study using data from five U.S. states for the years 2000-2007 examined the effectiveness of a heat vulnerability index (HVI) and found that the index identified areas of health vulnerability but not necessarily areas vulnerable to heat [360]. Therefore, for this study, a community-level approach was used to identifying modifiers of the heat-health relationship.

The potential modifiers were selected based on the literature and prior incorporation into an HVI or heat vulnerability assessment [9, 353, 361]. HVI and health vulnerability assessments are applied public health tools used for planning purposes to identify populations who are vulnerable (or resilient/adapted) to a particular hazard. After identification of these vulnerable populations, the effect disparity can potentially be mitigated. This analysis identified factors which can be incorporated into the current public health heat vulnerability assessments in Florida. To the best of our knowledge, this is the first study to assess factors which may modify the heat-health relationship among Florida residents and one of a handful of studies conducted in the southeastern U.S. [291, 295].

Each potential modifier was assessed one at a time, within the analysis. However, as alluded to when defining these factors in the methods section, they are not independently associated with a population's heat vulnerability and are mostly likely highly interrelated. A population's heat vulnerability, and the resulting morbidity and mortality, is affected by of a complex network of factors [7]. Further research examining the social and geographical make-up of the areas needs to be considered. For instance within this study, above the breakpoint of 101°F, the zip code-station groups with the highest percentage of forested land had the largest effect estimate for the relative increase in the HRI rates as heat index increases. Geographically speaking, the zip code-station groups that fell within this category are located in the northern part of Florida away from coast. In the summer, these areas can be sweltering without the benefit of cooling sea breezes. Further, much of Florida's timberlands are found in the northern part of the state [362]. Therefore, the discussion below is somewhat limited in the sense that the interrelatedness of these factors is not truly addressed. However, it is a much needed starting point for addressing population level vulnerability by examining the heat-HRI morbidity relationship in a humid subtropical climate.

Above the breakpoint of 101°F, the factor Black race alone modified the heat-HRI morbidity relationship. However, a significant difference in the magnitude of effect was only observed when the percent of the population reporting Black race alone was 30 percent (i.e., 2 standard deviations above the mean). This may be due to the geographic unit of analysis inappropriately averaging across each zip code-station group. By zip code, the factor ranges from 0 to 94.8 percent of the population

reporting Black race alone (mean = 13.6; sd = 16.2) while for zip code-station group the range is 0.4 to 44.3 percent. Therefore, in Florida, when the heat index is greater than 101°F, the relative increase in rates as heat index increases may be greatest in areas where the percentage of the zip code population reported as Black race alone is greater than 30 percent.

Within this analysis both percent of the population reporting other race and percent renter occupied houses were treated as binary. For both factors, below the breakpoint of 101°F, the largest effect estimate for the heat-HRI morbidity relationship was observed in the largest category. For other race, the distribution of the factor by a smaller geographic area, zip code (mean = 6.8; sd = 4.5; min = 0; max = 31.2), was similar to the distribution of the factor by the geographic area used in the analysis, zip code-station group. The distribution for the percent renter occupied houses by zip code also had a similar distribution as the zip code-station group, although the distribution by zip code had a larger right skew (mean = 28.6, sd = 16.5; min =0; max = 100). As such, it may also be possible to use both factors and corresponding cut-points (i.e., the median) for identification of populations which are vulnerable to HRI morbidity as heat increases at a smaller geographic level.

Areas with a small percentage of the population reporting speaking English less than ‘very well’ were assumed to be linguistically isolated and expected to have a higher relative rates of HRI as heat index increased [283, 353]. However, the specific percentage value that should be used to categorize a population as linguistically isolated was unclear. The zip code-station groups with the largest percentage of individuals having reported speaking English less than ‘very well’ were found in south Florida. In Florida overall, 27 percent of the population speaks a language other than English at home with 73 percent of those speaking Spanish [363]. Further, there is a large Hispanic population in south Florida (64.5% of population in Miami-Dade County) resulting in a large bilingual population [300, 301]. Therefore, we may assume that much of the population in south Florida (and the population within the highest category for the factor speaks English less than ‘very well’) are not linguistically isolated.

Below the breakpoint of 101°F, the smallest effect estimate for the heat-HRI morbidity relationship was found within the second quartile category of the factor, speaks English less than

‘very well’. These results were unexpected as this group was assumed to be linguistically isolated. However, it is possible that the population who speaks English less than ‘very well’ is not evenly distributed across the zip code-station group but instead is clustered within neighborhoods and therefore is not linguistically isolated. Further, within the state of Florida many of the public health messaging is in English and Spanish. As a result the use of percent of the population reporting speaking English less than ‘very well’ may be inappropriate. Investigation into why this area has the lowest effect estimates should be conducted. Additionally, it may be useful to restrict the factor by excluding those who speak Spanish at home.

The zip code-station groups with the largest proportion of impervious surfaces were found in the urban areas. Below a heat index of 101°F, the strongest relationship between HRI rates and heat was found in the areas with the largest proportion of impervious surfaces. Within the literature, urban areas, which have high proportions of impervious surfaces, have higher rates of heat-related outcomes as heat increases due to the urban heat island effect where the temperature in urban areas is higher than surrounding areas [6, 39, 77, 204, 208, 236, 240]. Therefore, it may be appropriate in Florida to identify vulnerable populations by mapping areas with a high proportion of impervious surfaces. However, as noted in prior studies, at a smaller geographical resolution, areas with a higher percentage of impervious surfaces may be commercial properties [353]. Therefore, the combination of percent impervious surfaces and housing density may more aptly identify populations vulnerable to heat-related outcomes as heat increases. Above a heat index value of 101°F, the areas with the largest proportion of impervious surfaces had the weakest association between HRI rates and increasing heat. This may suggest the occurrence of some form of HRI adaptation or behavioral modification. Further investigation into the potential resiliency at high heat values of Florida populations with a large proportion of impervious surfaces is required.

The heat-HRI morbidity relationship was weaker in the second quartile for high risk workers (i.e., the natural resource, construction, maintenance occupations) compared with the other quartile groups. This result was unexpected as it was hypothesized that the first quartile would also have a weaker association or similar association to the second quartile category for the heat-HRI morbidity

relationship. Examining the map in Figure 7-2, the zip code-station groups that fall within the factor's first quartile mostly include the urban centers of the state. And therefore this factor category may be a proxy for urban areas and the urban heat island effect. As such, this factor, percent high risk workers, may only be useful in identifying populations vulnerable to HRI as heat increases in non-urban settings. However, further exploration of this factor is necessary.

A small amount of variation in the estimates of exposure-response curve was observed across the zip code-station groups. These results indicate that, in general, the relative increase in rates as maximum daily heat index increases is similar across the state. Some of the variation was explained by the rates, suggesting that differing factors across the state may be mitigating (or increasing) actual individual heat exposure. Further, modification of the relationship between HRI and maximum daily heat index by data source (ED visits versus hospitalizations) was not seen, although, variation in the rates of HRI by data source was observed. Therefore, while identification of factors that modify the heat-HRI morbidity relationship is important, in order to reduce the burden of HRI, it is essential to identify factors which affect the magnitude of average HRI rates.

LIMITATIONS

Within this analysis the use of quartile groups was an efficient way to categorize the data from each of the susceptibility factors in order to prevent the assumption of linearity. However, it is possible that disparate groups were combined creating bins which inappropriately assumed the same exposure effect within the bin, resulting in modification not being detected or an attenuation of the detected effect. Additionally, while it is unclear of the clinical or biologic relevance of the quartile cut-points they do provide a distinct way to identify geographically areas that may be at higher or lower risk of HRI as heat index increase in order to implement targeted HRI prevention or intervention activities.

The meta-regression methodology employed in this analysis examined the variation between zip code-station groups. The geographic level used was the smallest unit for which the exposure-response curve could be individually estimated. This was done in order to minimize the area

assumed, for this analysis, to be homogenous. However, the averaging of each susceptibility factor across the zip code-station groups may have eliminated much of variation across the state that would have been observed at a smaller geographic level. As such, there may be factors in this study for which modification was not observed or where modification was observed but the magnitude was attenuated. While this analysis informed modification of the heat-HRI morbidity relationship across Florida, it would be beneficial, for intervention and prevention planning purposes, to examine modification of the heat-HRI morbidity relationship for smaller areas such as individual communities.

This analysis only looked at modifiers of the heat-health relationship by community-level factors which were comprised of individual level susceptibility (e.g., elderly individuals are more susceptible to heat therefore a population with a large proportion of elderly is vulnerable to heat). The assumption for each community-level factor was that at the individual level the factor modified the heat-health relationship making an individual more susceptible to increasing heat. However, this assumption may be invalid and the individual-level factor does not modify the heat-health relationship. As a result, the community-level factors that were analyzed in this study may represent a completely different set of individual-level susceptibility factors than previously thought, potentially invalidating some of discussion above. For instance, if the heat-HRI morbidity relationship is not modified by individuals who are linguistically isolated then zip codes-station groups that fall within the second quartile of the factor speaks English less than ‘very well’ may be associated other factor(s) that result in the weaker heat-HRI morbidity relationship as compared to the third quartile. It may therefore be irrelevant if those who speak English less than ‘very well’ are (or are not) clustered in neighborhoods. This unknown susceptibility factor may be more effective than the factor speaks English less than ‘very well’ at identifying populations affected by increasing heat. Finally, modification (or lack of modification) at the community-level does not indicate susceptibility to increasing heat for an individual group (e.g., those age 15-44). An analysis of modification of the heat-health relationship by individual-level susceptibility factors should be conducted.

Finally, it is possible that within zip code-station groups the number of HRI cases were too small for modification to be observed or for statistically significant differences to be observed within

the factor categories. This latter point is especially true for modification above the breakpoint where there were fewer cases at higher heat index values across the state.

CONCLUSION

Eight factors were observed to modify the relationship between daily HRI and maximum daily heat index. The eight community-level factors were percent of the population who: were age 15-44 years, reported black race alone, reported other race, resided in a renter occupied house, spoke English less than ‘very well’, had a high HRI risk job, percentage impervious surfaces, and percent forested land. However, for the majority of the factors the dose-response relationship was non-linear and statistical or substantial differences were only observed for some but not all categories. Florida is a diverse state and as such the impact of each of the chosen factors on the heat-HRI morbidity relationship may be different in different parts of the state (e.g., south Florida versus north Florida). The results of these analyses should therefore be used to target geographical areas for further sub-area analysis. Additionally, in order to reduce the burden of HRI in Florida there should be a strong focus on factors which explain the variation in average HRI rates.

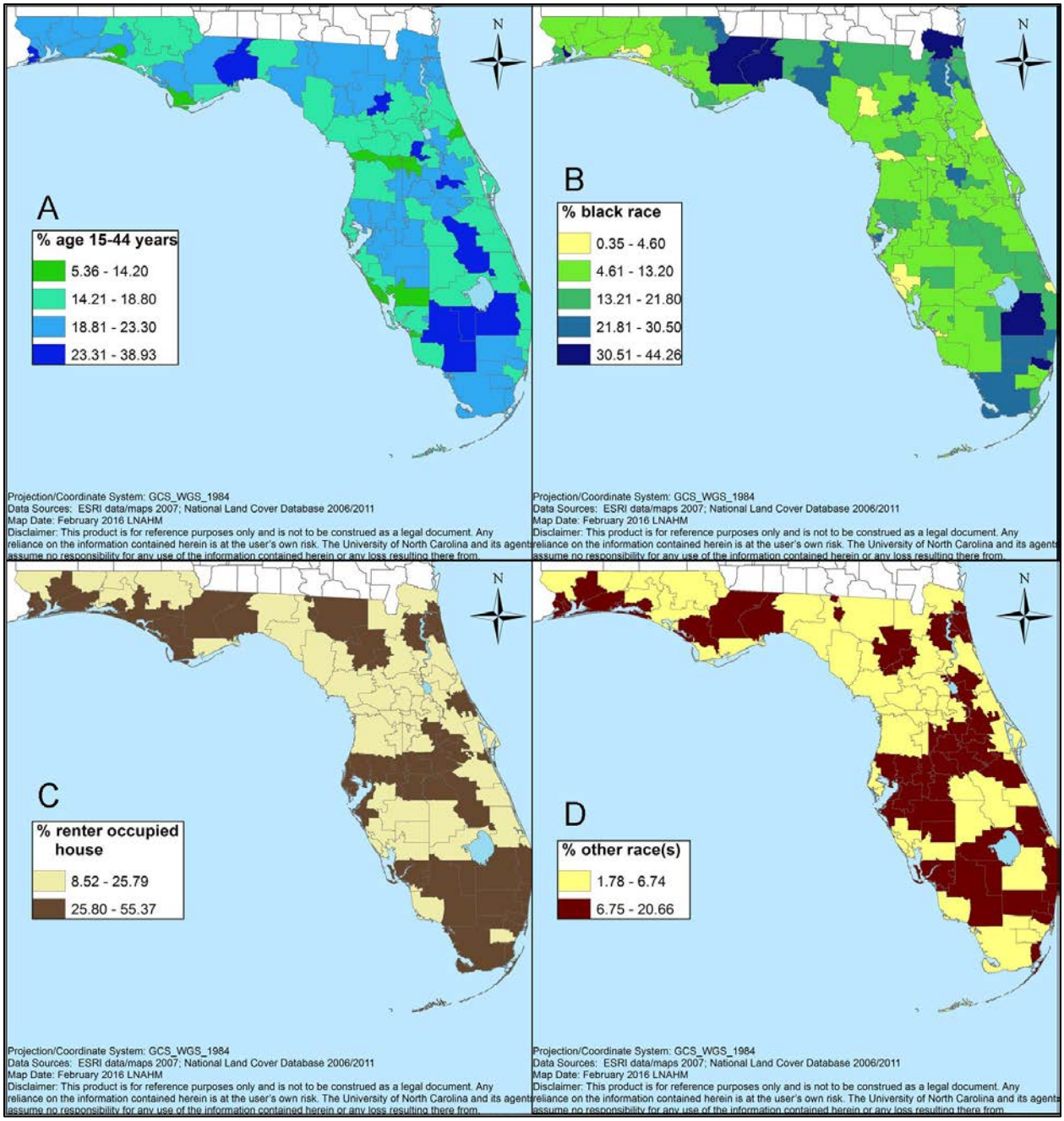


Figure 7-1. Factor specific maps with each factor stratified by the analysis categories where the geographic area is the zip code-station group: A) percent of the area population age 15-44 years, B) percent of the population who reported Black race alone, C) percent of houses which were renter occupied, D) percent of the population who reported other race.

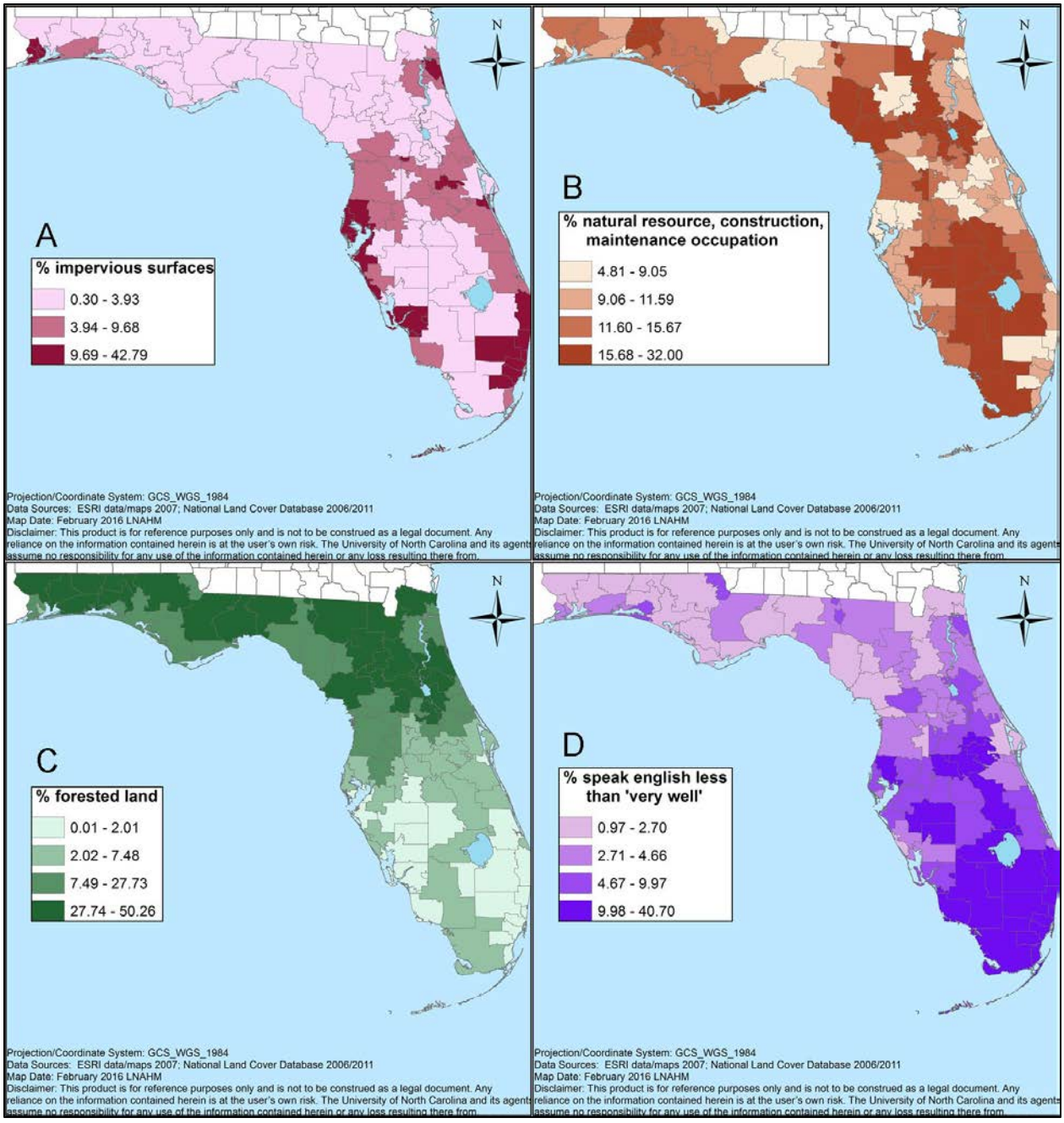


Figure 7-2. Factor specific maps with each factor stratified by the analysis categories where the geographic area is the zip code-station group: A) percent impervious surfaces in the area, B) percent of the population who reported natural resource, construction, maintenance occupation, C) percent forested land in the area, D) percent of the population who speaks another language other than English at home who also speak English less than 'very well'.

Table 7-1. Summary statistics related to the distribution of each the susceptibility factors

Susceptibility Factors	Mean (sd)	Minimum	Lower Quartile	Median	Upper Quartile	Maximum
% age <5 years	5.7 (1.25)	2.1	5.1	5.8	6.5	9.9
% age 15-44 years	18.8 (4.56)	5.4	16.7	18.8	21.1	38.9
% age 65 years or older	27.6 (3.37)	18.2	25.4	27.5	29.3	37.8
% one race: White alone	79.6 (9.67)	48.4	73.9	81.1	86.1	96.8
% one race: Black alone	13.2 (8.62)	0.4	7.9	11.1	16.7	44.3
% other race(s)	7.2 (3.50)	1.8	4.7	6.7	8.3	20.7
% Hispanic/Latino	15.0 (14.60)	1.5	5.3	8.9	19.5	75.4
% male house-holder age 65+ living alone	7.4 (2.69)	2.9	5.3	7.2	9.0	19.0
% renter occupied houses	27.2 (8.52)	8.5	21.6	25.8	30.9	55.4
% speak English less than ‘very well’	7.7 (7.55)	1.0	2.7	4.7	10.0	40.7
% below poverty level	15.7 (5.83)	5.2	12.0	14.2	19.1	30.9
% high heat risk occupation	37.7 (8.15)	21.8	31.2	36.5	42.1	58.9
% natural resource, construction, maintenance occupations	12.8 (5.19)	4.8	9.1	11.6	15.7	32.0
Latitude	28.6 (1.62)	24.6	27.4	28.7	30.07	30.9
% impervious surfaces	25.1 (23.21)	0.3	1.1	3.9	9.7	42.8
% cultivated crops	6.4 (9.79)	0.0	0.5	2.7	8.9	62.9
% forested land	15.4 (14.93)	0.01	2.0	7.5	27.73	50.3
Rural/urban status	Urban = 79 zip code-station groups; Micropolitan = 8 zip code-station groups; Rural = 16 zip code-station groups					

Table 7-2. Identification of population-level factors which modify the heat-health slope via comparison of factor-specific meta-regression models to an intercept only model

Meta-predictor†	Factor Parametrization‡	I^2	AIC	Log Likelihood	LRT (df)	LRT p-value	
Intercept only		25.697	-792.503	401.252			
% age <5 years	Linear	25.906	-790.555	402.278	2.05 (2)	0.4	
% age 15-44 years	Linear	23.603	-794.496	404.248	5.99 (2)	0.05	*
% age 65 years or older	Linear	24.989	-792.247	403.124	3.74 (2)	0.2	
% one race: White alone	Linear	24.092	-794.066	404.033	5.56 (2)	0.06	
% one race: Black alone	Linear	23.919	-795.053	404.526	6.55 (2)	0.04	*
% other race(s)	Binary	21.915	-797.997	405.998	9.49 (2)	0.01	*
% Hispanic/Latino	Linear	25.654	-791.026	402.513	2.52 (2)	0.3	
% male house-holder age 65+ living alone	4 categories	25.660	-786.274	404.137	5.77 (6)	0.5	
% renter occupied houses	Binary	21.672	-797.146	405.573	8.64 (2)	0.01	*
% speak English less than 'very well'	4 categories	21.376	-794.123	408.062	13.62 (6)	0.03	*
% below poverty level	Linear	24.796	-792.363	403.182	3.86 (2)	0.2	
% high heat risk occupation	4 categories	23.123	-790.870	406.435	10.37 (6)	0.1	
% natural resource, construction, maintenance occupations	4 categories	21.058	-794.635	408.318	14.13 (6)	0.03	*
Latitude	Linear	24.616	-793.807	403.904	5.3 (2)	0.07	
% impervious surfaces	3 categories	20.757	-797.712	407.856	13.21 (4)	0.01	*
% cultivated crops	4 categories	23.220	-789.978	405.989	9.48 (6)	0.2	
% forested land	Linear	22.773	-799.327	406.664	10.82 (2)	0.004	*
Rural/urban	3 categories	25.240	-790.476	404.238	5.97 (4)	0.2	

*Meta-predictors which met the *a-priori* definition of p-value less than 0.05. Note p-values are rounded to the first significant digit.
†Only the best fitting models for each factor are presented.
‡Parameterization in 4 categories was divided by quartile: minimum - <lowest quartile, lowest quartile - <median, median-<upper quartile, upper quartile to maximum. Parameterization in 3 categories combined the two lowest categories into one: minimum - <median, median-<upper quartile, upper quartile to maximum. Binary parameterization split the data into below the median and above the median.

Table 7-3a. Comparison of the magnitude of modification for the heat–HRI morbidity relationship within each susceptibility factor stratified by the linear components of the exposure-response curve.

