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Public health impacts of climate change in Washington State: projected mortality risks due to heat events and air pollution

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Abstract Illness and mortality related to heat and worsening air quality are core public health concerns associated with climate change projections. We examined the historical relationship between age- and cause-specific mortality rates from 1980 through 2006 and heat events at the 99th percentile of humidex values in the historic period from January 1, 1970 to December 31, 2006 in the greater Seattle area (King, Pierce and Snohomish counties), Spokane County, the Tri-Cities (Benton and Franklin counties) and Yakima County; the relative risks of mortality during heat events were applied to population and climate projections for Washington State to calculate number of deaths above the baseline (1980–2006) expected during

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projected heat events in 2025, 2045 and 2085. Three different warming scenarios were used in the analysis. Relative risks for the greater Seattle area showed a significant dose-response relationship between heat event duration and daily mortality rates for non-traumatic deaths for persons ages 45 and above, typically peaking at four days of exposure to humidex values above the 99th percentile. The largest number of projected excess deaths in all years and scenarios for the Seattle region was found for age 65 and above. Under the middle warming scenario, this age group is expected to have 96, 148 and 266 excess deaths from all non-traumatic causes in 2025, 2045 and 2085, respectively. We also examined projected excess deaths due to ground-level ozone concentrations at mid century (2045–2054) in King and Spokane counties. Current (1997–2006) ozone measurements and mid-twenty-first century ozone projections were coupled with dose-response data from the scientific literature to produce estimates overall and cardiopulmonary mortality. Daily maximum 8-h ozone concentrations are forecasted to be 16–28% higher in the mid twenty-first century compared to the recent decade of 1997–2006. By mid-century in King County the non-traumatic mortality rate related to ozone was projected to increase from baseline (0.026 per 100,000; 95% confidence interval 0.013–0.038) to 0.033 (95% CI 0.017–0.049). For the same health outcome in Spokane County, the baseline period rate of 0.058 (95% CI 0.030–0.085) was estimated increase to 0.068 (95% CI 0.035–0.100) by mid-century. The cardiopulmonary death rate per 100,000 due to ozone was estimated to increase from 0.011 (95% CI 0.005–0.017) to 0.015 (0.007–0.022) in King County, and from 0.027 (95% CI 0.013–0.042) to 0.032 (95% CI 0.015–0.049) in Spokane County. Public health interventions aimed at protecting Washington's population from excessive heat and increased ozone concentrations will become increasingly important for preventing deaths, especially among older adults. Furthermore, heat and air quality related illnesses that do not result in death, but are serious nevertheless, may be reduced by the same measures.

1 Introduction

Climate change is likely to have serious and long-term consequences for public health Researchers have identified a number of broad health issues associated with climate change, such as food security, large-scale migration and civil conflict (Frumkin et al. 2008). Other causes of morbidity and mortality that can be affected by climate change include water- and food-borne illnesses, vector-borne diseases, as well as traumatic injury and death from extreme weather events (Patz et al. 2001). Potential increases in vector-borne diseases are also of concern, but the large number of climate sensitive variables in their transmission makes the modeling of the effects of climate change very complex (NRC 2008a). The majority of food- and water-borne illnesses are not reportable conditions in Washington state, and while incidence of some of these illnesses are associated with summer months or flooding, little work has been done in this region to determine how incidence might be affected by climate change.

This report was not able to address these very important issues, although we hope to do so in subsequent work. Instead, our work has focused on a specific sub-set of core public health concerns related to climate change: heat-related mortality and worsening air quality (Luber and McGeehin 2008; Kinney 2008). Annual average temperatures in the USA and globally are rising, although the effects vary from

region to region (Mote and Salathé 2010 this issue). It is estimated that 400–700 people die from documented thermal stress, or hyperthermia, each year in the USA (Bernard and McGeehin 2004). Because the immediate cause of death is usually some form of cardiovascular failure, and hyperthermia is often not noted on the death certificate as an underlying factor, the number of heat-related deaths is underestimated (Wolfe 2001; CDC 2006).

Relatively short but intense heat waves over the last 30 years have been responsible for thousands of deaths in the USA and Canada, and thousands of deaths in Europe (Jones et al. 1982; Semenza et al. 1996; Whitman et al. 1997; Naughton et al. 2002; Kaiser et al. 2007). Climate projections suggest that these events will become more frequent, more intense and longer lasting in the remainder of the twenty-first century (Meehl and Tebaldi 2004). The greatest impacts will likely be in cities with milder summers, less air conditioning and higher population density (McGeehin and Mirabelli 2001). An aging population also will put more people at risk (Smoyer et al. 2000). Increased temperatures can also have an effect on the effectiveness of medications for the elderly as well as the general population.

Retrospective epidemiological research has identified groups most likely to be harmed by heat waves and suggests strategies to mitigate these harms through public interventions. The groups at greatest risk include the following: children, due to slower adaptation during exercise (AAP 2000); the elderly, due to changes in the physiological ability to maintain normal body temperature (Borrell et al. 2006; Basu et al. 2005; CDC 2005); poor and socially isolated populations, due to less access to mitigation measures (Greenberg et al. 1983; McGeehin and Mirabelli 2001; Browning et al. 2006); some urban dwellers, due to heat island effects and lack of vegetation (Grimmond and Oke 1999; DeGaetano and Allen 2002); outdoor laborers, due to extended exposures and lack of access to drinking water and shade (Greenberg et al. 1983; WA Dept Labor and Industries 2008); people with chronic illnesses (e.g., diabetes, heart disease), due to increased vulnerability to sustained heat (Medina-Ramon et al. 2006); and the mentally ill, due to behavioral factors and the effects of psychoactive medications (Kaiser et al. 2001).

Methods used for estimating mortality due to heat generally rely on an analysis of regional weather data in combination with daily mortality data. This typically requires large, dense urban areas for daily values to be sufficiently stable to support analyses. Most such studies consider the effects of both temperature and humidity. Studies of heat-related mortality in Philadelphia and Toronto have used synoptic climate modeling to identify regional conditions associated with elevated mortality (Kalkstein et al. 1996; Pengelly et al. 2005; Cheng et al. 2005). Regional and temporal differences in the effect of heat on mortality have been identified (Kalkstein and Davis 1989; Davis et al. 2003).

In addition to heat, adverse effects of climate change on air quality have recently come under investigation. The primary ambient air pollutants of concern for public health risk in Washington State include both fine particulate matter and ozone. An expanding evidence base regarding the relationship of these pollutants to adverse health outcomes has resulted in more stringent federal standards that require attainment of lower concentrations of these pollutants (US EPA 2006, 2008). Despite overall improvement in regional air quality over the past decade, future climate change related increases in ozone or $PM_{2.5}$ could lead to more days of exposure above health-based guidelines for Washington residents (PSCAA 2007).

The influence of meteorology on ozone and particulate matter concentrations is well recognized and critically important (EPRI 2005; Bernard et al. 2001). There is considerable evidence that ozone concentrations would increase in the USA as a result of climate change, if precursor emissions were held constant; data regarding influences of climate change on particulate matter are far fewer, precluding clear conclusions (CCSP 2008). For both of these pollutants regional-specific assessments of potential health impacts are few (Knowlton et al. 2004).

While ozone and fine particulate matter have been associated with multiple health outcomes, including increases in prevalence, clinical utilization, and severity of cardiac and respiratory disease, most studies have focused on premature mortality as an endpoint. This reflects recognition of this endpoint as the most serious outcome, as well as its status as the most accessible and reliable health outcome for which data are available for evaluation in large population based studies. Numerous epidemiologic studies in the USA and abroad have identified increased premature mortality in association with increased ozone exposure (Bell et al. 2004). The robustness of this evidence base, including several recent multi-city and meta-analyses, has been noted in a recent National Academy of Sciences report (NRC 2008b). While the effect estimates vary somewhat by study design and region, the studies viewed as a whole provide a pattern of consistency with generally comparable magnitude of effect estimates.

Increasingly, region-level modeling of ozone and other air pollutants under climate change scenarios is being conducted (Weaver et al. 2009). In the Pacific Northwest regional projections of future air quality at the resolution of approximately county level scales (36 km horizontal grids) have been developed. We sought to integrate knowledge of the ozone concentration–mortality response with Washington State ozone pollution projections to provide an initial quantitative assessment of potential mortality impacts in the mid twenty-first century. We selected to contrasting regions for this analysis: Spokane County and King County, Washington. Since the atmospheric chemical reactions that form ozone from precursors are driven by sunlight and increased temperature, Spokane's climatological conditions yield higher overall average summertime temperatures and episodes of high temperatures.

