

**Extreme Heat Events and Associated Health Outcomes in King County, WA: A Study of  
Historical Outcomes, Model Validation, and Heat-Risk Mapping**

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**Abstract**

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Increased mortality and morbidity have been associated with extreme-heat events, particularly in temperate climates. Climate change is predicted to increase the intensity and duration of these extreme heat events. Understanding the local heat-health relationship and its spatial distribution is important to predict future health-related burdens from climate change, and to assist local public health officials in emergency heat-preparedness efforts.

This study used a non-parametric Poisson regression model with a piece-wise linear function to estimate heat's effect on mortality and morbidity, beyond a model-derived threshold. Relative risk and time series analyses were conducted to explore extreme heat effects on mortality and morbidity outcomes for three different time frames: calibration (1980-2006 mortality/1990-2006 hospital admissions), validation (2007-2010), and complete (1980/1990-2010). The results from these analyses were used to test the validity of our piece-wise linear

approach to interpreting heat's effect on health outcomes. This study also provided translational materials to local public health practitioners, detailing heat-risk estimates, vulnerable populations, and heat-risk distribution.

The results demonstrate that heat, expressed as humidex, is associated with increased non-traumatic mortality and hospital admissions on heat days, and that the risk increases with heat intensity - especially for the older populations. The all-ages relative risk of mortality on a heat day (above the 99<sup>th</sup> percentile) was 10% greater than on a non-heat day, with risk increasing 2.12% for each degree increase in humidex above 36.0 °C. The all-ages relative risk of hospitalization on a heat day (above the 99<sup>th</sup> percentile) was 2% (non-significant) greater than on a non-heat day, with risk increasing 1.59% for each degree increase in humidex above 37.4 °C. This study found that with the available individual-level characteristics data, only age modified heat's effect on health outcomes. While the 65+ age groups were found to be at greater risk of poor health outcomes on an extreme heat day, younger age groups were also found to be at risk for specific causes of death and hospitalization.

We found that, overall, our method of using a piece-wise linear term to estimate heat's effect on mortality and hospital admission rates held up well, even when using a validation time frame that contained the most extreme heat event on record for King County, Washington. Our model's prediction estimates are conservative, and under predict mortality at the lower bound of the expected confidence interval. Future estimates using this method will likely predict at least as many deaths and hospitalizations as observed.

Our findings warrant additional research into the role heat exposure plays in several specific causes of death and hospitalization, such as diabetes and renal syndromes in the 45-64 age group and circulatory and cardiovascular conditions in the 85+ age group. Future areas of

study include improving exposure assessment and vulnerable population data, as well as assessing the next tier of heat-health effects: emergency response system/emergency room data. Finally, what we learn from additional research should improve of our understating of spatial heat-risk distribution and provide our public health partners with additional health related data to share with their constituents.

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## **Dedication**

This dissertation is dedicated to my children: Alexandra, Sophia, and Aidan.

May you go out and use your precious gifts to make a positive difference in the world.

## Specific Aim of Dissertation

Focused on King County, Washington, the main objectives for this dissertation were to evaluate a previously developed, county-specific heat/health projection model, update historical mortality and morbidity risk estimates associated with extreme heat, create a heat-risk mapping tool, and translate the research findings to the public health practice community. This research includes several epidemiological analyses of extreme heat effects on mortality and morbidity in King County, Washington. Results from these analyses were used to validate a previously developed heat-health model and to produce localized heat-risk maps. The findings of this study can be used to inform extreme heat preparedness efforts in King County, Washington, as well as support broad public health discussions on climate change.

### **The specific Aims were as follows:**

1. Use relative risk and time series analyses to quantify mortality rates associated with extreme heat in King County, WA for three time frames:
  - a. 1980-2006 (calibration time frame) used in Chapter 4 *Validating health outcome models associated with extreme heat events in King County, WA*
  - b. 2007-2010 (validation time frame) used in Chapter 4 *Validating health outcome models associated with extreme heat events in King County, WA*
  - c. 1980-2010 (complete time frame) used in Chapter 2 *Mortality associated with extreme heat events in King County, WA, 1980-2010*
2. Use relative risk and time series analyses to quantify hospital admission rates associated with extreme heat in King County, WA for three time frames:

- a. 1990-2006 (calibration time frame) used in Chapter 4 *Validating health outcome models associated with extreme heat events in King County, WA*
  - b. 2007-2010 (validation time frame) used in Chapter 4 *Validating health outcome models associated with extreme heat events in King County, WA*
  - c. 1990-2010 (complete time frame) used in Chapter 3 *Hospital admissions associated with extreme heat events in King County, WA, 1990-2010*
3. Test the validity of an extreme heat/health outcomes projection model by comparing model predictions to observed health outcomes using a time period not previously included in the model's development
  4. Produce heat-risk maps for vulnerable populations within King County, WA
  5. Translate the dissertation's findings into appropriate materials for use by local public health agencies

## Organization of Dissertation

The first chapter of this dissertation introduces the research area and provides background information for: historical health outcomes associated with extreme heat events; predicting health outcomes associated with extreme heat events; heat vulnerability; communicating health risks; extreme heat risk mapping; and historic meteorological data for King County, Washington. The next four chapters (Chapters 2-5) of this dissertation are composed as scientific manuscripts, intended for publication.

Chapter 2 is to be published as: Busch Isaksen TM, Hom EK, Ren Y, Fenske RA, Lyons H, Yost MG. *Historical mortality associated with extreme heat exposure in King County, Washington State*. Target Journal: International Journal of Biometeorology. This publication will describe the findings from the Epidemiological analysis of mortality rates associated with extreme heat events during 1980-2010 in King County, WA.

Chapter 3 is to be published as: Busch Isaksen TM, Hom EK, Ren Y, Yost MG, Lyons H, Fenske RA. *Historical morbidity associated with extreme heat exposure in King County, Washington State*. Target Journal: Environmental Health Perspectives. This publication will describe the findings from the Epidemiological analysis of hospitalization rates associated with extreme heat events during 1990-2010 in King County, WA.

Chapter 4 is to be published as: Busch Isaksen TM, Fenske RA, Lyons H, Yost MG. *Extreme Heat-Health Outcome model validation*. Target Journal: Environmental Health Perspectives. This publication will describe the findings from testing our heat-health relationship model with data not previously used in its development.

Chapter 5 is to be published as: Busch Isaksen TM, Stumbaugh M, Fenske RA, Yost MG. *Mapping heat-exposure risk for vulnerable populations in King County, WA*. Target Journal:

Journal of Environmental Health. This publication will describe the methods used to map the spatial distribution of heat-exposure risk for vulnerable populations.

Chapter 6 details the process and products developed to communicate the findings from specific Aims 1-4 to the public health practice community. Finally, Chapter 7 is a discussion and conclusions section tying the major findings back to the dissertation's overall objectives.

# Chapter 1

## Introduction and Background

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The evidence supporting climate change is unequivocal, and it is very likely that human activity has contributed significantly to the changes we are observing today and can expect to experience in the future (IPCC 2013). A warming world presents a real threat to the health and well-being of human populations in the United States and world-wide (IPCC 2007). Existing health risk data related to climate change are largely available as an enumeration of potential direct and indirect health consequences as a result of severe weather events and heat waves, worsening air pollution, changes in vector biology and associated infectious diseases, food and water contamination and shortages (Confalonieri et al. 2007), large-scale migration, and civil conflict (Frumkin et al. 2008; Balbus et al. 2008).

Health outcomes associated with extreme heat events has been one aspect of climate change research that receives considerable attention. Extreme heat events result in more fatalities than any other weather-related phenomenon (Centers for Disease Control and Prevention 2012). Mortality and morbidity associated with heat exposure is generally acute in presentation, occurring within the heat event's time frame, and therefore can be quantified well with current epidemiological and statistical methodologies (Barnett, Tong and Clements 2010). However, region-specific data addressing known and expected health outcomes, as well as identifying vulnerable populations at risk, are needed to inform public health preparedness efforts. Ultimately, the impact climate change will have on a population's health will vary depending on the region, the population's vulnerabilities and resiliency, and the public health system's ability to respond to the challenge (Patz et al. 2005; Gamble and Ebi 2008). An important step to attenuate impacts on health will be to place reliable information in the hands of on-the-ground policymakers.

## 1.1 Health outcomes associated with historic extreme heat events

In late 2011, the World Meteorological Organization released the *Provisional Statement on the Status of the Global Climate*, in which they cited 2011 to be the world's 10<sup>th</sup> warmest year on record, despite a global climate that was heavily influenced by one of the strongest La Niña events in the past 60 years (World Meteorological Organization 2011). In September 2013, the Intergovernmental Panel on Climate Change (IPCC) released its Summary for Policymakers Physical Science Basis: Working Group I Contribution to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. In this report, the IPCC states that “It is virtually certain that there will be more frequent hot and fewer cold temperature extremes over most land areas on daily and seasonal timescales as global mean temperatures increase. It is very likely that heat waves will occur with a higher frequency and duration.” (IPCC 2013).

In order to estimate future heat-related health consequences from a changing climate, we must first fully understand the association between heat exposure and health outcomes. Since the early 1980s, extreme heat events have contributed to thousands of deaths and hospitalizations in the U.S., Canada and European countries (Whitman et al. 1997; Naughton 2002; Ebi et al. 2004; Mastrangelo et al. 2007; Baccini et al. 2008; Basu, Feng and Ostro 2008; Anderson and Bell 2009; Knowlton et al. 2009; Jackson et al. 2010; Ye et al. 2012). The excess deaths and hospitalizations occur in concentrated time frames, rather than being spread across the summer months. As a result, these events can place a significant burden on the emergency response system (Golden et al. 2008; Garcia-Herrera et al. 2010). Davis et al. (2003) found that, of the 28 cities their study analyzed, the number of excess deaths occurring on heat days has declined over the years from  $41.0 \pm 4.8$  deaths/city/year (in the 1960s–1970s) to  $10.5 \pm 2.0$  (in the 1990s). The

authors suggest that acclimatization, the use of early warning systems, and preparedness efforts have all played a role in the reductions over time (Davis et al. 2003).

Studies that have analyzed historical extreme heat events, and their effects on health, have found that mortality risks increase as temperatures climb. Medina-Ramon and Schwartz's (2007) meta-analysis of 42 U.S. cities found a 3.85 % increase in mortality on extreme heat days (above the 99<sup>th</sup> percentile) compared to all other days. Similarly, in a study of 43 U.S. cities, Anderson and Bell (2011) found that mortality increased 3.74% (95% CI: 2.29-5.22%) on a heat wave day compared to a non-heat wave day, and that there was a 2.49% increase in mortality for each 1 °F increase in heat wave intensity. In a study of 15 European cities, Baccini et al. (2008) found that a 1 °C increase in maximum apparent temperature above a city-specific threshold was associated with a 1.84% increase (95% CI: 0.06-3.64%) in mortality in north-continental cities and a 3.12% increase (95% CI: 0.60-5.72%) in the Mediterranean region. Baccini et al.'s (2008) results suggest significant geographical variability observed in both heat thresholds and slopes. In the U.S., moderate climate zones may also be especially vulnerable to the effects of extreme heat on health. In 2002, Curriero et al. explored the relationship between temperature and mortality differences between northern and southern US cities. Their research demonstrated a stronger risk of mortality at lower temperatures in the north compared to southern cities (Curriero et al. 2002).

A similar heat-outcome relationship has been found when analyzing the impact extreme heat events have on morbidity. In a recent paper, Ye et al. (2012) reviewed 10 U.S. studies that assessed the association between extreme heat and morbidity risk. While the measure of both heat exposure and morbidity outcomes varied between studies, overall, they found a significant relationship between increasing temperatures and health outcomes. Knowlton et al. (2008)

analyzed California's July 2006 heat wave and found evidence that heat was responsible for 16,166 excess emergency department visits and 1,182 excess hospitalizations (RR 6.30, 95% CI: 5.67-7.01; RR 10.15, 95% CI: 7.79-13.43, respectively). Individuals greater than 65 years of age and children 0-4 years of age were found to be at greatest risk for an adverse health outcome (Knowlton et al. 2008). In 2009, the California Climate Change Center conducted a historic analysis of summer months (1999-2005), in nine California counties, and found that for each 10 °F increase in apparent temperatures there was a corresponding increase in hospitalization risk: 2.0% for respiratory, 3.7% for pneumonia, 3.1% for diabetes, 10.8% for dehydration, 404.0% for heat stroke, and -10.4% for hemorrhagic stroke causes of admission. While heat effect on admission rates differed by age, they found no effect modification with sex, ethnicity or air pollution levels (Green et al. 2010). Lastly, in the only published hospital-related study to use humidex as a measure of heat exposure, Mastrangelo et al. (2007) analyzed the Veneto, Italy Region over five consecutive heat waves. They found that a heat wave's duration, rather than its intensity, increased the risk of hospital admissions for heart and respiratory disease by 16% and 5%, respectively. A lag of 4 days was required to observe statistically significant results, and the first and last heat wave peaked equally. The results suggest that individuals were not able to avoid hospitalizations with acclimatization or behavioral changes over the course of the summer.

Factors previously identified as modifying heat's influence on mortality and morbidity rates include: race (Fletcher et al. 2012; Medina-Ramon et al. 2006; O'Neill, Zanobetti and Schwartz 2003; O'Neill, Zanobetti and Schwartz 2005), educational status (O'Neill, Zanobetti and Schwartz 2003; Jones 1982), socio-economic status (Lin et al. 2012; Portier 2010; Anderson and Bell 2009; Hajat et al. 2005; Jones et al. 1982; Kaiser et al. 2001), social isolation (Naughton 2002; Kaiser et al. 2001) and access to air conditioning (O'Neill, Zanobetti and Schwartz 2005;

Curriero et al. 2002; Naughton 2002). There is significant variability in the results among these studies, suggesting that *place* matters. Further supporting the notion that place matters is a recent review of literature (Yardley, Sigal, and Kenny 2011) which focused on the importance of social and community factors in heat-health planning. The authors found that the spatial distribution in heat-related mortality indicates more at play than just temperature and physiology. They further report that when comparing different locations with heat-related impacts on ethnicity, results were highly variable. Given that there is no physiological explanation for these differences in mortality rates, the authors conclude that the differences seen within various studies are more likely attributable to confounding by local characteristics - both physical environment and social structure (Yardley, Sigal, and Kenny 2010).

In Washington State, the State Legislature has been a leader in considering impacts from climate change. In 2008, a comprehensive analysis of climate change impacts on the Pacific Northwest was completed by the Washington Climate Impacts Group (University of Washington 2009). This report gave equal consideration to human health and other impacts, such as water, energy and agriculture. The human health chapter, published under Jackson et al. (2010), found that extreme-heat events were a significant environmental health hazard for communities in Washington State. Specifically, the authors found that in the Greater Seattle area (King, Pierce, Snohomish Counties) heat-related risk for all non-traumatic causes of death significantly increased in populations ages 45 years and above (with the highest for the 85+ age group, 1.5% (95% CI 1.2-1.8)). The highest cause-specific, increased risk of death, 1.5% (95% CI 1-1.2) was for circulatory causes in the 85+ population, although it was also statistically significant for all age groups 45+. Spokane, Benton, Franklin, and Yakima counties showed smaller, non-statistically significant increases in mortality (Jackson et al. 2010). More recently, Busch

Isaksen et al. (2014) found that in King County, a one-degree increase in the average county-wide daily maximum humidex above 35.7 °C was associated with 1.83% (95% CI: 0.77%, 2.91%) increase in non-traumatic mortality. Similar results were obtained for Clark County 1.08% (95% CI: 0.12%, 2.06%) and Spokane County 0.78% (95% CI: 0.01%, 1.55%) (Busch Isaksen et al. 2014).

Direct comparisons with findings in the literature are difficult because methods differ considerably across studies. Only a couple studies explore heat effects on mortality above a relative threshold (Jackson et al. 2010; Medina-Ramon et al. 2006). Instead, it is more common to find studies that explore relative risk effects from heat waves or heat wave days (Wu et al. 2014; Gasparrini and Armstrong 2011; Anderson and Bell 2009; Kaiser et al. 2007; Hajat et al. 2006; Le Tertre et al. 2006; and Naughton 2002). When estimating the intensity of heat effect on mortality and morbidity, many studies use a Poisson generalized additive model with nonparametric curves, or splines, to describe the relationship and to control for long-term changes in mortality or hospitalizations over time (Gasparrini and Armstrong 2011; Anderson and Bell 2009; Green et al. 2010; Basu, Feng and Ostro 2008; Medina-Ramon and Schwartz 2007; Mastrangelo et al. 2007; Curriero et al. 2002). However, the methods used to summarize and report the effects of heat on health vary considerably and include comparing changes in risks of relative percentiles (Gasparrini and Armstrong 2011; Anderson and Bell 2009; Medina-Ramon and Schwartz 2007), estimating changes in risk per 10 °F increase in mean daily apparent temperature (Green et al. 2010; Basu, Feng and Ostro 2008; Curriero et al. 2002) or comparing heat wave day/event risk against non-heat wave day/event risk (Knowlton et al. 2008; Mastrangelo et al. 2007). Finally, the effect of heat intensity on mortality and morbidity can be described by using a piece-wise linear function, which estimates changes in risk for each

additional °C unit of apparent temperature or humidex above a defined threshold (Chapters 2 & 3; Busch Isaksen et al. 2014; Lin et al. 2012; Lin et al. 2009; Baccini et al. 2008; Medina-Ramon and Schwartz 2007; Armstrong 2006).

## **1.2 Predicting health outcomes associated with extreme heat events**

Once a region's heat-health risk relationship has been defined, future climate scenarios can be used to project health outcomes. Huang et al. (2011) reviewed 14 studies that explored the future effect of extreme heat events on mortality and found the majority projected substantial increases in heat-related mortality. The methods used by the reviewed studies varied substantially. However, all studies achieved an understanding of the historical heat-mortality relationship and considered future changes in climate (e.g. climate scenarios). Some studies failed to take into consideration changes in demographics, nor did they all consider how acclimatization may affect results. The authors were unable to compare study findings through a meta-analysis, given the lack of standardized methods and results (Huang et al. 2011). Five of the 14 studies were conducted wholly in the United States. The largest US study, Kalkstein and Greene (1997), looked at 44 US urban areas with population over 1 million. The authors found historical increases in the frequency of air masses were associated with increased mortality. Their projected increases in mortality ranged from 70%, for the conservative global circulation model (GCM), to 100% for less conservative GCMs (Kalkstein and Greene 1997). Hayhoe et al. (2004) focused on four areas in California and projected the greatest increase (two to seven times) for the Los Angeles area's mid to end-of-century heat-related mortality compared to baseline. In 2007, Knowlton et al. projected increases in heat-related mortality for 31 county/metropolitan regions around New York City. Comparing 2050 projected to 1990 observed mortality, the authors estimate a 47% to 95% increase in mortality. They further took

into consideration the attenuating effects from acclimatization and project a ~25% reduction in expected mortality (Knowlton et al. 2007). Finally, a recent study published by Wu et al. (2014) examined heat events for the Eastern United States and projected 7.5-19.0 times higher mortality in 2057-2059 compared to their baseline 2002-2004 time frame.

In Washington State, Jackson et al. (2010) projected mortality rates for 2025, 2045, and 2085 based on age and cause-specific mortality rates associated with historic extreme heat. In 2025, the Greater Seattle area can expect an additional 68-211 (depending on climate scenario) excess non-traumatic deaths annually, while Eastern Washington counties can expect an excess 12-31 non-traumatic deaths annually (University of Washington 2009, Jackson et al. 2010). More recently, Busch Isaksen et al. (2014) estimates 2.3 to 8.0 times higher mortality in 2025 and 4.0 to 22.3 times higher mortality in 2045 for residents 85+ years of age living in King County, Washington, compared to 2002-2006 baseline rates.

### **1.3 Heat vulnerability**

There are finite limits to the amount of heat exposure a person can physiologically tolerate. In healthy adults, the body maintains thermoregulation by increasing radiant, convective and evaporative heat loss (Kovats and Hajat 2008; Kenny and Journeay 2010). Simply stated, when muscles work, they produce energy in the form of heat. The heat produced by muscles is transferred away from the work site via blood flow, resulting in an increase in core body temperature (heat storage). The hypothalamus and peripheral circulatory system work to keep core body temperatures at 37 °C, +/- 1 °C, by triggering cooling (or heating) mechanisms. Cooling mechanisms include: augmenting the skin's blood flow for convective heat loss, through cutaneous vasodilatation and increased heart rate; and controlling the sweating rate for evaporative heat loss, through sympathetic cholinergic stimulation (McKinnon and Utley 2005;

Rowland 2008; Kenny and Journey 2010). Factors that affect vasodilatation, cardiovascular performance, and sweating rates will, therefore, affect the body's ability to dissipate stored heat and will increase the risk for heat injury. Heat injury occurs when the body's cooling mechanisms are unable to dissipate enough of the energy through convective or evaporative cooling. Heat injury may manifest as minor as rash, swelling, or cramping, or as severe as heat exhaustion and stroke (Marshall 2010). Cell failure and breakdown begin to occur when core body temperatures exceed 40 °C (104 °F). The extent of cellular damage and potential reversibility depends upon how much the core body temperature exceeds 40 °C, as well as the duration spent above 40 °C (Kovats and Hajat 2008).

While there are finite limits to the amount of heat exposure a person can tolerate, this tolerance range is decreased for physiologically vulnerable populations such as the old, infirm, and young. The elderly experience age-related deterioration of natural homeostatic mechanisms, making it more difficult to maintain optimal thermoregulation. They also experience an increased prevalence of medical conditions requiring medications that negatively affect thermoregulation (Naughton 2002). On the opposite end of the age spectrum, children also have difficulty with optimal thermoregulation. Physiologically, there are important differences between children and adults that affect thermoregulation. Children and adolescents have greater heat exposure given their larger body surface area (BSA) to body mass ratio, they produce more metabolic heat per mass, and they have a lower sweating capacity than adults (American Academy of Pediatrics 2000). It is also thought that a greater increase in core body temperature must occur in younger populations before the onset of sweating (Naughton and Carlson 2008). These differences result in a reduced ability to dissipate body heat during periods of extreme heat and exertion. Literature also suggests that children take longer to acclimate to heat, are less

effective at thermoregulation when dehydrated, and are less likely to hydrate without proper supervision (Bar-Or 1995; Naughton and Carlson 2008).

In addition to age, gender, physical fitness and overall health status can modify thermoregulation. Differences in convective and evaporative cooling mechanisms have been found between males and females. These differences have been attributed to higher body surface area (BSA)-to-body mass (BM) ratio, reproductive cycle influences on resting core temperatures, and an increase in the threshold for the initiation of sweating found in females (Buresh, Berg and Noble 2005). A person's physical fitness can influence his/her body's response to heat dissipation. A more physically fit person can work harder with less cardiac output. The less the cardiovascular system must work to supply oxygen to the body, the more available it is to support convective cooling mechanisms (McKinnon and Utley 2005). There is a distinct difference between heart rate increasing due to a need for more oxygen and heart rate increasing in order to move more blood to the skin. Therefore, factors such as obesity and acclimatization to activity, as well as environmental conditions, all change the time it takes the body to reach limiting core temperatures (Buresh, Berg, and Noble 2005). Interestingly, because convective and evaporative cooling occur at the surface of the skin, individuals with a higher BSA relative to their body mass may dissipate heat more efficiently than individuals with less BSA/BM ratio. However, the BSA-to-body mass association is also affected by cardiovascular fitness, sex, type and duration of exercise/work, as well as external factors such as ambient temperature and humidity (Rowland 2008). The infirm have many of the same vulnerabilities as the old; they may present with illnesses that affect thermoregulation or utilize medications that affect their ability to adapt to extreme heat events (Kovats and Hajat 2008; Naughton 2002; Brown and Walker 2008). Examples of medications that can influence heat loss and blood supply include:

sleeping pills, which limit the sweating rate; beta-blockers, which affect heart rate regulation; and vasoactive drugs, which control blood vessel size (McKinnon and Utley 2005).

Additionally, prior history of heat injury, along with medical conditions such as the sickle cell trait and recent febrile illness place children at higher risk (Marshall 2010). Lastly, hydration status is important to the evaporative cooling mechanism, as it is a limiting factor in the production of sweat. A dehydrated individual will produce less sweat, thereby reducing evaporative cooling (McKinnon and Utley 2005; Kenny and Journeay 2010).

External factors that influence thermoregulation include ambient air temperature and humidity, clothing, and social and physical environment. As long as the ambient air temperature remains below core body temperature, radiative (through convective) cooling continues and accounts for approximately 65% of the body's cooling mechanisms (McKinnon and Utley 2005). When ambient air temperatures approach core body temperature, a reversal of the skin-to-air temperature gradient occurs. Individuals with higher BSA/BM ratio may no longer have a cooling advantage; instead their larger ratio may work against cooling by drawing additional ambient heat over the air-to-skin barrier (Rowland 2008). Also found to influence convective air currents, and subsequent radiative heat loss, is whether the individual is located indoors or outdoors (Buresh, Berg, and Noble 2005). Evaporative cooling, on the other hand, is reliant on water phase transfer across the surface of the skin-to-air boundary. Once humidity reaches 100%, evaporative cooling ceases, thereby eliminating approximately 30% of the body's cooling mechanism (McKinnon and Utley 2005). It is important to note that clothing can change the "microenvironment" next to the skin, increasing relative humidity. Clothing may reduce the maximum rate of evaporative cooling by impeding the skin-to-air phase transfer of water vapor, as well as reduce convective cooling by limiting radiative transfer via air movement (McKinnon

and Utley 2005). Clothing factors that impede cooling include: too much clothing, movement restrictive clothing, dark-colored clothing, and clothing fabric (Marshall 2010).

Lastly, the environment a person inhabits may play a significant role in reducing or increasing heat exposure, especially for the elderly. Studies have found that mortality risks for the elderly increases when they are housebound, living alone, or institutionalized (Kovats and Hajat 2008; Brown and Walker 2008). Brown and Walker (2008) found that, for the elderly, their ability to adapt was “inhibited by their spending prolonged periods in bed, being unable to care for themselves, and by not leaving home every day.” The literature also reports that the strongest protective factor against heat-related death for the elderly is a working air conditioner (Naughton 2002; Browning et al. 2006; Brown and Walker 2008). Naughton (2002) analyzed heat-related mortality during the 1999 heat wave in Chicago and found that the odds ratio for a working air conditioner was 0.02 (95% CI, 0.1-.07). While studies suggested that access to air conditioning prevents heat injury, others are beginning to think more broadly about the physical environment’s impact on health outcomes during extreme heat events. For example, in a study of 53 metropolitan regions, Stone, Hess, and Frumkin (2010) found that urban areas with sprawling development experienced more than double the rate of extreme heat events compared to their more compact counterparts. Considering that more than 50% of the world’s population lives in a city/metropolitan area, urban planning and design may play a significant role in mitigating the health effects from a warming planet (Stone, Hess, and Frumkin 2010; Cooney 2012; Huang et al. 2011).

#### **1.4 Communicating health risks**

The impact on health from climate change is a very complex, systems-based problem. Even with near consensus among scientists regarding the reality of climate change and human

contributions to it (IPCC 2013), and even with related media coverage, carbon emissions continue to rise in the absence of effective mitigation policies. Nerlich, Koteyko and Brown (2010) suggest that the lack of forward movement with regards to comprehensive mitigation policies may result from the ineffectiveness of current communication efforts. They argue that current methods have failed because they revolve largely around an outdated “deficit” model, where the communicator assumes that the audience is an empty vessel in need of information, and that once adequately informed they will be compelled to act. Instead, Nerlich, Koteyko and Brown (2010) recommend methods that include dialogue and audience engagement. Maibach et al. (2010) suggests reframing climate change communication from an environmental problem to a public health crisis.

Instead of conjuring mental images of polar bears on ever-shrinking ice floes, it would be more effective to illustrate vulnerable populations affected by extreme weather. Refocusing communication around the effects to public health reduces the distance between action and consequence; it creates an environment where serious and sustained public engagement can take place. Research conducted by Maibach et al. (2010) highlights the impact a public health frame can have even with individuals who are climate change doubters. Through a public health-framed/climate change essay, the authors demonstrate that statements focusing on the health impacts did have a mild influence (trending up towards positive perception) on the doubtful and dismissive individuals. When discussing the health benefits associated with mitigation activities, both the doubtful and dismissive groups were positively influenced by this type of communication. The authors go on to argue that public health is tied to positive solutions/co-benefits, and is therefore, well positioned to reframe the issue into one to which people can relate. However, reframing the issue and providing better, more relative information will not be

enough. Nerlich, Koteyko and Brown (2010) point out that individual action needs to be supported by changes to infrastructure and institutional norms.

## **1.5 Extreme heat risk mapping**

Geographic Information Systems (GIS) mapping of health risks can be a highly effective risk communication tool for planners and policymakers (Graham et al. 2011). However, only a few researchers have attempted to map heat-health risks. While Reid et al. (2009; 2012), Houghton et al. (2012), and Buscail, Upegui, and Viel (2012) have all spatially describe heat-related vulnerable populations, none have incorporated regionally-derived, heat-health outcome relationship data into their models. In Reid et al. (2009), the authors describe their development of a heat vulnerability index (HVI) for roughly 40,000 census tracts throughout the United States. They used 10 indicators to identify specific populations as being particularly vulnerable to extreme heat events; approximately 75% of the variance was explained by social/environmental vulnerability, social isolation, air conditioning prevalence, and proportion of elderly diabetics. In Reid et al. (2012), the hypothesis suggesting HVI would be predictive of poor health outcomes during heat events was tested. This study used an exposure and health outcomes spatial resolution (at a zip code level), combined with vulnerability factors (at a census tract and zip code level). The authors defined an abnormally hot day as a day deviant from the 30-year normal maximum temperature for a particular zip code. Health outcome counts for non-deviant days were compared to deviant days. The results showed an increase in poor health outcomes on both types of days, suggesting that the HVI may instead be predictive of health vulnerability in general (Reid et al. 2012).

In Houghton et al. (2012), the authors designed an interactive Web-based tool to help public officials in Austin, Texas visualize their heat-health risks. Their tool incorporates low

resolution exposure data (National Weather Service Alerts), pre-existing chronic disease rates (a proxy for health vulnerability), and six vulnerability variables (age, ethnicity, impervious surfaces, population density, social isolation, and surface temperature) to generate heat-risk maps. Similarly, Buscail, Upegui, and Viel (2012) spatially assess heat-related health risks by incorporating land surface temperature information, population density, and vulnerability variables (socio-economic status, age, population density, and building age). The resulting map product was a visual heat-wave, health-risk index for a medium-sized French city. Lastly, O’neill et al. (2013) provided simple examples of how to use census data to generate vulnerability maps for specific regions. Without specifically mapping heat distribution, they suggest that planners could overlay multiple indicators of risk to generate a visual representation of those census tracts at greater heat-health risk.

A common limitation for each of the aforementioned heat-risk mapping studies is that vulnerable populations are defined *a priori* based on literature and not based on region-specific epidemiological analyses. In all cases, vulnerable population data were combined to create a cumulative vulnerability score at the census tract-level, and further combined in varying ways with heat distribution to obtain a spatial description of heat risk. Defining heat risk in this way requires the following assumptions: the particular vulnerability factor statistically significantly modifies heat effect on health outcomes in the region of study and with similar magnitude.

## **1.6 Study location**

This dissertation details research conducted in King County, Washington. King County is the most populous county in Washington State. It is located in the western half of the state, is bordered on its west side by Puget Sound and covers 2,134 square miles (Figure 1.1). King County is the 14<sup>th</sup> most populous county in the U.S. and is nearly twice as large as the average

size for counties nation-wide (King County 2012). Its population has increased significantly over the past 31 years, from 1.26 million residents in 1980, to over 1.9 million in 2010, a 52% increase (Washington State Office of Financial Management 2012). Table 1.1 details the breakdown in King County's population, as well as the percent change, by the age groups used throughout this dissertation's epidemiological analyses.

**Figure 1.1** Washington State county boundaries



**Table 1.1** King County, Washington Population and Climate Demographics 1980–2010

	<i>King County</i>
<b>1980 Population</b>	Complete Count
State Total	4,132,156
King County Total	1,269,749
% of State Population	30.7%
0-4	78,525
5-14	170,657
15-44	643,707
45-64	247,446
65-84	115,910
85+	13,504
<b>2010 Population</b>	Complete Count (% Change)
WA State Total	6,724,540 (62.7%)
King County Total	1,931,249 (52.1%)
% of State Population	28.7% (-7.0%)
0-4	120,294 (53.2%)
5-14	224,084 (31.3%)
15-44	856,843 (33.1%)
45-64	519,349 (110%)
65-84	176,895 (52.6%)
85+	33,784 (150%)
<b>County-Wide Humidex °C (°F) (May-Sept.)</b>	
Min. Daily Humidex, Av. over time	6.66 °C (44.0 °F)
Max. Daily Humidex, Av. over time	22.4 °C (72.3 °F)

## 1.7 Meteorological data

This study used a historical (1915-2012) meteorological gridded (4 km x 7.5 km) data set produced by the University of Washington's Climate Impacts Group (Maurer et al. 2002). The climatologic foundation of this data set is the Parameter-elevation Relationships on Independent Slopes Model (PRISM), which was developed by Oregon State University (PRISM Climate Group). PRISM is considered to represent the most current knowledge on spatial climatic patterns for the United States (Daly et al. 2007). Daily temperature and relative humidity values for our meteorological data set were constructed using PRISM's regional spatial climatic patterns and weather station observations from the Global Historical Climate Network-Daily (GHCN). GHCN is a National Oceanic and Atmospheric Administration-maintained database of daily meteorological measurements from land surface stations across the globe (NOAA 2009). The resulting meteorology data used for this study contain daily maximum/minimum (max/min) temperature, precipitation and relative humidity values for each gridded (4 km x 7.5 km) meteorological center point. The county-wide daily maximum temperature and average relative humidity values were used to construct our exposure metric, *humidex*.

Exposure to heat was estimated as the average maximum county-wide humidex value for each day. Although unit less, humidex is expressed in units of °C (Masterton and Richardson 1979). Daily maximum humidex was first calculated for each meteorological center point and then averaged over all points within King County to obtain a daily average county-wide maximum value. Humidex combines the effects of temperature and humidity on the human body, and is calculated using the following equations:

$$f(T, H) = T + (5/9) \cdot (v - 10), \quad v = (6.112 \cdot 10^{\left[\frac{7.5T}{237.7+T}\right]}) \cdot H/100,$$

where  $T$  is the air temperature ( $^{\circ}\text{C}$ ),  $H$  is the humidity (%) and  $v$  is the vapor pressure (kPa) (Canadian Centre for Occupational Health and Safety 2011).

In King County, July and August tend to be the hottest months, while November and December tend to be the wettest, most humid months (Figure 1.2). Its summertime (May-September, 1980-2010) county-wide average daily humidex ranges from a minimum  $6.66^{\circ}\text{C}$  ( $44.0^{\circ}\text{F}$ ) to maximum  $22.4^{\circ}\text{C}$  ( $72.3^{\circ}\text{F}$ ) (Table 1.1). Finally, King County's overall climate is not homogeneous; it has three different climate zones transecting the length of the county: Puget Sound Lowlands, East Olympic Cascade Foothills, and the Cascade Mountains West (Figure 1.3) (National Climatic Data Center).

