

## The impact of extreme heat on morbidity in Milwaukee, Wisconsin

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**Abstract** Given predictions of increased intensity and frequency of heat waves, it is important to study the effect of high temperatures on human mortality and morbidity. Many studies focus on heat wave-related mortality; however, heat-related morbidity is often overlooked. The goals of this study are to examine the historical observed relationship between temperature and morbidity (illness), and explore the extent to which observed historical relationships could be used to generate future projections of morbidity under climate change. We collected meteorological, air pollution, and hospital admissions data in Milwaukee, Wisconsin, for the years 1989–2005, and employed a generalized additive model (GAM) to quantify the relationship between morbidity (as measured by hospital admissions) and high

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temperatures with adjustment for the effects of potential confounders. We also estimated temperature threshold values for different causes of hospital admissions and then quantified the associated percent increase of admissions per degree above the threshold. Finally, the future impact of higher temperatures on admissions for the years 2059–2075 was examined. Our results show that five causes of admission (endocrine, genitourinary, renal, accidental, and self-harm) and three age groups (15–64, 75–84, >85 years) were affected by high temperatures. Future projections indicate a larger number of days above the current temperature threshold leading to an increase in admissions. Our results indicate that climate change may increase heat-related hospital admissions in the US urban mid-West and that health systems should include heat wave planning.

## 1 Introduction

It is well known that heat waves can cause a substantial number of deaths. For example, the 1995 Upper Midwest heat wave resulted in 700 deaths in Chicago (Semenza et al. 1996). During the same heat wave, 91 deaths and 95 paramedic emergency medical service (EMS) runs in Milwaukee were attributed to heat. A recent study by Robine et al. (2008) disclosed that more than 70,000 additional heat-related deaths occurred in Europe during the summer of 2003, more than 45,000 of which occurred in August. Further, Meehl and Tebaldi (2004) showed that future heat waves in those areas of Europe and North America will become more intense, more frequent, and longer lasting in the second half of the twenty-first century. Hayhoe et al. (2010) estimated the frequency of heat wave events comparable to 1995 in Chicago using the future projections under SRES higher (A1FI) and lower (B1) emission scenarios, and found that before the end of the century such heat waves could occur every other year on average under lower emissions and as frequently as three times per year under higher levels. Moreover, annual average heat-related mortality rates are projected to equal those of 1995 under lower emissions and reach

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double the 1995 levels under higher ones. The increased mortality over past heat waves and the predicted growth of future heat waves raise urgent questions: what is the relationship between human health and extreme heat? And how do we expect the impact of future temperature on human morbidity to change?

Many studies have quantified heat-related mortality (Kalkstein and Greene 1997; Keatinge et al. 2000; Hajat et al. 2004; Curriero et al. 2002; O'Neill et al. 2003), yet few time-series studies have addressed the effects of weather on morbidity (hospital admissions, or primary care consultations). Excess admissions for various disease and age groups were documented during the 1995 Chicago heat wave (Semenza et al. 1999). Schwartz et al. (2004) examined the morbidity time series using Medicare data (only persons  $\geq 65$ ) across 12 US cities and showed that elevated temperature was associated with increased hospital admissions for cardiovascular disease, although a recent study in European cities found that high temperatures did not increase cardiovascular admissions (Michelozzi et al. 2009). In general, high temperatures have been shown to increase emergency hospital admissions for respiratory causes, particularly in the elderly (Kovats et al. 2004). There is also good evidence that admissions for endocrine and renal disorders increase during heat waves (Knowlton et al. 2009).

The objectives of this paper are to estimate the relationship between daily temperature and morbidity in Milwaukee, WI, a city with a moderate climate, and then to use these exposure-response functions to estimate the potential effects of climate change on additional admissions due to increased hot weather.

## 2 Methods and data

We used a generalized additive model (GAM) to quantify the relationship between morbidity (as measured by hospital admissions) and temperature in Milwaukee. This analysis is coupled with additional information on potential confounders, including relative humidity and the outdoor air pollutants of tropospheric ozone and particulate matter ( $PM_{10}$ ). GAM has become the most widely applied method in time-series studies of air pollution and health data because of its flexibility in allowing for nonparametric models for non-linear confounding variables (e.g., Dominici et al. 2000).

*Hospital inpatient discharge data* Data were reported by all Wisconsin's acute care facilities, including general medical/surgical, psychiatric, alcohol and other drug abuse, rehabilitation, and state institutions, with the exception of the federal Veterans Administration facilities. Data were extracted for all admissions for the years 1989–2005 and for all persons with residential zip codes corresponding to the city of Milwaukee. Cause of admission was the primary diagnosis coded according to International Classification of Diseases (ICD) 9th revision (Table 1). The information contained age and diagnosis (cause of admission). We used the admission date in the analysis.

*Daily meteorological data* Data from 1989–2005 for a single weather station in Milwaukee were obtained from the National Climate Data Center's cooperative daily meteorological data set available at the National Center for Atmospheric Research. The location of this weather station is shown in Fig. 1. Variables obtained