Increased levels of $PM_{2.5}$ are an important factor in poor air quality conditions in the State of Washington. Climate change, however, has not been shown conclusively to be a significant factor in future projected levels of $PM_{2.5}$. In an attribution study of various contributing factors to future air quality, Avise et al. (2008) showed that projected changes in weather patterns for the 2050s produced an insignificant (0.2 $\mu g/m^3$) reduction in $PM_{2.5}$ for EPA Region 10 (Alaska, Idaho, Oregon, Washington). Nevertheless, future changes in local and Asian emissions are projected to increase $PM_{2.5}$ levels by 2 $\mu g/m^3$ (from a current value of 4 $\mu g/m^3$) over the same period in this region, and interaction between this increase and climate change may have an amplified impact on human health in the future. Such interactions are beyond the scope of the current project but merit future research given the increasing evidence for adverse public health consequences of $PM_{2.5}$ exposure.

This study had three goals. First, we estimated the historical relationship between extreme heat events and mortality in different regions of Washington State for selected age groups and causes of death. Second, we used these estimates to project the number of excess deaths by age group and cause during projected heat events in years 2025, 2045 and 2085; these time points were selected to be consistent with

other studies being conducted in Washington State. Finally, we used estimates of the relationship between ozone concentration and mortality available from the scientific literature to predict the number of excess deaths in King and Spokane counties at mid-century (2045–2054) due to ozone under a changing climate, and assuming a growing population.

2 Methods

2.1 Estimates of relative risk of mortality due to heat events, 1980–2006

Four study areas were selected for the heat event analysis (Fig. 1): greater Seattle area (King, Pierce and Snohomish counties), Tri-Cities (Benton and Franklin counties), Spokane County, and Yakima County. Daily historic weather data were drawn from the 16th degree downscaled models (Mote and Salathé 2010). Grid points falling within study area counties (grid size ~7.2 by 4.8 km) were identified by spatially joining the grid center points and county boundaries using ESRI ArcMap software. Heat events were defined using humidex values. The humidex is a measure of the combined effect of heat and humidity on human physiology (Masterton and Richardson 1979; Environment Canada 2008), and has been used in other mortality studies and as a basis for declaring heat warnings (Smoyer-Tomic and Rainham

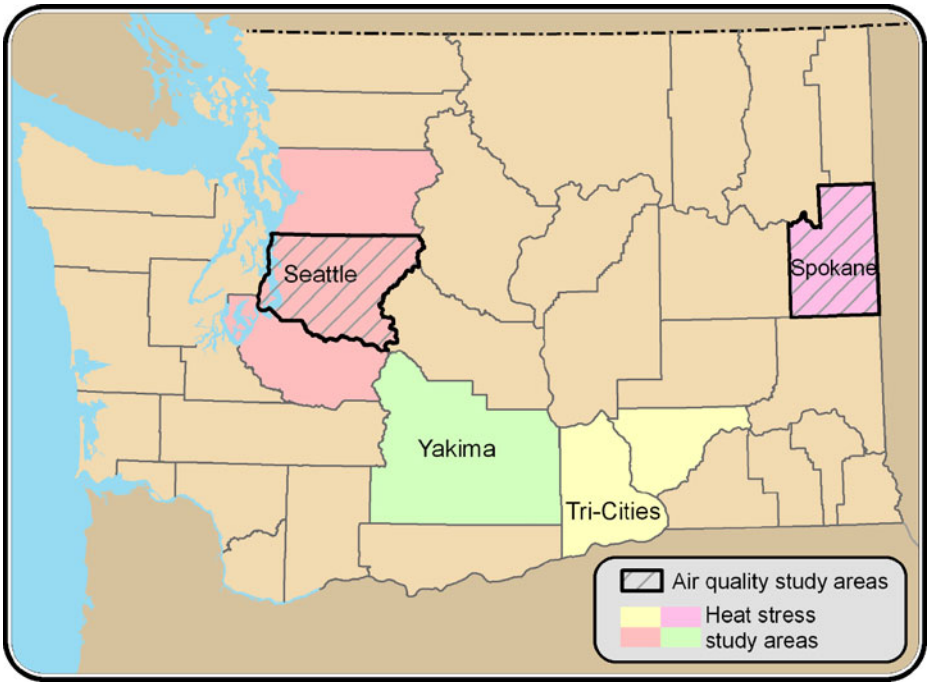

Fig. 1 Map of study areas. Figure: Robert Norheim

2001). The humidex value was calculated for each grid point from daily maximum temperature and relative humidity data using the following formula:

$$\text{Humidex} = T + 5/9 * (v - 10) \quad (1)$$

where T = air temperature (degrees Celsius), v = vapor pressure = (6.112 × 10^(7.5*T/(237.7 + T)) * H/100), and H = humidity (%).

Grid point humidex values were averaged across all grids in each county to yield a county-level humidex value for each day from January 1, 1970 to December 31, 2006. Thresholds at the humidex 99th percentile were identified for this entire historical period in each study area. After finding the 99th percentile value, we then determined which months in the historical record had heat events and used an observation frame for the analysis. This approach allowed us to unambiguously define both a humidex-based threshold and the months for observing heat events. Heat events were defined as one or more consecutive days of the humidex above these thresholds; the number and duration of heat events were counted in each study area over the period. Since only daily observations of mortality were available, it was not possible or necessary to resolve the heat event time periods to less than 1 day intervals.

Annual county population estimates by age group from 1980 through 2006 were obtained from Washington State's Office of Financial Management (OFM 2008a). Complete mortality data from 1980 through 2006 were obtained from the Washington State Department of Health. Computerized mortality data were not available for earlier periods prior to 1980. Daily numbers of deaths for each year were aggregated by cause, age group, and county of residence.

Heat has been cited frequently as a contributing factor in deaths due to failure of the circulatory and respiratory systems. Therefore, the following cause-of-death categories were examined: all non-traumatic causes (ICD-9: 001–799; ICD-10: A00–R99), circulatory (ICD-9: 390–459; ICD-10: I00–I99, G45, G46), respiratory (ICD-9: 460–519; ICD-10: J00–J99), cardiovascular (ICD-9: 393–429; ICD-10: I05–I52), and ischemic (ICD-9: 410–414; ICD-10: I20–I25). Cardiovascular and ischemic are subsets of circulatory. The ICD groups used are from a study of heat and air quality related mortality in Toronto (Cheng et al. 2005). ICD-9 was changed to ICD-10 in 1999, so our analysis has used each coding system for the appropriate years.

Observed and expected crude daily mortality rates for age and cause-of-death specific groupings were calculated for heat event days (days 1 to day 5+) and non-heat event days (all defined as day 0) during the years 1980–2006. Only data in the months of May–September between 1980 and 2006 were used in the analysis. Daily mortality observed during heat events in the months of May–September were accumulated in five time periods of roughly 5-year duration: 1980–1984; 1985–1989; 1990–1994; 1995–1999; and 2000–2006. A mortality rate for non-heat event days was computed in six age-specific categories of 0–4, 5–14, 15–44, 45–64, 65–84, and 85+ years. Heat events have been shown to present increased mortality risks for older persons, so most of our analysis focused on the following age categories: 45 years and older, 65 years and older and 85 years and older.

Deaths occurring on each consecutive day of a heat event were counted for each study area, and classified according to the duration of potential heat exposure prior to

the day of death for day 1 through day 5+ of heat events. The average daily mortality rates on days between May and September with no defined heat event (all designated as day 0) were treated as the baseline mortality rates for each time period. Expected values for the number of deaths in each day of a heat event in an annual period were calculated by applying the average daily mortality rate for non-heat event days to the number of days observed in each exposure duration (day 1, day 2, etc.) for heat events during a year. The total observed and expected deaths were then summed for each exposure duration category for all heat events. The mortality relative risks by heat event duration, specific age and disease categories were computed from the ratios of observed over expected duration-specific mortality. Calculating separate relative risks for each elapsed day of a heat event (starting with day 1 of the heat event) allows evaluation of the influence of a single day versus more prolonged heat events on mortality.

Confidence intervals were computed assuming Poisson intervals for the observed number of cases as recommended by the Washington State Department of Health (DOH 2002). Exact 95% confidence intervals were computed using Poisson distribution percentiles when the number of observed deaths was <500; for >500 observed deaths, intervals were computed using a normal approximation method (Breslow and Day 1987). This procedure was repeated separately for each heat study area in order to control for regional differences in the effect of heat events on mortality. Given the smaller population in Eastern Washington, a combined analysis of Benton, Franklin, Spokane and Yakima county study areas was also performed.

2.2 Population projections for Washington State in the twenty-first century

Projected county population estimates by age group were obtained from the Washington Office of Financial Management for the years 2005–2030 (OFM 2008b). In predicting future excess deaths during extreme heat events, population was held constant at 2025 projected estimates, allowing differences in excess deaths between years to be interpreted as the component due to climate change. However, this analysis was not able to incorporate other factors that may have been at play; e.g., access to health care, demographic shifts. For the analysis of excess deaths related to ozone concentrations, calculated total and age-group populations were calculated by extending the Office of Financial Management linear projections to 2045 through 2054. Washington State population forecasts are developed from a cohort component demographic forecast model that accounts for births, deaths and net migration. Projected births are derived from a natural change model component of the childbearing population, applying historical trends in fertility rates by county. Annual deaths, in terms of life expectancy generally follow national trends, and survival expectations were adjusted to follow Social Security Administration projections in 2007. Migration is the most important variable component of the population forecasts. The state's future net migration is based on an econometric model where Washington's relative attractiveness to job seekers is weighed against the attractiveness of California and other state destinations. A historical comparison of the actual and fitted net migration for 1978–2008 using OFM's migration model found an R^2 of 0.91, indicating reasonably good agreement.

