

Dear Author,

Here are the proofs of your article.

- You can submit your corrections **online**, via **e-mail** or by **fax**.
- For **online** submission please insert your corrections in the online correction form. Always indicate the line number to which the correction refers.
- You can also insert your corrections in the proof PDF and **email** the annotated PDF.
- For fax submission, please ensure that your corrections are clearly legible. Use a fine black pen and write the correction in the margin, not too close to the edge of the page.
- Remember to note the **journal title**, **article number**, and **your name** when sending your response via e-mail or fax.
- **Check** the metadata sheet to make sure that the header information, especially author names and the corresponding affiliations are correctly shown.
- **Check** the questions that may have arisen during copy editing and insert your answers/ corrections.
- **Check** that the text is complete and that all figures, tables and their legends are included. Also check the accuracy of special characters, equations, and electronic supplementary material if applicable. If necessary refer to the *Edited manuscript*.
- The publication of inaccurate data such as dosages and units can have serious consequences. Please take particular care that all such details are correct.
- Please **do not** make changes that involve only matters of style. We have generally introduced forms that follow the journal's style. Substantial changes in content, e.g., new results, corrected values, title and authorship are not allowed without the approval of the responsible editor. In such a case, please contact the Editorial Office and return his/her consent together with the proof.
- If we do not receive your corrections **within 48 hours**, we will send you a reminder.
- Your article will be published **Online First** approximately one week after receipt of your corrected proofs. This is the **official first publication** citable with the DOI. **Further changes are, therefore, not possible.**
- The **printed version** will follow in a forthcoming issue.

#### **Please note**

After online publication, subscribers (personal/institutional) to this journal will have access to the complete article via the DOI using the URL: [http://dx.doi.org/\[DOI\]](http://dx.doi.org/[DOI]).

If you would like to know when your article has been published online, take advantage of our free alert service. For registration and further information go to: <http://www.springerlink.com>.

Due to the electronic nature of the procedure, the manuscript and the original figures will only be returned to you on special request. When you return your corrections, please inform us if you would like to have these documents returned.

# Metadata of the article that will be visualized in OnlineFirst

---

ArticleTitle	Seasonal variability in heat-related mortality across the United States	
--------------	---	--

---

Article Sub-Title		
-------------------	--	--

---

Article CopyRight	Springer Science+Business Media B.V. (This will be the copyright line in the final PDF)	
-------------------	--	--

---

Journal Name	Natural Hazards	
--------------	-----------------	--

---

Corresponding Author	Family Name	<b>Sheridan</b>
	Particle	
	Given Name	<b>Scott C.</b>
	Suffix	
	Division	Department of Geography
	Organization	Kent State University
	Address	413 McGilvrey Hall, Kent, OH, 44242, USA
	Email	ssherid1@kent.edu

---

Author	Family Name	<b>Kalkstein</b>
	Particle	
	Given Name	<b>Adam J.</b>
	Suffix	
	Division	Department of Geography and Environmental Engineering
	Organization	United States Military Academy
	Address	West Point, NY, USA
	Email	

---

Schedule	Received	13 January 2010
	Revised	
	Accepted	16 March 2010

---

Abstract	<p>This study examines the seasonal variability in the heat mortality relationship across 29 US metropolitan areas from 1975 to 2004 to discern the seasonal cycle of the health risk from anomalously high temperatures (relative to the time of season). Mortality data for the 30-year period are standardized to account for population trends and overall seasonal and interannual variability. On days when a city experienced an “oppressive” air mass, mean anomalous mortality was calculated. Results show that while the greatest overall health impact is found mid-summer in many locations due to the peak frequency of hot weather occurring at this time, the relative increase in acute mortality on oppressive air mass days is actually just as large in spring as it is in summer, and in some cases is larger. Late summer and autumn vulnerability to anomalously warm or hot days is much less significant than spring days in all areas except along the Pacific coast. Results show significant spatial variability, with the most consistent results across the more ‘traditionally’ heat vulnerable areas of the Midwestern and northeastern US, along with the Pacific Coast. Elsewhere, the seasonal cycle of the correlation between anomalously high temperatures and human health is more ambiguous.</p>	
----------	--	--

---

Keywords (separated by '-')	Heat watch-warning system - Atmospheric hazards - Heat-related mortality - Seasonal cycle - Heat vulnerability	
-----------------------------	--	--

---

Footnote Information		
----------------------	--	--

---

Journal: 11069  
Article: 9526



## Author Query Form

**Please ensure you fill out your response to the queries raised below  
and return this form along with your corrections**

Dear Author

During the process of typesetting your article, the following queries have arisen. Please check your typeset proof carefully against the queries listed below and mark the necessary changes either directly on the proof/online grid or in the 'Author's response' area provided below

Query	Details required	Author's response
1.	Following citation IDs are not cited in text: [CR22 CR39] {CR22} A Le Tertre, A Lefranc, D Eilstein, C Declercq, S Medina, M Blanchard, B Chardon, P Fabre, L Filleul, JF Jusot, L Pascal, H Prouvost, S Cassadou, M Ledrans, (2006) {CR39} P Vaneckova, PJ Beggs, CR Jacobson, (2010)	



2 **Seasonal variability in heat-related mortality across**  
3 **the United States**

4 **Scott C. Sheridan · Adam J. Kalkstein**

5 Received: 13 January 2010 / Accepted: 16 March 2010  
6 © Springer Science+Business Media B.V. 2010

7 **Abstract** This study examines the seasonal variability in the heat mortality relationship  
8 across 29 US metropolitan areas from 1975 to 2004 to discern the seasonal cycle of the  
9 health risk from anomalously high temperatures (relative to the time of season). Mortality  
10 data for the 30-year period are standardized to account for population trends and overall  
11 seasonal and interannual variability. On days when a city experienced an “oppressive” air  
12 mass, mean anomalous mortality was calculated. Results show that while the greatest  
13 overall health impact is found mid-summer in many locations due to the peak frequency of  
14 hot weather occurring at this time, the relative increase in acute mortality on oppressive air  
15 mass days is actually just as large in spring as it is in summer, and in some cases is larger.  
16 Late summer and autumn vulnerability to anomalously warm or hot days is much less  
17 significant than spring days in all areas except along the Pacific coast. Results show  
18 significant spatial variability, with the most consistent results across the more ‘tradition-  
19 ally’ heat vulnerable areas of the Midwestern and northeastern US, along with the Pacific  
20 Coast. Elsewhere, the seasonal cycle of the correlation between anomalously high  
21 temperatures and human health is more ambiguous.