Figure 1.2 King County meteorological averages, 1980-2010

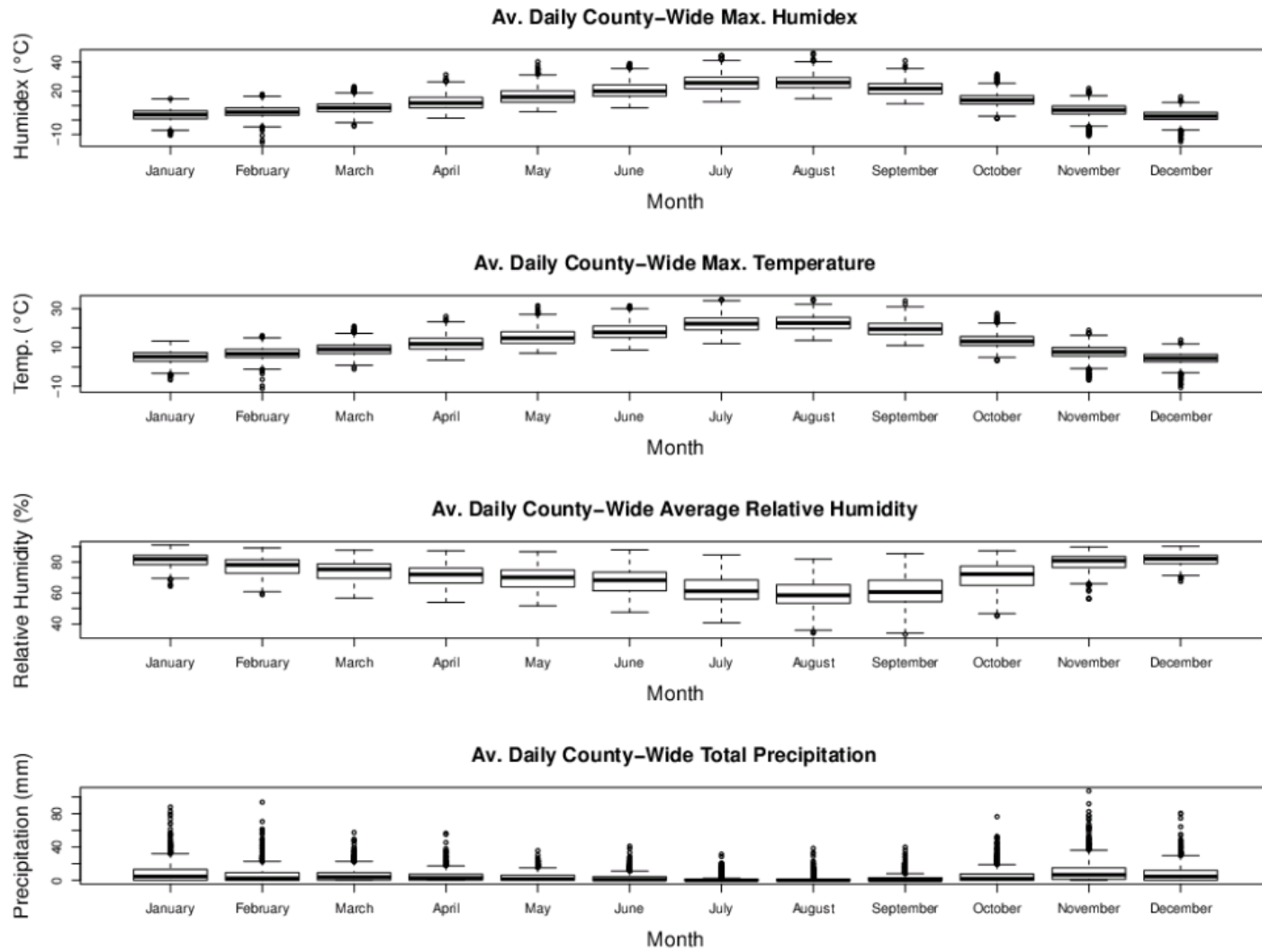
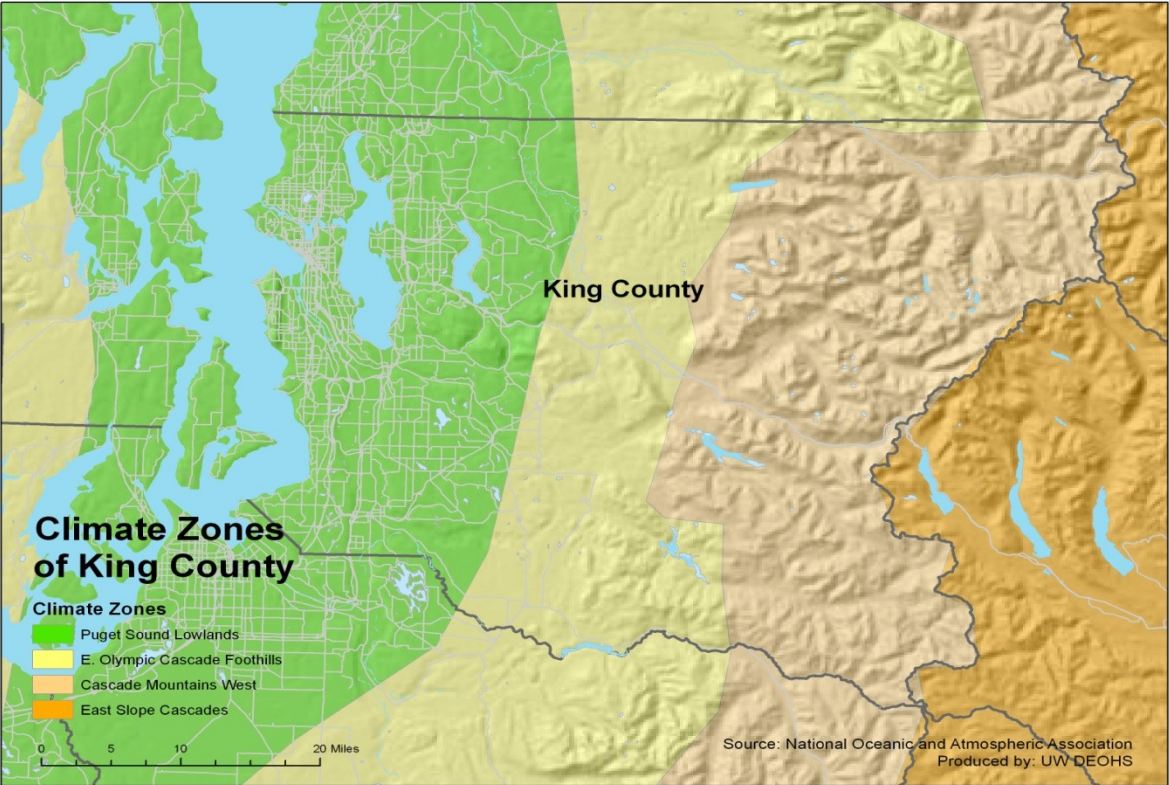


Figure 1.3 King County, Washington climate zones



## Notes to Chapter 1

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## Chapter 2

### Mortality associated with extreme heat exposure in King County, WA, 1980-2010

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## Chapter 2 abstract

### Background

Extreme heat has been associated with increased mortality, particularly in temperate climates. The frequency and intensity of extreme heat events are projected to increase in the future due to climate change. An important first step to mitigating the changing climate's heat impact on health is to understand the historical heat-mortality relationship on a regional scale. Few epidemiologic studies, however, have considered the Pacific Northwest region in their analyses.

### Methods

This study quantifies the historical (May to September, 1980-2010) heat-mortality relationship in King County, Washington using both relative risk and time series analyses. Data were obtained from the Washington State Department of Health (mortality) and the University of Washington Climate Impacts group (temperature and relative humidity). A relative risk (RR) analysis was used to explore the association between heat and all-cause mortality on 99<sup>th</sup> percentile heat days compared to non-heat days. Additionally, a time series analysis using a piece-wise linear model fit was conducted to estimate the effect heat's intensity has on mortality, adjusted for temporal trends and day of the week. Risk of mortality was estimated by age group and specific non-traumatic and traumatic underlying causes of death for both analyses.

### Results

We found for all-ages, all-causes, a 10% [1.10 (95% CI: 1.06, 1.14)] increase in risk of death on a heat day versus a non-heat day in King County, Washington. When considering the intensity effect of heat on mortality, we found a 1.69% (95% CI: 0.69, 2.70) increased risk of

death per degree increase in average county-wide daily maximum humidex above 36.0 °C for all-ages, all-causes. Mortality stratified by cause and age produced statistically significant results with both types of analyses for non-traumatic, circulatory, cardiovascular, cerebrovascular, and diabetes causes. This study identified a younger, vulnerable diabetic population (45-64 age group), having a 78% [1.78 (95% CI: 1.12, 2.83)] greater risk of death on a heat day than on a non-heat day, with risk increasing 14.22% (95% CI: 2.21, 27.64) for each degree increase in humidex above 36.0 °C. Additional, unique age-adjusted results were observed for each type of analysis: an increased risk of death from renal failure, natural heat exposure, mental health and accident causes using the relative risk analysis; and an increased risk of death from suicide, homicide and ischemic causes using the time series analysis. While individual-level characteristics (age being an exception) or other heat effects such as cool-down, duration and lag were not found to affect risk, the synoptic weather mass classification on a heat day significantly influenced mortality.

## **Conclusions**

These results demonstrate that heat, expressed as humidex, is associated with increased mortality on heat days, and that the risk increases with heat's intensity. When stratified by age and cause of death, the contrast between the relative risk and time series analyses is notable; the relative risk estimates for younger age groups are significant for several sub-categories of all cause mortality, whereas, conversely, heat's intensity almost exclusively affects the 85+ age group.

## **2.1 Introduction**

Extreme heat events have contributed to thousands of deaths in the US, Canada and Europe since the early 1980s (Whitman et al. 1997; Naughton 2002; Baccini et al. 2008; Basu,

Feng and Ostro 2008; Anderson and Bell 2009; Jackson et al. 2010; Anderson and Bell 2011). V-, U-, or J-shaped relationships have been identified where there is a minimum mortality temperature (also called “threshold” or “turning point) beyond which mortality increases significantly with increasing heat (Baccini et al. 2008; Curriero et al. 2002; Kim, Ha, and Park 2006). Studies have also suggested that the heat-mortality relationship may not be the same in all locations (Baccini et al. 2008; Curriero et al. 2002). Curriero et al.’s 2002 research demonstrated a stronger association in mortality risk for northern cities in the US, at lower temperatures, when compared to southern cities.

Other factors have been identified as modifying heat’s influence on mortality rates including intensity and duration of the described heat event (Anderson and Bell 2011; Baccini et al. 2008), synoptic weather patterns (Kalkstein et al. 2011; Sheridan, Kalkstein and Kalkstein 2009), access to air conditioning (Naughton 2002; O’Neill, Zanobetti, and Schwartz 2005), social isolation (Naughton 2002; Kaiser et al. 2001), socio-economic status (Jones et al. 1982, Kaiser et al. 2001), and ethnicity and educational status (O’Neill, Zanobetti, and Schwartz 2003). It is important to note that across all of the above referenced studies, there is significant variability in the results - suggesting that *place*, or regionality matters. Yardley, Sigal, and Kenny (2010)’s literature review further supports the notion that place matters. The authors looked at the importance of social and community factors in heat-health planning. They found that the spatial distribution in heat-related mortality reported in the literature indicates more at play than just temperature and physiology. As average temperatures and the frequency of extreme heat events are predicted to increase with climate change, understanding the regional heat-mortality relationship becomes increasingly important to direct adaptation-related policy decisions.

The current study investigated the relationship between heat and mortality in the 14<sup>th</sup> most populous county in the United States, King County, Washington (King County 2012). Two different modeling approaches were used. We first built upon our previous work that found significant increases in non-traumatic mortality associated with heat days compared to non-heat days in the Pacific Northwest region (Jackson et al. 2010). We used a relative risk (RR) model to explore an expanded list of age adjusted, cause-of-death categories requested by the local health jurisdiction. Second, we added a time series analysis to study heat intensity effects on mortality separately from effects associated with duration. Specifically, we quantified the percentage increase in mortality associated with a one degree increase in humidex for the same expanded list of age-adjusted, cause-of-death categories. Using the time series model, we were able to explore effect modification from individual-level characteristics, as well as analyze other effects from heat, including cool down, duration, and lag effects. To our knowledge, no other study has looked at such a comprehensive list of mortality categories in the Pacific Northwest, using two methods of analyses.

## **2.2 Methods**

### **2.2.1 Mortality and population data**

King County death certificate data for all causes, 1980 to 2010, were obtained from the Washington State Department of Health. Only deaths which occurred during the warmer summer months of May through September were analyzed. Colder months were excluded to minimize potential confounding by infectious diseases typically seen during these months. There are 153 days per constrained calendar year, a total of 4,743 days for the entire study period. Death certificates were coded using the International Classification of Diseases (ICD). ICD-9 codes were used from 1980 to 1998 and ICD-10 codes from 1999 to 2010.

This study expanded the cause-of-death categories analyzed by Jackson et al. (2010) and Busch Isaksen et al. (2014) from only non-traumatic causes (ICD-9 001-799, ICD-10 A01-R99) to including all causes of mortality (ICD-9 000, A00+). We also investigated select subsets of all-cause mortality that were determined *a priori* through the literature (Jackson et al. 2010; Busch Isaksen et al. 2014; Cheng et al. 2005) or that were specifically requested by our community partners. In addition to the overall groupings of all-cause and non-traumatic mortality, we investigated the relationship between heat and diabetes (ICD-9 250, E08-E13), circulatory (ICD-9 390-459, ICD-10 I00-I99, G45, G46), cardiovascular (ICD-9 393-429, ICD-10 I05-I52), ischemic (ICD-9 410-414, ICD-10 I20-I25), cerebrovascular (ICD-9 430-438, ICD-10 I67), respiratory (ICD-9 460-519, ICD-10 J00-J99), nephritis and nephrotic (ICD-9 580-589, ICD-10 N17-N19), acute renal failure (ICD-9 584, ICD-10 N17), mental health (ICD-9 290-316, ICD-10 F01-F69) and natural heat exposure including dehydration (ICD-9 E900.0 or E900.9 & 992, 276.51, ICD-10 X30 & T67, E86.0). Our analyses also made use of the death certificate database variable, *Inj\_cause*, that describes whether a death is classified as natural, accidental, suicide, or homicide. Table 2.1 lists the specific ICD-9 and 10 codes used in this study.

**Table 2.1** Underlying causes of death analyzed and associated death certificate ICD-9 (1980 to 1998) & ICD-10 (1999-2010) codes

<i>Category</i>	<i>ICD-9 Code</i>	<i>ICD-10 Code</i>
<b>All Causes</b>	000+	A00+
<b>Non-traumatic</b>	001-799	A00-R99
<b>Select Non-Traumatic Causes</b>		
<b>Diabetes</b>	250	E08-E13
<b>Circulatory</b>	390-459	I00-I99, G45, G46
<b>Cardiovascular</b>	393-429	I05-I52
<b>Ischemic</b>	410-414	I20-I25
<b>Cerebrovascular</b>	430-438	I67
<b>Respiratory</b>	460-519	J00-J99
<b>Nephritis and Nephrotic</b>	580-589	N17-N19
<b>Acute Renal Failure</b>	584	N17
<b>Mental Health</b>	290-316	F01-F69
<b>Select Traumatic Causes</b>		
<b>Natural Heat Exposure<sup>1</sup></b>	E900.0 or E900.9 & 992	X30 & T67
<b>Accident<sup>2</sup></b>	2	2
<b>Suicide<sup>2</sup></b>	3	3
<b>Homicide<sup>2</sup></b>	4	4

<sup>1</sup> While mortality attributed to natural heat-related exposure is classified as a traumatic external injury, we have included the non-traumatic dehydration code ICD-9 276.51/ICD-10 E86.0 with this sub-grouping.

<sup>2</sup> The Washington State Department of Health codes the variable “Inj\_caus” as the type of external injury indicated by the underlying cause of death: Accident=2; Suicide=3; and Homicide=4.

*A priori* we anticipated that several individual-level characteristics might identify populations that are vulnerable to heat-related mortality: age, non-White race, less than a high school education, Hispanic origin, and tobacco contribution to death. Population data, by age groups (0-4, 5-14, 15-44, 45-64, 65-84, 85+), were obtained from the Washington State Office of Financial Management (OFM) (Washington State Office of Financial Management 2012).

### **2.2.2 Meteorology data**

This study used a historical (1915-2012) 1/16° gridded resolution meteorological data set produced by the University of Washington's Climate Impacts Group (Maurer et al. 2002). The climatologic foundation of this data set is the Parameter-Elevation Relationships on Independent Slopes Model (PRISM), developed by Oregon State University (PRISM Climate Group). PRISM is considered to represent the most current knowledge on spatial climatic patterns for the United States (Daly et al. 2007). Daily temperature and relative humidity values for our meteorological data set were constructed using PRISM's regional spatial climatic patterns and weather station observations from the Global Historical Climate Network-Daily (GHCN). GHCN is a National Oceanic and Atmospheric Administration database of daily meteorological measurements from land surface stations across the globe (NOAA 2009). The resulting meteorology data used for this study contain daily max/min temperature, precipitation and relative humidity values for each meteorological center point. The county-wide daily maximum temperature and average relative humidity values were used to construct our exposure metric, humidex.

### **2.2.3 Exposure assessment**

As with our previous studies (Jackson et al. 2010; Busch Isaksen et al. 2014), we used humidex as the measure of exposure. Humidex is a feels-like index that measures the combined effects of temperature and humidity on the human body (Masterton and Richardson 1979).

An average daily maximum humidex was computed over all meteorological center points located in King County. Exposure to heat was then estimated as the average maximum county-wide humidex value for each day within the study's time frame. Humidex is defined by the following formula and is expressed in units of °C:

$$\mathbf{Humidex} = \mathbf{T} + \left(\frac{\mathbf{5}}{\mathbf{9}}\right) \cdot (\mathbf{v} - \mathbf{10}), \text{ where} \tag{1}$$

$$\mathbf{v} = \left( \mathbf{6.112} \cdot \mathbf{10}^{\left[\frac{\mathbf{7.5T}}{\mathbf{237.7+T}}\right]} \right) \cdot \frac{\mathbf{H}}{\mathbf{100}}$$

where  $T$  is the air temperature (degrees Celsius),  $H$  is the average relative humidity (%), and  $v$  is the vapor pressure (kPa) (Canadian Centre for Occupational Health and Safety 2011). In this analysis, “humidex” refers to the “county-wide average daily maximum humidex”.

#### 2.2.4 Association between humidex and mortality

##### *Relative risk analysis*

A heat day was defined as a day in which the average King County-wide daily maximum humidex exceeded a specified threshold. Jackson et al. (2010) used the 99<sup>th</sup> percentile of the average greater Seattle area-wide (King, Pierce, and Snohomish counties) maximum humidex as the threshold to define a heat day. In this analysis, we tried the 99<sup>th</sup>, 95<sup>th</sup> and 90<sup>th</sup> percentiles for King County and chose the one that gave the best fit to the data (with a maximum likelihood or a minimum deviance). The following Poisson regression model was used:

$$E(Y_j) = P_j \lambda_j + \beta_1 I\{\mathbf{humidex}_j > \mathbf{threshold}\} \tag{2}$$

where  $Y_j$  is the mortality count on day  $j$ ,  $P_j$  is the population,  $\lambda_j$  is the mortality incidence rate and  $\beta_1 I\{\mathbf{humidex}_j > \mathbf{threshold}\}$  is the indicator of a heat day.

This approach modeled the expected mortality count after controlling for population growth.

### *Time Series Analysis*

A Poisson model was built to explore the relationship between daily humidex and mortality rates. Similar to other studies (Anderson and Bell 2009; Baccini et al. 2008; Curriero et al. 2002), we used nonparametric splines to model the log-mortality rate over time and humidex. Specifically, we assumed that:

$$Y_j \sim \text{Poisson}(P_j \lambda_j), \text{ with} \\ \log \frac{\mu_j}{\text{popu}} = \beta_0 + s(\mathbf{h}_j) + s(\mathbf{t}_j) + \sum_{l=6}^9 \beta_l I_{\{\text{month}_j=l\}} \quad (3)$$

where  $\mu_j$  is the expected mortality count on day  $j$ ,  $s(\mathbf{h}_j)$  is a penalized regression spline modeling the effects of humidex,  $s(\mathbf{t}_j)$  is a penalized regression spline modeling the overall trend of mortality over 31 years, and  $(\beta_l$ 's) is the adjustment for seasonal monthly effects.

To increase interpretability and usefulness for public health practitioners and policymakers, we simplified the nonparametric spline model with a piece-wise linear model, fit with two knots. The first knot was set at the 50<sup>th</sup> percentile of summer-time humidex values. The maximum likelihood for the second knot, or “optimal alert threshold” for humidex, was identified by exploring 0.1 degree incremental changes starting at 20 °C and continuing through 44 °C humidex. The following model represents the threshold tuning process:

$$\log \frac{\mu_j}{\text{popu}} = \beta_0 + \beta_1(\mathbf{h}_j - \mathbf{h}_{q50})_+ + \beta_2(\mathbf{h}_j - \hat{\mathbf{h}}_0)_+ + s(\mathbf{t}_j) + \sum_{l=6}^9 \beta_l I_{\{\text{month}_j=l\}} \quad (4)$$

where  $\mathbf{h}_j$  is the county-wide average daily maximum humidex value on day  $j$ ,  $\mathbf{h}_{q50}$  is the 50<sup>th</sup> percentile of humidex from May to September, 1980-2010,  $\hat{\mathbf{h}}_0$  is the optimal alert threshold,  $s(\mathbf{t}_j)$  is a natural cubic spline modeling the overall trend of mortality over 31 years, and  $I_{\text{month}}$  is the indicator variable for months May through September. A heat day was then defined as a

day in which the average county-wide daily maximum humidex exceeded the optimal alert threshold. The impact of heat intensity on mortality was assessed by the slope of the line above the threshold. The “mgcv” and “GAM” packages were used with the statistical software R version 2.14.1 to determine the model’s degrees of freedom and to tune the threshold, respectively (R Core Team 2012).

### 2.2.5 Effect modification with individual-level characteristics

Individual-level characteristic data obtained from death certificates were evaluated for differences in mortality risk. These covariates included age, gender, race, high school graduation, marital status, Hispanic origin, and tobacco use. Effect modification was examined by adding each covariate into the model along with an interaction term. Mortality rates for all covariate groups, except age, were not adjusted by population size of the covariate group, because the data were not available. An example of exploring effect modification can be illustrated by the following example of the covariate “Hispanic origin”:

$$\mathbf{Log}\left(\frac{\mu_{ij}}{\text{popu}}\right) = \beta_0 + \beta_1(h_j - h_{q50}) + \beta_2 \mathbf{Hisp}_i + \beta_3(h_j - h_{q50}) \cdot \mathbf{Histp}_i \quad (5)$$

where  $\mu_{ij}$  is the expected mortality count for sub-population with covariate level  $i$  on day  $j$ . In this example, the covariate “Hispanic origin” has two levels “Hispanic” and “non-Hispanic”. The parameter of interest is  $\beta_3$ , the coefficient of the interaction between heat intensity indicator and the covariate. By testing the significance of the interaction between demographic variable and the heat variable, we can identify whether specific subpopulations are more vulnerable to heat intensity effects.

## 2.2.6 Other heat effects on mortality

Several studies have suggested that cooler night-time temperatures help minimize the effect of heat on mortality (Schwartz 2005), that lengthier heat events increase mortality risk (Daniela et al. 2010; Anderson and Bell 2011), and that the type of synoptic weather pattern may influence mortality rates on heat days (Kalkstein and Green 1997; Sheridan, Kalkstein and Kalkstein 2009).

This study evaluated the data to see if there was a “cool down effect,” whereas an elevated average county-wide minimum humidex on a hot day would contribute to an increase in mortality beyond the effect of the maximum humidex during the day. For data on heat days, cool down effect is explored using the following two models:

$$\log \frac{\mu_j}{popu} = \beta_0 + \beta_2 \mathbf{difference} \quad (6)$$

$$\log \frac{\mu_j}{popu} = \beta_0 + \beta_1 \mathbf{aboveThres} + \beta_2 \mathbf{difference} \quad (7)$$

where **difference** is defined as the daily maximum humidex - daily minimum humidex for a given heat day, and **aboveThres** is defined as the daily maximum humidex - threshold.

Similarly, this study examined the relationship between mortality count and heat event duration (number of consecutive heat days). For data on heat days, a duration effect is explored using the following two models:

$$\log \frac{\mu_j}{popu} = \beta_0 + \beta_2 \mathbf{duration} \quad (8)$$

$$\log \frac{\mu_j}{popu} = \beta_0 + \beta_1 \mathbf{aboveThres} + \beta_2 \mathbf{duration} \quad (9)$$

where **duration** is defined as the day's order in a given heat event.

This study also explored whether or not there was a lag effect between humidex and mortality over several days. A lag effect can best be described as the total heat effect on mortality spread over several days or weeks. Following the methods described in Armstrong (2006) we explored distributed lag effects using the following model:

$$E(Y_j) = \exp\{\alpha + f(\mathbf{h}_{\sim j}) + \text{covariates} + s(\text{time})\},$$

$$f(\mathbf{h}_{\sim j}) = \sum_{l=0}^L \beta_l \mathbf{h}_{j-l}, \quad (10)$$

where  $Y_j$  is the mortality count on day  $j$ ,  $s(\text{time})$  is a spline curve over time, and  $f(\mathbf{h}_{\sim j})$  is a function of historic county-wide average maximum humidex values on days up to day  $j$ , which takes a weighted effect of humidex on day  $j$  and the previous  $L$  days. By assuming different constraints or temporal structures for  $\beta_l, l = 0, \dots, L$ , we examined the evidence of lag effects. This was implemented using the “dlnm” (distributed lag non-linear model) package in R version 2.14.1 (Gasparrini 2011).

Finally, we investigated whether or not mortality rates were influenced by the type of synoptic weather mass on a given heat day. Previous research indicates that moist and dry tropical air masses are associated with increased mortality (Kalkstein et al. 2011; Sheridan, Kalkstein and Kalkstein 2009). Using daily spatial synoptic classification data for the Seattle/Tacoma station (Sheridan 2013), effect modification was explored by adding synoptic classification as a covariate into the model along with an interaction term, Equation (5).

## **2.3 Results**

### **2.3.1 King County population and climate**

King is the largest county in Washington State and accounts for approximately 29%-31% of the State's population throughout the study's time frame (Washington State Office of Financial Management 2012). From 1980 to 2010 the county's population increased over 52%, with age groups 45-64, 65-84, and 85+ increasing 110%, 52.6%, 150%, respectively. King County is located in Western Washington and is characterized by relatively cool summers. Its summer humidex values range from a minimum average of 6.66 °C (44.0 °F) to a maximum average of 22.4 °C (72.3 °F). From 1980 to 2010 (May-September), King County experienced 135,333 deaths, 34.2% of the overall death count. On average, there were 28.5 deaths per day. Table 2.2 reports population by age and percent change, meteorological ranges, and mortality counts for individual level characteristics, 1980-2010.

### **2.3.2 Association between humidex and mortality**

The plotted relationship between summertime daily mortality count and humidex was u-shaped, illustrating an increase in mortality with extreme humidex values (Figure 2.1).

#### ***Relative risk analysis***

As defined by Equation (2), a heat day for the relative risk analysis is a day that exceeds the 99<sup>th</sup> percentile (36.1 °C (97.0 °F) humidex). During 1980-2010, King County experienced 114 days over the 99<sup>th</sup> percentile. The average humidex on heat days was 38.7 °C (101.7 °F) (Table 2.2). For all-ages, all-causes of mortality, we found that the relative risk of death on a heat day was 10% [1.1 (95% CI: 1.06, 1.14)] greater than on a non-heat day. Statistically significant all-age results were found for non-traumatic 10% [1.1 (95% CI: 1.06, 1.14)], circulatory 9%

**Table 2.2** King County, Washington population and climate demographics, 1980–2010

<b>1980 Population<sup>1</sup></b>	Complete Count
State Total	4,132,156
King County Total	1,269,749
% of State Population	30.7%
0-4	78,525
5-14	170,657
15-44	643,707
45-64	247,446
65-84	115,910
85+	13,504
<b>2010 Population<sup>2</sup></b>	Complete Count (% Change)
WA State Total	6,724,540 (62.7%)
King County Total	1,931,249 (52.1%)
% of State Population	28.7% (-7.0%)
0-4	120,294 (53.2%)
5-14	224,084 (31.3%)
15-44	856,843 (33.1%)
45-64	519,349 (110%)
65-84	176,895 (52.6%)
85+	33,784 (150%)
<b>County-Wide Humidex °C(°F) (May-Sept.)</b>	
Min. Daily Humidex, Av. over time	6.66 °C (44.0 °F)
Max. Daily Humidex, Av. over time	22.4 °C (72.3 °F)
<b>Heat Event Days (May-Sept.)</b>	
<b>Heat Days above Relative Risk Threshold</b> (99 <sup>th</sup> percentile 36.1 °C (97.0°F))	114 / 4,743 total days (2.40%)
<b>Average County-Wide Max. on Heat Days</b>	
Humidex	38.7 °C (101.7 °F)
Temperature	30.9 °C (87.6 °F)
<b>Minimum County-Wide Max. on Heat Days</b>	
Humidex	36.1 °C (97 °F)

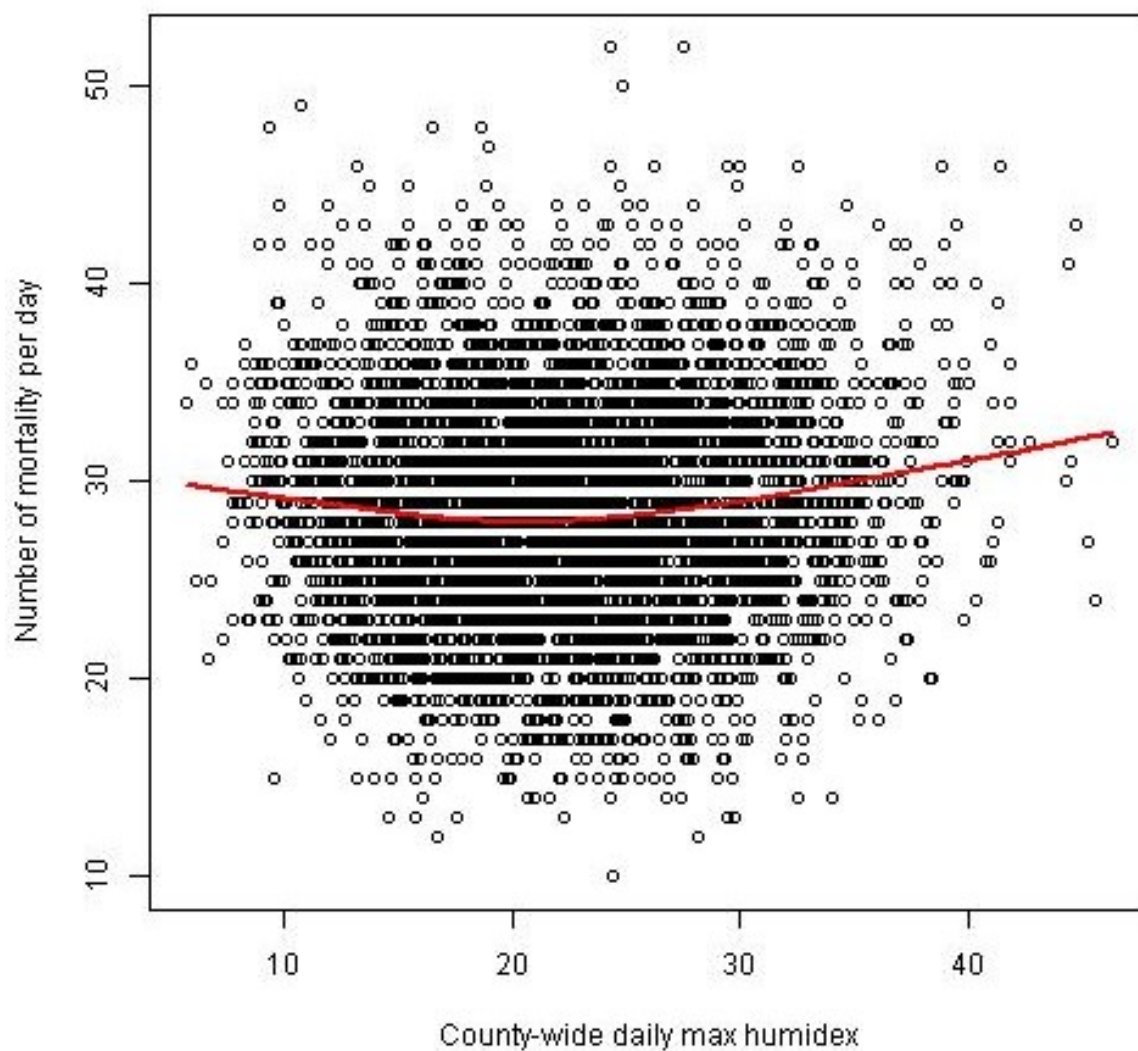
<sup>1</sup> Source: Washington State Department of Health mortality data set

<sup>2</sup> Source: Washington State Office of Financial Management <http://www.ofm.wa.gov/pop/census2010/data.asp>

Temperature	28.0 °C (82.4 °F)
<b>Maximum County-Wide Max. on Heat Days</b>	
Humidex	46.3 °C (115.3 °F)
Temperature	34.9 °C (94.8 °F)
<b>Heat Days above Time Series Threshold</b> (36.0 °C (96.8 °F))	117 days /4,743 total days (2.47%)
<b>Average County-Wide Max. on Heat Days</b>	
Humidex	38.6 °C (101.4 °F)
Temperature	30.8 °C (87.4 °F)
<b>Minimum County-Wide Max. on Heat Days</b>	
Humidex	36.0 °C (96.8 °F)
Temperature	28.0 °C (82.4 °F)
<b>Maximum County-Wide Max. on Heat Days</b>	
Humidex	46.3 °C (115.3 °F)
Temperature	34.9 °C (94.8 °F)
<b>Total King County Deaths 1980-2010</b>	<b>395,138</b>
<b>May - September 1980-2010</b>	
Total Deaths (% of total)	135,333 (34.2%)
Average Daily Mortality	28.5 deaths/day
<b>Individual Level Characteristics</b>	
<b>Gender</b>	
Male	68,203 (50.4%)
Female	67,130 (49.6%)
<b>High School Graduate Status (% of total)</b>	
High School Diploma or better	79,298 (58.6%)
No High School Diploma	53,217 (39.3%)
Status Unknown	2,818 (2.1%)
<b>Hispanic Ethnicity (% of total)</b>	
Hispanic	1,557 (1.2%)
Non-Hispanic	133,505 (98.6%)
Unknown	271 (0.2%)
<b>Smoking Status (% of total)</b>	
Smoker	27,287 (20.2%)
Non-smoker	94,774 (70.0%)
Unknown	13,272 (9.8%)

<b>Race (% of total)</b>	
White	118,987 (87.9%)
Black	7,027 (5.2%)
Asian/Pacific Islander	6,935 (5.1%)
Native American	1,184 (0.87%)
Other	1,031 (0.76%)
Unknown	169 (0.17%)
<b>Marital Status (% of total)</b>	
Married or Domestic Partner	52,098 (38.5%)
Other	83,235 (61.5%)

**Figure 2.1** Lowess nonparametric regression curve modeling county-wide maximum humidex effect over daily mortality counts, 1980-2010



[1.09 (95% CI: 1.02, 1.16)], cerebrovascular 40% [1.4 (95% CI: 1.15, 1.69)], and accident 19% [1.19 (95% CI: 1.02, 1.39)]. When investigating mortality stratified by age, statistically significant increases in risk were found for: the 0-4 age group, renal 904% [10.04 (95% CI: 1, 100.42)]; the 45-64 age group, diabetes 78% [1.78 (95% CI: 1.12, 2.83)], and natural heat exposure 2,261% [23.61(95% CI: 4.45, 125.14)]; the 65-84 age group, all-causes 6% [1.06 (95% CI: 1.01, 1.12)], non-traumatic 6% [1.06 (95% CI: 1, 1.12)], cerebrovascular 37% [1.37 (95% CI: 1.08, 1.74)], mental health 43% [1.43 (1.02, 2.01)], and accident 43% [1.43 (95% CI: 1.03, 1.98)]; and the 85+ age group, all causes 18% [1.18 (95% CI: 1.11, 1.26)], non-traumatic 18% [1.18 (95% CI: 1.11, 1.26)], circulatory 18% [1.18 (95% CI: 1.06, 1.3)], cardiovascular 17% [1.17 (95% CI: 1.04, 1.31)], and cerebrovascular 53% [1.53 (95% CI: (1.17, 2.01)]. Relative risk estimates and 95% confidence intervals for all age groups and categories of death are reported in Table 2.3. It should be noted that both the 0-4 age group's nephritis and nephrotic syndromes and the 45-64 age group's natural heat exposure estimates are based on a small number of cases as reported in Table 2.3

**Table 2.3** Relative risk analysis results: Increased risk (95% CI) in mortality on a 99<sup>th</sup> percentile (36.1 °C) heat day compared to a non-heat day, by age group and cause of death <sup>1</sup>

	<i>All Ages</i>	<i>0-4</i>	<i>5-14</i>	<i>15-44</i>	<i>45-64</i>	<i>65-84</i>	<i>85+</i>
<b>All Causes</b>	<b>1.1</b> <b>(1.06, 1.14)</b>	0.73 (0.51, 1.03)	1.11 (0.61, 2.04)	1.02 (0.9, 1.17)	1.03 (0.94, 1.13)	<b>1.06</b> <b>(1.01, 1.12)</b>	<b>1.18</b> <b>(1.11, 1.26)</b>
<b>Non-traumatic</b>	<b>1.1</b> <b>(1.06, 1.14)</b>	0.75 (0.53, 1.08)	1.07 (0.44, 2.63)	0.98 (0.82, 1.18)	1.04 (0.94, 1.14)	<b>1.06</b> <b>(1, 1.12)</b>	<b>1.18</b> <b>(1.11, 1.26)</b>
<b>Select Non-Traumatic Causes</b>							
<b>Diabetes</b>	1.2 (0.93, 1.55)	0.99 (0.82, 1.19)	0 (0, Inf)	0.64 (0.15, 2.64)	<b>1.78</b> <b>(1.12, 2.83)</b>	1.09 (0.76, 1.56)	0.93 (0.47, 1.85)
<b>Circulatory</b>	<b>1.09</b> <b>(1.02, 1.16)</b>	1.54 (0.38, 6.26)	0 (0, Inf)	1.11 (0.82, 1.5)	0.94 (0.78, 1.13)	1.03 (0.94, 1.14)	<b>1.18</b> <b>(1.06, 1.3)</b>
<b>Cardiovascular</b>	1.07 (0.99, 1.15)	0.93 (0.13, 6.73)	0 (0, Inf)	1.06 (0.76, 1.48)	0.99 (0.82, 1.2)	0.98 (0.88, 1.1)	<b>1.17</b> <b>(1.04, 1.31)</b>
<b>Ischemic</b>	1.06 (0.97, 1.16)	0 (0, Inf)	1 (0.83, 1.2)	1.1 (0.54, 2.23)	1.09 (0.87, 1.37)	0.98 (0.86, 1.12)	1.12 (0.97, 1.3)
<b>Cerebrovascular</b>	<b>1.4</b> <b>(1.15, 1.69)</b>	0 (0, Inf)	0 (0, Inf)	1.64 (0.66, 4.09)	0.59 (0.26, 1.33)	<b>1.37</b> <b>(1.08, 1.74)</b>	<b>1.53</b> <b>(1.17, 2.01)</b>
<b>Respiratory</b>	1.11 (0.99, 1.25)	0 (0, Inf)	0 (0, Inf)	0.98 (0.4, 2.38)	1.23 (0.88, 1.73)	1.05 (0.89, 1.23)	1.15 (0.95, 1.4)
<b>Nephritis and Nephrotic</b>	1.31 (0.91, 1.88)	<b>10.04</b> <b>(1, 100.42)<sup>2</sup></b>	0 (0, Inf)	0 (0, Inf)	0.99 (0.31, 3.15)	1.22 (0.71, 2.11)	1.48 (0.83, 2.63)
<b>Acute renal failure</b>	1.08 (0.39, 2.94)	2.28e+9 (0, Inf)	1 (0.83, 1.2)	0 (0, Inf)	3.03 (0.41, 22.34)	0 (0, Inf)	1.27 (0.33, 4.95)
<b>Mental Health</b>	1.21 (0.97, 1.5)	0.99 (0.82, 1.19)	1 (0.83, 1.2)	0.37 (0.05, 2.57)	0.7 (0.26, 1.88)	<b>1.43</b> <b>(1.02, 2.01)</b>	1.16 (0.86, 1.56)
<b>Select Traumatic Causes</b>							
<b>Natural Heat Exposure</b>	1.89 (0.82, 4.39)	0 (0, Inf)	0 (0, Inf)	0 (0, Inf)	<b>23.61</b> <b>(4.45, 125.14)<sup>3</sup></b>	0 (0, Inf)	1.6 (0.45, 5.69)

<sup>1</sup> Bolded relative risk values are significantly greater than 1 ( $p < 0.05$ )

<sup>2</sup> While statistically significant, the estimate is based on a small number of cases [4 cases on non-heat days, 1 case on a heat day]

<sup>3</sup> While statistically significant, the estimate is based on a small number of cases [5 cases on non-heat days, 3 cases on a heat day]

	<i>All Ages</i>	<i>0-4</i>	<i>5-14</i>	<i>15-44</i>	<i>45-64</i>	<i>65-84</i>	<i>85+</i>
<b>Accident</b>	<b>1.19</b> <b>(1.02, 1.39)</b>	0.58 (0.14, 2.51)	1.24 (0.51, 3.02)	1.16 (0.92, 1.46)	1.14 (0.83, 1.55)	<b>1.43</b> <b>(1.03, 1.98)</b>	1.17 (0.75, 1.84)
<b>Suicide</b>	0.87 (0.66, 1.13)	0.99 (0.82, 1.19)	0 (0, Inf)	0.91 (0.63, 1.31)	0.83 (0.51, 1.36)	0.52 (0.22, 1.24)	2.68 (0.93, 7.69)
<b>Homicide</b>	0.83 (0.52, 1.34)	0 (0, Inf)	1.76 (0.23, 13.34)	0.9 (0.54, 1.52)	0.44 (0.11, 1.81)	1.74 (0.36, 8.29)	0 (0, Inf)

### *Time series analysis*

In King County, the time series relationship between humidex and log-mortality rates is J-shaped. Figure 2.2 illustrates a penalized cubic regression spline modeling effects of humidex over mortality (Equation 3). The relationship suggests an increased risk of mortality from exposure to humidex exceeding approximately 30 °C humidex. An optimal heat day threshold for the time series analysis was derived by setting the first knot at  $h_{q50} = 22.1$  °C and incrementally increasing the second knot by 0.1 °C. The optimal threshold for King County is just shy of the 99<sup>th</sup> percentile, at 36.0 °C (96.8 °F) (Figure 2.3). During 1980-2010, King County experienced 117 days that exceeded this threshold. The average humidex on these exceedance days was 38.6 °C (101.4 °F).