2.3 Projected excess mortality due to heat events

Projected heat events were determined for 3 years: 2025, 2045 and 2085. Three climate change scenarios were selected for high, moderate and low summer (May–Sept.) warming, for a total of nine modeled future heat regimes. The low scenario chosen was the PCM1-B1 model, the high scenario chosen was the HADCM-A1B model, and the middle scenario was the mean of the two composite models using either the A1B or B1 emissions scenario (Mote and Salathé 2010). Expected monthly temperature deviations in Celsius for each scenario and time period were added to the observed daily temperature and relative humidity distributions in each study area from 1970 to 1999; the daily humidex was then calculated for each of the new temperature distributions. Historical humidex thresholds at the 99th percentile were applied to the estimated future distributions, and the number and duration of expected heat events in 2025, 2045 and 2085 were calculated for each scenario.

Projections of heat-related mortality were developed by applying the baseline mortality rate and duration-specific relative risks derived from the historical analysis to the expected future population structure and expected number and duration of heat events in each of three heat scenarios for time periods of 2025, 2045 and 2085. The climate modeling produced 30 realizations of daily maximum temperature changes for each time period. Excess deaths, which are the number of expected deaths above the baseline number of deaths, were calculated for each heat realization for each time period. This resulted in an estimate of the mean annual excess deaths in a sample of 30 simulated realizations for each region and future time period.

2.4 Projected excess mortality due to air pollution

We adapted a health risk assessment modeling approach described by Knowlton et al. (2004) in their effort to assess the impact of ozone on mortality in the northeastern USA. We selected two populous but distinct climatological areas of the State for this initial assessment. Using the following formula, we estimated ozone-related mortality for King County and Spokane County in the recent decade (1997–2006) and at mid-century (2045–2054):

$$M = \left(P/100,000\right) * B * \mathrm{CR} * E \tag{2}$$

where M is the excess mortality due to ozone, P is the estimated population in the county for the period of interest, B is the baseline county-level mortality rate, CR is the concentration-response function that describes the expected change in daily mortality per incremental increase in ozone, and E is the concentration of ozone during the period of interest. We calculated overall non-traumatic mortality as well as mortality specific to cardiopulmonary causes.

The population (P) data were derived from annual population size estimates available from the US Census for King and Spokane County for 1997–2006 and projections of the annual population for these counties in 2045–2054, as described above. For each decade, the mean annual average was calculated. These data demonstrated that from the period of 1997–2006 to mid-century (2045–2054), the annual average population size for King County is expected to increase from 1,758,260 to 2,629,160 (50% increase). In Spokane County, the population is expected to grow from 424,636 to 712,167 (68% increase).

The county-level non-traumatic (categorized as above) and cardiopulmonary (ICD-9: 393–429, 460–519; ICD-10: I05–I52, J00–J99) mortality rates were calculated by dividing the daily average number of total non-traumatic deaths and cardiopulmonary deaths in the baseline decade of each county by its annual population average. For 1997–2006, the mean daily total non-traumatic and cardiopulmonary death rates per 100,000 for King County were 1.55 and 0.57, respectively. For Spokane County, these rates were 2.03 and 0.78, respectively.

We examined concentration-response (CR) functions for ozone based on three meta-analyses, two multi-city time series, and one case-crossover study of populations in the USA, all of which were reviewed in a recent National Academy of Science report which summarized estimates of the percentage increase in mortality from short-term increases in ozone (NRC 2008b). We decided to apply the analysis by Bell et al. (2004) to our data. This analysis included data and methods developed for the National Mortality and Morbidity Air Pollution Study (NMMAPS; see Bell et al. 2004). This landmark study estimated a national average relative rate of mortality (non-injury mortality and cardiopulmonary mortality) associated with short-term average ambient ozone concentrations in 1987–2000 based on 95 large US urban communities made up of almost 40% of the US population (including Spokane and Seattle). Of note, the city-specific estimates for King and Spokane County within the NMMAPS analyses were nearly identical to the combined multi-city concentration-response function employed in this assessment, further supporting its appropriateness. Estimates available per 24-h average ozone concentration were converted to 8-h maximum concentrations based on the recommended ratio of 8-h ozone to a 24-h average of 1.53 (NRC 2008b). The concentration-responses for ozone-related non-injury mortality and cardiopulmonary mortality derived from this analysis were 0.80% (95% confidence interval 0.41%–1.18%), and 0.98% (95% CI 0.47%–1.50%), respectively per 10 parts per billion (ppb) increase in 8-h maximum daily ozone concentration over the previous week.

For each county, exposure to ozone ($E_{1997-2006}$) in the recent decade of each county was assessed based on 8-h maximum daily ozone (ppb) concentration data drawn from the Washington State Department of Ecology air quality monitoring network for the months May–September (warm season) from 1997–2006. A warm season "baseline" decadal daily average was calculated.

We then estimated future comparable measurements of ozone in the mid-century decade ($E_{2045-2054}$). To accomplish this, we derived the change in ozone concentration predicted from a modeling framework which calculated both daily 8-h maximum concentrations for the baseline decade of this century (1990–1999) as well as for 2045–2054. Specifically, daily 8-h maximum daily average ozone concentrations for May–September of the mid-century decade (2045–2054) were derived by coupling a global climate model projection with regional meteorology and chemistry models for the 36 km grids that coincide with King and Spokane Counties.

The modeling framework is described in detail in Chen et al. (2008). Briefly, the regional Mesoscale Meteorological model version 5 (MM5) was used to downscale the Parallel Climate Model (PCM) to produce regional meteorological fields. These fields were used to drive the Community Multi-scale Air Quality (CMAQ) model, which downscaled the Model for Ozone and Related Chemical Tracers, version 2.4 (MOZART2 outputs) and accounted for regional pollutant emissions to predict photochemical ozone and PM levels. The MM5/CMAQ modeling treats increased ozone formation under climate change as a direct effect of increasing temperature

as well as broad indirect effects. The 2050's projections were based on the IPCC A2 scenario, changes in US emissions due to population growth and economic expansion, and alterations in land use/land cover that can affect both meteorological conditions and biogenic emissions important for ozone formation. Future chemical boundary conditions were obtained through downscaling of MOZART-2 based on the IPCC A2 emissions scenario. Projected changes in US anthropogenic emissions were estimated using the EPA Economic Growth Analysis System (EGAS), and changes in land-use were projected using data from the community land model (CLM) and the spatially explicit regional growth model (SERGOM; Mote and Salathé 2010).

It is important to recognize that the county monitoring data are influenced by fresh nitrogen oxide emissions largely derived from traffic sources which cause titration (loss) of ozone in the urban areas, while the model results, based upon 36 km grids, tend to minimize this effect since the NO_x emissions are diluted significantly due to the size of the grid. This is clear from evaluation of the modeling system which consistently shows that the model overestimates concentrations at the low end of the ozone concentration distribution. Consequently, urban monitors will record relatively low ozone concentrations while nearby more rural monitors will record higher ozone concentrations. The model results will not correctly reflect these differences. This is clear from evaluation of the modeling system which consistently shows that the model overestimates low ozone levels (Chen et al. 2008). Because of this bias in the model we employed the model results in a relative sense, where the change in predicted ozone levels between the baseline period and the future decade were added to the baseline measured values at each site to yield an estimate of future levels. This is essentially the same approach that EPA uses for analysis of ozone control strategies where it is recognized that the models perform better in predicting the change in ozone due to a control compared to predictions of absolute levels.

3 Results

3.1 Estimates of excess mortality due to heat events, 1980–2006

The heat study areas accounted for approximately two-thirds of Washington State's population in 2006; King, Pierce and Snohomish counties combined made up just over half of the state's 2006 population of 6.3 million (Table 1). Persons ages 85 and over made up approximately one percent of the total population in most study areas, and one half of one percent in the Tri-Cities region in 1980; by 2006 this age group had roughly doubled in all areas as a proportion of total population. Among study areas, the mean daily maximum humidex from May to September, 1970–2006, was lowest in the greater Seattle area (23.2°C, 73.8°F) and highest in the Tri-Cities (28.1°C, 82.6°F). The 99th percentile for annual daily maximum humidex ranged from 10°C to 12°C (18–20°F) higher than the May–September mean daily maximum. The number of heat events at or above the 99th percentile averaged 1.6 to 1.8/year, with a mean duration of 2.0 to 2.3 days, and maximum duration from 6 days (greater Seattle area) to 10 days (Yakima).