Factor	Percent of population	Below the breakpoint		Above the breakpoint	
		RR for 5°F increase (95% CI)	Ratio of RRs*	RR for 5°F degree (95% CI)	Ratio of RRs*
% age 15-44 years	14.2 (-sd)	1.67 (1.61, 1.73)		1.44 (1.37, 1.51)	
	18.8 (mean)	1.70 (1.65, 1.74)	1.035	1.49 (1.44, 1.54)	1.065
	23.3 (+sd)	1.73 (1.67, 1.79)		1.54 (1.47, 1.61)	
% one race: Black alone	4.6 (-sd)	1.68 (1.62, 1.75)		1.42 (1.35, 1.50)	
	13.2 (mean)	1.70 (1.65, 1.74)	1.017	1.48 (1.43, 1.53)	1.083
	21.8 (+sd)	1.71 (1.66, 1.77)		1.54 (1.48, 1.61)	
% other race(s)	1.78 - <6.74	1.63 (1.57, 1.69)	1	1.50 (1.43, 1.58)	1
	6.74 - 20.66	1.76 (1.70, 1.81)	1.080	1.48 (1.41, 1.55)	0.985
% renter occupied houses	8.52 - < 25.8	1.63 (1.57, 1.69)	1	1.52 (1.44, 1.60)	1
	25.8 - 55.37	1.75 (1.70, 1.80)	1.076	1.47 (1.41, 1.54)	0.970
% speak English less than ‘very well’	0.97 - < 2.7	1.70 (1.61, 1.79)	0.958	1.55 (1.46, 1.66)	1.038
	2.7 - < 4.66	1.60 (1.53, 1.68)	0.904	1.48 (1.39, 1.58)	0.989
	4.66 - < 9.97	1.77 (1.70, 1.85)	1	1.50 (1.40, 1.60)	1
	9.97 - 40.7	1.73 (1.64, 1.81)	0.974	1.41 (1.30, 1.53)	0.942

*For the linear parameterized factors = RR at 1 sd above the mean/RR at 1 sd below the mean; for categorical parameterized factors = RR in category x/RR in the reference category

Table 7-3b. Comparison of the magnitude of modification for the heat–HRI morbidity relationship within each susceptibility factor stratified by the linear components of the exposure-response curve.

Factor	Percent of population	Below the breakpoint		Above the breakpoint	
		RR for 5°F increase (95% CI)	Ratio of RRs*	RR for 5°F degree (95% CI)	Ratio of RRs*
% natural resource, construction, maintenance occupations	4.81 - < 9.05	1.72 (1.65, 1.79)	0.979	1.57 (1.48, 1.67)	1.047
	9.05 - < 11.59	1.62 (1.56, 1.69)	0.924	1.42 (1.34, 1.5)	0.945
	11.59 - < 15.67	1.76 (1.68, 1.84)	1	1.5 (1.42, 1.59)	1
	15.67 - 32	1.72 (1.60, 1.86)	0.982	1.46 (1.32, 1.63)	0.973
% impervious surfaces	0.30 - <3.93	1.70 (1.64, 1.77)	1.052	1.55 (1.47, 1.64)	1.051
	3.93 - <9.68	1.62 (1.56, 1.69)	1	1.48 (1.39, 1.57)	1
	9.68-42.79	1.78 (1.71, 1.85)	1.097	1.42 (1.34, 1.51)	0.965
% forested land	0.01 - < 2.01	1.76 (1.68, 1.85)	1.033	1.39 (1.29, 1.49)	0.932
	2.01 - < 7.48	1.66 (1.58, 1.74)	0.972	1.44 (1.33, 1.55)	0.967
	7.48 - < 27.73	1.71 (1.63, 1.79)	1	1.49 (1.4, 1.58)	1
	27.73 - 50.26	1.65 (1.57, 1.73)	0.965	1.62 (1.53, 1.72)	1.091

*For categorical parameterized factors = RR in category x/RR in the reference category

CHAPTER 8. DISCUSSION

HRI and any resulting health complications are highly preventable, as such; broad public health interventions may potentially have substantial impacts on reducing the population burden of HRI and mitigating the severity of HRI impacts. In order to design effective public health interventions to accomplish this task, it is important to understand the relationship between temperature/heat index and HRI, as well as the population burden and identification of the most susceptible groups. Prior to this study this information was not available in Florida.

There were two aims addressed in this dissertation: (1) modeling of the relationship between daily ambient outdoor heat and daily HRI morbidity and mortality and (2) identification of factors that modified the relationship identified in the first aim. The first aim examined the heat-health relationship from three perspectives, ambient outdoor heat, heat waves, and an analysis that assessed the additional impact of heat waves after accounting for ambient outdoor heat. A component of these aims was the separation of HRI cases into non-work-related and work-related HRI cases. This was especially important as there is little epidemiologic research about the heat-health relationship in the civilian work-force.

AIM 1 DISCUSSION

The modeling of the relationship between ambient outdoor heat and HRI in Florida indicated, as hypothesized, a positive relationship. In other words, the average statewide rates of HRI increased with increasing heat index or temperature. The magnitude of effect for the heat-health relationship was allowed to vary below and above the breakpoint. For both non-work-related and work-related HRI the relationship between heat and HRI was strongest below the breakpoint. The weaker relationship above the breakpoint may indicate that at higher heat index (or temperature) values there may be some form of HRI prevention or adaptation that is occurring. As such, prevention

and intervention activities should include a focus on times when the heat index is equivalent to or slightly above the average value. This is especially relevant for sub-populations who due to their activities or protective gear may have increased internal metabolic heat production or a reduced physiologic ability to disperse heat (e.g., marathon runners, landscapers, or firefighters).

Heat index as opposed to temperature was determined to be a better metric of heat in Florida when examining the relationship between HRI morbidity/mortality and heat. The heat index is a metric which incorporates both humidity and temperature. Florida is the most humid state in the nation [339]. As the amount of water content of the air increases (i.e., the humidity) the temperature feels warmer to the individual and the ability of an individual to cool themselves via evaporation decreases [50]. Further, for each temperature degree, dependent on water content in the air, the heat index could vary by up to ± 16°F. Therefore, as demonstrated by the statistical models, for each temperature degree, the rates of HRI compared with the reference value can vary substantially dependent on the amount humidity. This finding has implications related to projecting the future HRI burden associated with climate change. Incorporating effect estimates based only on temperature will most likely under-estimate the projected future burden of disease.

After adjusting for ambient outdoor heat on the current and prior day, no effect on the rates of HRI were observed for additional contiguous days of high heat (i.e., heat wave). In fact, the majority of HRI cases were observed to have occurred outside of a heat wave. For public health purposes, this finding suggests that it may not be cost-effective to invest in Heat-Health Warning Systems (HHWS) in Florida. HHWS are designed to activate local response plans to mitigate the adverse heat effects during excessive heat events. In order to reduce the HRI burden and mitigate the effects within vulnerable populations, instead of focusing on heat waves, resources should be spent on targeted sub-population intervention and prevention activities in Florida and potentially the southeastern U.S..

While the focus of HRI public health activities should be shifted, there is still a benefit to episodic analysis, especially when studying other heat-related outcomes (e.g., acute renal failure or cardiovascular disease). As these other outcomes have many causes, identifying an exposure

window may be useful in understanding the burden of other heat-related outcomes. According to the results of this study, heat waves should be defined as two or more days above a relative threshold, although, a constant threshold value is also appropriate when the threshold is defined using the heat index.

The modeling of the heat-health relationship conducted in my study, including modification of the ambient outdoor heat by heat waves provides a population level understanding of the heat-health relationship in Florida which may be generalizable to other humid subtropical and tropical climates. At the state level, the results of this study may provide a more optimal and nuanced timing for interventions. Further, long term projections (e.g., 2050, 2100) of the population burden of adverse outcomes under different climate scenarios have numerous sources of uncertainty. The results of this study will help to minimize some of this uncertainty when assessing the future burden of HRI in Florida by providing morbidity and mortality estimates specific to Florida's climate and population.

AIM 2 DISCUSSION

Given that an additional heat wave effect was not observed, the relationship between maximum daily heat index and daily HRI morbidity was modeled when assessing the community-level susceptibility factors in the second aim. Susceptibility factors, included in this analysis, were identified from the literature and in prior heat vulnerability assessments [9, 353, 361]. In this manner, the goal was to evaluate if these factors identified populations vulnerable to increasing heat in Florida. Only 8 of the 18 factors evaluated were identified as modifiers of the heat-health relationship in Florida. In general, the pattern of modification within each category differed below and above the breakpoint value (i.e., heat index = 101°F). Above the breakpoint, modification was observed for six of the factors, suggesting that the potential adaptive behavior observed in the first aim may be differential by susceptibility factor. For three of the six factors, the variance of the HRI-heat dose-response relationship, within each factor category, was very large resulting in non-statistically significant results. These results may be reflective of the smaller proportion of HRI cases at the higher heat index values. Further research is required into the potential adaptive behavior of communities at high heat values is required.

The factor specific categories where the heat-health relationship was the strongest could be used to identify populations vulnerable to increasing heat. Strategies could be developed to mitigate the effect disparity within these vulnerable populations. Vulnerable populations may include communities with a high proportion of impervious surfaces, a high percentage of the population reporting Black race alone, or a high proportion of the population residing in renter occupied homes. The result of aim two highlights the importance of area specific vulnerability assessments. Additional research should evaluate the susceptibility factors at a regional or community level as the analysis methodology within this study may have obscured some modification effects. Further, research needs to be conducted examining the factors in the presence of other factors in addition to the uni-variable analysis conducted in this study.

Finally, the HRI rates appear to vary at the sub-county level and explain some of the variation in the heat-health relationship. Therefore, while the second aim may have identified populations vulnerable to increasing heat, in order to reduce the overall burden of HRI, an investigation into how the average rates differ by susceptibility factors defined at the individual-level was also conducted. The results indicated that the sub-populations with the highest rates of HRI, regardless of work-related status or data source, were males or residents of rural counties. Hispanics had lower rates of non-work-related HRI and work-related HRI ED visits as compared with non-Hispanics. As discussed in chapter five, further investigation into HRI by Hispanic origin sub-groups is necessary given the regional diversity of nativity for Hispanics in Florida. Additionally, the highest rates of HRI were found in the Florida panhandle and central Florida.

The analysis of factors that may affect a community's vulnerability to HRI as heat increases provides information for identification of vulnerable sub-populations which can be incorporated into heat vulnerability assessments and heat mitigation planning in Florida. The analysis of individual susceptibility factors can be used to target and evaluate interventions aimed at reducing the burden and severity of HRI within the Florida population. Further, the results of this study provide a solid basis for future research which may include cross-sectional studies of HRI in sub-populations (e.g., workers or the Hispanic population), exploration of the heat-health relationship in other data sets

(e.g., syndromic surveillance), or the relationship between heat and cause-specific conditions (i.e., acute renal failure) that may be associated with heat exposure.

STRENGTH AND LIMITATIONS

The greatest strength and contribution to this dissertation is the large and diverse study population which has not previously been well explored. There have only been eight studies which examined the heat-health relationship in the southeastern U.S. [198, 263, 291, 295, 336, 342, 343, 364], five of which were conducted in North Carolina [198, 263, 295, 343, 364]. Comparatively few studies have examined the heat-health relationship in non-urban communities [343, 344]. My analysis included all Florida residents and, therefore, includes both rural and urban communities. To the best of my knowledge, this is the first study of heat and work-related HRI in the civilian population conducted in a humid sub-tropical environment. Further, the demographic and medical (e.g., severity) distribution of HRI cases varies by data source (see Chapter 5) [87, 155, 270]. As such, the assessment of the outcome using three sources (i.e., ED, hospitalizations, and death certificates) allowed for a more complete estimate of the burden of disease across the spectrum of severity than has been provided by prior studies. An additional strength of the study was the inclusion of meteorological data from Mesonet sites in addition to data from the NWS stations. The inclusion of the Mesonet sites allowed for a more even coverage of weather stations across the state, especially in rural and agricultural areas [365]. Finally, this is the first study is the southeastern U.S. to examine multiple aspects of the heat-health relationship and the first in Florida to examine individual susceptibility to HRI and population vulnerability to increasing heat. However, even with the aforementioned strengths there are still a number of limitations which are discussed below along with some suggestions for additional research.

As with all population studies of the heat-health relationship, the use of ambient outdoor temperature and heat index as proxies for heat exposure has inherent measurement bias as individuals (and populations) do not spend all of their time outdoors nor do they remain in one place. A few articles have been published examining the relationship between indoor and outdoor temperature. A positive correlation has been found between the two with indoor temperatures

typically being higher and less variable than outdoor temperatures [345-347]. A study of 32 homes in New Zealand found that outdoor temperature and seasonality explained 75 percent of the variation in indoor temperature [347], while a study of 30 homes in Detroit, Michigan observed that 34 percent of the variation in indoor temperature was explained by outdoor temperature [346]. A larger study in Montreal, Canada (72 homes) found 22 percent of the variation in indoor temperature was explained by outdoor temperature, while adding the preceding 24-hour average temperature to the model explained a total of 40 percent of the variation in indoor temperature [345]. These studies suggest that, regardless of the individual's location (or movements), as outdoor temperature increases, personal exposure will also increase. However, both North American studies noted that the use of air conditioning, the surrounding environment (e.g., park or parking lot), and type of building, including building materials and location within the structure (e.g., top floor versus bottom) also contributed to variation in indoor temperature [345, 346], potentially biasing the estimated heat-health relationship. The additional modifiers of the relationship between indoor and outdoor temperature may be especially important in Florida. For instance, the use of air conditioning may result in a negative relationship between indoor and outdoor temperature. This is demonstrated in a study of personal heat exposure, measured by pendent monitors, and ambient temperature obtained from weather stations conducted among 81 rural and urban participants in Alabama between July and August 2012 [348]. The authors report that, in general, temperature from weather stations overestimated heat exposure, although the overestimate was less for outdoor workers and was modified by additional factors such as income level and percent body fat [348]. Therefore, while classical measurement error is present when using data from the weather stations, due to the correlation, the results of the analyses are still valid, although the observed relationship between temperature/heat index from weather stations and HRI may be attenuated [366]. Further research estimating the magnitude of measurement error in different geographical areas when using weather stations to represent personal exposure is necessary.

Additional measurement error in the study's main exposure is due to the fact that the heat (i.e., ambient outdoor temperature/heat index) varies by location of the weather station and is assumed to be homogenous over a large area (e.g., zip code). For this aspect of the study, Berkson's

error can be assumed, potentially leading to decreased precision with negligible bias in the effect estimate [366]. Additionally, the reduction in precision will most likely be greater for rural areas than for urban areas, as the distribution of weather stations is differential; a greater number of stations are located in urban areas and, therefore, a smaller area is assumed to be homogenous.

Air pollution was not considered in this analysis as a modifier of the relationship between ambient outdoor heat and HRI morbidity and mortality. Instead, the total effect of heat (as estimated by temperature/heat index) on HRI morbidity and mortality was assessed. This may be viewed as a limitation since air pollution may also exacerbate preexisting conditions making an individual more susceptible to HRI. However, as previously mentioned (see Chapter 2: Environmental Parameters), there is little information in the literature on effect measure modification by air pollution and the magnitude of the observed effect measure modification (when present) in prior studies varied by locale and the outcome under study [92, 130-135]. As such, there does not appear to be enough evidence in the literature to include air pollution as part of a heat mitigation/adaption strategy. Since the overall purpose of this study was to inform policy, analysis of air pollution modification was left to future studies.

There is the potential for exposure misclassification in the morbidity and mortality data, as the residential address of the individual was used instead of the zip code where the exposure actually occurred. It is unclear if this misclassification is associated with the outcome or to a susceptibility factor. For instance, it is possible that individuals with severe chronic medical conditions who are at higher risk of a heat-related outcome may stay close to home (e.g., within their zip code). Alternatively, it is possible among exertional heat-related outcomes that occupational cases may be exposed away from their residential zip codes, while non-occupational cases may be exposed at home (e.g., working in garden). Regardless, for those whose exposure occurred outside their residential zip code, it is impossible to know if that exposure was higher or lower than the exposure in their residential zip code. As a result, the direction and the estimated magnitude of the bias are unclear. However, a review of Florida death certificates may shed some light on the magnitude of the exposure misclassification related to residential address. Of the 163 death certificates with deaths in

the warm season from or with HRI, sixty-one percent (n = 101) contained the residential zip code and the zip code of injury (i.e., where the exposure occurred). Of those certificates with both residential and injury zip codes, 83.1 percent (n = 84) were in the same county. Further stratified, 51 were in the same county and also in the same zip code, and 33 were in the same county but in a different zip code. Among those records that had different zip codes, the mean approximate distance between zip code centroids was 27 miles (SE = 9; median = 12; range = 1-357). Therefore, if we assume that the relationship between residential address and zip code of injury is similar for the mortality and morbidity data, then if may also be possible to assume that a large proportion of HRI morbidity cases are not misclassified at the zip code level. Of course, the distribution of among HRI deaths is different than the distribution for HRI ED visits or HRI hospitalizations and additional research is required before the assumption should be made.

Zip codes were designed to most efficiently deliver the mail. They were not designed for research or to group households in any way other than for efficient mail delivery. The shape files with geographical zip code boundaries are created by a company external to the U.S. Postal Service [367]. As such, the zip code polygon to which each individual is assigned may not be the correct location; therefore, their exposure assignment (via weather station) may also be incorrect. The misclassification is further magnified by the standardization process, as each yearly zip code shape file suffers from the same potential misclassification. This misclassification is non-differential, but the direction and magnitude of this bias is unclear.

Additional bias may be present due to the geographical mismatch between the Zip Code Tabulation Areas (ZCTAs) and zip codes [367]. The Florida population (i.e., denominator) data obtained from the 2007–2011 ACS was stratified by ZCTAs. The purpose of the ZCTA is to be a representation of the zip code. However, ZCTAs were created based on census tracts by the U.S. Census Bureau and are recalculated every 10 years. As such, there will most likely be some amount of mismatch between the geographical area for the zip codes and the geographic area of the ZCTAs. In other words, the denominator data for a zip code in the analysis (based on the ZCTA) may not be

an accurate estimation of the denominator information for that zip code, potentially biasing the rates. Again, the bias is non-differential, and the direction or magnitude of the problem is unclear.

Within my dissertation, the amount of bias created by ZCTA/zip code mismatch, zip code instability (e.g., changing boundaries), and zip code-induced misclassification is less concerning than the bias that would be created by assuming exposure, demographic, and susceptibility factor homogeneity across a larger, more stable, geographical area, such as a county. Further, analysis at a larger geographical area may obscure relevant associations that were observed in this project.

The use of ICD-9-CM codes to identify the outcome within the study has innate limitations due to the original purpose of the codes (e.g., billing). The assignment of codes varies from facility to facility (or medical examiner). No strict uniform guidelines exist to inform the assignment of each particular code, resulting in intermingling of actual diagnoses. This misclassification should be non-differential with respect to the exposure, and as a result, an attenuation of the effect estimates is expected.

A further limitation of the outcome specific to HRI is that doctors may be more likely to assign a heat-related diagnosis (represented by ICD-9-CM codes) if they are aware of a prolonged increase in temperature or an intensely hot day. However, the degree to which this occurs (if it does occur) will most likely vary by region and facility. The direction and the magnitude of the differential measurement bias on the effect estimates between temperature/heat index and HRI is unclear [366].

The outcome used in this study was diagnosed failure of the thermoregulatory system (i.e., HRI). However, HRI or clinical outcomes biologically caused by heat (e.g., acute renal failure) may not be the only possible outcomes associated with increasing heat exposure. For instance, during hot weather the time till food spoilage may decrease as temperature increases leading to potential increased consumption of spoiled food. As individuals are trying to escape from the heat they may end up in places where they have increased exposure to vector-borne-illnesses (e.g., time at the pool or hiking). Additionally, disorientation among workers due to heat may lead to injuries, such as falls. The public health interventions related to these and other outcomes not related to HRI, but associated

with increased heat exposure, will be different from those interventions related to HRI reduction/prevention. Therefore, additional studies that look at non-traditional outcomes associated with heat in Florida are recommended.

Due to data availability, air-conditioning usage/availability was not included in this dissertation even though the factor has a strong level of agreement across the literature as modifying the risk of heat-related outcomes [9, 350]. The American Housing Survey currently collects data on air-conditioning availability; however it is only available at the national level, regional level (i.e., Northeast, South, Mideast, and West) and for metropolitan areas. For Florida, only the 2013 survey contains data on the metropolitan areas of Jacksonville, Miami, Orlando, and Tampa. The U.S. Energy Information Administration also collects information on air conditioning usage at the national, regional, and state level. Finally, Florida is collecting air conditioning usage information via the Behavioral Risk Factor Surveillance (BRFSS) survey. However, the collection process was not completed in time to be included in this dissertation document. It is clear in the literature that the ability to cool ones-self is important in preventing HRI [9, 38, 39, 350]. According to the 2013 American Housing survey the proportion of occupied homes with central air-conditioning in Florida metropolitan areas was 90 percent or higher [368]. Due to the humid subtropical/tropical Florida climate it is reasonable to assume that the rest of Florida also has a high proportion of air conditioning availability. Air-conditioning usage may vary by a number of factors including socio-economic status, house type and age, and personal feelings about usage (e.g., dislike of air-conditioning) [171, 233, 234, 369]. Further research on the usage and non-usage of air conditioning in Florida is required, as well as how air conditioning usage impacts the rates of HRI as heat index increases over the summer.

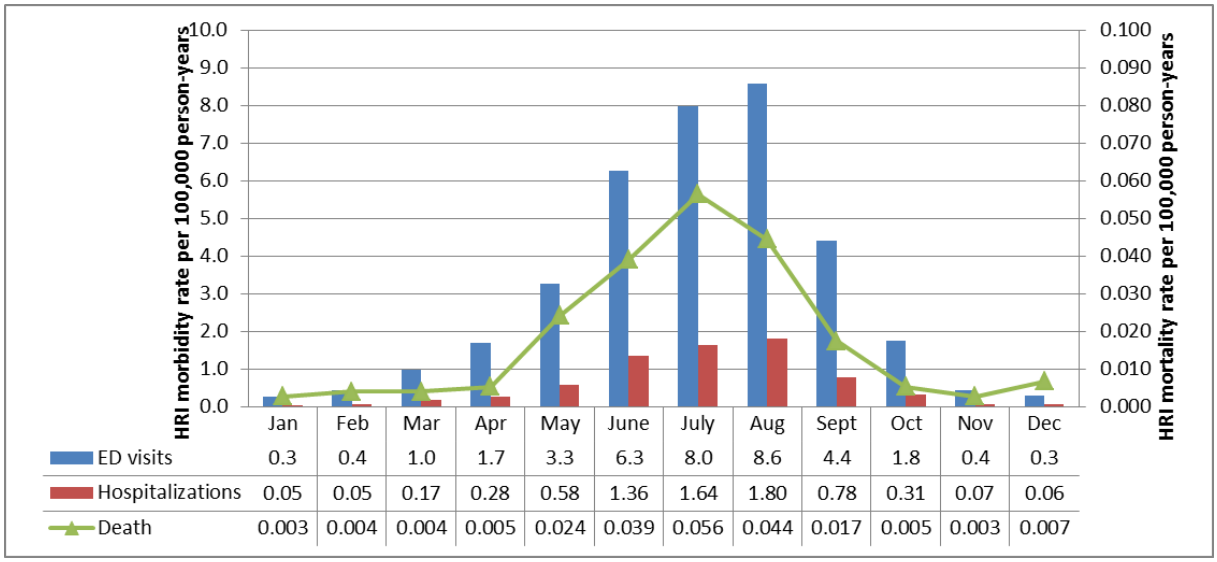
SUMMARY

In summary, the rates of HRI do increase with increasing heat index (and temperature). Adaptation or prevention activities are potentially occurring in Florida at higher heat index and temperature values. The amount and effectiveness of these adaption activities may vary by geographic region or susceptibility factor(s). Only some of the community-level susceptibility factors

identified in other studies and used in prior heat vulnerability assessments may be appropriate for use in Florida. However, this study only provided a basic evaluation of these factors and further research is required. Individual-level factors were also identified which modify the burden of HRI. This work found that while rates of HRI are higher during a heat wave compared to non-heat wave days, after adjusting for the current and prior days' heat, the effect disappears. Finally, as Florida is in a humid subtropical climate, the use of heat index when modeling the heat-health relationship is a better metric of heat than temperature alone.

