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Accessibility

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**Introduction**

Heat waves, or extreme heat events, are predicted to increase in frequency, intensity, and duration with climate change (Intergovernmental Panel on Climate Change 2007). The elderly are more vulnerable to heat-related mortality, so the changing population structure (Vincent and Velkoff 2010) may also lead to an increased health burden from heat. The associations between temperature and mortality are well studied (Gosling et al. 2009; Hajat and Kosatsky 2010). In the United States, associations between heat and daily mortality are stronger in cities with milder summers and lower air conditioning prevalence (Anderson and Bell 2009, 2011; Curriero et al. 2002; Medina-Ramón and Schwartz 2007; O’Neill and Ebi 2009). Although extreme heat events in the United States are associated with increased deaths (Ostro et al. 2009; Semenza et al. 1996), the role of heat-wave duration versus intensity as a health determinant remains unclear (Anderson and Bell 2011; Gasparrini and Armstrong 2011).

Studies examining associations between heat and hospital admissions have had mixed results (Turner et al. 2012; Ye et al. 2012). For example, in London, England, a 1995 heat wave was not associated with total hospital admissions but was associated with hospitalizations for respiratory and renal diseases and increased mortality (Kovats et al. 2004). On the other hand, heat waves in Adelaide, Australia, have been associated with increased hospitalizations but not increased mortality (Nitschke et al. 2007). In 12 European cities, respiratory admissions increased with temperature in Mediterranean and North-Continental cities, but associations between temperature and cardiovascular admissions tended to be negative and nonsignificant (Michelozzi et al. 2009). A recent analysis of 213 U.S. counties found a 4% increase in respiratory admissions for each 1°F increase in same-day temperature (Anderson et al. 2013).

Understanding associations between heat waves and hospital admissions in the United States is important to predict how climate change may increase the future burden of heat-related morbidity; to identify vulnerable subpopulations for potential interventions; and to refine activation thresholds for heat-health warning systems.

We analyzed Medicare inpatient billing records from 114 cities across five U.S. climate zones to evaluate a) the associations between moderate and extreme heat measured as daily mean apparent temperature (AT) and all-cause and cause-specific hospital admissions among elderly Medicare beneficiaries residing in five U.S. climate zones, b) the added effect of extreme heat durations of 2–8 days, c) whether observed associations are confounded by ambient ozone levels and holidays, and d) the sensitivity of our findings to the choice of temperature metric and the modeling of the exposure–response relationship.

**Methods**

**Study cities and climate zones.** The 200 counties with the highest number of cardiovascular hospital admissions in 2004–2006 were assigned to their respective Metropolitan Statistical Areas to form the study cities, which were then assigned to the U.S. Department of Energy, Energy Information Administration’s (EIA) five climate zones (EIA 2010), as previously described (Zanobetti et al. 2012; see also Supplemental Material, “Study Cities and Climate Zones Methods”). These climate zones were based on numbers of cooling-degree days (annual sum of daily mean temperatures > 18.3°C) and heating-degree days (annual sum of daily mean temperatures < 18.3°C).

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Health outcomes. We obtained emergency hospital admissions for individuals ≥ 65 years of age from 1992 through 2006 from the U.S. Centers for Medicare and Medicaid Services MedPAR billing records (CMMS 2006). Ninety-seven percent of Americans ≥ 65 years of age receive Medicare insurance coverage. We categorized admissions according to the primary admission International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM; Centers for Disease Control and Prevention 2013) codes as all-cause (all ICD-9-CM codes < 800), cardiovascular (CVD; 390–429), heat (992 and E900.0), respiratory (580–589), and environmental (Resp; 480–487, 490–492, and 494–496). The “heat” category also included secondary causes of admission related to heat. The research was approved by the institutional review boards at Harvard University and University of Michigan, and waivers of the informed consent requirement were granted.

Environmental variables. We obtained daily temperature and dew point data for each city from the National Climatic Data Center Cooperative Summary of the Day station files [National Climatic Data Center (NCDC) 2010b]. Data from a single monitor for each city were used except in 6 cities where multiple observations were missing from the nearest station. We obtained dew point data from the National Climatic Data Center’s Integrated Surface Database Lite (NCDC 2010a) converted to daily values. For 25 stations missing dew point data, dew point data were obtained from the nearest station with dew point data. We calculated AT using daily mean temperature (MT) and dew point [AT(°C) = −2.653 + (0.994 × MT) + 0.0153 × (dew point°)] (Kalkstein and Valimont 1986). We excluded from our analysis 20 cities that were missing AT measurements on at least 15% of the study days.

