

Mortality related to temperature and persistent extreme temperatures: a study of cause-specific and age-stratified mortality

Joacim Rocklöv, 1 Kristie Ebi, 2 Bertil Forsberg 1

¹Division of Occupational and Environmental Medicine, Department of Public Health and Clinical Medicine, Umeå University, Umeå, Sweden ²Department of Global Ecology, Carnegie Institution for Science, Stanford, California, USA

Correspondence to

Dr Joacim Rocklöv, Department of Public Health and Clinical Medicine, Occupational and Environmental Medicine, Umeå University, SE 901 87 Umeå, Sweden; joacim.rocklov@ envmed.umu.se

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ABSTRACT

Objectives High and low ambient temperatures are associated with large numbers of deaths annually. Many studies show higher mortalities during heatwaves. However, such effects are rarely explicitly incorporated in models of temperature and mortality, although dehydration followed by cardiovascular stress is more likely to occur. The authors aim to establish time-series models in which the effects of persistent extreme temperature and temperature in general can be disentangled.

Methods The authors established time-series Poisson regression models based on cause-specific mortality and age-stratified mortality in Stockholm County (Sweden), 1990—2002, adjusting for time trends and potential confounders, and studied the effects of temperature and persistence of extreme temperature.

Results Persistent extremely high temperature was associated with additional deaths, and the risk of death increased significantly per day of extended heat exposure. Extreme exposure to heat was associated with higher death rates in adults and for cardiovascular causes of death, compared with a rise in temperature. Warmer temperatures increase daily mortality from natural causes, while decreasing colder temperatures increase the risk of cardiovascular deaths. Furthermore, the impact of warm and cold temperatures decreases within the season, while the impact of persistent extremely high temperatures remains similar throughout the summer.

Conclusions The authors found the mortality impact of persistence of extreme high temperatures to increase proportionally to the length of the heat episode in addition to the effects of temperature based on the temperature—mortality relationship. Thus, the additional effect of persistent extreme heat was found to be important to incorporate for models of mortality related to ambient temperatures to avoid negatively biased attributed risks, especially for cardiovascular mortality. Moreover, the effects associated with non-extreme temperatures may decline as the pool of fragile individuals shrink as well as due to acclimatisation/adaptation. However, a similar decline was not observed for the effects associated with extreme heat episodes.

BACKGROUND

Exposure to heat and cold constitutes a public health threat. ¹⁻⁴ The number of deaths attributed to warm or cold weather are not straightforward to assess but can be estimated in time-series regression models as the relationship between temperature fluctuations and daily death rates adjusted for

What this paper adds

- ► The study from a cold region shows that episodes of extremely high temperatures can accelerate the mortality effect of ambient heat resulting in effects proportional to the number of days in row of such extreme conditions. The accelerating effect is not captured to any great degree by the overall daily temperature—mortality relation, but adds to the increase in mortality explained by the general temperature mortality relation in Stockholm.
- ► In summer, cardiovascular deaths were associated with extreme heat episodes only, and during such conditions risks increased in individuals aged 45 years or older, while deaths related to warm temperatures in general occurred in individuals aged 80 years and older.
- Extreme cold episodes contributed no additional risk compared with the risks associated with cold in general. Deaths due to cold exposure appeared mainly in cardiovascular causes.
- ► The approach used in this study is relevant in understanding the effects of heat from climate change, as extremes of heat are expected to become more frequent in some areas and because such events based on the results from our study are associated with substantial additional risks.
- ► This study also contributes to understanding what population is at risk at which type of heat exposure.

several confounding factors and calendar time.⁵ The impacts of temperature on mortality should be understood in the relative context of the local temperatures and the level of adaptation of a population.^{1 6 7} Exposure to warm temperatures increases cardiovascular and respiratory deaths more in the older population.^{4 8 9} During heatwaves, however, excess mortality is elevated in all age groups.¹⁰ Cold temperatures also elevate death rates of cardiovascular and respiratory causes. For cold, the relative risks are larger in younger adults compared with those for heat.^{3 8 9}

Many studies use mean, maximum or minimum daily temperatures to predict mortality.² ⁷ ⁸ However, such exposure metrics may not well describe the combined impacts of temperature and humidity, but separate modelling of temperature and humidity may induce bias due to the close

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correlation. Therefore, measures that weigh the effect of several parameters based on thermophysiological models, such as apparent temperature, can help reduce model complexity and bias. The mortality impacts following ambient temperature exposure can be lagged over several days or weeks. For high temperatures, the lag between exposure and effect is typically immediate or short. Provided weather, the time between exposure and increased mortality can lag up to several weeks. Significantly 12 in the several weeks.