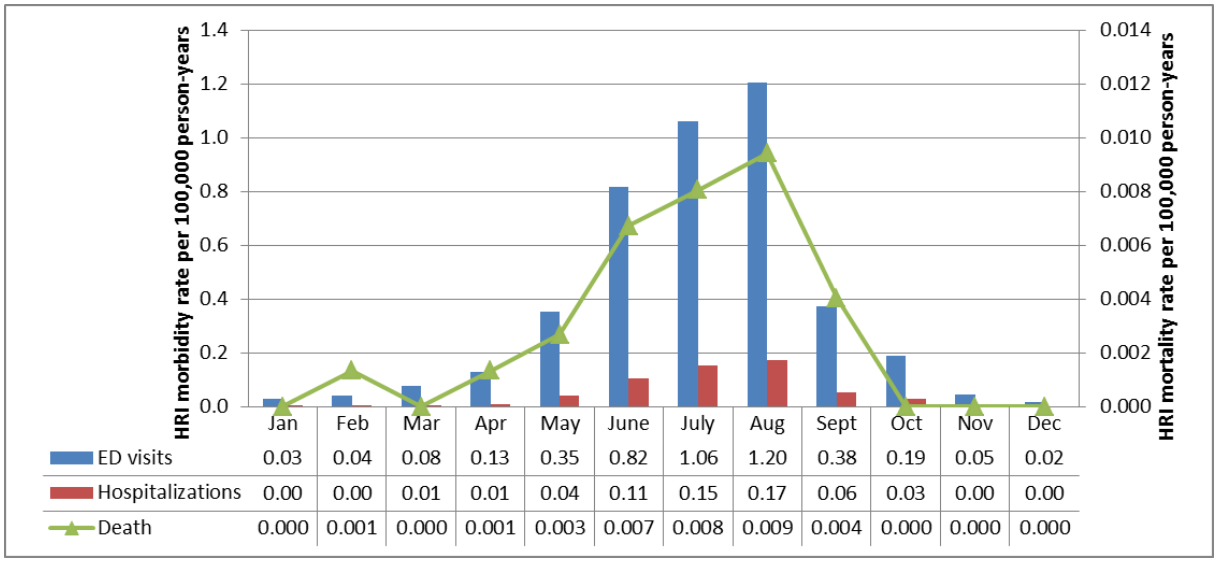
The work conducted in this dissertation provides a comprehensive look among Florida residents at the association between ambient outdoor heat and HRI. The completed assessment of exposure-response relationship between heat and HRI will provide guidance on which exposure metrics to use for future research and inform the direction of policy development. Further, the analysis and identification of factors associated with higher rates of HRI or modification of the exposure-response relationship will help policy makers to more effectively use limited resources by identifying and targeting vulnerable sub-populations/areas. Finally, my research has established a number of areas for future research.

APPENDIX A. SUPPLEMENTAL FIGURES AND TABLES



	Jan	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec
ED visits	0.3	0.4	1.0	1.7	3.3	6.3	8.0	8.6	4.4	1.8	0.4	0.3
Hospitalizations	0.05	0.05	0.17	0.28	0.58	1.36	1.64	1.80	0.78	0.31	0.07	0.06
Death	0.003	0.004	0.004	0.005	0.024	0.039	0.056	0.044	0.017	0.005	0.003	0.007

Figure A-1. *Non-work-related* HRI morbidity and mortality rates for Florida residents stratified by month and data source, 2005–2012 (Total cases: ED visits = 27,028; Hospitalizations = 5324; Deaths = 158). All rates are per 100,000 person-years. Note that the morbidity rates (left y-axis) are two orders of magnitude larger than the mortality rates (right y-axis).



	Jan	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec
ED visits	0.03	0.04	0.08	0.13	0.35	0.82	1.06	1.20	0.38	0.19	0.05	0.02
Hospitalizations	0.00	0.00	0.01	0.01	0.04	0.11	0.15	0.17	0.06	0.03	0.00	0.00
Death	0.000	0.001	0.000	0.001	0.003	0.007	0.008	0.009	0.004	0.000	0.000	0.000

Figure A-2. *Work-related* HRI morbidity and mortality rates for Florida residents stratified by month and data source, 2005–2012 (Total cases: ED visits = 3,234; Hospitalizations = 432; Deaths = 25). All rates are per 100,000 person-years. Note that the morbidity rates (left y-axis) are two orders of magnitude larger than the mortality rates (right y-axis).

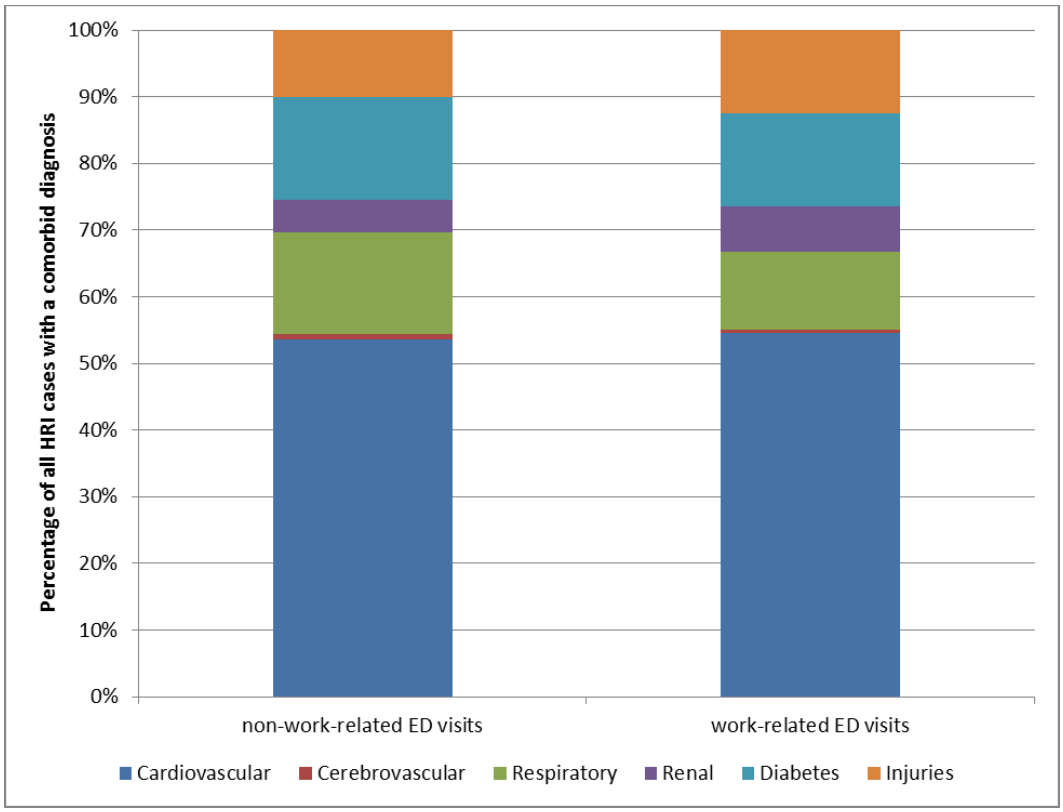


Figure A-3. Distribution of co-morbid outcomes among Florida residents identified with HRI in the ED stratified by work-related status (2005–2012)

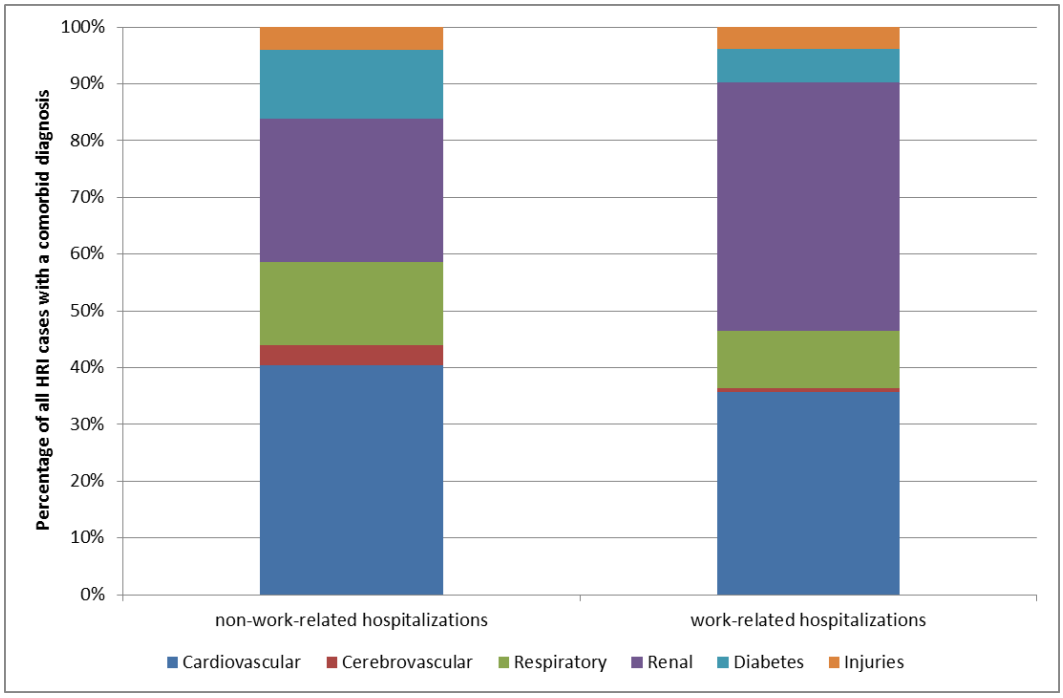


Figure A-4.Distribution of co-morbid outcomes among Florida residents identified with HRI in the hospital stratified by work-related status (2005–2012)

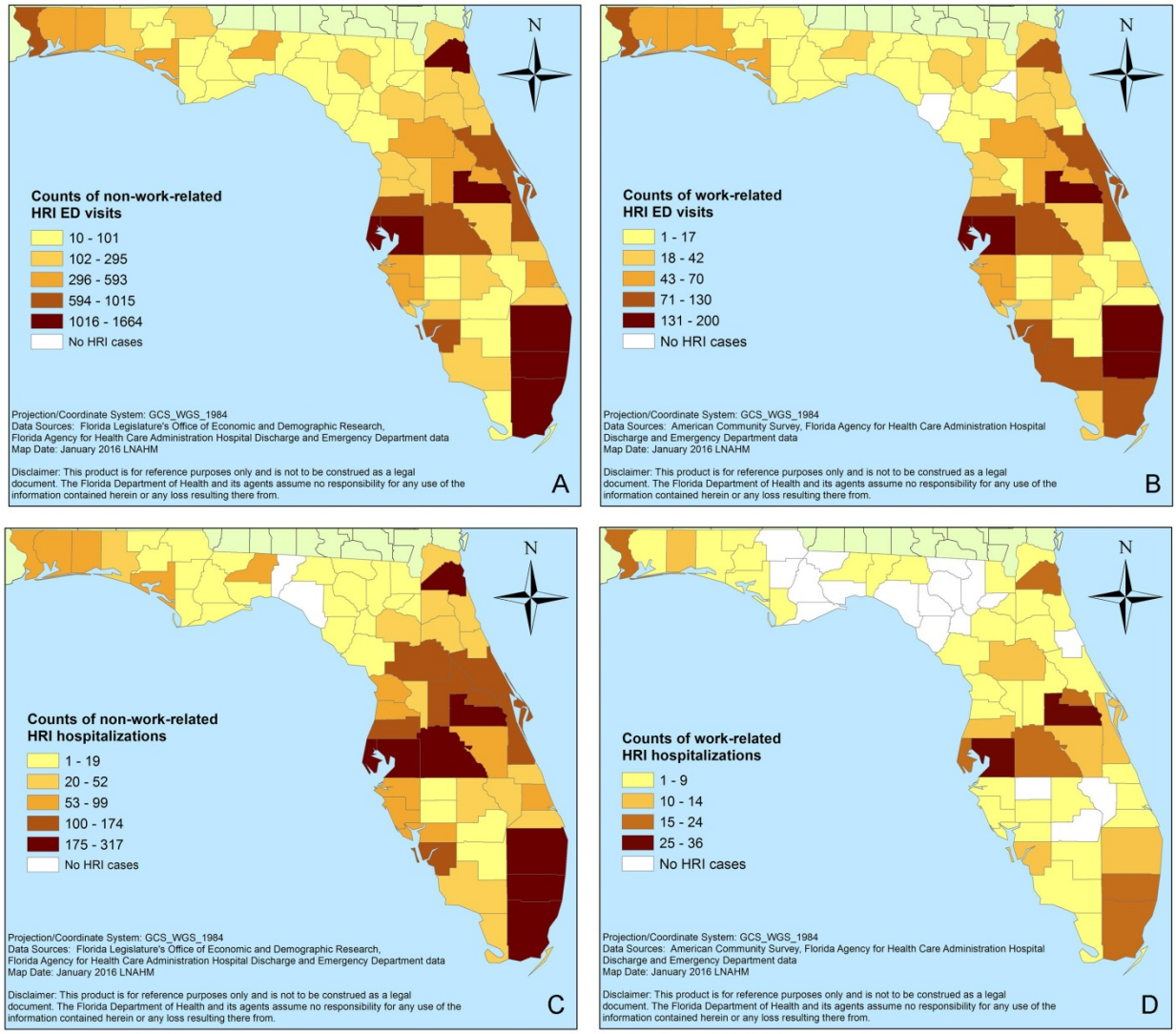


Figure A-5. County-specific counts of HRI ED visits and hospitalizations among Florida residents during the warm season (2005–2012): (A) non-work-related HRI ED visits; (B) work-related HRI ED visits; (C) Non-work-related HRI hospitalizations; (D) work-related HRI hospitalizations

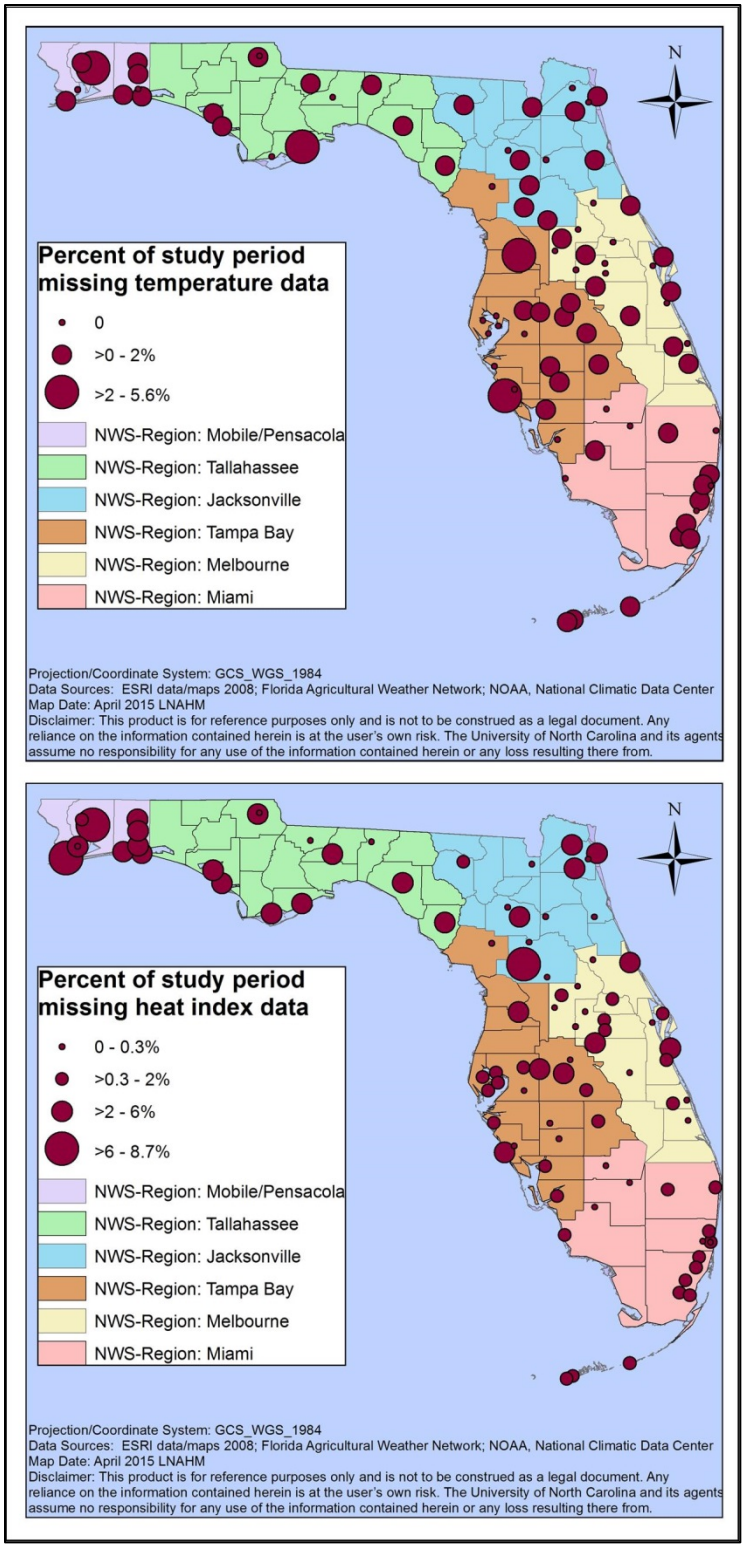


Figure A-6. Percent of missing data for daily temperature (top panel) and daily heat index (bottom panel) over the entire study period, May-October 2005–2012, by weather station.

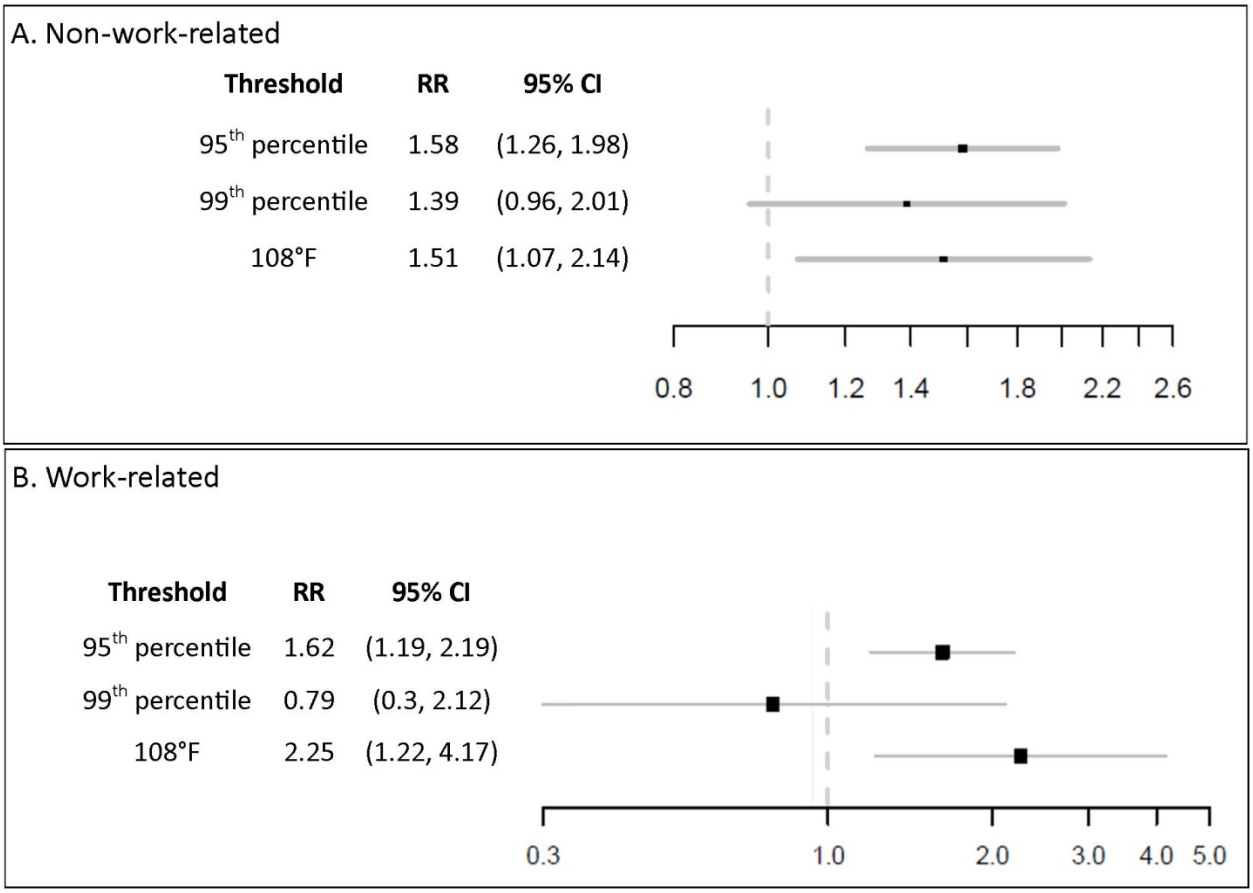


Figure A-7. Forest plot of the rate ratio (RR) and 95% confidence intervals for non-work-related (A) and work-related (B) HRI morbidity rates occurring during a four-day or longer heat wave compared to rates on non-heat wave days, stratified by varying heat wave definitions. The range of threshold values for the 95th percentile of maximum daily heat index was 101-109°F and for the 99th percentile was 101-115°F. Note that reference group, non-heat wave days, varied by definition.