22 **Keywords** Heat watch-warning system · Atmospheric hazards · Heat-related mortality ·  
23 Seasonal cycle · Heat vulnerability

24 **1 Introduction**

25 The body of literature that evaluates the effects of oppressive heat upon human health has  
26 grown substantially over recent years (Kovats and Hajat 2008; O’Neill and Ebi 2009),  
27 especially in the wake of infamous heat events, such as the Chicago heat wave of 1995

---

A1 S. C. Sheridan (✉)  
A2 Department of Geography, Kent State University, 413 McGilvrey Hall, Kent, OH 44242, USA  
A3 e-mail: ssherid1@kent.edu

A4 A. J. Kalkstein  
A5 Department of Geography and Environmental Engineering, United States Military Academy,  
A6 West Point, NY, USA



Author Proof

28 (e.g. Palecki et al. 2001), the European heat wave of 2003 (e.g. Le Tertre et al. 2006), and  
29 the California heat wave of 2006 (e.g. Knowlton et al. 2009). Studies have approached the  
30 heat–health relationship from a variety of frameworks, from public health (e.g. Ebi and  
31 Schmier 2005) to epidemiological (e.g. Anderson and Bell 2009) and applied climato-  
32 logical (e.g. Vaneckova et al. 2008). Vulnerability has been historically assessed largely  
33 through mortality data aggregated to the city level or greater. However, within-city  
34 analyses are becoming increasingly common along with improved geo-coded health data  
35 availability (e.g. Harlan et al. 2006; Bassil et al. 2009; Vaneckova et al. 2010). Non-lethal  
36 outcomes, such as emergency room visits or ambulance calls, are also becoming more  
37 readily studied as data availability continues to improve (e.g. Knowlton et al. 2009;  
38 Michelozzi et al. 2009). Spatial variability has been assessed across different metropolitan  
39 areas within the developed world (e.g. Medina-Ramon and Schwartz 2007); international  
40 comparisons are also becoming more common, including locations within the developing  
41 world (e.g. McMichael et al. 2008; Hajat et al. 2005).

42 Numerous studies have highlighted a general winter-high/summer-low mortality cycle  
43 across much of the planet (e.g. Davis et al. 2004; Moore 2006), yet the impact of weather  
44 on this pattern remains poorly understood. The human response to extremes in temperature  
45 vary considerably; excessive heat often results in large, abrupt increases in human mor-  
46 tality while the response to cold is less pronounced, making direct weather–health rela-  
47 tionships between cold and human mortality difficult to detect (e.g. Laschewski and  
48 Jendritzky 2002).

49 Despite the strong relationship between heat and human health, interannual, inter-, and  
50 intra-seasonal variability in the heat–health response has been less frequently studied.  
51 Research has shown a significant decrease in heat-related mortality over time (e.g. Davis  
52 et al. 2003; Donaldson et al. 2003; Sheridan et al. 2009) a trend attributed to increased air-  
53 conditioning and improved health care, especially in developed nations. More specifically,  
54 Toulemon and Barbieri (2008) analyzed death rates in France before and after the Euro-  
55 pean heat wave of 2003 and suggest that a new baseline was established in the wake of the  
56 heat event, suggesting that greater concern for the elderly in the wake of the event may  
57 play a significant role for years afterward. Two recent studies, Rocklöv et al. (2009) and  
58 Stafoggia et al. (2009), uncovered for Sweden and Italy, respectively, an inverse rela-  
59 tionship between the previous winter’s mortality and the following summer’s heat–health  
60 relationship. Their research strongly suggests that there is a most-susceptible pool of  
61 individuals that oscillates in size over time, becoming smaller after significant extreme  
62 weather events.

63 Beyond epidemiological interests, understanding temporal variability in the heat–health  
64 relationship is important for intervention purposes as well as validation of predictive  
65 models. The flourishing of heat watch-warning systems (HWWS) throughout the devel-  
66 oped world represents an important component in programs to reduce heat-related vul-  
67 nerability (e.g. Sheridan and Kalkstein 2004; Kovats and Ebi 2006). HWWS, though based  
68 on a variety of methodologies for identifying a heat event (Hajat et al. 2010), include  
69 mitigation activities that have been shown to reduce heat vulnerability (e.g. Ebi et al. 2004;  
70 Palecki et al. 2001). Heat mortality and morbidity mitigation activities are likely to  
71 increase in importance, as demographic and likely climate changes increase the potentially  
72 vulnerable population over the course of the twenty-first century (e.g. Luber and McGeehin  
73 2008).

74 In this study, we follow up on our earlier research (Sheridan et al. 2009) and assess the  
75 seasonal cycle of heat-related mortality for 29 cities across the US (Fig. 1; Table 1). We  
76 use the word “heat” to maintain consistency, although as we discuss below, many early



**Fig. 1** The 29 US cities whose metropolitan areas were studied in this research

77 season events with well above average temperatures may be better described as “warm”.  
78 We approach this study from an applied climatological framework, and in particular for the  
79 understanding of the seasonality of heat vulnerability for planning and decision purposes  
80 within heat stress mitigation efforts.

## 81 2 Materials and methods

### 82 2.1 Mortality data

83 At each location depicted in Fig. 1, all-cause mortality data were obtained from *National*  
84 *Center for Health Statistics* for each day over the period 1975–2004. As noted in Sheridan  
85 et al. (2009), locations with relatively complete data were selected to include all significant  
86 climate zones of the US so that the spatial variability in the heat–health relationship across  
87 US cities can be evaluated. Nearest neighbor analysis indicates that the selected cities are  
88 randomly to uniformly distributed geographically with a nearest neighbor index of 1.31.

89 All deaths that occurred in each metropolitan area (as defined by the 2000 US Census)  
90 were included. Due to the significant undercount that would result if this study solely  
91 examined deaths that a coroner explicitly attributed to heat (Dixon et al. 2005), all-cause  
92 mortality is used here, and it is a method commonly used in similar studies.

93 Daily mortality counts were standardized in several ways. First, values were age-  
94 adjusted to the population distribution of the United States in 2000 across 10 age groups  
95 (see Anderson and Rosenberg 1998 for a full description of this methodology). Thus,  
96 locations with a disproportionately high percentage of elderly inhabitants, for whom heat  
97 vulnerability is above the population mean, will have this subset population proportionally  
98 underweighted so that the overall vulnerability is not unduly biased. To adjust for popu-  
99 lation changes throughout the study period of record, US Census population totals for each



**Table 1** List of cities whose metropolitan areas are examined in this study, along with regional identification, code utilized in other tables and graphs, and the annual mean number of occurrences of oppressive air masses per season (based on the 1975–2004 study period; see Fig. 1 for map)