The observed impacts of a heatwave can be larger than expected, based on models of mortality and morbidity predicted from ambient temperature exposure. 13-17 This has also been observed as unexpected, dramatic consequences following several heatwaves—for example, the 2003 European heatwave. To date, only a few studies have tried to simultaneously model the additional impacts due to the length of extreme heat exposure with the impacts of high temperatures. $^{\rm 13\ 15\ 19\ 20}$ Most such studies estimate an additional heatwave effect including an indicator when temperature increases above a threshold for at least a prespecified number of days and, thus, force the additional RR to be at the same level on the third day of a heatwave as on the tenth day of the heatwave. However, studies of heatwaves have shown increasing risk with increasing length of the heatwave. 10 14 Currently, little research has indicated a similar increase corresponding to the persistence of exposure during cold spells. 13

The effects of heat exposure have been reported to be greater in the beginning of the warm season. $^{4 \ 14}$ Earlier studies attribute this to acclimatisation; however, this may also be due to a greater number of susceptible individuals earlier in the summer season. It has been shown that the size of the fragile pool of individuals may influence the effect estimates downward when the pool of fragile individuals is smaller. 17 $^{21-23}$

OBJECTIVES

The objective is to study the association between daily temperature and mortality in Stockholm, Sweden, and the additional mortality associated with persistence of extreme temperatures. We examine total mortality (excluding external causes); deaths due to cardiovascular causes, respiratory causes and other causes; and deaths stratified by age. We estimate the association between temperature and deaths as a function of time within summer and winter season. We also aim to predict the consequences of a heatwave according to the estimates from analyses of total mortality.

METHODS Health data

We collected data for Stockholm County from the Swedish Cause of Death Register at the Swedish National Board of Health and Welfare for the period 1990—2002. The mortality data was used to construct bases of daily total mortality excluding external causes (ICD-9:800-999 and ICD-10:E), cardiovascular mortality (ICD-9:390-459 and ICD-10:I), respiratory mortality (ICD-9:460-519 and ICD-10:J) and non-cardiorespiratory deaths. We divided deaths into age strata of 0—44 years, 45—64 years, 65—79 years and 80 years or older. To control estimates associated with temperature from potential confounding from flu, we also acquired the daily number of flu deaths in Stockholm County in the winter period (ICD-9:487 and ICD-10:J10-11).

Environmental variables

We collected meteorological and air pollution data from the main monitoring station at Torkel Knutssongatan in Central

Stockholm, which is managed by the Environment and Health Administration. The variables collected included daily mean temperature, daily maximum temperature, daily minimum temperature, daily mean relative humidity, daily mean levels of nitrogen oxides and daytime 8 h mean ozone levels.

We computed indexes of the maximum and minimum apparent temperature by first calculating the dew point temperature using an improved Magnus form approximation. The discomfort index of apparent temperatures was approximated by the formula: $AT=-2.653+0.994T+0.0153(DT)^2$, where T is the daily maximum or minimum temperature, and DT is the daily mean dew point temperature.

We constructed variables for lagged effects of exposure as average exposure by lag 0-1, lag 0-6 and lag 0-13. These lag periods have been shown to satisfactorily describe mortality associated with weather in the study area. 13 Additionally, in order to incorporate effects associated with prolonged heat and cold stress without time for physiological recovery, we created variables describing the number of days with persistent extreme temperatures taking the values: 0, or the day number in sequence of consecutive days above the 98th percentile or below the second (cold) percentile value of the daily temperature. We assigned the value, 0, to the first day because the accumulation of effects had not started. For example, the variable was assigned the values (0, 0, 2, 3, 4, 0 and 0) in a week if temperatures were below the 98th percentile on day 1, above the 98th percentile on days 2-5 and below the 98th percentile on the last 2 days of the week. Percentiles corresponding to the extreme are often used to describe the effect of extreme temperature on human health. We based our heat effect threshold on the daily mean temperature of 21.6°C, which coincides with the 98th percentile of daily mean temperature over the study period; it is also the heat-effect threshold estimate identified in a prior study. ²⁷ The day sequence of consecutive extreme temperature describes the effects of extreme exposure as a function of the time of persistence.