Table A-1. Comparison of Akaike information criterion (AIC) values for different heat metric parametrization stratified by model specification and National Weather Service (NWS) region.*

Model Specification	Region	AIC value for heat metric parametrization				
		Linear	Quadratic	Spline	Piecewise†	Categorical
Outcome = non-work-related HRI morbidity Exposure = Temperature	Pensacola	13976.6	13966.5	13948.7	**13948.0**	13952.8
	Tallahassee	**18395.2**	18397.2	18400.7	18395.3	18414.2
	Jacksonville	36655.5	36656.7	36656.6	**36646.8**	36682.7
	Melbourne	53717.0	53718.9	53717.7	**53711.4**	53761.8
	Tampa	78624.9	78618.8	78605.1	**78604.8**	78664.2
	Miami	53756.2	53747.5	**53724.9**	53735.8	53784.3
	Key West	**1073.2**	1075.0	1080.2	1075.1	1083.4
Outcome = work-related HRI morbidity Exposure = Heat Index	Pensacola	13866.8	13866.9	13867.7	**13866.3**	13885.3
	Tallahassee	18155.6	18153.8	18158.4	**18147.3**	18171.4
	Jacksonville	36434.3	**36415.2**	36419.8	36415.7	36455.6
	Melbourne	53483.8	53475.9	53470.1	**53464.2**	53495.8
	Tampa	78591.2	78538.8	78531.9	**78526.5**	78551.4
	Miami	53868.1	53863.8	**53858.3**	53858.9	53862.2
	Key West	1069.9	1070.1	1073.0	**1064.9**	1087.4
Outcome = work-related HRI morbidity Exposure = Temperature‡	Pensacola	3072.7	**3068.2**	3074.0	3068.2	3077.7
	Tallahassee	3253.2	3255.0	3257.7	**3252.9**	3262.9
	Jacksonville	5778.8	5780.5	5780.9	**5774.5**	5778.8
	Melbourne	8399.4	8401.0	8401.6	**8398.5**	8416.0
	Tampa	14902.0	14904.0	14902.6	**14900.9**	14912.7
	Miami/Key West	8383.6	8385.3	8387.5	**8383.6**	8390.3
Outcome = work-related HRI morbidity Exposure = Heat Index	Pensacola	3058.3	3053.4	3058.6	**3051.8**	3069.9
	Tallahassee	3214.1	3215.6	3219.1	**3213.8**	3222.3
	Jacksonville	5713.8	5715.8	5716.9	**5713.5**	5725.5
	Melbourne	8380.1	8374.3	8377.8	**8371.7**	8394.6
	Tampa	14852.7	14852.5	14851.5	**14849.8**	14871.3
	Miami/Key West	8358.6	8359.6	8360.0	**8357.5**	8375.4
Outcome = non-work-related HRI mortality Exposure = Temperature	Pensacola	**144.6**	146.6	148.6	146.4	**---**
	Tallahassee	**168.6**	170.2	172.1	170.6	**---**
	Jacksonville	**422.0**	424.0	424.8	423.9	**---**
	Melbourne	**648.3**	649.0	649.6	649.9	**---**
	Tampa	735.3	736.0	737.7	**735.1**	**---**
	Miami/Key West	605.9	605.8	**603.1**	607.8	**---**
Outcome = non-work-related HRI mortality Exposure = Heat Index	Pensacola	144.6	143.1	144.9	**141.3**	---
	Tallahassee	**174.7**	176.2	178.3	176.3	---
	Jacksonville	**424.0**	425.8	424.7	426.0	---
	Melbourne	**644.0**	645.1	644.3	645.3	---
	Tampa	**733.0**	734.5	735.6	734.8	---
	Miami/Key West	**608.4**	609.2	609.6	610.2	---

*Bold values indicate minimum AIC value for the NWS region/model specification (i.e row).
†The breakpoint for the piecewise parametrization was NWS region-specific
‡For 4 of the region-specific models the difference between the linear and piecewise models was <2.

Table A-2. Assessment of an added heat-wave effect in Florida (2005–2012) via comparison of the model of ambient heat index (i.e., no heat wave term) and models with an additional term for a four-day or longer heat wave stratified by heat wave definition and work-related status .

Model with heat wave term	Non-work-related			Work-related		
	Heat wave effect*	Z Score (p-value)†	AIC‡	Heat wave effect*	Z Score (p-value)†	AIC‡
No heat wave term	---	---	-145.93	---	---	-95.43
95th percentile	0.89 (0.76,1.05)	-1.35 (0.2)	-136.77	0.78 (0.57,1.06)	-1.6 (0.1)	-82.72
99th percentile	0.78 (0.59,1.03)	-1.73 (0.08)	-123.16	0.47 (0.18,1.26)	-1.49 (0.1)	-38.47
108°F	0.74 (0.55,0.99)	-2.04 (0.04)	-125.95	0.85 (0.44,1.65)	-0.47 (0.6)	-68.49

*The pooled relative effect and 95% confidence interval for HRI morbidity rates during heat waves compared to non-heat wave days after adjusting for ambient heat index

†Z-test H0: the effect estimate is equal to zero

‡Akaike information criterion (AIC) is a test of model fit, the smallest value indicates the best model

APPENDIX B. RESULTS OF ALL TEMPERATURE ANALYSES

In this study, heat index was determined to be the best metric when assessing the heat-health relationship. However, temperature is the most widely available metric of heat [111] and many studies use temperature when assessing the heat-health relationship [62, 66, 343]. In order to provide information comparable to other studies, the results of the analysis examining the relationship between HRI and maximum daily temperature from three angles are provided below. Although the effect estimates and variance are different, the analysis conclusions of the temperature and HRI morbidity/mortality relationship are the same as the heat-index and HRI morbidity/mortality relationship. With the only difference being that only the relative intensity threshold should be used when defining a heat wave using temperature.

AMBIENT HEAT: NON-WORK-RELATED HRI

The distribution of maximum daily temperature can be found in Figure B-1. For all HRI outcomes, the final model included the temperature on the current day (lag0) and the prior day (lag1). Appendix C contains the results of the model specific comparisons for inclusion of lag days. The modeled relationship between maximum daily temperature and the HRI outcome can be found in Figure B-2, Figure B-3, and Figure B-4. The strongest relationship between HRI and temperature was observed in relation to the current day's temperature regardless of outcome (e.g., morbidity) or work-related status.

HEAT WAVES: NON-WORK-RELATED

The number of distinct heat waves occurring in the state varied by threshold value and geographic location (Table B-1). Regardless of definition, a small proportion of to study days were classified as heat wave days (Figure B-5).

For non-work-related HRI, regardless of definition, a small proportion of HRI cases occurred during a heat wave. The proportion of non-work-related HRI morbidity cases by region can be found in Table B-2. For a heat wave with a duration of two or more days, 9 (6.5%), 2 (1.4%), or 1 (0.7%) HRI death(s) occurred when the intensity was at the 95th percentile, 99th percentile, or constant threshold, respectively. However, according to information of the death certificate only two, one, and zero deaths had onset of symptoms occurring during the respectively defined heat wave. The majority of non-work-related HRI cases for a heat wave of two or more days above the threshold occurred on the first (i.e., two consecutive days above intensity threshold) and second day of the heat wave (95th percentile: day 1 = 50.8%, day 2 = 24.4%; 99th percentile: day 1 = 66.1%, day 2 = 20.2%; 98°F: day 1 = 57.4%, day 2 = 19.8%). The results of the non-work-related HRI morbidity rates on heat wave days compared to HRI rates on non-heat wave days by heat wave definition can be found in Figure B-5.

Less than a half a percent of all work-related HRI cases occurred when a heat wave was defined by the 99th percentile or by an intensity of 98°F. As such, these intensity definitions were not used to analyze work-related HRI rates during heat waves. Fifty-one percent of work-related HRI cases occurred on the first day of a two day or longer heat wave with intensity defined by the 95th percentile and 24.4 percent occurred on the second day. There was one heat-related death that occurred on a heat wave day; however, according to information on the death certificate, onset of symptoms occurred prior to the heat wave. A heat wave with duration of two days or more days and an intensity at the 95th percentile produced a rate 1.66 times as high as the rate on non-heat wave

days (95% CI = 1.32, 2.08). When duration was defined as four days or more days above the threshold, the rate was 1.33[60] times as high as the rate on non-heat wave days (95% CI = 0.82, 2.15).

AMBIENT HEAT AND HEAT WAVES

For both non-work-related and work-related morbidity, the best fitting model did not include a heat wave term. Further, for each of the six heat wave definitions, the null hypothesis that the natural log of the heat wave term was equal to zero could not be rejected (Table B-3).

[60] There were zero work-related cases for the Miami/Key West NWS regions.

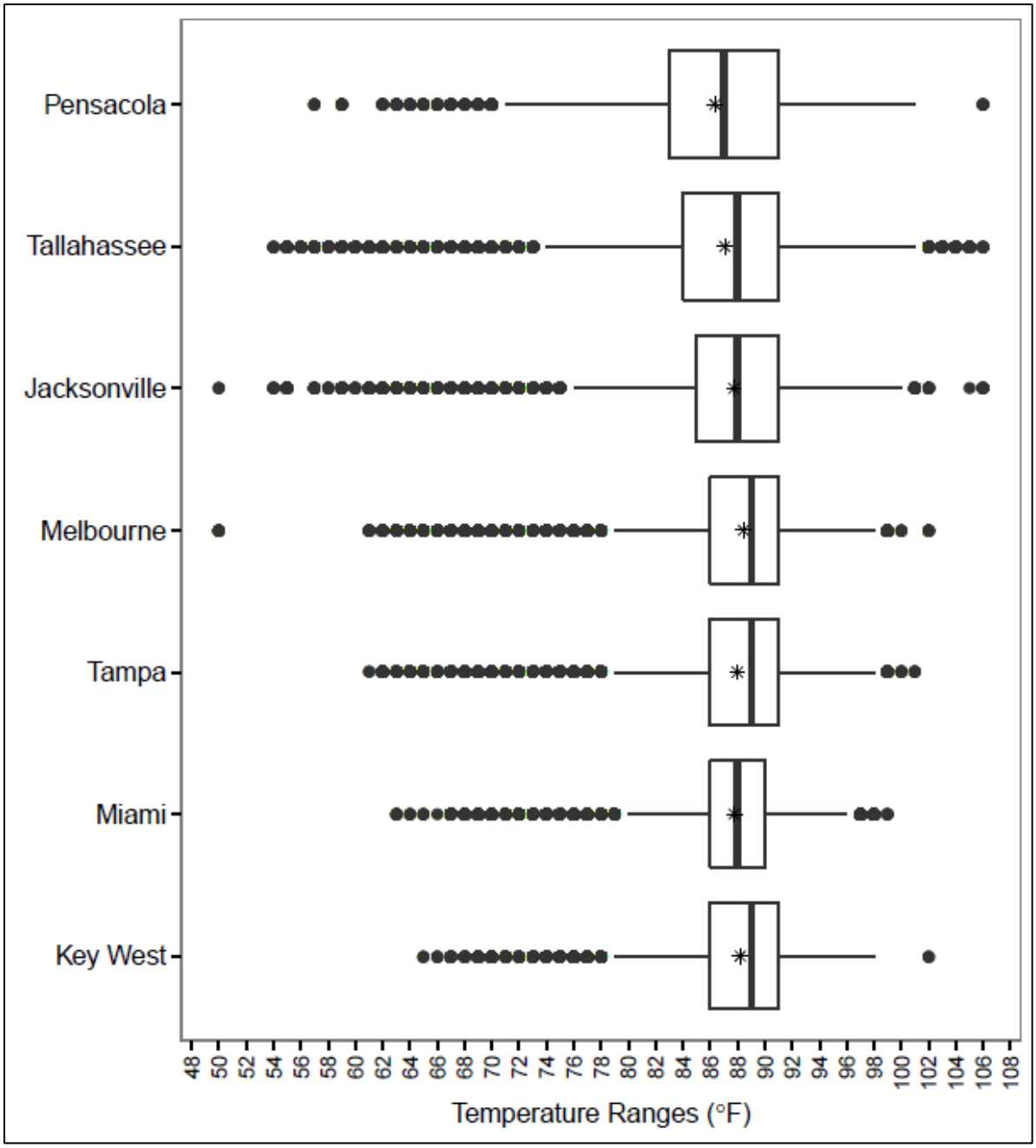


Figure B-1. Box-and-whisker plot of maximum daily temperature May-October (2005–2012) stratified by National Weather Service region. The vertical line represents the median temperature value for each region, and the asterisk represents the mean temperature value for each region. The median and mean temperature value for the state is 88°F.

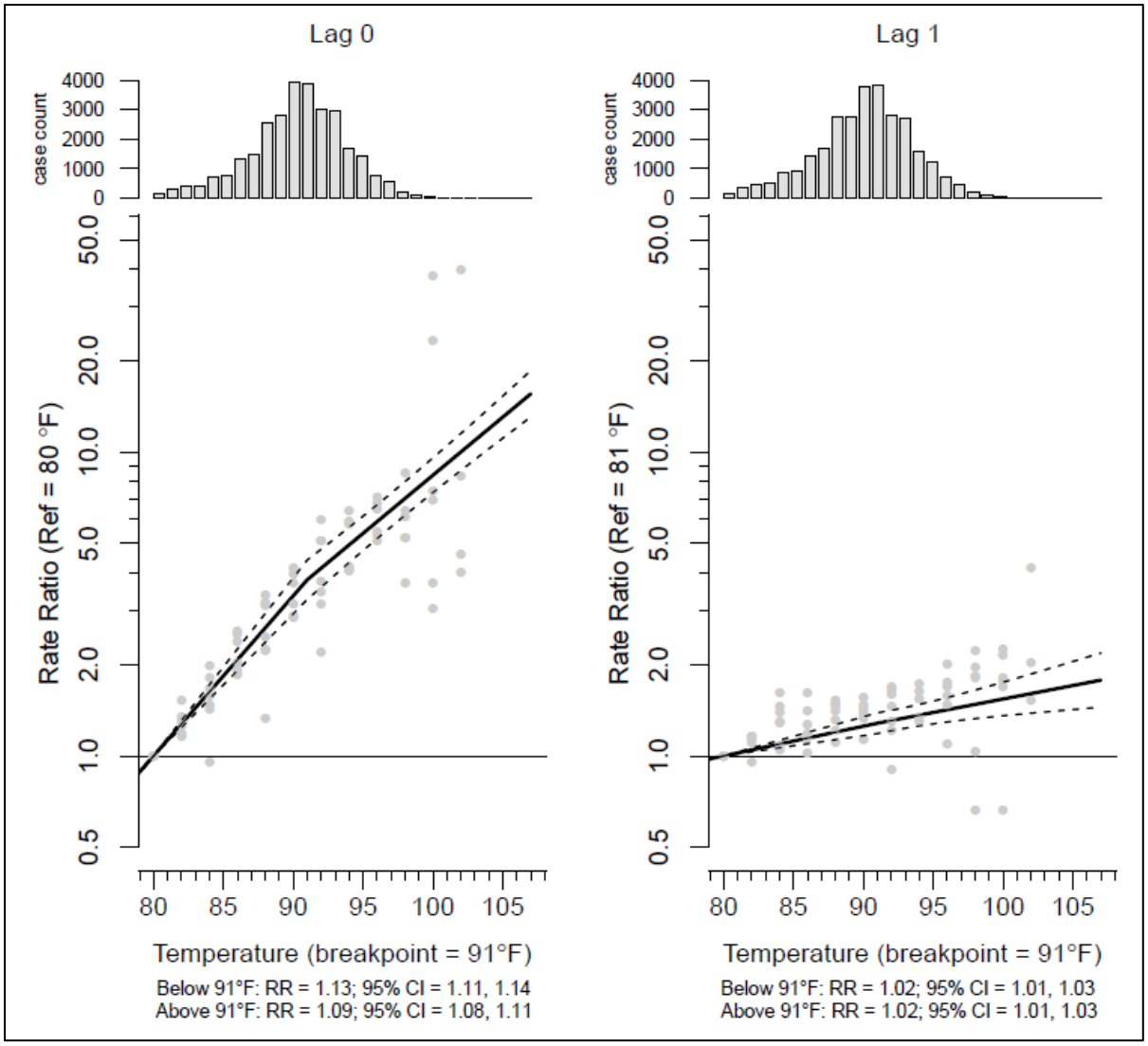


Figure B-2. Modeled relationship between non-work-related HRI morbidity rates and maximum daily temperature on the current day, lag 0, and the prior day, lag 1, for May-October, 2005–2012 (solid line). The hashed lines are the 95% confidence intervals. The grey dots are the region-specific rate ratios for every two degrees of temperature (e.g., 80-81, 82-83…)[61]. The histogram represents the observed number of cases occurring at each temperature degree. All other temperature values had at least four cases during the study period except a temperature of 106°F which had one case.

[61] For the Key West region, the region-specific rate ratios were for every 4 degrees of temperature.

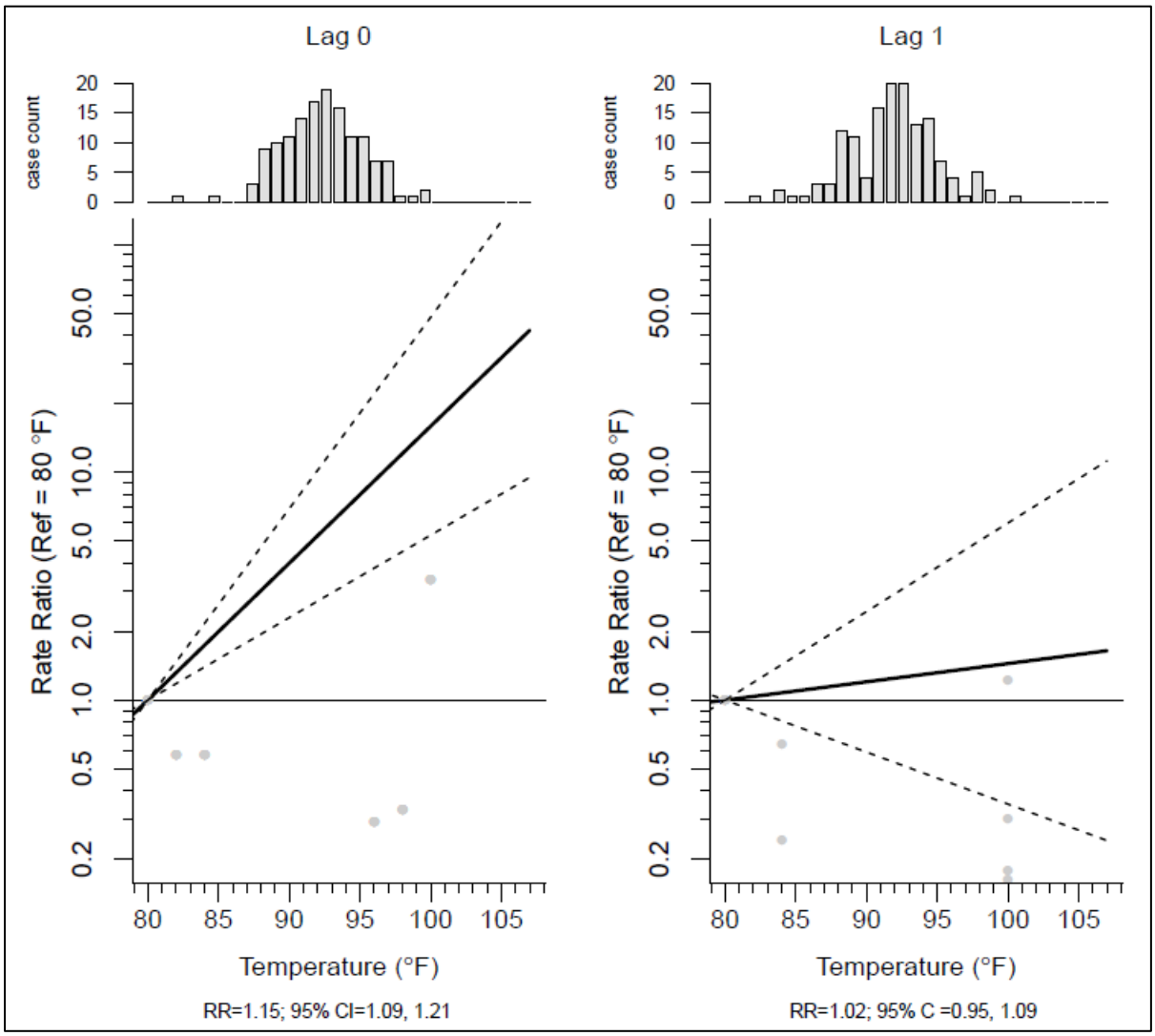


Figure B-3. Modeled relationship between non-work-related HRI death rates and maximum temperature on the current day, lag 0, and the prior day, lag 1, for May-October, 2005–2012 (solid line). The hashed lines are the 95% confidence intervals. The grey dots are the region-specific rate ratios for every two degrees of temperature (e.g., 80-81, 82-83…). The histogram represents the observed number of cases occurring at each temperature degree. Note that the majority of the region-specific rate ratios are unstable and considerably far outside the y-axis range.

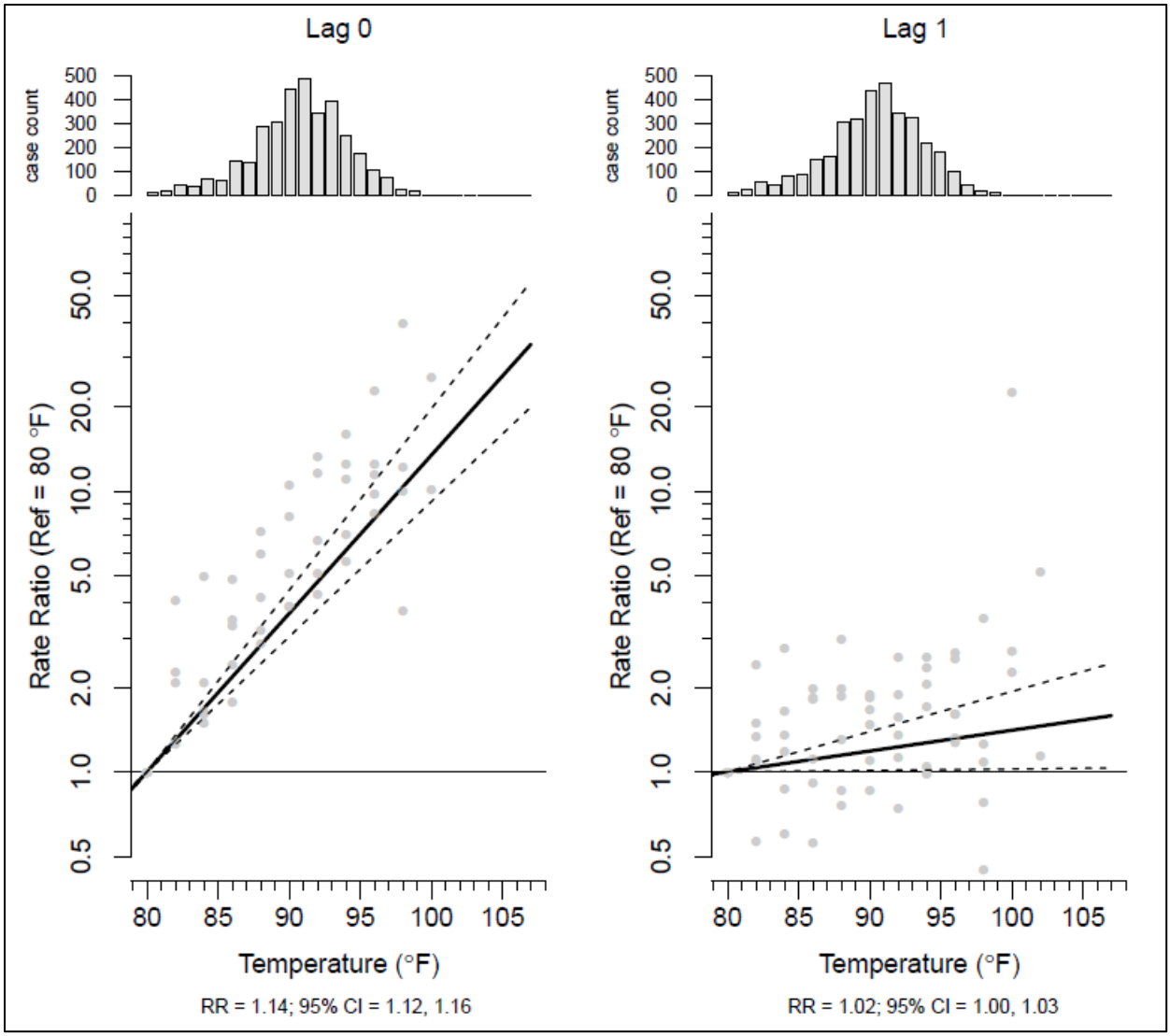


Figure B-4. Modeled relationship between work-related HRI morbidity rates and maximum daily temperature on the current day, lag 0, and the prior day, lag 1, for May-October, 2005–2012 (solid line). The hashed lines are the 95% confidence intervals. The grey dots are the region-specific rate ratios for every two degrees of temperature (e.g., 80-81, 82-83…). The histogram represents the observed number of cases occurring at each temperature degree. Above a temperature of 101°F there was only one case which occurred at a temperature of 105°F.