City	Region	Code	Threshold		Mean annual number of occurrences				
			DT <sup>a</sup>	MT <sup>b</sup>	Dec–Feb	Mar–May	Jun–Aug	Sep–Nov	Year
Albuquerque, New Mexico	Desert	ALB	+	All	0.3	0.5	2.6	2.4	5.8
Atlanta, Georgia	Central	ATL	All	+	7.8	14.0	8.1	5.8	35.6
Baltimore, Maryland	Northeast	BAL	All	+	2.4	10.1	11.6	5.1	29.2
Boston, Massachusetts	Northeast	BOS	All	+	1.0	4.8	12.8	4.5	23.1
Buffalo, New York	Midwest	BUF	All	+	1.2	7.8	4.6	2.5	16.1
Chicago, Illinois	Midwest	CHI	All	+	0.7	8.1	10.5	3.5	22.9
Cincinnati, Ohio	Midwest	CIN	All	+	3.3	11.7	4.3	4.3	23.6
Cleveland, Ohio	Midwest	CLE	All	+	1.3	9.3	7.3	3.3	21.2
Dallas, Texas	Central	DAL	All	+	11.0	14.2	18.5	11.8	55.5
Denver, Colorado	Central	DEN	+	All	0.3	0.7	1.9	0.4	3.3
Detroit, Michigan	Midwest	DET	All	+	0.6	8.6	9.1	4.2	22.5
Houston, Texas	Southeast	HOU	All	+	12.2	20.3	16.8	15.9	65.2
Kansas City, Missouri	Central	KC	All	+	4.1	10.9	12.7	7.3	35.0
Las Vegas, Nevada	Desert	LV	+	All	3.7	3.6	16.2	3.7	27.3
Los Angeles, California	Pacific	LA	All	All	32.8	19.6	7.8	18.7	78.9
Miami, Florida	Southeast	MIA	All	+	21.6	15.9	17.6	23.6	78.7
Minneapolis, Minnesota	Midwest	MIN	All	+	0.1	6.4	10.1	4.6	21.2
New Orleans, Louisiana	Southeast	NO	All	+	9.7	12.4	10.0	12.5	44.6
New York, New York	Northeast	NYC	All	+	1.7	6.7	11.9	3.9	24.2
Philadelphia, Pennsylvania	Northeast	PHI	All	+	1.7	10.8	14.5	4.9	31.9
Phoenix, Arizona	Desert	PHX	+	+	2.8	2.9	22.2	5.7	33.6
Pittsburgh, Pennsylvania	Midwest	PIT	All	+	3.2	11.8	5.9	3.4	24.2
Portland, Oregon	Pacific	POR	All	All	3.9	5.4	5.7	4.0	19.1
Saint Louis, Missouri	Central	STL	All	+	4.9	15.1	18.0	9.8	47.8
Salt Lake City, Utah	Desert	SLC	+	All	0.8	1.1	4.7	1.1	7.7
San Diego, California	Pacific	SD	All	All	24.4	16.2	10.1	16.2	66.8
San Francisco, California	Pacific	SF	All	All	16.4	14.8	2.7	6.5	40.3
Seattle, Washington	Pacific	SEA	All	All	5.8	5.8	4.3	0.8	16.6
Tampa, Florida	Southeast	TAM	All	+	16.0	15.3	7.7	12.9	52.0

<sup>a</sup> Dry Tropical; <sup>b</sup> Moist Tropical; ‘+’ signifies only the plus subset (i.e. MT+, DT+) of the air mass was considered oppressive; ‘all’ signifies that the entire air mass was analyzed. See text for additional details

age group were acquired for the years 1970, 1980, 1990, and 2000, with linear interpolation used to estimate population counts for the intervening years (see Davis et al. 2004 for a more detailed description). Last, there were 25 instances (across the 29 cities) in which known singular events unrelated to heat (e.g. high-fatality accident) produced significant outliers in daily mortality totals. These instances were identified by examining all days for which the daily adjusted mortality total for injury and poisoning deaths was greater than 10 standard deviations above the mean. These 25 days were removed from the analysis.

Author Proof



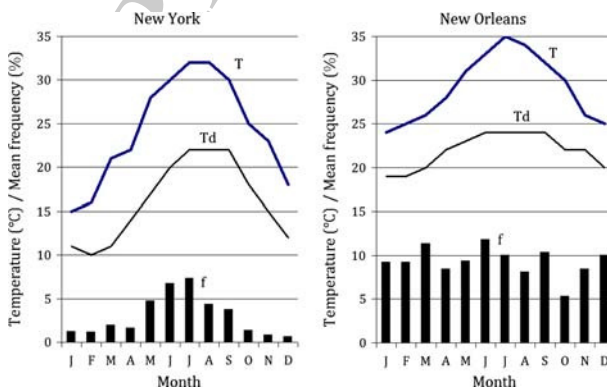
Author Proof

108 Data were then further standardized to account for the mean seasonal cycle in mortality  
109 along with interannual variability and long-term trend in overall mortality rates. The mean  
110 seasonal cycle was calculated by averaging 11-day centered-mean mortality over the entire  
111 period of record. (Other periods from 7 to 15 days were also examined, with little change  
112 to the results.) This seasonal mean mortality value for each day is then subtracted from the  
113 age-adjusted daily mortality total to create a mortality value with the seasonal trend  
114 removed. As mean mortality rates have decreased over the decades, to account for these  
115 long-term trends, the de-seasoned values are then further standardized by subtracting the  
116 3-year (centered) running mean de-seasoned mortality. This final anomalous mortality  
117 value, expressed in deaths per standardized million (DSM) population, is what is used in  
118 this study.

## 119 2.2 Weather data

120 The spatial synoptic classification (SSC) classifies each day at a location into one of a  
121 several air masses or weather types. These classifications are based on historical rela-  
122 tionships between the air masses and weather variables, namely, temperature, dew-point  
123 temperature, pressure, wind speed and direction, and cloud opacity. A thorough description  
124 of the SSC methodology is found in Sheridan (2002). In this research, the SSC is utilized to  
125 identify “oppressive air masses” (air masses or weather types for which mean mortality is  
126 statistically significantly above the summer baseline). The SSC is designed so that air mass  
127 character changes both spatially and temporally; that is, it will vary from place to place and  
128 month to month (e.g. Fig. 2). As a result, seasonal variability can be readily accounted for,  
129 and weather’s impact is determined in a relative way. This facet is key in understanding the  
130 seasonal variability in the heat–health relationship; as evidence has suggested that early  
131 season heat events may have more impact, even with lower absolute temperature values  
132 (e.g. Hajat et al. 2005; Sheridan and Kalkstein 2004).

133 Two SSC primary air masses are most commonly associated with increases in mortality,  
134 Moist Tropical (MT) and Dry Tropical (DT) (Sheridan et al. 2009; Sheridan and Kalkstein  
135 2004). MT is warm, partly cloudy, and humid, with high overnight temperatures, whereas  
136 DT is hotter and sunnier, with lower humidity. Where a primary air mass is very common,  
137 a plus subset (MT+, DT+) exists to extract only those days at least one standard deviation



**Fig. 2** Mean 1600 EST temperature ( $T$ ), dew-point temperature ( $T_d$ ), and frequency of occurrence for Moist Tropical Plus, New York and New Orleans. Climatological values of all SSC types for all cities can be found at <http://sheridan.geog.kent.edu/ssc.html>





138 above SSC seed-day means for apparent temperature. Thus, MT+ represents the warmest  
139 and most humid subset of MT days. The selection of whether to utilize the plus subsets or  
140 the entire air mass is based largely upon their usage in HWWS, and corresponds to a  
141 historical summer frequency threshold of 10%. For each city in this study, the nearest  
142 airport to downtown for which the SSC was available was utilized; Table 1 lists whether  
143 the plus thresholds were used for each city.