The environmental data are presented in table 1. Observations missing were not imputed.

Statistical analysis

Data were analysed separately according to a summer and winter period to have the observations on each side of the minimum mortality temperature point separating heat effects from cold effects. Winter included December to February, and

Table 1 Environmental variables and number of deaths in Greater Stockholm Country, 1990—2002

| Environmental variables | June to August | December to February |
|---------------------------------|----------------------------|----------------------------|
| Temperature (°C) | Missing: 8% | Missing: 6% |
| Daily mean ± SD | 16.9 ± 3.2 | $-0.43\!\pm\!4.2$ |
| Minimum, 1990-2002 | 5.8 | -18.5 |
| Maximum, 1990-2002 | 33.5 | 12.9 |
| Apparent temperature (°C) | Missing: 9% | Missing: 8% |
| $Mean \! \pm \! SD$ | 16.1 ± 4.1 | $-2.6\!\pm\!3.6$ |
| Minimum, 1990-2002 | 3.2 | -17.0 |
| Maximum, 1990-2002 | 34.6 | 10.8 |
| NO_x (24 h, $\mu g/m^3$) | Missing: 32% | Missing: 29% |
| $Mean \pm SD$ | 23.0 ± 12.0 | 40.4 ± 30.5 |
| Ozone (8 h, μg/m ³) | Missing: 7% | Missing: 31% |
| $Mean \! \pm \! SD$ | 57.2 ± 15.2 | 34.2 ± 14.1 |
| Total no of deaths over the stu | dy period in age strata (e | excluding external causes) |
| 0—44 | 1512 | 1500 |
| 45-64 | 5551 | 5921 |
| 65-79 | 15258 | 17117 |
| 80+ | 22417 | 26897 |

summer included June to August. For each calendar time period, we established time-series Poisson regression models to describe the effects of the explanatory variables on the mortalities, which made allowances for overdispersion. The calendar variables adjusted for were: year; month; weekday; national holiday; and the day prior to the national holiday. In addition, we ran models to evaluate the sensitivity of the prior model to adjustment for within-year and between-year time trends using a natural cubic spline approach allowing each season three different maximum degrees of freedom. The degrees of freedom used for the natural cubic spline basis were 2, 3 and 4 for each summer or winter season (26, 39 and 52 respectively).

The predictors of mortality considered in this study (temperature, apparent temperature, NO_x ozone and flu) were first modelled as smooth functions. We identified the best predictors of daily total mortality using the Akaike Information criterion (AIC) in terms of temperature, apparent temperature, ozone and NO_x in the prespecified lag strata. If the smooth curves showed that linear relationships were suitable, and the model performance according to AIC was equal or higher for such a model, then the final models were based on a linear relationship. A priori, we decided to keep confounders and interaction between explanatory variables in the model only if they had a significant (p<0.1) impact on the rates of mortality.

We controlled for flu when modelling the effects of cold weather on mortality in winter by including the daily number of flu deaths as a linear predictor in the regression model.

The effect of persistence of extreme temperatures and the effect of temperature were modelled simultaneously in all the regression models. We presented estimates from models as relative risks (RR). CIs are presented at the $\alpha{=}0.05$ level. Model diagnostic tests such as partial autocorrelation, heteroscedasticity and normality were assessed to ensure modelling assumptions were not violated.

Using the estimates from this study, we predicted the attributed relative increase in mortality using a temperature scenario. The temperature scenario consists of 15 days with the 4th to the 10th day (7 days) exceeding the heat episode threshold. See figure 1, for information on the maximum and minimum apparent temperature used in the scenario created for illustrative purposes.