**Table 1** The list of selected causes with their ICD9 codes and abbreviations

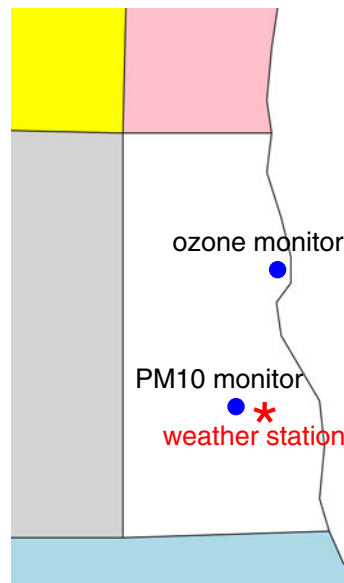
Disease	ICD9 code	Abbreviations
<b>All cardiovascular</b>	<b>390–459</b>	<b>Cardiovascular</b>
Stroke	430–438	Stroke
Ischaemic heart disease	410–414	Ischaem heart
Myocardial infarction	410–412	Myocard infarc
<b>Endocrine, nutritional and metabolic diseases</b>	<b>240–279</b>	<b>Endocrine</b>
Diabetes mellitus	250–250	Diabetes
<b>Mental and behavioural disorders</b>	<b>290–319</b>	<b>Mental disorder</b>
Mental and behavioural disorders due to psychoactive substance use of alcoholism	291–294	Psychoact mental
Schizophrenia, schizotypal and delusional disorders	295–299	Schizophrenia
<b>Diseases of the genitourinary system</b>	<b>580–629</b>	<b>Genitourinary</b>
Diseases of the renal system, including renal failure, kidney stones	580–599	Renal failure
<b>Diseases of the respiratory system</b>	<b>460–519</b>	<b>Respiratory</b>
COPD	490–496	COPD
Asthma	493–493	Asthma
Infectious respiratory	460–487	Infec Respiratory
<b>Diseases of the nervous system</b>	<b>320–359</b>	<b>Nervous</b>
<b>Accidents, injuries</b>	<b>E</b>	<b>Accidents</b>
Intentional self harm	E8, E90–94	Intention harm

The disease in bold font groups the following items in that group

included maximum and minimum temperature and maximum and minimum relative humidity (RH) for the time period 1989–2005. There were no missing values.

*Daily air pollution data* Data, which include daily observations of ozone and PM<sub>10</sub> from 1989 through 2006, were obtained from the EPA Air Quality System database.

**Fig. 1** Map of Milwaukee and locations of the weather station, the ozone monitor and the PM<sub>10</sub> monitor at which we observe meteorological data and pollution data



We analyzed pollutant data from two monitor stations that are located in central Milwaukee and have the full temporal coverage of 1989–2005. See Fig. 1 for the locations of these stations. We used the maximum of the 8-h moving average of ozone and the daily mean concentrations of PM<sub>10</sub> in our analysis. Ozone observations are only available in summer (roughly from April to October), therefore during the 17 summer seasons 286 days lack observations. The missing values mostly occur either in early May or late October, and we removed those days in our data analysis. PM<sub>10</sub> was recorded at approximately 5 to 6 day intervals; therefore we use natural cubic spline interpolation to estimate the missing daily values. This produces a good approximation of daily information for PM<sub>10</sub>, and thus permits more efficient data usage than if we were to simply ignore the missing data. The natural cubic spline interpolation is a very commonly used practice (see e.g., Ruppert et al. 2003).

For both meteorological and pollution data, we use observations at a single monitor site to represent the entire county. Monitors are typically sited to ensure that they are not biased by measuring a very localized source; furthermore, the stations available to us were centrally located and representative of an urban core area of

**Table 2** Summary statistics of daily hospital admission counts in Milwaukee from 1989 to 2005 for different causes of admission and age groups over three durations: 17 entire years, only summer seasons and only the days with temperatures above 25°C which is about the 95th percentile of the mean temperature

	Entire years				Summers (May–Oct.)				Days >25°			
	%	Mean	5th	95th	%	Mean	5th	95th	%	Mean	5th	95th
<b>All causes</b>	100	347.7	221	465	100	345.4	221.4	461	100	354.1	224.7	471.8
<b>Cardiovascular</b>	13.9	48.3	25	70	13.6	47	25	68	13.3	47	24.6	68
Stroke	2.2	7.7	3	13	2.2	7.6	3	13	2.1	7.4	3	13
Ischaem heart	4.5	15.6	7	25	4.4	15.2	6	25	4.4	15.6	6	26.3
Myocard infrac	1.8	6.2	2	12	1.8	6.1	2	12	1.8	6.4	2	13
<b>Endocrine</b>	3.1	10.6	4	18	2.9	10.1	4	17	3	10.7	4.7	18
Diabetes	1.3	4.4	1	9	1.3	4.3	1	9	1.2	4.4	1	9
<b>Mental disorder</b>	9.8	34.1	16	56	9.9	34.1	16	55	9.8	34.7	17	55
Psychoact mental	1	3.6	0	8	1.1	3.8	0	8	1	3.6	1	7
Schizophrenia	4.1	14.3	6	23	4.1	14.2	6	23	4	14.3	6.7	21
<b>Genitourinary</b>	3.7	12.7	4	23	3.7	12.9	5	23	3.9	14	4	24
Renal failure	2	7	3	12	2.1	7.2	3	13	2.3	8	3	13
<b>Respiratory</b>	7.4	25.6	14	41	6.4	22.1	12	34	5.8	20.4	12	30
COPD	1	3.3	0	7	0.8	2.9	0	6	0.8	2.8	0	7
Asthma	1.7	5.9	2	11	1.7	5.8	1	12	1.3	4.7	1	10
Infec Respiratory	3.2	11	4	21	2.4	8.2	3	14	2.2	7.8	3.7	13
<b>Nervous</b>	0.9	3.2	0	7	0.9	3.2	0	7	0.9	3.2	0	7
<b>Accidents</b>	7.3	25.4	0	54	7.5	26	0	54	7.5	26.4	0	54.3
Intention harm	6.5	22.6	0	49	6.6	22.9	0	49	6.6	23.3	0	49
0–4 years	14.6	50.7	34	69	14.5	49.9	34	67	14.6	51.7	34.7	69
5–14 years	2.7	9.5	4	16	2.8	9.7	4	17	2.7	9.4	3	15.3
15–64 years	52.3	182	110	252	52.9	182.8	112	250.6	53.5	189.4	116	256.4
65–74 years	12	41.8	20	65	11.9	41	19	63	11.9	42.3	20	64.3
75–84 years	12.5	43.3	23	62	12.3	42.3	22	62	11.9	42	22	60.7
>85 years	5.9	20.4	11	31	5.7	19.7	10	30	5.5	19.3	10	29