For all-ages, all-causes, we observed a 1.69% (95% CI: 0.69, 2.7) increase in mortality per degree increase in average county-wide daily maximum humidex above 36.0 °C. Statistically significant all-age results were also found for non-traumatic 2.12% (95% CI: 1.07, 3.19), circulatory 1.98% (95% CI: 0.33, 3.66), ischemic 2.49% (95% CI: 0.08, 4.96), cerebrovascular 6.23% (95% CI: 2.03, 10.61), and respiratory 4.37% (95% CI: 1.11, 7.74) causes of death. When investigating mortality stratified by age, statistically significant results were found for the: 45-64 age group, diabetes 14.22% (95% CI: 2.21, 27.64); the 65-84 age group, homicide 43.73% (95% CI: 0.61, 105.31); and the 85+ age group, all-causes 3.74% (95% CI: 1.77, 5.74), non-traumatic 3.83% (95% CI: 1.85, 5.86), circulatory 4.14% (95% CI: 1.3, 7.06), cardiovascular 4.27% (95% CI: 0.91%, 7.74%), ischemic 4.22% (95% CI: 0.36%, 8.24%), cerebrovascular 9.68% (95% CI: 3.79, 15.92), and suicide 43.39% (95% CI: 11.85, 83.84). Intensity estimates and 95% confidence intervals for all age groups and categories of death are reported in Table 2.4. It should

be noted that both the 65-84 age group's homicide and the 85+ age group's suicide estimates are based on a small number of cases as reported in Table 2.4.

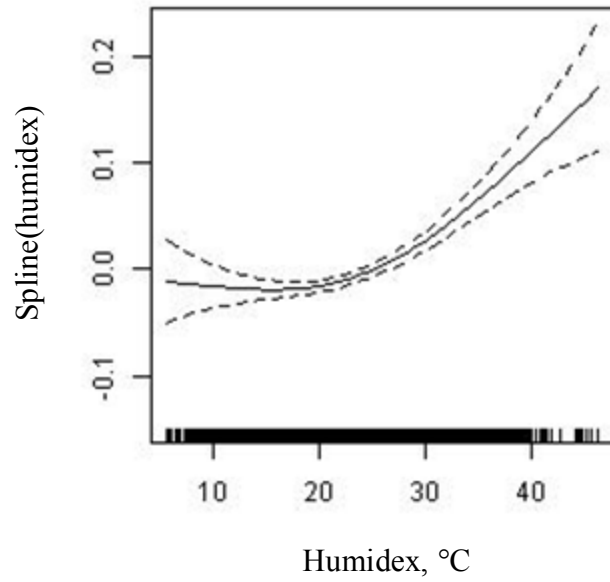
### **2.3.3 Effect modification with individual-level characteristics**

We did not find that the following individual-level characteristics altered the risk of dying on a heat day: gender, race, high school graduation, marital status, Hispanic origin, or whether or not tobacco use contributed to death. However, we did find that age has a statistically significant relationship with the risk of dying on a heat day. In both the relative risk and time series analysis we found the 85+ age group to be at the greatest risk for all causes of mortality.

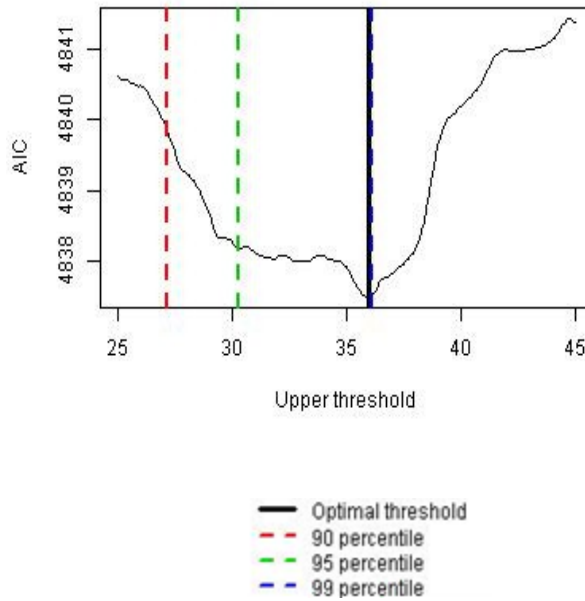
### **2.3.4 Other heat effects on mortality**

We did not find a statistically significant cool-down effect on mortality; the difference between minimum and maximum humidex on a given heat day did not significantly influence mortality rates. Likewise, we found no effect on mortality from duration of consecutive heat days above threshold. Additionally, we found no significant effect on the mortality rate from the lagged humidex, while the acute association between excess humidex above the upper threshold and mortality on the same day remained statistically significant. Lastly, mortality associated with the type of synoptic weather mass on a given heat day was explored. A day classified as having either a moist or dry tropical air mass was associated with a 39.6% (95% CI: 28.1, 52.1) increase in all-age, all-cause mortality, per one degree increase above 36.0 °C, as compared to a day with a non-moist/dry tropical air mass classification.

**Figure 2.2** Penalized cubic regression spline modeling humidex effect over log-mortality



**Figure 2.3** The optimal mortality threshold for King County identified by the Akaike Information Criterion (AIC), maximum likelihood of the model fit



**Table 2.4** Time series analysis results: Percentage (95% CI) increase in mortality per degree increase in county-wide average daily maximum humidex °C above 36.0 °C, by age group and underlying cause of death<sup>1</sup>

	<i>All Ages</i>	<i>0-4</i>	<i>5-14</i>	<i>15-44</i>	<i>45-64</i>	<i>65-84</i>	<i>85+</i>
<b>All Causes</b>	<b>1.69%</b> ( <b>0.69%</b> , <b>2.7%</b> )	-5.92% (-16%, 5.38%)	4.31% (- 11.84%, 23.42%)	-4.17% (-8.07%, -0.1%)	-0.33% (-2.9%, 2.31%)	0.97% (-0.66%, 2.62%)	<b>3.74%</b> ( <b>1.77%</b> , <b>5.74%</b> )
<b>Non-traumatic</b>	<b>2.12%</b> ( <b>1.07%</b> , <b>3.19%</b> )	-4.26% (-14.76%, 7.53%)	12.68% (-13.63%, 47.02%)	1.66% (-3.92%, 7.57%)	-0.25% (-2.97%, 2.54%)	0.91% (-0.73%, 2.59%)	<b>3.83%</b> ( <b>1.85%</b> , <b>5.86%</b> )
<b>Select Non-Traumatic Causes</b>							
<b>Diabetes</b>	2.53% (-4.73%, 10.33%)	-0.05% (-100%, 1.99e+82%)	-92.04% (-100%, 3.68e+98%)	13.29% (-13.91%, 49.09%)	<b>14.22%</b> ( <b>2.21%</b> , <b>27.64%</b> )	-3.66% (-13.37%, 7.13%)	-14.06% (-31.21%, 7.36%)
<b>Circulatory</b>	<b>1.98%</b> ( <b>0.33%</b> , <b>3.66%</b> )	-11.16% (-58.49%, 90.16%)	-100% (-100%, Inf%)	0.03% (-9.04%, 10.01%)	-0.95% (-5.77%, 4.12%)	0.59% (-2.02%, 3.27%)	<b>4.14%</b> ( <b>1.3%</b> , <b>7.06%</b> )
<b>Cardiovascular</b>	1.74% (-0.17%, 3.69%)	-8.56% (-63.53%, 129.26%)	5.23% (-100%, 1.23e+8%)	-3.11% (-13.1%, 8.03%)	0.54% (-4.59%, 5.96%)	0.06% (-2.97%, 3.18%)	<b>4.27%</b> ( <b>0.91%</b> , <b>7.74%</b> )
<b>Ischemic</b>	<b>2.49%</b> ( <b>0.08%</b> , <b>4.96%</b> )	-7.69% (-100%, 1.03e+41%)	-0.1% (-100%, 1.45e+49%)	7.6% (-13.53%, 33.89%)	4.25% (-1.32%, 10.12%)	0.38% (-2.85%, 3.72%)	<b>4.22%</b> ( <b>0.36%</b> , <b>8.24%</b> )
<b>Cerebrovascular</b>	<b>6.23%</b> ( <b>2.03%</b> , <b>10.61%</b> )	7.64e+206% (-100%, Inf%)	-100% (-100%, Inf%)	1.56% (-22.8%, 33.62%)	-6.51% (-22.02%, 12.09%)	3.21% (-2.41%, 9.15%)	<b>9.68%</b> ( <b>3.79%</b> , <b>15.92%</b> )
<b>Respiratory</b>	<b>4.37%</b> ( <b>1.11%</b> , <b>7.74%</b> )	-100% (-100%, Inf%)	-100% (-100%, Inf%)	10.73% (-15.37%, 44.88%)	5.36% (-4.96%, 16.8%)	3.1% (-1.82%, 8.28%)	3.59% (-2.12%, 9.64%)
<b>Nephritis and Nephrotic</b>	7.74% (-2.22%, 18.72%)	18.34% (-32.76%, 108.24%)	-100% (-100%, Inf%)	-100% (-100%, Inf%)	-7.99% (-34.79%, 29.82%)	5.48% (-9.72%, 23.24%)	15.72% (-1.59%, 36.07%)

<sup>1</sup> Bolded time series estimates are significantly greater than 0 ( $p < 0.05$ )

	<i>All Ages</i>	<i>0-4</i>	<i>5-14</i>	<i>15-44</i>	<i>45-64</i>	<i>65-84</i>	<i>85+</i>
<b>Acute renal failure</b>	10.94% (-10.67%, 37.77%)	-100% (-100%, Inf%)	-1.29% (-100%, Inf%)	-100% (-100%, Inf%)	29.14% (-20.59%, 110%)	-91.41% (-99.99%, 6896.47%)	14.2% (-11.76%, 47.81%)
<b>Mental Health</b>	-2.88% (-9.31%, 4.01%)	-0.2% (-100%, 5.2e+127%)	-0.26% (-100%, 1.8e+127%)	-8.3% (-35.76%, 30.9%)	-18.87% (-39.4%, 8.61%)	2.39% (-6.79%, 12.48%)	-7.11% (-14.7%, 1.14%)
<b>Select Traumatic Causes</b>							
<b>Natural Heat Exposure</b>	12.72% (-6.55%, 35.98%)	-100% (-100%, Inf%)	-100% (-100%, Inf%)	-100% (-100%, Inf%)	36.38% (-24.58%, 146.6%)	-100% (-100%, Inf%)	-2.79% (-47.54%, 80.14%)
<b>Accident</b>	-1.45% (-5.82%, 3.13%)	-10.42% (-42.94%, 40.64%)	4.71% (-16.55%, 31.38%)	-10.15% (-16.83%, - 2.95%)	2.77% (-6.25%, 12.67%)	3.74% (-6.08%, 14.59%)	-4.38% (-17.14%, 10.35%)
<b>Suicide</b>	-6.97% (-14.66%, 1.41%)	-0.61% (-100%, Inf%)	-100% (-100%, Inf%)	-7.81% (-17.6%, 3.16%)	-12.85% (-26.98%, 4.01%)	-14.54% (-34.99%, 12.35%)	<b>43.39%</b> <b>(11.85%, 83.84%)<sup>2</sup></b>
<b>Homicide</b>	-7.4% (-20.52%, 7.88%)	-100% (-100%, Inf%)	-10.71% (-53.79%, 72.52%)	-12.58% (-28.7%, 7.19%)	-3.11% (-31.18%, 36.4%)	<b>43.73%</b> <b>(0.61%, 105.31%)<sup>3</sup></b>	-100% (-100%, Inf%)

<sup>2</sup> While statistically significant, the estimate is based on a small number of cases [58 cases on days below optimal threshold, 4 cases on days exceeding optimal threshold]

<sup>3</sup> While statistically significant, the estimate is based on a small number of cases [45 cases on days below optimal threshold, 3 cases on days exceeding optimal threshold]

## 2.4 Discussion

This study characterized King County, Washington's historic heat-mortality relationship using two different statistical methods. Our relative risk analysis quantified the excess mortality on a heat day compared to a non-heat day, while our time series analysis quantified the intensity effect of heat on mortality for each one degree increase in humidex over the threshold. We further characterized risk by age groups and subcategories of all-cause mortality. This study explored cool-down, duration, lag, and synoptic weather pattern effects on mortality, and it quantified effect modification from available individual-level characteristics. The results demonstrate that heat, expressed as humidex, is associated with increased mortality on heat days, and that the risk increases with heat's intensity.

Our study design offers advantages over others, as it allows for direct comparison between two commonly used analyses. First, we are able to compare two ways of defining a heat day: a relative threshold calculated from a fixed percentile to an absolute threshold estimated using a fitted model. Our relative risk analysis uses the 99<sup>th</sup> percentile as the definition of an extreme heat day; the threshold for this is 36.1 °C. In comparison, our time series threshold was calculated at 36.0 °C using a more complicated piece-wise linear model. From a practical standpoint, the similarity between the two thresholds offers support to choosing a fixed percentile as a simple way to define extreme heat. Second, our study design offers a more complete picture of regional heat effects. The relative risk analysis provides a robust analysis of heat's overall contribution to excess mortality on heat days, while the time series analysis allows for a nuanced understanding of the effect of heat on mortality and the role of potential effect modifiers.

This study found a statistically significant increase in all-age, all-cause mortality with both analyses. The relative risk of death on a heat day was 10% greater than on a non-heat day, with risk increasing 1.69% for each degree increase in humidex above 36.0 °C. We also found a statistically significant increase in all-age, non-traumatic mortality for both analyses. The relative risk of death on a heat day was 10% greater than on a non-heat day, with risk increasing 2.12% for each degree increase in humidex above 36.0 °C. Jackson et al. (2010) found a similar 10% increase in mortality risks for the 65+ and 75+ age groups living in the Greater Seattle area. Comparatively, Medina-Ramon and Schwartz's (2007) meta-analysis of 42 cities found a 3.85 % increase in mortality on extreme heat days (above the 99<sup>th</sup> percentile) compared to all other days, while Anderson and Bell (2011) found in their meta analysis of 43 cities that there was a 2.49% increase in heat-wave mortality risk for every 1 °F increase in the intensity of the heat wave.

When investigating a log-linear increase in mortality above an absolute threshold, our previous research using a shorter time frame (1980-2006) found similar threshold and intensity results; each one degree increase in humidex above 35.7 °C was associated with 1.83% increase in non-traumatic causes of death (Busch Isaksen et al. 2014). However, other US-based studies conducted in 9 California counties, 9 U.S. cities, and 20 U.S. cities have found smaller (1-3%) increases in daily non-traumatic mortality per 10 °F increase in daily apparent temperature, equivalent to approximately a 0.2-0.5% increase in mortality per 1 °C increase (Basu, Dominici and Samet 2005; Basu, Feng and Ostro 2008; Zanobetti and Schwartz 2008). Compared to European results, similar magnitude, albeit lower thresholds, were found (1.8% and 3.1% increases per 1 °C apparent temperature for thresholds of 23.3 °C and 29.4 °C in north-continental and Mediterranean regions of Europe, respectively) (Baccini et al. 2008). The variability among studies further supports the importance of regional analysis.

When stratifying all-ages by cause of mortality, we found both relative risk and time series analyses showed statistically significant increases for circulatory and cerebrovascular causes of death. Additionally, the relative risk analysis found a statistically significant increase in mortality risk from accidents, while the time series analysis found a significant increase in risk associated with ischemic and respiratory causes.

When stratifying cause-of-death categories by age group in the relative risk analysis, we found that heat's overall effect does not impact the elderly (85+ age group) exclusively. Statistically significant results were found in the 0-4 (nephritis and nephrotic), 45-64 (diabetes, and natural heat exposure), 65-84 (all causes, non-traumatic, cerebrovascular, mental health, and accident) and 85+ (all causes, non-traumatic, circulatory, cardiovascular, and cerebrovascular) age groups. However, when examining heat's intensity effect using the time series analysis, we found that, with two exceptions (45-64 diabetes and 65-84 homicide), heat's intensity almost exclusively affects the 85+ age group (all-causes, non-traumatic, circulatory, cardiovascular, ischemic, cerebrovascular, and suicide). Busch Isaksen et al. (2014) found similar statistically significant age-stratified results for the 85+ age group when comparing heat's intensity effect on circulatory (4.8% (95% CI: 2.2, 7.46)) and cardiovascular (4.22% (95% CI: 1.11, 7.43)) causes of death in King County.

Results from both analyses in this study suggest that a vulnerable population of younger diabetic patients exists. For the 45-64 age group we found that the relative risk of death from diabetes on a heat day was 78% greater than on a non-heat day, with risk increasing 14.22% for each degree increase in humidex above 36.0 °C. In comparison, Shuman (1972) found a 117% increase in diabetic-related mortality during a New York heat wave, while Basagaña et al. (2011) observed a 20% increase in mortality risk for diabetes on extreme heat days in the Catalonia

region of Spain. Schwartz (2005) found that the relative odds of death, on a 99<sup>th</sup> percentile day, increased 17% for those persons previously hospitalized for diabetes. One possible explanation for the increased diabetic mortality risk is suggested by Akanji and Oputa's (1991) study findings that both non-diabetic and diabetic participants' plasma glucose levels significantly increased with a 10 °C increase in ambient temperature. Given that an estimated 8% of 45-64 year-olds and 15% of 65+ year-olds living in King County have diabetes, our findings are an important consideration for outreach and prevention programs (Public Health - Seattle & King County 2013).

This study also highlights the vulnerability of older age groups to circulatory deaths and its subcategories, cardiovascular and cerebrovascular causes. Similarly, Jackson et al. (2010) found in the Greater Seattle area that the highest elevated risk for circulatory mortality on a heat day belonged to the 65+ (30%) and 85+ (50%) age groups. Busch Isaksen et al. found that, in King County, the 85+ age group's risk for circulatory mortality increased 4.8% for each degree above 35.7 °C humidex. Studies from other geographic locations have also shown an increased risk in heat-related mortality for the elderly (Basu, Feng and Ostro 2008; Ishigami et al. 2008). Age-related vulnerability has been attributed to changes in the body's natural homeostatic mechanisms, greater likelihood of taking medications that inhibit the body's thermoregulation, being homebound, living alone, and not being able to care for one's self (Brown and Walker 2008; Kovats and Hajat 2008; Naughton et al. 2002).

Aside from age, this study did not find that the individual covariates investigated modified the effect of heat on mortality. However, other studies have found that individuals of Black race or non-White race (Basu 2009; O'Neill, Zanobetti and Schwartz 2003; O'Neill Zanobetti and Schwartz 2005) those with less education, and those who are socially isolated are

more vulnerable to heat-related death (Medina-Ramon et al. 2006; O'Neill Zanobetti and Schwartz 2003; Naughton 2002). It is possible that other individual characteristics not collected through death certificate data are more relevant predictors of vulnerability to heat-related mortality than are the characteristics we were able to consider. In addition, neighborhood or area-level characteristics such as community poverty, available green space, prevalence of air conditioning, and urban heat islands would be worth investigation (O'Neill Zanobetti and Schwartz 2005; Stone, Hess and Frumkin 2010; Tan et al. 2007).

This study found that the same-day humidex exposure had the strongest association with mortality, and that there was no evidence of a lag or cooling effect. These findings are similar to those of other studies in which either the same-day apparent temperature (Gasparrini and Armstrong 2011; Anderson and Bell 2009; Zanobetti and Schwartz 2008) or recent lags of 1-3 days were found to be most relevant to the heat-mortality relationship (Basagaña et al. 2011; Basu, Feng and Ostro 2008; Curriero et al. 2002). We also found that days classified as having either a moist or dry tropical synoptic weather pattern were associated with a significant increased risk of mortality than days that were not classified as moist or dry tropical. Our research supports Kalkstein and Green (1997) and Kalkstein et al. (2011) findings of increased mortality on days with oppressively hot air masses.

Limitations of this study include possible exposure misclassification and inappropriate geographical boundary selection. Our study uses an average daily county-wide maximum humidex value to estimate heat exposure, which may result in exposure misclassification when a disproportionate number of cases are below or above the average value. Improved exposure assessment could be obtained by using a population-weighted temperature value, or by assigning each case a maximum humidex value from the closest meteorological grid center point. Data

regarding access to air conditioning and behavioral/lifestyle choices would further refine heat exposure. Personal monitoring of time-activity patterns from a representative sample of vulnerable populations could help clarify these factors, as was done in a previous study of elderly individuals (Basu and Samet 2002).

This study used political jurisdictions (county boundary) to assess the heat-mortality relationship. This geographical unit of analysis may not accurately reflect how the effects of heat on mortality vary spatially. An alternative method, and area for future research, would be to combine populations that experience similar climate zones and, therefore, should have similar levels of acclimatization. Combining populations into climate zones may also reduce type II error, by ensuring adequate power, allowing the examination of heat-related health effects in rural areas.

This study did not correct for multiple comparisons. A Type 1 error may occur when many subgroups are analyzed for effect difference; the more comparisons analyzed, the more opportunity there is to identify by chance a result that appears significant, even when no statistically significant difference exists. A multiple testing correction, such as Bonferroni, could be applied to our analyses (Koepsell and Weiss 2003). However, multiple testing corrections have their disadvantages by reducing study power, resulting in false negative results. Instead, we looked at all the statistically significant results and examined them for expected dose-response patterns, concurrence with existing literature, and influence of small counts. When results were found to depend on a small number of outcomes ( $N < 20$ ), we flagged these results in the data presentation and discussion.

This study did not adjust for air pollution. Studies suggest that air pollution may confound the association between heat and mortality and that it is a phenomenon that may vary

geographically (Daniela et al. 2010; Anderson and Bell 2009; Medina-Ramon and Schwartz 2007; Hajat et al. 2005; O'Neill, Zanobetti and Schwartz 2005; O'Neill, Zanobetti and Schwartz 2003). Anderson and Bell (2009), Basu, Feng and Ostro (2008) and Zanobetti and Schwartz (2008) have all reported that the association between heat and mortality persists even after controlling for ozone and particulate matter. We contend that it is unnecessary to control for air pollution because it does not meet the definition of a confounder. A confounder is a characteristic that must not only be related to both the exposure and the outcome but must also affect the exposure (Koepsell and Weiss 2003). While ambient temperatures may affect the levels of air pollution, air pollution levels do not affect ambient temperatures. Buckley, Samet and Richardson (2014) argue this point through the use of directed acyclic graphs (DAGs). They conclude that greater care should be taken in clarifying causal assumptions prompting model adjustments.

## **2.5 Conclusion**

This study characterized King County, Washington's historic heat-mortality relationship using two different statistical methods. The results demonstrate that heat, expressed as humidex, is associated with increased mortality on heat days, and that the risk increases with heat's intensity. This study has found that when stratifying by age and cause of death, younger age groups are at an increased risk of death for several causes of death, particularly diabetes. Additionally, we have found that heat's intensity almost exclusively affects the 85+ age group. While individual-level characteristics (age being an exception) and other heat effects (cool-down, duration, lag) were not found to affect mortality rates, the synoptic weather mass classification was found to increase mortality rates on a heat day.

Future research is needed to validate the methods used to model our heat-mortality relationship, as our piece-wise linear model fits linear slopes to an otherwise non-linear relationship. Improving heat exposure assessment is another area where additional research would improve the understanding of our region's heat-mortality relationship. Our findings warrant additional investigation into the role heat exposure plays in diabetic patient health and care. Lastly, a better understanding of the full range of effects that air mass type has on mortality would support the use of synoptic air mass classification by the National Weather Service's extreme weather warning system.

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## Chapter 3

### Hospitalizations associated with extreme heat exposure in King County, WA, 1990-2010

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## Chapter 3 abstract

### Background

Increased morbidity and mortality have been associated with extreme heat events, particularly in temperate climates. The frequency and intensity of these events are projected to increase in the future due to climate change. Understanding the historical heat-health relationship on a regional scale is an important first step to managing the impact on health outcomes. Few epidemiologic studies have considered the impact of extreme heat events on hospitalization rates in the Pacific Northwest region.

### Methods

This study quantifies the historical (May to September 1990-2010) heat-morbidity relationship in King County, Washington using both relative risk and time series analyses. Data were obtained from the Comprehensive Hospital Abstract Reporting System (CHARS) (hospitalization admissions) and the University of Washington Climate Impacts group (temperature and relative humidity). A relative risk (RR) analysis was used to explore the association between heat and all non-traumatic hospitalizations on 99<sup>th</sup> percentile heat days. Additionally, a time series analysis using a piece-wise linear model fit was conducted to estimate the effect that heat's intensity has on admissions, adjusted for temporal trends and day of the week. Risk of hospitalization was estimated by age group and specific unplanned, non-traumatic causes of admission for both analyses.

### Results

We found for all-ages, all non-traumatic causes, a non-statistically significant 2% [95% CI: 1.02 (0.98, 1.05)] increase in risk of hospitalization on a heat day versus a non-heat day in

King County, Washington. When considering the effect that heat intensity has on hospitalizations, we found a statistically significant 1.59% (95% CI: 0.9%, 2.29%) increased risk of admission per degree increase in average county-wide daily maximum humidex above 37.4 °C. Admission stratified by cause and age produced statistically significant results with both relative risk and time series analyses for nephritis and nephrotic syndromes, acute renal failure and natural heat exposure admissions. Additional, unique age-adjusted results were observed for each type of analysis: an increased risk of admission from mental health causes, using the relative risk analysis; and an increased risk of admission from circulatory, cardiovascular, respiratory, COPD, and asthma causes, using the time series analysis. Individual-level characteristics (age being an exception) or other heat effects, such as cool-down, duration, lag, or synoptic weather mass classification, were not found to alter hospitalization risk.

## **Conclusions**

Our study demonstrates that heat, expressed as humidex, is associated with increased hospitalizations on heat days, and that the risk increases with heat's intensity. When stratified by age and cause of admission, younger age groups are at a significant risk for the sub-categories of unplanned, non-traumatic hospitalizations: mental health, nephritis and nephrotic syndromes, acute renal failure, natural heat exposure, COPD and asthma.

## **3.1 Introduction**

The health-related impacts associated with extreme heat events are of growing concern given predicted increases in both frequency and duration of these events as a result of climate change (IPCC 2013). Numerous studies indicate that higher temperatures, or other indices of the physiological effect of heat and humidity, are associated with increased mortality (Whitman et al. 1997; Naughton 2002; Baccini et al. 2008; Basu, Feng and Ostro 2008; Anderson and Bell

2009; Jackson et al. 2010; Anderson and Bell 2011). While not as extensively studied as mortality, research focused on morbidity has also found an increased risk of hospitalization and emergency room visits associated with increasing temperatures (Semenza 1999; and Semenza et al. 1999; Mastrangelo et al. 2007; Knowlton et al. 2008; Kovats and Hajat 2008). Studies conducted in the United States have shown an increased risk of hospitalization for diverse conditions such as heat stroke or heat exhaustion (Knowlton et al. 2009), acute renal failure (Fletcher et al. 2012), diabetes (Ostro et al. 2010), respiratory (Lin et al. 2009), and cardiovascular diseases (Koken et al. 2003; Green et al. 2010).

Intensity and duration of heat events have been found to modify heat's effect on mortality (Baccini et al. 2008; Anderson and Bell 2010) and morbidity (Fletcher et al. 2012).

Demographic factors found to influence mortality and morbidity risks include social isolation (Kaiser et al. 2001; Naughton 2002), socio-economic status (Jones et al. 1982; Kaiser et al. 2001), and ethnicity and educational status (O'Neill, Zanobetti, and Schwartz 2003; Fletcher et al. 2012). Additionally, access to air conditioning has been found to decrease mortality (Naughton 2002; O'Neill, Zanobetti, and Schwartz 2005) and morbidity (Ostro et al. 2010) risks. It is important to note the significant variability in study results that suggests that *place* matters. The heat-health relationship is affected by area-specific characteristics, such as underlying disease burden, vulnerable population demographics, and public health infrastructure (Kinney et al. 2008). As average temperatures and the frequency of extreme heat events are predicted to increase with climate change, understanding the regional heat-morbidity relationship becomes increasingly important for directing adaptation-related policy decisions.

This study investigated the relationship between heat and morbidity in the 14<sup>th</sup> most populous county in the U.S., King County, Washington (King County 2012). The first analysis

used a Poisson, relative risk (RR) model to explore age-adjusted causes of hospitalization. Specific causes were selected *a priori* through literature or by request from the local health jurisdiction (non-traumatic, respiratory, COPD, Asthma, circulatory, cardiovascular, ischemic, cerebrovascular, diabetes, heat-related illness, mental health, and chronic and acute renal failure.) We also conducted a time series analysis to study the effect of heat's intensity on morbidity. Here, we quantified the percentage increase in hospital admissions associated with one degree increase in humidex for the same list of causes of admission, age adjusted. A time series analysis allowed us to investigate potential effect modification from individual-level characteristics. It also provided the opportunity to test the following additional effects from heat on hospitalization: cool down, duration, lag, and synoptic weather mass classification. Lastly, we were able to examine length-of-hospital stay and admission costs.

## **3.2 Methods**

### **3.2.1 Hospital admissions and population data**

King County hospitalization discharge data for all non-traumatic illnesses and external injury due to heat causes, 1990 to 2010, were obtained from the Comprehensive Hospital Abstract Reporting System (CHARS) maintained by the Washington State Department of Health. Only admissions during the warmer summer months of May through September were analyzed. Colder months were excluded to minimize potential confounding by infectious diseases typically seen during these months. There are 153 days per constrained calendar year, a total of 3,213 days for the entire study period. In addition, this study only included those visits categorized as “emergency” (requiring immediate medical intervention) and “urgent” (requiring immediate attention), by the Washington Department of Health (Washington State Department of Health, Office of Hospital and Patient Data Systems April 2, 2010). “Elective”

hospitalizations were excluded from this analysis. A total of 1,384,251 non-traumatic hospital admissions occurred during the warmer months from 1990 to 2010; 54% were unplanned. Admissions were coded using the International Classification of Diseases, Clinical Modification (ICD-9-CM) codes.

In addition to unplanned, non-traumatic hospitalizations (ICD-9 001-799), we investigated the relationship between heat and the following disease conditions analyzed in Chapter 2: diabetes (ICD-9 250), circulatory (ICD-9 390-459), cardiovascular (ICD-9 393-429), ischemic (ICD-9 410-414), cerebrovascular (ICD-9 430-438), respiratory (ICD-9 460-519), COPD (ICD-9 490-496), asthma (ICD-9 493), nephritis and nephrotic (ICD-9 580-589), acute renal failure (ICD-9 584), mental health (ICD-9 290-316) and natural heat exposure, including dehydration (ICD-9 992, E900.0, E900.9, 276.51). Table 3.1 lists the specific ICD-9 codes used in this study.

*A priori* we anticipated that several individual-level characteristics may identify populations vulnerable to heat-related hospitalizations: age, gender, admission source (emergency room referral or non-ER referral), admission type (emergency or urgent) and socioeconomic status (probable low SES, defined as cases where primary payer indicated Medicaid or Charity care, all-ages, or non-low SES). Population data, by age groups (0-4, 5-14, 15-44, 45-64, 65-84, 85+) were obtained from the Washington State Office of Financial Management (OFM) (Washington State Office of Financial Management 2012).