RESULTS

Descriptive statistics of the environmental data used in the analysis are presented in table 1, which shows the mean daily

frequency of deaths (excluding external causes) by age strata during the study period. In the summer period, the mean of the minimum apparent temperature (lag 0-1) best described mortality according to AIC. The shape of the temperatureresponse curve was slightly steeper at higher temperatures, but showed no significant effect modification of the slope according to piecewise linear models. The variable describing persistent heat was also satisfactorily described by a linear variable. In winter, the optimal predictor of mortality was a linear 7-day mean of minimum apparent temperature (lag 0-6). The differences between maximum, minimum, and mean temperature and apparent temperature as predictors of daily mortality were minimal. However, when modelling the effect of persistent exposure to temperatures above the 98th percentile and below the 2nd percentile, the best predictor was the daily maximum apparent temperature. The 98th percentile of the maximum apparent temperature during the study period was 27.5°C, while the 2nd percentile of maximum apparent temperature was -5.9°C. The number of days in sequence with persistent heat and cold exposure is presented in table 2. Both summer and winter models included a term of minimum apparent temperature for the everyday effects of temperature and a term based on maximum apparent temperature that estimated effects associated with persistent extreme heat or cold exposure as a function of the number of consecutive days in a row with high or low temperature.

The relative risks associated with apparent temperature in summer and winter are presented separately for the temperature variable and for the variable corresponding to persistence of extreme temperature, but were estimated simultaneously. For total mortality, the RR associated with a one-unit increase in minimum apparent temperature in summer (lag 0–1) was statistically significant with RR=1.006 (95% CI 1.001 to 1.010), and the additional RR associated with the day number in sequence of extreme heat (beyond day 2) was statistically significant with RR=1.024 (95% CI 1.010 to 1.038). For cardiovascular mortality, the RR associated with the persistence of extreme heat was statistically significant, while indicating an effect on respiratory causes (table 3). Mortality in non-cardiorespiratory causes was associated with heat but not cold exposure.

The relative increase in total mortality associated with a 1° C decrease in minimum apparent temperature in winter (lag 0-6) was statistically significant with RR=1.006 (95% CI 1.001 to 1.010), while the RR associated with the day number in sequence of extreme cold (beyond day 2) was non-significant

Figure 1 Expected increase in total mortality excluding external causes (bars) compared with a baseline minimum apparent temperature at 14°C. Above this arbitrarily chosen baseline level, the mortality increases with RR=1.006 (95% CI 1.001 to 1.010) per degree increase in minimum apparent temperature. The finely dotted line corresponds to the threshold of extreme heat exposure of 27.5°C of maximum apparent temperature. Above this line, the risk increases with RR=1.024 (95% CI 1.010 to 1.038) per day starting on day 2 of the heat event.

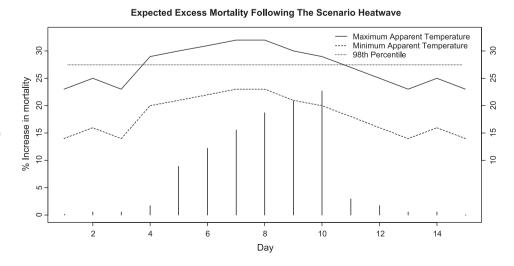


Table 2 Occurrence of persistent extreme temperature in Stockholm, 1990–2002

| No of consecutive days | 2 | 3 | 4 | 5 | 6 | 7 |
|--|----|----|---|---|---|---|
| No of days with consecutive extreme heat exposure (apparent maximum $>$ 98th pct or 27.5°C) | 20 | 12 | 8 | 6 | 3 | 2 |
| No of days with consecutive extreme cold exposure (apparent maximum <2nd pct or -5.9°C) | 22 | 10 | 6 | 4 | 1 | 0 |

with RR=1.007 (95% CI 0.990 to 1.025). However, for respiratory mortality, there was an indication of a large increase in RR with the persistence of cold extremes. For other causes of death, the RR associated with cold was statistically non-significant.

The relative risks associated with apparent temperature in the summer by age strata are presented in table 3. The RR corresponding to a 1°C increase in minimum apparent temperature in summer was statistically significant for the oldest age strata (80 years and above). For persistent exposure to extreme high apparent maximum temperature, there was a statistically significant risk increase per additional day in a sequence for the age groups from 65 years and above. For the ages 45–64 years, there was an indication of a larger risk of death per additional day of extreme exposure (p=0.07).

A 1°C decrease in apparent minimum temperature in winter was associated with a borderline significant increase in mortality in the age strata 45–64 years, as well as a statistically significant increase for ages 80 years and above. There was also an indication of increasing risks in ages 65–79 years. The persistence of extreme cold exposure shows no additional effect on mortality.