The disease in bold font groups the following items in that group. We focus on this list of causes as they are suspected to respond to high temperatures from the health perspective

**Table 3** Summary of observed daily meteorological and pollution statistics over entire year and summer (May–October) from 1989 to 2005, and of projected temperatures and relative humidity from 2059 to 2075

		Mean		5th percentile		95th percentile	
		Annual	Summer	Annual	Summer	Annual	Summer
Observed (1989–2005)	Temperature (°C)	9.1	17.4	−8.1	7.2	24.7	26.1
	Relative humidity (%)	71.0	70.9	51.5	53.0	90.0	89.0
	Ozone (ppm)	–	0.045	–	0.022	–	0.078
	PM10 (µg/m <sup>2</sup> )	–	24.8	–	10.0	–	49.0
Projected temperatures (2059–2075)	Mean-adjusted (°C)	11.7	20.3	−5.5	9.5	27.8	29.2
	Mean- and variance- adjusted (°C)	11.8	20.5	−5.5	9.2	28.6	30.6
Projected relative humidity (2059–2075)	Mean-adjusted (%)	71.1	69.8	51.8	52.4	90.7	88.6
	Mean- and variance- adjusted (%)	71.2	69.9	52.7	51.6	89.2	89.0

Milwaukee. However, small scale variabilities are expected and they could add some uncertainty to our results. We created additional indicator variables to allow for other (non-weather) factors that affect daily hospital admissions. The 17-year time series of hospital admission counts exhibits clear holiday effects on New Year's Day, Martin Luther King Day, Memorial Day, Independence Day, Labor Day, Thanksgiving and Christmas, and a very strong weekly pattern with more admissions on Mondays and Tuesdays and fewer admissions on Saturdays and Sundays. There was also an overall upward trend across the 17 years, possibly due to population growth.

Table 2 gives the basic summary statistics of hospital admissions for different causes of admission and age groups, and compares those statistics between 17 years as a whole, 17 summer seasons and the total days with temperatures above 25°C, which is about the 95th percentile of the mean temperature. We focus on this list of causes as they are suspected to respond to high temperatures from the health perspective. Small differences are seen between the entire year and the summer season; slight differences are also observed for days above 25°C. Daily meteorological and pollution data are summarized in Table 3.

### 3 Method

In the first part of the analysis we use the data from only the months of May to October, the warmest months in Wisconsin, as we are focusing on the impact of extreme heat. To evaluate the relationship between daily temperature and hospital admissions, we fitted a Generalized Additive Model (GAM) following the methodology of Kovats et al. (2004) and Hajat et al. (2007).

Assuming the daily admission counts are Poisson distributed, we have

$$\begin{aligned} \text{Log}(\mu) = & \beta_0 + \mu_{\text{year}} + \mu_{\text{dow}} + \mu_{\text{holiday}} + \beta_1 OZ \\ & + \beta_2 PM_{10} + s(\text{doy}) + s(\text{RH}) + s(T_{\text{avg}}), \end{aligned} \quad (1)$$

where  $\mu$  is the expectation of daily admission counts, *dow* is day of week and *doy* is day of year.  $\mu_{\text{year}}$ ,  $\mu_{\text{dow}}$  and  $\mu_{\text{holiday}}$  are treated as factors to adjust the effect from

year, day of week and holidays.  $OZ$  is the maximum of the 8-h moving average of ozone, and  $RH$  is the daily average of Relative Humidity.  $PM_{10}$  is the estimated daily average obtained by spline interpolation since the actual  $PM_{10}$  is observed only every 5 to 6 days.  $T_{avg}$  is the average temperature over the index and previous 2 days, and  $s(x)$  is a smooth function of  $x$ . All smooth terms use cubic regression spline with 3 d.f./6 months in  $s(doy)$ , 1 d.f./5<sup>0</sup> in  $s(T_{avg})$  and 1 d.f./12 in  $RH$ . We followed Kovats et al. (2004) to choose the average temperature, and the choice of the d.f. allows adequate control for unmeasured confounders such as seasonal cycles, while it leaves sufficient information from which to estimate temperature effects. More arguments and sensitivity analyses of different d.f. are discussed in Kovats et al. (2004). In principle, we should model the admission rate rather than the admission counts in the Poisson model, but here we simply model the counts since we assume the denominator is the entire population of Milwaukee. The  $\beta_0 + \mu_{year}$  can be used to adjust for the annual population variation and also  $s(doy)$  to account for the seasonal changes in the population.

Still following Kovats et al. (2004) and Hajat et al. (2007), we quantify the high temperature effects by assuming a “hockey stick” shape to the temperature-health association when the current day temperature is above a threshold. In other words, this assumes a log linear increase in risk above a temperature threshold for each cause in addition to the smooth relationship between the risk and the average temperature  $T_{avg}$ .

Then we have

$$\begin{aligned} \text{Log}(\mu) = & \beta_0 + \mu_{year} + \mu_{dow} + \mu_{holiday} + \beta_1 OZ + \beta_2 PM_{10} \\ & + \beta_3 (T - t_h) I(T - t_h \geq 0) + s(doy) + s(RH) + s(T_{avg}), \end{aligned} \quad (2)$$

where  $T$  is the current daily average temperature,  $t_h$  denotes the temperature threshold for excess morbidity, and  $I(x)$  is an indication function. The other notations are adopted directly from model (1). In model (2) we assume a log linear increase in risk when the current temperature is above  $t_h$ . To estimate this threshold, we in turn assign seven different values (24.7, 26.1, 26.65, 27.2, 27.8, 28.9, 29.45) to  $t_h$ , which correspond to the (90, 95, 96, 97, 98, 99, 99.5)th percentiles of summer daily temperatures of the entire 17 years, and then for each value we assess the significance and examine the sign of the estimated  $\beta_3$ , denoted by  $\hat{\beta}_3$ . A temperature value is considered a reasonable threshold only if  $\hat{\beta}_3$  is significantly positive for all the other temperatures in the list that are greater than or equal to such value, and additionally the p-values are significant from this value onward.