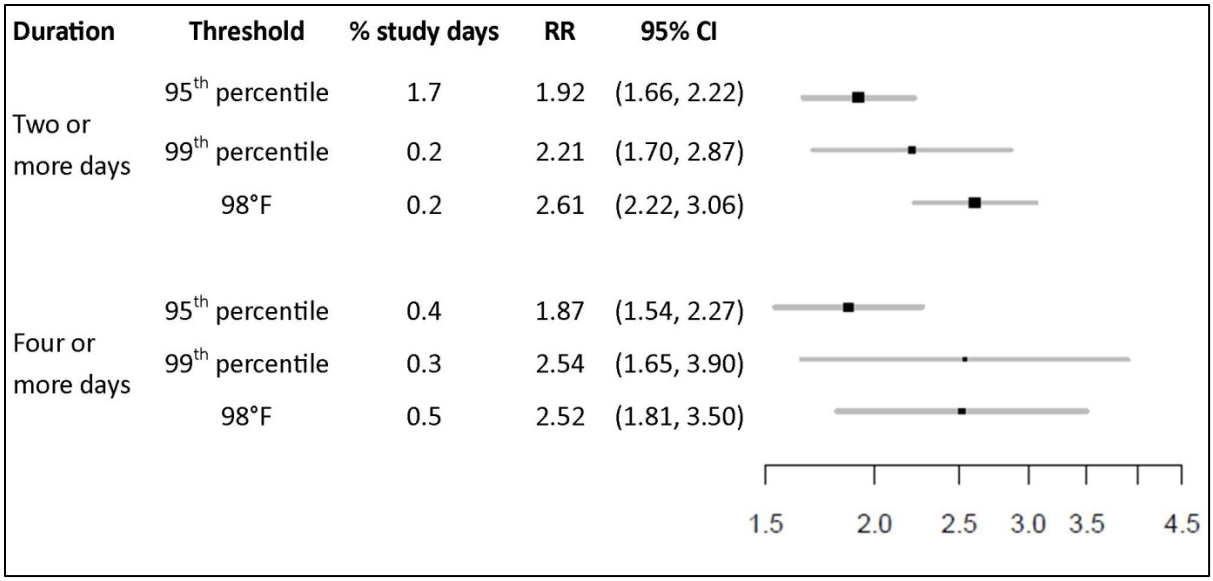


Figure B-5. Forest plot of the rate ratios (RR) and 95% confidence intervals for non-work-related HRI morbidity rates on heat wave days versus non heat wave stratified by varying heat wave definitions. The range of threshold values for the 95th percentile was 93-97°F and for the 99th percentile was 94-100°F. Note that the comparison group for a four day or longer (4-day) heat wave includes two and three consecutive days above the threshold. There were zero 4-day heat waves with a threshold at the 99th percentile in the Key West Region during the study period. There were also zero heat waves, regardless of duration, with a threshold of 98°F for the Miami and Key West regions during the study period.

Table B-1. Number of distinct heat waves in Florida stratified by National Weather Service (NWS) region, duration, and maximum daily temperature intensity thresholds (2005–2012)

NWS Region	HW Threshold = 95th Percentile			HW Threshold = 99th Percentile			HW Threshold = 98°F		
	no. 2-day HW	*no. 4-day HW*	*Max HW days*	*no. 2-day HW*	*no. 4-day HW*	*Max HW days*	*no. 2-day HW*	*no. 4-day HW*	*Max HW days*
Pensacola	78	29	15	15	5	7	19	6	7
Tallahassee	133	**58**	14	33	**10**	8	**47**	**14**	13
Jacksonville	136	43	10	15	2	8	24	5	8
Melbourne	133	43	14	12	4	8	11	2	8
Tampa	**179**	55	14	38	7	7	10	1	4
Miami	142	33	11	**40**	7	6	0	0	1
Key West	39	11	5	3	0	3	0	0	1
*Florida**	*390*	*150*	*15*	*102*	*25*	*8*	*68*	*21*	*13*

* Heat waves defined as two or more days (2-day) above an intensity threshold or four or more days (4-day) above the intensity threshold. The number of distinct Florida heat waves is less than the sum of the regional heat waves. A regional heat wave may have occurred at the same time as a heat wave in another region and, therefore, the heat wave was counted only once when calculating the number of distinct heat waves at the state level. A regional heat wave may not have occurred over the whole region but only for zip codes associated with a few weather stations.

Table B-2. Proportion of total non-work-related heat-related illness (HRI) morbidity cases occurring on a heat wave day stratified by heat wave definition* and National Weather Service (NWS) region (2005–2012)

NWS Region	HW Threshold = 95th Percentile		HW Threshold = 99th Percentile		HW Threshold = 98°F	
	2-day HW	4-day HW	2-day HW	4-day HW	2-day HW	4-day HW
Pensacola	105 (6%)	35 (1.9%)	22 (1.2%)	6 (0.3%)	26 (1.4%)	8 (0.4%)
Tallahassee	233 (12.8%)	102 (5.2%)	48 (2.4%)	12 (0.6%)	109 (5.6%)	30 (1.5%)
Jacksonville	303 (7.8%)	62 (1.5%)	20 (0.5%)	0 (0%)	36 (0.9%)	2 (0%)
Melbourne	210 (3.5%)	42 (0.7%)	8 (0.1%)	2 (0%)	5 (0.1%)	2 (0%)
Tampa	266 (3.2%)	53 (0.6%)	41 (0.5%)	2 (0%)	7 (0.1%)	0 (0%)
Miami	200 (3.6%)	33 (0.6%)	44 (0.8%)	3 (0.1%)	---	---
Key West	2 (1.7%)	0 (0%)	0 (0%)	---	---	---
Florida	*1319 (4.6%)*	*327 (1.1%)*	*183 (0.6%)*	*25 (0.1%)*	*183 (0.6%)*	*42 (0.1%)*

*Heat wave intensity defined by maximum daily temperature. Heat wave duration defined as two or more days (2-day) above the intensity threshold or four or more days (4-day) above the intensity threshold.

Table B-3. Assessment of an added heat-wave effect in Florida (2005–2012) via comparison of the model of ambient temperature (i.e., no heat wave term) and models with an additional term for a two-day or longer heat wave stratified by temperature definition and work-related status.

Duration	Model with heat wave term	Non-work-related			Work-related		
		Heat wave effect*	Z Score (p-value)†	AIC‡	Heat wave effect*	Z Score (p-value)†	AIC‡
Two-day or longer	No heat wave term	---	---	-128.942	---	---	-53.1728
	95th percentile	0.96 (0.86,1.08)	-0.70 (0.5)	-125.353	0.69 (0.54,0.89)	-2.90 (0.004)	-32.3878
	99th percentile	0.93 (0.74,1.19)	-0.56 (0.6)	-104.278	---	---	---
	98°F	1.02 (0.76,1.38)	0.16 (0.9)	-106.916	---	---	---
Four-day or longer	95th percentile	0.94 (0.81,1.10)	-0.75 (0.5)	-107.003	0.51 (0.29,0.89)	-2.35 (0.02)	-12.9075
	99th percentile	0.90 (0.57,1.42)	-0.46 (0.6)	-98.977	---	---	---
	98°F	0.91 (0.64,1.28)	-0.56 (0.6)	-98.3419	---	---	---

*The pooled relative effect and 95% confidence interval for HRI morbidity rates on heat wave days compared to non-heat wave days after adjusting for ambient temperature

†Z-test H0: the natural log of the effect estimate is equal to zero

‡Akaike information criterion (AIC) is a test of model fit, the smallest value indicates the best model

APPENDIX C. LAG RESULTS

Lags of 1-3 days were originally included when modeling maximum daily temperature or maximum daily heat index. A lag-stratified approach was used; lag day 2 and lag day 3 were constrained to be equal [312]. The prior day was considered separately from the 2-day constrained term (i.e., lag1, lag2-3). The likelihood ratio test with an alpha level of 0.05 was used to determine the number of lags to be included in the final model. Lag status was assessed for each NWS region individually. This was done to expose any variation in model specification that was occurring prior to employing the meta-analytic techniques used to obtain the statewide summary estimate.

TEMPERATURE: NON-WORK-RELATED HRI

The likelihood ratio test indicated that the HRI-temperature model with a lag of three days was the best fit for all regions except Pensacola and Key West. For Pensacola the best fit was a lag of one day while the best fit for Key West did not include lags. Since the majority of regions include three lag days, the analysis was run with all lag terms. However, no effect was observed for lag2 and lag3. There was very little change in the effect estimates and variance for lag0 and lag1 when the additional lag days were removed from the model. As there were no changes, a simpler model was used instead.

Table C-1. Assessment of the number of lag days to be included when modeling the relationship between non-work-related HRI and maximum daily temperature (2005–2012)

NWS Region	Model Lag Terms	N	AIC*	Likelihood	LRT†	p-value
	lag0, lag1, lag23	51700	13651.94	-6801.97		
Pensacola	lag0, lag1	51700	13650.71	-6803.36	2.8	0.10
	lag 0	51700	13663.44	-6811.72	16.7	<0.001
	lag0, lag1, lag23	119369	18295.70	-9124.85		
Tallahassee	lag0, lag1	119369	18301.60	-9128.80	7.9	0.02
	lag 0	119369	18305.17	-9132.59	7.6	0.02
	lag0, lag1, lag23	201314	36411.65	-18181.83		
Jacksonville	lag0, lag1	201314	36414.10	-18185.05	6.4	0.04
	lag 0	201314	36438.77	-18199.39	28.7	<0.001
	lag0, lag1, lag23	257874	53544.14	-26748.07		
Melbourne	lag0, lag1	257874	53562.72	-26759.36	22.6	<0.001
	lag 0	257874	53568.08	-26764.04	9.4	0.01
	lag0, lag1, lag23	422081	78191.84	-39071.92		
Tampa	lag0, lag1	422081	78257.28	-39106.64	69.4	<0.001
	lag 0	422081	78279.03	-39119.52	25.7	<0.001
	lag0, lag1, lag23	305230	53683.01	-26817.51		
Miami	lag0, lag1	305230	53689.02	-26822.51	10.0	0.01
	lag 0	305230	53694.42	-26827.21	9.4	0.01
	lag0, lag1, lag23	7321	1070.82	-511.41		
Key West	lag0, lag1	7321	1068.78	-512.39	2.0	0.4
	lag 0	7321	1067.17	-513.58	2.4	0.3

*Akaike information criterion: a lower value indicates a better model fit.
†Likelihood ratio test. Each row is compared to the prior row (e.g., lag0 versus lag0-1).

Table C-2. Non-work-related HRI rate ratio and 95% confidence interval for a 1°F increase in maximum daily temperature on the current or on each of the prior three days (2005–2012)*

	Below 91°F RR (95% CI)	Above 91°F RR (95% CI)
Lag 0	1.132 (1.116, 1.148)	1.091 (1.076, 1.106)
Lag 1	1.036 (1.027, 1.046)	1.027 (1.014, 1.041)
Lag 2	0.985 (0.981, 0.990)	1.001 (0.991, 1.011)
Lag 3	0.985 (0.981, 0.990)	1.001 (0.991, 1.011)

*Summarized statewide estimates from seven regional models. Lag2 and Lag3 are constrained to be equal.

TEMPERATURE: WORK-RELATED HRI

The likelihood ratio test indicated that the HRI-temperature model with zero lags was not substantially different from a model with lags of three days except for the Melbourne and Miami/Key West regions. For these two regions the best fit was a lag of three days. To confirm the results from the likelihood ratio test, the analysis was run with all lag terms. However, no effect was observed for lag2 and lag3. There was very little change in the effect estimates and variance for lag0 and lag1 when the additional lag days were removed from the model. As there were no changes, the simpler model was used.

Table C-3. Assessment of the number of lag days to be included when modeling the relationship between work-related HRI and maximum daily temperature (2005–2012)

NWS Region	Model Lag Terms	N	AIC*	Likelihood	LRT†	p-value
Pensacola	lag0, lag1, lag23	51700	3015.26	-1486.63		
	lag0, lag1	51700	3014.39	-1487.20	1.1	0.3
	lag 0	51700	3012.52	-1487.26	0.1	0.7
Tallahassee	lag0, lag1, lag23	119369	3215.38	-1586.69		
	lag0, lag1	119369	3214.85	-1587.42	1.5	0.2
	lag 0	119369	3214.75	-1588.37	1.9	0.2
Jacksonville	lag0, lag1, lag23	201314	5756.10	-2857.05		
	lag0, lag1	201314	5754.41	-2857.20	0.3	0.6
	lag 0	201314	5754.31	-2858.15	1.9	0.2
Melbourne	lag0, lag1, lag23	257874	8358.00	-4158.00		
	lag0, lag1	257874	8365.20	-4162.60	9.2	0.002
	lag 0	257874	8363.54	-4162.77	0.3	0.6
Tampa	lag0, lag1, lag23	422081	14796.56	-7377.28		
	lag0, lag1	422081	14798.35	-7379.17	3.8	0.05
	lag 0	422081	14802.81	-7382.41	6.5	0.01
Miami	lag0, lag1, lag23	312551	8374.10	-4166.05		
	lag0, lag1	312551	8384.76	-4172.38	12.7	<0.001
	lag 0	312551	8382.82	-4172.41	0.1	0.8

*Akaike information criterion: a lower value indicates a better model fit.
†Likelihood ratio test. Each row is compared to the prior row (e.g., lag0 versus lag0-1).

Table C-4. Work-related HRI rate ratio and 95% confidence interval for a 1°F increase in maximum daily temperature on the current or on each of the prior three days (2005–2012)*

	RR (95% CI)
Lag 0	1.139 (1.116, 1.162)
Lag 1	1.032 (1.013, 1.052)
Lag 2	0.988 (0.978, 0.998)
Lag 3	0.988 (0.978, 0.998)

*Summarized statewide estimates from six regional models (the Miami and Key West NWS regions were combined). Lag2 and Lag3 are constrained to be equal.

HEAT INDEX: NON-WORK-RELATED HRI

The likelihood ratio test indicated that the HRI-temperature model with a lag of three days was the best fit for all regions except Tallahassee and Key West. For Tallahassee the best fit was a lag of one day while the best fit for Key West did not include lags. Since the majority of regions include three lag days, the analysis was run with all lag terms. However, no effect was observed for lag2 and lag3. There was very little change in the effect estimates and variance for lag0 and lag1 when the additional lag days were removed from the model. As there were no changes, a simpler model was used instead.

Table C-5. Assessment of the number of lag days to be included when modeling the relationship between non-work-related HRI and maximum daily heat index (2005–2012)

NWS Region	Model Lag Terms	N	AIC*	Likelihood	LRT†	p-value
Pensacola	lag0, lag1, lag23	47797	13241.6	-6596.8		
	lag0, lag1	47797	13245.41	-6600.7	7.8	0.02
	lag 0	47797	13258.77	-6609.38	17.4	<0.001
Tallahassee	lag0, lag1, lag23	113406	17756.4797	-8854.2399		
	lag0, lag1	113406	17754.3659	-8855.183	1.9	0.39
	lag 0	113406	17758.7696	-8859.3848	8.4	0.01
Jacksonville	lag0, lag1, lag23	193670	35735.9968	-17844.00		
	lag0, lag1	193670	35742.1254	-17849.06	10.1	0.01
	lag 0	193670	35763.1402	-17861.57	25.0	<0.001
Melbourne	lag0, lag1, lag23	251998	52815.4815	-26383.74		
	lag0, lag1	251998	52818.92	-26387.46	7.4	0.02
	lag 0	251998	52845.8127	-26402.91	30.9	<0.001
Tampa	lag0, lag1, lag23	413812	77537.297	-38744.65		
	lag0, lag1	413812	77582.4707	-38769.24	49.2	<0.001
	lag 0	413812	77601.0382	-38780.52	22.6	<0.001
Miami	lag0, lag1, lag23	300633	53605.2063	-26778.60		
	lag0, lag1	300633	53613.191	-26784.60	12.0	0.002
	lag 0	300633	53618.032	-26789.016	8.8	0.01
Key West	lag0, lag1, lag23	7207	1057.71	-506.86		
	lag0, lag1	7207	1055.81	-506.91	0.1	0.75
	lag 0	7207	1054.71	-507.35	0.9	0.34

*Akaike information criterion: a lower value indicates a better model fit.
†Likelihood ratio test. Each row is compared to the prior row (e.g., lag0 versus lag0-1).

Table C-6. Non-work-related HRI rate ratio and 95% confidence interval for a 1°F increase in maximum daily heat index on the current or on each of the prior three days (2005–2012)*

	Below 101°F RR (95% CI)	Above 101°F RR (95% CI)
Lag 0	1.088 (1.082, 1.093)	1.057 (1.045, 1.069)
Lag 1	1.022 (1.015, 1.028)	1.019 (1.009, 1.030)
Lag 2	0.996 (0.992, 1.000)	0.995 (0.987, 1.003)
Lag 3	0.996 (0.992, 1.000)	0.995 (0.987, 1.003)

*Summarized statewide estimates from seven regional models. Lag_2 and Lag_3 are constrained to be equal.

HEAT INDEX: WORK-RELATED HRI

The likelihood ratio test indicated that for the Melbourne NWS region the HRI-heat index model with three lag days provided the best fit. For the Tallahassee and Miami/Key West regions a single lag day was not substantially different from a model with three lag days. Within all other regions, the likelihood ratio test supported a model with zero lag days. To confirm the results from the likelihood ratio test, the analysis was run with all lag terms. However, no effect was observed for lag2 and lag3. There was very little change in the effect estimates and variance for lag0 and lag1 when the additional lag days were removed from the model. As there were no changes, the simpler model was used.

Table C-7. Assessment of the number of lag days to be included when modeling the relationship between work-related HRI and maximum daily heat index (2005–2012)

NWS Region	Model Lag Terms	N	AIC*	Likelihood	LRT†	p-value
Pensacola	lag0, lag1, lag23	47797	2960.25	-1456.13		
	lag0, lag1	47797	2961.22	-1458.61	4.97	0.08
	lag 0	47797	2958.30	-1459.15	1.08	0.6
Tallahassee	lag0, lag1, lag23	113406	3093.52	-1522.76		
	lag0, lag1	113406	3090.63	-1523.31	1.11	0.6
	lag 0	113406	3103.50	-1531.75	16.87	<0.001
Jacksonville	lag0, lag1, lag23	193670	5637.73	-2794.86		
	lag0, lag1	193670	5634.39	-2795.20	0.66	0.7
	lag 0	193670	5631.49	-2795.75	1.10	0.6
Melbourne	lag0, lag1, lag23	251998	8266.52	-4109.26		
	lag0, lag1	251998	8272.26	-4114.13	9.74	0.01
	lag 0	251998	8269.50	-4114.75	1.24	0.5
Tampa	lag0, lag1, lag23	413812	14630.57	-7291.28		
	lag0, lag1	413812	14631.82	-7293.91	5.25	0.07
	lag 0	413812	14633.08	-7296.54	5.26	0.07
Miami	lag0, lag1, lag23	307840	8293.35	-4122.67		
	lag0, lag1	307840	8294.60	-4125.30	5.25	0.07
	lag 0	307840	8297.01	-4128.50	6.41	0.04

*Akaike information criterion: a lower value indicates a better model fit.
†Likelihood ratio test. Each row is compared to the prior row (e.g., lag0 versus lag0-1).

Table C-8. Work-related HRI rate ratio and 95% confidence interval for a 1°F increase in maximum daily heat index on the current or on each of the prior three days (2005–2012)*

	Below 99°F RR (95% CI)	Above 99°F RR (95% CI)
Lag 0	1.129 (1.104, 1.155)	1.068 (1.044, 1.093)
Lag 1	1.029 (1.007, 1.050)	1.038 (1.010, 1.067)
Lag 2	0.989 (0.980, 0.998)	0.994 (0.979, 1.010)
Lag 3	0.989 (0.980, 0.998)	0.994 (0.979, 1.010)

*Summarized statewide estimates from seven regional models. Lag2 and Lag3 are constrained to be equal.

APPENDIX D. MODIFICATION BY MORBIDITY DATA SOURCE

Modification of the relationship between maximum daily heat index and daily HRI morbidity by data source was assessed at the individual-level and is summarized below. The morbidity data sources were ED visit data and hospital discharge data. Individuals treated in the ED and released were included in the ED visit data while those admitted to the hospital regardless of source were captured in the hospital discharge data.

The population averaged heat-health relationship modified by data source. A binary interaction term was included in each zip code-station group model to represent the data source: $log[E(Y_i)] = \log(py) + \alpha + \sum_{j=1}^{p} g_j(x_{ij}) + m(\cdot) + z_i + m(\cdot) \times z_i$ where z_i is the indicator term (hospitalization versus ED visit) and as described in the methods section for Chapter 7 $m(\cdot)$ is the exposure term. The heat-health relationship modified by data source was then summarized at the state level using an intercept only random effects meta-regression analysis. Note that there were five zip code-station groups where the model did not converge and were therefore not included in the meta-regression analysis. The results indicate that the slope of the exposure-response curve did not vary by data source (the interaction terms in the Table D-1). However, the intercept of the exposure-response curve did vary by data source, with lower HRI hospitalization rates than ED rates HRI. The same difference in HRI rates by data source was also observed in Chapter 5.