**Table 3.1** Cause of hospital admissions analyzed and associated admission ICD-9-CM codes

<i>Category</i>	<i>ICD-9 Code</i>
<b>Non-traumatic</b>	001-799
<b>Select Non-Traumatic Causes</b>	
<b>Diabetes</b>	250
<b>Circulatory</b>	390-459
<b>Cardiovascular</b>	393-429
<b>Ischemic</b>	410-414
<b>Cerebrovascular</b>	430-438
<b>Respiratory</b>	460-519
<b>COPD and allied conditions</b>	490-496
<b>Asthma</b>	493
<b>Nephritis and Nephrotic<sup>11</sup></b>	580-589
<b>Acute Renal Failure</b>	584
<b>Mental Health</b>	290-316
<b>Select Traumatic Cause</b>	
<b>Natural Heat Exposure w/Dehydration<sup>12</sup></b>	E900.0, E900.9, 992, & 276.51

<sup>11</sup> Nephritis and Nephrotic syndromes and acute renal failure have been found to be a significant cause of hospital admissions associated with extreme heat events. (Mastrangelo et. al. 2007, Hansen et al. 2008, Knowlton et al. 2008, Reid et al. 2012, Lin et al. 2012)

<sup>12</sup> While morbidity attributed to natural heat-related exposure is classified as a traumatic external injury, we have included the non-traumatic dehydration code ICD-9 276.51 with this sub-grouping.

### **3.2.2 Meteorology data**

This study used a historical (1915-2012) 1/16° resolution gridded meteorological data set produced by the University of Washington's Climate Impacts Group (Maurer et al. 2002). The climatologic foundation of this data set is the Parameter-Elevation Relationships on Independent Slopes Model (PRISM), developed by Oregon State University (PRISM Climate Group). PRISM is considered to represent the most current knowledge on spatial climatic patterns for the United States (Daly et al. 2007). Daily temperature and relative humidity values for our meteorological data set were constructed using PRISM's regional spatial climatic patterns and weather station observations from the Global Historical Climate Network-Daily (GHCN). GHCN is a National Oceanic and Atmospheric Administration database of daily meteorological measurements from land surface stations across the globe (NOAA 2009). The resulting meteorology data used for this study contain daily max/min temperature, precipitation and relative humidity values for each meteorological center point. The county-wide daily maximum temperature and average relative humidity values were used to construct our exposure metric, humidex.

### **3.2.3 Exposure assessment**

As with our previous heat-mortality studies (Jackson et al. 2010; Busch Isaksen et al. 2014; Chapter 2), we used humidex as the measure of exposure. Humidex is an apparent temperature or "feels-like" index that measures the combined effects of temperature and humidity on the human body (Masterton and Richardson 1979). An average daily maximum humidex was computed over all meteorological center points located in King County. Exposure to heat was then estimated as the average maximum county-wide humidex value for each day within the study's time frame.

Humidex is defined by the following formula and is expressed in units of °C:

$$\begin{aligned} \mathbf{Humidex} &= \mathbf{T} + \left(\frac{\mathbf{5}}{\mathbf{9}}\right) \cdot (\mathbf{v} - \mathbf{10}), \text{ where} \\ \mathbf{v} &= \left( \mathbf{6.112} \cdot \mathbf{10}^{\left[\frac{\mathbf{7.5T}}{\mathbf{237.7+T}}\right]} \right) \cdot \frac{\mathbf{H}}{\mathbf{100}} \end{aligned} \tag{1}$$

where  $T$  is the air temperature (degrees Celsius),  $H$  is the average relative humidity (%), and  $v$  is the vapor pressure (kPa) (Canadian Centre for Occupational Health and Safety 2011).

### 3.2.4 Association between humidex and morbidity

#### *Relative risk analysis*

A heat day was defined as a day in which the average King County-wide daily maximum humidex exceeded a specified threshold. In previous mortality studies, the 99<sup>th</sup> percentile has been used as the Greater Seattle area-wide (Jackson et al. 2010) and King County-wide (Chapter 2, Busch Isaksen et al. 2014) maximum humidex threshold, defining a heat day. In this analysis, we tried the 99<sup>th</sup>, 95<sup>th</sup> and 90<sup>th</sup> percentiles for King County and chose the one that gave the best fit to the data (with a maximum likelihood or a minimum deviance). The following Poisson regression model was used:

$$E(Y_j) = P_j \lambda_j + \beta_1 I\{\mathbf{humidex}_j > \mathbf{threshold}\} \tag{2}$$

where  $Y_j$  is the hospital admission count on day  $j$ ,  $P_j$  is the population,  $\lambda_j$  is the morbidity incidence rate and  $\beta_1 I\{\mathbf{humidex}_j > \mathbf{threshold}\}$  is the indicator of a heat day. This approach modeled the expected admission count after controlling for population growth.

### *Time series analysis*

For our time series analysis, we used a Poisson regression with nonparametric splines to model the log-admission rate over time and humidex. Specifically, we assumed that:

$$Y_j \sim \text{Poisson}(P_j \lambda_j), \text{ with}$$

$$\log \frac{\mu_j}{\text{popu}} = \beta_0 + s(\mathbf{h}_j) + s(\mathbf{t}_j) + \sum_{l=1}^7 \beta_l I_{\{\text{dayofweek}_j=l\}} + \sum_{l=6}^9 \beta_l I_{\{\text{month}_j=l\}} \quad (3)$$

where  $\mu_j$  is the observed hospitalization count on day  $j$ ,  $s(\mathbf{h}_j)$  is a penalized regression spline modeling the effects of humidex,  $s(\mathbf{t}_j)$  is a penalized regression spline modeling the overall trend of admissions over 21 years, and  $(\beta_l$ 's) are the adjustments for day of the week and seasonal monthly effects.

As with our heat-mortality relationship we increased interpretability and usefulness for public health practitioners and policymakers by simplifying the nonparametric spline model with a piece-wise linear model. However, unlike our mortality analysis, we used a penalized regression spline to model heat effect on morbidity under the model-derived threshold and a linear piece to summarize heat's effect on morbidity beyond the threshold. The maximum likelihood for the optimal humidex threshold was identified by exploring 0.1 degree incremental changes starting at 20 °C and continuing through 44 °C humidex. The following represents the final model:

$$\log \frac{\mu_j}{\text{popu}} = \beta_0 + s(\hat{h}_0 - h_j) + \beta_1 (h_j - \hat{h}_0)_+ + s(\mathbf{t}_j) + \sum_{l=1}^7 \beta_l I_{\{\text{dayofweek}_j=l\}} + \sum_{l=6}^9 \beta_l I_{\{\text{month}_j=l\}} \quad (4)$$

where  $h_j$  is the county-wide average daily maximum humidex value on day  $j$ ,  $\hat{h}_0$  is the optimal alert threshold,  $s(\hat{h}_0 - h_j)$  is the penalized regression spline below the optimal threshold,  $\beta_1$  is the estimated heat effect on mortality for each degree C above  $\hat{h}_0$ ,  $s(t_j)$  is a penalized regression spline modeling the overall trend of hospitalizations over 21 years, and  $(\beta_l's)$  are adjustments for day of the week and seasonal effects. A heat day was then defined as a day in which the average county-wide daily maximum humidex exceeded the optimal alert threshold. The impact of humidex intensity on admissions was assessed by the slope of the line above the threshold ( $\beta_1$ ). The “mgcv” and “GAM” packages were used with the statistical software R version 2.14.1 to determine the model’s degrees of freedom and to tune the threshold, respectively (R Core Team 2012). Similar methods were used to study heat and hospitalizations in New York City (Lin et al. 2009; Lin et al. 2012).

### **3.2.5 Effect modification with individual-level characteristics**

Individual-level characteristic data, obtained from CHARS, were evaluated for differences in hospitalization risk. The analyzed covariates included age, gender, admission source (emergency room or other), admission type (emergency or urgent) and socio-economic status (probable low SES, defined as cases where primary payer indicated Medicaid or Charity care for all-ages, or non-low SES). Effect modification was examined by adding each covariate into the model along with an interaction term. Hospitalization rates for all covariate groups, except age, were not adjusted by population size of the covariate group because the data were not available.

### 3.2.6 Other heat effects on morbidity

While not fully analyzed with respect to morbidity, studies have suggested that cooler nighttime temperatures help minimize the effect of heat on mortality (Schwartz 2005) and that lengthier heat events increase mortality risk (Daniela et al. 2010; Anderson and Bell 2011). Furthermore, the type of synoptic weather pattern has been shown to influence mortality (Kalkstein and Green 1996; Sheridan, Kalkstein and Kalkstein 2009) and morbidity (Morabito et al. 2006) rates on heat days.

This study evaluated the data to see if there was a “cool down effect,” wherein warmer evenings (or an elevated minimum humidex) would contribute to an increase in hospitalizations beyond the effect of heat during the day. For data on heat days, cool down effect is explored using the following two models:

$$\log \frac{\mu_j}{popu} = \beta_0 + \beta_2 \mathbf{difference} \quad (5)$$

$$\log \frac{\mu_j}{popu} = \beta_0 + \beta_1 \mathbf{aboveThres} + \beta_2 \mathbf{difference} \quad (6)$$

where *difference* is defined as the daily maximum humidex - daily minimum humidex for a given heat day, and *aboveThres* is defined as the daily maximum humidex - threshold.

Similarly, this study examined the relationship between admission count and heat event duration (number of consecutive heat days). For data on heat days, a duration effect is explored using the following two models:

$$\log \frac{\mu_j}{popu} = \beta_0 + \beta_2 \mathbf{duration} \quad (7)$$

$$\log \frac{\mu_j}{popu} = \beta_0 + \beta_1 \mathbf{aboveThres} + \beta_2 \mathbf{duration} \quad (8)$$

where *duration* is defined as the day's order in a given heat event.

This study also explored whether there was a lag effect between humidex and hospitalizations over several days. Following the methods described in Armstrong (2006) we explored distributed lag effects using the following model:

$$E(Y_j) = \exp\{\alpha + f(\mathbf{h}_{\sim j}) + \text{covariates} + s(\text{time})\},$$

$$f(\mathbf{h}_{\sim j}) = \sum_{l=0}^L \beta_l h_{j-l}, \quad (9)$$

where  $Y_j$  is the hospital admission count on day  $j$ ,  $s(\text{time})$  is a spline curve over time, and  $f(\mathbf{h}_{\sim j})$  is a function of historic county-wide average maximum humidex values on days up to day  $j$ , which takes a weighted effect of humidex on day  $j$  and the previous  $L$  days. By assuming different constraints or temporal structures for  $\beta_l, l = 0, \dots, L$ , we examined the evidence of lag effects. This was implemented using the “dlnm” (distributed lag non-linear model) package in R version 2.14.1 (Gasparrini 2011).

Finally, we investigated whether hospitalization rates were influenced by the type of synoptic weather mass on a given heat day. Previous research indicates that moist and dry tropical air masses are associated with increased mortality, but have not been analyzed for effects on morbidity (Sheridan, Kalkstein and Kalkstein 2009; Kalkstein et al. 2011). Using daily spatial synoptic weather mass classification data for the Seattle/Tacoma station (Sheridan 2013), effect modification was explored by applying the same method used for individual-level characteristics.

### **3.3 Results**

#### **3.3.1 King County population and climate**

King is the largest county in Washington State, the 14<sup>th</sup> largest county in the United States, and accounted for approximately 30% of the state's population throughout the study's time frame (Washington State Office of Financial Management 2012; King County 2013). From 1990 to 2010 the county's population increased 28%, with age groups 45-64, 65-84, and 85+ increasing 88%, 19%, 101%, respectively. King County is located in Western Washington and is characterized by relatively cool summers. Its summer daily average humidex values range from a minimum of 6.90 °C (44.4 °F) to a maximum of 22.6 °C (72.7 °F). From 1990 to 2010, King County experienced 752,151 unplanned hospitalizations during the warmer months of May-September, 54% of the overall, non-traumatic admission count. On average, there were 234 admissions per day and the length of stay averaged approximately 5 days (Table 3.2). Over the 21-year study time frame, total hospital charges in King County have grown exponentially. Over 1990-2010, the average cost per day of stay was \$3,388.50, while the average total cost for all of King County was approximately 4 million dollars per day. Table 3.2 reports population by age and percent change, meteorological ranges, admission costs, and admission counts for individual level characteristics, 1990-2010.

#### **3.3.2 Association between humidex and morbidity**

The plotted relationship between summertime daily admission count and humidex was u-shaped, illustrating an increase in hospitalizations at extreme humidex values (Figure 3.1).

**Table 3.2** King County, Washington population and climate demographics, 1990–2010

	<i>King County</i>
<b>1990 Population<sup>1</sup></b>	Complete Count
WA State Total	4,866,692
King County Total	1,507,319
% of State Population	30.9%
0-4	106,999
5-14	185,933
15-44	772,361
45-64	276,070
65-84	149,170
85+	16,786
<b>2010 Population<sup>2</sup></b>	Complete Count (% Change)
WA State Total	6,724,540 (38.2 %)
King County Total	1,931,249 (28.1 %)
% of State Population	28.7% (-7.0 %)
0-4	120,294 (12.4 %)
5-14	224,084 (20.5 %)
15-44	856,843 (10.9 %)
45-64	519,349 (88.1 %)
65-84	176,895 (18.6 %)
85+	33,784 (101 %)
<b>County-Wide Humidex °C(°F) (May-Sept.)</b>	
Min. daily humidex, av. over time	6.90 °C (44.4 °F)
Max. daily humidex, av. over time	22.6 °C (72.7 °F)
<b>Heat Event Days (May-Sept.)</b>	
<b>Heat Days above Relative Risk Threshold</b> (99 <sup>th</sup> percentile 36.2 °C (97.2 °F))	77 / 3213 total days (2.4 %)
<b>Average County-Wide Max. on Heat Days</b>	
Humidex	38.7 °C (101.7 °F)
Temperature	30.8 °C (87.4 °F)
<b>Minimum County-Wide Max. on Heat Days</b>	

<sup>1</sup> Source: Washington State Department of Health

<sup>2</sup> Source: Washington State Office of Financial Management <http://www.ofm.wa.gov/pop/census2010/data.asp>

Humidex	36.2 °C (97.2 °F)
Temperature	28.0 °C (82.5 °F)
<b>Maximum County-Wide Max. on Heat Days</b>	
Humidex	44.7 °C (112.5 °F)
Temperature	34.6 °C (94.3 °F)
<b>Heat Days above Time Series Threshold</b> (37.4 °C (99.3 °F))	50 days /3213 total days (1.6 %)
<b>Average County-Wide Max. on Heat Days</b>	
Humidex	39.7 °C (103.5 °F)
Temperature	31.4 °C ( 88.5 °F)
<b>Minimum County-Wide Max. on Heat Days</b>	
Humidex	37.4 °C (99.3 °F)
Temperature	29.7 °C (85.5 °F)
<b>Maximum County-Wide Max. on Heat Days</b>	
Humidex	44.7 °C (112.5 °F)
Temperature	34.6 °C (94.3 °F)
<b>King County Hospital Admissions</b>	
<b>May - September 1990-2010</b>	
Total non-traumatic admissions <sup>3</sup>	1,384,251
Total unplanned admissions <sup>4</sup> (% of total)	752,151 (54%)
Average daily admission rate	234 (1990=219; 2010=250)
Average length of admission stay	5.01 days (1990=6.2; 2010=5.0)
Average charge/day of stay	\$3,388.50 (1990= \$1,072; 2010=\$7,328)
Total hospital charges/day	\$3.97 million (2009= \$1.5m; 2010=\$9.2 m)
<b>Individual Level Characteristics</b>	
<b>Gender</b> (% of total)	
Male	419,861 (55.8%)
Female	332,285 (44.2%)
<b>Admission Source</b> (% of total)	
Emergency room	476,698 (63.4%)
Other	275,453 (36.6%)

<sup>3</sup> This analysis only considered non-traumatic causes of admissions and one traumatic cause of admission, natural heat exposure.

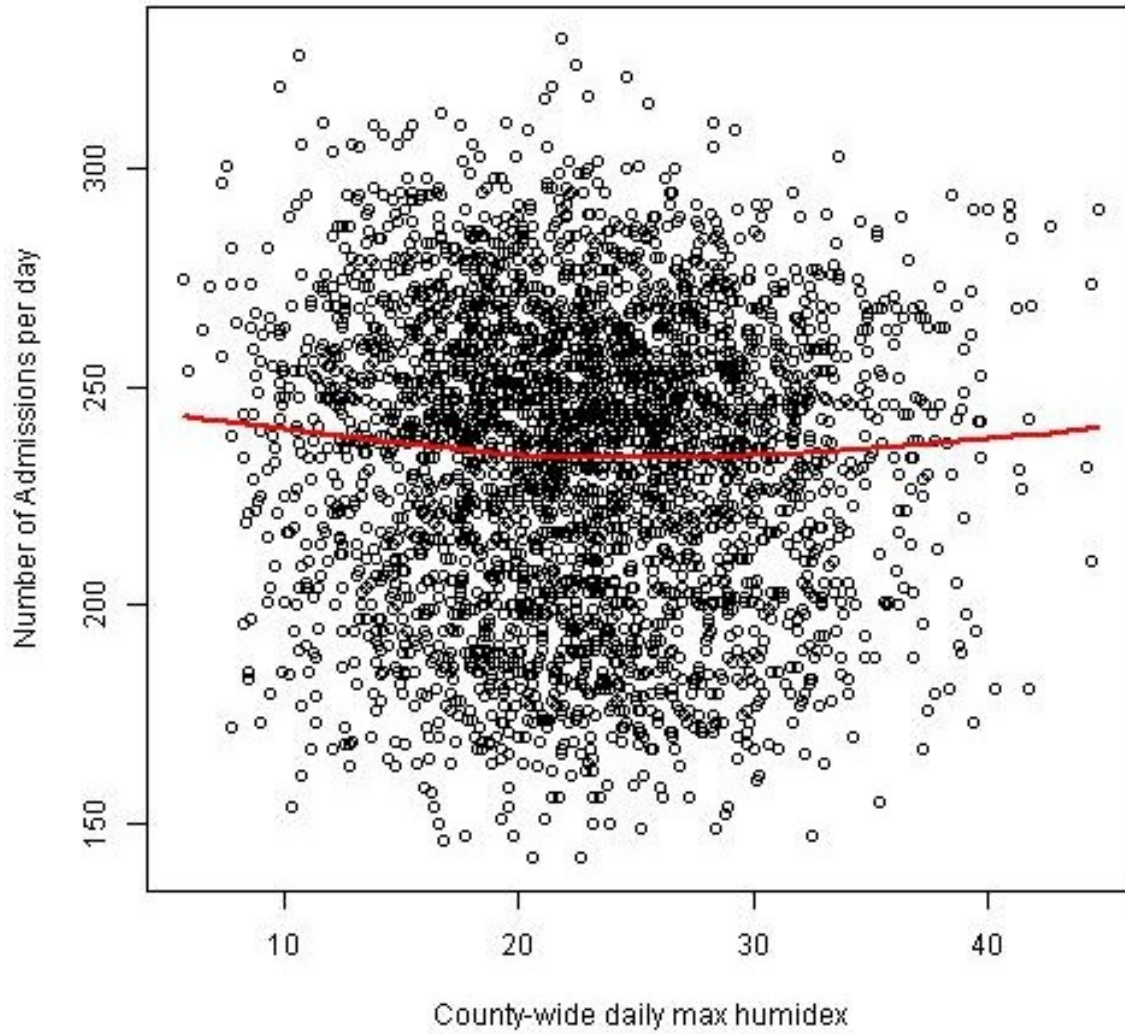
<sup>4</sup> This analysis only considered unplanned admissions, May-September

<b>Socio Economic Status</b> (% of total)	
Probable low SES <sup>5</sup>	121,844 (16.2%)
Non-low SES	630,307 (83.8%)
<b>Admission Type</b> (% of total)	
Emergency	486,195 (64.6%)
Urgent	265,956 (35.4%)

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<sup>5</sup> Probable Low SES was defined as cases where primary payer indicated Medicaid (all ages,) or Charity care (all ages.)

**Figure 3.1** Lowess non-parametric regression curve modeling county-wide maximum humidex effect over daily hospital admission counts, 1990-2010



### ***Relative risk analysis***

A heat day for the relative risk analysis was defined by Equation (2) as a day that exceeded the 99<sup>th</sup> percentile of all days January-December (36.2 °C (97.2 °F) humidex). During 1990-2010, King County experienced 77 days over the 99<sup>th</sup> percentile, 2.4% of all May-September days. The average humidex on heat days was 38.7 °C (101.7 °F) (Table 3.2). Relative risk estimates and 95% confidence intervals for all age groups and categories of admissions are reported in Table 3.3. We found that for all-ages, all non-traumatic causes of hospitalizations, the relative risk of admission on a heat day was not statistically significantly greater, 2% [95% CI: 1.02 (0.98, 1.05)], than on a non-heat day. While the all-age, all non-traumatic results were statistically non-significant, when examining subcategories of admission, significant all-age results were found for: nephritis and nephrotic syndromes 57% [95% CI: 1.57 (1.35, 1.83)], acute renal failure 68% [95% CI: 1.68 (1.41,2.01)], and natural heat exposure (including dehydration) 244% [3.44 (95% CI: 2.56, 4.64)].

When investigating hospitalizations stratified by cause and age, significant increases in risk were found for the: 0-4 age group, mental health 318% [4.18 (95% CI: 1.29, 3.57)]; the 15-44 age group, natural heat exposure (including dehydration) 399% [4.99 (95% CI: 2.89, 8.6)]; the 45-64 age group, nephritis and nephrotic syndromes 76% [1.76 (95% CI: 1.42, 2.18)], acute renal failure 99% [1.99 (95% CI: 1.58, 2.5)], and natural heat exposure (including dehydration) 142% [2.42 (95% CI: 1.45, 4.06)]; the 65-84 age group, nephritis and nephrotic syndromes 60% [1.6 (95% CI: 1.3, 1.97)], acute renal failure 67% [1.67 (95% CI: 1.34, 2.07)], and natural heat exposure (including dehydration) 242% [3.42 (95% CI: 2.3, 5.08)]; and the 85+ age group, all non-traumatic 8% [1.08 (95% CI: 1.03, 1.14)], nephritis and nephrotic syndromes 49% [1.49 (95% CI: 1.12, 1.99)], acute renal failure 55% [1.55 (95% CI: 1.16, 2.07)] and natural heat

exposure (including dehydration) 343% [4.43 (95% CI: 2.99, 6.56)]. It should be noted that the 0-4 age group's mental health estimate, and both the 15-44 and 45-64 age groups' natural heat exposure estimates are based on a small number of cases, as reported in Table 3.3.

### ***Time series analysis***

In King County, the time series relationship between humidex and log-admission rates is J-shaped. Figure 3.2 illustrates a penalized cubic regression spline modeling effects of humidex over morbidity (Equation 3). The relationship illustrates an increased risk of morbidity from exposure to humidex exceeding approximately 33 °C humidex. An optimal heat day threshold for the time series analysis was determined to be (Equation 4) 1.2 °C above the 99<sup>th</sup> percentile at 37.4 °C (99.3 °F) (Figure 3.3). During 1990-2010, King County experienced 50 days that exceeded the time series analysis threshold. The average humidex on these exceedance days was 39.7 °C (103.5 °F) (Table 3.2).

Intensity estimates and 95% confidence intervals for all age groups and categories of admission are reported in Table 3.4. For all-ages, all non-traumatic causes, we observed a 1.59% (95% CI: 0.9%, 2.29%) increase in hospitalizations per degree increase in average county-wide daily maximum humidex above 37.4 °C. Significant all-age results were found for respiratory 2.32% (95% CI: 0.4%, 4.29%), nephritis and nephrotic syndromes 6.76% (95% CI: 2.59 %, 11.11 %), acute renal failure 7.6% (95% CI: 3.21%, 12.18%), and natural heat exposure (including dehydration) 17.5% (95% CI: 12.14%, 23.1%). When investigating morbidity stratified by cause and age, significant results were found for the: 15-44 age group, COPD 10.01% (95% CI: 0.02%, 21%) and asthma 11.8% (95% CI: 0.54%, 24.33%); the 45-64 age group, cardiovascular -4.28% (95% CI: -7.44%, -1.01%), nephritis and nephrotic syndromes

**Table 3.3** Relative risk analysis results: Increased risk (95% CI) in morbidity on a 99<sup>th</sup> percentile (36.2 °C) heat day compared to a non-heat day, by age group and cause of admission <sup>1</sup>

	<i>All Ages</i>	<i>0-4</i>	<i>5-14</i>	<i>15-44</i>	<i>45-64</i>	<i>65-84</i>	<i>85+</i>
<b>Non-traumatic</b>	1.02 (0.98, 1.05)	0.98 (0.9, 1.06)	0.97 (0.88, 1.08)	0.99 (0.93, 1.05)	1.03 (0.98, 1.07)	1.01 (0.97, 1.05)	<b>1.08</b> <b>(1.03, 1.14)</b>
<b>Select Non-Traumatic Causes</b>							
<b>Diabetes</b>	1.01 (0.9, 1.14)	0.22 (0.03, 1.65)	1.07 (0.71, 1.63)	0.99 (0.82, 1.19)	1.11 (0.91, 1.35)	0.95 (0.74, 1.23)	0.78 (0.37, 1.66)
<b>Circulatory</b>	0.98 (0.93, 1.02)	0.86 (0.52, 1.44)	1.03 (0.6, 1.79)	0.96 (0.85, 1.08)	0.98 (0.9, 1.06)	0.95 (0.89, 1.01)	1.01 (0.92, 1.1)
<b>Cardiovascular</b>	0.97 (0.91, 1.02)	0.92 (0.47, 1.8)	1.03 (0.48, 2.18)	0.97 (0.84, 1.12)	0.92 (0.84, 1.01)	0.96 (0.89, 1.04)	1.01 (0.91, 1.13)
<b>Ischemic</b>	0.97 (0.89, 1.06)	0 (0, Inf)	0 (0, Inf)	1.02 (0.79, 1.31)	0.93 (0.81, 1.07)	0.97 (0.87, 1.09)	1.01 (0.82, 1.23)
<b>Cerebrovascular</b>	1.03 (0.96, 1.1)	1.35 (0.33, 5.44)	0.87 (0.21, 3.54)	0.96 (0.72, 1.29)	1.14 (1, 1.31)	0.96 (0.87, 1.07)	1.02 (0.86, 1.21)
<b>Respiratory</b>	0.97 (0.91, 1.03)	0.71 (0.58, 0.86)	0.67 (0.49, 0.91)	1.05 (0.93, 1.19)	0.99 (0.9, 1.1)	0.97 (0.89, 1.05)	1.11 (0.97, 1.25)
<b>COPD</b>	0.87 (0.78, 0.97)	0.68 (0.51, 0.9)	0.54 (0.35, 0.85)	1.04 (0.81, 1.32)	0.96 (0.8, 1.15)	0.88 (0.75, 1.03)	1.21 (0.86, 1.69)
<b>Asthma</b>	0.8 (0.68, 0.94)	0.68 (0.51, 0.91)	0.55 (0.35, 0.86)	0.98 (0.75, 1.28)	0.95 (0.7, 1.28)	0.93 (0.65, 1.32)	1.11 (0.53, 2.34)
<b>Nephritis and Nephrotic</b>	<b>1.57</b> <b>(1.35, 1.83)</b>	0.46 (0.06, 3.25)	1.68 (0.69, 4.11)	1.13 (0.78, 1.63)	<b>1.76</b> <b>(1.42, 2.18)</b>	<b>1.6</b> <b>(1.3, 1.97)</b>	<b>1.49</b> <b>(1.12, 1.99)</b>
<b>Acute renal failure</b>	<b>1.68</b> <b>(1.41, 2.01)</b>	1.84 (0.25, 13.38)	0 (0, Inf)	0.99 (0.58, 1.69)	<b>1.99</b> <b>(1.58, 2.5)</b>	<b>1.67</b> <b>(1.34, 2.07)</b>	<b>1.55</b> <b>(1.16, 2.07)</b>
<b>Mental Health</b>	0.93 (0.83, 1.03)	<b>4.18</b> <b>(1.29, 3.57)<sup>2</sup></b>	0.82 (0.6, 1.11)	0.92 (0.82, 1.03)	0.92 (0.8, 1.06)	1.05 (0.86, 1.28)	0.76 (0.48, 1.2)
<b>Select Traumatic Cause</b>							

<sup>1</sup> Bolded relative risk values are significantly greater than 1 ( $p < 0.05$ )

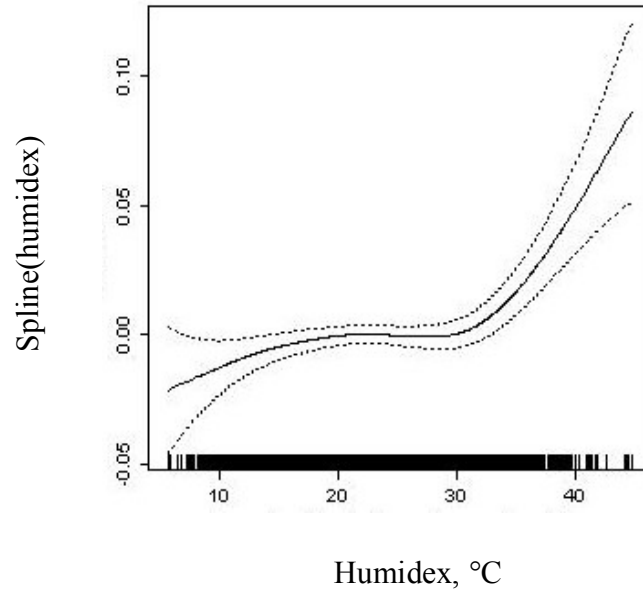
<sup>2</sup> While statistically significant, the estimate is based on a small number of cases [103 cases on non-heat days, 5 cases on a heat day]

	<i>All Ages</i>	<i>0-4</i>	<i>5-14</i>	<i>15-44</i>	<i>45-64</i>	<i>65-84</i>	<i>85+</i>
<b>Natural Heat Exposure w/Dehydration</b>	<b>3.44</b> <b>(2.56,4.64)</b>	1.96 (0.76, 5.03)	2.52 (0.76, 8.32)	<b>4.99</b> <b>(2.89, 8.6)<sup>3</sup></b>	<b>2.42</b> <b>(1.45, 4.06)<sup>4</sup></b>	<b>3.42</b> <b>(2.3, 5.08)</b>	<b>4.43</b> <b>(2.99, 6.56)</b>

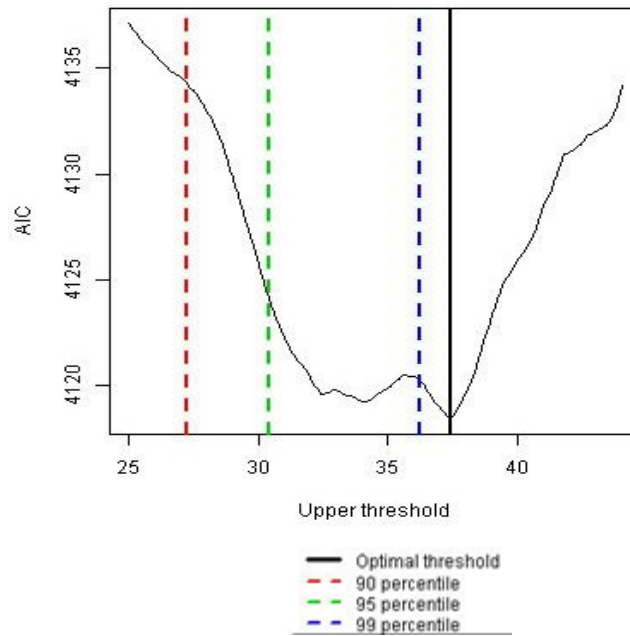
<sup>3</sup> While statistically significant, the estimate is based on a small number of cases [139 cases on non-heat days, 17 cases on a heat day]

<sup>4</sup> While statistically significant, the estimate is based on a small number of cases [264 cases on non-heat days, 16 cases on a heat day]

**Figure 3.2** Penalized cubic regression spline modeling humidex effect over log-hospital admissions



**Figure 3.3** The optimal morbidity threshold for King County identified by the Akaike Information Criterion (AIC), maximum likelihood of the model fit



9.54% (95% CI: 1.77%, 17.91%), and acute renal failure 9.19% (95% CI: 0.45%, 18.68%); the 65-84 age group, all non-traumatic 1.62% (95% CI: 0.28%, 2.97%), nephritis and nephrotic syndromes 8.46% (95% CI: 1.77%, 15.6%), acute renal failure 8.49% (95% CI: 1.16%, 16.34%), and natural heat exposure (including dehydration) 18.31% (95% CI: 10.71%, 26.42%); and the 85+ age group, all non-traumatic 6.28% (95% CI: 4.12%, 8.49%), circulatory 4.8% (95% CI: 1.39%, 8.33%), respiratory 9.96% (95% CI: 5.21%, 14.92%), COPD 17.38% (95% CI: 4.79%, 31.49%), and natural heat exposure (including dehydration) 27.52% (95% CI: 17.76%, 38.08%). It should be noted that the 85+ group's natural heat exposure estimate is based on a small number of cases, as reported in Table 3.4.

### **3.3.3 Effect modification with individual-level characteristics**

We did not find that individual-level characteristics analyzed (gender, admission source, admission type and socio-economic status) altered the risk of admissions on a heat day. However, we did find that age had a statistically significant influence on a person's risk of being admitted to a hospital on a heat day. In both the relative risk and time series analysis we found the 85+ age group to be at greatest risk for all non-traumatic causes of admission.

### **3.3.4 Other heat effects on hospitalization**

We did not find a statistically significant effect on admissions when considering the difference between minimum and maximum humidex (cool-down) on a given heat day. Likewise, we found no effect on admissions from duration of consecutive heat days above threshold. Additionally, we explored the relationship between humidex and hospitalizations spread over several days and found no evidence of a lag effect. Lastly, synoptic weather mass classification was not found to affect hospitalization rates.