As shown in table 4 the effect estimates for minimum apparent temperature in the warm season stratified by month show statistically significant relative risks in the beginning of the summer (June and July) that diminish in the last summer month (August). When estimating the decline in the effect as a function of time in days in summer, the baseline RR of minimum apparent temperature was estimated to be 1.011 (1.005 to 1.016) for the first of June. For each day later in the season, the effect declines with RR corresponding to 0.99992 (0.99987 to 0.99998). This means that the effect 62 days after first of June (1 August) corresponds to 1.011×0.99992⁶² or an RR of 1.006 per degree temperature increase in summer.

In contrast, the estimates for persistence of extreme high maximum apparent temperature were approximately the same for July and August, but could not be estimated for June because of a lack of long, warm periods earlier in the summer season (see table 4).

The estimates associated with minimum apparent temperatures in the cold season showed statistically significant relative

risks in the early cold season that diminish later in the cold season. For persistence of extreme temperatures, the effect was non-significant in the winter months (see table 4).

Confounding or interaction with air pollution in lag strata up to 14 days was not apparent, and the effect of air pollution on mortality was non-significant (p>0.1).

The validation using a different control for time trend did not cause the estimate to change from statistically significant to non-significant or vice versa. However, some point estimates changed to a small extent, and standard deviations (p values) were slightly different.

Figure 1 illustrates the predicted impacts of a 1-week hypothetical heatwave in the study area given increases in minimum apparent temperature (baseline 14°C) and maximum apparent temperature (baseline 23°C). The threshold defining a persistent extreme heat event is the maximum apparent temperature greater than 27.5°C. The predicted impacts of the temperatures graphed correspond to the relative risks estimated for high temperatures, RR=1.006 (95% CI 1.001 to 1.010) per 1°C; and for persistent extreme temperatures, RR=1.024 (95% CI 1.010 to 1.038) per day longer episode. We only have data for the impact of persistent hot temperatures up to 7 days, and so we set the maximum apparent daily temperature to above 27.5°C for a maximum of 7 days without extrapolation.

DISCUSSIONS

We separated the effects associated with temperature in general with the effects associated with extreme persistent temperature using time-series models. The results based on a single Nordic population, the inhabitant in Stockholm Country, show that persistent exposure to extreme temperature is associated with additional effects in hot condition in contrast to cold conditions, and that the effects on mortality increase with the duration of the heat episode. The results further show that persistent extreme heat and heat in general affect deaths differently with respect to age and cause of death. In particular, cardiovascular deaths seem to be more related to persistent extreme heat than to heat in general. Moreover, deaths during extreme persistent heat appear to occur in people from 45 years of age and above, while the effect of high temperature only is seen for deaths in ages 80 years and above. Our results support the idea that extreme persistence of exposure is a key component for inclusion in models when relating high ambient temperatures (in a relative context) to mortality, and the variable has a strong physiological basis in the effects of prolonged heat stress (dehydration corresponding to increased stress on the cardiovascular system and kidneys). Several prior studies

Table 3 Relative risks (RR) associated with temperature and persistent extreme temperatures in cause-specific groups and age strata adjusted for potential confounding, calendar patterns, seasonality and long-term time trends.