Table D-1. Multi-variate meta-regression model results summarizing the coefficients from each zip code-station group model

Model coefficients	Estimate	Standard Error	z-score (p-value
ln(RR) for 1°F increase in heat index below the breakpoint of 101°F	0.105	0.003	40.32 (<0.001)
ln(RR) for 1°F increase in heat index above the breakpoint of 101°F	-0.026	0.005	-5.28 (<0.001)
ln(RR) comparing data sources (Hospital versus ED)	-1.638	0.036	-46.20 (<0.001)
Interaction term below breakpoint	0.002	0.004	0.50 (0.5)
Interaction term above breakpoint	0.006	0.010	0.57 (0.6)

REFERENCES

1. Basu, R., *High ambient temperature and mortality: a review of epidemiologic studies from 2001 to 2008.* Environ Health, 2009. **8**: p. 40.

2. Basu, R. and J.M. Samet, *Relation between Elevated Ambient Temperature and Mortality: A Review of the Epidemiologic Evidence.* Epidemiologic Reviews, 2002. **24**(2): p. 190-202.

3. Goldman, R.F., *Introduction to heat-related problems in military operations*, in *Medical aspects of harsh environments*, K.B. Pandolf, et al., Editors. 2001, Office of The Surgeon General; Department of the Army, United States of America: Washington, D.C. p. 3-49.

4. Guo, Y., et al., *The impact of temperature on mortality in Tianjin, China: a case-crossover design with a distributed lag nonlinear model.* Environ Health Perspect, 2011. **119**(12): p. 1719-25.

5. National Weather Service. *Weather Fatalities*. Natural Hazard Statistics 18 March 2016 30 March 2016]; Available from: http://www.nws.noaa.gov/om/hazstats.shtml.

6. Kovats, R.S. and L.E. Kristie, *Heatwaves and public health in Europe.* Eur J Public Health, 2006. **16**(6): p. 592-9.

7. Klinenberg, E., *Heat wave : a social autopsy of disaster in Chicago*. 2002, Chicago: University of Chicago Press. xvii, 305 p.

8. Kosatsky, T., *The 2003 European heat waves.* Euro surveillance, 2005. **10**(7): p. 148.

9. Romero-Lankao, P., H. Qin, and K. Dickinson, *Urban vulnerability to temperature-related hazards: A meta-analysis and meta-knowledge approach.* Global Environmental Change, 2012. **22**(3): p. 670-683.

10. Gubernot, D.M., G.B. Anderson, and K.L. Hunting, *The epidemiology of occupational heat exposure in the United States: a review of the literature and assessment of research needs in a changing climate.* Int J Biometeorol, 2014. **58**(8): p. 1779-88.

11. Gosling, S.N., et al., *Associations between elevated atmospheric temperature and human mortality: a critical review of the literature.* Climatic Change, 2008. **92**(3-4): p. 299-341.

12. Choudhary, E. and A. Vaidyanathan, *Heat stress illness hospitalizations--environmental public health tracking program, 20 States, 2001-2010.* MMWR Surveill Summ, 2014. **63**(13): p. 1-10.

13. Luber, G. and M. McGeehin, *Climate change and extreme heat events.* Am J Prev Med, 2008. **35**(5): p. 429-35.

14. McGeehin, M.A. and M. Mirabelli, *The potential impacts of climate variability and change on temperature-related morbidity and mortality in the United States.* Environ Health Perspect, 2001. **109 Suppl 2**: p. 185-9.

15. IPCC (Intergovernmental Panel on Climate Change), *Climate Change 2007: Impacts, Adaptation and Vulnerability*, in *Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change, 2007*, M.L. Parry, et al., Editors. 2007, Cambridge University Press: Cambridge, UK.

16. Kinney, P.L., et al., *Approaches for estimating effects of climate change on heat-related deaths: challenges and opportunities.* Environmental Science & Policy, 2008. **11**(1): p. 87-96.

17. Curriero, F.C., et al., *Temperature and mortality in 11 cities of the eastern United States.* Am J Epidemiol, 2002. **155**(1): p. 80-7.

18. Anderson, B.G. and M.L. Bell, *Weather-related mortality: how heat, cold, and heat waves affect mortality in the United States.* Epidemiology, 2009. **20**(2): p. 205-13.

19. Kalkstein, L.S. and R.E. Davis, *Weather and Human Mortality: An Evaluation of Demographic and Interregional Responses in the United States.* Annals of the Association of American Geographers, 1989. **79**(1): p. 44-64.

20. IPCC (Intergovernmental Panel on Climate Change), *Climate Change 2007: Synthesis Report. Contribution of Working Groups I, II and III to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*, R.K. Pachauri and A. Reisinger, Editors. 2007, IPCC: Geneva, Switzerland.

21. IPCC (Intergovernmental Panel on Climate Change), *Climate Change 2007: The Physical Science Basis.*, in *Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change.* 2007, Cambridge University Press: Cambridge, UK.

22. *A roman experience with heat stroke in 24 B.C.* Bull N Y Acad Med, 1967. **43**(8): p. 767-8.

23. Blackburn, S., *The Oxford dictionary of philosophy*. 2nd ed. 2008, Oxford ;: Oxford University Press. 407 p.

24. Wilson, W.C., et al., *Hypothermia and Heat Related Injuries*, in *Trauma: Emergency Resuscitation, Perioperative Anesthesia, Surgical Management*, W.C. Wilson, C.M. Grande, and D.B. Hoyt, Editors. 2007, Informa Healthcare: New York. p. 795-820.

25. Bricknell, M.C., *Heat illness--a review of military experience (Part 1).* J R Army Med Corps, 1995. **141**(3): p. 157-66.

26. Levick, J.J., *Remarks on Sunstroke*. 1859: Philadelphia, United States, Philadelphia. p. 40-40.

27. Marx, J.A., et al., *Rosen's emergency medicine concepts and clinical practice*. 2010, Mosby/Elsevier,: Philadelphia. p. 1 online resource ill. (chiefly col.).

28. Mettler, C.C., F.A. Mettler, and B.R. Wilcox, *History of medicine; a correlative text, arranged according to subjects*. 1947, Philadelphia etc.: Blakiston. xxix, 1215 p.

29. Kiple, K.F., *The Cambridge world history of human disease*. 1993, Cambridge ; New York: Cambridge University Press. xxiv, 1176 p.

30. Spellissy, J.M., *A review of the study and treatment of heatstroke at the pennsylvania hosptial and elsewhere, 1751-1870*. 1902: Philadelphia, United States, Philadelphia. p. 485-485.

31. Wakefield Eg, H.W., *Heat injuries: A preparatory study for experimental heat-stroke.* JAMA: The Journal of the American Medical Association, 1927. **89**(2): p. 92-95.

32. North, S.N.D., W.F. Willcox, and H. Gannett, *Abstract of the twelfth census of the United States, 1900*. 1902: Govt. print. off.

33. Shattuck, G.C. and M.M. Hilferty, *Sunstroke and Allied Conditions in the United States.* The American Journal of Tropical Medicine and Hygiene, 1932. **s1-12**(3): p. 223-245.

34. Shattuck, G.C. and M.M. Hilferty, *Causes of Deaths from Heat in Massachusetts.* Trans Am Clin Climatol Assoc, 1933. **49**: p. 161-84.

35. Gover, M., *Mortality during Periods of Excessive Temperature.* Public Health Reports (1896-1970), 1938. **53**(27): p. 1122-1143.

36. Schuman, S.H., *Patterns of urban heat-wave deaths and implications for prevention: data from New York and St. Louis during July, 1966.* Environ Res, 1972. **5**(1): p. 59-75.

37. Schickele, E., *Environment and fatal heat stroke; an analysis of 157 cases occurring in the Army in the U.S. during World War II.* Mil Surg, 1947. **100**(3): p. 235-56.

38. Kravchenko, J., et al., *Minimization of heatwave morbidity and mortality.* Am J Prev Med, 2013. **44**(3): p. 274-82.

39. Hajat, S., M. O'Connor, and T. Kosatsky, *Health effects of hot weather: from awareness of risk factors to effective health protection.* Lancet, 2010. **375**(9717): p. 856-63.

40. Hanna, E.G., et al., *Climate change and rising heat: population health implications for working people in Australia.* Asia Pac J Public Health, 2011. **23**(2 Suppl): p. 14S-26.

41. Sawaka, M.N., C.B. Wenoer, and K.B. Pandolf, *Thermoregulatory responses to acute exercise-heat stress and heat acclimation.*, in *Handbook of physiology: Environmental physiology*, M.J. Fregly and C.M. Blatteis, Editors. 1996, Published by the American Physiological Society by Oxford University Press: New York. p. 157-185.

42. Jay, O. and G.P. Kenny, *Heat exposure in the Canadian workplace.* Am J Ind Med, 2010. **53**(8): p. 842-53.

43. Seeley, R.R., T.D. Stephens, and P. Tate, *Anatomy & physiology*. 8th ed. 2008, Boston: McGraw Hill. xxi, 1116, 42, 32, 2, 45 p.

44. Parsons, K.C., *Human thermal environments : the effects of hot, moderate, and cold environments on human health, comfort, and performance*. 2nd ed. 2003, London ; New York: Taylor & Francis. xxiv, 527 p.

45. Gagge, A.P. and R.R. Gonzalez., *Mechanisms of Heat Exchange: Biophysics and Physiology*, in *Handbook of physiology, Environmental physiology*, M.J. Fregly and C.M. Blatteis, Editors. 1996, Published by the American Physiological Society by Oxford University Press: New York. p. 60.

46. Wenger, C.B., *Human Adaption to hot environments*, in *Medical aspects of harsh environments*, K.B. Pandolf, et al., Editors. 2001, Office of The Surgeon General; Department of the Army, United States of America: Washington, D.C. p. 51-86.

47. Guyton, A.C. and J.E. Hall, *Textbook of medical physiology*. 11th ed. 2006, Philadelphia: Elsevier Saunders. xxxv, 1116 p.

48. Lugo-Amador, N.M., T. Rothenhaus, and P. Moyer, *Heat-related illness.* Emerg Med Clin North Am, 2004. **22**(2): p. 315-27, viii.

49. Hall, J.E. and A.C. Guyton, *Guyton and Hall textbook of medical physiology*. 12th ed. 2011, Philadelphia, PA: Saunders/Elsevier. xix, 1091 p.

50. Vander, A.J., J.H. Sherman, and D.S. Luciano, *Human physiology : the mechanisms of body function*. 8th ed. 2001, Boston, Mass.: McGraw-Hill. xxviii, 800 p.

51. Charkoudian, N., *Skin blood flow in adult human thermoregulation: how it works, when it does not, and why.* Mayo Clin Proc, 2003. **78**(5): p. 603-12.

52. Bouchama, A. and J.P. Knochel, *Heat stroke.* N Engl J Med, 2002. **346**(25): p. 1978-88.

53. Cabanac, M., *Heat stress and behavior.*, in *Handbook of physiology, Environmental physiology*, M.J. Fregly and C.M. Blatteis, Editors. 1996, Published by the American Physiological Society by Oxford University Press: New York. p. 261-284.

54. Falk, B., *Effects of thermal stress during rest and exercise in the paediatric population.* Sports Med, 1998. **25**(4): p. 221-40.

55. Grubenhoff, J.A., K. du Ford, and G.E. Roosevelt, *Heat-Related Illness.* Clinical Pediatric Emergency Medicine, 2007. **8**(1): p. 59-64.

56. Hales, J.R.S., R.W. Hubbard, and S.L. Gaffin, *Limitation of heat tolerance*, in *Handbook of physiology, Environmental physiology*, M.J. Fregly and C.M. Blatteis, Editors. 1996, Published by the American Physiological Society by Oxford University Press: New York. p. 285-355.

57. Levine, M., et al., *Influence of drug use on morbidity and mortality in heatstroke.* J Med Toxicol, 2012. **8**(3): p. 252-7.

58. Howe, A.S. and B.P. Boden, *Heat-related illness in athletes.* Am J Sports Med, 2007. **35**(8): p. 1384-95.

59. Simon, H.B., *Hyperthermia.* N Engl J Med, 1993. **329**(7): p. 483-7.

60. Barrow, M.W. and K.A. Clark, *Heat-related illnesses.* Am Fam Physician, 1998. **58**(3): p. 749-56, 759.

61. Shen, T., et al., *Toward a broader definition of heat-related death: comparison of mortality estimates from medical examiners' classification with those from total death differentials during the July 1995 heat wave in Chicago, Illinois.* Am J Forensic Med Pathol, 1998. **19**(2): p. 113-8.

62. Hajat, S. and T. Kosatky, *Heat-related mortality: a review and exploration of heterogeneity.* J Epidemiol Community Health, 2010. **64**(9): p. 753-60.

63. Yeo, T.P., *Heat stroke: a comprehensive review.* AACN Clin Issues, 2004. **15**(2): p. 280-93.

64. Schwartz, J., *Who is Sensitive to Extremes of Temperature?* Epidemiology, 2005. **16**(1): p. 67-72.

65. Hansen, A.L., et al., *The effect of heat waves on hospital admissions for renal disease in a temperate city of Australia.* Int J Epidemiol, 2008. **37**(6): p. 1359-65.

66. Ye, X., et al., *Ambient temperature and morbidity: a review of epidemiological evidence.* Environ Health Perspect, 2012. **120**(1): p. 19-28.

67. Basu, R. and B.D. Ostro, *A multicounty analysis identifying the populations vulnerable to mortality associated with high ambient temperature in California.* Am J Epidemiol, 2008. **168**(6): p. 632-7.

68. Rey, G., et al., *The impact of major heat waves on all-cause and cause-specific mortality in France from 1971 to 2003.* Int Arch Occup Environ Health, 2007. **80**(7): p. 615-26.

69. Kovats, R.S., S. Hajat, and P. Wilkinson, *Contrasting patterns of mortality and hospital admissions during hot weather and heat waves in Greater London, UK.* Occup Environ Med, 2004. **61**(11): p. 893-8.

70. Wang, X.Y., et al., *The impact of heatwaves on mortality and emergency hospital admissions from non-external causes in Brisbane, Australia.* Occup Environ Med, 2012. **69**(3): p. 163-9.

71. Michelozzi, P., et al., *High temperature and hospitalizations for cardiovascular and respiratory causes in 12 European cities.* Am J Respir Crit Care Med, 2009. **179**(5): p. 383-9.

72. Linares, C. and J. Diaz, *Impact of high temperatures on hospital admissions: comparative analysis with previous studies about mortality (Madrid).* Eur J Public Health, 2008. **18**(3): p. 317-22.

73. Kilbourne, E.M., *The spectrum of illness during heat waves.* Am J Prev Med, 1999. **16**(4): p. 359-60.

74. Vaneckova, P., et al., *Effect of temperature on mortality during the six warmer months in Sydney, Australia, between 1993 and 2004.* Environ Res, 2008. **108**(3): p. 361-9.

75. Semenza, J.C., et al., *Excess hospital admissions during the July 1995 heat wave in Chicago.* Am J Prev Med, 1999. **16**(4): p. 269-77.

76. Keatinge, W.R., et al., *Increased platelet and red cell counts, blood viscosity, and plasma cholesterol levels during heat stress, and mortality from coronary and cerebral thrombosis.* Am J Med, 1986. **81**(5): p. 795-800.

77. Kilbourne, E., *Heat waves and hot environments*, in *The Public Health Consequences of Disasters*, E. Noji, Editor. 1997, Oxford University Press: New York. p. 245-269.

78. Basu, R., et al., *The effect of high ambient temperature on emergency room visits.* Epidemiology, 2012. **23**(6): p. 813-20.

79. Mastrangelo, G., et al., *Pattern and determinants of hospitalization during heat waves: an ecologic study.* BMC Public Health, 2007. **7**: p. 200.

80. Schwartz, J., J.M. Samet, and J.A. Patz, *Hospital admissions for heart disease: the effects of temperature and humidity.* Epidemiology, 2004. **15**(6): p. 755-61.

81. Lin, S., et al., *Extreme high temperatures and hospital admissions for respiratory and cardiovascular diseases.* Epidemiology, 2009. **20**(5): p. 738-46.

82. Green, R.S., et al., *The effect of temperature on hospital admissions in nine California counties.* Int J Public Health, 2010. **55**(2): p. 113-21.

83. Nitschke, M., G.R. Tucker, and P. Bi, *Morbidity and mortality during heatwaves in metropolitan Adelaide.* Med J Aust, 2007. **187**(11-12): p. 662-5.

84. Basagana, X., et al., *Heat waves and cause-specific mortality at all ages.* Epidemiology, 2011. **22**(6): p. 765-72.

85. Gasparrini, A., et al., *The effect of high temperatures on cause-specific mortality in England and Wales.* Occup Environ Med, 2012. **69**(1): p. 56-61.

86. Williams, S., et al., *Heat and health in Adelaide, South Australia: assessment of heat thresholds and temperature relationships.* Sci Total Environ, 2012. **414**: p. 126-33.

87. Knowlton, K., et al., *The 2006 California heat wave: impacts on hospitalizations and emergency department visits.* Environ Health Perspect, 2009. **117**(1): p. 61-7.

88. Ye, F., et al., *Effects of temperature and air pollutants on cardiovascular and respiratory diseases for males and females older than 65 years of age in Tokyo, July and August 1980-1995.* Environ Health Perspect, 2001. **109**(4): p. 355-9.

89. Koken, P.J., et al., *Temperature, air pollution, and hospitalization for cardiovascular diseases among elderly people in Denver.* Environ Health Perspect, 2003. **111**(10): p. 1312-7.

90. Braga, A.L., A. Zanobetti, and J. Schwartz, *The effect of weather on respiratory and cardiovascular deaths in 12 U.S. cities.* Environ Health Perspect, 2002. **110**(9): p. 859-63.

91. Medina-Ramon, M. and J. Schwartz, *Temperature, temperature extremes, and mortality: a study of acclimatisation and effect modification in 50 US cities.* Occup Environ Med, 2007. **64**(12): p. 827-33.

92. Basu, R., W.Y. Feng, and B.D. Ostro, *Characterizing temperature and mortality in nine California counties.* Epidemiology, 2008. **19**(1): p. 138-45.

93. Kenny, G.P., et al., *Heat stress in older individuals and patients with common chronic diseases.* CMAJ, 2010. **182**(10): p. 1053-60.

94. Noakes, T.D., *A modern classification of the exercise-related heat illnesses.* J Sci Med Sport, 2008. **11**(1): p. 33-9.

95. Fletcher, B.A., et al., *Association of summer temperatures with hospital admissions for renal diseases in New York State: a case-crossover study.* Am J Epidemiol, 2012. **175**(9): p. 907-16.

96. Armstrong, L.E., et al., *Exertional Heat Illness during Training and Competition.* Medicine & Science in Sports & Exercise, 2007. **39**(3): p. 556-572 10.1249/MSS.0b013e31802fa199.

97. Yardley, J.E., et al., *Is whole-body thermoregulatory function impaired in type 1 diabetes mellitus?* Curr Diabetes Rev, 2013. **9**(2): p. 126-36.

98. US EPA, *Excessive Heat Events Guidebook*, Office of Atmospheric Programs, Editor. 2006: Washington DC.

99. McLellan, T.M. and G.A. Selkirk, *The management of heat stress for the firefighter: a review of work conducted on behalf of the Toronto Fire Service.* Ind Health, 2006. **44**(3): p. 414-26.

100. Nunneley, S.A. and M.J. Reardon, *Prevention of heat illness*, in *Medical aspects of harsh environments*, K.B. Pandolf and R.E. Burr, Editors. 2001, Office of The Surgeon General; Department of the Army, United States of America: Washington, D.C. p. 209-230.

101. United States. Occupational Safety and Health Administration., *Health Hazard: Heat Stress*, in *OSHA technical manual*. 1999, Occupational Safety & Health Administration,: Washington, D.C.

102. Dolney, T.J. and S.C. Sheridan, *The relationship between extreme heat and ambulance response calls for the city of Toronto, Ontario, Canada.* Environ Res, 2006. **101**(1): p. 94-103.

103. Michelozzi, P., et al., *Temperature and summer mortality: geographical and temporal variations in four Italian cities.* J Epidemiol Community Health, 2006. **60**(5): p. 417-23.

104. Rainham, D.G., et al., *Synoptic weather patterns and modification of the association between air pollution and human mortality.* Int J Environ Health Res, 2005. **15**(5): p. 347-60.

105. Sheridan, S.C. and T.J. Dolney, *Heat, mortality, and level of urbanization: measuring vulnerability across Ohio, USA.* Climate research, 2003. **24**: p. 255-265.

106. Sheridan, S.C. and L.S. Kalkstein, *Progress in Heat Watch–Warning System Technology.* Bulletin of the American Meteorological Society, 2004. **85**(12): p. 1931-1941.

107. Kalkstein, L., S. Sheridan, and A. Kalkstein, *Heat/Health Warning Systems: Development, Implementation, and Intervention Activities*, in *Biometeorology for Adaptation to Climate Variability and Change*, K. Ebi, I. Burton, and G. McGregor, Editors. 2009, Springer Netherlands. p. 33-48.

108. Synoptic Climatology Lab. *Operational Heat/Health Watch-Warning Systems*. 2009 December 2009 [cited 2012 29 November]; Available from: http://www.as.miami.edu/geography/research/climatology/OtherWWS.html.

109. Sheridan, S.C., *The redevelopment of a weather-type classification scheme for North America.* International Journal of Climatology, 2002. **22**(1): p. 51-68.

110. Hajat, S., et al., *Heat-health warning systems: a comparison of the predictive capacity of different approaches to identifying dangerously hot days.* Am J Public Health, 2010. **100**(6): p. 1137-44.

111. Kininmonth, W., *Climate change: a natural hazard*. 2004: multi-science publishing.

112. Hajat, S., et al., *Impact of high temperatures on mortality: is there an added heat wave effect?* Epidemiology, 2006. **17**(6): p. 632-8.

113. Barnett, A.G., S. Tong, and A.C. Clements, *What measure of temperature is the best predictor of mortality?* Environ Res, 2010. **110**(6): p. 604-11.

114. Kalkstein, L.S., *A new approach to evaluate the impact of climate on human mortality.* Environ Health Perspect, 1991. **96**: p. 145-50.

115. Laaidi, K., et al., *The impact of heat islands on mortality in Paris during the August 2003 heat wave.* Environ Health Perspect, 2012. **120**(2): p. 254-9.

116. Lim, Y.H., A.K. Park, and H. Kim, *Modifiers of diurnal temperature range and mortality association in six Korean cities.* Int J Biometeorol, 2012. **56**(1): p. 33-42.

117. Steadman, R.G., *The Assessment of Sultriness. Part I: A Temperature-Humidity Index Based on Human Physiology and Clothing Science.* Journal of Applied Meteorology, 1979. **18**(7): p. 861-873.

118. Rothfusz, L.P., *The Heat Index "Equation" (or, More Than You Ever Wanted to Know About Heat Index)*, Scientific Services Divisions (NWS Southern Region Headquarters), Editor. 1990: Fort Worth, TX.

119. Kalkstein, L.S. and K.M. Valimont, *An Evaluation of Summer Discomfort in the United State Using a Relative Climatological Index.* Bulletin of the American Meteorological Society, 1986. **67**(7): p. 842-848.

120. Anderson, G.B., M.L. Bell, and R.D. Peng, *Methods to calculate the heat index as an exposure metric in environmental health research.* Environ Health Perspect, 2013. **121**(10): p. 1111-9.