**Table 3.4** Time series analysis results: Percentage (95% CI) increase or decrease in morbidity per degree increase in county-wide average daily maximum humidex °C above 37.4 °C, by age group and underlying cause of admission<sup>1</sup>

	<i>All Ages</i>	<i>0-4</i>	<i>5-14</i>	<i>15-44</i>	<i>45-64</i>	<i>65-84</i>	<i>85+</i>
<b>Non-traumatic</b>	<b>1.59 %</b> ( <b>0.9 %</b> , <b>2.29 %</b> )	1.11% (-2.32%, 4.66%)	1.21% (-3.05%, 5.66%)	0.6% (-0.71%, 1.92%)	0.97% (-0.42%, 2.39%)	<b>1.62%</b> ( <b>0.28%</b> , <b>2.97%</b> )	<b>6.28%</b> ( <b>4.12%</b> , <b>8.49%</b> )
<b>Diabetes</b>	-1.23 % ( -5.82 % , 3.58 % )	-21.91% (-64.47%, 71.64%)	-11.87% (-30.31%, 11.45%)	-0.78% (-8.51%, 7.6%)	-2.06% (-9.99%, 6.57%)	2.6% (-6.82%, 12.97%)	-3.02% (-28.59%, 31.71%)
<b>Circulatory</b>	0.14 % ( -1.28 % , 1.59 % )	-1.95% (-22.39%, 23.88%)	15.57% (-3.64%, 38.62%)	-0.55% (-5.54%, 4.69%)	-2.16% (-4.77%, 0.53%)	-0.02% (-2.1%, 2.11%)	<b>4.8%</b> ( <b>1.39%</b> , <b>8.33%</b> )
<b>Cardiovascular</b>	-0.23 % ( -1.97 % , 1.53 % )	-14.62% (-43.01%, 27.92%)	2.18% (-26.83%, 42.68%)	-1.23% (-7.4%, 5.36%)	<b>-4.28%</b> ( <b>-7.44%</b> , <b>-1.01%</b> )	1.06% (-1.4%, 3.59%)	<b>4.44%</b> ( <b>0.18%</b> , <b>8.87%</b> )
<b>Ischemic</b>	1.46 % ( -1.12 % , 4.11 % )	-98.01% (-100%, 3.87 e+86%)	-100% (-100%, 1.19 e+115%)	-1.64% (-13.65%, 12.04%)	-0.62% (-5.2%, 4.18%)	2.28% (-1.83%, 6.56%)	7.13% (-1.32%, 16.3%)
<b>Cerebrovascular</b>	0.6 % ( -2.32 % , 3.62 % )	-19.41% (-73.3%, 143.28%)	2.02% (-37.79%, 67.31%)	2.25% (-9.02%, 14.91%)	2.22% (-3.42%, 8.19%)	-2.22% (-6.53%, 2.3%)	4.44% (-1.74%, 11%)
<b>Respiratory</b>	<b>2.32 %</b> ( <b>0.4 %</b> , <b>4.29 %</b> )	-4.93% (-12.49%, 3.28%)	-2.98% (-13.64%, 9%)	4.67% (-0.53%, 10.14%)	3.54% (-0.46%, 7.7%)	-1.22% (-4.6%, 2.28%)	<b>9.96%</b> ( <b>5.21%</b> , <b>14.92%</b> )
<b>COPD</b>	3.4 % ( -0.48 % , 7.42 % )	-4.41% (-15.21%, 7.76%)	-9.17% (-24.54%, 9.35%)	<b>10.01%</b> ( <b>0.02%</b> , <b>21%</b> )	4.82% (-2.55%, 12.74%)	1.12% (-5.71%, 8.45%)	<b>17.38%</b> ( <b>4.79%</b> , <b>31.49%</b> )
<b>Asthma</b>	5.01 %	-0.96%	-5.55%	<b>11.8%</b>	8.25%	4.58%	19.41%

<sup>1</sup> Bolded time series estimates are significantly greater than 0 ( $p < 0.05$ )

	<i>All Ages</i>	<i>0-4</i>	<i>5-14</i>	<i>15-44</i>	<i>45-64</i>	<i>65-84</i>	<i>85+</i>
	(-0.68 % , 11.02 % )	(-12.04%, 11.51%)	(-21.33%, 13.39%)	<b>(0.54%, 24.33%)</b>	(-3.32%, 21.2%)	(-10.47%, 22.17%)	(-7.05%, 53.39%)
<b>Nephritis and Nephrotic</b>	<b>6.76 %</b> <b>( 2.59 % , 11.11 % )</b>	-25.62% (-82.5%, 216.12%)	-15.61% (-53.53%, 53.27%)	-7.11% (-21.44%, 9.83%)	<b>9.54%</b> <b>(1.77%, 17.91%)</b>	<b>8.46%</b> <b>(1.77%, 15.6%)</b>	7.16% (-2.9%, 18.27%)
<b>Acute renal failure</b>	<b>7.6 %</b> <b>( 3.21 % , 12.18 % )</b>	11.5% (-66.8%, 274.51%)	-100% (-100%, Inf%)	-3.5% (-22.02%, 19.42%)	<b>9.19%</b> <b>(0.45%, 18.68%)</b>	<b>8.49%</b> <b>(1.16%, 16.34%)</b>	7.14% (-3.71%, 19.22%)
<b>Mental Health</b>	1.88 % (-0.54 % , 4.36 % )	17.8% (-31.36%, 102.16%)	-10.95% (-23.6%, 3.81%)	2.23% (-0.88%, 5.44%)	2.39% (-2.27%, 7.27%)	2.36% (-5.52%, 10.89%)	2.32% (-13.07%, 20.43%)
<b>Natural Heat Exposure w/dehydration</b>	<b>17.5 %</b> <b>( 12.14 % , 23.1 % )</b>	17.66% (-4.19%, 44.49%)	21.24% (-1.83%, 49.75%)	9.96% (-3.49%, 25.28%)	4.85% (-7.05%, 18.28%)	<b>18.31%</b> <b>(10.71%, 26.42%)</b>	<b>27.52%</b> <b>(17.76%, 38.08%)<sup>2</sup></b>

<sup>2</sup> While statistically significant, the estimate is based on a small number of cases [230 cases on non-heat days, 18 cases on a heat day]

### 3.4 Discussion

This study characterized King County, Washington's historic heat-morbidity relationship using two different statistical methods. Our relative risk analysis quantified the excess admissions on a heat day compared to a non-heat day, while our time series analysis quantified the intensity effect of heat on admissions for each one degree increase in humidex over the threshold. We further characterized risk by age groups and subcategories of all unplanned, non-traumatic causes of admission. This study explored cool-down, duration, lag, and synoptic weather pattern effects on admissions, and it quantified effect modification from available individual-level characteristics. The results demonstrate that heat, expressed as humidex, is associated with increased hospital admissions on heat days, and that the risk increases with heat's intensity.

Our study is unique, as it provides the opportunity to compare two ways of defining a heat day: a relative threshold calculated from a fixed percentile, and an absolute threshold estimated using a fitted model. We believe the results provide a more complete picture of regional heat effects. The relative risk analysis captures heat's overall contribution to excess morbidity on heat days, while the time series analysis allows for a nuanced understanding of the effect of heat on hospitalization and the role of potential effect modifiers.

This study did not find a statistically significant increase in all-age, all non-traumatic hospitalizations for both analyses. The relative risk of hospitalization on a heat day was 2% (statistically non-significant) greater than on a non-heat day, with risk increasing 1.59% (statistically significant) for each degree increase in humidex above 37.4 °C. Similarly, Knowlton et al. (2008) found a non-significant 1% increase in the relative risk of California hospitalizations on a heat wave day, compared to a non-heat wave day. When investigating a

linear increase in morbidity above an absolute threshold, Linares and Diaz (2008) found a 4.6% increase in unplanned, hospital admissions for each degree increase in maximum daily temperature above 36.6 °C in Madrid, Spain.

When we stratified all ages by cause of hospital admission, both relative risk and time series analyses showed statistically significant increases in risk for nephritis and nephrotic syndromes (57% increase in risk on a heat day with risk increasing 6.76% for each degree increase in humidex above 37.4 °C), acute renal failure (68% increased risk on a heat day, increasing 7.6% for each degree above threshold), and natural heat exposure (244% increased risk on a heat day, increasing 17.5% for each degree above threshold). Additionally, the time series analysis found a statistically significant increase in all-ages respiratory-related hospitalizations (2.3% increased risk for each degree above threshold). Comparatively, Knowlton et al. (2008) found all-age hospitalizations increased by 5% for nephritis and nephrotic syndromes, 11% for acute renal failure, 950% for heat-related illness, and 9% for electrolyte imbalance, during the July 15 - August 1, 2006 California heat wave as compared to a referent period.

When stratifying cause-of-admission categories by age group, we found with the relative risk analysis that heat's overall effect does not exclusively impact the elderly (85+ age group). Rather, the results suggest hospitalizations impact a younger population including: 0-4 age group (mental health), 15-44 age group (natural heat exposure), and 45+ age groups (nephritis and nephrotic syndromes, acute renal failure, and natural heat exposure). Similar younger age-vulnerable populations were found (Chapter 2) when we examined the relative risk of mortality associated with extreme heat in King County, Washington. However, unlike the time series mortality results (Chapter 2) where heat intensity almost exclusively affected the 85+

age group, the hospitalization results revealed heat intensity effects for the 15-44, 45-64, and 65-84 year-old age groups.

This study continues to highlight the vulnerability of the elderly, 85+ population to circulatory and its subcategory cardiovascular health outcomes. The same general modeling approach and geographic study area was used in our previous analysis of heat and mortality (Chapter 2). Our mortality results showing increased circulatory (4.1%) and cardiovascular deaths (4.3%) with each degree increase in humidex above the threshold (36.0 °C), parallel the association we found between increased circulatory (4.8%) and cardiovascular hospitalizations (4.4%). It has been hypothesized that the time between heat exposure and resulting cardiovascular death is short (Linares and Diaz 2008), and that social isolation increases the risk of death (Kovats, Hajat and Wilkinson 2004; Linares and Diaz 2008; Michelozzi et al. 2009). It has also been found that cardiovascular mortality observed on extreme heat days was higher for out-of-hospital deaths than in-hospital deaths (Medina-Ramon et al. 2006). Our mortality (Chapter 2) and morbidity findings suggest that the impact of extreme heat on cardiovascular health is much greater than any one study finding taken alone.

Results from both analyses in this study identify a non-elderly population vulnerable to renal-impairment. For the 45-64 year-old age group, we found that the relative risk of hospitalization on a heat day from nephritis and nephrotic syndromes was 76%, with risk increasing 9.5% for each degree increase in humidex above 37.4 °C. Similarly, this age group experienced a 99% increased risk of acute renal failure admissions, with risk increasing 9.2% for each degree increase in humidex above 37.4 °C. With both analyses, we found significant increases in nephritis and nephrotic syndromes and acute kidney failure hospitalizations for the 64-84 year-old age group as well, although not as large. While the mortality analysis did not find

a parallel increase in nephritis and nephrotic syndromes and acute renal failure mortality, for the 45-64 year-old age group we found a 78% greater risk of death from diabetes on a heat day than on a non-heat day, with risk increasing 14.22% for each degree increase in humidex above 36.0 °C. Comparatively, Hansen et al. (2008) found that 15-64 year-olds living in Adelaide, Australia, had a 13% increased risk of hospitalization for renal disease and a 25% increased risk for acute renal failure during heat wave days (compared to non-heat wave days). They also found that diabetic females 50-54 and 85+ years of age were at an increased risk for hospitalization due to renal disease (Hansen et al. 2008). In California, Ostro et al.'s (2010) meta-analysis found an elevated risk of diabetes and acute renal failure hospitalizations, 4% and 10.2%, respectively. In our study, we did not have access to comorbid data, such as diabetes prevalence. However, with roughly 44% of new kidney-failure cases nationwide originating from diabetic patients (United States Renal Data System 2007), our findings suggest that King County's diabetic population could be at an increased risk for kidney-related health outcomes. Given that 8% of 45-64 year-olds and 15% of 65+ year-olds living in King County are estimated to have diabetes, our findings are an important consideration for outreach and prevention programs (Public Health - Seattle & King County 2013).

Aside from age, this study did not find that the individual-level covariates of gender, admission source, admission type, and socio-economic status, modified the effect of heat on hospitalizations. Other individual-level characteristics, not collected by CHARS, may be more relevant predictors of vulnerability to heat-related morbidity. Studies have found that race and ethnicity (Fletcher et al. 2012), income level (Lin et al. 2012), educational achievement, and social isolation affect risks for hospitalization and death (O'Neill, Zanobetti and Schwartz 2003; Medina-Ramon et al. 2006). This study found that the same-day humidex exposure had the

strongest association with hospitalization, and that there was no evidence of a lag or cooling effect. These findings are similar to those of other studies in which either the same-day apparent temperature (Green et al. 2010) or recent lags of 0-2 days were found to be most relevant to the heat-morbidity relationship (Fletcher et al. 2012).

This study did not adjust for air pollution, specifically ozone and PM<sub>2.5</sub>. Given that this study found a statistically significant increase in respiratory hospitalizations for the all-ages, 15-44 and 85+ age groups, confounding effects from air pollution may seem to be of concern. Numerous studies have assumed that air pollution may confound the association between heat and morbidity and have subsequently controlled for pollution levels in their respective studies (Lin et al. 2012; Li et al. 2011; Lin et al. 2009; Green et al. 2010; Linares and Diaz 2008; and Kovats, Hajat and Wilkinson 2004) We contend that it is unnecessary to control for air pollution because it does not meet the definition of a confounder. A confounder is a characteristic that must not only be related to both the exposure and the outcome but must also affect the exposure (Koepsell and Weiss 2003). While ambient temperatures may affect the levels of air pollution, air pollution levels do not affect ambient temperatures. Buckley, Samet and Richardson (2014) argue this point through the use of directed acyclic graphs (DAGs). They conclude that greater care should be taken in clarifying causal assumptions underlying model adjustments.

This study did not correct for multiple comparisons. A Type 1 error may occur when many subgroups are analyzed for effect difference; the more comparisons analyzed, the more opportunity there is to identify by chance a result that appears significant, even when no statistically significant difference exists. A multiple testing correction, such as Bonferroni, could be applied to our analyses (Koepsell and Weiss 2003). However, multiple testing corrections

have their disadvantages by reducing study power, resulting in false negative results. Instead, we looked at all the statistically significant results and examined them for expected dose-response patterns, concurrence with existing literature, and influence of small counts. When results were found to depend on a small number of outcomes ( $N < 20$ ), we flagged these results in the data presentation and discussion.

Potential limitations to our study include using an average county-wide metric for heat exposure and limiting our study area to a political geographical boundary. Using an average daily county-wide maximum humidex value to estimate heat exposure may result in exposure misclassification when a disproportionate number of cases are below or above the average value. In this study, we did not have access to geocoded address information; however improved exposure assessment could be obtained by assigning each case a maximum humidex value from the meteorological grid center point closest to the hospital used.

Lastly, this study used political jurisdictions (county boundary) to assess the heat-morbidity relationship. This geographical unit of analysis may not accurately reflect how the effects of heat on hospitalization vary spatially. An alternative method, and area for future research, would be to combine populations that experience similar climate zones and therefore should have similar levels of acclimatization. An example of this type of analysis is provided by Lin et al. (2012); they examined 14 different New York State climate regions for associations with excessive heat and respiratory hospitalizations, and then predicted future health burdens given climate change.

### **3.5 Conclusion**

This study characterized King County, Washington's historic heat-morbidity relationship using two different statistical methods. The results demonstrate that heat, expressed as humidex,

is associated with increased hospitalizations on heat days, and that the risk increases with heat intensity. This study found that in addition to the elderly, younger age groups have an increased relative risk of hospitalization for several of the admission categories, particularly nephritis and nephrotic syndromes, acute renal failure, and natural heat exposure. When considering heat's intensity, we found effects on health outcomes in age groups as young as 15-44 years-old; this contrasts our mortality findings, where intensity almost exclusively affected the 85+ age group. Individual-level characteristics (age being an exception) and other heat effects from nighttime cooling, heat event duration, lags, and synoptic air mass classification were not found to affect hospitalization rates.

Future research is needed to validate the methods used to model our heat-morbidity relationship, as our piece-wise linear model fits a linear slope to an otherwise non-linear relationship. Consideration should be given to the overall circulatory health of the 85+ population on extreme heat days, as improvements would affect both hospitalization admission and mortality rates. Lastly, our findings warrant additional investigation into the role heat exposure plays in the diabetic patient's health and care, as well as the connection between diabetes and renal syndromes in our area.

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## Chapter 4

# Validating health outcome models associated with extreme heat exposure in King County, WA

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## Chapter 4 abstract

### Background

Climate change is predicted to increase the intensity and duration of extreme heat events. Already, poor health outcomes have been associated with historic extreme heat. Understanding the local heat-health relationship is important when predicting future burdens from climate change. There have been very few studies that test the performance of the underlying heat-health relationship model.

### Methods

This study uses a non-parametric Poisson regression model with a piece-wise linear function to estimate heat's effect on mortality and morbidity, beyond a model-derived threshold. Calibration and validation analyses were performed using death certificate and hospital admission data for two distinct time periods: 1980-2006 mortality/1990-2006 hospital admissions (calibration) and 2007-2010 (validation). We then used two general approaches to test the performance of the extreme heat-health outcome model.

The first approach used a two-sample *t*-test, with unequal variances, to test for a significant difference between the average observed mortality and hospitalization counts for extreme heat days and the average expected health outcome counts predicted by our calibration model estimates. The second approach compared slope estimates from the calibration time frames to the validation time frame using a two-sample *Z*-test with pooled standard errors.

### Results

This study found a 1.79% (95% CI: 0.63%, 2.97%) increased risk of all-age, all non-traumatic mortality for each 1 °C increase in humidex above 35.8 °C during the calibration time

frame, and a 2.77% (95% CI: 0.65%, 4.94%) increased risk for the same threshold during the validation time frame. For morbidity, a 2.23% (95% CI: 1.31 %, 3.15 %) increased risk of all-age, all unplanned non-traumatic hospital admissions was found for each 1 °C increase in humidex above 37.5 °C during the calibration time frame, and a 1.56 % (95% CI: 0.59 %, 2.55 %) increase in risk for the validation time frame. A two-sample *t*-test, with unequal variances, indicates that our model statistically significantly underestimated ( $p = 0.01$ ) the expected mortality for the lower bound of the model's confidence interval, yet it performed well for expected hospital admission counts. A two-sample *Z*-test with pooled standard errors compared each validation coefficient to the calibration time frames' coefficient and did not find a statistically significant difference between the estimates of heat effect on health, with one exception. When comparing the validation time frame's hospitalization model-derived estimate to the calibration time frame's estimate, a statistically significant difference was found.

## **Conclusions**

Our study validated the performance of a heat-health outcome relationship model developed to aid in the prediction of climate change-related mortality and morbidity for King County, Washington. The results suggest that, overall, the piece-wise linear function describing heat's effect on health outcomes performed well. Our model is conservative, and will likely result in predictions that have at least as many deaths and hospitalizations as observed.

## **4.1 Introduction**

Solid scientific evidence indicates that climate change is occurring and that human activity has played a significant role in the warming of the planet (IPCC 2013). Further, there is widespread consensus that the effects from climate change present a serious threat to the public's health and well-being (Balbus et al. 2008; Confalonieri et al. 2007). Numerous studies have

found that extreme heat is associated with increased mortality (Busch Isaksen et al. 2014, Anderson and Bell 2011; Jackson et al. 2010; Anderson and Bell 2009; Basu, Feng and Ostro 2008; Baccini et al. 2008; O’Neill, Zanobetti, and Schwartz 2005; Naughton et al. 2002; Whitman et al. 1997) and hospitalizations (Ye et al. 2012; Fletcher et al. 2012; Ostro et al. 2010; Green, Basu and Malig 2010; Lin et al. 2009; Knowlton et al. 2008; Kovats and Hajat 2008; Mastrangelo et al. 2007; Semenza 1999; and Semenza et al. 1999). Additionally, several studies emphasize the necessity to explore heat-health relationships on a regional or place-specific basis, suggesting that characteristics unique to a location make a significant difference in this relationship (Yardley et al. 2010; Baccini et al. 2008; Kinney et al. 2008; Davis et al. 2004; Curriero et al. 2002).

The relationship between heat and health outcomes has often been described as a non-linear V-, U-, or J-shaped curve, where there is a “threshold” or “turning point” beyond which mortality and hospitalizations increase (Lin et al. 2012; Baccini et al. 2008; Kim et al. 2006; Curriero et al. 2002; Kalkstein and Davis 1989). While many studies use a Poisson generalized additive model with nonparametric curves or splines to describe this relationship and to control for long-term changes in mortality or hospitalizations over time (Gasparrini and Armstrong 2011; Anderson and Bell 2009; Green, Basu and Malig 2010; Basu, Feng and Ostro 2008; Medina-Ramon and Schwartz 2007; Mastrangelo et al. 2007; Curriero et al. 2002), the methods used to summarize and report the effects of heat on health vary. Methods used to interpret heat’s impact on health include: comparing changes in risks of relative percentiles (Gasparrini and Armstrong 2011; Anderson and Bell 2009; Medina-Ramon and Schwartz 2007), estimating changes in risk per 10 °F increase in mean daily apparent temperature (Green, Basu and Malig

2010; Basu, Feng and Ostro 2008; Curriero et al. 2002) and comparing heat wave day/event risk against non-heat wave day/event risk (Knowlton et al. 2008; Mastrangelo et al. 2007).

Others have chosen to model the effect of heat on mortality and morbidity as a piece-wise linear function, estimating changes in risk for each additional 1 °C unit of apparent temperature or humidex above a defined threshold (Chapters 2 & 3; Busch Isaksen et al. 2014; Lin et al. 2012; Lin et al. 2009; Baccini et al. 2008; Medina-Ramon and Schwartz 2007; Armstrong 2006). Several studies have suggested that the use of a piece-wise linear fit for an otherwise non-linear relationship increases the ease of interpretation and reporting of meaningful results to public health practitioners and policymakers (Lin et al. 2012; Lin et al. 2009; Armstrong 2006). Finding ways to effectively communicate results to policymakers has become increasingly important in order to direct mitigation action and adaptation-related policy decisions (Confalonieri 2007).

Understanding the local heat-health relationship is also important when estimating future outcomes associated with climate change. Of the 14 projection studies reviewed by Huang et al. (2011), all started with a basic understanding of the historical heat-health relationship in the study area. While there are a variety of ways authors have modeled historical heat-health relationships, very few studies have validated the methods used to model and interpret heat's effect on health (Dessai 2002; Gosling, McGregor and Paaldy 2007; Fouillet et al. 2007).

This study validates the performance of a heat-health outcome relationship model developed to aid in the prediction of climate change-related mortality and morbidity for the 14<sup>th</sup> most populous county in the United States - King County, Washington (King County 2012). King County's heat-health relationship was described using a nonparametric Poisson regression model with penalized cubic splines to control for long-term changes in health outcomes over

time (not attributable to heat) and effects from month and day of the week. A piece-wise linear function was added to estimate heat's impact on mortality and morbidity, beyond a model-derived threshold. These historical heat-health outcome estimates were used with population projections and three different future climate scenarios to project mortality (Busch Isaksen et al. 2014) and morbidity (Hom et al. 2013) for 2025 and 2045. To our knowledge, no other study has validated a heat-health outcome relationship model using a piece-wise linear approach to define heat's effect on health outcomes.

## **4.2 Methods**

### **4.2.1 Health outcome and population data**

Daily total death and hospitalization counts were obtained for King County, Washington from the Washington State Department of Health. Death certificate data for all non-traumatic causes of mortality were obtained for 1980 to 2010, while Comprehensive Hospital Abstract Reporting System (CHARS) discharge data for all unplanned, non-traumatic admissions were obtained for 1990 to 2010. Only deaths and hospitalizations that occurred during the warmer summer months of May through September were analyzed; colder months were excluded to minimize potential confounding by infectious diseases typically seen during these months. All non-traumatic deaths (ICD-9 001-799 and ICD-10 A00-R99) and hospitalizations (ICD-9 001-799) were coded using the International Classification of Diseases (ICD). Population data, by age groups (0-4, 5-14, 15-44, 45-64, 65-84, 85+), were obtained from the Washington State Office of Financial Management (OFM) (Washington State Office of Financial Management 2012).

#### **4.2.2 Meteorology and exposure data**

A historical (1915-2012) 1/16° gridded meteorological data set was produced for our study area by the University of Washington's Climate Impacts Group (Maurer et al. 2002). This data set was derived using the Parameter-Elevation Relationships on Independent Slopes Model (PRISM), a spatial climatologic model developed by Oregon State University (PRISM Climate Group). Representing the most current knowledge on spatial climatic patterns for the United States (Daly et al. 2007), PRISM was used along with weather station observations from the Global Historical Climate Network-Daily (GHCN), to produce daily temperature and relative humidity values for our geographical study area. GHCN is a National Oceanic and Atmospheric Administration database of daily measurements from land surface stations across the globe (NOAA 2009). The resulting meteorology data set contains daily max/min temperature, precipitation and relative humidity values for each gridded center point. Daily average county-wide maximum temperature and average relative humidity values were used to construct our exposure metric, humidex. Humidex is an apparent “feels-like” index that measures the combined effects of temperature and humidity on the human body (Masterton and Richardson 1979). Exposure to heat was estimated as the average maximum county-wide humidex value for each day within the study's time frame.

#### **4.2.3 Model construction and validation**

Model construction and identification of mortality and hospitalization thresholds followed the methodology reported in Chapters 2 and 3, as well as Busch Isaksen et al. (2014). Similar to methods used to study heat and hospitalizations in New York City (Lin et al. 2012; Lin et al. 2009), our analyses used a Poisson regression with nonparametric splines to model the changes in log-admission and mortality rate over time not associated with heat. Figure 4.1

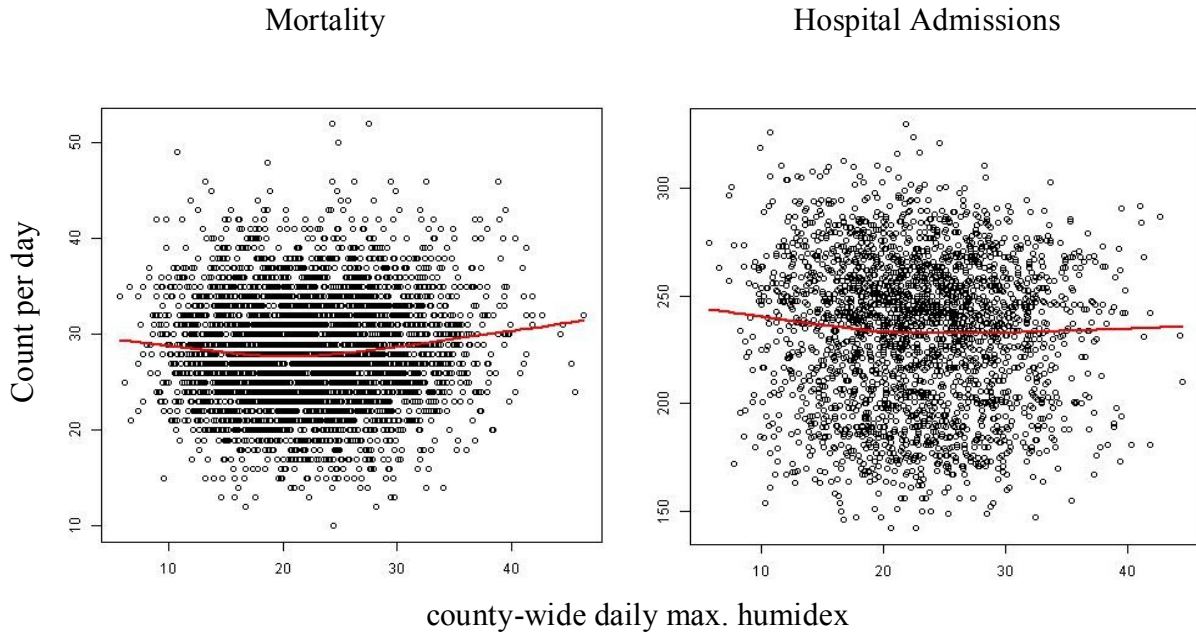
illustrates a Lowess curve modeling the humidex effect over mortality and hospitalization counts per day for both the calibration and validation time frames, while Figure 4.2 illustrates the penalized cubic regression spline, modeling the humidex effect over log-mortality and hospital admissions.

To increase interpretability, we replaced the penalized cubic spline modeling humidex, and instead estimated heat's effect on log-mortality and hospital admission rates with a piece-wise linear function, fit with two knots. The first knot was set at the 50<sup>th</sup> percentile of summer-time humidex values. The maximum likelihood for the second knot, or "optimal alert threshold" for humidex, was identified by exploring 0.1 degree incremental changes starting at 20 °C and continuing through 44 °C humidex. A heat day was then defined as a day in which the average county-wide daily maximum humidex exceeded the optimal alert threshold. The impact of humidex intensity on mortality and admissions was assessed by the slope of the line above the threshold and interpreted as the percent increase in risk for each 1 °C unit increase in humidex. Analyses were performed on the data for two distinct time periods: 1980-2006 (calibration) and 2007-2010 (validation) for mortality, and 1990-2006 and 2007-2010 for hospitalizations. The calibration time frames represent previous study results for mortality (Busch Isaksen et al. 2014) and hospitalization (Hom et al. 2013), while the validation time frame represents new data not previously available during the model's development.

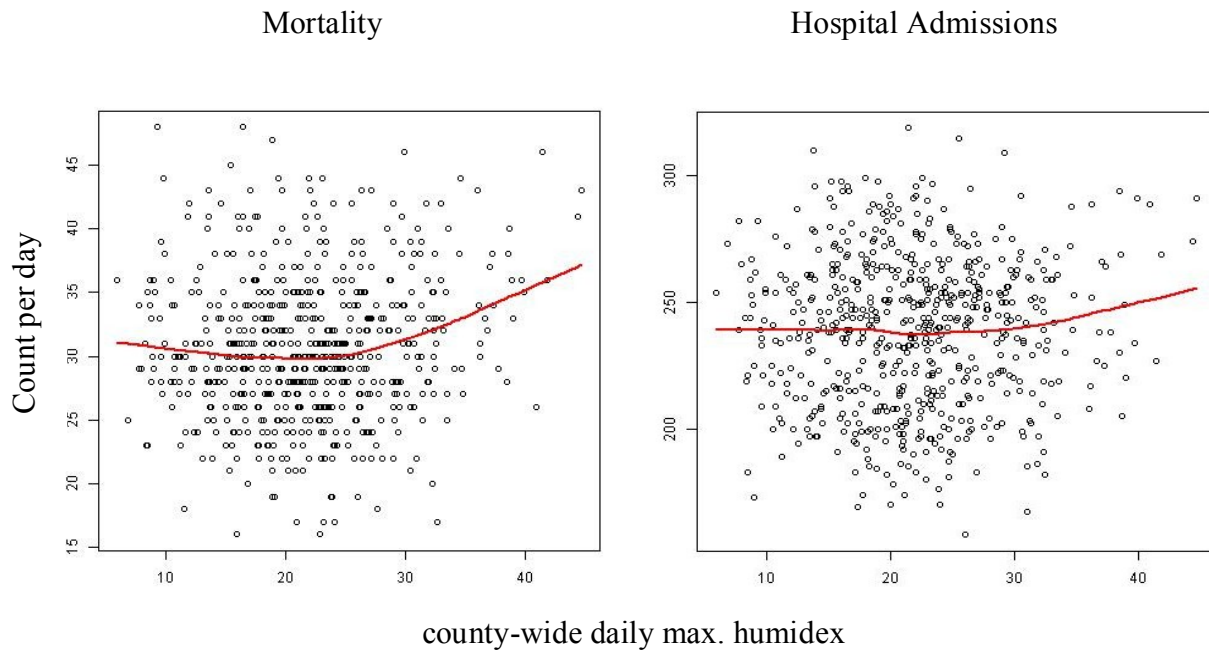
Two general approaches were used to test the validity of modeling the underlying heat-health outcome relationship. The first approach uses an expected versus observed method to investigate heat days during the validation time frame that exceed King County's mortality and morbidity calibration time frames' thresholds (35.8 °C and 37.5 °C humidex, respectively).

**Figure 4.1** Lowess non-parametric regression curve modeling humidex effect over daily mortality and hospitalization counts

**Calibration 1980/1990-2006**

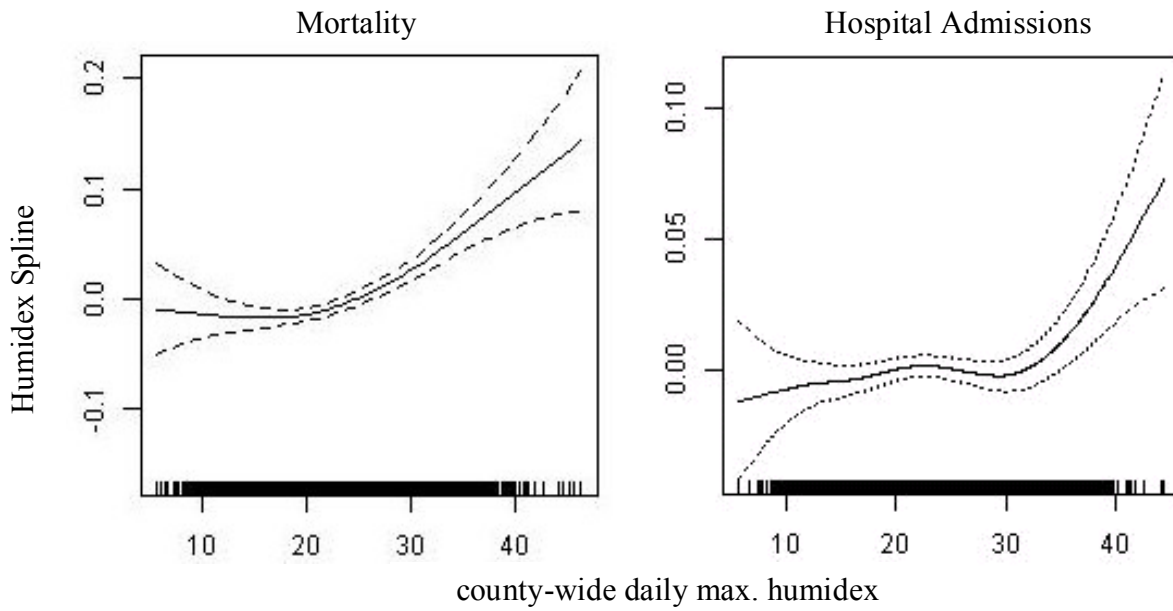


**Validation 2007-2010**

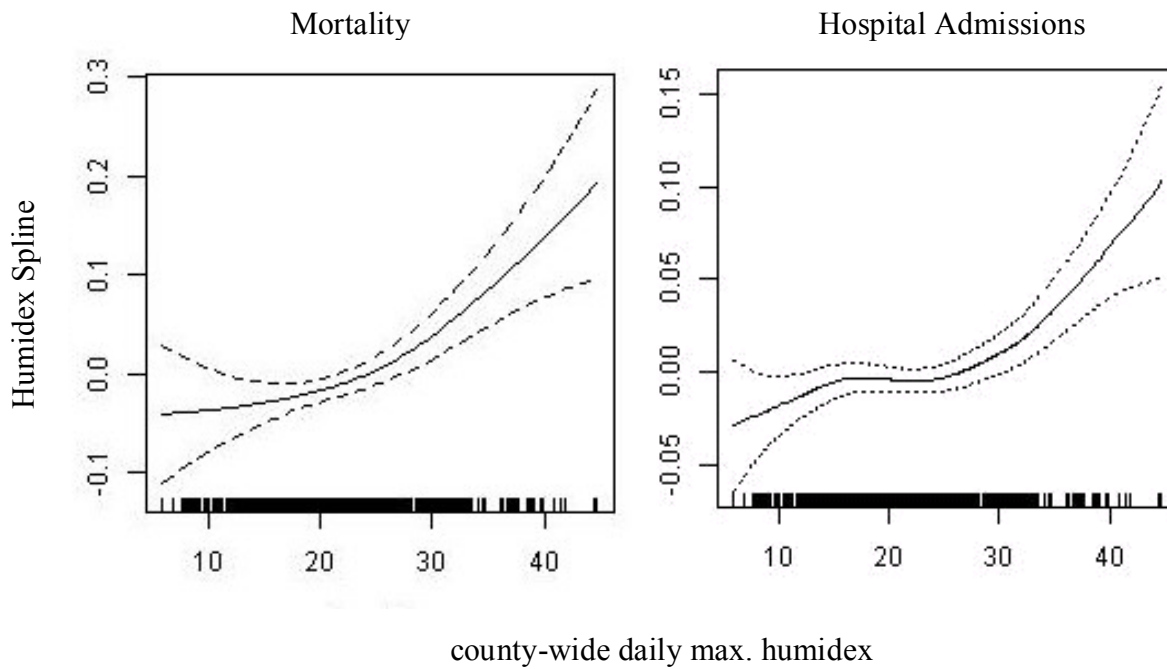


**Figure 4.2** Penalized cubic regression spline modeling humidex effect over log-mortality and log-hospital admissions

**Calibration 1980/1990-2006**



**Validation 2007-2010**



We compared the observed mortality and hospitalization counts for each heat day to the expected health outcome count predicted by our calibration model estimates. Expected mortality and hospitalization counts for each heat day above calibration thresholds were calculated factoring in cumulative effects of temperature increase by using the following formula:

$$x_j = \bar{x} \cdot \exp\{\widehat{\beta}_1 t\} \quad (1)$$

where  $x_j$  is the average expected count on day  $j$ ,  $\bar{x}$  is the average count on non-heat days, 2007-2010,  $\widehat{\beta}_1$  is the calibration time frame estimate and  $t$  is the increase above threshold. The average mortality count for non-heat days during the validation time period was used for the baseline,  $\bar{x}$ , to control for confounding associated with changes in population or medical access. A two-sample  $t$ -test with unequal variances was used to compare mean, minimum and maximum expected from observed counts aggregated over all heat days.

The second validation approach compares slope estimates from the calibration time frames to the validation time frame. We estimated two different slopes for the validation time frame; the first slope coefficient represents a model-derived estimate, while the second slope coefficient represents a forced slope estimate derived by using the threshold from the calibration time frame. We then used a two-sample  $Z$ -test to compare each coefficient in the validation time frame, to the corresponding coefficient in the calibration time frame:

$$Z = \frac{(\widehat{\beta}_1 - \widehat{\beta}_2) - 0}{\sqrt{SE_1^2 + SE_2^2}} \quad (2)$$

where  $\widehat{\beta}_1$  and  $\widehat{\beta}_2$  are the validation and calibration coefficient estimates (respectively),  $0$  is the hypothesized difference between the estimates, and  $SE_1$  and  $SE_2$  are the pooled standard errors (Tauritz 2002).

### 4.3 Results

King County, located in Western Washington, is characterized by relatively mild summers. It is the 14<sup>th</sup> most populous county in the United States (King County 2012) and has seen its population increase over 52% from 1980 to 2010 (Washington State Office of Financial Management 2012). Its summertime, 1980-2006, county-wide maximum humidex values range from a 50<sup>th</sup> percentile of 13.0 °C (55.4 °F) to a 99<sup>th</sup> percentile of 35.8 °C (96.44 °F). As table 4.1 shows, the validation time frame's 99th maximum humidex percentile is 1.3 - 1.5 °C humidex greater, at 37.3 °C (99.1 °F), than the calibration time frames.