| | Estimated simultaneously | in summer model | Estimated simultaneously in winter model | | |
|--|--|---|--|---|--|
| | RR associated with a 1°C increase in minimum apparent temperature (lag 0—1) | RR associated with day number in sequence of persistent extreme hot temperature | RR associated with a 1°C decrease in minimum apparent temperature (lag 0—1) | RR associated with day number in sequence of persistent extreme cold temperature | |
| All causes (excluding external) | 1.006 (1.001 to 1.010) | 1.024 (1.010 to 1.038) | 1.006 (1.001 to 1.010) | 1.007 (0.990 to 1.025) | |
| Cardiovascular causes | 1.004 (0.998 to 1.009) | 1.020 (1.000 to 1.041) | 1.014 (1.008 to 1.020) | 0.994 (0.970 to 1.019) | |
| Respiratory causes | 1.014 (0.997 to 1.030) | 1.039 (0.987 to 1.094) | 1.000 (0.987 to 1.014) | 1.022 (0.970 to 1.077) | |
| Other causes | 1.007 (1.001 to 1.013) | 1.023 (1.003 to 1.042) | 1.002 (0.996 to 1.008) | 1.002 (0.996 to 1.008) | |
| All causes (excluding external) ages 0-44 | 0.994 (0.972 to 1.018) | 0.977 (0.892 to 1.071) | 1.010 (0.983 to 1.036) | 0.939 (0.833 to 1.058) | |
| All causes (excluding external) ages 45-64 | 0.992 (0.980 to 1.004) | 1.036 (0.996 to 1.079) | 1.012 (0.999 to 1.023) | 0.995 (0.947 to 1.046) | |
| All causes (excluding external) ages 65-79 | 1.004 (0.996 to 1.010) | 1.028 (1.004 to 1.052) | 1.004 (0.996 to 1.011) | 0.996 (0.967 to 1.027) | |
| All causes (excluding external) ages 80+ | 1.011 (1.005 to 1.017) | 1.021 (1.002 to 1.040) | 1.006 (1.000 to 1.011) | 1.019 (0.996 to 1.042) | |

Table 4 Relative risks (RR) associated with temperature stratified by month and persistent extreme temperatures in cause-specific groups and age strata adjusted for potential confounding, calendar patterns, seasonality and long-term time trends.

| | Summer | | | Winter | | |
|--------|--|--|----------|--|---|--|
| Month | RR associated with a 1°C increase in minimum apparent temperature (lag 0—1) | RR associated with day number in sequence of persistent extreme hot temperature | Month | RR associated with a 1°C decrease in minimum apparent temperature (lag 0—1) | RR associated with day number in sequence of persistent extreme cold temperature | |
| June | 1.008 (1.001 to 1.014) | _ | December | 1.010 (1.003 to 1.017) | 0.996 (0.963 to 1.031) | |
| July | 1.008 (1.001 to 1.015) | 1.023 (1.006 to 1.040) | January | 1.007 (1.000 to 1.014) | 1.016 (0.986 to 1.047) | |
| August | 1.003 (0.997 to 1.009) | 1.026 (1.005 to 1.047) | February | 1.001 (0.993 to 1.008) | 1.007 (0.982 to 1.032) | |

have found death rates to increase rapidly and in addition to an average effect of hot temperatures. S 13 17 19 28 29 However, our approach contributes to an easily comprehended RR of how the risk increases for each day of a heatwave. Knowing this relationship can help public health actions during heatwaves given an accurate weather forecast. The results further suggest that the public health could have large benefits of such actions, even if actions are started after the heatwave onset, as the largest death rates can be expected at the end of the heatwave. Further, multicity studies are needed to repeat this finding separating the effects associated with hot weather in different climate regimes with varying population characteristics.

Heat and cold exposure were found to impact death rates significantly, but differently according to underlying cause of death. We found that heat elevated death rates for cardiovascular, respiratory and non-cardiorespiratory causes, while cold exposure mainly elevated the risk of cardiovascular deaths. The increase in non-cardiorespiratory deaths with hot temperatures appeared to be relatively larger than the increase in cardiovascular deaths, with respiratory deaths increasing most, but the number of deaths is fewer and effect estimates more uncertain. Previous studies support the fact that respiratory deaths increase significantly with warm ambient temperatures, 4 13 as well as among adults during persistent extreme heat exposure. 10 A European multicity study found no increases in cardiovascular mortality in north Europe with elevated temperature. 4 However, the authors may have found stronger increases in north Europe and in younger age groups if the impacts of persistent extreme temperature had been taken into account.

Some studies found risks related to heat to be more lagged at higher temperatures, which could affect the estimate of persistent heat exposure. We conducted additional analyses to verify this by fitting a non-linear distributed lag model. From such a model, the relative risks associated with persistent extreme temperature were statistically significant and of a magnitude similar to the estimates in this study, 1.02 (non-published results). In studies that found a change in the timing of the event above a certain threshold, the estimates may capture parts of the effect associated with prolongation of heatwaves shown in this study. However, as a non-linear distributed lag model does not contain a constraint of consecutive days above a certain extreme temperature, such a model is unlikely to explain such effects well. Therefore, the results provide strong evidence that the persistent of heat is a key component to incorporate in models to avoid negatively biased estimates of the effect of heat on mortality. Moreover, a very favourable feature of the approach taken in this study is the simplicity in the interpretation and estimation of the effects, and the comparability between estimates to help identify high-risk individuals.