121. Masterton, J.M. and F.A. Richardson, *Humidex ; a method of quantifying human discomfort due to excessive heat and humidity*. 1979, Downsview, Ont.: Environment Canada, Atmospheric Environment.

122. Smoyer-Tomic, K.E. and D.G. Rainham, *Beating the heat: development and evaluation of a Canadian hot weather health-response plan.* Environ Health Perspect, 2001. **109**(12): p. 1241-8.

123. Metzger, K.B., K. Ito, and T.D. Matte, *Summer heat and mortality in New York City: how hot is too hot?* Environ Health Perspect, 2010. **118**(1): p. 80-6.

124. Service, N.W. *The Heat Index Equation*. 08/03/2013 12/09/2013]; Available from: http://www.hpc.ncep.noaa.gov/html/heatindex_equation.shtml.

125. National Weather Service. *What is the Coop Program?* Cooperative Observer Program 25 April 2012 6 October 2012]; Available from: http://www.nws.noaa.gov/om/coop/what-is-coop.html.

126. National Weather Service Southern Region Headquarters. *Automated Surface Observing Systems (ASOS)*. JetStream - Online School for Weather 5 January 2010 6 October 2012]; Available from: http://www.srh.noaa.gov/jetstream/remote/asos.htm.

127. Pope, C.A., 3rd, *Mortality and air pollution: associations persist with continued advances in research methodology.* Environ Health Perspect, 1999. **107**(8): p. 613-4.

128. Ayres, J.G., R. Maynard, and R. Richards, *Air pollution and health*. Air pollution reviews. 2006, London/Hackensack, N.J.: Imperial College Press; distributed by World Scientific. xv, 248 p.

129. Reid, C.E., et al., *The Role of Ambient Ozone in Epidemiologic Studies of Heat-Related Mortality.* Environ Health Perspect, 2012.

130. Filleul, L., et al., *The Relation Between Temperature, Ozone, and Mortality in Nine French Cities During the Heat Wave of 2003.* Environmental Health Perspectives, 2006. **114**(9): p. 1344-1347.

131. Katsouyanni, K., et al., *Evidence for interaction between air pollution and high temperature in the causation of excess mortality.* Arch Environ Health, 1993. **48**(4): p. 235-42.

132. Pattenden, S., B. Nikiforov, and B.G. Armstrong, *Mortality and temperature in Sofia and London.* J Epidemiol Community Health, 2003. **57**(8): p. 628-33.

133. Ren, C., et al., *Ozone modifies associations between temperature and cardiovascular mortality: analysis of the NMMAPS data.* Occup Environ Med, 2008. **65**(4): p. 255-60.

134. Ren, C., G.M. Williams, and S. Tong, *Does Particulate Matter Modify the Association between Temperature and Cardiorespiratory Diseases?* Environmental Health Perspectives, 2006.

135. Zanobetti, A. and J. Schwartz, *Temperature and mortality in nine US cities.* Epidemiology, 2008. **19**(4): p. 563-70.

136. Centers for Disease Control Prevention, *Heat-related deaths--United States, 1999-2003.* MMWR Morb Mortal Wkly Rep, 2006. **55**(29): p. 796-8.

137. HCUPnet. *Healthcare Cost and Utilization Project. Agency for Healthcare Research and Quality*. [cited 2012 14 February]; Available from: http://hcupnet.ahrq.gov/.

138. Centers for Disease Control and Prevention, *Heat illness among high school athletes --- United States, 2005-2009.* MMWR Morb Mortal Wkly Rep, 2010. **59**(32): p. 1009-13.

139. Bureau of Labor Statistics, U.S.D.o.L., *1. Employment status of the civilian noninstitutional population, 1942 to date*, H.D.A. Averages, Editor. 2013.

140. Bureau of Labor Statistics, U.S.D.o.L. *Occupational Injuries/Illnesses and Fatal Injuries Profiles*. 16 January 2013]; Available from: http://data.bls.gov/gqt/InitialPage.

141. Kovats, R.S. and S. Hajat, *Heat stress and public health: a critical review.* Annu Rev Public Health, 2008. **29**: p. 41-55.

142. Knowlton, K., et al., *Six climate change-related events in the United States accounted for about $14 billion in lost lives and health costs.* Health Aff (Millwood), 2011. **30**(11): p. 2167-76.

143. Anderson, G.B. and M.L. Bell, *Heat waves in the United States: mortality risk during heat waves and effect modification by heat wave characteristics in 43 U.S. communities.* Environ Health Perspect, 2011. **119**(2): p. 210-8.

144. Bobb, J.F., F. Dominici, and R.D. Peng, *A Bayesian model averaging approach for estimating the relative risk of mortality associated with heat waves in 105 U.S. cities.* Biometrics, 2011. **67**(4): p. 1605-16.

145. Whitman, S., et al., *Mortality in Chicago attributed to the July 1995 heat wave.* Am J Public Health, 1997. **87**(9): p. 1515-8.

146. Robine, J.M., et al., *Death toll exceeded 70,000 in Europe during the summer of 2003.* C R Biol, 2008. **331**(2): p. 171-8.

147. Bonauto, D., E. Rauser, and L. Lim, *Occupational Heat Illness in Washington State, 2000-2009*, W.S.D.o.L.a. Industries, Editor. 2010.

148. Kark, J.A., et al., *Exertional heat illness in Marine Corps recruit training.* Aviat Space Environ Med, 1996. **67**(4): p. 354-60.

149. Bassil, K.L., et al., *The relationship between temperature and ambulance response calls for heat-related illness in Toronto, Ontario, 2005.* J Epidemiol Community Health, 2011. **65**(9): p. 829-31.

150. Golden, J.S., et al., *A biometeorology study of climate and heat-related morbidity in Phoenix from 2001 to 2006.* Int J Biometeorol, 2008. **52**(6): p. 471-80.

151. Hartz, D.A., et al., *Climate and heat-related emergencies in Chicago, Illinois (2003-2006).* Int J Biometeorol, 2012. **56**(1): p. 71-83.

152. Schaffer, A., et al., *Emergency department visits, ambulance calls, and mortality associated with an exceptional heat wave in Sydney, Australia, 2011: a time-series analysis.* Environ Health, 2012. **11**(1): p. 3.

153. Rydman, R.J., et al., *The rate and risk of heat-related illness in hospital emergency departments during the 1995 Chicago heat disaster.* J Med Syst, 1999. **23**(1): p. 41-56.

154. Jackson, L.L. and H.R. Rosenberg, *Preventing heat-related illness among agricultural workers.* J Agromedicine, 2010. **15**(3): p. 200-15.

155. Nitschke, M., et al., *Impact of two recent extreme heat episodes on morbidity and mortality in Adelaide, South Australia: a case-series analysis.* Environ Health, 2011. **10**: p. 42.

156. Yip, F.Y., et al., *The impact of excess heat events in Maricopa County, Arizona: 2000--2005.* Int J Biometeorol, 2008. **52**(8): p. 765-72.

157. Gomez-Acebo, I., et al., *Extreme temperatures and mortality in the North of Spain.* Int J Public Health, 2012. **57**(2): p. 305-13.

158. Kunst, A.E., C.W. Looman, and J.P. Mackenbach, *Outdoor air temperature and mortality in The Netherlands: a time-series analysis.* Am J Epidemiol, 1993. **137**(3): p. 331-41.

159. Chung, J.Y., et al., *Ambient temperature and mortality: an international study in four capital cities of East Asia.* Sci Total Environ, 2009. **408**(2): p. 390-6.

160. Yu, W., et al., *Daily average temperature and mortality among the elderly: a meta-analysis and systematic review of epidemiological evidence.* Int J Biometeorol, 2012. **56**(4): p. 569-81.

161. Yu, W., et al., *Is the association between temperature and mortality modified by age, gender and socio-economic status?* Sci Total Environ, 2010. **408**(17): p. 3513-8.

162. Baccini, M., et al., *Heat Effects on Mortality in 15 European Cities.* Epidemiology, 2008. **19**(5): p. 711-719.

163. Hajat, S., et al., *Impact of hot temperatures on death in London: a time series approach.* J Epidemiol Community Health, 2002. **56**(5): p. 367-72.

164. Hajat, S., et al., *Mortality Displacement of Heat-Related Deaths.* Epidemiology, 2005. **16**(5): p. 613-620.

165. Hashizume, M., et al., *The effect of temperature on mortality in rural Bangladesh--a population-based time-series study.* Int J Epidemiol, 2009. **38**(6): p. 1689-97.

166. Rocklov, J., K. Ebi, and B. Forsberg, *Mortality related to temperature and persistent extreme temperatures: a study of cause-specific and age-stratified mortality.* Occup Environ Med, 2011. **68**(7): p. 531-6.

167. Kan, H.D., J. Jia, and B.H. Chen, *Temperature and daily mortality in Shanghai: a time-series study.* Biomed Environ Sci, 2003. **16**(2): p. 133-9.

168. Ballester, F., et al., *Mortality as a function of temperature. A study in Valencia, Spain, 1991-1993.* Int J Epidemiol, 1997. **26**(3): p. 551-61.

169. Son, J.Y., et al., *The impact of heat waves on mortality in seven major cities in Korea.* Environ Health Perspect, 2012. **120**(4): p. 566-71.

170. Bell, M.L., et al., *Vulnerability to heat-related mortality in Latin America: a case-crossover study in Sao Paulo, Brazil, Santiago, Chile and Mexico City, Mexico.* Int J Epidemiol, 2008. **37**(4): p. 796-804.

171. O'Neill, M.S., A. Zanobetti, and J. Schwartz, *Disparities by race in heat-related mortality in four US cities: the role of air conditioning prevalence.* J Urban Health, 2005. **82**(2): p. 191-7.

172. Kaiser, R., et al., *The effect of the 1995 heat wave in Chicago on all-cause and cause-specific mortality.* Am J Public Health, 2007. **97 Suppl 1**: p. S158-62.

173. Kysely, J., *Mortality and displaced mortality during heat waves in the Czech Republic.* Int J Biometeorol, 2004. **49**(2): p. 91-7.

174. de'Donato, F.K., et al., *Airport and city-centre temperatures in the evaluation of the association between heat and mortality.* Int J Biometeorol, 2008. **52**(4): p. 301-10.

175. Tobias, A., et al., *Mortality on extreme heat days using official thresholds in Spain: a multi-city time series analysis.* BMC Public Health, 2012. **12**: p. 133.

176. Huynen, M.M., et al., *The impact of heat waves and cold spells on mortality rates in the Dutch population.* Environ Health Perspect, 2001. **109**(5): p. 463-70.

177. Curriero, F.C., J.M. Samet, and S.L. Zeger, *Re: "On the use of generalized additive models in time-series studies of air pollution and health" and "Temperature and mortality in 11 cities of the eastern United States".* Am J Epidemiol, 2003. **158**(1): p. 93-4.

178. Yu, W., et al., *The effect of various temperature indicators on different mortality categories in a subtropical city of Brisbane, Australia.* Sci Total Environ, 2011. **409**(18): p. 3431-7.

179. Michelozzi, P., et al., *The impact of the summer 2003 heat waves on mortality in four Italian cities.* Euro Surveill, 2005. **10**(7): p. 161-5.

180. Goodman, P.G., D.W. Dockery, and L. Clancy, *Cause-specific mortality and the extended effects of particulate pollution and temperature exposure.* Environ Health Perspect, 2004. **112**(2): p. 179-85.

181. Khalaj, B., et al., *The health impacts of heat waves in five regions of New South Wales, Australia: a case-only analysis.* Int Arch Occup Environ Health, 2010. **83**(7): p. 833-42.

182. National Institute for Occupational Safety and Health. *Heat Stress*. NIOSH Workplace Safety and Health Topics May 18, 2012 2/20/2013]; Available from: http://www.cdc.gov/niosh/topics/heatstress/.

183. Bricknell, M.C., *Heat illness--a review of military experience (Part 2).* J R Army Med Corps, 1996. **142**(1): p. 34-42.

184. Wasterlund, D.S., J. Chaseling, and L. Burstrom, *The effect of fluid consumption on the forest workers' performance strategy.* Appl Ergon, 2004. **35**(1): p. 29-36.

185. Wallace, R.F., et al., *The effects of continuous hot weather training on risk of exertional heat illness.* Med Sci Sports Exerc, 2005. **37**(1): p. 84-90.

186. Al-Tawheed, A.R., et al., *Anuria secondary to hot weather-induced hyperuricaemia: diagnosis and management.* Ann Saudi Med, 2003. **23**(5): p. 283-7.

187. Carter, R., 3rd, et al., *Epidemiology of hospitalizations and deaths from heat illness in soldiers.* Med Sci Sports Exerc, 2005. **37**(8): p. 1338-44.

188. Epstein, Y., D.S. Moran, and Y. Shapiro, *Exertional heatstroke in the Israeli Defence Forces*, in *Medical aspects of harsh environments*, K.B. Pandolf, et al., Editors. 2001, Office of The Surgeon General; Department of the Army, United States of America: Washington, D.C. p. 281-292.

189. Dickinson, J.G., *Heat illness in the services.* J R Army Med Corps, 1994. **140**(1): p. 7-12.

190. Dellinger, A.M., et al., *Risk of heat-related injury to disaster relief workers in a slow-onset flood disaster.* J Occup Environ Med, 1996. **38**(7): p. 689-92.

191. Donoghue, A.M., M.J. Sinclair, and G.P. Bates, *Heat exhaustion in a deep underground metalliferous mine.* Occup Environ Med, 2000. **57**(3): p. 165-74.

192. Donoghue, A.M., *Heat illness in the U.S. mining industry.* Am J Ind Med, 2004. **45**(4): p. 351-6.

193. Donoghue, A.M. and G.P. Bates, *The risk of heat exhaustion at a deep underground metalliferous mine in relation to surface temperatures.* Occup Med (Lond), 2000. **50**(5): p. 334-6.

194. Hunt, A.P., A.W. Parker, and I.B. Stewart, *Symptoms of heat illness in surface mine workers.* Int Arch Occup Environ Health, 2012.

195. Bonauto, D., et al., *Occupational heat illness in Washington State, 1995-2005.* Am J Ind Med, 2007. **50**(12): p. 940-50.

196. Prudhomme JC, N.A., *Cal/OSHA investigations of heat related illnesses 2005*. 2006, State of California.

197. Prudhomme JC, N.A., *Cal/OSHA investigations of heat related illnesses 2006*. 2007, State of California.

198. Mirabelli, M.C. and D.B. Richardson, *Heat-related fatalities in North Carolina.* Am J Public Health, 2005. **95**(4): p. 635-7.

199. Centers for Disease Control and Prevention, *Heat-related deaths among crop workers--United States, 1992--2006.* MMWR Morb Mortal Wkly Rep, 2008. **57**(24): p. 649-53.

200. Wu, P.C., et al., *Cardiovascular mortality during heat and cold events: determinants of regional vulnerability in Taiwan.* Occup Environ Med, 2011. **68**(7): p. 525-30.

201. Gouveia, N., S. Hajat, and B. Armstrong, *Socioeconomic differentials in the temperature-mortality relationship in Sao Paulo, Brazil.* Int J Epidemiol, 2003. **32**(3): p. 390-7.

202. Reid, C.E., et al., *Mapping community determinants of heat vulnerability.* Environ Health Perspect, 2009. **117**(11): p. 1730-6.

203. Harlan, S.L., et al., *Neighborhood effects on heat deaths: social and environmental predictors of vulnerability in maricopa county, Arizona.* Environ Health Perspect, 2013. **121**(2): p. 197-204.

204. Bi, P., et al., *The effects of extreme heat on human mortality and morbidity in Australia: implications for public health.* Asia Pac J Public Health, 2011. **23**(2 Suppl): p. 27S-36.

205. Nelson, N.G., et al., *Exertional heat-related injuries treated in emergency departments in the U.S., 1997-2006.* Am J Prev Med, 2011. **40**(1): p. 54-60.

206. Rhea, S., et al., *Using near real-time morbidity data to identify heat-related illness prevention strategies in North Carolina.* J Community Health, 2012. **37**(2): p. 495-500.

207. Centers for Disease Control and Prevention, *Heat-related illnesses, deaths, and risk factors--Cincinnati and Dayton, Ohio, 1999, and United States, 1979-1997.* MMWR Morb Mortal Wkly Rep, 2000. **49**(21): p. 470-3.

208. Lundgren, K., et al., *Effects of heat stress on working populations when facing climate change.* Ind Health, 2013. **51**(1): p. 3-15.

209. Ishigami, A., et al., *An ecological time-series study of heat-related mortality in three European cities.* Environ Health, 2008. **7**: p. 5.

210. O'Neill, M.S., A. Zanobetti, and J. Schwartz, *Modifiers of the temperature and mortality association in seven US cities.* Am J Epidemiol, 2003. **157**(12): p. 1074-82.

211. Wolf, K., et al., *Air temperature and the occurrence of myocardial infarction in Augsburg, Germany.* Circulation, 2009. **120**(9): p. 735-42.

212. Stafoggia, M., et al., *Vulnerability to heat-related mortality: a multicity, population-based, case-crossover analysis.* Epidemiology, 2006. **17**(3): p. 315-23.

213. Stafoggia, M., et al., *Factors affecting in-hospital heat-related mortality: a multi-city case-crossover analysis.* J Epidemiol Community Health, 2008. **62**(3): p. 209-15.

214. Piver, W.T., et al., *Temperature and air pollution as risk factors for heat stroke in Tokyo, July and August 1980-1995.* Environ Health Perspect, 1999. **107**(11): p. 911-6.

215. Kerr, Z.Y., et al., *Epidemiology of exertional heat illness among U.S. high school athletes.* Am J Prev Med, 2013. **44**(1): p. 8-14.

216. Centers for Disease Control and Prevention, *Nonfatal sports and recreation heat illness treated in hospital emergency departments--United States, 2001-2009.* MMWR Morb Mortal Wkly Rep, 2011. **60**(29): p. 977-80.

217. Bureau of Labor Statistics, U.S.D.o.L. *2009 Annual Averages - Household Data - Tables from Employment and Earnings*. Labor Force Statistics from the Current Population Survey 2010 February 26, 2014 March 10, 2014]; Available from: http://www.bls.gov/cps/cps_aa2009.htm.

218. Medina-Ramon, M., et al., *Extreme temperatures and mortality: assessing effect modification by personal characteristics and specific cause of death in a multi-city case-only analysis.* Environ Health Perspect, 2006. **114**(9): p. 1331-6.

219. Gardner, J.W., et al., *Risk factors predicting exertional heat illness in male Marine Corps recruits.* Med Sci Sports Exerc, 1996. **28**(8): p. 939-44.

220. Centers for Disease Control and Prevention, *Heat-related deaths--Los Angeles County, California, 1999-2000, and United States, 1979-1998.* MMWR Morb Mortal Wkly Rep, 2001. **50**(29): p. 623-6.

221. Greenberg, J.H., et al., *The epidemiology of heat-related deaths, Texas--1950, 1970-79, and 1980.* Am J Public Health, 1983. **73**(7): p. 805-7.

222. Naughton, M.P., et al., *Heat-related mortality during a 1999 heat wave in Chicago.* Am J Prev Med, 2002. **22**(4): p. 221-7.

223. Kim, Y. and S. Joh, *A vulnerability study of the low-income elderly in the context of high temperature and mortality in Seoul, Korea.* Sci Total Environ, 2006. **371**(1-3): p. 82-8.

224. Borrell, C., et al., *Socioeconomic position and excess mortality during the heat wave of 2003 in Barcelona.* Eur J Epidemiol, 2006. **21**(9): p. 633-40.

225. Martinez, B.F., et al., *Geographic distribution of heat-related deaths among elderly persons. Use of county-level dot maps for injury surveillance and epidemiologic research.* JAMA, 1989. **262**(16): p. 2246-50.

226. Hajat, S., R.S. Kovats, and K. Lachowycz, *Heat-related and cold-related deaths in England and Wales: who is at risk?* Occup Environ Med, 2007. **64**(2): p. 93-100.

227. Hansen, A., et al., *The effect of heat waves on mental health in a temperate Australian city.* Environ Health Perspect, 2008. **116**(10): p. 1369-75.

228. Epstein, Y., *Heat intolerance: predisposing factor or residual injury?* Med Sci Sports Exerc, 1990. **22**(1): p. 29-35.

229. O'Connor, F.G., et al., *American College of Sports Medicine Roundtable on exertional heat stroke--return to duty/return to play: conference proceedings.* Curr Sports Med Rep, 2010. **9**(5): p. 314-21.

230. Shapiro, Y., et al., *Heat intolerance in former heatstroke patients.* Ann Intern Med, 1979. **90**(6): p. 913-6.

231. Gomez, J.E., A.J. Grove, and W. McBride, *Environmental Stress: Heat Illness*, in *DeLee & Drez's orthopaedic sports medicine principles and practice*, J. DeLee, et al., Editors. 1994, Saunders/Elsevier: Philadelphia.

232. Braga, A.L., A. Zanobetti, and J. Schwartz, *The time course of weather-related deaths.* Epidemiology, 2001. **12**(6): p. 662-7.

233. Lane, K., et al., *Extreme Heat Awareness and Protective Behaviors in New York City.* J Urban Health, 2013.

234. U.S. Census Bureau, *American Housing Survey for the United States: 2011*. 2013, U.S. Government Printing Office: Washington, DC 20401.

235. O'Neill, M.S., *Air conditioning and heat-related health effects.* Appl Environ Sci Public Health, 2003. **1**: p. 9-12.

236. US EPA, Climate Protection Partnership Division, *Reducing Urban Heat Islands: Compendium of Strategies*, Office of Atmospheric Programs, Editor. 2008: Washington DC.

237. Reardon, C., C. McGee, and G. Milne, *Thermal Mass*, in *Your Home: Australia's guide to environmentally sustainable homes*, Commonwealth of Austrailia, Department of Industry, Editor. 2013. p. 178-187.

238. Vandentorren, S., et al., *August 2003 heat wave in France: risk factors for death of elderly people living at home.* Eur J Public Health, 2006. **16**(6): p. 583-91.

239. Hansen, A., et al., *Vulnerability to extreme heat and climate change: is ethnicity a factor?* Glob Health Action, 2013. **6**: p. 21364.

240. Balbus, J.M. and C. Malina, *Identifying vulnerable subpopulations for climate change health effects in the United States.* J Occup Environ Med, 2009. **51**(1): p. 33-7.

241. Loughnan, M., N. Nicholls, and N. Tapper, *Mortality-temperature thresholds for ten major population centres in rural Victoria, Australia.* Health Place, 2010. **16**(6): p. 1287-90.

242. Simón, F., et al., *Mortality in Spain during the heat waves of summer 2003.* Euro surveillance : bulletin européen sur les maladies transmissibles 2005. **10**(7): p. 156.

243. Gabriel, K.M. and W.R. Endlicher, *Urban and rural mortality rates during heat waves in Berlin and Brandenburg, Germany.* Environ Pollut, 2011. **159**(8-9): p. 2044-50.

244. Centers for Disease Control and Prevention, *Heat-related mortality--Latium Region, Italy, summer 1983.* MMWR Morb Mortal Wkly Rep, 1984. **33**(37): p. 518-21.