We chose AT as our main heat exposure metric to jointly account for effects of temperature and humidity, but we also addressed the sensitivity of our results to the alternative metric choices of MT, daily minimum temperature (MI), and diurnal temperature range (DTR; the difference between MA and MI).

Because ambient ozone levels rise during hot weather and are associated with increased rates of hospital admissions, ozone can potentially confound associations between heat and hospital admissions (Medina-Ramón et al. 2006). We obtained ozone data from the U.S. Environmental Protection Agency’s Air Quality System (U.S. EPA 2014), and daily 8-hr averages were calculated and standardized as described previously (Medina-Ramón et al. 2006). For ozone analyses, we excluded an additional 10 cities that were missing ozone measurements on at least 15% of the study days.

Main statistical analysis. We used a time-stratified case-crossover design to evaluate the association between heat and hospital admissions in each city using data on hospital admissions occurring between May and September. Control days were chosen such that cases and controls were matched on calendar month and day of week. We applied Poisson regression and accounted for over-dispersion using a quasi-Poisson model in the glm package in R (R Development Core Team, Vienna, Austria) (Guo et al. 2011).

We modeled temperature effects as natural cubic splines (NCSs) of AT with 3 degrees of freedom (df). We expected greater changes in the effect estimates at the extremes of temperature, so we placed the internal knots at the 10th and 90th city-specific percentiles of warm-season AT. We centered the splines at the 75th percentiles of warm-season AT, which served as the reference temperatures. A priori, moderate heat was defined as the 90th percentile of warm-season AT, and extreme heat was defined as the 99th percentile of warm-season AT. To reduce collinearity between temperature lags in our models, we modeled AT exposure as 2-day sums of lags from lags 0–7, instead of individual lags, or as four 2-day lag strata. This is a constrained distributed lag model where lags 0 and 1 are constrained to have the same effect, lags 2 and 3 are constrained to have the same effect, and so on (Armstrong 2011; Schwartz et al. 2004). We used the dlnm package in R to generate the temperature-lag crossbases (Gasparrini et al. 2010).

To distinguish between effects of heat intensity and duration, we included a term for heat waves, or extreme heat duration, in additional models. This term was an indicator variable for 2-day mean AT above the 95th percentile of city-specific warm-season AT for at least 2, 4, 6, or 8 days in duration. For example, the city-specific model of the effect on hospital admissions of being in a heat wave for at least 8 days is as follows:

\[
\text{ln(hospital admissions)} = \alpha_{1}YMW_{1} + \ldots + \alpha_{3}YMW_{3},
\]

where \(YMW_{1–3}\) were the indicator variables for each year/month/day-of-week combination (how seasonal and long-term effects in a case-crossover design using Poisson regression are controlled for); \(NCS_{0}, NCS_{3}, NCS_{5}, NCS_{7}\) were the four natural cubic splines of AT (one for each of four lag strata: days 0–1, 2–3, 4–5, and 6–7) each with 3 df; and \(HW\) was the single heat wave indicator variable for an 8-day heat wave. The cumulative effects over 8 days following a day of moderate or extreme heat and the effects of 2–8 day-long heat waves were calculated as the sums of the lag-specific effects (and heat-wave effect for the heat wave models) within each city.

For each city, we estimated a single moderate or extreme heat effect at lag day 0 or over lag days 0–7 separately. We then pooled city-specific results in each of the five climate zones and overall in a random-effects meta-analysis using inverse variance weighting (DerSimonian and Laird 1986) using the meta package in R, and calculated Q statistics for heterogeneity within and between climate zones. To pool the cumulative effects of extreme heat plus the added heat-wave effect across cities in models with a heat-wave term, we first estimated the main extreme heat effect and the added heat-wave effect for each city, or 2 effects per city, and their corresponding covariances. Then we performed a restricted maximum likelihood multivariate meta-analysis of these effects using the mvmeta package in R (Gasparrini et al. 2012). All analyses were performed in R version 2.15 or 3.0.