## 144 2.3 Analysis

145 Research has consistently shown that the impacts of heat are most significantly observed  
146 close to the heat event itself (e.g. Anderson and Bell 2009), often with 0- or 1-day lag. Both  
147 0-day and 1-day lag were assessed in the preliminary evaluations done for this research.  
148 While the results were similar, the 1-day lag results were slightly weaker in magnitude  
149 compared to the 0-day lag results. Thus, we use a 0-day lag in this research. Mean  
150 anomalous mortality in DSM per occurrence of oppressive air mass is calculated for each  
151 subset, and the significance of its difference from 0 is tested using bootstrap resampling  
152 with  $n = 10,000$ . As with our previous research, we aggregate both oppressive air masses  
153 to increase sample size. To evaluate the seasonality of the heat–health relationship, both  
154 semi-monthly and rolling 15-day subsets are examined for the calendar year. Semi-  
155 monthly analysis includes all oppressive air mass days over the 30-year period of record  
156 that fall within each of the periods shown in Table 2. The rolling 15-day subsets include all  
157 oppressive air mass days over the period of record; e.g. the mortality response in a given  
158 city for 1 June includes all oppressive air mass days between 25 May and 8 June over the  
159 period 1975–2004.

160 In addition to calculating the mean response, the overall health ‘burden’ was assessed  
161 by multiplying the anomalous mortality response by the frequency of oppressive air mass  
162 occurrence. This metric is similar to that undertaken in other research (e.g. Chestnut et al.  
163 1998) in which total heat-related mortality is estimated, although we use this different term  
164 to reflect the significant uncertainty in the level of mortality displacement. Thus, as  
165 mortality displacement is not assessed (discussed further in Sect. 4), values for ‘burden’  
166 should be thought of as relative, not absolute measures.

## 167 3 Results

168 The overall mean mortality response to oppressive air mass days varies significantly from  
169 city to city (Table 2). The highest annual mean is found in Denver (1.3 DSM), and the  
170 lowest at Salt Lake City with a value slightly below zero. Looking at the traditional  
171 summer months of June, July, and August, a wider range is observed, with Houston below  
172 0, and all other cities positive, reaching a maximum of 3.7 DSM in Chicago. Overall, in  
173 support of much previous research, the region of the US extending from the Northeast to  
174 the central plains shows the most consistent and significant response, along with several  
175 cities along the Pacific Coast. Inland west and southeastern locations are associated with a  
176 lower mean response.

### 177 3.1 Northeast

178 Of all regions, the four coastal Northeast cities are associated with the most significant and  
179 consistent increases in mortality on oppressive air mass days (Table 2; Fig. 3). These cities



**Table 2** Mean anomalous mortality by city during oppressive air mass days by semi-month

	Northeast										Midwest										Central				
	BOS	NYC	PHI	BAL	PIT	BUF	CLE	DET	CHI	MIN	CIN	BAL	PIT	BUF	CLE	DET	CHI	MIN	CIN	STL	ATL	DAL	DEN	KC	
	1-15 Jan				3.9	0.6														2.3	0.8	1.7		0.2	
16-31 Jan					-0.1															-0.3	0.0	0.4		1.5	
1-14 Feb					0.2															-0.9	1.0	0.0		-2.0	
15-29 Feb				1.9	0.2	0.1	0.4													-0.1	0.1	1.5		0.2	
1-15 Mar	-0.8	0.2	0.3	0.8	0.8	-1.1	0.4	0.1	-0.1										0.8	0.8	1.2		1.1		
16-31 Mar	0.3	0.6	1.2	1.1	0.8	1.1	-0.2	1.2	0.4										0.0	1.0	0.7		0.9		
1-15 Apr	0.0	0.4	0.1	1.9	0.2	0.0	0.4	0.8	0.4	0.6									-0.1	-0.1	1.0		0.6		
16-30 Apr	1.2	0.9	0.9	1.1	1.2	0.3	1.5	1.1	1.0	0.5									0.8	0.8	0.8		0.4		
1-15 May	1.5	1.1	1.3	1.4	0.7	1.2	1.2	1.2	1.2	0.0									1.8	0.9	0.3		0.2		
16-31 May	1.1	1.4	1.1	0.7	0.6	0.2	0.9	1.3	0.5	1.0									1.1	-0.2	0.4	2.2	-0.1		
1-15 Jun	1.9	1.7	1.0	1.5	0.6	1.2	1.0	1.3	1.0	0.2									-0.2	-0.1	0.3		1.2		
16-30 Jun	1.4	1.2	0.8	0.8	1.0	3.2	1.9	1.2	1.0	1.0									1.0	0.0	0.6	2.0	0.9		
1-15 Jul	1.3	2.1	0.6	1.7	0.7	1.9	1.4	2.1	2.3	1.0									1.2	1.4	0.8	3.4	2.4		
16-31 Jul	1.6	1.8	1.5	1.1	1.7	2.0	2.4	2.6	1.7	0.7									4.9	1.9	0.3	1.0	1.2		
1-15 Aug	1.2	1.6	1.2	0.5	0.5	3.2	1.7	2.5	1.8	0.9									2.2	0.0	0.5	-0.2	1.2		
16-31 Aug	1.6	0.7	0.2	0.2	0.6	3.6	1.8	2.6	1.5	1.6									4.0	0.2	0.6	0.8	1.1		
1-15 Sep	0.5	1.1	0.5	1.0	-0.5	0.9	0.7	1.0	0.7	-0.2									1.1	0.2	0.1	0.1	2.1		
16-30 Sep	1.1	0.6	0.7	-0.3			1.0	0.4	0.3	1.1									0.9	0.6	-0.2	0.8	-0.9		
1-15 Oct	0.2	0.6	0.7	0.0		-1.4	1.0	0.6	0.7	0.1									1.2	0.7	1.5		-1.3		
16-31 Oct	1.2	0.4	0.3	0.8	0.0	1.8	0.2	0.6	0.8	-0.1									-0.4	0.5	1.3		0.8		
1-15 Nov	0.0	-0.1	0.0	0.8	0.2		-0.6		0.8	0.7									2.0	0.1	0.6		0.0		
16-30 Nov	-0.6	-0.3	-0.4	2.5		-0.8													2.3	1.2	-0.2		0.2		
1-15 Dec				1.3	0.8														0.6	-0.4	-1.3		-1.5		
16-31 Dec					-0.4														-0.8	0.3	0.2	2.3	0.6		