#### 4.3.1 Model construction

The nonparametric, piece-wise linear Poisson regression estimates for each time frame, are presented in Table 4.2. We found that, when the validation time frames were forced with calibration time frame parameters, the effect estimates were roughly similar. The calibration time frame estimated a 1.79% (95% CI: 0.63%, 2.97%) increased risk of all-age, all non-traumatic mortality for each 1 °C increase in humidex above 35.8 °C, while the validation time frame estimated a 2.77% (95% CI: 0.65%, 4.94%) increased risk for the same threshold. Similarly for hospitalization risks, the calibration time frame estimated a 2.23 % (95% CI: 1.31 %, 3.15 %) increased risk of all-age, all unplanned non-traumatic admissions for each 1 °C increase in humidex above 37.5 °C, while the validation time frame estimated a 1.56 % (95% CI: 0.59 %, 2.55 %) increased risk. Allowing the model to tune mortality and hospitalization thresholds for the validation time frame, we found a 5 °C difference between calibration and validation thresholds, 40.9 °C and 32.4 °C, respectively.

**Table 4.1** May - September average daily outcome count and humidex measures in King County, Washington for calibration and validation time frames.

<b>Calibration 1980-2006, 1990-2006</b>	
1980-2006	
Average mortality count	26.2 deaths/ non-heat day
50 <sup>th</sup> Percentile Maximum Humidex	13.0 °C (55.4 °F)
99 <sup>th</sup> Percentile Maximum Humidex	35.8 °C (96.4 °F)
1990-2006	
Average hospitalization count	230.1 admissions/non-heat day
50 <sup>th</sup> Percentile Maximum Humidex	13.0 °C (55.4 °F)
99 <sup>th</sup> Percentile Maximum Humidex	36.0 °C (96.8 °F)
<b>Validation 2007-2010</b>	
50 <sup>th</sup> Percentile Maximum Humidex	11.7 °C (53.1 °F)
99 <sup>th</sup> Percentile Maximum Humidex	37.3 °C (99.1 °F)
Heat days above mortality threshold, 35.8 °C humidex	23
Heat days above hospitalization threshold, 37.5 °C humidex	14

**Table 4.2** Time series analysis results: Percentage increase (95% CI) in mortality and hospitalizations per degree increase in county-wide average daily maximum humidex °C above threshold (°C), by time frame and threshold<sup>1</sup>

<i>All ages, All non-traumatic causes</i>	
<b>Calibration</b>	
<b>Mortality</b>	
1980-2006 (35.8 °C)	<b>1.79% (0.63%, 2.97%)</b>
<b>Morbidity</b>	
1990-2006 (37.5 °C)	<b>2.23 % (1.31%, 3.15 %)</b>
<b>Validation</b>	
<b>Mortality</b>	
2007-2010 (35.8 °C) Forced	<b>2.77% (0.65%, 4.94%)</b>
2007-2010 (40.9 °C) Model fit	<b>7.47% (0.95%, 14.4%)</b>
<b>Morbidity</b>	
2007-2010 (37.5 °C) Forced	<b>1.56 % (0.59%, 2.55 %)</b>
2007-2010 (32.4 °C) Model fit	<b>0.78 % (0.38%, 1.19 %)</b>

<sup>1</sup> Bolded, time series estimates are significantly greater than 0 ( $p < 0.05$ )

### 4.3.2 Model performance

There were 23 days during the validation time frame that exceeded the calibration time frame's mortality threshold of 35.8 °C humidex, and there were 14 days that exceeded the hospitalization threshold of 37.5 °C humidex (Table 4.1). The average 2007-2010 non-heat day mortality count was 27.9/day, while the average hospitalization count was 233/day (Table 4.3). Comparatively, the average 1980 (mortality) and 1990 (hospitalization) - 2006 non-heat day count was 26.2 and 230.1 per day, respectively. The expected/observed analysis found that for the 23 days above the mortality threshold of 35.8 °C humidex, the average observed count was 7.7% greater than the average expected count, with a range of 11.60% - 3.83%, based on the predicted Poisson regression 95% confidence interval [1.79% (95% CI: 0.63%, 2.97%)] (Table 4.3). Likewise, the average observed hospitalization count was 3.2% greater than the average expected count for the 14 days above the hospitalization threshold of 37.5 °C humidex, with a range of 5.9% - 0.6% for the predicted estimate and confidence interval, 2.23 % (95% CI: 1.31%, 3.15%) (Table 4.3).

A two-sample *t*-test with unequal variances was used to compare expected counts, along with the confidence interval's lower (minimum expected count) and upper bounds (maximum expected count), to observed counts aggregated over all heat days. The results, provided in Table 4.4, indicate that, overall, our model performed well at predicting health outcome counts. However, our model does statistically significantly underestimate expected mortality at the lower bound of the estimate's confidence interval. Conversely, our model appears to perform well across the estimate's confidence interval when predicting hospital admissions associated with extreme heat above the threshold of 37.5 °C humidex (Table 4.4).

**Table 4.3** Expected versus observed mortality and hospitalization counts on extreme heat days

	<b>Av. observed non-heat day (2007-2010) count</b>	<b>Expected count</b>	<b>Lower bound CI:95% expected count</b>	<b>Upper bound CI:95% expected count</b>	<b>Av. observed heat day (2007-2010) count</b>
<b>Mortality</b> - All ages, all non-traumatic 1.79% (95% CI: 0.63%, 2.97%) n=23 days					
Mortality count	27.9	29.5	28.4	30.6	31.7
% greater than expected		7.7%	11.6%	3.8%	
<b>Hospitalizations</b> - All ages, all non-traumatic 2.23 % (95% CI: 1.31%, 3.15%) n=14 days					
Hospitalization count	233.0	247.8	241.5	254.2	255.7
% greater than expected		3.2%	5.9%	0.6%	

**Table 4.4** Expected versus observed validation results, ages, all non-traumatic causes

<b>Mortality<sup>1</sup></b>	<b>Av. Expected vs. Observed Count</b>		<b>Min. Expected vs. Observed Count</b>		<b>Max. expected vs. Observed Count</b>	
	<i>Av. Expected</i>	<i>Observed Count</i>	<i>Av Min Expected</i>	<i>Observed Count</i>	<i>Av Max. Expected</i>	<i>Observed Count</i>
Mean	29.47	31.74	28.44	31.74	30.60	31.74
Observations	23	23	23	23	23	23
P(T<=t) two-tail	0.06		<b>0.01</b>		0.35	

<b>Hospital Admissions<sup>2</sup></b>	<b>Av. Expected vs. Observed Count</b>		<b>Min. Expected vs. Observed Count</b>		<b>Max. expected vs. Observed Count</b>	
	<i>Av. Expected</i>	<i>Observed Count</i>	<i>Av Min Expected</i>	<i>Observed Count</i>	<i>Av Max. Expected</i>	<i>Observed Count</i>
Mean	247.75	255.71	241.53	255.71	254.19	255.71
Observations	14	14	14	14	14	14
P(T<=t) two-tail	0.38		0.11		0.87	

<sup>1</sup> 1980-2006 time series analysis all ages, all non-traumatic estimate 1.79% (95% CI: 0.63%, 2.97%)

<sup>2</sup> 1990-2006 time series analysis all ages, all non-traumatic estimate 2.23 % (95% CI: 1.31%, 3.15%)

The second test for model performance compared estimates of heat effects on health outcomes from the calibration time frames to the validation time frame (Table 4.2). We used a two-sample Z-test with pooled standard errors for comparing each validation coefficient to the calibration time frames' coefficients. We found that, when comparing the estimates derived from the forced model parameters to the calibration time frame's estimates, there was no significant difference between the estimates of heat effects on health (Table 4.5). However, when comparing the validation time frame's hospitalization model-derived estimate to the calibration time frame's estimate, a statistically significant difference was found.

#### **4.4 Discussion**

This study characterized King County, Washington's historic heat-health outcome relationship using a nonparametric Poisson regression to model the changes in health outcomes over time, not associated with heat, and using a piece-wise linear term to estimate heat's effect on mortality and hospital admission rates. The model construction results demonstrate that heat, expressed as humidex, is associated with increased mortality and hospital admissions on days above corresponding thresholds, and that the risk increases with heat's intensity. Furthermore, this study assessed the performance of the heat-health outcome model using two methods: expected versus observed and heat-effect estimate comparison. To our knowledge, only a few heat-health outcome relationship validation studies have been conducted previously (Dessai 2002; Gosling, McGregor and Paaldy 2007; Fouillet et al. 2007), and only one has used Poisson regression model construction methods similar to our own (Fouillet et al. 2007). Our study is unique in that it tests how a piece-wise linear function performs in estimating heat's effect on health outcomes.

**Table 4.5** Time series analyses: Z-test for model performance, all ages, non-traumatic causes<sup>1</sup>

<b>Mortality (Threshold)</b>	<b>TSA Estimate</b>	<b>Z score</b>	<b>Two-tailed p-value</b>
1980-2006 (35.8 °C)	<b>1.79%</b> <b>(95% CI: 0.63%, 2.97%)</b>		
2007-2010 (35.8 °C)	<b>2.77%</b> <b>(95% CI: 0.65%, 4.94%)</b>	0.79	0.43
Forced 2007-2010 (40.9 °C)	<b>7.47%</b> <b>(95% CI: 0.95%, 14.4%)</b>	1.63	0.10
<b>Hospital Admissions (Threshold)</b>	<b>TSA Estimate</b>	<b>Z score</b>	<b>Two-tailed p-value</b>
1990-2006 (37.5 °C)	<b>2.23%</b> <b>(95% CI: 1.31%, 3.15%)</b>		
2007-2010 Forced (37.5 °C)	<b>1.56 %</b> <b>(95% CI: 0.59%, 2.55%)</b>	-0.977	0.329
2007-2010 (32.4 °C)	<b>0.78 %</b> <b>(95% CI: 0.38%, 1.19 %)</b>	-2.827	<b>0.005</b>

<sup>1</sup> Bolded time series analysis estimates are significantly greater than 0 ( $p < 0.05$ )

Our model construction with Poisson model-derived thresholds differs considerably from both Dessai (2002) and Gosling, McGregor, and Paaldy (2007), where mortality thresholds were identified using an expected versus observed method. Fouillet et al. (2007) used a similar Poisson model with nonparametric splines to control for changes in mortality not associated with heat. However, their main focus was to identify and validate the optimal combination of temperature indicators that explained the daily mortality fluctuations in France, 1975-2003, rather than to validate the Poisson model performance.

This study found a statistically significant increase in all-ages, non-traumatic mortality and hospitalizations. Mortality was found to increase 1.79% and 2.77% for each 1 °C increase in humidex above 35.8 °C, for the calibration and validation time frames, respectively. Other US-based studies have found smaller, 1-3% increases in daily non-traumatic mortality per 10 °F increase in daily apparent temperature, equivalent to approximately a 0.2-0.5% increase in mortality per 1 °C increase above corresponding thresholds and varying study time frames (Basu, Dominici and Samet 2005; Basu, Feng and Ostro 2008; Zanobetti and Schwartz 2008). However, in the Mediterranean regions of Europe, Baccini et al. (2008) found 1.8% and 3.1% increased mortality per 1 °C increase in apparent temperature for thresholds of 23.3 °C and 29.4 °C, respectively. In our study, hospital admissions were found to increase 2.23% and 1.56% for each 1 °C increase in humidex above 37.5 °C for the calibration and validation time frames, respectively. Comparatively, Linares and Diaz (2008) found a 4.6% increase in unplanned hospital admissions for each degree increase in maximum daily temperature above 36.6 °C in Madrid, Spain, 1995-2000.

For the identified 23 and 14 extreme heat days, we expected a 5.6% and 6.3% increase in mortality and hospitalization counts compared to non-heat days. Instead we observed 13.8% and

10% increases in mortality and hospital admissions, respectively, compared to non-heat days. A test to determine whether there was a statistically significant difference between the expected and observed counts found that our model significantly under-predicts at the lower confidence bounds for expected mortality, while it performs well over the entire confidence interval for hospital admission counts. Figure 4.1 suggests one possible explanation for our mortality model's performance; the effect of humidex over daily mortality count is very different for the two time frames. The difference between these time frames could be attributable to the fact that, during the validation time frame, King County experienced its hottest, longest extreme heat event on record. The July/August, 2009 heat event spanned a total of 10 days, with maximum temperatures of 40.6 °C (105 °F) accompanied by 44% humidity for the Seattle area; the combined effect of temperature and humidity translated into a “feels like” humidex value of 53°C (127 °F) (Weather Underground 2011).

When comparing the validation time frame's mortality and hospitalization heat effect estimates (derived using model parameters (forced)), to the calibration time frame estimates, the results were not significantly different. This suggests that the forced model parameters can be expected to perform reasonable well at predicting future extreme heat-related health outcomes. However, when comparing validation estimates derived from the time period itself, against the calibration time frames' estimates, we found the hospitalization estimates to be significantly different. Figure 3.3 (Chapter 3) offers a possible explanation for the significant difference. Over the entire time frame from 1990-2010, there doesn't appear to be a sharp dip in the Akaike Information Criterion (AIC) over humidex, which is used to indicate the maximum likelihood of the model fit. Instead, there appears to be a 5 degree span where there is little difference. This

5-degree span happens to ~32-37 °C, the same span as our model-derived validation and calibration thresholds.

When considering the entire time frame, the optimal thresholds derived by the model were nearly identical to the mortality and hospitalization calibration/forced validation time frames, 36.0 °C versus 35.8 °C (mortality) and 37.4 °C versus 37.5 °C (hospitalization), respectively (Chapters 2 & 3). Furthermore, we observed a similar intensity estimate for all years mortality and hospitalizations (2.12% and 1.59%) compared to the validation time frame's forced model intensity estimate (2.77% and 1.56%) for each 1 °C above threshold, respectively (Chapters 2 & 3). This finding suggests that the extreme heat event is affecting the validation time frame's model-derived threshold and subsequent outcome estimates.

The purpose for interpreting an otherwise non-linear relationship using a piece-wise linear function is to increase the ease of interpretation and reporting of meaningful results to public health practitioners and policymakers. While scientifically it may seem to be more suitable to model heat's effect on health outcomes with a complex spline model, Hurley et al. (2004) found that the more complex models were not always appropriate for the situation, especially when prediction was of interest. Furthermore, even after acknowledging that the assumption of linearity is almost always incorrect, Buis (2009) offers that "The aim of a model is to simplify the situation such that mere mortals can understand the patterns present in the data." If the duration and intensity of extreme heat events increase as predicted (Meehl and Tebaldi 2004), and on the order of what was seen in 2009, our model projections will likely be conservative, with mortality and morbidity outcomes at least as great as our model predictions. This information will allow local public health practitioners and policymakers to understand the

patterns of heat effects on mortality and morbidity and will support preparedness decisions (Confalonieri 2007).

One potential limitation of this study is that our calibration and validation time frames were created out of necessity; the calibration time frames (27 years for mortality and 17 years for morbidity) had already been used to develop the model, while the validation time frame (4 years) was the most recent data available and included the most extreme heat event on record. It is possible that the validation time frame did not include enough data to conduct a robust analysis and that estimates were strongly influenced by the most extreme heat event on record. In comparison, Dessai (2001) split the 18 years of data available into two groups, 9 years for the calibration and 8 for the validation time frame. Similarly, Gosling, McGregor and Paaldy (2007) divided the data for the 6 cities under study into roughly two equal time frames. Only Fouillet et al. (2007) used considerably fewer years in their validation time frame (summer of 2003) versus their 28-year calibration time frame. An alternative validation method would be to re-calibrate our estimates using the entire time frame 1980-2010 and 1990-2010 and employ a cross-validation approach to test the model's performance.

One other potential limitation is the choice of baseline parameters. This study used an expected baseline derived from the validation time frame's average mortality and hospital admission count on non-heat days. It is possible that this was not the correct baseline. A sensitivity analysis, adjusted for changes in population, was conducted using a baseline derived from the calibration time frame. Results showed poorer model performance the further the baseline for expected counts was from the validation time frame.

## **4.5 Conclusion**

This study validates the performance of a heat-health outcome relationship model developed to aid in the prediction of climate change-related mortality and morbidity for King County, Washington. The results suggest that, overall, the use of a piece-wise linear function to describe heat's effect on health outcomes performs well when predicting future extreme heat-related health outcomes. This study did find that the model significantly under-predicts the lower bounds of expected death count on extreme heat days, and that a significant difference occurred between the calibration and the validation's model-derived hospitalization estimates. However, when examining the entire time frame, the resulting threshold and effect estimate further supported the use of a piece-wise linear model to describe heat effects on health outcomes.

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## Chapter 5

### Mapping mortality risks from extreme heat exposure in King County, WA 2007-2010

*List of figures:*

**Figure 5.1** Meteorological data center point locations for each 1/16° grid cell, King County, Washington

**Figure 5.2** Heat distribution z-scores, King County, Washington

**Figure 5.3** Heat-risk distribution - All-ages proportion of expected mortality per 1000 deaths given a heat day, by census tract

**Figure 5.4** Heat-risk distribution - 65+ age group's proportion of expected mortality per 1000 deaths given a heat day, by census tract

## Chapter 5 abstract

### Background

Climate change is predicted to increase the frequency and duration of extreme heat events. Already, poor health outcomes have been associated with historic extreme heat. Understanding the spatial heat distribution and location of vulnerable populations is important when preparing for future events. This is the first study, to our knowledge, that incorporates local heat-mortality relationship data into vulnerability mapping methods.

### Methods

King County's spatial heat distribution was determined using heat days, defined as exceeding 36.0 °C, during 2007-2010. Spatial correlation between expected and observed maximum humidex values for each meteorological grid center point (over all heat days) was determined. Each grid cell humidex value was interpolated to census tract scale, and then mapped. Vulnerable population data were combined with the heat distribution to create heat-risk maps estimating proportion of cases per 1000 persons, by census tract.

### Results

Twenty-three extreme heat days were identified during 2007-2010. The overall coefficient of determination ( $R^2$ ) between expected and observed maximum humidex values was 70 %. Heat distribution was mapped for King County, by census tract. Vulnerable populations identified in the historic heat-mortality analyses were considered for mapping heat-risk. Given spatially available population data, only the 65+ age-group was added to our heat distribution map, creating an overall heat-risk map for King County, by census tract.

## **Conclusions**

Heat-risk maps are visual tools that can be used by planners, public health practitioners, and emergency management professionals to prepare for future extreme heat events. This study uses what we know about the local heat-mortality relationship to construct heat-risk maps for our largest vulnerable population, the 65+ age group. Additional data sources are necessary to create heat-risk maps for other vulnerable populations identified by our epidemiological analyses.

### **5.1 Introduction**

Exposure to extreme heat has been found to have deleterious health effects. Numerous studies report significant associations between extreme heat and increased mortality (Busch Isaksen et al. 2014; Anderson and Bell 2011; Jackson et al. 2010; Anderson and Bell 2009; Basu, Feng and Ostro 2008; Baccini et al. 2008; O'Neill, Zanobetti, and Schwartz 2005; Naughton 2002; Whitman et al. 1997) and hospitalizations (Ye et al. 2012; Fletcher et al. 2012; Ostro et al. 2010; Green et al. 2010; Lin et al. 2009; Knowlton et al. 2008; Kovats and Hajat 2008; Mastrangelo et al. 2007; Semenza 1999; and Semenza et al. 1999). Climate change threatens to increase the duration and intensity of extreme heat events (Meehl and Tebaldi 2004), leading to a serious threat to the public's health and well-being (IPCC 2013, Confalonieri et al. 2007, Balbus et al. 2008). All locations will not be similarly affected by climate change or by heat's effect on health outcomes (IPCC 2013). Local climatic conditions, coupled with unique environmental and population characteristics, result in significant differences in heat-health outcome relationships (Yardley et al. 2010; Baccini et al. 2008; Kinney et al 2008; Davis et al 2004; Curriero et al. 2002).

Several studies have defined the heat-mortality relationship for parts of Washington State. Jackson et al. (2010) explored relative risks for non-traumatic mortality in the Greater Seattle area. They found 1.0 (95% CI: 1,1.1), 1.1 (95% CI: 1,1.1) and 1.1 (95% CI: 1,1.1) times greater mortality risks above the 99<sup>th</sup> percentile of humidex values (heat days) compared to non-heat days, for the 45+, 65+ and 75+ age groups, respectively. Our analysis (Chapter 2) found similar 99<sup>th</sup>-percentile results for King County, Washington; the relative risk of death for all-ages, non-traumatic causes was 10% [1.1 (95% CI: 1.06, 1.14)] greater on a heat day. Heat intensity effects on King County non-traumatic mortality were explored by both Busch Isaksen et al. (2014) and in Chapter 2. Mortality increased by 1.83% (95% CI: 0.77, 2.91) for each one degree increase in humidex above an absolute threshold (35.7 °C), and by 2.12% (95% CI: 1.07, 3.19) for each degree above 36.0 °C, respectively.

Translating local study results into policy action requires more than publishing the results it requires effective communication efforts. Nerlich, Koteyko, and Brown (2010) recommend methods that include dialogue and audience engagement, while Maibach et al. (2010) emphasize the need to frame climate change communications as a public health problem. GIS mapping tools offer a visual way to display health risks and vulnerable population characteristics, and can be an effective means to engage planners, policymakers and the public at large (Graham et al. 2011).

Several studies have mapped heat-vulnerability using different methods. Martinez et al. (1989) used dots placed on a map of the United States to display individual cases of heat-related deaths occurring 1968-1985. Malik, Awan, and Khan (2012), and Reid et al. (2009) used location-specific knowledge about vulnerable populations and adaptive capacity to develop a vulnerability index that identifies spatial variability. However, in validating the heat-vulnerability index described by Reid et al. (2009), Reid et al. (2012) found an increase in poor

health outcomes for both heat and non-heat days, suggesting that their heat vulnerability index may instead be predictive of health vulnerability in general. Houghton et al. (2012) used heat-exposure data, pre-existing chronic disease rates and population vulnerability variables to generate interactive web-based heat-risk maps. Buscail, Upegui, and Viel (2012) included land surface temperature information, population density, and population vulnerability characteristics to develop a health-risk index. Finally, O'Neill et al. (2009) demonstrated how to use census data to generate vulnerability maps, and suggested that planners could simply overlay multiple indicators of risk to generate a visual representation of those census tracts at greater heat-health risk. While all of the described studies have spatially modeled heat-related vulnerable populations, it is important to note that these vulnerable populations were identified through literature and not through location-specific epidemiological analysis. The authors have provided no evidence that these populations are heat-vulnerable in their particular study location.

Our study details the methods used to design a heat-mortality risk map for the 14<sup>th</sup> most populous county in the United States, King County, Washington (King County 2012). When compared to other studies, our methods are unique in that we use information obtained from King County's own historic heat-mortality analysis to define the extreme heat day threshold and to identify the vulnerable populations for mapping the distribution of heat and heat-vulnerability. To our knowledge, no other study uses local heat-health outcome relationships in their heat-risk mapping methodologies.

## **5.2 Methods**

### **5.2.1 Meteorology data**

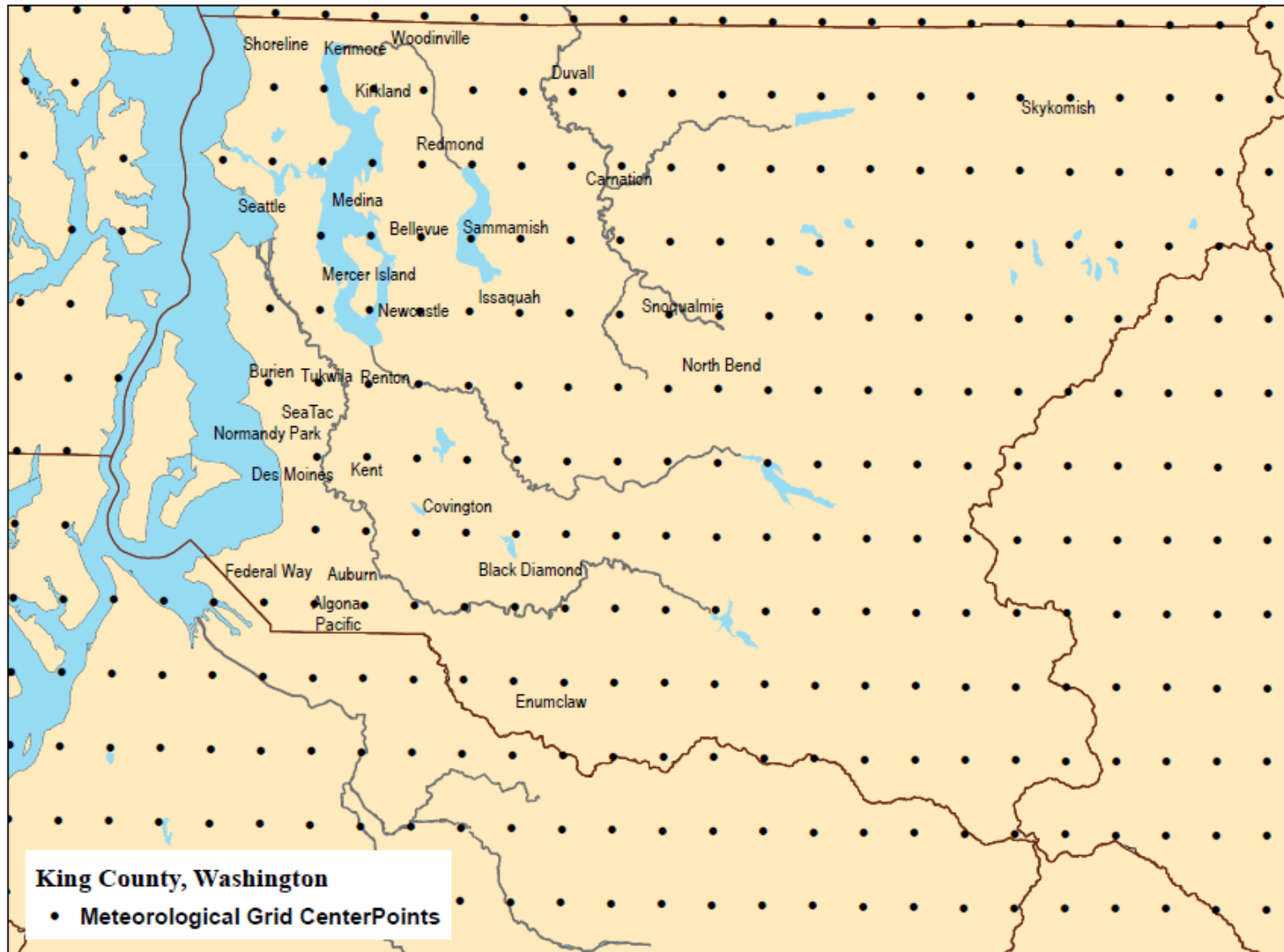
The Parameter-elevation Relationships on Independent Slopes Model (PRISM), a climatologic model developed by Oregon State University, was used by the University of

Washington's Climate Impacts Group to develop a Pacific Northwest, historical (1915-2012) meteorological data set (Maurer et al. 2002). Representing the most current knowledge on spatial climatic patterns for the United States (Daly et al. 2007), PRISM was used along with weather station observations from the Global Historical Climate Network-Daily (GHCN) to produce a gridded record of daily temperature and relative humidity values. GHCN is a National Oceanic and Atmospheric Administration database of daily measurements from land surface stations across the globe (NOAA 2009). The resulting meteorology data set contains daily max/min temperature, precipitation and average relative humidity values for the center point of each 4 km x 7.5 km cell (Figure 5.1). The daily average county-wide maximum temperature and average relative humidity values were used to construct our exposure metric, humidex. Humidex is a feels-like index measuring the combined effects of temperature and humidity on the human body (Masterton and Richardson 1979).

### **5.2.2 Heat-mortality relationship**

Death certificate data for all non-traumatic causes of mortality were obtained from the Washington State Department of Health for 1980 to 2010. Only deaths that occurred during the warmer summer months of May through September were analyzed. Colder months were excluded to minimize potential confounding by infectious diseases typically seen during these months. Population data, by age groups (0-4, 5-14, 15-44, 45-64, 65-84, 85+) were obtained from the Washington State Office of Financial Management (OFM) (Washington State Office of Financial Management 2012).

**Figure 5.1** Meteorological data center point locations for each 1/16° grid cell, King County, Washington



King County's historical (1980-2010) heat-mortality relationship was described using two different methodological approaches. The first approach used a Poisson regression analysis to model relative risk of mortality for a heat day, defined as a day exceeding the 99<sup>th</sup> percentile of average county-wide maximum humidex values. The second approach used a Poisson generalized additive model with nonparametric curves, to control for long-term changes in mortality over time, not attributable to heat. A piece-wise linear function was then added to estimate heat's impact on mortality, beyond a model-derived threshold. Individual-level characteristics were evaluated for differences in mortality risk; and vulnerable populations were identified. Model construction and identification of relative and absolute mortality thresholds, heat effect estimates, and vulnerable populations, follow methodology reported in Chapter 2, as well as by Busch Isaksen et al. (2014). Our methods are also similar to Lin et al. (2012) and Lin et al's. (2009) study of heat and hospitalizations in New York City. All analyses used the statistical software R, version 2.14.1 (R Core Team 2012).

### **5.2.3 Base map and vulnerable population data**

Base map features were obtained from King County GIS Center's online data portal, and include shape files titled: *2010 Census Tracts for King County - Conflated to Parcels, Open water for King County and portions of adjacent counties, Washington Counties, and Cities and Unincorporated King County*. Additionally, *2010 Census SF1 table: Population Substituted - Total Population for Tracts* was obtained from the same online data portal and contains census data for the vulnerable populations identified by our heat-mortality relationship analyses (KCGIS Center, 2010).

#### 5.2.4 Mapping procedure

We first determined whether or not King County's spatial heat distribution is relatively similar for any given heat day. Heat days exceeding the modeled temperature-mortality threshold were identified for 2007-2010. Correlation between observed and expected maximum daily humidex for each cell center point, on each given heat day, was checked and the coefficient of determination ( $R^2$ ) was calculated.  $R^2$  measures the proportion of variability shared between the two variables. The expected maximum daily humidex values for each cell point on each given heat day were calculated using the following formula:

$$e_{ij} = x_i \cdot \frac{x_j}{\bar{x}} \quad (1)$$

where  $e_{ij}$  is the expected maximum humidex for cell  $i$  on heat day  $j$ ;  $x_i$  is the observed maximum humidex for cell  $i$ , averaged over all heat days;  $x_j$  is the observed maximum humidex for heat day  $j$ , average over all cells; and  $\bar{x}$  is the grand mean maximum humidex, over all cells and all heat days.

We then took the observed average maximum humidex for each cell ( $x_i$ ) and modeled humidex distribution across space and time using ArcGIS Geostatistical Analyst tools. Specifically, a geostatistical layer was created using inverse-distance-weighted interpolation methods. The resulting product is a continuous surface layer representing average maximum humidex values on any given heat day. The geostatistical layer was then used to derive average maximum humidex values for each census tract. Humidex  $z$ -scores were also calculated for each census tract using the following formula:

$$z = \frac{x_i - \bar{x}}{\sigma} \quad (2)$$

where  $x_i$  is the average maximum humidex for cell  $i$ ,  $\bar{x}$  is the grand average maximum humidex over all heat days and all cell center points, and  $\sigma$  is the standard deviation with respect to the grand mean. Heat distribution, by census tract, was mapped using ArcGIS ArcMap software, version 10.1(ESRI 2012).

We then combined heat distribution and heat-vulnerable population information to create heat-risk maps illustrating expected cases per 1000 for each census tract. First, expected mortality counts for any given heat day were calculated using the following formula:

$$x_j = \bar{x} \cdot p_j \exp\{\widehat{\beta}_1 t\} \quad (3)$$

where  $x_j$  is the expected mortality count in census tract  $j$ ,  $\bar{x}$  is the average mortality count on non-heat days for the vulnerable population at risk, 2007-2010,  $p_j$  is the fraction of vulnerable population at risk in census tract  $j$ ,  $\widehat{\beta}_1$  is the vulnerable population's mortality estimate derived from Chapter 2, and  $t_j$  is the difference from threshold in census tract  $j$ . Next, we calculated the proportion of expected heat-day mortality cases for each census tract using the following formula:

$$\frac{x_j}{\sum x_j} \cdot 1000 \quad (4)$$

Where  $x_j$  is the expected mortality count in census tract  $j$ , and  $\sum x_j$  is the sum of expected mortality count over all census tracts within King County. Finally, the proportion of mortality risk, given a heat day, was mapped using ArcGIS ArcMap software, version 10.1(ESRI 2012).

## 5.3 Results

King County, located in Western Washington, is characterized by a relatively mild climate. Its summertime, 1980-2010, humidex values range from an average minimum of 6.7 °C (44.0 °F) to a maximum of 22.4 °C (72.3 °F). King is the largest county in Washington State and accounts for approximately 31% of the State's population. From 1980 to 2010 the county's population increased over 52%, with age groups 45-64, 65-84, and 85+ increasing 110%, 52.6%, and 150%, respectively (Washington State Office of Financial Management 2012).

### 5.3.1 Heat-mortality relationship and vulnerable populations

King County's historical heat-mortality relationship was defined using two different statistical methods. Our relative risk analysis quantified the excess mortality on a heat day compared to a non-heat day, while our time series analysis quantified the intensity effect of heat on mortality for each one degree increase in humidex over the threshold. We further characterized risk by age groups and subcategories of non-traumatic mortality. This study also explored effect modification from available individual-level characteristics.

Model-derived thresholds from both the relative risk and time series analyses were similar, at 36.1 °C (99<sup>th</sup> percentile) and 36.0 °C, respectively. Days where the average county-wide maximum humidex exceeded thresholds were considered heat-days by the respective analysis. We found a statistically significant increase in all-age, non-traumatic mortality with both analyses. The relative risk of death on a heat day was 10% [1.1 (95% CI: 1.06, 1.14)] greater than on a non-heat day, with risk increasing 2.12% (95% CI: 1.07, 3.19) for each degree increase in humidex above 36.0 °C. When stratifying non-traumatic mortality by age group, we found that heat increases the risk of mortality for the 65-84 age group by 6% [1.06 (95% CI: 1, 1.12)] on a heat day compared to a non-heat day, with risk increasing 0.91% (95% CI: -0.73%,

2.59%) for each degree increase in humidex above 36.0 °C. We also found the 85+ age group's risk of dying on a heat day is 18% [1.18 (95% CI: 1.11, 1.26)] greater than a non-heat day, with risk increasing 3.83% (1.85%, 5.86%) for each degree increase in humidex above 36.0 °C. Furthermore, when stratifying cause of death by age category, we found that a vulnerable population of non-elderly diabetic patients exists. For the 45-64 age group, we found that the relative risk of death from diabetes on a heat day was 78% [1.78 (95% CI: 1.12, 2.83)] greater than on a non-heat day, with risk increasing 14.22% (95% CI: 2.21, 27.64) for each degree increase in humidex above 36.0 °C. Lastly, the analyses of effect modification by individual-level characteristics did not identify additional vulnerable populations, aside from age. Both relative risk and time series analysis results can be found in Tables 2.3 and 2.4, Chapter 2 of this document.

### **5.3.2 Heat-risk maps**

For the purpose of this study, we chose the lower mortality threshold of 36.0 °C to identify extreme heat days during 2007-2010. This 4-year time frame was chosen because it represents the most recent years that health outcome data were available, while also being recent enough to control for large spatial changes that may affect heat distribution and render the maps unusable by public health practitioners. During 2007-2010, 23 heat days exceeded 36.0 °C. The overall coefficient of determination ( $R^2$ ) between expected and observed maximum humidex values was 70 %, indicating that our map will perform well in predicting heat distribution on an event basis. Average observed maximum humidex values for each cell center point were interpolated into a geospatial layer using ArcGIS ArcMap. The *Zonal Statistics as Table* tool was then used to calculate average maximum humidex values for each census tract. These values were standardized to a *z*-score and mapped (Figure 5.2). The *z*-scores reflect how much

cooler or hotter the particular census tract is predicted to be than the overall average, on any given heat day.