Sensitivity analyses. We evaluated confounding by ozone and the holidays Memorial Day, Independence Day, and Labor Day (which all occur in the first or last week of the month) by comparing the risk ratios (percent increase/100 + 1) for AT from models with and without inclusion of the confounders (ozone or holidays). We examined the sensitivity of our results to temperature metric by modeling different metrics for temperature (MT, MI, MA, or DTR) in place of AT.

In a time-stratified case-crossover design, each case’s controls are selected from the same time stratum as the case, and seasonal effects and long-term time trends are assumed to vary inconsequentially within each time stratum. In our main models, this time stratum was a month, for a total of five time strata per year, each 30 or 31 days long. To examine the sensitivity of our results to the length of the time stratum, we used either six time strata (each 25 or 26 days long) or four time strata (each 38 or 39 days long) per year in separate models.

We assumed a functional form of the association between daily temperature and hospital admission to be more complex than that modeled by a natural cubic spline with 3 df (2 internal knots). However, we examined the sensitivity of our results to the modeling of the functional form in the largest city in each of the five climate zones by varying knot locations in natural cubic splines as well as in piecewise linear splines (which are more easily interpreted). We also examined the sensitivity of our results to lag structure by using a natural cubic spline lag structure with 3 internal
knobs as opposed to four discrete lag strata. We also compared the case-crossover results using Poisson regression with a quasi-Poisson distribution to those of a case-crossover design using Cox proportional hazards regression with robust standard errors. Finally, we attempted to identify heat and cold thresholds, or knot locations, using the SiZer method, which identifies significant increases or decreases in locally weighted polynomial smoothers for different spans of the smoother (see Supplemental Material, “Alternative Threshold Identification Using SiZer”).

Results
We examined the association between heat and rates of hospitalization in 114 cities broadly distributed across the contiguous United States (see Supplemental Material, Figure S1).

Cardiovascular, respiratory, and renal admissions accounted for approximately 24%, 8%, and 1% of all-cause admissions in each climate zone from May through September (Table 1). Heat-related admissions were uncommon, accounting for only 0.1% of all-cause admissions. In hotter climates (climate zones 4 and 5), the differences between the 99th and 75th percentiles of warm-season AT were smaller than in cooler climates (climate zones 1–3), though the number of days meeting the definitions for 2-, 4-, 6-, and 8-day long heat waves were similar between climates. Mean daily ozone levels were slightly higher in climate zone 4.

The functional form of the association between AT and hospital admissions in the largest city in each climate zone (Minneapolis, MN; Chicago, IL; New York City, NY; Los Angeles, CA; and Houston, TX) varied widely by city, admissions cause, and lag day (Figure 1). For all-cause, renal, and respiratory diseases, the association between AT at lag 0 and hospital admission was approximately linear (Figure 1A,C,D). However, at subsequent lags, the association was U-shaped for these three causes of admission, and for the cumulative effects of AT over 8 days, the form of the association was U-shaped (Figure 1E,G,H). This difference in functional form by lag persisted regardless of whether the form was modeled as a piecewise linear spline versus a natural cubic spline and regardless of knot placement (see Supplemental Material, Figure S2, for all-cause admissions in New York City). Focusing only on the range of AT above the 75th percentile, there was a positive association between hospital admissions