Table 2 continued

Year	Midwest										Central									
	Northeast					Midwest					Central					Central				
	BOS	NYC	PHI	BAL	PIT	BUF	CLE	DET	CHI	MIN	CIN	STL	ATL	DAL	DEN	KC				
1-15 Jan	-0.9	1.1	1.2	0.8	1.1	0.6	0.9	1.0	1.0	1.3	1.0	0.6	0.9	0.5	0.5	0.6	1.3	0.8		
16-31 Jan	0.6	1.0	1.0	1.0	0.7	0.5	1.0	1.1	0.7	1.1	0.5	0.5	1.1	0.4	0.7	0.5	0.7	0.4		
1-14 Feb	1.2	1.6	1.2	0.8	0.9	2.1	1.5	1.9	3.7	0.8	0.8	0.8	0.8	0.6	1.2	0.6	1.4	1.4		
15-29 Feb	-0.2	0.7	0.4	0.4	0.2	0.9	0.8	0.7	0.6	0.4	0.6	0.5	0.6	0.5	0.4	0.5	0.1	0.5		
1-15 Mar	0.7	0.6	0.4	1.0	0.4	0.4	-1.0	1.9	0.8	1.9	0.5	0.5	0.5	0.2	0.2	0.1	0.1	0.1		
16-31 Mar	0.2	-0.2	-0.2	-0.1	-4.2	-1.6	-0.4	1.2	1.0	1.0	-1.2	0.0	0.0	0.0	0.2	0.3	0.3	0.3		
1-15 Apr	-0.4	0.2	0.5	0.1	0.3	0.2	-0.5	0.1	1.3	1.3	0.4	0.2	0.2	0.7	0.1	0.2	0.2	0.2		
16-30 Apr	0.2	0.8	0.3	0.2	0.3	0.2	-0.3	1.1	0.4	0.8	0.8	0.3	0.2	0.2	0.2	0.3	0.9	0.9		
1-15 May	0.7	0.8	0.5	0.5	0.5	0.5	-1.0	-1.0	0.8	0.8	0.8	1.5	2.1	1.1	0.1	0.2	0.2	0.2		
16-31 May	0.4	1.0	0.2	0.4	0.2	0.2	-0.3	0.2	1.6	0.9	0.9	2.0	2.0	1.1	0.2	0.5	0.5	0.5		
1-15 Jun	1.1	1.3	-0.1	0.6	-0.1	1.5	1.4	0.4	3.4	3.1	1.7	2.5	2.1	1.7	0.6	0.6	0.6	0.6		
16-30 Jun	0.9	0.9	0.0	0.7	0.3	1.6	0.3	0.0	-0.1	1.7	0.4	1.0	2.1	0.4	0.1	0.1	0.1	0.1		
1-15 Jul	1.2	0.1	0.7	0.9	0.9	0.3	0.0	0.0	-0.3	2.2	2.7	0.4	2.7	1.5	-0.3	0.7	-0.3	-0.3		
16-31 Jul	2.0	-2.3	-0.2	0.7	3.7	0.4	0.0	0.0	-0.6	3.2	3.8	1.5	3.8	1.5	0.7	0.7	0.7	0.7		
1-15 Aug	0.9	-0.7	0.7	0.9	1.4	0.8	0.5	1.3	1.2	1.2	1.3	0.8	1.3	0.8	0.4	0.4	0.4	0.4		
16-31 Aug	-0.5	0.1	-0.2	0.6	1.5	0.2	0.3	0.3	-0.6	1.0	2.3	0.9	1.0	0.9	0.4	0.4	0.4	0.4		



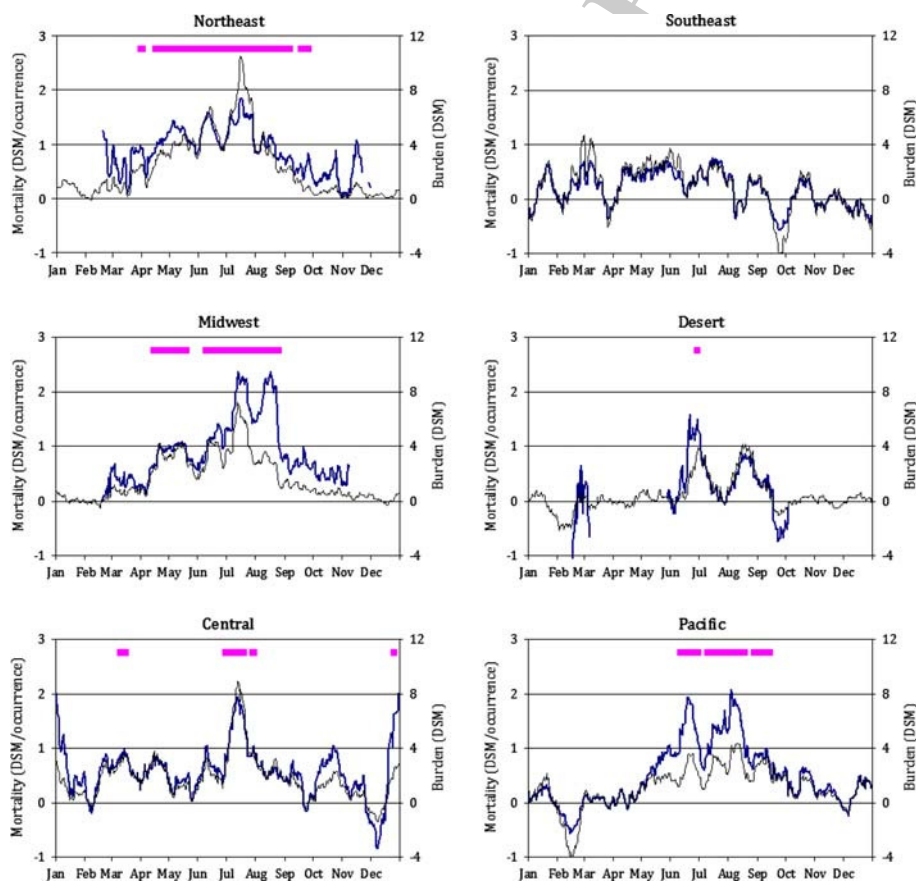
Table 2 continued

	Southeast										Desert					Pacific				
	NO	HOU	TAM	MIA	ALB	LV	PHX	SLC	SEA	POR	SF	LA	SD							
	16-30 Sep	<b>-0.9</b>	<b>-1.8</b>	-0.1	0.2	-2.7	-0.7	-0.3			-0.7	0.7	0.2	<b>1.2</b>						
1-15 Oct	0.0	-0.5	-0.1	0.4	-0.1	-0.8	0.0			1.4	<b>1.5</b>	0.2	-0.4							
16-31 Oct	0.3	0.2	0.1	<b>0.6</b>	2.6		-0.5			0.2	0.9	0.3	0.6							
1-15 Nov	0.1	-0.6	-0.1	0.2	0.3					0.7	0.1	<b>0.5</b>	-0.2							
16-30 Nov	<b>1.5</b>	0.0	<b>-0.5</b>	-0.3							0.0	0.1	<b>0.9</b>							
1-15 Dec	-1.1	0.2	0.0	-0.1						-1.2	0.1	-0.2	0.5							
16-31 Dec	-1.0	0.3	-0.4	0.1			0.2			-1.5	-0.1	<b>0.8</b>	-0.1							
Year	0.3	0.1	0.1	0.2	<b>1.1</b>	0.2	0.2	-0.1	0.3	<b>0.8</b>	0.1	0.3	0.3							
Apr-May	0.2	0.7	0.4	0.3	-1.4	-0.2	0.5	1.4	0.5	0.6	0.4	0.1	0.4							
Jun-Aug	<b>1.1</b>	-0.3	0.5	0.5	<b>1.9</b>	0.7	0.2	0.2	<b>2.1</b>	<b>2.5</b>	<b>1.2</b>	<b>1.1</b>	0.3							
Sep-Oct	-0.3	<b>-0.5</b>	-0.1	0.4	<b>0.7</b>	-0.2	0.1	-0.3	<b>1.3</b>	0.3	<b>1.2</b>	0.4	0.5							

Anomalies are deaths per standardized million. Bold values represent mortality values that are statistically significant ( $p < .05$ ). Where no value is listed, an oppressive air mass occurs less than 3% of all days within sample

180 are associated with a long heat-related mortality 'season', as all cities are collectively  
181 statistically significant from 16 April to 21 August, with several smaller periods before and  
182 after. Individually, there is an observable north–south gradient, with statistically significant  
183 increases in mortality appearing earlier (late March) in the southernmost cities of Phila-  
184 delphia and Baltimore. Similarly, heat vulnerability 'ends' earlier as well, with statistically  
185 significant increases consistent in Baltimore and Philadelphia only through early August, in  
186 comparison with late August and early September for the more northern cities of Boston  
187 and New York.