Table 1 Baseline climate and population parameters 1980–2006

	Greater Seattle Area	Spokane	Tri-Cities	Yakima
Counties included	King, Pierce, Snohomish	Spokane	Benton, Franklin	Yakima
1980 Population				
Total	2,236,898	367,867	157,983	187,226
45 to 64	395,521	62,823	25,928	32,670
65 to 84	184,078	35,232	9,141	19,009
85 and above	20,398	4,221	739	1,912
2006 Population				
Total	3,488,123	471,872	242,781	251,381
45 to 64	847,217	113,889	55,611	52,829
65 to 84	288,330	46,746	19,633	22,134
85 and above	51,580	9,502	2,774	4,493
Humidex, °C (°F)				
Mean daily high, May–Sep	23.2 (73.8)	26.2 (79.2)	28.1 (82.6)	24.9 (76.8)
99th pctl of daily high, annually	33.6 (92.5)	38.1 (100.6)	38.3 (100.9)	35.5 (95.9)
Heat events above 99th pctl				
Mean annual number	1.7	1.8	1.6	1.6
Mean (max) duration in days	2.2 (6)	2.0 (9)	2.2 (9)	2.3 (10)

Residents of the greater Seattle area experienced 14,250 deaths from all non-traumatic causes in all months of 1980, and 19,341 in 2006; in the Spokane, Tri-Cities and Yakima areas combined, there were 4,676 deaths from non-traumatic causes in 1980, and 6,264 in 2006 (not shown in tables). Annual mortality rates by non-traumatic causes in all study areas ranged from 36 to 130 per 100,000 for persons ages 0–14 and from 36 to 58 per 100,000 for those ages 15–44. Deaths for specific causes (e.g. ischemic disease) in these age groups were on the order of 20 per 100,000 or fewer annually in all study areas.

Mortality rates for all non-traumatic causes, circulatory causes and respiratory causes increased with age, and were highest for persons 85 years of age or older. In the greater Seattle area, the non-traumatic annual mortality rate among those ages 85 and above was 14,937 per 100,000 in 1980 and 12,460 per 100,000 in 2006; in the other study areas combined there were similar rates in this age group: 14,871 per 100,000 and 12,517 per 100,000 in 1980 and 2006, respectively. Annual mortality rates for all causes but respiratory were higher for all age groups in 1980 than in 2006. About half of all non-traumatic deaths in 1980, and about one third in 2006, were from circulatory causes, the bulk of these from cardiovascular causes. Only about one-tenth of non-traumatic deaths occurred due to respiratory causes annually (not shown in tables).

In the greater Seattle area, risk of death due to all non-traumatic causes and circulatory causes rose for the overall population ages 45 years and above beginning on day 1 of heat events, peaked on day 4, and declined slightly for day 5 and beyond (Table 2; Fig. 2). The highest relative risk (RR) estimated for non-traumatic deaths was 1.3 (95% confidence interval [CI] 1.2–1.5) for persons ages 65 and above, and 1.5 for those ages 85 and above (95% CI 1.2–1.8). Relative risk of death due to

Table 2 Mortality relative risks for selected causes and age groups by heat event duration, Greater Seattle Area and the Spokane, Tri-Cities and Yakima combined, 1980–2006[a]

Day of heat event	Greater Seattle Area					Spokane, Tri-Cities, Yakima				
	1	2	3	4	5+	1	2	3	4	5+
All non-traumatic causes										
Aged 45+	**1.0** (1,1.1)	**1.2** (1.1,1.3)	**1.1** (1,1.2)	**1.3** (1.1,1.5)	**1.2** (1,1.4)	1.0 (0.9,1.1)	1.1 (1,1.2)	1.1 (1,1.3)	1.0 (0.8,1.3)	1.0 (0.9,1.3)
Aged 65+	**1.1** (1,1.1)	**1.2** (1.1,1.3)	**1.1** (1,1.2)	**1.3** (1.2,1.5)	**1.2** (1,1.4)	1.0 (0.9,1.1)	1.1 (0.9,1.2)	1.1 (0.9,1.3)	1.1 (0.8,1.3)	1.0 (0.8,1.2)
Aged 85+	1.1 (1,1.1)	**1.3** (1.2,1.5)	**1.3** (1.1,1.5)	**1.5** (1.2,1.8)	1.1 (0.8,1.5)	1.0 (0.8,1.1)	1.1 (0.9,1.3)	1.1 (0.8,1.5)	1.1 (0.7,1.6)	1.0 (0.6,1.4)
Circulatory										
Aged 45+	1.0 (1,1.1)	**1.2** (1.1,1.3)	**1.2** (1,1.3)	**1.3** (1.1,1.6)	1.1 (0.8,1.3)	1.0 (0.9,1.1)	1.1 (0.9,1.2)	1.1 (0.9,1.4)	1.0 (0.7,1.4)	1.1 (0.8,1.4)
Aged 65+	**1.1** (1,1.2)	**1.2** (1.1,1.3)	**1.2** (1,1.3)	**1.4** (1.1,1.7)	1.1 (0.9,1.4)	1.0 (0.9,1.2)	1.1 (0.9,1.3)	1.2 (0.9,1.5)	1.1 (0.8,1.5)	1.0 (0.7,1.4)
Aged 85+	1.1 (1,1.2)	**1.4** (1.2,1.6)	**1.3** (1.1,1.6)	**1.5** (1.1,2)	1.2 (0.8,1.7)	1.1 (0.9,1.3)	1.1 (0.8,1.4)	1.2 (0.8,1.8)	1.1 (0.6,1.8)	1.1 (0.6,1.7)
Respiratory										
Aged 45+	1.0 (0.8,1.1)	**1.3** (1.1,1.5)	**1.4** (1.1,1.7)	1.1 (0.7,1.7)	1.5 (0.9,2.3)	0.9 (0.7,1.1)	1.0 (0.7,1.4)	0.9 (0.5,1.5)	0.6 (0.2,1.3)	0.8 (0.3,1.5)
Aged 65+	1.0 (0.8,1.1)	**1.3** (1,1.5)	**1.4** (1.1,1.8)	1.2 (0.7,1.8)	1.6 (0.9,2.5)	0.8 (0.6,1.1)	1.0 (0.7,1.4)	1.0(0.6,1.7)	0.5 (0.1,1.4)	0.8 (0.3,1.6)
Aged 85+	0.8 (0.6,1)	1.3 (0.9,1.7)	1.3 (0.9,2)	1.4 (0.6,2.7)	1.5 (0.5,3.2)	0.6 (0.3,1)	1.3 (0.7,2.2)	0.7 (0.1,2)	0.8 (0.1,2.9)	0.6 (0.1,2.3)

[a] Bolded relative risk values are significantly greater than 1 ($p < 0.05$)

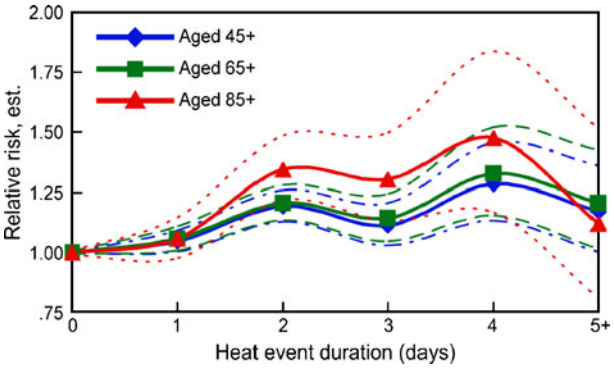

Fig. 2 Mortality relative risk estimates (*solid lines*) for all non-traumatic causes (ICD-9: 001–799; ICD-10: A00–R99) by heat event duration (at 99th percentile), Greater Seattle Area (King, Pierce and Snohomish counties), 1980–2006. *Dotted lines* show estimated 95% confidence limits

circulatory causes followed a similar pattern for persons ages 65 and above and 85 and above, with the highest effect observed in association with 4 days of exposure (RR = 1.4, 95% CI 1.1–1.7, and 1.5, 1.1–2.0, respectively; Fig. 3). Risk of death from non-traumatic and circulatory causes was significantly elevated for all ages on most days of heat events. Duration-specific RRs due to respiratory causes were less likely to reach statistical significance and were based on smaller sample sizes (Fig. 4); the risk was greatest on day 3 for persons ages 45 and over (RR = 1.4; 95% CI 1.1–1.7) and 65 and over (RR = 1.4; 95% CI 1.1–1.8). However, the highest effect estimates were observed on day 5 for all age ranges, and confidence intervals suggest the possibility of substantially elevated risks on day 5 and beyond for anyone ages 45 and above (RR = 1.5; 95% CI 0.9–2.3), and particularly for persons ages 65 and above (RR = 1.6; 95% CI 0.9–2.5). The overall relative risk of death for non-traumatic causes was 1.1 for persons ages 65 and above and 1.2 for persons ages 85 and above (which can also be expressed as elevated risks of death during heat events of 10% and 20%, respectively), compared with more temperate periods; overall RRs were similar for circulatory causes (not shown in tables).