<table>
<thead>
<tr>
<th>Variable</th>
<th>Zone 1 (13 cities)</th>
<th>Zone 2 (24 cities)</th>
<th>Zone 3 (22 cities)</th>
<th>Zone 4 (24 cities)</th>
<th>Zone 5 (21 cities)</th>
<th>All zones (114 cities)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital admissions (mean daily count)**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All natural causes</td>
<td>53 (19–97)</td>
<td>241 (18–571)</td>
<td>286 (21–570)</td>
<td>142 (16–336)</td>
<td>98 (18–201)</td>
<td>186 (16–571)</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>13 (6–24)</td>
<td>59 (4–133)</td>
<td>65 (5–135)</td>
<td>33 (4–77)</td>
<td>24 (4–53)</td>
<td>45 (4–135)</td>
</tr>
<tr>
<td>Heat-related</td>
<td>0.0 (0.0–0.1)</td>
<td>0.2 (0.0–0.7)</td>
<td>0.3 (0.0–0.6)</td>
<td>0.1 (0.0–0.2)</td>
<td>0.1 (0.0–0.2)</td>
<td>0.2 (0.0–0.7)</td>
</tr>
<tr>
<td>Renal</td>
<td>0.6 (0.2–1.2)</td>
<td>2.8 (0.1–6.0)</td>
<td>3.0 (0.2–6.2)</td>
<td>1.8 (0.1–4.1)</td>
<td>1.2 (0.2–2.0)</td>
<td>2.2 (0.1–6.2)</td>
</tr>
<tr>
<td>Respiratory</td>
<td>5 (2–8)</td>
<td>21 (2–47)</td>
<td>22 (2–49)</td>
<td>13 (1–31)</td>
<td>8 (2–16)</td>
<td>16 (1–49)</td>
</tr>
<tr>
<td>Annual no. of days in a heat wave (consecutive 2-day means each above 75th percentile of AT) by heat-wave duration</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-day</td>
<td>7.6 (7.3–7.9)</td>
<td>7.7 (7.1–8.1)</td>
<td>7.7 (7.1–7.8)</td>
<td>7.7 (7.4–8.1)</td>
<td>7.7 (7.4–7.9)</td>
<td>7.7 (7.1–8.1)</td>
</tr>
<tr>
<td>4-day</td>
<td>2.6 (1.8–3.4)</td>
<td>2.9 (2.3–3.7)</td>
<td>2.9 (2.3–4.7)</td>
<td>3.4 (2.2–4.3)</td>
<td>3.1 (2.4–4.0)</td>
<td>3.0 (1.8–4.7)</td>
</tr>
<tr>
<td>6-day</td>
<td>0.8 (0.2–1.2)</td>
<td>1.0 (0.3–2.3)</td>
<td>1.0 (0.3–3.1)</td>
<td>1.7 (0.5–2.6)</td>
<td>1.4 (0.5–2.7)</td>
<td>1.2 (0.2–3.1)</td>
</tr>
<tr>
<td>8-day</td>
<td>0.2 (0.0–0.6)</td>
<td>0.4 (0.0–1.4)</td>
<td>0.4 (0.0–2.1)</td>
<td>0.9 (0.0–1.7)</td>
<td>0.6 (0.1–2.2)</td>
<td>0.5 (0.0–2.2)</td>
</tr>
<tr>
<td>Mean daily ozone (ppb)†</td>
<td>45 (37–49)</td>
<td>45 (36–54)</td>
<td>46 (33–56)</td>
<td>50 (27–71)</td>
<td>44 (29–57)</td>
<td>46 (27–71)</td>
</tr>
</tbody>
</table>

*Population-weighted means of the city-specific means. **ICD-9-CM codes: all natural causes (all ICD-9-CM codes < 800), cardiovascular (390–429), heat (including 992 [effects of heat and light] and E900.0 [excessive heat due to weather conditions]), renal (580–598), and respiratory (480–487, 490–492, and 494–498). The “heat” category also includes admissions with any secondary causes related to heat. †Number of cities contributing to ozone calculations: 11, 32, 22, 20, and 19 for zones 1–5, respectively.

Figure 1. Percent increases for the largest city in each of five climate zones in hospital admissions among U.S. elderly for apparent temperature (AT) versus the 75th percentile of AT, May–September, 1992–2006, at lag 0 (A–D) and over lags 0–7 (E–H) for all causes (A,E), cardiovascular (CVD) diseases (B,F), renal diseases (C,G), and respiratory (Resp) diseases (D,H).
and AT in most instances at lag 0 and over
lags 0–7. In contrast to the other causes of
admission, for cardiovascular admissions, the
association at lag 0 was an inverse U-shape;
and above the 75th percentile of AT, there was
a weak inverse association between hospital
admission and AT in Minneapolis, Chicago,
New York City, and Los Angeles. Although
this inverse U-shape became a U-shape in
subsequent lags, the cumulative effect over
8 days for AT above the 75th percentile and
hospital admission was still weakly protective.