188 In terms of the magnitude of mortality response, mean mortality response (Table 2; blue  
189 line in Fig. 3) is generally above 1 DSM/occurrence (signifying a mean increase in one  
190 death per million population on the day an oppressive SSC type occurs) from April through  
191 August, with the same spatial variability noted earlier. It is interesting to note that in the  
192 marginal seasons (hereafter defined as April and May in spring, and September and  
193 October in autumn), a much larger response is observed in spring than in autumn, with  
194 mean values at all four cities at or above 1 DSM in April and May, and only around half of



**Fig. 3** Mean mortality response on oppressive SSC days (thick blue line) in deaths per standardized million (DSM); mean mortality burden (thin black line) in DSM; and statistical significance ( $p < .05$ ; magenta bar across top) over rolling 15-day periods for each of the six aggregated regions



195 that (and in many cases not statistically significant) in September and October, even though  
196 in the latter 2 months temperatures are several degrees warmer. The mean mortality  
197 increases in April and May are on par with those in mid-summer, suggesting a similar  
198 acute impact per occurrence. The 'burden', in which the frequency of occurrence is  
199 multiplied by the response, is largest in mid-summer, in particular in July, when the  
200 frequency of MT+ days is greatest.

### 201 3.2 Midwest

202 Across the seven Midwest cities, the heat response is qualitatively similar to that of the  
203 Northeast cities. Collectively, the cities exhibit statistically significant increases from 19  
204 April to 19 May, and 9 June to 25 August. There is somewhat greater variability across these  
205 cities; with the three largest cities (Cleveland, Detroit, and Chicago) more consistently  
206 associated with increases in mortality throughout the warm season than the other four.

207 In contrast to the Northeast cities, across most Midwest cities the mean mortality  
208 response is significantly higher in July and August, with mean response exceeding 2 DSM/  
209 occurrence for some portion of these months at all cities except Pittsburgh and Minne-  
210 apolis; values exceed 3 DSM/occurrence at Buffalo and Cincinnati. Though April and May  
211 heat responses are less, they are still above September and October responses at six of  
212 seven cities, and are statistically significant at five (compared to zero in September and  
213 October). The health burden is similar in shape to that of the Northeast, with a peak in July,  
214 and higher values earlier in the season compared with later.

### 215 3.3 Central

216 The seasonal cycle across the five cities of the Central region is different from the previous  
217 two regions in many aspects. In terms of aggregate significance, aside from two smaller  
218 periods, roughly only in the month of July is the increase in mortality statistically sig-  
219 nificant. In examining the cities individually, except for the July peak, there is little  
220 consistency from city to city, with a spring period of statistically significant increases in  
221 mortality occurring in St. Louis, Atlanta, and Dallas, although at different times. Other  
222 than a short period spanning the year's end, the increase in mortality in Dallas is most  
223 significant in late February and early March, among the earliest observed.

224 In terms of mean mortality response, the Central stations do not exhibit the strong  
225 seasonal pattern seen in Northeast and Midwest cities, except for the July peak. Rather,  
226 mean anomalous mortality on SSC days hovers between 0 and 1 DSM per occurrence  
227 across the aggregate of the cities, with individual cities showing substantial variability. St.  
228 Louis and Atlanta are similar in terms of response to many Northeast and Midwest cities,  
229 although with less consistency through the seasonal cycle; Denver and Kansas City are  
230 more similar to the Pacific cities discussed below; and Dallas shows greater mean  
231 responses outside the hottest time of year. As oppressive SSC types occur over a broader  
232 portion of the year here than in the Northeast and Midwest, the health burden is similarly  
233 spread out over the seasonal cycle, aside from the July peak.

### 234 3.4 Southeast

235 Across the Southeast cities collectively, the overall increase in mortality on MT+ and DT  
236 days is largely not statistically significant. Several exceptions are noted at individual cities,



237 including a period from late April through May in Houston, a general summertime period  
238 in New Orleans, and a late summer period in Miami. The mean mortality response and  
239 health burden is overall lower than with the previous regions discussed, though similar to  
240 the Central region, with the exception that there is no July peak apparent. There is,  
241 interestingly, a general downward slope throughout the warm season that is most readily  
242 apparent at all cities except for Miami. Indeed, at Tampa, New Orleans, and Houston, the  
243 oppressive SSC types are actually associated with decreases in mortality for portions of the  
244 late summer and autumn.

### 245 3.5 Desert

246 Of the six regions evaluated, the Desert region shows the least consistent results in terms of  
247 statistical significance, mortality response, and 'burden', suggesting a fairly acclimatized  
248 population. Across the cities, marginally statistically significant mortality increases are  
249 observed in Phoenix from late February through early April, in Salt Lake City in June, Las  
250 Vegas from late June to mid-July, and Albuquerque in August. Mean mortality response  
251 peaks at these same times as well. However, given the variability seen from city to city,  
252 across the region as a whole, aside from a modest double peak in late June and early  
253 August, the mortality response is near zero for the remainder of the year.

### 254 3.6 Pacific

255 In contrast to all of the other regions in this study, the Pacific region is unique in that many  
256 stations have a maximum frequency of oppressive SSC days in the winter, largely due to  
257 warm humid advection from Pacific cyclones, or downsloping Santa Ana winds in southern  
258 California. During this time of year, these well above normal temperature days are not  
259 associated with any systematic increase in mortality. Across the aggregate of the five cities,  
260 the increase in mortality is significant on nearly all days between 11 June and 14 September.  
261 At individual cities, this seasonality is largely reflected, except in San Diego and San  
262 Francisco, the latter of which experiences nearly no oppressive SSC days in mid-summer.

263 Mean mortality response is among the highest in the Pacific cities, with values near or  
264 above 3 DSM/occurrence for portions of the summer at Portland and Seattle, and 2 DSM/  
265 occurrence at Los Angeles and San Francisco. Interestingly, this is the only region for  
266 which the imbalance toward early season mortality does not appear, rather, at 4 of the 5  
267 cities, mean mortality response to oppressive weather is greater in autumn than spring, with  
268 the study's largest mean mortality response to oppressive air mass days in September and  
269 October occurring in Seattle and San Francisco.

## 270 4 Discussion and conclusions

271 It is unsurprising that across the country as a whole, mortality response on oppressive  
272 weather days is greatest in mid-summer, along with the health burden, as oppressive  
273 weather days are most common at that time of year. Across many Northeast, Midwest, and  
274 Pacific cities, this peak is fairly broad, extending across most of the traditional summer  
275 months of June through August; in other regions the peak is generally narrower.