Relative risks were derived for Eastern Washington study areas combined as a group (Table 2). For residents of these areas, the risk of death by any cause on any given day of a heat event was not significantly elevated for any age group. However,

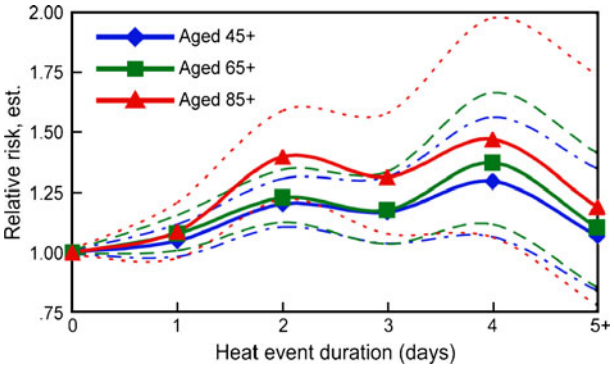

Fig. 3 Mortality relative risk estimates (*solid lines*) for circulatory causes (ICD-9: 390–459; ICD-10: I00–I99, G45, G46) by heat event duration (99th percentile), Greater Seattle Area (King, Pierce and Snohomish counties), 1980–2006. *Dotted lines* show estimated 95% confidence limits

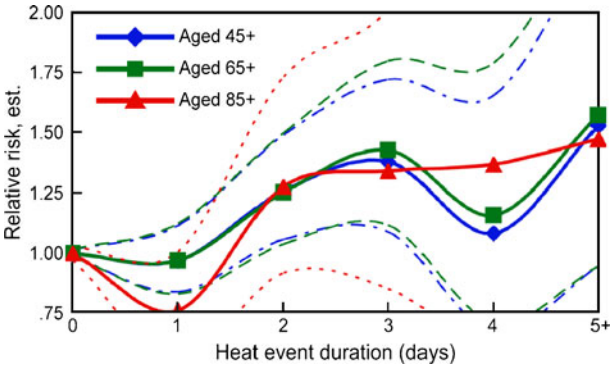

Fig. 4 Mortality relative risk estimates (*solid lines*) for respiratory causes (ICD-9: 460–519; ICD-10: J00–J99) by heat event duration (99th percentile), Greater Seattle Area (King, Pierce and Snohomish counties), 1980–2006. *Dotted lines* show estimated 95% confidence limits

risk estimates for death due to all non-traumatic causes, and for circulatory causes specifically, initially increased as the duration of heat event increased, rising from approximately 1.0 on day 1 to 1.1–1.2 on days 2 and 3, and falling back to about 1.0 on day 5 and beyond, for all age ranges. Non-traumatic death risk estimates on days 2 and 3 for persons ages 45 and above approached statistical significance (RR = 1.07 95% CI 0.96–1.19 and 1.12 95% CI 0.96–1.31, respectively). Relative risks were more variable for death due to respiratory causes and followed no clear pattern. The overall relative risk of death due to non-traumatic causes was 1.03 for persons ages 65 and above and 1.02 for persons ages 85 and above, for elevated risks of death during heat events of 3% and 2%, respectively, compared with more temperate periods. For circulatory causes, overall RRs were 1.06 for persons ages 65 and over and 1.10 for those ages 85 and over, indicating elevated risks during heat events of 6% and 10%, respectively (not shown in tables).

Relative risks of death during heat events were examined for all three eastern study areas individually as well (Table 3). No statistically significant excess risk for the cause- and age-groups considered was observed and confidence intervals were much wider due to smaller population size, although a few patterns emerged. In Spokane, relative risks for non-traumatic cause-of-death remained close to 1.0, for all age ranges, wherein point estimates for the RR were approximately 1.0 on day 1, they increased to 1.1 on days 2 and 3 (95% CI 0.9–1.4 for ages 45+ and 65+) and then decreased to 0.9 on day 5 and beyond. Relative risks for circulatory cause-of-death followed a similar pattern. In the Tri-Cities, elevated relative risk of death by all non-traumatic or circulatory causes for persons 45 years of age and older approached statistical significance on day 1 (RR = 1.1; 95% CI 0.9–1.3 and RR = 1.1; CI 0.9–1.4, respectively). In Yakima, relative risk of death for all non-traumatic causes or by circulatory causes peaked on day 5 for persons ages 45 and above (RR = 1.3 and 1.4; 95% CI 0.9–1.8 and 0.8–2.1, respectively). In general, although not statistically significant, the estimates suggested an increased risk of death for all non-traumatic causes and circulatory causes among persons ages 45 and above.

3.2 Projected mortality due to heat events: 2025–2085

Projected population and climate factors are shown in Table 4. Population projections for Washington State indicate an expected increase in total population between

Table 3 Mortality relative risks for selected causes and age groups by heat event duration, Spokane, Tri-Cities and Yakima, 1980–2006

Day	Spokane					Tri-Cities					Yakima				
of heat event	1	2	3	4	5+	1	2	3	4	5+	1	2	3	4	5+
All non-traumatic causes															
Aged	1.0	1.1	1.1	0.9	0.9	1.1	1.1	1.0	1.0	1.3	1.0	1.0	1.1	1.2	1.3
45+	(0.9,1.1)	(0.9,1.3)	(0.9,1.4)	(0.6,1.3)	(0.6,1.1)	(0.9,1.3)	(0.8,1.4)	(0.7,1.5)	(0.6,1.6)	(0.8,2.1)	(0.8,1.1)	(0.8,1.3)	(0.8,1.5)	(0.8,1.7)	(0.9,1.8)
Aged	1.0	1.1	1.1	0.9	0.9	1.0	1.1	1.2	1.1	1.1	0.9	1.0	1.1	1.2	1.2
65+	(0.9,1.1)	(0.9,1.3)	(0.9,1.4)	(0.6,1.3)	(0.6,1.2)	(0.8,1.2)	(0.8,1.5)	(0.7,1.8)	(0.6,1.9)	(0.6,2)	(0.8,1.1)	(0.8,1.2)	(0.8,1.5)	(0.8,1.8)	(0.8,1.7)
Aged	1.0	1.1	1.1	1.0	0.9	1.0	1.0	1.5	0.7	1.5	0.9	0.9	1.0	1.4	1.0
85+	(0.8,1.2)	(0.8,1.4)	(0.7,1.6)	(0.5,1.7)	(0.5,1.5)	(0.7,1.4)	(0.6,1.7)	(0.7,2.9)	(0.2,2.2)	(0.4,3.7)	(0.7,1.3)	(0.6,1.4)	(0.5,1.7)	(0.7,2.5)	(0.4,2.1)
Circulatory															
Aged	1.0	1.1	1.1	0.9	0.8	1.1	1.1	1.1	1.3	1.4	1.0	0.9	1.2	1.0	1.4
45+	(0.9,1.2)	(0.9,1.4)	(0.8,1.5)	(0.5,1.5)	(0.5,1.2)	(0.9,1.4)	(0.7,1.6)	(0.6,2)	(0.6,2.4)	(0.5,2.6)	(0.8,1.2)	(0.6,1.2)	(0.8,1.8)	(0.5,1.8)	(0.8,2.1)
Aged	1.1	1.1	1.1	1.0	0.8	1.0	1.2	1.3	1.3	1.0	1.0	0.9	1.2	1.1	1.3
65+	(0.9,1.2)	(0.9,1.4)	(0.8,1.6)	(0.6,1.7)	(0.5,1.3)	(0.7,1.3)	(0.8,1.7)	(0.7,2.3)	(0.6,2.6)	(0.3,2.4)	(0.7,1.2)	(0.6,1.3)	(0.7,1.8)	(0.5,1.9)	(0.7,2.1)
Aged	1.1	1.1	1.2	1.0	0.8	1.2	0.8	1.7	0.9	1.8	1.0	1.0	1.0	1.2	1.4
85+	(0.8,1.4)	(0.8,1.6)	(0.7,2)	(0.4,2.1)	(0.3,1.6)	(0.8,1.8)	(0.3,1.7)	(0.6,3.8)	(0.1,3.1)	(0.4,5.2)	(0.7,1.5)	(0.6,1.7)	(0.4,2)	(0.4,2.8)	(0.5,3.1)
Respiratory															
Aged	0.9	1.0	0.9	0.2	0.9	0.8	1.7	1.4	1.3	0.0	0.7	0.5	0.7	0.8	0.8
45+	(0.7,1.3)	(0.6,1.6)	(0.4,1.8)	(0,1.2)	(0.3,1.9)	(0.4,1.5)	(0.8,3.2)	(0.3,4)	(0.2,4.9)	(0,3.4)	(0.3,1.2)	(0.1,1.3)	(0.1,2.1)	(0.1,2.9)	(0,2.5)
Aged	0.9	1.1	1.0	0.0	0.8	0.8	1.7	1.6	1.6	0.0	0.6	0.3	0.8	0.9	0.9
65+	(0.6,1.3)	(0.7,1.6)	(0.4,2)	(0,0.9)	(0.3,1.9)	(0.3,1.5)	(0.8,3.4)	(0.3,4.6)	(0.2,5.7)	(0,4)	(0.3,1.2)	(0,0.9)	(0.2,2.3)	(0,3.3)	(0.1,3.3)
Aged	0.6	1.3	0.8	0.0	0.5	0.4	2.5	0.0	0.0	0.0	0.7	0.8	0.8	2.6	1.3
85+	(0.2,1.1)	(0.5,2.5)	(0.1,2.7)	(0,2.6)	(0,2.6)	(0,2.1)	(0.5,7.2)	(0,7.6)	(0,11.3)	(0,17.4)	(0.1,1.9)	(0.1,2.8)	(0,4.2)	(0.3,9.4)	(0,7.1)