Our second goal is to conduct a sensitivity analysis to estimate a potential increase in future admissions that would occur under an illustrative climate change scenario. Here, we first describe how we estimate a future temperature distribution based on a global climate model simulation, then discuss how to quantify the impact of these temperature changes on hospital admissions.

### 3.1 Estimating future temperatures and relative humidity

To assess future climate trends we used results from time-slice experiments that were conducted for the North American Regional Climate Change Assessment Program (NARCCAP, Mearns et al. 2009). In this experiment the atmospheric component of

the Geophysical Fluid Dynamics Laboratory (GFDL) global climate model (AM2.1) (GFDL GAMDT 2004) was run for the period 1971–2000 using observed sea surface temperatures (SSTs) and sea ice boundaries. For the future period, 2041–2070, the model used the A2 SRES emissions scenario (Nakićenović et al. 2000) and sea surface temperatures based on modifications of the observed SSTs based on changes in SSTs and sea ice from the fully coupled global model (CM2.1) simulations. The spatial resolution of the time slice experiments is 50 m. These data are available at: <http://www.gfdl.noaa.gov/narccap-am2-data>.

We chose to use these global time slice experiments for our illustrative climate scenario because they are produced with relatively high spatial resolutions for the entire globe, and their current climate simulations are often less biased than those of fully coupled global climate experiments (Duffy et al. 2003), since they use observed SSTs and sea ice boundaries. The future simulation from the time slice experiments may also have a less biased response because their future lower boundary conditions are based on adjustments to the observed lower boundary conditions (Govindasamy et al. 2003).

It is important to note that we are using only one future climate scenario, and thus are not exploring all the uncertainties regarding future climate that have been identified. Chief among these are uncertainties regarding the future trajectory of emissions and concentrations of greenhouse gases and other pollutants, the uncertainties regarding how the climate system will respond to the future concentrations (usually explored through use of multiple climate models), and the natural internal variability of the climate system (i.e. the unpredictable component of the system) (Meehl et al. 2007). In future efforts we plan to consider these uncertainties, but in this paper we use a single scenario as a plausible future climate for illustrative purposes. An important element of this scenario is its high spatial resolution which brings it closer to the metropolitan scale relevant for this study.

Our purpose is to estimate the monthly temperature and relative humidity change 70 years apart using the GFDL simulation data and then to apply the estimated difference to the real data during 1989–2005 and project them to 2059–2075. This is by no means the precise temperature and humidity during 2059–2075, but rather a reasonable approach to approximate what could be the future climate given the available information. In order to achieve a credible projection, we use only the simulation data during the overlapped period 1989–2000 and its 70-year shifted period 2059–2070 to estimate the temperature and humidity change. Although 2071–2075 is beyond the range of the GFDL future period, we retain these 5 years to make adequate use of the hospital data.

We illustrate how to project the future climate variables using temperature as the example. The monthly mean temperature difference (future minus current period) from the time-slice experiments is applied to our actual temperature data set for 1989–2005 to project the future temperature in 2059–2075. This gives the mean-adjusted future temperature projection. Furthermore, the variance of the future temperature is expected to change as well; thus, in addition to mean adjustment, we can also use the variance of the past temperature to adjust our projected monthly temperatures. Note that in order to focus on only the variation of the temperatures, we consider just the variance of the detrended series obtained by deducting a smooth function from the original temperature series. We then compute the monthly ratio of the future standard deviation to past standard deviation. Finally,



we inflate the standard deviation of our detrended actual temperature data set by these monthly ratios to adjust the variance of the past temperature. Combining the two procedures of adjusting mean and variance, we obtain the mean and variance jointly adjusted temperature. Our approach to projecting the future temperature is essentially an adaptation of the ‘delta’ method, which has long been used to project future temperature in impact studies (Mearns et al. 2001). Here the same method is applied to project the future relative humidity.

### 3.2 Estimating the future impact of climate change on hospital admissions

Because we have no reliable information about future pollution, we assume that all pollution remains unchanged in the future. In addition, we assume that the estimated exposure-response model (2) still holds in the future. With these assumptions, projecting the future hospital admissions can be simply made by replacing the  $T_{avg}$ ,  $T$  and RH in model (2) by those calculated from the projected temperature and humidity, while inheriting all the parameter estimates including the threshold estimates from the observed data. Although these assumptions may not seem realistic, this exercise is useful to assess the sensitivity of hospital admissions to temperature and humidity change.

Since our paper aims to assess the sensitivity of excess hospital admissions to extremely hot temperatures, which is reflected by  $\beta_3$  in model (2), we will show the results of the future impact of climate change on hospital admissions only for the causes that have a reasonable threshold  $t_h$ .