276 More interesting is the asymmetry around the summer peak. Across most Northeast and  
277 Midwest cities, a distinct early season asymmetry is observable, with increases in mortality  
278 statistically significant in April and May, when afternoon temperatures on oppressive days



Author Proof

279 peak at values that would be close to or below normal during summer (e.g. mean 1600 EST  
280 temperature in Baltimore on oppressive air mass days in April is 27°C, whereas mean 1600  
281 EST temperature in July across all days is 30°C). In many of these cases, mean mortality is  
282 similar in magnitude to mid-summer values when the oppressive air mass occurs, although  
283 the overall health burden is lower since their occurrence is less common in April and May.  
284 Late summer heat events in these locations do not statistically significantly increase  
285 mortality in most cases.

286 This early season heat vulnerability is also observed in other areas in the Central,  
287 Southeast, and Desert regions, although there is little consistency from one city to another.  
288 Periods of statistically significant increases are observed in Phoenix and Dallas in March,  
289 and St. Louis and Atlanta in April, and Houston in late April and May. What is distinct  
290 about several of these cases (Phoenix, Dallas, and Houston) is that a summertime peak  
291 does not occur, and statistically significant increases in mortality are largely confined to  
292 spring.

293 Pacific cities, in contrast, are the only broad area where early season heat events do not  
294 seem to increase mortality; in contrast, around a summer peak similar to cities in the  
295 Midwest and Northeast, mortality tends to stay significantly above zero in the California  
296 cities through September. This may be to the asymmetry of summer weather there, with  
297 late-season Santa Ana downsloping winds leading to heat events in September that can be  
298 warmer than those during mid-summer.

299 These results are important for understanding how the population perceives heat  
300 warnings. Heat-related mortality at many cities increases significantly on oppressive days  
301 before those days' temperature values would normally lead people to believe that they may  
302 be vulnerable. Given the observations that people tend to decide whether it is too hot  
303 themselves (e.g. Sheridan 2007) and perceive the risks to others over themselves (e.g.  
304 Abrahamson et al. 2009), this early season vulnerability may need to be addressed more  
305 thoroughly in heat mitigation plans, in particular since heat watch-warning systems in the  
306 US typically begin official operation only in May.

307 Several limitations of this work need to be addressed. Foremost, this study only eval-  
308 uates acute (0-day lag) mortality occurring on the day of an oppressive air mass. It does not  
309 take into account mortality displacement, that is, an assessment of the proportion of short-  
310 term increases in mortality that would have occurred within the near-term future anyway.  
311 Current research presents mixed results on the proportion of deaths on hot days that are  
312 compensated for by decreased mortality in the coming weeks; with results that vary by  
313 place (e.g. Hajat et al. 2005) and severity of event (e.g. Anderson and Bell 2009). Observed  
314 displacement ranges from near 100% (e.g. London in Hajat et al. 2005)—suggesting that  
315 virtually all short-term heat-related mortality is compensated for by mortality decreases in  
316 future weeks—to near 25% in others (Rey et al. 2007; Kaiser et al. 2007; Toulemon and  
317 Barbieri 2008). More research is warranted on evaluating this issue.

318 The seasonal asymmetry observed at most cities supports the notion of there existing a  
319 susceptible pool of vulnerable individuals; whether this is a function of seasonal accli-  
320 matization or that the number of susceptible people decreases following prior heat events  
321 or winter mortality events (e.g. Rocklöv et al. 2009; Stafoggia et al. 2009) should be further  
322 explored as well.

323 It should also be stated that a lack of a statistically significant response in this research  
324 does not mean that there is no heat-related mortality, or that a city's population is not  
325 vulnerable. In addition to the seasonal aspect of the heat–health response, there are also the  
326 related issues of intensity and duration of a heat event. Some oppressive weather type days  
327 are hotter than others within a given time of year. There are instances where oppressive air





328 masses persist for several days; this ‘heat wave effect’ has been already studied by a  
329 number of researchers (e.g. Hajat et al. 2006; Anderson and Bell 2009; Sheridan and  
330 Kalkstein 2004). For example, while the city of Phoenix has no observed statistically  
331 significant increase when all DT+ days are averaged in mid-summer, the analysis done by  
332 one author (Sheridan) in the development of the HWWS for Phoenix showed statistically  
333 significant increases in mortality when the hottest DT+ days persist for 2 days or longer.  
334 Thus, the results presented herein should be considered to be relative levels of vulnera-  
335 bility, not absolute determinants.

336 Last, our previous research (Sheridan et al. 2009) showed that, following a steep decline  
337 in heat-related mortality from the 1970s to the 1990s, the mortality response has largely  
338 remained the same, which may be related to the general decrease in day-to-day mortality  
339 variability (e.g. Carson et al. 2006). We explored whether the seasonal cycle of heat  
340 mortality relationship has changed over our 30-year period of study; however, due to  
341 different seasonal frequencies of oppressive air masses over time, a consistent trend could  
342 not be identified. Rather, the mortality response appeared to be generally somewhat weaker  
343 later in the period throughout the calendar year. As it is likely that heat events will increase  
344 in frequency in the twenty-first century (Meehl and Tebaldi 2004), seasonal changes in the  
345 heat–health relationship should be further explored.

## 348 References

- 349 Abrahamson V, Wolf J, Lorenzoni I, Fenn B, Kovats S, Wilkinson P, Adger WN, Raine R (2009)  
350 Perceptions of heatwave risks to health: interview-based study of older people in London and Norwich,  
351 UK. *J Publ Health* 31:119–126
- 352 Anderson BG, Bell ML (2009) Weather-related mortality: How heat, cold, and heat waves affect mortality  
353 in the United States. *Epidemiol* 20:205–213
- 354 Anderson RN, Rosenberg HM (1998) Age standardization of death rates: implementation of the year 2000  
355 standard. Technical Report 47. National Vital Statistics Reports, Hyattsville, MD
- 356 Bassil KL, Cole DC, Moineddin R, Craig AM, Wendy Lou WY, Schwartz B, Rea E (2009) Temporal and  
357 spatial variation of heat-related illness using 911 medical dispatch data. *Environ Res* 109:600–606
- 358 Carson C, Hajat S, Armstrong B, Wilkinson P (2006) Declining vulnerability to temperature-related  
359 mortality in London over the 20th century. *Am J Epidemiol* 164:77–84
- 360 Chestnut LG, Breffle WS, Smith JB, Kalkstein LS (1998) Analysis of differences in hot-weather-related  
361 mortality across 44 U.S. metropolitan areas. *Environ Sci Policy* 1:59–70
- 362 Davis RE, Knappenberger PC, Novicoff WM, Michaels PJ (2003) Decadal changes in summer mortality in  
363 U.S. cities. *Int J Biometeorol* 47:166–175
- 364 Davis RE, Knappenberger PC, Michaels PJ, Novicoff WM (2004) Seasonality of climate-human mortality  
365 relationships in US cities and impacts of climate change. *Clim Res* 26:61–76
- 366 Dixon PG, Brommer DM, Hedquist C, Kalkstein AJ, Goodrich GB, Walter JC, CC Dickerson IV, Penny SJ,  
367 Cerveny RS (2005) Heat mortality versus cold mortality: a study of conflicting databases in the United  
368 States. *Bull Am Meteorol Soc* 86:937–943
- 369 Donaldson GC, Keatinge WR, Nayha S (2003) Changes in summer temperature and heat-related mortality  
370 since 1971 in North Carolina, South Finland, and Southeast England. *Environ Res* 91:1–7
- 371 Ebi KL, Schmier JK (2005) A stitch in time: improving public health early warning systems for extreme  
372 weather events. *Epidemiol Rev* 27:115–121
- 373 Ebi KL, Teisberg TJ, Kalkstein LS, Robinson L, Weiher RF (2004) Heat watch/warning systems save lives:  
374 estimated costs and benefits for Philadelphia 1995–1998. *Bull Am Meteorol Soc* 85:1067–1074
- 375 Hajat S, Armstrong BG, Gouveia N, Wilkinson P (2005) Mortality displacement of heat-related deaths.  
376 *Epidemiology* 16:613–620
- 377 Hajat S, Armstrong B, Baccini M, Biggeri A, Bisanti L, Russo A, Paldy A, Menne B, Kosatsky T (2006)  
378 Impact of high temperatures on mortality: Is there an added “heat wave” effect? *Epidemiology* 17:  
379 632–638