Table 4 Projected climate[a] and population parameters

	Greater Seattle Area			Spokane			Tri-Cities			Yakima		
	2025	2045	2085	2025	2045	2085	2025	2045	2085	2025	2045	2085
Population (in thousands)												
Total	4,091	–	–	561	–	–	293	–	–	287	–	–
45 to 64	980	–	–	131	–	–	62	–	–	59	–	–
65 to 84	638	–	–	86	–	–	36	–	–	33	–	–
85 and above	73	–	–	11	–	–	4	–	–	5	–	–
Low summer warming												
Mean high humidex,	24.0	24.4	25.1	26.9	27.2	27.8	28.7	29.0	29.6	25.6	25.9	26.5
°C (°F), May–September	(75.2)	(75.9)	(77.2)	(77.2)	(81.0)	(82.0)	(83.7)	(84.2)	(85.3)	(78.1)	(78.6)	(79.7)
Expected annual heat events	2.6	3.1	3.8	2.5	2.9	3.2	2.5	2.9	3.3	2.5	3.0	3.4
Mean(max) event duration in days	2.2 (6)	2.3 (7)	2.3 (8)	2.3 (9)	2.6 (9)	2.7 (9)	2.4 (9)	2.5 (12)	2.6 (13)	2.4 (11)	2.5 (13)	2.6 (13)
Moderate summer warming												
Mean high humidex,	24.8	25.8	27.5	27.6	28.5	30.1	29.4	30.2	31.7	26.2	27.1	28.6
°C (°F), May–September	(76.6)	(78.4)	(81.5)	(81.7)	(83.3)	(86.2)	(84.9)	(86.4)	(89.1)	(79.2)	(80.8)	(83.5)
Expected annual heat events	3.6	4.7	7.2	3.2	4.1	6.0	3.2	4.2	5.9	3.2	4.3	5.9
Mean (max) event duration in days	2.3 (7)	2.6 (14)	2.9 (18)	2.6 (9)	3.0 (14)	3.4 (17)	2.7 (13)	3.0 (14)	3.6 (17)	2.8 (13)	2.9 (14)	3.5 (17)
High summer warming												
Mean high humidex,	26.3	28.1	31.3	29.0	30.6	33.5	30.6	32.2	34.8	27.5	29.1	31.8
°C (°F), May–September	(79.3)	(82.6)	(88.3)	(84.2)	(87.1)	(92.3)	(87.1)	(90.0)	(94.6)	(81.5)	(84.4)	(89.2)
Expected annual heat events	5.8	8.8	10.1	4.8	6.6	8.4	4.9	6.9	8.9	5.2	6.8	9.4
Mean(max) event duration in days	2.7 (18)	3.2 (18)	6.1 (57)	3.4 (16)	3.8 (17)	5.6 (50)	3.5 (16)	3.9 (24)	5.6 (50)	3.4 (17)	3.9 (24)	5.4 (42)

[a] See companion paper for description of low, medium, and high summer warming scenarios (Mote and Salathé 2010)

2006 and 2025 of 14% to 21%. The 65–84 age group is expected to grow fastest in all areas; this age group is expected to grow by 121% in the greater Seattle area, by 84% in Spokane and the Tri-Cities, and by 49% in Yakima. The expected number and duration of heat events above the humidex historical 99th percentile thresholds are also expected to increase. Under the moderate warming scenario, the greater Seattle area can expect 3.6 heat events with a mean duration of 2.3 days, and in 2085 this will increase to 7.2 heat events of 2.9 days mean duration. Spokane can expect approximately 3.2 heat events of 2.6 days mean duration in 2025, and 6.0 heat events of 3.4 days mean duration in 2085.

The mean numbers of excess deaths that can be expected annually from heat events above the 99th percentile are presented in Table 5 for the greater Seattle area, and for Spokane, the Tri-Cities and Yakima combined, holding population constant at 2025 projected levels. Holding the population level constant allows for the comparison of excess deaths due to heat events alone, without introducing uncertainty in the population projections beyond 2025, which are increasingly speculative. Under a climate scenario that yields relatively low summer (May–September) warming, during heat events the greater Seattle area can expect 68 excess deaths in 2025, and 89 excess deaths in 2045 and 107 excess deaths in 2085 from all non-traumatic causes among persons 45 years of age and older, as compared with more temperate periods. Under the moderate warming scenario, which is considered the most reliable estimate, Seattle can expect 101 excess deaths in 2025, 156 excess deaths in 2045 and 280 excess deaths in 2085 from all non-traumatic causes among adults 45 and above. Under the highest warming scenario, 211 excess deaths in 2025, 401 excess deaths in 2045 and 988 excess deaths in 2085 are expected during extreme heat in the same cause- and age-group. The bulk of all non-traumatic deaths will happen in persons 65 years old or older, with approximately one third to one half of these occurring among those ages 85 and above. Under the moderate scenario, just under half of all excess deaths in the greater Seattle area will occur by circulatory failure, and about 1 in 7 will be due to respiratory failure.

In the combined eastern study areas, 12 to 31 excess deaths by non-traumatic causes in persons ages 45 and older are expected in 2025, depending on the scenario. By 2085, this same age-cause group is expected to yield between 17 and 76 excess deaths. As in Seattle, the greatest number of non-traumatic annual deaths among the population ages 45 and above will occur among persons ages 65 to 84. Comparatively few deaths would be expected to occur in persons 85 years of age or older due to the smaller population in this age group.

3.3 Projected mortality due to air pollution

Using the modeling framework, the forecasted change (delta) in ozone for the mid century was determined to be +5.8 ppb in King County and +6.1 ppb in Spokane County. These projections were then applied to the baseline decade measurements made at monitoring stations. The baseline decade summertime (May–September) average 8 h average maximum daily ozone concentrations for King County based on regulatory monitoring measurements was 20.7 ppb for 1997–2006. So, applying the model delta, the future ozone concentrations in the mid century are forecasted to be approximately 26.5 ppb, a 28% increase. In Spokane County, the measured

Table 5 Projected annual excess deaths by cause and age group for low, middle and high warming scenarios, population held constant at 2025 projections

	Low			Middle			High		
	2025 mean (SE)	2045 mean (SE)	2085 mean (SE)	2025 mean (SE)	2045 mean (SE)	2085 mean (SE)	2025 mean (SE)	2045 mean (SE)	2085 mean (SE)
Greater Seattle Area									
Non-traumatic deaths									
Aged 45+	68 (10)	89 (12)	107 (13)	101 (12)	156 (17)	280 (22)	211 (20)	401 (26)	988 (32)
Aged 65+	64 (9)	84 (11)	102 (12)	96 (12)	148 (17)	266 (21)	200 (19)	382 (25)	956 (32)
Aged 85+	32 (4)	40 (5)	48 (6)	46 (5)	68 (7)	117 (8)	89 (8)	160 (9)	304 (8)
Circulatory deaths									
Aged 45+	34 (5)	43 (6)	52 (6)	49 (6)	72 (7)	124 (8)	95 (8)	170 (9)	326 (8)
Aged 65+	35 (5)	45 (6)	54 (6)	51 (6)	75 (8)	130 (9)	99 (9)	178 (10)	351 (9)
Aged 85+	20 (3)	26 (3)	31 (3)	30 (3)	44 (5)	76 (5)	58 (5)	105 (6)	215 (5)
Respiratory deaths									
Aged 45+	9 (1)	11 (2)	14 (2)	13 (2)	22 (3)	44 (5)	31 (4)	66 (6)	218 (11)
Aged 65+	8 (1)	11 (2)	13 (2)	13 (2)	22 (3)	42 (5)	30 (4)	64 (6)	213 (11)
Aged 85+	1 (0)	2 (0)	2 (1)	2 (1)	4 (1)	8 (1)	6 (1)	14 (2)	53 (3)
Spokane, Tri-Cities, Yakima									
Non-traumatic deaths									
Aged 45+	12 (2)	15 (2)	17 (2)	17 (2)	24 (2)	37 (2)	31 (2)	45 (2)	76 (2)
Aged 65+	9 (1)	11 (1)	13 (1)	13 (1)	18 (2)	27 (2)	23 (2)	32 (1)	45 (2)
Aged 85+	1 (0)	1 (0)	1 (0)	1 (0)	2 (0)	3 (0)	3 (0)	4 (0)	4 (1)

ozone concentrations were higher than in King County, with a 35.5 ppb average 8 h maximum ozone concentration based on regulatory monitor data for 1997–2006. Applying the model delta predicts future ozone concentration at approximately 41.6 ppb in Spokane County, a 17% increase.