In meta-analyses, the magnitude of the
associations between moderate heat and
hospital admissions for all causes, cardiovascular
diseases, and respiratory diseases were small,
with increases in admissions at the 90th versus
the 75th percentile of AT of –0.4% to 1.3%
over lags 0–1 and lags 0–7 (Table 2). The
associations between moderate heat over lags 0–1 and
lags 0–7 and admissions for renal diseases were
stronger, with increases in admissions of 3.9%
(95% CI: 2.9%, 4.9%) and 4.3% (95% CI:
3.0%, 5.6%), respectively. The associations
between extreme heat (9th vs. 75th percentile
of AT) and hospital admissions were stronger
than for moderate heat over lags 0–1 and lags
0–7. Specifically, the “main” (as opposed to
heat-wave) extreme heat-associated increases
in respiratory admissions over these time periods were 3.3% (95% CI: 1.3%, 5.4%)
and 4.3% (95% CI: 1.8%, 6.9%), respec-
tively; and extreme heat-associated increases
in renal admissions were even higher at 9.3%
(95% CI: 4.3%, 14.5%) and 14.2% (95% CI:
8.5%, 20.1%), respectively. We failed to
observe a decline in the effects of extreme heat
in the 7 days following the day of extreme heat,
with the cumulative lag 0–7 effects similar to or
higher than the lag 0–1 effects.

For renal and respiratory causes of
admission, we observed a significant “added
heat-wave effect” for heat waves defined as
at least 6 days of AT above the 95th percen-
tile of AT, with increases in admissions of
10.7% and 2.8%, respectively. In pooling
the city-specific cumulative main effects of
extreme heat over 6 days and the added
heat-wave effect, we estimated increases in
admissions for renal and respiratory diseases
for a 6-day heat wave to be 23.2% (95% CI:
14.2%, 32.8%) and 8.5% (95% CI:
4.8%, 12.2%), respectively.

When comparing the effects of extreme
heat by climate zone, we found significant
deregion heterogeneity between climate zones in the
cumulative 8-day effect estimates for admissions
for respiratory diseases, with pooled increases of
11.8% (95% CI: 2.3%, 22.2%) in climate
zone 1 and –0.5% (95% CI: –4.0%, 3.1%)
in climate zone 5 (Table 3). For admissions for
both respiratory and renal diseases, we found
significant heterogeneity in effect estimates
within climate zones 1, 2, 3, and/or 4.

Results of sensitivity analyses.

Confounding by ozone of the association
between AT and hospital admissions was
minimal. Both moderate heat and extreme
heat risk ratios at lag 0 and over lags 0–7 from
the models with ozone deferred from those in
the models without ozone by < 10% in all
cities (results not shown). Confounding by
holidays was also minimal. In models with
terms for holidays versus models without
holiday terms, the risk ratios for moderate and
extreme heat at lag 0 and over lags 0–7 also
did not differ by > 10% in any city.

When we varied the length of the time
stratum, the extreme heat effect estimates were
similar, but associations over lag days 0–7
between moderate heat and hospital admis-
sions were slightly stronger [e.g., for respira-
tory diseases, 1.0% (95% CI: 0.3%, 1.6%) vs.
0.2% (95% CI: –0.5%, 0.8%) when seasonal
dependent week effect was included].