For exposure to non-persistent high and low minimum apparent temperatures, the impacts decrease in the last month of the summer and winter seasons, respectively. However, the risks associated with persistent, extreme temperatures remain

throughout the summer season. This indicates that the previously suggested acclimatisation effect may not apply for heatwaves.⁴ ¹⁴ An explanation for this could be that the acclimatisation effect suggested in prior studies with larger effects of heat in the early season may result from a larger pool of fragile individuals in the early season. 21 23 27 This reasoning is further strengthened by the estimates of elevated summer temperatures in this study, being significant only on noncardiovascular causes, while acclimatisation corresponds physiologically to a more efficient sweat production reducing the cardiovascular stress. Moreover, the winter temperature estimates indicate a similar pattern, while there is little evidence in the literature that acclimatisation to cold temperatures occurs. Our results may suggest that when exposed to extreme, persistent high temperatures, the fragile pool would likely empty at the same rate as it would fill by new individuals from the population transferred into a more fragile state due to the extreme environmental conditions. However, when exposed to non-persistent extreme temperatures in summer and winter, this pool becomes smaller over the course of the season. This may be further supported by the contrast in relative risks by age groups between non-persistent and persistent extreme heat, as we found persistent heat episodes to affect lower ages than does heat in general, and since the susceptible pool is likely to be determined by the number of very elderly in the population, as are the main risk group to non-extreme persistent temperatures. A similar situation was observed after the killer smog event in London in December 1952; a high mortality was observed many months after the smog event. 30 However, adaptation (eg, behavioural responses) within summer and winter can also influence these estimates.

Similar to a prior study, our study found rather small differences among the temperature predictors. The predictors indirectly incorporating the effects of temperature and humidity described mortality slightly better. However, the small difference between apparent temperature and ordinary temperature in explaining daily mortality may result because the ecological design is not finely tuned enough for such exposure measures. An interesting observation in our results is that the minimum apparent temperature better explains mortality in summer and winter; however, when the exposure is extreme and persistent, the maximum apparent temperature describes the impacts on summer mortality better. Minimum daily temperature may be a better predictor because it describes if there is relief from the heat during the night.

We did not find a significant influence of air pollution on the impacts of heat and cold, and neither did a prior study. However, some studies have found that air pollution is influential and possibly biases the temperature impacts even if only to a very small extent. 19 31

A prior study tested the sensitivity of selecting the threshold when estimating the additional heatwave effect, and found that the impact estimate increased with increasing thresholds. It also

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found that the effects depended on the minimum number of days in a row used when defining a heatwave.¹⁹ The approach taken in this study incorporates the increase in risk with the length of a heatwave but has not tested different cut points. However, it is likely that changing the cut point upward and downward would influence the RR estimate. This needs to be explored in future studies.

Climate change is projected to change the frequency, intensity and duration of heatwaves. There is considerable interest in projecting how heat-related mortality could change with a changing climate. Our study suggests that projections of possible future mortality based only on changes in mean temperature may underestimate the magnitude of impacts. Improvements in the ability of climate models to project climate variability can be used to generate better estimates of possible future health impacts under different climate scenarios.

CONCLUSIONS

Mortality related to high ambient temperature is not sufficiently described by simple variables of the daily or lagged temperature; such models should include variables of the persistence of extreme temperatures as well. The relative risks associated with high temperatures show that older persons are at greatest risk, while a much broader age range face elevated risks of death during persistent extreme heat events. During persistent extreme exposure, the daily relative risks increase with the persistence of the episode. Persistent extreme temperatures appeared to be homogeneous in summer, while the relative risks associated with a one-unit change in heat and cold diminish in the later part of the season. This may be related to a smaller population at high risk (fragile pool) or due to adaptation/ acclimatisation. Future research should focus on identifying risk groups to temperature and persistent extreme temperatures and verify the finding from this study in other settings. Moreover, improved health impact models are needed to better understand the possible consequences of projected increases in the frequency, intensity and duration of heatwaves in a changing climate.

Competing interests None.

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