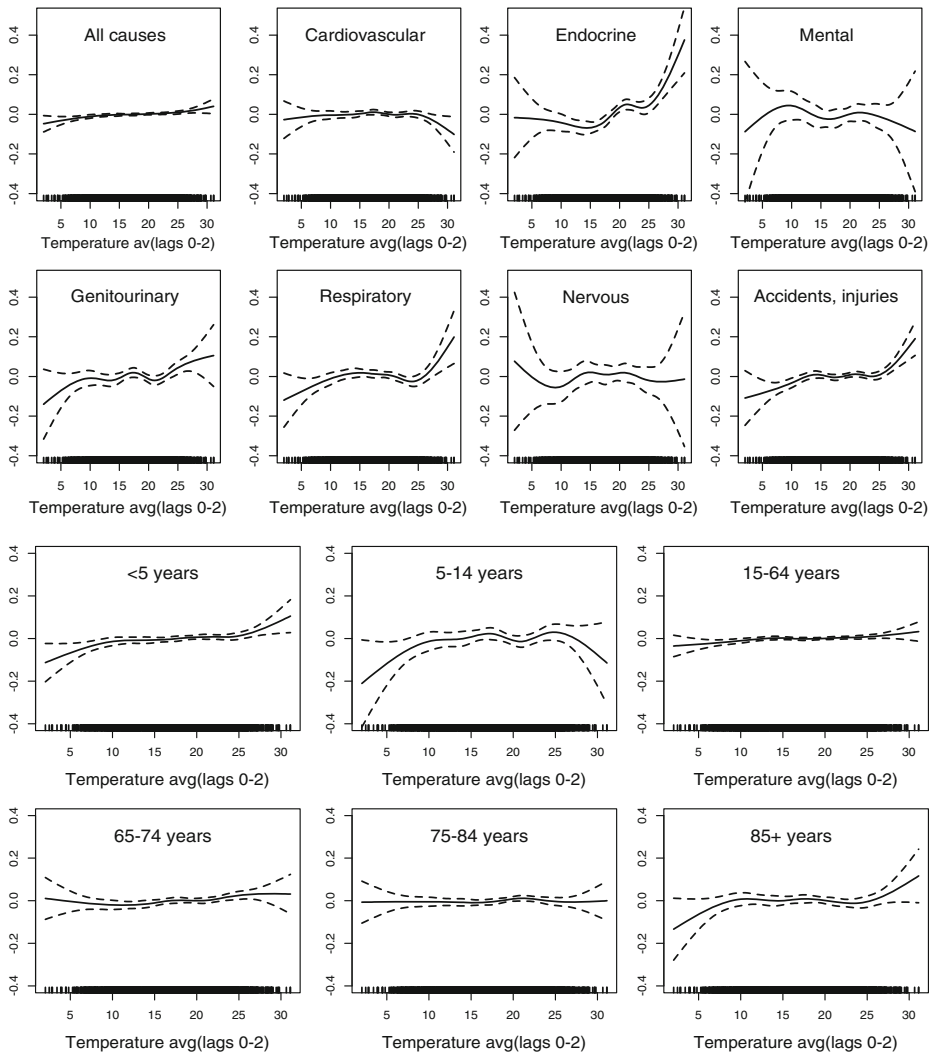
## 4 Results

### 4.1 The association between temperature and hospital admissions

Figure 2 shows the fitted relationship between hospital admissions and temperature by cause and age group, respectively. Many of these plots share the common feature that the fitted curve is flat in the middle, and then shows some pattern as temperature increases. A slight elevation of risk for high temperature is seen for all causes, and a strong increase in admissions occurs in endocrine, genitourinary, respiratory, and accidental causes. Several causes of admission are not affected by high temperature, including cardiovascular, mental and nervous system. The relationships for three age groups between 15–84 years old are basically flat, while an obvious increase is seen for children under five and the elderly over 85.

### 4.2 Quantifying the relationship

We fit model (2) for all 19 causes and six age groups listed in Table 1. For each category we examine the p-value and sign of  $\hat{\beta}_3$ , the slope parameter corresponding to the log linear term, at each of the seven percentiles of summer temperatures to determine the threshold. Table 4 illustrates how we determine the threshold using the p-value of  $\hat{\beta}_3$  corresponding to each of the 19 causes and six age groups in Table 2 and each of the seven temperature values. We see that for threshold,



**Fig. 2** Smoothed relationship between hospital admissions and temperature by cause and age group. The temperature here refers to the average of mean temperature 0–2 days preceding admission. The spline curves show the relative risk of hospital admission for temperatures across their range. The *solid line* is the estimated spline curve and the *dashed lines* wrapping the *solid line* are its 95% confidence limits. The ticks in the x-axis represent the occurrence of each “x”

$t_h = 29.45^\circ\text{C}$ , endocrine, genitourinary, renal, accidental and self-harm have positive and significant  $\hat{\beta}_3$ . Furthermore, for all  $t_h = 28.9, 27.8, 27.2$ , accidents and self-harm still have positive and significant  $\hat{\beta}_3$ . This implies that the threshold for these two causes is  $27.2^\circ\text{C}$ , while for endocrine, genitourinary and renal causes the threshold is  $29.45^\circ\text{C}$ . In summary, there is evidence of heat effects for five causes and three age groups. Their estimated thresholds and the estimated increase in admission percentages for each degree above the identified threshold value are reported in Table 5. No sensible threshold is found for all the other diseases even after we

**Table 4**  $P$ -values for  $\hat{\beta}_3$ , the slope parameter corresponding to the log linear term in model (2), for all causes and age groups in Table 2 and for seven different temperature values assigned to threshold  $t_h$ , respectively