<table>
<thead>
<tr>
<th>Variable</th>
<th>All causes</th>
<th>Cardiovascular</th>
<th>Renal</th>
<th>Respiratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sum of the main effects of moderate heat</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lag days 0–1</td>
<td>0.7 (0.5, 0.9)</td>
<td>–0.4 (–0.6, –0.2)</td>
<td>3.9 (2.9, 4.9)</td>
<td>1.3 (0.8, 1.8)</td>
</tr>
<tr>
<td>Lag days 0–7</td>
<td>0.5 (0.3, 0.7)</td>
<td>–1.3 (–1.6, –1.0)</td>
<td>4.3 (3.0, 5.6)</td>
<td>0.0 (–0.6, 0.7)</td>
</tr>
<tr>
<td>Sum of the main effects of extreme heat</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lag days 0–1</td>
<td>1.4 (0.8, 2.0)</td>
<td>–1.6 (–2.7, –0.6)</td>
<td>9.3 (4.3, 14.5)</td>
<td>3.3 (1.3, 5.4)</td>
</tr>
<tr>
<td>Lag days 0–3</td>
<td>2.1 (1.5, 2.8)</td>
<td>–2.0 (–2.9, –1.0)</td>
<td>12.6 (8.2, 17.3)</td>
<td>4.8 (2.6, 6.9)</td>
</tr>
<tr>
<td>Lag days 0–5</td>
<td>2.9 (2.2, 3.6)</td>
<td>–2.0 (–3.0, –1.0)</td>
<td>11.3 (6.0, 16.8)</td>
<td>5.5 (3.2, 7.8)</td>
</tr>
<tr>
<td>Lag days 0–7</td>
<td>3.2 (2.4, 4.0)</td>
<td>–1.8 (–2.7, –0.8)</td>
<td>14.2 (8.5, 20.1)</td>
<td>4.3 (1.8, 6.9)</td>
</tr>
<tr>
<td>Added heat-wave effect for 4 heat-wave durations</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-day</td>
<td>0.2 (–0.3, 0.7)</td>
<td>–0.4 (–1.3, 0.5)</td>
<td>2.6 (1.0, 4.4)</td>
<td>0.4 (–1.2, 2.3)</td>
</tr>
<tr>
<td>4-day</td>
<td>1.5 (0.9, 2.2)</td>
<td>0.6 (–0.6, 1.8)</td>
<td>4.6 (0.5, 8.9)</td>
<td>2.1 (0.1, 4.3)</td>
</tr>
<tr>
<td>6-day</td>
<td>0.9 (–0.1, 1.9)</td>
<td>–0.6 (–2.4, 1.2)</td>
<td>10.7 (3.1, 18.8)</td>
<td>2.8 (0.1, 5.9)</td>
</tr>
<tr>
<td>8-day</td>
<td>0.8 (–2.3, 0.6)</td>
<td>–0.9 (–3.5, 0.8)</td>
<td>12.8 (16.25, 0.0)</td>
<td>1.1 (–3.6, 5.9)</td>
</tr>
<tr>
<td>Sum of the main effects of extreme heat and the added heat-wave effect for 4 heat-wave durations</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-day</td>
<td>1.6 (1.1, 2.1)</td>
<td>–2.0 (–2.8, –1.2)</td>
<td>12.1 (8.4, 15.9)</td>
<td>3.7 (1.8, 5.7)</td>
</tr>
<tr>
<td>4-day</td>
<td>3.7 (2.9, 4.4)</td>
<td>–1.4 (–2.4, –0.3)</td>
<td>17.8 (12.8, 23.0)</td>
<td>7.0 (4.3, 9.8)</td>
</tr>
<tr>
<td>6-day</td>
<td>3.8 (2.7, 4.9)</td>
<td>–2.6 (–4.3, –1.0)</td>
<td>23.2 (14.2, 32.8)</td>
<td>8.5 (4.8, 12.2)</td>
</tr>
<tr>
<td>8-day</td>
<td>2.3 (0.8, 3.9)</td>
<td>–2.7 (–5.2, –0.1)</td>
<td>28.9 (16.1, 43.0)</td>
<td>5.4 (0.0, 11.1)</td>
</tr>
</tbody>
</table>

*Significant heterogeneity within climate zone categories (p < 0.05 in Q-test for heterogeneity).
Heat and hospital admissions among U.S. elderly

We found a slight decrease in hospital admissions for cardiovascular diseases with moderate heat or extreme heat (1–2%) and an increase in admissions for respiratory diseases of 4% in the 8 days following extreme heat. Other studies of associations between cardiovascular and respiratory admissions and heat and heat waves in the United States have found increased risks of cardiovascular and respiratory diseases on lag days 0–1 (Ye et al. 2012). Studies using time series of admissions and temperature data from earlier time periods (e.g., Schwartz et al. 2004) tended to find stronger associations between cardiovascular admissions and heat than our study. These differences in results by time period may be related to the implementation of heat-health warning systems in several U.S. cities and increased awareness of the dangers of heat to the elderly after the 1995 Chicago heat wave (Bassil and Cole 2010). More similar to our findings, a study in California from 1999–2005 (Green et al. 2010) and a study in New York State from 1991–2004 (Lin et al. 2009) did not find increases in cardiovascular hospital admissions with heat on lag days 0 or 1. In a meta-analysis of the associations between heat and admissions for cardiovascular and respiratory diseases from studies worldwide, Turner et al. (2012) also did not find significant associations for cardiovascular admissions, though their results were suggestive of an association between respiratory admissions and heat (for a 1°C increase in temperature, risk ratio > 1.020; 95% CI: 0.986, 1.055).