Using the health risk assessment framework, estimates of the total ozone related non-traumatic mortality and cardiopulmonary mortality as rates (per 100,000) and numbers of deaths for each county for each decade were summarized (Table 6). We estimated that the total non-traumatic mortality rate due to ozone in the recent and mid-century period for King County will increase from 0.026 (95% confidence interval 0.013–0.038) to 0.033 (95% confidence interval 0.017–0.049) (Table 6). For the same health outcome in Spokane County, the rate was 0.058 (0.030–0.085) in the recent decade and increased to 0.068 (0.035–0.100) in the mid century. The estimated annual number of May–September excess deaths in King County due to ozone in 1997–2006 is 69 (95% CI 35–102). Using projections of the future population size and ozone concentrations, this was estimated to increase to 132 (95% CI 68–195). For Spokane County the warm season excess deaths due to ozone in the recent decade were estimated to be 37 (95% CI 19–55). In mid-century this is projected to be 74 (95% CI 38–109).

The cardiopulmonary death rate per 100,000 due to ozone was estimated to increase from 0.011 (95% CI 0.005–0.017) to 0.015 (0.007–0.022) in King County, comparing the recent decade to mid-century. In Spokane, the daily cardiopulmonary death rate attributed to ozone was estimated to increase from 0.027 (95% CI 0.013–0.042) to 0.032 (95% CI 0.015–0.049) across the decades. This translates to an estimated annual 31 (95% CI 14.7–47) excess deaths in King County due to ozone in the 1997–2006 baseline period, and a projected increase in mid century annual deaths to 59 (95% CI 28–90). For Spokane, there were 18 (95% CI 9–27) estimated annual excess deaths for the baseline period due to ozone, and in the mid century this number is expected to increase to 35 (95% CI 17–54).

Table 6 Baseline decade (1997–2006) and mid-century decade (2045–2054) estimates of population size, daily ozone concentration, mortality rate due to ozone, and excess deaths due to ozone (May–September)

Estimates	King county		Spokane county	
May–September	1997–2006	2045–2054	1997–2006	2045–2054
O_3 (ppb)[a]	20.7	26.5	35.5	41.6
Population	1,758,260	2,629,160	424,636	712,617
O_3 Non traumatic mortality rate (95% CI)[b]	0.026 (0.013–0.038)	0.033 (0.017–0.049)	0.058 (0.030–0.085)	0.068 (0.035–0.100)
O_3 Cardiopulmonary mortality rate (95% CI)[b]	0.011 (0.005–0.017)	0.015 (0.007–0.022)	0.027 (0.013–0.042)	0.032 (0.015–0.049)
O_3 Non traumatic deaths (95% CI)[c]	69 (35–102)	132 (68–196)	37 (19–55)	74 (38–109)
O_3 Cardiopulmonary deaths (95% CI)[c]	31 (15–47)	59 (28–90)	18 (9–27)	35 (17–54)

[a] Average daily maximum 8 h ozone concentration
[b] Rate expressed per 100,000 for May–September with 95% confidence interval
[c] Number of deaths May–September

4 Discussion

4.1 Mortality and heat events

We found a clear relationship between heat events and elevated risk of mortality for persons ages 45 and above for the greater Seattle area during the period 1980–2006. The elevated risk was apparent for non-traumatic causes in general, and for circulatory and respiratory causes specifically. The majority of circulatory deaths were due to cardiovascular causes; an analysis of cardiovascular deaths (not presented) showed that the relative risks associated with circulatory cause-of-death were driven primarily by cardiovascular deaths. Respiratory deaths were too small in number to allow for an analysis of more specific causes. The highest relative risks were for persons ages 65 and above; relative risks for persons ages 45 to 64 were smaller (not presented) and this age group contributed relatively few excess deaths in the historical period (not shown). Analyses of age groups younger than 45 were inconclusive, as there were insufficient numbers of deaths to produce stable relative risk values (not presented). We did not attempt to extend the mortality analysis beyond the duration of the heat event itself. This approach may have missed some latent deaths if they occurred after the heat event ended. However, by limiting the analysis just to the heat event, the calculated risk estimates should be conservative because they would tend to understate the deaths attributable to the event.

In the Spokane, Tri-Cities and Yakima study areas, separately or combined, no relative risks were statistically significant. Some patterns in relative risk, however, suggest real differences in mortality rates during heat events, but with samples perhaps too small to support statistical significance.

Projected annual numbers of excess deaths in the greater Seattle area were substantial under some conditions; even under moderate summer (May–September) warming, the area can expect around 100 excess non-traumatic deaths in 2025 and more than 150 excess in 2045. The projections for the eastern Washington study areas combined were much smaller. Even when projected population were taken into account, excess deaths were much lower in Spokane, Tri-Cities and Yakima than in the greater Seattle area. This could be explained in a number of ways. The urban heat island effect may be stronger in the more densely settled Seattle area. To the extent that socioeconomic inequality is greater in urban portions of the Seattle area, this may explain the higher relative risks for mortality during heat waves.

Perhaps the best explanation is adaptation through use of residential air conditioning. Hamlet et al. (2010) recently reported that market penetration of residential air conditioning is significantly higher in the study areas east of the Cascade Mountains. Data from 1970–1999 indicated that the Spokane (24%), Tri-Cities (54%), and Yakima (21%) study areas had significantly higher percentages of residential air conditioning than the greater Seattle area (8%). According to projections for the 2020s, the disparity will grow even more as the relatively cooler Seattle study area will still have significantly lower percentages of residential air conditioning (10%) than the relatively warmer Spokane (41%), Tri-Cities (68%), and Yakima (30%) study areas. An association between lowered risks for heat related illness and higher prevalence of residential air conditioning has also been cited by a number of authors (McGeehin and Mirabelli 2001; Chestnutt et al. 1998) as a mitigating factor on heat related illness during heat events.

The numbers of excess deaths shown in Table 5 were computed from 30 realizations of daily maximum temperatures in each future time period (2025, 2045, and 2085). The variability in the estimates, due to the changing frequency and duration of heat events in the annual scenarios, is reflected in the standard error term for each value. We recognize that in using the inter-annual variation as a measure of uncertainty, not all sources of uncertainty may have been included, and therefore the standard errors likely will be artificially small. In some cases, age-specific mortality rates for certain disease categories are very close to baseline, and may not indicate a net excess. For example, the projections for circulatory deaths in the greater Seattle area show slightly fewer excess deaths in the 45+ category than in the 65+ category, because the relative risk point estimates are slightly less than unity in the age group 45–64. This probably reflects statistical uncertainties in the age-specific relative risk calculations, which in this case have confidence limits that overlapped unity. However, in the remaining categories where the relative risk estimates were significantly elevated, there are consistent trends in excess deaths across projection scenarios.

A limitation of this analysis was the use of the county as the geographic level at which mortality data were linked with climate data. This decision was driven by the ready availability of both death certificate and population data at the county level, and the substantial difficulty of creating smaller areas of analysis that were geographically stable (and therefore containing a consistent population base) for each year over the historical period. The necessity of averaging climate variables over a comparatively large area meant that local extremes in temperature and humidity were dampened, and the estimated effect of heat on mortality may have been attenuated. However, this suggests that our analysis yielded conservatively-biased estimates of the relationship between heat and mortality, and that the actual effects may be larger.

In addition, the reliability of the projections for excess deaths in each of the nine future heat regimes depends upon the reliability of both climate and population projections. The middle 2025 scenario, combining the closest time period with the average climate scenario, is the most reliable of the nine simulations. Excess death estimates using the low and high warming scenarios must be interpreted cautiously, and considered extremes bracketing the best estimate. Estimates of excess deaths for 2045 and 2085 were made using 2025 projected populations. To the extent that population continues to grow beyond 2025, particularly if more growth occurs in higher age ranges, excess death estimates reported here will prove to be conservative.

Other issues that should be mentioned concern our use of ICD-9 and ICD-10 codes to categorize deaths by cause. First, ICD-9 and ICD-10 codes are not perfectly comparable, so cause-specific rates may appear to change between years when different coding schemes were in use for no other reason than deaths are grouped somewhat differently in each system. However, we did not aim to analyze changing mortality rates over time, so the change in coding scheme is not central to the analysis. Second, since deaths are not classified as being caused by heat exposure, some inference is necessary in choosing cause-of-death groupings that are believed to be influenced by heat events. Since we cannot precisely isolate cause of deaths that are due solely or substantially to heat, inaccurate cause of death information could create potential non-differential misclassification, resulting in conservatively biased estimates of the effect of heat on mortality.