Causes (abbreviation)	$t_h$						
	24.7	26.1	26.65	27.2	27.8	28.9	29.45
<b>All causes</b>	<b>0.180</b>	0.177	0.150	0.079	0.097	0.255	0.333
<b>Cardiovascular</b>	<b>0.247</b>	<b>0.850</b>	<b>0.907</b>	<b>0.801</b>	<b>0.775</b>	<b>0.529</b>	<b>0.245</b>
Stroke	0.379	0.375	0.346	0.376	0.410	0.705	<b>0.868</b>
Ischaem heart	<b>0.438</b>	0.750	0.640	0.764	0.791	0.715	0.957
Myocard infarac	0.984	0.254	0.208	0.230	0.215	0.286	0.443
<b>Endocrine</b>	0.216	0.415	0.556	0.888	<b>0.694</b>	<b>0.212</b>	0.045
Diabetes	<b>0.960</b>	<b>0.673</b>	<b>0.664</b>	<b>0.552</b>	<b>0.482</b>	<b>0.392</b>	<b>0.276</b>
<b>Mental disorder</b>	0.350	0.619	0.693	0.735	0.805	0.863	<b>0.989</b>
Psychoact mental	0.787	0.980	0.973	0.896	0.610	0.653	0.746
Schizophrenia	0.917	<b>0.924</b>	0.983	0.748	0.577	0.512	0.646
<b>Genitourinary</b>	0.936	0.711	0.672	0.778	<b>0.808</b>	<b>0.060</b>	0.004
Renal failure	<b>0.424</b>	<b>0.995</b>	0.795	0.764	0.931	<b>0.122</b>	0.011
<b>Respiratory</b>	<b>0.189</b>	<b>0.482</b>	<b>0.574</b>	<b>0.553</b>	<b>0.397</b>	<b>0.270</b>	<b>0.191</b>
COPD	<b>0.324</b>	<b>0.338</b>	<b>0.402</b>	<b>0.476</b>	<b>0.505</b>	<b>0.635</b>	<b>0.524</b>
Asthma	0.819	0.505	0.337	0.324	0.388	0.792	0.993
Infec Respiratory	<b>0.074</b>	<b>0.317</b>	<b>0.344</b>	<b>0.342</b>	<b>0.262</b>	<b>0.264</b>	<b>0.241</b>
<b>Nervous</b>	<b>0.140</b>	<b>0.228</b>	<b>0.291</b>	<b>0.318</b>	<b>0.364</b>	<b>0.201</b>	<b>0.164</b>
<b>Accidents</b>	<b>0.949</b>	<b>0.248</b>	<b>0.135</b>	<b>0.048</b>	0.004	0.000	0.000
Intention harm	<b>0.847</b>	<b>0.327</b>	<b>0.149</b>	0.032	<b>0.002</b>	0.000	0.000
0–4 years	<b>0.594</b>	<b>0.892</b>	0.914	0.711	0.670	0.884	0.942
5–14 years	<b>0.931</b>	<b>0.667</b>	<b>0.735</b>	<b>0.739</b>	<b>0.946</b>	0.595	0.466
15–64 years	<b>0.169</b>	<b>0.294</b>	<b>0.354</b>	<b>0.299</b>	<b>0.113</b>	0.009	0.002
65–74 years	<b>0.567</b>	<b>0.485</b>	<b>0.539</b>	<b>0.444</b>	<b>0.257</b>	0.095	<b>0.087</b>
75–84 years	<b>0.603</b>	0.947	<b>0.982</b>	<b>0.703</b>	<b>0.324</b>	0.020	0.002
>85 years	0.797	0.826	0.779	0.894	<b>0.684</b>	<b>0.071</b>	<b>0.012</b>

The  $p$ -value in roman indicates a corresponding positive  $\hat{\beta}_3$  and italic indicates a negative  $\hat{\beta}_3$ .  $P$ -values less than 0.05 are emphasized in bold. The disease in bold font groups the following items in that group

expanded the searching range for  $t_h$ . The fact that respiratory causes and the under 5-years-of-age group were not associated with a threshold might seem to contradict Fig. 2. However, this may not be surprising since Fig. 2 is used to show the smooth relationship between admissions and averaged temperature, that is,  $s(T_{avg})$  vs.  $T_{avg}$

**Table 5** Estimated thresholds and the estimated increase in admission (%) for each degree above the identified threshold value, for those causes and age groups that have a sensible threshold detected

Causes (abbreviation)	Threshold $t_h$ (°C)	% increase per °C above $t_h$
Endocrine	29.45	1.09 (1.00, 1.19)
Genitourinary	29.45	1.12 (1.04, 1.21)
Renal failure	29.45	1.13 (1.03, 1.25)
Accidents	27.2	1.02 (1.00, 1.05)
Intention harm	27.2	1.03 (1.00, 1.05)
15–64 years	28.9	1.03 (1.00, 1.05)
75–84 years	28.9	1.05 (1.01, 1.09)
>85 years	29.45	1.09 (1.02, 1.16)

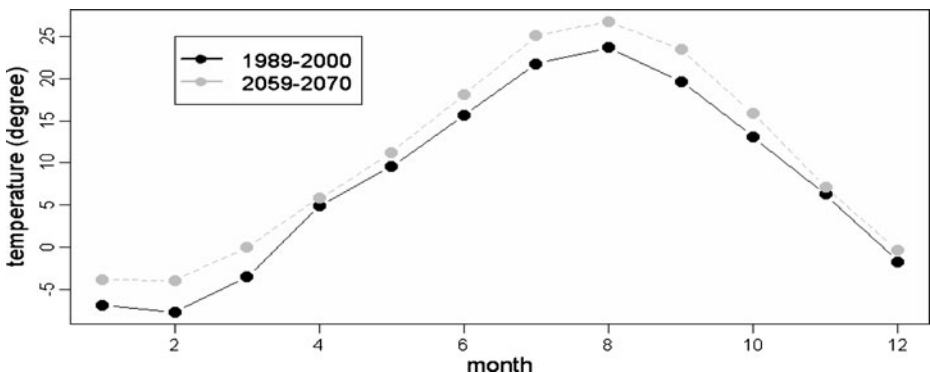
in model (1), whereas  $t_h$  in model (2) is used to describe the response to extreme current day temperatures in addition to the effect of  $s(T_{avg})$ , which have already captured the smooth increase, if any, due to the high temperatures. If the impact of high temperatures is completely captured by  $s(T_{avg})$ , there will be no evidence of an additional effect for temperature above a particular threshold on a given day.