Using a data set very similar to ours, Anderson et al. (2013) found an association between respiratory hospital admissions and a 10°F increase in temperature of 4.3% (95% posterior interval: 3.8%, 4.8%). This effect is similar though slightly higher than our effect estimate of 1.7% (95% CI: 0.9%, 2.6%) (Table 3) for extreme heat (99th vs. 75th percentile of AT), which corresponds to an average of 11°F across cities. Additionally, they found a cumulative association between a 10°F increase in the previous week’s temperature and respiratory admissions of 2.2% (95% posterior interval: 1.3%, 3.1%) which was similar to our estimate of 4% for extreme heat over the subsequent 8 days, though they modeled the association between temperature and hospital admissions as linear at all lags. In contrast, we found a linear association at early lags (0–1) and a U-shaped association at later lags for respiratory admissions.

The associations between temperature and hospital admissions for all natural causes were robust to knot placement and time strata length and were similar between metrics, though moderate DTR effects were slightly higher. Differences in hypothesized underlying biological mechanisms, such as an inability to cool off through sweating versus adjust to sudden large changes in temperature, may account for small differences observed between DTR and the other metrics.

The associations between cardiovascular and respiratory hospital admissions and heat were weaker than between cardiovascular and respiratory mortality and heat found in previous U.S. studies. Anderson and Bell (2009) found a 5% increase in cardiovascular mortality and 6% increase in respiratory mortality among individuals ≥ 75 years of age for an increase in mean daily temperature from the 90th to 99th percentile on lag days 0–1. In the United States, deaths associated with extreme heat were higher among individuals dying out of hospital as well as individuals with preexisting atrial fibrillation (Medina-Ramón et al. 2006; Zanobetti et al. 2013). Therefore, heat may have a strong, deleterious association with cardiovascular mortality and a protective association with hospital admissions, because heat-related deaths may tend to be more sudden and individuals who might have been hospitalized for cardiovascular diseases die instead as a result of extreme heat.

Consistent with studies of the added heat-wave effect in the association between heat and mortality in the United States (Anderson and Bell 2011; Gasparini and Armstrong 2011), we did not see a substantial added heat-wave effect for cardiovascular and respiratory admissions. In a multicity U.S. study modeling temperature as a spline and heat wave as an indicator variable, Gasparini and Armstrong (2011) and Barnett et al. (2012) found added heat-wave effects of 0–7% in the United States, with the greatest added heat-wave effect when the temperature spline had the fewest df, as did Rocklov et al. (2012) in Sweden. This finding can be explained by the fact that intensity and duration of extreme heat are correlated, so estimates of heat-wave effects diminish as the independent effects of extreme heat are allowed to be more intense in the model. In our model, with a temperature spline with only 3 df (and main effect results that were insensitive to knot location), we still found only a small added heat-wave effect of 1–3% for respiratory diseases. In contrast to admissions for cardiovascular and respiratory diseases, we found a more significant added heat-wave effect in the association between heat and hospital admissions for renal diseases (for heat waves at least 6 days long, 10.7% 95% CI: 3.1%, 18.8%).

Even after accounting for displacement, or the harvesting effect, whereby the exposure advances a health outcome by only a few days because the affected individuals are already very frail (Schwartz et al. 2004), a day of extreme heat was still associated with a significant increase in admissions for renal and respiratory diseases in many cities over the subsequent 7 days. In contrast, for cardiovascular admissions, the cumulative effects of extreme or moderate AT over the subsequent 7 days were slightly protective. Mortality displacement patterns have varied widely in studies of heat-associated cardiovascular and respiratory mortality in the United States and Europe, with some studies finding the sum of effects over multiple lags to be deleterious.
The associations between extreme heat and hospital admissions for all causes over lag days 0–1 as well as 0–7 were statistically significant and of public health significance in the United States in all climate zones. Elderly individuals with respiratory and especially renal conditions, and providers of services to such people, may benefit from taking additional precautions when heat warnings are issued.

**Conclusion**

The associations between extreme heat and hospital admissions for all causes lag days 0–1 as well as 0–7 were statistically significant and of public health significance in the United States in all climate zones. Elderly individuals with respiratory and especially renal conditions, and providers of services to such people, may benefit from taking additional precautions when heat warnings are issued.

**References**