245. Jones, T.S., et al., *Morbidity and mortality associated with the July 1980 heat wave in St Louis and Kansas City, Mo.* JAMA, 1982. **247**(24): p. 3327-31.

246. Josseran, L., et al., *Syndromic surveillance and heat wave morbidity: a pilot study based on emergency departments in France.* BMC Med Inform Decis Mak, 2009. **9**: p. 14.

247. Dear, K., *Commentary: On Hashizume et al.'s 'The effect of temperature on mortality in rural Bangladesh'.* Int J Epidemiol, 2009. **38**(6): p. 1697-9.

248. Toulemon, L. and M. Barbieri, *The mortality impact of the August 2003 heat wave in France: investigating the 'harvesting' effect and other long-term consequences.* Popul Stud (Camb), 2008. **62**(1): p. 39-53.

249. Tong, S., X.Y. Wang, and A.G. Barnett, *Assessment of heat-related health impacts in Brisbane, Australia: comparison of different heatwave definitions.* PLoS One, 2010. **5**(8): p. e12155.

250. Robinson, P.J., *On the Definition of a Heat Wave.* Journal of Applied Meteorology, 2001. **40**(4): p. 762-775.

251. Gasparrini, A. and B. Armstrong, *The impact of heat waves on mortality.* Epidemiology, 2011. **22**(1): p. 68-73.

252. Weisskopf, M.G., et al., *Heat wave morbidity and mortality, Milwaukee, Wis, 1999 vs 1995: an improved response?* Am J Public Health, 2002. **92**(5): p. 830-3.

253. Mirchandani, H.G., et al., *Heat-related deaths in Philadelphia--1993.* Am J Forensic Med Pathol, 1996. **17**(2): p. 106-8.

254. Smoyer, K.E., *A comparative analysis of heat waves and associated mortality in St. Louis, Missouri--1980 and 1995.* Int J Biometeorol, 1998. **42**(1): p. 44-50.

255. Diaz, J., et al., *Heat waves in Madrid 1986-1997: effects on the health of the elderly.* Int Arch Occup Environ Health, 2002. **75**(3): p. 163-70.

256. Joubert, D., J. Thomsen, and O. Harrison, *Safety in the Heat: a comprehensive program for prevention of heat illness among workers in Abu Dhabi, United Arab Emirates.* Am J Public Health, 2011. **101**(3): p. 395-8.

257. McMichael, A.J., et al., *International study of temperature, heat and urban mortality: the 'ISOTHURM' project.* Int J Epidemiol, 2008. **37**(5): p. 1121-31.

258. Centers for Disease Control and Prevention, *Heat-related deaths -- Dallas, Wichita, and Cooke counties, Texas, and United States, 1996.* MMWR Morb Mortal Wkly Rep, 1997. **46**(23): p. 528-31.

259. Kottek, M., et al., *World Map of the Köppen-Geiger climate classification updated.* Meteorologische Zeitschrift, 2006. **15**(3): p. 259-263.

260. Ellis, F.P., et al., *Mortality and morbidity in Birmingham during the 1976 heatwave.* Q J Med, 1980. **49**(193): p. 1-8.

261. Kilbourne, E.M., et al., *Risk factors for heatstroke. A case-control study.* JAMA, 1982. **247**(24): p. 3332-6.

262. Applegate, W.B., et al., *Analysis of the 1980 heat wave in Memphis.* J Am Geriatr Soc, 1981. **29**(8): p. 337-42.

263. Lippmann, S.J., et al., *Ambient temperature and emergency department visits for heat-related illness in North Carolina, 2007-2008.* Environ Res, 2013. **124**: p. 35-42.

264. Pan, W.H., L.A. Li, and M.J. Tsai, *Temperature extremes and mortality from coronary heart disease and cerebral infarction in elderly Chinese.* Lancet, 1995. **345**(8946): p. 353-5.

265. Wang, X.Y., et al., *Temperature variation and emergency hospital admissions for stroke in Brisbane, Australia, 1996-2005.* Int J Biometeorol, 2009. **53**(6): p. 535-41.

266. Fouillet, A., et al., *A predictive model relating daily fluctuations in summer temperatures and mortality rates.* BMC Public Health, 2007. **7**: p. 114.

267. United States Public Health Service and Centers for Medicare & Medicaid Services (U.S.), *International classification of diseases, ninth revision, clinical modification*. 2010, Thomson Reuters,: St. Paul, Minn.

268. Cerutti, B., et al., *Temperature related mortality and ambulance service interventions during the heat waves of 2003 in Ticino (Switzerland).* Sozial- und Präventivmedizin SPM, 2006. **51**(4): p. 185-193.

269. Petitti, D.B., et al., *Multiple Trigger Points for Quantifying Heat-Health Impacts: New Evidence from a Hot Climate.* Environ Health Perspect, 2016. **124**(2): p. 176-83.

270. Mastrangelo, G., et al., *Contrasting patterns of hospital admissions and mortality during heat waves: are deaths from circulatory disease a real excess or an artifact?* Med Hypotheses, 2006. **66**(5): p. 1025-8.

271. Smith, S. and S. Cody, *Methodology for Constructing Estimates of Total Population for Counties and Subcounty Areas in Florida*, Bureau of Economic and Business Research, University of Florida, Editor. 2014.

272. U.S. Census Bureau, *Current Population Survey Design and Methodology Technical Paper 66*. 2006.

273. Office, A.C.S. *Guidance for Data Users*. American Community Survey 1 February 2013 2 April 2013]; Available from: http://www.census.gov/acs/www/guidance_for_data_users/guidance_main/.

274. Fogleman, M., L. Fakhrzadeh, and T.E. Bernard, *The relationship between outdoor thermal conditions and acute injury in an aluminum smelter.* International Journal of Industrial Ergonomics, 2005. **35**(1): p. 47-55.

275. World Health Organization., *International statistical classification of diseases and related health problems*. 10th revision, 2010 ed. 2011, Geneva: World Health Organization.

276. Alamgir, H., et al., *An evaluation of hospital discharge records as a tool for serious work related injury surveillance.* Occup Environ Med, 2006. **63**(4): p. 290-6.

277. Harduar Morano, L., et al., *Occupational heat-related illness emergency department visits and inpatient hospitalizations in the southeast region, 2007-2011.* Am J Ind Med, 2015. **58**(10): p. 1114-25.

278. Godsey, K., *heat advisory/warning questions*, L. Harduar Morano, Editor. 2012.

279. Griffin, M., L. Harduar Morano, and K. Godsey, *Examining Heat Advisories and Health Impacts in Florida*, in *NOAA CDC Heat and Health Summit*. 2014: Silver Spring, Maryland.

280. Winsberg, M.D. and M. Simmons. *An Analysis of the Beginning, End, Length, and Strength of Florida's Hot Season*. 2015 6/3/2015]; Available from: http://climatecenter.fsu.edu/topics/specials/floridas-hot-season.

281. Wellenius, G.A., et al., *Ambient air pollution and the risk of acute ischemic stroke.* Arch Intern Med, 2012. **172**(3): p. 229-34.

282. Fechter-Leggett, E.D., A. Vaidyanathan, and E. Choudhary, *Heat Stress Illness Emergency Department Visits in National Environmental Public Health Tracking States, 2005-2010.* J Community Health, 2015.

283. Gronlund, C.J., *Racial and socioeconomic disparities in heat-related health effects and their mechanisms: a review.* Curr Epidemiol Rep, 2014. **1**(3): p. 165-173.

284. Semenza, J.C., et al., *Heat-related deaths during the July 1995 heat wave in Chicago.* N Engl J Med, 1996. **335**(2): p. 84-90.

285. Florida Department of Health Office of Rural Health. *Rural county list*. 1/28/2015]; Available from: http://www.floridahealth.gov/programs-and-services/community-health/rural-health/_documents/rual-counties-2000-2010.pdf.

286. Morabito, M., et al., *Relationship between work-related accidents and hot weather conditions in Tuscany (central Italy).* Ind Health, 2006. **44**(3): p. 458-64.

287. Centers for Disease Control and Prevention. *Warning signs and symptoms of heat-related illness*. Extreme heat and your health 6/20/2011 5/26/2015]; Available from: http://www.cdc.gov/extremeheat/warning.html.

288. Occupational Safety & Health Administration. *Occupational Heat Exposure: heat illness and first aid*. Back to Safety and Health Topics Page 5/27/2015]; Available from: https://www.osha.gov/SLTC/heatstress/heat_illnesses.html.

289. Arizona Department of Health and Human Services, *Trends in Morbidity and Mortality from Exposure to Excessive Natural Heat in Arizona, 2012 report*, The Bureau of Epidemiology and Disease Control, Editor. 2014: Phoenix, Arizona.

290. Hess, J.J., S. Saha, and G. Luber, *Summertime acute heat illness in U.S. emergency departments from 2006 through 2010: analysis of a nationally representative sample.* Environ Health Perspect, 2014. **122**(11): p. 1209-15.

291. Pillai, S.K., et al., *Heat illness: predictors of hospital admissions among emergency department visits-Georgia, 2002-2008.* J Community Health, 2014. **39**(1): p. 90-8.

292. Winsberg, M.D., *Florida Weather*. second ed. 2003, Gainesville, Florida: University Press of Florida.

293. Florida Department of Agriculture and Consumer Services, *Florida Agricultural Statistical Directory*, Florida Department of Agriculture and Consumer Services, Editor. 2009.

294. Calvin, L. and M. Philip, *The U.S. produce industry and labor: facing the future in a global economy*, US Department of Agriculture Economic Research Service, Editor. 2010.

295. Kovach, M.M., C.E. Konrad, and C.M. Fuhrmann, *Area-level risk factors for heat-related illness in rural and urban locations across North Carolina, USA*. Applied Geography, 2015. **60**: p. 175-183.

296. D'Ippoliti, D., et al., *The impact of heat waves on mortality in 9 European cities: results from the EuroHEAT project.* Environ Health, 2010. **9**: p. 37.

297. Mississippi State Department of Health, *Heat-Related Illness*. 2008: Jackson, Mississippi.

298. Culp, K., et al., *Preventing heat-related illness among Hispanic farmworkers.* AAOHN J, 2011. **59**(1): p. 23-32.

299. Utterback, D.F. and T.M. Schnorr, *Use of Workers' Compensation Data for Occupational Injury & Illness Prevention*, P.H.S. U.S. Department of Health and Human Services, Centers for Disease Control, National Institute for Occupational Safety and Health, Editor. 2010: Cincinnati, OH.

300. Brown, A. and M.H. Lopez *Mapping the Latino Population, by State, County, and City*. Pew Research Center's Hispanic Trends Project, 2013.

301. Pew Research Center's Hispanic Trends Project, *Population Distribution of Hispanic Origin Groups by County, 2010*, all_counties_by_top_six_groups.xlsx, Editor. 2012, PewResearchCenter: Washington, D.C.

302. Xiang, J., et al., *Health impacts of workplace heat exposure: an epidemiological review.* Ind Health, 2014. **52**(2): p. 91-101.

303. Emrich, C.T., et al., *Climate-Sensitive Hazards in Florida: Identifying and Prioritizing Threats to Build Resilience against Climate Effects*. 2014, Hazard and Vulnerability Research Institute.

304. Xiang, J., et al., *Extreme heat and occupational heat illnesses in South Australia, 2001-2010.* Occup Environ Med, 2015. **72**(8): p. 580-6.

305. Johnson, R.F. and J.L. Kobrick, *Psychological Aspects of military performance in hot environments*, in *Medical aspects of harsh environments*, K.B. Pandolf, et al., Editors. 2001, Office of The Surgeon General; Department of the Army, United States of America: Washington, D.C. p. 135-159.

306. Dixon, D., *Florida tourism numbers break records again for the third quarter of this year*, in *The Florida Times Union*. 2015, Jackonville.com: Jacksonville, Florida.

307. U.S. Census Bureau, *OnTheMap Application and LEHD Origin-Destination Employment Statistics*. 2005-2014.

308. Davis, L.K., et al., *Use of statewide electronic emergency department data for occupational injury surveillance: a feasibility study in Massachusetts.* Am J Ind Med, 2012. **55**(4): p. 344-52.

309. Groenewold, M.R. and S.L. Baron, *The proportion of work-related emergency department visits not expected to be paid by workers' compensation: implications for occupational health surveillance, research, policy, and health equity.* Health Serv Res, 2013. **48**(6 Pt 1): p. 1939-59.

310. Centers for Disease Control and Prevention (CDC), *Proportion of workers who were work-injured and payment by workers' compensation systems - 10 states, 2007.* MMWR Morb Mortal Wkly Rep, 2010. **59**(29): p. 897-900.

311. Li, M., et al., *Heat waves and morbidity: current knowledge and further direction-a comprehensive literature review.* Int J Environ Res Public Health, 2015. **12**(5): p. 5256-83.

312. Armstrong, B., *Models for the relationship between ambient temperature and daily mortality.* Epidemiology, 2006. **17**(6): p. 624-31.

313. Environmental Systems Research Institute (Redlands Calif.), *ESRI data & maps 9.3, 2008*. 2008, Environmental Systems Research Insitute,: Redlands, CA. p. 5 DVD-ROMs 4 3/4 in. + 1 guide.

314. Service, N.W., *WFO Non-precipitation weather products specification*. 2011.

315. Burnham, K.P., D.R. Anderson, and K.P. Burnham, *Model selection and multimodel inference : a practical information-theoretic approach*. 2nd ed. 2002, New York: Springer. xxvi, 488 p.

316. Muggeo, V.M., *Modeling temperature effects on mortality: multiple segmented relationships with common break points.* Biostatistics, 2008. **9**(4): p. 613-20.

317. Muggeo, V.M., *segmented: An R Package to Fit Regression Models with Broken-Line Relationships.* R News, 2008. **8**(1): p. 20-25.

318. Gasparrini, A., B. Armstrong, and M.G. Kenward, *Multivariate meta-analysis for non-linear and other multi-parameter associations.* Stat Med, 2012. **31**(29): p. 3821-39.

319. Jackson, D., R. Riley, and I.R. White, *Multivariate meta-analysis: potential and promise.* Stat Med, 2011. **30**(20): p. 2481-98.

320. Gasparrini, A., *Distributed lag linear and non-linear models in R: the package dlnm.* Journal of Statistical Software, 2011. **43**(8): p. 1-20.

321. R Core Team, *R: A language and environment for statistical computing*. 2015, R Foundation for Statistical Computing: Vienna, Austria.

322. Dahl, D.B., *xtable: Export Tables to LaTeX or HTML.* 2015.

323. Dragulescu, A.A., *xlsx: Read, write, format Excel 2007 and Excel 97/2000/XP/2003 files.* 2014.

324. Harrell, F.E. and C. Dupont, *Hmisc: Harrell Miscellaneous*. 2015.

325. Robotham, A., *magicaxis: Pretty Scientific Plotting with Minor-Tick and log Minor-Tick Suppor*. 2015.

326. Wickham, H., *ggplot2: Elegant Graphics for Data Analysis*. 2009: Springer-Verlag New York.

327. National Weather Service. *Heat Index*. Heat Index 9 September 2015]; Available from: http://www.wrh.noaa.gov/psr/general/safety/heat/heatindex.png.

328. Bai, L., et al., *The effects of summer temperature and heat waves on heat-related illness in a coastal city of China, 2011-2013.* Environ Res, 2014. **132**: p. 212-9.

329. Kim, Y.M., et al., *Comparison of temperature indexes for the impact assessment of heat stress on heat-related mortality.* Environ Health Toxicol, 2011. **26**: p. e2011009.

330. Harlan, S.L., et al., *Heat-related deaths in hot cities: estimates of human tolerance to high temperature thresholds.* Int J Environ Res Public Health, 2014. **11**(3): p. 3304-26.

331. Toloo, G.S., et al., *The impact of heatwaves on emergency department visits in Brisbane, Australia: a time series study.* Crit Care, 2014. **18**(2): p. R69.

332. Fortune, M., C. Mustard, and P. Brown, *The use of Bayesian inference to inform the surveillance of temperature-related occupational morbidity in Ontario, Canada, 2004-2010.* Environ Res, 2014. **132**: p. 449-56.

333. Li, B., et al., *The impact of extreme heat on morbidity in Milwaukee, Wisconsin.* Climatic Change, 2011. **110**(3-4): p. 959-976.

334. Lin, Y.-K., et al., *High-temperature indices associated with mortality and outpatient visits: Characterizing the association with elevated temperature.* Science of The Total Environment, 2012. **427-428**: p. 41-49.

335. Bureau of Meteorology. *Climate statistics for Australian locations: Adelaide*. Climate Data Online, Montlhy Statistics 2015 16 September 2015 19 September 2015]; Available from: http://www.bom.gov.au/climate/averages/tables/cw_023090_All.shtml.

336. Kent, S.T., et al., *Heat waves and health outcomes in Alabama (USA): the importance of heat wave definition.* Environ Health Perspect, 2014. **122**(2): p. 151-8.

337. Mayner, L., P. Arbon, and K. Usher, *Emergency department patient presentations during the 2009 heatwaves in Adelaide.* Collegian, 2010. **17**(4): p. 175-182.

338. Vaneckova, P. and H. Bambrick, *Cause-specific hospital admissions on hot days in Sydney, Australia.* PLoS One, 2013. **8**(2): p. e55459.

339. Zierden, D. and M. Griffin. *Humidity in Florida*. 2015; Available from: http://climatecenter.fsu.edu/topics/humidity.

340. Rocklov, J., A.G. Barnett, and A. Woodward, *On the estimation of heat-intensity and heat-duration effects in time series models of temperature-related mortality in Stockholm, Sweden.* Environ Health, 2012. **11**: p. 23.

341. Xiang, J., et al., *Association between high temperature and work-related injuries in Adelaide, South Australia, 2001-2010.* Occup Environ Med, 2014. **71**(4): p. 246-52.

342. Leary, E., et al., *Identifying Heat Waves in Florida: Considerations of Missing Weather Data.* PLoS ONE, 2015. **10**(11): p. e0143471.

343. Sugg, M.M., C.E. Konrad, 2nd, and C.M. Fuhrmann, *Relationships between maximum temperature and heat-related illness across North Carolina, USA.* Int J Biometeorol, 2015.

344. Bishop-Williams, K.E., et al., *A spatial analysis of heat stress related emergency room visits in rural Southern Ontario during heat waves.* BMC Emerg Med, 2015. **15**(1): p. 17.

345. Smargiassi, A., et al., *Prediction of the indoor temperatures of an urban area with an in-time regression mapping approach.* J Expo Sci Environ Epidemiol, 2008. **18**(3): p. 282-8.

346. White-Newsome, J.L., et al., *Climate change and health: indoor heat exposure in vulnerable populations.* Environ Res, 2012. **112**: p. 20-7.

347. Schluter, P.J., P.M. Macey, and R.P. Ford, *The relationship between inside and outside ambient temperatures in Christchurch, New Zealand.* Paediatr Perinat Epidemiol, 2000. **14**(3): p. 275-82.

348. Bernhard, M.C., et al., *Measuring personal heat exposure in an urban and rural environment.* Environmental research, 2015. **137**: p. 410-418.

349. Burkart, K., et al., *The effects of season and meteorology on human mortality in tropical climates: a systematic review.* Trans R Soc Trop Med Hyg, 2014. **108**(7): p. 393-401.

350. Benmarhnia, T., et al., *Review Article: Vulnerability to Heat-related Mortality: A Systematic Review, Meta-analysis, and Meta-regression Analysis.* Epidemiology, 2015. **26**(6): p. 781-93.

351. Chuang, W.C. and P. Gober, *Predicting hospitalization for heat-related illness at the census-tract level: accuracy of a generic heat vulnerability index in Phoenix, Arizona (USA).* Environ Health Perspect, 2015. **123**(6): p. 606-12.

352. Chow, W.T.L., W.-C. Chuang, and P. Gober, *Vulnerability to Extreme Heat in Metropolitan Phoenix: Spatial, Temporal, and Demographic Dimensions.* The Professional Geographer, 2012. **64**(2): p. 286-302.

353. Uejio, C.K., et al., *Intra-urban societal vulnerability to extreme heat: the role of heat exposure and the built environment, socioeconomics, and neighborhood stability.* Health Place, 2011. **17**(2): p. 498-507.

354. Joint Center for Housing Studies, *Evolving markets and needs*, in *America's rental housing*, M. Fernald, Editor. 2013, Harvard University: Cambridge, MA.

355. Loughnan, M., N. Nicholls, and N.J. Tapper, *Mapping Heat Health Risks in Urban Areas.* International Journal of Population Research, 2012. **2012**: p. 1-12.

356. Fry, J., et al., *Completion of the 2006 National Land Cover Database for the Conterminous United States.* Photogrammetric Engineering and Remote Sensing, 2011. **77**(9): p. 858-864.

357. Morrill, R., J. Cromartie, and G. Hart, *Metropolitan, urban, and rural commuting areas: toward a better depiction of the United States settlement system.* Urban Geography, 1999. **20**(8): p. 727-748.

358. Higgins, J.P., et al., *Measuring inconsistency in meta-analyses.* BMJ, 2003. **327**(7414): p. 557-60.

359. Arizona Department of Health and Human Services. *Heat Safety - Heat-Related Maps*. Extreme Weather and Public Helaht 3/12/2016]; Available from: http://www.azdhs.gov/preparedness/epidemiology-disease-control/extreme-weather/index.php#heat-maps.

360. Reid, C.E., et al., *Evaluation of a heat vulnerability index on abnormally hot days: an environmental public health tracking study.* Environ Health Perspect, 2012. **120**(5): p. 715-20.

361. Bao, J., X. Li, and C. Yu, *The Construction and Validation of the Heat Vulnerability Index, a Review.* Int J Environ Res Public Health, 2015. **12**(7): p. 7220-34.

362. Florida Department of Agriculture and Consumer Services, *Florida Agricultural by the Numbers 2012*, Florida Department of Agriculture and Consumer Services, Editor. 2012.

363. U.S. Census Bureau, *2011 American Community Survey - 5-year estimates, Table S1601*, in *American FactFinder*

364. Fuhrmann, C.M., et al., *Impact of Extreme Heat Events on Emergency Department Visits in North Carolina (2007-2011).* J Community Health, 2015.

365. Lusher, W.R., J.L. Jackson, and K.T. Morgan. *The Florida Automated Weather Network: Ten Years of Providing Weather Information to Florida Growers*. in *Proc. Fla. State Hort. Soc*. 2008.

366. Rothman, K.J., S. Greenland, and T.L. Lash, *Modern epidemiology*. 2008: Lippincott Williams & Wilkins.

367. Grubesic, T.H. and T.C. Matisziw, *On the use of ZIP codes and ZIP code tabulation areas (ZCTAs) for the spatial analysis of epidemiological data.* Int J Health Geogr, 2006. **5**: p. 58.

368. U.S. Census Bureau, *2013 American Housing Survey; Heating, Air Conditioning, and Appliances - All Housing Units (SELECTED METROPOLITAN AREAS), Table C-03-AH-M*, in *American FactFinder*

369. RECS 2009. *Air conditioning in nearly 100 million U.S. homes*. Residential Energy Consumption Survey (RECS) 2011 8/19/2011 2/14/2016]; Available from: https://www.eia.gov/consumption/residential/reports/2009/air-conditioning.cfm.