- 380 Hajat S, Sheridan SC, Allen MJ, Pascal M, Laaidi K, Yagouti A, Bickis U, Tobias A, Bourque D, Armstrong  
381 BG, Kosatsky T (2010) Which days of hot weather are identified as dangerous by Heat-Health Warning  
382 Systems? A comparison of the predictive capacity of different approaches. *Am J Public Heal* (in press)
- 383 Harlan SL, Brazel AJ, Prashad L, Stefanov WL, Larsen L (2006) Neighborhood microclimates and vul-  
384 nerability to heat stress. *Soc Sci Med* 63:2847–2863
- 385 Kaiser R, Le Tertre A, Schwartz J, Gotway C, Daley R, Rubin CH (2007) The effect of the 1995 heatwave in  
386 Chicago on all-cause and cause-specific mortality. *Am J Public Health* 97:S158–S162
- 387 Knowlton K, Rotkin-Ellman M, King G, Margolis HG, Smith D, Solomon G, Trent R, English P (2009) The  
388 2006 California heat wave: impacts on hospitalizations and emergency department visits. *Environ Heal*  
389 *Perspect* 117:61–67
- 390 Kovats RS, Ebi KL (2006) Heatwaves and public health in Europe. *Eur J Pub Health* 16:592–599
- 391 Kovats RS, Hajat S (2008) Heat stress and public health: a critical review. *Annu Rev Public Health* 29:  
392 41–55
- 393 Laschewski G, Jendritzky G (2002) Effects of the thermal environment on human health: an investigation of  
394 30 years of daily mortality data from SW Germany. *Clim Res* 21:91–103
- 395 Le Tertre A, Lefranc A, Eilstein D, Declercq C, Medina S, Blanchard M, Chardon B, Fabre P, Filleul L,  
396 Jusot JF, Pascal L, Prouvost H, Cassadou S, Ledrans M (2006) Impact of the 2003 heatwave on  
397 all-cause mortality in 9 French cities. *Epidemiology* 17:75–79
- 398 Luber G, McGeehin M (2008) Climate change and extreme heat events. *Am J Prev Med* 35:429–435
- 399 McMichael AJ, Wilkinson P, Kovats RS, Pattenden S, Hajat S, Armstrong B, Vajanapoom N, Niciu EM,  
400 Mahomed H, Kingkeow C, Kosnik M, O'Neill MS, Romieu I, Ramirez-Aguilar M, Barreto ML,  
401 Gouveia N, Nikiforov B (2008) International study of temperature, heat and urban mortality: the  
402 'ISOTHERM' project. *Int J Epidemiol* 37:1121–1131
- 403 Medina-Ramon M, Schwartz J (2007) Temperature, temperature extremes, and mortality: a study of  
404 acclimatization and effect modification of 50 US cities. *Occup Environ Med* 64:827–833
- 405 Meehl GA, Tebaldi C (2004) More intense, more frequent, and longer lasting heat waves in the 21st century.  
406 *Science* 305:994–997
- 407 Michelozzi P, Accetta G, De Sario M, D'Ippoliti D, Marino C, Baccini M, Biggeri A, Anderson HR,  
408 Katsouyanni K, Ballester F, Bisanti L, Cadum E, Forsberg B, Forastiere F, Goodman PG, Hojs A,  
409 Kirchmayer U, Medina S, Paldy A, Schindler C, Sunyer J, Perucci CA (2009) High temperature and  
410 hospitalizations for cardiovascular and respiratory causes in 12 European Cities. *Am J Respir Crit Care*  
411 *Med* 179:383–389
- 412 Moore SE (2006) Patterns in mortality governed by the seasons. *Int J Epidemiol* 35:435–437
- 413 O'Neill MS, Ebi KL (2009) Temperature extremes and health: impacts of climate variability and change in  
414 the United States. *J Occup Environ Med* 51:13–25
- 415 Palecki MA, Changnon SA, Kunkel KE (2001) The nature and impacts of the July 1999 heat wave in the  
416 midwestern United States: learning from the lessons of 1995. *Bull Am Meteorol Soc* 82:1353–1367
- 417 Rey G, Jouglu E, Fouillet A, Pavillon G, Bessemoulin P, Frayssinet P, Clavel J, Hémon D (2007) The impact  
418 of major heat waves on all-cause and cause-specific mortality in France from 1971 to 2003. *Int Arch*  
419 *Occup Environ Health* 80:615–626
- 420 Rocklöv J, Forsberg B, Meister K (2009) Winter mortality modifies the heat-mortality association the  
421 following summer. *Eur Respir J* 33:245–251
- 422 Sheridan SC (2002) The redevelopment of a weather-type classification scheme for North America. *Int J*  
423 *Climatol* 22:51–68
- 424 Sheridan SC (2007) A survey of public perception and response to heat warnings across four North  
425 American cities: an evaluation of municipal effectiveness. *Int J Biometeorol* 52:3–15
- 426 Sheridan SC, Kalkstein LS (2004) Progress in heat watch-warning system technology. *Bull Am Meteorol*  
427 *Soc* 85:1931–1941
- 428 Sheridan SC, Kalkstein AJ, Kalkstein LS (2009) Trends in heat-related mortality in the United States,  
429 1975–2004. *Nat Haz* 50:145–160
- 430 Stafoggia M, Forastiere F, Michelozzi P, Perucci CA (2009) Summer temperature-related mortality: effect  
431 modification by previous winter mortality. *Epidemiology* 20:575–583
- 432 Toulemon L, Barbieri M (2008) The mortality impact of the August 2003 heat wave in France: Investigating  
433 the 'harvesting' effect and other long-term consequences. *Populat Stud* 62:39–53
- 434 Vaneckova P, Hart MA, Beggs PJ, de Dear RJ (2008) Synoptic analysis of heat-related mortality in Sydney,  
435 Australia, 1993–2001. *Int J Biometeorol* 52:439–451
- 436 Vaneckova P, Beggs PJ, Jacobson CR (2010) Spatial analysis of heat-related mortality among the elderly  
437 between 1993 and 2004 in Sydney, Australia. *Soc Sci Med* 70:293–304
- 438