#### 4.3 Distribution of future temperature and relative humidity

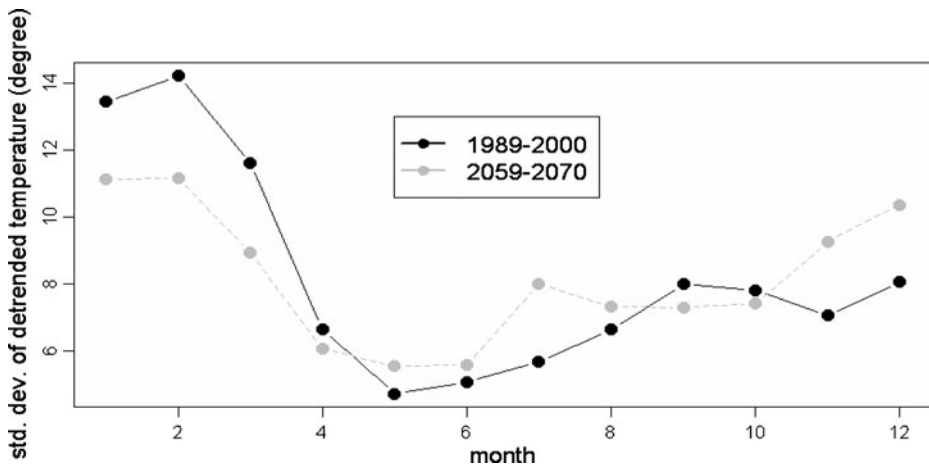
Figure 3 shows the monthly average temperature for both the past (1989–2000) and future (2059–2070) based on the future simulation described previously. A consistent increase in future temperature is observed, and in particular, the amount of increase is pronounced in July, August and September. The monthly difference is then added to our temperature data set to obtain the mean-adjusted projected temperature for the time period 2059–2075. Figure 4 shows the standard deviation of detrended past and future model temperatures. The monthly ratios of the two standard deviation sets are used to adjust the variation of our observed data set. Since only the ratios of standard deviations are of interest, the result is insensitive to the detrending procedure. The variation of future temperature is not uniformly larger than past temperature; the months May through August have greater variations. July, in particular, displays a big jump. The past and future relative humidity show somewhat different patterns in that future humidity is relatively lower in June to August but exhibits a greater variation in June and July. Table 3 gives the statistical summary of the projected temperatures and relative humidity, and it provides a comparison to variables computed using observed data over 1989–2005.

#### 4.4 Assessing the future impact of climate change on hospital admissions

Both mean-adjusted temperatures and temperatures jointly adjusted by mean and variance result in more days above the threshold, and as a consequence more hospital admissions could be expected in the future. Table 6 gives the number of days above the threshold under three different separate temperature scenarios,



**Fig. 3** Monthly average temperature over 1989–2000 and 2059–2070 from time-slice experiments conducted with the Geophysical Fluid Dynamics Laboratory (GFDL) AM2.1 model



**Fig. 4** Monthly standard deviation of detrended temperature over 1989–2000 and 2059–2070 from time-slice experiments conducted with the Geophysical Fluid Dynamics Laboratory (GFDL) AM2.1 model

and the excess admissions for both mean-adjusted and mean and variance-adjusted temperatures and relative humidity. We see that in general, adjusted temperatures come with positive admission excess as the result of future warming. Specifically, the temperature projection with both mean and variance adjusted is associated with more days above the threshold and a larger admission excess relative to the mean-adjusted temperatures. These results only assess the sensitivity of hospital admissions to change in temperature and humidity since we assumed the pollution level remains the same as in the past. We also evaluated the sensitivity to temperature change alone and found the results to be very similar.

**Table 6** Estimated daily hospital admissions by selected causes corresponding to projected temperatures and relative humidity in 2059–2075 by adjusting either only the mean or both mean and variance of temperatures and relative humidity in 1989–2005 (assuming no changes in population or age distribution)

Causes (abbreviation)	nod ( $> t_h$ )			Daily admission		
	Past	Mean-adjusted	Mean and variance-adjusted	Past	Mean-adjusted	Mean and variance-adjusted
Endocrine	18	146	235	10.13	10.55	10.86
Genitourinary	18	146	235	12.91	13.34	13.58
Renal failure	18	146	235	7.19	7.52	7.71
Accidents	95	387	486	25.98	25.86	26.13
Intention harm	95	387	486	22.93	22.84	23.13
15–64 years	33	190	278	182.76	184.71	184.98
75–84 years	33	190	278	42.32	42.25	42.27
>85 years	18	146	235	19.67	19.75	19.83

Number of days (nod) with projected temperatures above the corresponding threshold  $t_h$  for each cause and age group is also reported

## 5 Discussion

Seventeen years of hospital admission, climate and pollution data in Milwaukee, WI were analyzed to quantify the association between hospital admissions and temperatures, taking into account the effect of pollution and other meteorological parameters. Based on our analysis, admissions for a number of causes were detected to be significantly related to high temperature, and a significant relationship was also observed for several age groups.

There are many studies on the effects of heat waves on mortality, but this study focuses on the often overlooked effect of extreme heat on morbidity. Kovats et al. (2004) conducted a similar analysis of hot weather impacts on morbidity in the United Kingdom and concluded that heat waves had a small effect on total emergency hospital admissions. Both the Kovats et al. study and ours found no significant threshold for increased morbidity for all causes (total admissions); however, our study provides evidence of a potential high temperature threshold for several age groups (15–64 year, 75–84 year, and >85 year) and specific causes for hospital admissions: endocrine, genitourinary, renal, accidental injuries, and intentional self-harm. Another strength of this study is that it includes measurements of ozone and particulate matter, thus avoiding the potential confounding effect of air pollution.