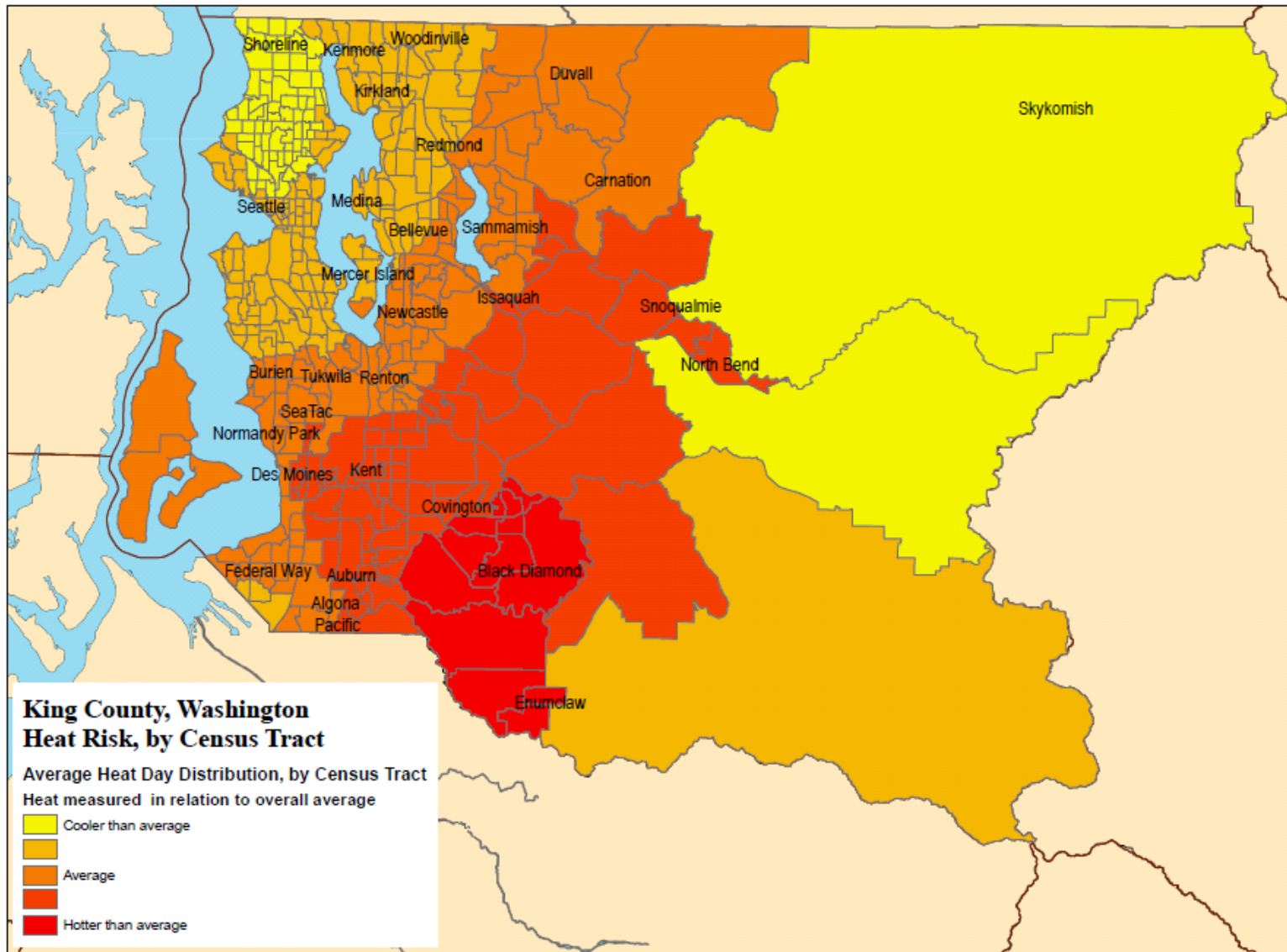
Our epidemiological analyses found that only age modified the heat-mortality relationship. Looking at the results in whole, we conclude that the primary vulnerable population was age 65+. The proportion of mortality risk per 1000 deaths was calculated and mapped for both all-ages and the vulnerable 65+ year-old age group (Figure 5.3 and 5.4, respectively). An additional vulnerable population, diabetics in the 45-64 year-old age group, was identified; however we were unable to map heat risk for this population due to the lack of census tract-level diabetes prevalence data.

#### **5.4 Discussion**

This project used the threshold derived from King County, Washington's historic (1980-2010) heat-mortality time series analysis to map average heat distribution for extreme heat days occurring 2007-2010. We then used heat-vulnerable populations identified in both the relative risk and time series analyses to create census tract-level heat risk maps. Our methods are unique, and to our knowledge, no other study uses local heat-health outcome relationships in their heat-risk mapping methodologies.

While still relatively few in number, existing studies that have mapped heat risk/vulnerability typically use findings from the literature to identify potentially vulnerable populations. For example, Ried et al. (2009) selected their vulnerability variables from epidemiologic literature to design a national map illustrating a heat vulnerability index, by county. Ten initial vulnerability variables were explained by four factors in their final model: social/environmental (including a combination of education, poverty, race, and green space),

Figure 5.2 Heat distribution z-scores, King County, Washington



**Figure 5.3** Heat-risk distribution - All-ages proportion of expected mortality per 1000 deaths given a heat day, by census tract

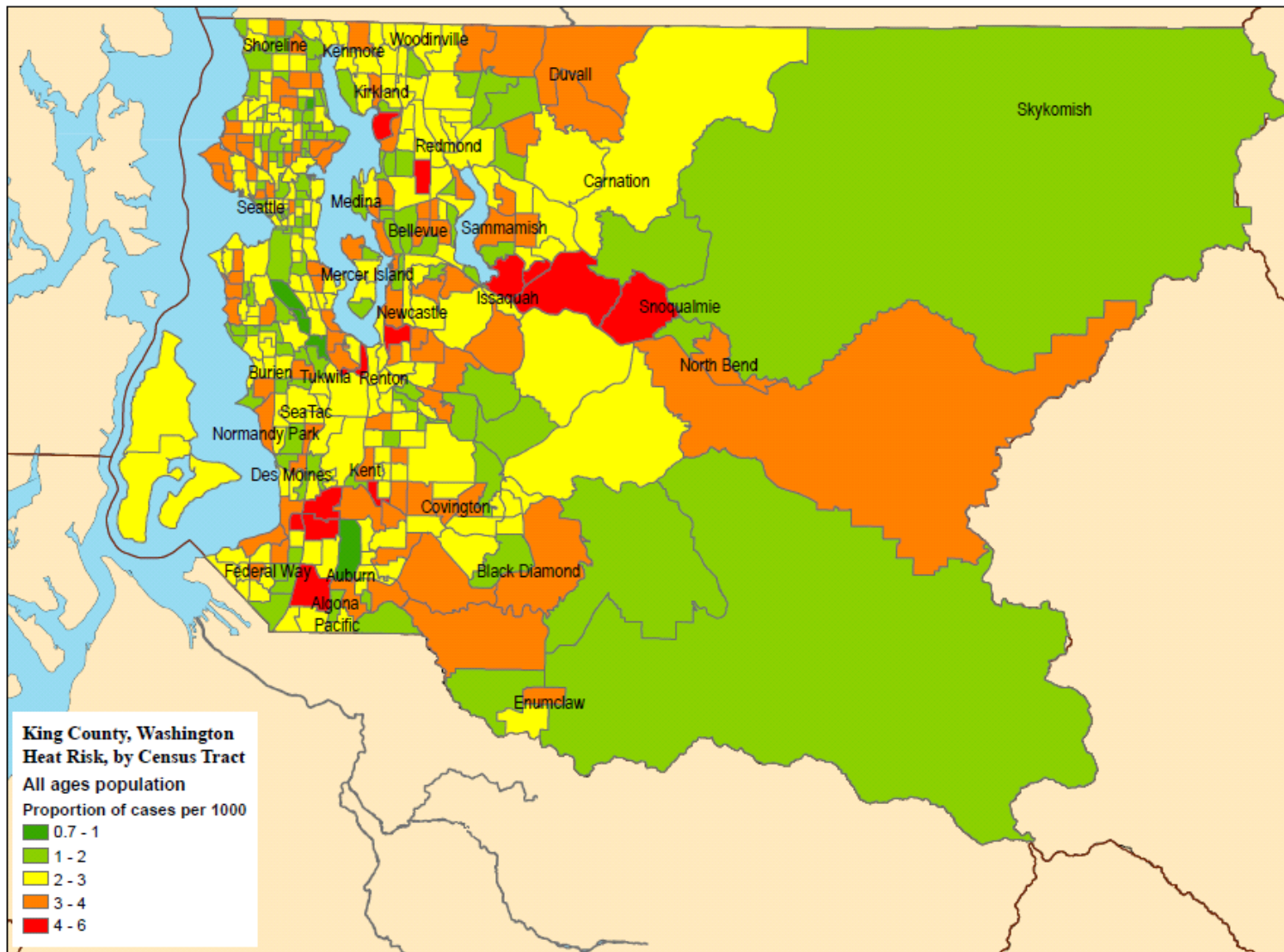
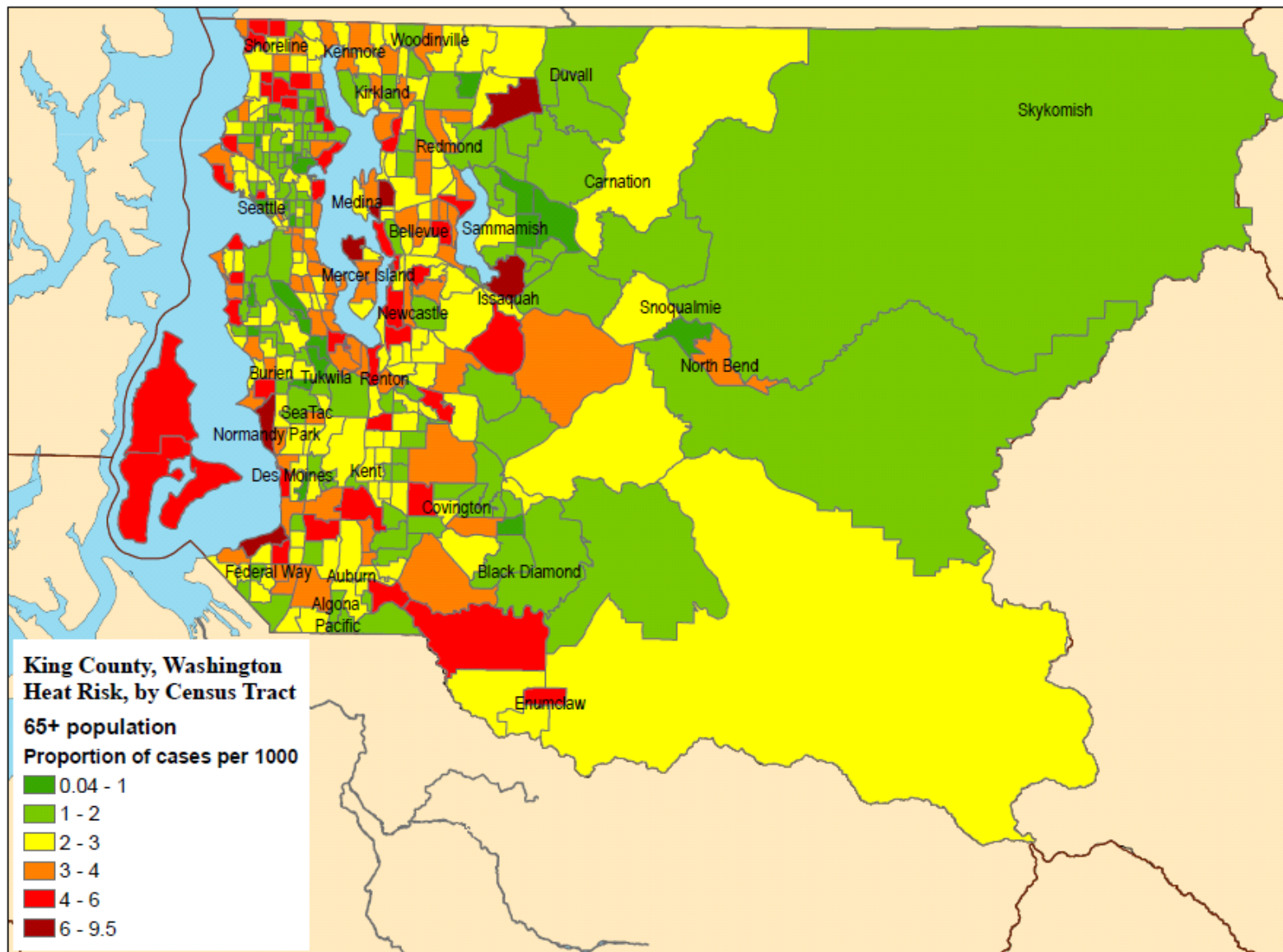


Figure 5.4 Heat-risk distribution - 65+ age group's proportion of expected mortality per 1000 deaths given a heat day, by census tract



social isolation, air conditioning prevalence, and proportion of elderly with diabetes (Reid et al. 2009). Later validation of their heat-vulnerability index found an increase in poor health outcomes for both heat and non-heat days, suggesting that their index may be predictive of health vulnerability in general (Reid et al. 2012). In comparison, our historical heat-mortality analysis explored the effect that age, gender, educational achievement, race, ethnicity, and social isolation (as measured by marital status) had on mortality rates. We found that none of these individual-level characteristics, aside from age, affected mortality rates in King County (Chapter 2). Furthermore, with King County's air conditioning prevalence rate, universally low at approximately 8% (Hamlet et al. 2009), the association between socio-economic status and access to air conditioning, a strong protective factor, (O'Neill et al. 2003, Naughton et al. 2002, Curriero et al. 2002) becomes less observable. We argue that by tailoring our heat-risk maps to local study findings, we are better able to map location-specific heat vulnerability.

A potential limitation of our study is that the individual-level characteristics used in our epidemiologic analyses may not have been adequate surrogates to identify heat-vulnerable populations. Furthermore, the inability to adjust individual-level data by population may have contributed to type II error. Other historic heat-mortality studies have found that individuals of Black race or non-White race (Medina-Ramon et al. 2006, O'Neill, Zanobetti, and Schwartz 2003, O'Neill, Zanobetti, and Schwartz 2005), those with less education (O'Neill, Zanobetti, and Schwartz 2003), lower socio-economic status (Anderson and Bell 2009), and who are more socially isolated (Naughton et al. 2002) are more vulnerable to heat-related mortality. However, all studies cited above, except Naughton et al. (2002), used the same unadjusted data source (death certificates) that we used for individual-level characteristic data, suggesting that our community is, in fact, different from previous study locations. While other mapping projects

have included these potential vulnerable populations, it is possible that by mapping non-statistically significant vulnerability variables, the resulting statistical noise masks true heat-vulnerability.

Another possible limitation of this study is that the epidemiological analysis used an average daily county-wide maximum humidex value to estimate heat's effect on mortality. Using an average value may result in exposure misclassification when a disproportionate number of cases are below or above the average value. We also did not consider available green space or urban heat island effects when creating the meteorological grid and assessing exposure, both of which can influence spatial distribution of heat (Stone, Hess, and Frumkin 2010, Tan et al. 2007). Improvement in exposure assessment could be obtained by using a population-weighted humidex value or by assigning each case a maximum humidex value interpolated from the closest meteorological grid center points. Indices of available green space and heat island effects could be included in our epidemiological methodology as potential effect modifiers of mortality. However, it should be noted that our use of maximum humidex, averaged over a meteorological grid, already improves upon most studies' use of only the closest airport meteorological data to calculate maximum exposure for the entire study area.

Further research is needed to refine our heat-risk maps to include other vulnerable populations with pre-existing disease conditions, such as diabetes. Our epidemiological analyses found that death from diabetic causes increased by 78% on extreme heat days for the 45-64 year-old age group. Currently, data for diabetes prevalence are available at a much coarser than census tract level. Public Health-Seattle King County is working on methodology to interpolate the available health reporting area data into census tract, or finer, geographical scale. Once the data becomes available, a heat-risk map can be created for the 45-64 year-old diabetic

population, using methods described in this chapter. Furthermore, as new vulnerable population data becomes available, existing maps can be easily updated or new heat-risk maps can be created.

## **5.5 Conclusion**

This study creates heat-risk maps for vulnerable populations residing in King County, Washington. These maps provide planners, public health practitioners, and emergency management professionals with visual tools to prepare for future extreme heat events. Whether used for locating cooling centers, directing planning efforts to increase tree canopy, or targeting specific vulnerable populations with behavioral interventions, these maps will provide spatial guidance that currently does not exist. Furthermore, mapping heat-vulnerability is another way to easily communicate climate change impacts to policymakers. Constructing our mapping methodology in a way that could be easily updated, as new information becomes available, has been an important research-to-practice goal for this overall dissertation. Incorporating census tract-level diabetes prevalence data is an important next step towards improving the understanding of heat-risk distribution in King County, Washington.

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## Chapter 6

### Communicating findings to the public health practice community

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## 6.1 Purpose

Climate change is a serious threat to the public's health and well-being (IPCC 2013; Balbus et al. 2008; Confalonieri et al. 2007). One of the direct and measurable impacts of climate change is an increase in extreme heat events, particularly in moderate climates (Curriero et al. 2002). Already, exposure to excessive heat kills more people than any other weather-related phenomenon (CDC 2012). Numerous studies report significant associations between extreme heat and increased mortality (Busch Isaksen et al. 2014; Anderson and Bell 2011; Jackson et al. 2010; Anderson and Bell 2009; Basu, Feng and Ostro 2008; Baccini et al. 2008; O'Neill, Zanobetti and Schwartz 2005; Naughton 2002; and Whitman et al. 1997) and hospitalization admissions (Ye et al. 2012; Fletcher et al. 2012; Ostro et al. 2010; Green et al. 2010; Lin et al. 2009; Knowlton et al. 2008; Kovats and Hajat 2008; Mastrangelo et al. 2007; Semenza 1999; and Semenza et al. 1999).

Climate change is predicted to increase the duration and intensity of extreme-heat events (Meehl and Tebaldi 2004), resulting in a significant burden on our existing public health resources. However, not all locations will be similarly affected by climate change or by heat's effect on health outcomes (IPCC 2013). Local climatic conditions coupled with unique environmental and population characteristics result in significant differences in heat-health outcome relationships (Yardley et al. 2010; Baccini et al. 2008; Kinney et al 2008; Davis et al 2004; Curriero et al. 2002). Additionally, the public health system's ability to respond to the challenge is predicted to influence outcomes (Patz et al. 2005, Climate Change Science Program 2008). An important step to reduce vulnerabilities and increase a community's resiliency is to place reliable and locally-specific information in the hands of public health practitioners and policymakers.

This dissertation has built on previous research efforts to identify heat-health relationships in Washington State, and to translate those findings for the public health community's use (Busch Isaksen et al. 2014; Hom 2013). The specific goals were to evaluate the impact of extreme-heat events on human health in King County, and to produce translational science products that provide King County public health officials with a solid basis for communicating the health risks of climate change. This chapter describes these products, how they were developed, and the lessons learned from translating the dissertation's findings into audience-appropriate communication materials. These tools are intended to increase the likelihood that the data will be used to support climate change-related mitigation and adaptation programs and policies in King County.

## **6.2 Process**

### **6.2.1 Formative research activities**

Formative research data were collected during a previous Centers for Disease Control and Prevention-funded heat-health project (Busch Isaksen et al. 2013). This included data on local climate change-related activities, stakeholder knowledge/attitudes, and political currents. Relevant news articles and county documents were reviewed, the county's government websites were consulted, and public health officials were questioned about their goals and about perceived support for/opposition against climate action, within the organization and in the larger community.

Public Health-Seattle King County (PHSKC)'s primary goal was to be able to collaborate with other county departments engaged in climate change mitigation activities. In other words, PHSKC wanted a seat at the county's climate change discussion table. The local, climate change-related public health impact data was seen as helping to legitimize Public Health's role within

the larger climate change community. In addition, key informant interviews with representatives from other county departments were conducted to determine how PHSKC might engage these departments. In these interviews, we explored whether health-related/climate change messaging could support existing climate change mitigation policies and programs. The key informant interview results (Table 6.1) reflect the wide variety of questions that other county representatives had regarding public health impacts from climate change.

### **6.2.2 Collaboration**

Involving practitioners early on in the research process established collaborative relationships and allowed for research activities to be adjusted or tailored to meet the practice world's needs. Several meetings were held (in person, webinar, and video conference) to: orient practitioners to the research collected; explore interest in the study and intended use of research findings; share findings and get feedback on additional data needs and formats; identify other audiences for the findings; and plan presentations. A good example of how this collaborative process added to the dissertation results is the suggestion from PHSKC staff that we explore impacts specific to the diabetic population. During a presentation detailing the CDC study findings, staff members inquired as to whether we could look at the diabetic population's response to heat. Unfortunately, for that research project, there was no time to re-analyze the data. However, in the dissertation research, we made sure to include diabetes and renal outcomes in both our mortality and morbidity analyses. As a result, we were able to observe that, in the 45-64 year-old age group, the relative risk of death from diabetes on a heat day was 78% greater than on a non-heat day, with risk increasing 14.22% for each degree increase in humidex above 36.0 °C. Furthermore, we found that the same age group has a 76% increased risk of hospitalization from nephritis and nephrotic causes, and a 99% increased risk from acute

**Table 6.1:** King County Key Informant Interview Questions and Responses, n=11

<b>Question</b>	<b>Response</b>
<i>What are your specific concerns regarding the human impacts from climate change?</i>	Absence of cooling centers and AC
	Elderly
	Poor, low income, homeless
	Asthma and allergy problems
	Ethnic minorities, new immigrants/limited English proficiency
	Residents of flood-prone areas - flood plains and coastal
	New illnesses related to heat and flooding
	Decreasing food sources and water in the summers
	People in poorer health
	Manual and outside laborers
	Ozone level leading to more skin cancer and poorer indoor air
	Fire potential
	Children
	Those dependent on well water
<i>How could information on the projected local health effects of climate change be useful?</i>	Raise awareness among city and county council members
	Influence county policies about employee work conditions
	Influence county planning, decision making, prioritizing, as well as details of specific projects
	Influence citizens to engage in mitigating behaviors and to vote for the right people
	Provide a single source of new information on mitigation and climate change impacts
<i>What specific information interests you?</i>	Which neighborhoods will be most affected by climate change?
	What are the numbers of vulnerable people and where do they live?
	Where are the biggest heat and air quality problems?
	What increases in morbidity and mortality can be expected? How do we know that these are related to climate change?
	What strategies will reduce carbon emissions? (zoning, building transportation policies)
	What about migration?
	What can we expect in terms of flooding and water quality?
	What is an example of how a policy could potentially reduce projected morbidity and mortality and by how much?
<i>Who should the scientific data target and how?</i>	King County Board of Health
	City Council
	County Council
	Other decision makers
<i>Other comments</i>	Use different formats for different audiences
	Use as many formats/channels as possible
	Provide concrete examples, e.g., how a policy could have positive effects
	Time the information -- pay attention to what's going on that could affect how seriously people take the information
	Make only statements that you're confident about
	Focus on the Executive's priorities and on news-making events

renal failure on a heat day compared to a non-heat day, with risk increasing by 9.5% and 9.2%, respectively, for each degree increase in humidex above 37.4 °C.

### **6.2.3 Research translation**

Translating local study results into policy action requires effective communication methods. Maibach et al. (2010) argue that there is a need to frame climate change communication as a public health problem and that the public health community can be an important part of the solution. Nerlich, Koteyko, and Brown (2010) encourage the use of tools that include dialogue and audience engagement. In collaboration with PHSKC, it was decided that presenting the data in slide deck format was the best way to convey the main research findings, while promoting dialogue and audience engagement. Using PowerPoint®, initial research results were presented to practitioners. Comments and general feedback regarding the visual representation of data were noted during each meeting and used to further refine the slide deck. The finalized PowerPoint® was formatted as a stand-alone presentation; information included in the notes pane feature was designed to support practitioners' understanding and their communication of the findings to others. While PowerPoint® is typically used as a visual accompaniment to a speaker's oral presentation, its use has expanded to a stand-alone communication tool. The value of this type of tool, if designed properly, is that it broadens the access to material without the need to attend a presentation or sift through a dense report (Farkas 2005).

The use of imagery in climate change communication is common, as recently reviewed by O'Neill and Smith (2014). The authors found that the use of images is an important tool to communicate time (historic and future response visualization) and to portray 'truths.' They argue that scientific images, while ethically constructed, become attached to the common narrative of

fear, symbolism and myth when consumed by the audience (O'Neill and Smith 2014). Bostrom, Anselin and Farris (2008) emphasize the need for these tools to achieve effective visual representation of numbers and statistics.

Previous King County heat-health study results have been reported in bar chart format (Figure 6.1). However, this type of display does not convey the proportionality of a significant subcategory cause of death within all non-traumatic deaths, and it does not allow for the full comparison of mortality burden. In other words, in Figure 6.1, there is no way to relate the total burden of circulatory compared to respiratory mortality on a heat day; we just know that (for all age groups) there is a greater increase in respiratory causes of death for each degree increase in humidex above 36.0 ° C (4.4%), than for circulatory causes (2%). When trying to target vulnerable populations with shrinking public health funds, it could be valuable to know which causes of death account for the larger burden of mortality and morbidity. Figure 6.2 shows how the proportion of mortality on a heat day can be illustrated. The size of the bubble represents the proportion of all non-traumatic deaths. At a glance the viewer is able to assess estimated % risk increase (y-axis) for each cause-of-death category (x-axis), while also being able to distinguish between the higher risk of mortality from respiratory causes and the higher proportion of deaths attributed to circulatory causes.

Additionally, GIS mapping tools offer a visual way to display health risks and vulnerable population characteristics and can be an effective means to engage planners, policymakers and the public at large (Graham et al. 2011). This dissertation improved upon past risk communication efforts by mapping King County, Washington's average heat distribution for extreme-heat days occurring 2007-2010. We then used heat-vulnerable populations, identified in the dissertation's epidemiological analyses, to create census tract-level heat-risk maps. These

Figure 6.1 Visual presentation: Results using bar charts

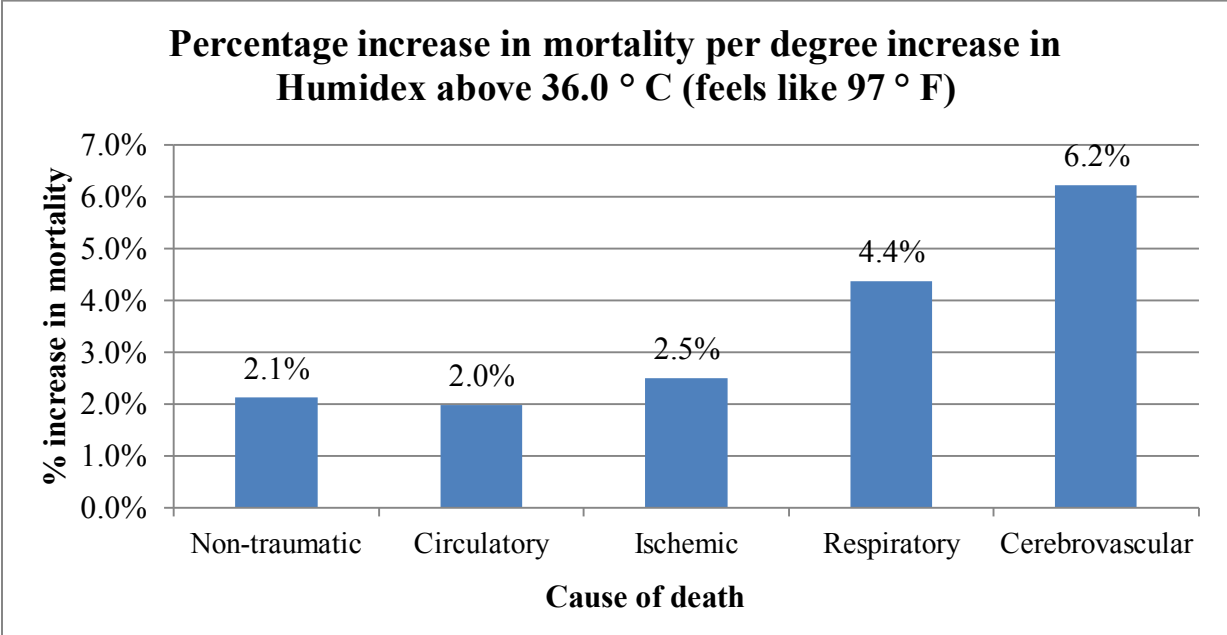
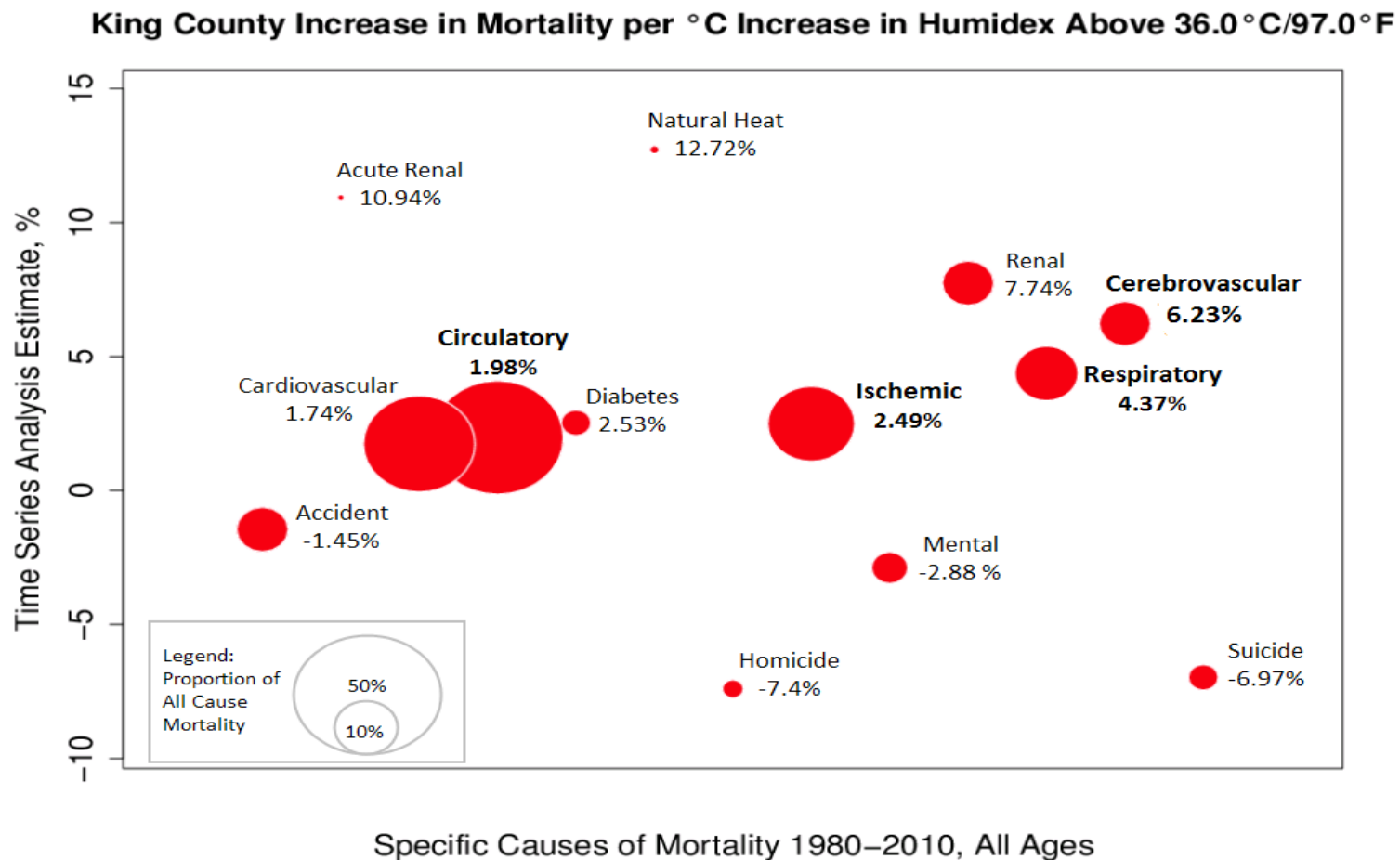


Figure 6.2 Visual presentation: Results using proportional bubble graphics, by cause of death<sup>1,2</sup>



<sup>1</sup> Statistically significant results are in bold.

<sup>2</sup> Proportionality represents the proportion of all-cause mortality for all ages.

maps were added to the county's PowerPoint© slide deck, and the note panes were used to describe the findings.

#### **6.2.4 Process evaluation**

We sought feedback about the information and tools provided to Public Health - Seattle King County throughout the research process. While feedback for this dissertation's communication methods was more informal, the initial heat-health relationship study sought comments using survey and structured group discussion methods to examine: the strengths and weaknesses of the collaborative process; the practitioners' use of the research findings; and factors that facilitated or hindered use. During one structured conference call, county partners agreed that the slide presentations provided useful information that they intended to present to others. They commented that having local information about heat trends/projections and health outcomes gave them a way to talk about current and anticipated local impacts and participate in interdepartmental county discussions on climate change. They also felt more comfortable taking the lead on climate change and developing partnerships for mitigation and adaptation activities. As one health official put it, "We've been saying that health should be part of this. This research gives us the data to back that."

Additional feedback about the presentation's effectiveness was collected via an online survey. The results are presented in Table 6.2. A total of 7 practitioners responded to the presentation survey. All participants strongly agreed (57%) or agreed (43%) that the presentation provided useful information regarding heat-related outcomes. Eighty-six percent strongly agreed or agreed that the content of the slides answered all their heat-health questions. All participants strongly agreed or agreed that they felt comfortable using the notes pane to discuss the findings internally, while 28% of participants were neutral or disagreed that they felt comfortable using

the notes pane to discuss the findings with external groups. Finally, 24% either disagreed or were neutral when asked whether the notes pane readability/use of jargon was appropriate for them.

The feedback gathered from this evaluation informed the design of this dissertation's PowerPoint© slide deck. Once again, slides were created to stand alone and to include additional information in the notes pane; however, special care was taken to fully define technical jargon and new concepts. Results were displayed in both bar chart and proportional bubble graphic formats, and heat-risk maps were included for their most vulnerable population, the 65+ age group. The updated slides were shared with PHSKC, and informal feedback was solicited. Practitioners preferred the proportional bubble graphic slides over the bar charts for displaying results, and they requested that the remaining results be converted to display proportionality. The heat-risk maps were also well received, and practitioners requested that additional information describing the application of these maps be included in the notes pane. PHSKC staff stated that the notes for the majority of the slides were invaluable and helped them discuss the information correctly with others. They asked that a "bottom line" statement be added to each slide, as well as a "definition" section for unfamiliar terminology.

Additional written feedback on the proportional bubble graphs was obtained during a climate change workshop held at the Washington State Public Health Association's Joint Conference on Health, October 14, 2013. Of approximately 25 people viewing slides of the proportional bubble graphs, seven commented on their use. Six stated that they preferred this type of presentation over the typical bar chart or table formats, while one individual commented that he preferred a table with confidence intervals over a more visual representation of the data.