Finally, the analytic method we chose relies upon a dense population with substantial numbers of deaths each day. Members of smaller, more isolated populations may also experience elevated risk of mortality during heat events. This analysis is not sensitive enough to determine relative risks for smaller, rural locales.

4.2 Mortality and ozone

We assessed the potential health impacts of climate-related ozone changes at a locally relevant regional scale, the county, for two highly populated regions of Washington State: King and Spokane counties. Given the assumptions of our models, projected increases in ozone concentrations are expected to increase the mortality rate for non-traumatic and cardiopulmonary causes. The higher ozone concentrations and underlying mortality rates observed in Spokane County yield higher current and future decade mortality rates due to ozone than seen in the greater Seattle area. However, the relative change in ozone-related mortality is projected to be greater in King County, due to a larger relative increase in predicted ozone concentrations by mid-century.

The availability of regionally downscaled climate models and meteorological and air pollution models has provided an opportunity for this initial public health assessment of climate change and ozone in Washington State. However, the models and subsequent estimates are subject to influence based on assumptions for the underlying components, as well as the scope of available data sources. We applied a single climate change scenario-ozone model to forecast future ozone concentrations that incorporates the range of influences on ozone formation through both direct and indirect meteorological changes. Previous applications of climate change related ozone forecasting to estimate health impacts have relied on ozone projections focused on the direct impacts of climate change and do not incorporate land use/land cover projections, anthropogenic emission changes, and future boundary conditions (Knowlton et al. 2004; Bell et al. 2007).

We drew the concentration-response function for our analysis from the NMMAPS study. Several features support its selection. The effect estimates fall within the range of those reported by the National Academy of Sciences in its recent review of US based studies. The studies reviewed included analyses with multiple cities or meta-analyses. In these studies the point estimates ranged between 0.46%–1.50% increase in mortality per 10 ppb increase in 8 h ozone concentrations, with the lower and upper bounds of the confidence intervals ranging from 0.23%–2.10% (Thurston and Ito 2001; Levy et al. 2005; Stieb et al. 2002; Bell et al. 2004, 2006; Schwartz 2005; NRC 2008b). NMMAPS and the studies cited include temperature and particulate matter air pollution in the ozone concentration-response model, to remove confounding by the influence of these factors on mortality.

There is an ongoing need for better data on the portion of mortality that represents people who are at risk of death within a few days, irrespective of ozone exposure—the so-called harvesting effect. A similar pattern of latent deaths may also be occurring in respect to heat events. However, the current evidence for ozone-related health effects suggests that mortality due to ozone is not restricted to this subgroup of individuals (NRC 2008b). While individuals within the population with pre-existing disease, particularly cardiopulmonary conditions, and at the extremes of the age range are likely more vulnerable to the effects of higher ozone, the

distribution of ozone-mortality effects on subpopulations is not well characterized, unlike the overall (population-weighted) average concentration effects such as those applied in this study.

In the first study of this kind to apply regional climate model outputs to address county level public health risks (Knowlton et al. 2004), the estimated baseline decade (1990s) mortality for 31 northeast US counties was between 5 and 123 (for June- August period). This was calculated based on modeling the baseline 1990s decade ozone concentrations using a regional climate ozone model under the IPCC A2 scenario. Our baseline 1990s ozone-related mortality estimates for King and Spokane County yielded findings within this range (69 and 37, respectively for May–September period), although our baseline decade ozone concentrations were based on regulatory monitoring network measurements, rather than application of the regional model for the 1990s. We project slightly larger increases between the current decade and the mid century for Spokane County (+6.1 ppb) and for King County (+5.8 ppb) as compared to the 1–4 ppb increases reported in the northeastern county based analysis. This likely reflects that the climate change ozone model employed in the northeastern analysis did not incorporate indirect effects included in our model, such as land use/land cover projections, anthropogenic emission changes, and future boundary conditions (Knowlton et al. 2004; Bell et al. 2007). These effects would be expected to increase future ozone concentrations above the influence of more direct effects of climate on ozone.

The application of projected population increases on mortality rates had a strong influence on future mortality projections. This demonstrates the potential public health impact that even modest increases in ozone concentrations may have as the population grows, but also underscores the uncertainties inherent in an assessment such as that presented here. In the future, we plan to employ several models of climate change-ozone concentrations that differ in their underlying assumptions as they become available for our region.

5 Research gaps and recommendations for future research

Social and economic factors have been shown to influence mortality during periods of excessive heat (Greenberg et al. 1983; McGeehin and Mirabelli 2001; Browning et al. 2006). A logical next stage in the study of the effect of heat events on mortality in Washington State would be to consider socioeconomic factors (e.g., race/ethnicity, income, occupation) that influence exposure to heat and the ability to mitigate adverse effects. It would also be worthwhile to devote additional effort to the study of mitigation measures; for example, evaluation of the distribution of residential air conditioning and access to cooling at work or leisure. Such access is unlikely to be equally distributed across the state or adequately available to persons most at risk of serious illness or death.

A refinement of the estimated relationship between heat events and mortality could be made by reducing the size of the geographic unit used to link climate variables with mortality, so that a more precise approximation of the local heat history surrounding the decedent could be made. If fatalities were geocoded to census blocks, then climate variables at the grid level could be assigned to specific blocks individually, rather than averaged over a much larger area. In addition, a

variety of block-level contextual factors (e.g., neighborhood characteristics) available from census data that might be relevant to heat-related mortality risk could be linked and analyzed in concert with other factors.

Finally, this analysis considered only fatalities, the end stage of a progression of heat-induced morbidity that many individuals will not reach. A more sensitive and perhaps more revealing analysis of the effects of heat on the health and welfare of a population would consider other outcomes, such as emergency room and hospital admissions for heat-related morbidity, and even lost income and productivity due to illness.

Complexities not considered in the analysis of ozone and mortality include differences within population subgroups regarding vulnerability, housing characteristics, and activity patterns which are known to vary and may in the future vary to a greater extent. As the climate warms, people may spend more time indoors or in air conditioned settings, thereby decreasing exposure. We applied a single baseline mortality rate based on current decade, but this may change due to medical advances, access to medical care and changes in other risk factors such as smoking and diet, and aging of the population. Some acclimatization may occur but quantifying this was outside the scope of this study.

We focused on short term mortality increases due to increased ozone, but other important but less severe health conditions that are known to be influenced by short term increases in ozone include hospitalization for asthma and other chronic respiratory disease, lost work and school days due to respiratory symptoms. The adverse health consequences of chronic elevated ozone exposure on health is less well-studied, although an expanding literature suggests such exposures increase the prevalence of asthma and asthma symptoms (McConnell et al. 2002; Lin et al. 2008). Additionally, there is a well-known potential for interaction effects between high temperatures and air pollutants such as ozone. This analysis was not able to explore that, although other authors have done so (Ren et al. 2008).

In regard to ozone and mortality, the following issues need to be addressed:

- Development of a range of climate-ozone projections reflecting different assumptions regarding population growth, emission changes and land use changes would allow consideration of the range of potential changes in ozone concentration and the influence of potential future policy-making options on those changes.
- Consideration of other important health outcomes and medical/public health system burdens due to increases in ozone, such as asthma hospitalizations, asthma prevalence, lost work and school days, and cardiovascular disease events should be applied to future policy-making options
- Development of robust models forecasting regional scale changes in particulate matter (e.g. $PM_{2.5}$) and application in health risk studies in Washington State would further enhance climate-preparedness efforts.
- Better understanding of the effects of ozone on vulnerable subpopulations such as those with pre-existing diseases and certain age groups, particularly the very young and elderly.

Finally, a great deal more study is needed to understand the multiple effects of climate change on the incidence of death or illness from causes not considered in the analysis reported here. For example, the currently observed wintertime

increases in cardiopulmonary disease may be lessened with future decreases in wintertime temperatures. Characterizing this will be helpful to fully understand the global context of climate change and health in the population. These include food- and water-borne illnesses, vector-borne disease, and exposure to risk of traumatic injury and death from extreme weather events such as flooding, storm surges and sea-level rise.

6 Conclusions

Heat events are a significant factor in mortality rates during the warmer months in Washington State, especially for persons ages 65 and above. As temperatures increase and the population grows, Washington can expect an increase in the number of heat-related deaths annually. More research should be done to explore other important factors influencing the effect of heat on mortality risk in Washington, including individuals' socioeconomic status and access to cooling in very hot weather. In the last decades, overall ambient air quality has improved in Washington State through regulatory policy, but adverse health impacts have not been fully prevented. Climate change may threaten the gains that have been made in this regard. A better understanding of climate change and its effect on ambient air quality is critical to prepare for and alleviate potential public health impacts.

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