The finding that renal causes of morbidity (specifically acute renal failure) are exacerbated by high temperatures is corroborated by various other studies (Knowlton et al. 2009; Nitschke et al. 2007; Kovats and Ebi 2006; Semenza et al. 1999; Hansen et al. 2008a). Not only is the renal association verified by these studies, it also makes sense physiologically: severe dehydration and hyperthermia instigated by a heat wave can damage the kidneys, leading to acute renal failure.

The association between high temperatures and endocrine admissions is also verified in the studies of Knowlton et al. (2009) and Semenza et al. (1999). Endocrine-related admissions may be affected by heat phenomena since the endocrine ICD category encompasses such causes as fluid disorders, and electrolyte and acid-base balance. These include dehydration, a diagnosis significantly related to heat waves (Semenza et al. 1999).

The accidents and injuries, and intentional self-harm causes of admissions are not consistently correlated to heat waves in other studies. Nitschke et al. (2007) found that assault-related injuries among 15–64 year-olds increased significantly during heat waves, and Page et al. (2007) suggested a relationship between high temperatures and increased risk of suicide, with a threshold effect at 18°C (Page et al. 2007). However, Semenza et al. (1999) did not observe a significant increase in accidents and injuries, and Nitschke et al. found a significant decrease in automobile accidents during heat waves in the 75+ age group (2007). Clearly, further studies are needed to assess these health outcomes.

Contrary to expectations, cardiovascular admissions were not statistically significant in relation to high temperatures since several studies have confirmed their importance in heat-related morbidity (Schwartz et al. 2004; Knowlton et al. 2009; Nitschke et al. 2007). In theory, cardiovascular admissions (versus mortality) are not higher because of the high case-fatality rate of myocardial infarctions. Our study did not include mortality data, so we cannot be certain of the comparative effects of extreme heat on morbidity versus mortality. Another somewhat surprising result was the non-significance of mental health illnesses, since some studies found them

to be an important factor involved in heat-related admissions (Nitschke et al. 2007; Hansen et al. 2008b; Kovats and Ebi 2006), but not all (e.g., Semenza et al. 1999).

It is well established that elderly populations are more susceptible to hospital admissions during heat waves, so this result is consistent with other studies (McGeehin and Mirabelli 2001; Knowlton et al. 2009; Kovats and Ebi 2006; Jones et al. 1982). Elderly people have diminished sweating capabilities (Foster et al. 1976), decreased ability for sufficient cardiac output (Wheeler 1976), and often have underlying health conditions (Jones et al. 1982), which make them more vulnerable to high temperatures.

Due to some contradictory findings and relatively small numbers of studies that focus on the morbidity effects of heat waves, more research is needed to establish the correlation between high temperatures and accidents, injuries, and mental health. A potential source of bias in comparing morbidity studies is the differing methods of determining hospital admissions: some studies only use primary discharge diagnoses whereas others use both primary and secondary diagnoses. This could create bias if, for example, a doctor diagnoses a “fluid disorder” at admission, but cardiovascular disease is the masked co-morbid condition (Kilbourne 1999). In the study by Semenza et al., inclusion of secondary diagnoses caused cardiovascular disease to be a significant cause of hospital admissions, but it was not significant when primary diagnoses alone were examined; this bias may also apply to our study.

Hospital admissions can vary over time for other reasons. For example, the “harvesting effect” and acclimation are the best known factors. The harvesting effect is due to a compensatory decrease in overall mortality during the weeks following a heat wave, when a substantial number of deaths occur acutely within 1 to 2 days. Such compensatory reduction suggests that heat affects especially those who are so frail that they “would have died in the short term anyway.” See Zanobetti et al. (2000) and Schwartz (2001) for an account of harvesting effect. Acclimatization is the process by which an organism adjusts to chronic change in its environment (Kalkstein and Davis 1989); this often involves temperature, moisture and food, and is related to seasonal climate changes. Knowlton et al. (2009) stated that the acclimatized assumptions for the 2050s reduced the potential increases in mortality by about 25% relative to the nonacclimatized results, similar to the 20% to 25% acclimatization effects in Hayhoe et al. (2004). The potential impacts of harvesting effect and acclimatization remain highly uncertain, therefore we exclude these in our analysis.

Our evaluation of the impact of future temperature change is a sensitivity analysis using just one illustrative future climate scenario. We have sought to estimate the sensitivity of morbidity projections within the range of what might be expected due to global climate change. No general conclusions regarding the likelihood of a specific magnitude of increased morbidity can be made on the basis of one scenario but this work does provide the foundation for a next step to develop a suite of projections using a range of climate model simulations and scenarios.

It is a fairly standard practice in climate impact studies to use the “delta” method (Mearns et al. 2001), that is, to apply changes of climate at a grid box scale to a single observational point. This raises concerns that the variability of the real climate at the single point is underestimated. However, we used the NARCCAP results because of the relatively high resolution of the simulations, specifically a 50 km grid (Most AOGCMs have grid sizes between 200 and 300 km). There may be some discrepancy between the NCARCCAP gridded data when compared to a point

location. We have used the highest resolution simulations available for this area. Rigorous consideration of spatial misalignment can be seen in Peng and Bell (2010).

### 5.1 Future studies/implications

The Intergovernmental Panel on Climate Change predicts higher temperatures, more frequent heat waves (Meehl et al. 2007) and an increase in heat-related morbidity (Confalonieri et al. 2007). Similarly, the global climate model from this study indicates that monthly average temperatures are projected to increase in the 2059–2070 time period, especially in the summer months. Given the link between high temperatures and certain hospital admissions, public health strategies should focus on prevention efforts by targeting groups at risk, especially the elderly and those populations with preexisting co-morbidities.

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