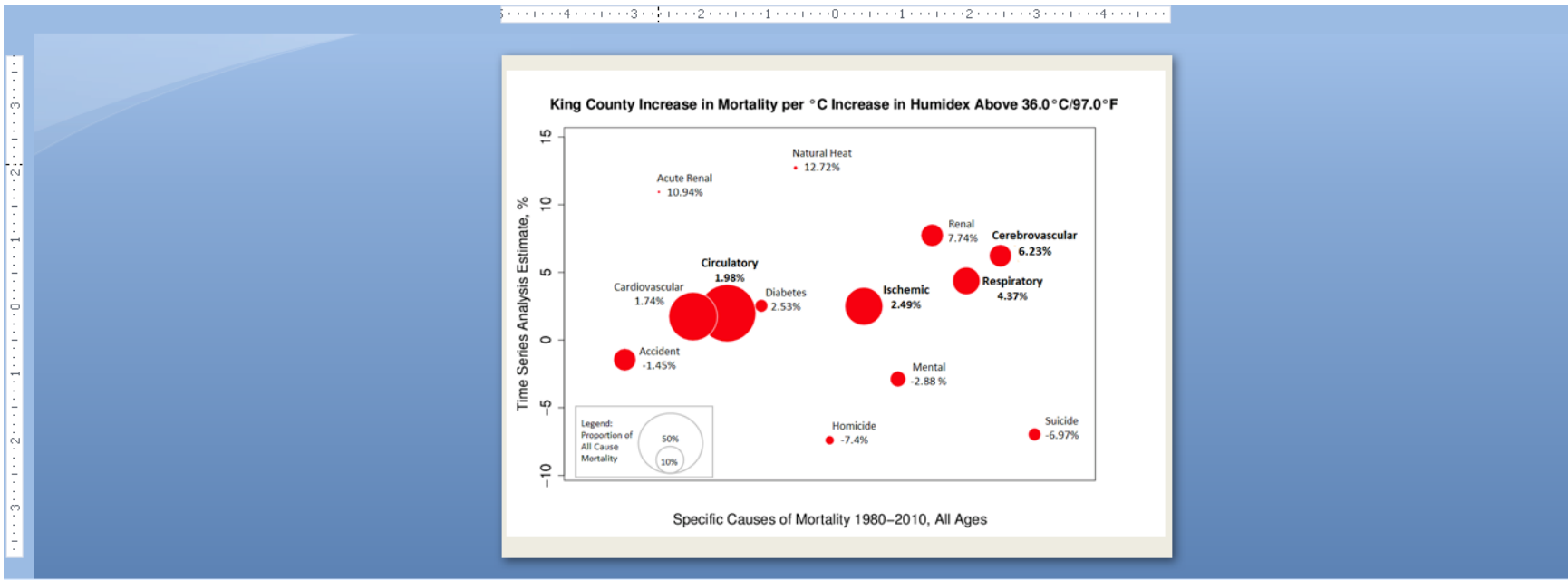
**Table 6.2:** PowerPoint© evaluation survey (n=7)

<b>Question</b>	<b>Strongly agree</b>	<b>Agree</b>	<b>Neutral</b>	<b>Disagree</b>	<b>Strongly Disagree</b>
1. Overall, the PowerPoint© presentation (PPP) provided useful information.	57%	43%	0%	0%	0%
2. The slides were well balanced between graphics and written text.	33%	67%	0%	0%	0%
3. The slides successfully answered my heat-related mortality/morbidity outcomes questions.	29%	57%	14%	0%	0%
<i>The following questions specifically pertain to the <u>notes pane</u></i>					
4. The PPP notes pane, containing additional explanation/information, is a helpful tool to further my understanding about the findings.	57%	43%	0%	0%	0%
5. The PPP notes pane’s readability/use of jargon is appropriate for me.	47%	29%	14%	10%	0%
6. The PPP notes pane content is (will be) helpful to me when presenting the findings to <u>internal</u> groups.	57%	43%	0%	0%	0%
7. The PPP notes pane content is (will be) helpful to me when presenting the findings to <u>external</u> groups.	72%	0%	14%	14%	0%
8. The supplemental information provided in the notes pane increases my comfort/confidence level when presenting our findings to interested partners.	57%	43%	0%	0%	0%

### **6.3 Communication Product**

A single PowerPoint© file was provided to PHSKC containing the results for both the mortality and morbidity analyses. The notes pane for each slide was used to describe the results and to provide bottom-line meanings and definitions needed for practitioners to correctly interpret the data and present it to others. Figure 6.3 is an example of a results slide with notes pane. A total of seven different proportional bubble graphics were made to display each set of statistically significant findings for mortality and morbidity heat effects. Two additional examples are provided in Figures 6.4 and 6.5. Additionally, heat-risk maps locating heat-vulnerable populations were included in the final presentation (Chapter 5, Figures 5.3 and 5.4).

**Figure 6.3** Notes panel use in a stand-alone PowerPoint© presentation



**How to explain the graph: Stratified by Cause of Mortality, All Ages**

The statistically significant Time Series Analysis estimates shown in this graphic are Circulatory, Ischemic, Respiratory and **Cerebrovascular** causes of death. All Causes and Non-traumatic causes are also statistically significant but are not shown in this graphic as their proportion to the others renders several causes of death too small to visualize.

The bubble's size is proportional to All Causes of death; Circulatory = ~40% of all causes of death for all ages on a day above the threshold of 36.0° C.

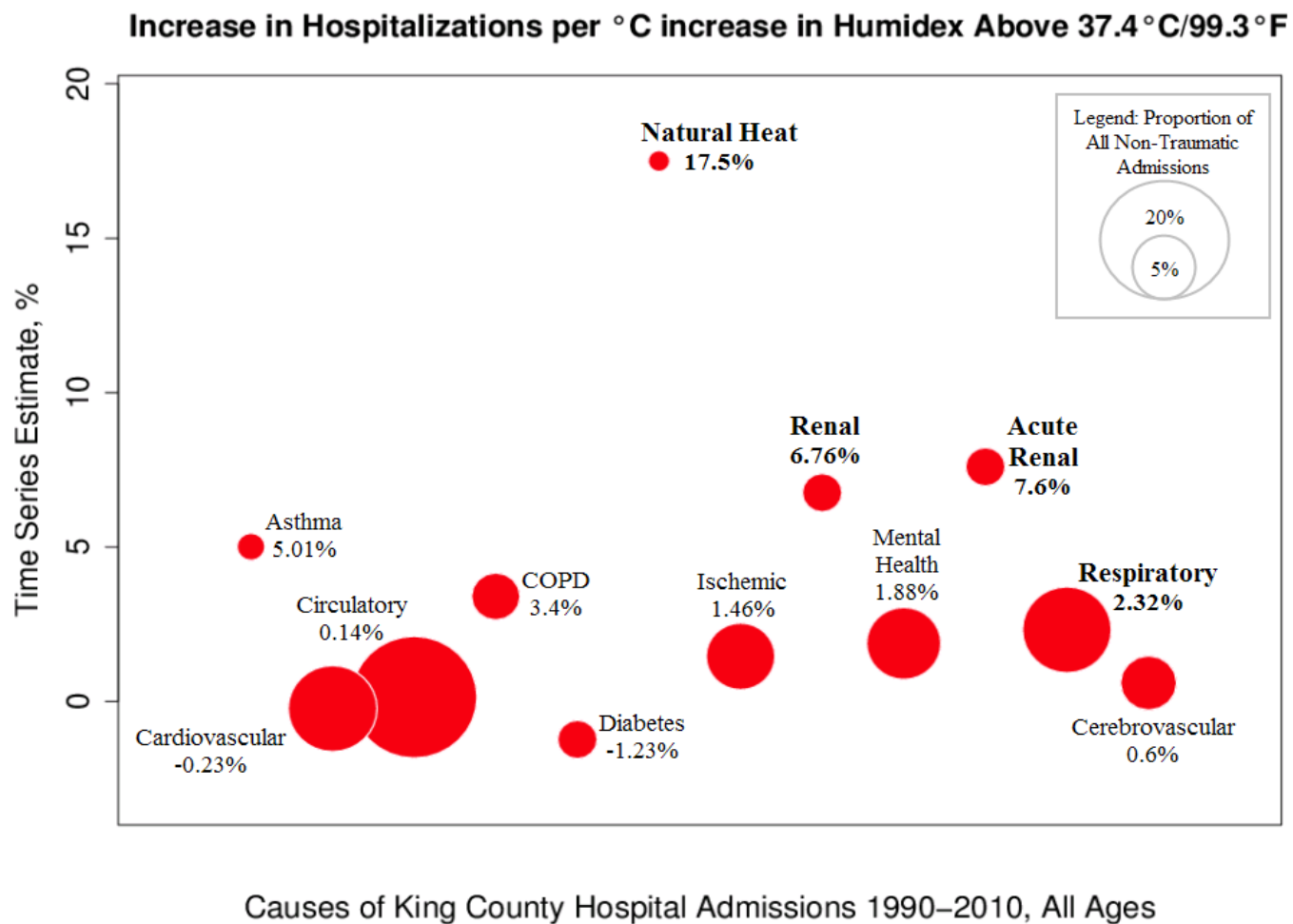
**How to read the graphic:**

Mortality from ALL causes, for all ages, increases, on average, 1.69% (0.69%, 2.7%)\* for each 1 degree increase in humidex above 36.0° C. (not shown in this graphic)

Mortality from all non-traumatic causes, for all ages, increases, on average, 2.12% (1.07%, 3.19%)\* for each 1 degree increase in humidex above 36.0° C. (not shown in this graphic)

Mortality from Respiratory causes, for all ages, increases, on average, 4.37% (1.11%, 7.74%)\* for each 1 degree increase in humidex above 36.0° C.

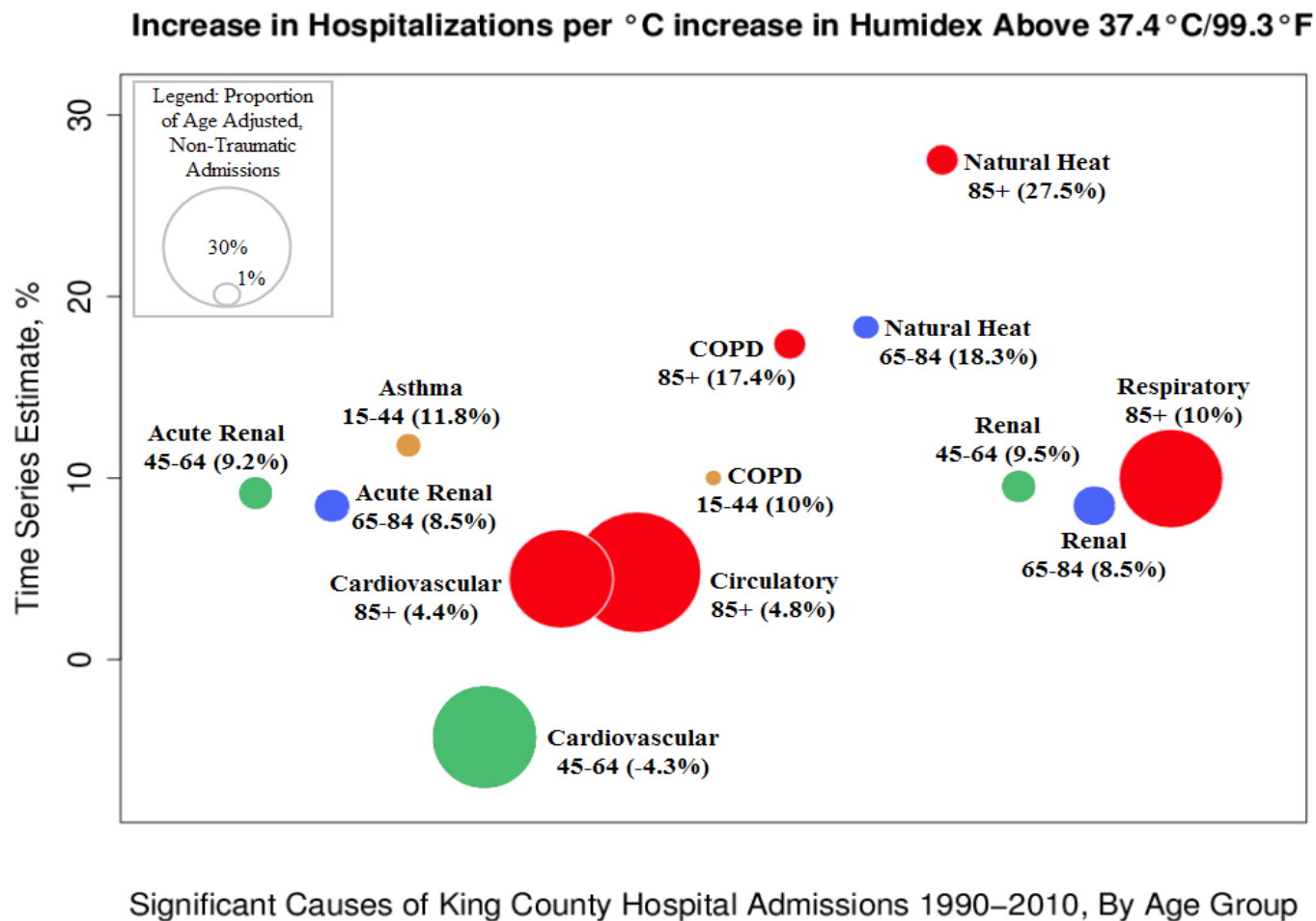
**Figure 6.4** Visualizing proportional hospitalization risk, by cause of admission<sup>1,2</sup>



<sup>1</sup> Statistically significant results are in bold.

<sup>2</sup> Proportionality represents the proportion of non-traumatic hospitalizations for all ages.

**Figure 6.5** Visualizing proportional hospitalization risk, by statistically significant age group and cause of admission<sup>1,2,3</sup>



<sup>1</sup> All results are statistically significant.

<sup>2</sup> Each age group is noted by a different color: red=85+ age group, blue=65-84 age group, green=45-64 age group, and gold=15-44 age group.

<sup>3</sup> Proportionality represents the proportion of non-traumatic hospitalizations for the particular age group.

## 6.4 Conclusion

PHSKC's primary goal was to focus on climate change mitigation and build collaborations with other county departments that were positioned to develop mitigation policies and programs (e.g., Department of Transportation, Department of Natural Resources and Parks, etc.). Presentation and discussion of findings with staff from other county departments articulated why public health should be involved in climate change discussions, demonstrated the potential power of health consequences in climate change messaging, and laid some groundwork for future cross-department collaborations. The PHSKC team has worked with other King County officials to build support for climate action and to incorporate the research findings into preparedness planning.

Several anticipated next steps have been identified. These include: a pilot project to record and understand community perceptions and differences with regard to climate change and extreme heat preparedness; an analysis of emergency response data for heat-related calls; and an increase in advocacy for community-level interventions (e.g., increase in cooling centers and tree canopy). Additionally, the results from this dissertation will be used by PHSKC as they update their King County Health and Human Services Transformation Plan. This is a five-year strategic plan that charts the path to improving health and human services for King County (King County Health and Human Services 2013). It is hoped that our locally derived heat-health findings not only substantially contribute to the Transformation Plan, but also help PHSKC secure an all-important seat at the climate change discussion table.

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## **Chapter 7**

### **Discussion and Conclusion**

The overall objective of this dissertation was to test the methods used to model and interpret historic extreme-heat effects on mortality and morbidity in King County, Washington. A concurrent goal was to translate the epidemiological findings into materials that are understandable and usable by the public health practice community. The body of work contains 12 separate epidemiological analyses; both relative risk and time series models were used to explore extreme-heat effects on mortality and morbidity outcomes for calibration (1980-2006 mortality/1990-2006 hospital admissions), validation (2007-2010), and complete (1980/1990-2010) time frames. Results from these analyses were used to test a piece-wise linear approach to interpreting heat's effect on health outcomes, provide updated heat-risk estimates, and produce localized heat-risk maps. The findings of this study can be used to prepare for extreme-heat events in King County, Washington, and to support the broader public health discussions on climate change.

#### **7.1 Model validation**

We found that our method of using a piece-wise linear function to describe heat effects on health outcomes performed well overall. However, at the lower bound of the expected death count, we found that mortality is significantly under-predicted. Additionally, when comparing time frame estimates, we found a statistically significant difference between the calibration estimate and the validation's hospitalization model-derived estimate. Reasons for the model's conservative performance may be that the validation time frame is relatively short compared to

the calibration time frames and/or that the validation time frame includes the most extreme heat event on record. When comparing the entire study time frame thresholds and estimates, as reported in Chapter 2 and 3, to our calibration and validation time frames, we found that the overall thresholds and estimates are very similar to that of the calibration/forced validation results. This finding suggests that the 2009 extreme-heat event is affecting the validation time frame's model-derived threshold and subsequent outcome estimates. A sensitivity analysis that removed the most extreme heat event from the data might provide additional information as to the model's fit; however it may come at a cost to the validation study's power. As an alternative, we could use a cross-validation approach to test the use of a piece-wise linear function, thereby using data from the entire time frame for both estimation and validation.

The purpose for interpreting an otherwise non-linear relationship (using a piece-wise linear function) is to increase the understanding and reporting of meaningful results to public health practitioners and policymakers (Lin et al. 2012; Lin et al. 2009; Armstrong 2006). Even when using a spline over temperature to summarize heat effects on health, authors choose to present the results in a way that the audience can understand. Varying methods include: comparing changes in risks of relative percentiles (Gasparrini and Armstrong 2011; Anderson and Bell 2009; Medina-Ramon and Schwartz 2007), estimating average changes in risk per 10 °F increase in heat above a threshold (Green et al. 2010; Basu, Feng and Ostro 2008; Curriero et al. 2002) and comparing heat wave day/event risk against non-heat wave day/event risk (Knowlton et al. 2008; Mastrangelo et al. 2007).

Scientifically, it may seem to be more appropriate to model heat's effect on health outcomes with a complex spline. However, Hurley et al. (2004) found that the more complex models were not always appropriate for the situation, especially when prediction was of interest.

One important use of an absolute threshold is to initiate a local heat-health warning system. In King County, the Public Health-Seattle King County's (PHSKC) Preparedness Section receives broadcasted National Weather Service (NWS) alerts based on temperature thresholds set by the NWS. When a heat watch or warning is issued, it is forwarded through PHSKC's communications network to healthcare organizations and to hundreds of community-based organizations across the county. PHSKC's local emergency management partners also broadcast alerts widely across their own jurisdictions (Loehr 2012). Without a heat-health alert threshold and subsequent public health preparedness action, the public may experience substantial adverse health effects demonstrated in Chapters 2 and 3 of this dissertation.

In fact, the efficacy of the NWS alert system has been studied not only in the United States (Kalkstein et al. 2011; Alberini, Mastrangelo and Pitcher 2008; Ebi et al. 2004) but also worldwide (Hajat et al. 2010). Ebi et al. (2004) evaluated the effect on observed mortality from the use of Philadelphia's Heat-Health Warning System (HHWS), and found that 2.6 fewer people died on days that a warning was issued than not issued. Kalkstein et al. (2011) evaluated 40 major cities and found that, since 1995, when the NWS guidelines were broadly implemented, great reductions in extreme heat-related mortality have occurred. Alberini, Mastrangelo and Pitcher (2008) found a 25% reduction in mortality when heat alerts were issued versus days without alerts. Finally, Hajat et al. 2010 found that among the four types of heat-health warning systems they evaluated, all identified excess mortality. They also found that the heat-health system using a temperature-mortality relationship identified the most days with excess mortality than days identified by the other three approaches.

## 7.2 Mortality and morbidity analysis

This dissertation also reanalyzed heat effects on mortality and morbidity using all data years available (1980-2010 mortality and 1990-2010 morbidity). The epidemiological analyses demonstrated that all non-traumatic mortality and hospital admissions associated with extreme heat increased significantly in all ages, and that the risk increased with heat intensity - especially for the older populations. The relative risk of mortality on a heat day (above the 99<sup>th</sup> percentile) was 10% greater than on a non-heat day, with risk increasing 2.12% for each 1 degree increase in humidex above 36.0 °C. The relative risk of hospitalization on a heat day (above the 99<sup>th</sup> percentile) was 2% (non-significant) greater than on a non-heat day, with risk increasing 1.59% for each degree increase in humidex above 37.4 °C. Our results are consistent with those studies whose methodologies were similar to ours: exploring heat effects on mortality above a relative threshold (Medina-Ramon et al. 2006; Medina-Ramon and Schwartz 2007; Jackson et al. 2010) and using a piece-wise linear function to estimate changes in risk for each additional 1 °C unit of heat above a defined threshold (Busch Isaksen et al. 2014; Lin et al. 2012; Lin et al. 2009; Baccini et al. 2008; Medina-Ramon and Schwartz 2007; Armstrong 2006; Kovats, Hajat, and Wilkinson 2004).

The epidemiological relative risk analyses identified several unexpected results, including an increased risk of mortality from diabetes (78%) for the 45-64 age group, and from cerebrovascular (37%), mental health (43%), and accidental (43%) causes for the 65-84 age group. When considering our morbidity analyses, we found unexpected results for nephritis and nephrotic causes of admission for the 45-64 (76%), 65-84 (60%), and 85+ (49%) age groups and for acute renal failure for the same age groups, 99%, 67%, 55%, respectively. Additionally,

exposure to natural heat increased hospital admission risks for all but two age groups: 0-4 and 5-14.

Unexpected results were also discovered with the time series analyses. We found increases in heat intensity effects per degree increase in humidex above the threshold for diabetic mortality (14.2%) in the 45-64 age group, COPD and asthma hospital admissions (10.0% and 11.8%, respectively) in the 15-44 age group, nephritis and nephrotic admissions (9.5% and 8.5%, respectively) for the 45-64 and 65-84 age groups, acute renal failure admissions (9.2% and 8.5%, respectively) for the 45-64 and 65-84 age groups, and natural heat-exposure admissions (18.3%) for the 65-84 age group. Lastly, we found a decreased risk of cardiovascular hospitalizations per degree increase in humidex above the threshold for the 45-64 year-old age group.

### **7.2.1 Vulnerable populations**

Aside from age, this study did not find that the individual covariates investigated for mortality (gender, race, high school graduation, marital status, Hispanic origin, and tobacco use) or morbidity (gender, admission source, admission type, and primary payer) modified the effect of heat on death or admission counts. Our results are different from other studies that have found effect modification from race (Fletcher et al. 2012; Medina-Ramon et al. 2006; O'Neill, Zanobetti and Schwartz 2003; O'Neill, Zanobetti and Schwartz 2005), socio-economic status (Lin et al. 2012; Portier 2010; Anderson and Bell 2009; Hajat et al. 2005), educational achievement (O'Neill, Zanobetti and Schwartz 2003; Jones 1982), and social isolation (Naughton 2002; Klinenberg 2002). However, even among all these aforementioned studies, there are considerable differences in which populations were defined as vulnerable and what their corresponding effect modifications were on mortality and morbidity.

Differences in vulnerable population findings have led several studies to suggest that *place* matters. Yardley, Sigal, and Kenny 2010, suggest that the spatial distribution in heat-related mortality indicates more at play than just temperature and physiology, and that the addition of neighborhood or area-level characteristics would further explain local heat-risk differences. For example, Harlan et al. (2006) found that microclimates, or urban heat-islands, affect mortality risk and that lower socio-economic status groups were more likely to occupy these areas. Davis et al. (2003) found that communities concentrating on planning improvements to increase shade and access to water decreased their heat-related mortality. Finally, Stone, Hess and Frumkin (2010) found that metropolitan sprawl affects the rate of increase in annual number of extreme-heat events. In addition to community infrastructure, it is believed that the social connectedness of *place* affects heat-related health risks. Smoyer 1998; Klinenberg 2002; and Naughton 2002 have all found that social connections, perception of safety and the community's walkability affect heat-mortality. Klinenberg (2002) attributes differences in the community's social environment to findings from the Chicago 1995 heat wave, where mortality rates for African-Americans were significantly lower than those found for Whites.

In King County, the lack of identified vulnerable populations could be a result of our selection and use of individual-level data. It is possible that the individual-level characteristics used in our epidemiologic analyses did not adequately identify heat-vulnerable populations, or that the inability to adjust by population count contributed to type II error. Using other sources of data to estimate vulnerable population count, by year, would reduce the possibility of type II error; however it would not address whether our individual-level data were adequate surrogates of vulnerability. For example, the level of education listed on death certificates was used as the mortality surrogate for socio-economic status. An alternative way to determine socio-economic

status would be to use neighborhood-level average real-estate appraisal data. Since mortality data is geo-coded, it allows for the calculation and use of residential-specific data, but this is not the case with morbidity data, which is spatially available only at the hospital location-level.

Alternative explanations for the lack of identified vulnerable populations in this study could derive from our region's low prevalence of air conditioning and/or other characteristics of specific neighborhoods. The availability and use of air conditioning is considered the strongest factor in preventing heat-related mortality (O'Neill, Zanobetti, and Schwartz et al. 2003; Davis et al. 2003; Naughton 2002; Curriero et al. 2002). It is possible that our region's general lack of residential air conditioning (Hamlet et al. 2009) is masking an otherwise noticeable vulnerable population. Additional research pertaining to the use of air conditioning and other types of cooling methods is needed in order to disentangle the relationships in our area. Adding data pertaining to tree canopy coverage and distance to shore lines or major water bodies would also improve the understanding of the modifying effect neighborhood-level characteristics have on mortality.

### **7.2.2 Contrasting and comparing mortality and morbidity results**

This dissertation is one of a few studies that jointly investigate mortality and morbidity outcomes using two different types of analyses. Concurrent investigations grant a unique opportunity to compare and contrast risk, and to identify patterns and connections between health outcomes and extreme-heat exposure. Kovats, Hajat, and Wilkinson (2004) analyzed six years of data for Greater London, UK, and found results similar to ours, where the relative risk of mortality statistically significantly increased by 10.8%, while unplanned hospital admissions increased by a non-statistically significant 1.9%. Their time series analysis used a similar linear fit to explain heat intensity effects on mortality (3.3% increase for each degree increase above

21.5 °C) and hospital admissions (no statistically significant increase). As in our study, they found respiratory and renal causes of hospital admissions increased significantly with heat exposure. The considerable contrast between mortality and hospitalization results led the authors to conclude that “many deaths occur rapidly or in isolated people before they come to anyone’s attention,” and that, in comparison, hospital admissions are less affected by exposure to heat (Kovats, Hajat, and Wilkinson 2004). Mastrangelo et al. (2006) explored this theory and reported that some conditions specifically related to circulatory causes, rapidly lead to death before hospitalization can occur. This theory has since been echoed by Michelozzi et al. (2009) and Linares and Diaz (2008). The authors recommend a concurrent investigation of mortality and hospital admission outcomes associated with extreme-heat exposure.

Overall, our findings support Mastrangelo et al.’s (2006) theory; there is considerable contrast between all-ages circulatory mortality and hospital admission results, suggesting that those at risk for circulatory events are more likely to die rather than be admitted to a hospital. However, this dissertation also found that the 85+ year-old age group experienced increases in circulatory hospital admissions associated with increasing heat intensity, even though statistically significant increases in admissions were not seen when comparing a heat day to a non-heat day. With increasing heat above threshold, increased rates of circulatory (4%) and cardiovascular (4%) deaths paralleled the observed increased rates of circulatory (5%) and cardiovascular (4%) hospital admissions. Our results highlight the particular vulnerability of the elderly, 85 + age group to heat intensity effects on adverse circulatory and, its subcategory, cardiovascular health outcomes. More research is necessary to discern the factors that influence why part of this population experiences a rapidly acute event resulting in death, while others are hospitalized. Death certificate data could be analyzed for place of death, to determine the

proportion that occurred in hospital. This analysis would begin to answer the question of whether or not we are seeing the same population reflected in both the mortality and morbidity findings. Lastly, supplemental individual-level data could be collected with a survey tool, similar to that used by Naughton (2002), to improve our understanding of how social isolation, socio-economic status, and the use of air conditioning affect the difference between mortality and morbidity rates.

In this study, we observed a contrast between diabetes, nephritis and nephrotic syndromes, and acute renal failure causes of mortality and hospital admissions in the relatively young, 45-64 age group. To our best of our knowledge, this contrast has not been identified and discussed in the literature. Our findings show a statistically significant increase in mortality from diabetes (78%) with no corresponding increase in mortality from nephritis and nephrotic or acute renal failure causes. Conversely, we observed a statistically significant increase in nephritis and nephrotic (76%) and acute renal failure (99%) hospital admissions, with no corresponding increase in diabetes admissions. Given that roughly 44% of all new kidney failure cases originate from diabetic patients (United States Renal Data System 2007) we would expect these causes of mortality and morbidity to be more connected. A partial explanation to these findings may lie in how the mortality and morbidity data were coded. Death Certificate and Comprehensive Hospital Abstract Reporting System (CHARS) data use the International Classification of Diseases (ICD-9) and the ICD Clinical Modification (ICD-9-CM) coding processes, respectively. On the death certificate, ICD-9 code is used to identify the underlying cause of death, while CHARS uses the codes to classify diagnoses and procedures (Harriss et al. 2011; O'Malley et al. 2005). Additional research is needed to fully understand the association between diabetes and renal outcomes. Future research could explore *multiple causes of death*,

included in the death certificate database, to identify diabetic mortality influenced by nephritis and nephrotic and/or acute renal failure. Likewise, future analyses that include pre-existing health conditions for renal-related hospital admissions would shed light on connections to diabetes.

This dissertation highlights a younger, potentially vulnerable group: the 45-64 year-old diabetic population. Our findings suggest that King County's diabetic population could be at an increased risk for chronic and acute kidney hospitalizations, as well as mortality, when exposed to extreme heat. In King County, an estimated 8% of 45-64 year-olds and 15% of 65+ year-olds have diabetes (Public Health-Seattle & King County 2013). Diabetes varies by race, income, and neighborhood: compared with Whites, African-Americans and American Indians have roughly double the diabetes prevalence; households with the lowest income have nearly three times the diabetes prevalence as households with the highest income; and households located in South and North King County have a higher prevalence of diabetes than those households located in Seattle or the East part of the county (Public Health-Seattle & King County 2013). Nationwide, diabetes accounts for approximately 23% of the total healthcare costs. The 45-64 and 65+ year-old age groups represent 33% and 59% of all diabetic-related health care expenditures, respectively. In Washington State, the 2012 cost burden of diabetes-related direct medical care is estimated at \$3.75 billion dollars (American Diabetes Association 2013). Given our findings and the substantial burden that diabetes places on the economy, public health funding of targeted extreme-heat preparedness interventions for the diabetic population is warranted and easily justified.

It is anticipated that the Affordable Care Act (ACA) will have a significant effect on health outcomes and may subsequently affect population heat-vulnerabilities. In 2011,

approximately 17% (217,000) of King County residents between the ages of 18 and 64 were uninsured. Insurance status differed among race, ethnicity and geographical location. Hispanics had nearly four times the uninsured status as compared with Non-Hispanics, while American Indian and African-Americans were both twice as likely as Whites to be uninsured. Additionally, groups living in South King County were more likely to be uninsured than those living in the rest of the county. The ACA should improve access for all ethnic and racial groups, and for low-income individuals. An estimated 83,000 uninsured King County residents will become eligible for Medicaid, with the homeless population accounting for approximately 7,000. Spatially, it is predicted that the highest concentration of Medicaid and subsidy-eligible adults live in South King County (King County 2013).

Additionally, the ACA provides new public health funding for tobacco cessation, obesity, nutrition and physical activity intervention programs. According to Waidmann, Ormond and Bovbjerg (2011), these funded interventions target “primarily chronic diseases like diabetes, hypertension, heart disease, stroke, and renal disease.” As King County citizen’s access to and use of health care and preventative services increases, reduction in prevalence of these chronic diseases is likely to occur. Given that those with these disease causes were also found to be at higher risk of mortality and morbidity during an extreme heat event, we would expect heat-risk estimates to decline as well. Additionally, it is possible that the ACA will result in an increase in medical service utilization during extreme heat events among those who previously delayed care due to lack of coverage. This may also attenuate the differences we currently find between the mortality and morbidity analyses. While the 65+ age group already receives health care coverage under Medicare, their heat-risk vulnerability is still likely to improve given the ACA’s increased funding for chronic disease intervention programs.

### 7.2.3 Threshold comparison

A final benefit from the concurrent epidemiological analyses is the opportunity to compare relative and absolute thresholds. This study used the 99<sup>th</sup> percentile and a model-derived absolute humidex value to determine excess heat risk for both mortality and morbidity. For mortality, the relative and absolute thresholds were nearly identical. For morbidity, the absolute threshold was 1.2 °C higher than the relative, 99<sup>th</sup> percentile threshold. Furthermore, comparing the absolute mortality and morbidity thresholds, we observed a 1.4 °C higher threshold for morbidity. There is no standard definition of a heat day or heat event. However, from a practical standpoint, setting a public health response threshold at the lowest of the significant thresholds would allow interventions to have the biggest public health impact.

In King County, as with the rest of the United States, the U.S. National Weather Service (NWS) is responsible for issuing forecast alerts related to extreme weather. Traditionally, the NWS may choose to issue a heat advisory if the heat index (apparent temperature) is forecasted to reach  $\geq 100$  °F for one or more days, and the minimum nighttime lows remain above 75° F. An excessive heat warning can be issued if the heat index is forecasted to reach  $\geq 105$  °F for at least two consecutive days, and the minimum nighttime lows remain above 75 °F (National Weather Service 2005). In King County, we found that mortality risks statistically significantly increase at a humidex equivalent to 36.0 °C (97.0 °F). While the humidex value is approximately 4.5 °C (8 °F) lower than the NWS advisory threshold, the values are not a direct comparison. In fact, at this heat, our humidex value is equivalent to a heat index approximately 7 °C higher. Therefore, it would appear that the NWS heat advisory alert threshold is sufficiently below our lowest statistically significant threshold. However, our average county-wide nighttime humidex lows are approximately 13 °C (55 °F), well below the NWS's minimum

nighttime heat index. It should be noted that the issuance of heat advisories and warnings are at the discretion of the local weather forecasting office, and that they are specific to the climate zone in which they are issued.

Our study also explored and found effect modification from oppressive weather mass types with mortality but not morbidity outcomes. Weather data are classified into one of seven different air mass types. According to Sheridan and Kalkstein (2004), two of the seven are related to mortality: dry tropical (DT), and moist tropical (MT). The authors also found that the more temperate the location, the greater the increase in mortality on oppressively hot weather days. A movement towards including synoptic forecasting data into advisory and warning decisions has been quickly gaining ground; this information is now being used by local weather forecasting offices in 27 U.S. and 19 international metropolitan areas, including the Greater Seattle Area (Sheridan and Kalkstein 2010).

This movement has been supported by evaluation research. Ebi et al. (2004) evaluated the effect that the city of Philadelphia's use of synoptic-based heat-health warning systems (HHWS) had on observed mortality count. They estimated that within the 4-year study period, 117 lives had been saved by the synoptic warning system, as compared to the traditional NWS alert thresholds. Kalkstein et al. (2011) evaluated 40 major cities and found that, since 1995, those cities with a more aggressive, synoptic-based HHWS, experienced greater reductions in mortality than using non-synoptic-based alert systems. While the decision to consider synoptic forecasting data in the alert process is still optional, our findings support routine use of these data at the Seattle weather forecasting office. Additional research could determine if heat alerts are consistently issued for days that this study has found to exceed the lowest mortality threshold, and whether or not the inclusion of synoptic weather data increased frequency of alert issuance.

### 7.3 Communication

The final goal for this dissertation was to communicate our findings in a way that was useful for our public health practice community. Since our public health partner's (Public Health-Seattle King County) primary goal was to collaborate with other internal county departments, we provided them with a stand-alone PowerPoint© slide deck that they could use for these purposes. The PowerPoint© slide deck was unique in several ways: it used the notes pane, for each slide, to fully describe the study results; it included proportional bubble graphics to communicate heat-health risk estimates; and it included heat-risk maps identifying the geographical distribution of heat and vulnerable populations. It is hoped that this packaged information will increase the likelihood that the data will be used to support climate change-related mitigation and adaptation programs and policies in King County.

While we were unable to fully evaluate the value of our translation and communication efforts, the preliminary discussions with PHSKC have been encouraging. Not only do they perceive the PowerPoint© as useful in their future communication plans, but they plan to use our data as they update their *King County Health and Human Services Transformation Plan*. This five-year strategic plan charts the path to improving the health and human services for King County (King County 2014). Our heat-risk vulnerability maps and associated outcome estimates will be valuable resources as they consider health impacts to various regions within the county. In particular, South King County has: the highest concentration of individuals that qualify for Medicaid and subsidized health care under the Affordable Care Act, a higher prevalence of diabetes, and some of the hottest average humidex values on a given heat day. Time for policy action and additional research is necessary before the full extent of the dissertation's impact is known.

## 7.4 Conclusion

This dissertation tested the performance of a heat-health outcome relationship model developed to aid in the prediction of climate change-related mortality and morbidity for King County, Washington. The results suggest that, overall, the piece-wise linear function describing heat's effect on health outcomes performed well. This study also characterized King County's historical heat-health outcome relationship using two different statistical methods. The results suggest that heat, expressed as humidex, is associated with increased mortality and morbidity on heat days, and that the risk increases with heat's intensity. This study has found that, when stratifying by age and cause of death, younger age groups are at an increased risk of death and hospitalization for several different causes, particularly diabetes, nephritis and nephrotic syndromes, acute renal failure, and natural heat exposure. The findings from this dissertation were translated and packaged using a PowerPoint© slide deck, which communicated estimated heat effects on mortality and morbidity, as well as the spatial distribution of heat risk for vulnerable populations. While additional time is needed to fully understand and evaluate potential effects on policy action, it is hoped that this dissertation has, at a minimum, provided PHSKC with the data needed to obtain a valued seat at their own county's climate change discussion table.

Our findings warrant additional investigation into the role heat exposure plays in diabetic patients' health and care, as well as the connection between diabetes and renal syndromes in our area. Incorporating census tract-level diabetes prevalence data is an important next step to improving the understanding of heat-risk distribution. Our findings also warrant a more in-depth look at the circulatory and cardiovascular health-outcome connections for the elderly, 85+ group.

A logical next step for research would be to examine the next tier in heat-health impacts: emergency calls and emergency room visits.

This dissertation's design might be improved if exposure assessment was refined to reflect a population-weighted humidex value, or by utilizing the geo-coded data to assign each case a maximum humidex value from the closest meteorological grid center point. Our understanding of vulnerable populations could be improved by adding additional sources of information to our model, such as neighborhood-level average income, prevalence and use of air conditioning, and tree canopy or impervious service indices. Finally, what we learn from additional research may result in the refinement of our heat-risk maps to include other heat-vulnerable populations, and may provide our public health partners with additional health-related data to share with their constituents.

## Notes to chapter 7

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