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Effect of night-time temperatures on cause and age-specific mortality in London

Peninah Murage*, Shakoor Hajat, R. Sari Kovats

Background: High ambient temperatures are associated with an acute increase in mortality risk. Although heat exposure during the night is anecdotally cited as being important, this has not been rigorously demonstrated in the epidemiological literature.

Methods: We quantified the contribution of nighttime temperatures using time-series quasi-Poisson regression on cause and age-specific daily mortality in London between 1993 and 2015. Daytime and nighttime exposures were characterized by average temperatures between 9 am and 9 pm and between 4 am and 8 am, respectively, lagged by 7 days. We also examined the differential impacts of hot and cool nights preceded by very hot days. All models were adjusted for air quality, season, and day of the week. Nighttime models were additionally adjusted for daytime exposure.

Results: Effects from nighttime exposure persisted after adjusting for daytime exposure. This was highest for stroke, RR (relative risk) = 1.65 (95% confidence interval (CI) = 1.27 to 2.14) estimated by comparing mortality risk at the 80th and 99th temperature percentiles. Compared to daytime exposure, nighttime exposure had a higher mortality risk on chronic ischemic and stroke and in the younger age groups. Respiratory mortality was most sensitive to daytime temperatures. Hot days followed by hot nights had a greater mortality risk than hot days followed by cool nights.

Conclusions: Nighttime exposures make an additional important contribution to heat-related mortality. This impact was highest on warm nights that were preceded by a hot day, which justifies the alert criteria in heat-health warning system that is based on hot days followed by hot nights. The highest mortality risk was from stroke; targeted interventions would benefit patients most susceptible to stroke.

Keywords: Climate change, Environment, Heat wave warning system, London, Mortality, Night time, Temperature, Urban Heat Island

Introduction

The effect of extreme heat on health is well documented; very high ambient temperatures have been associated with an increase in mortality,^{1–4} in hospital admissions,^{5–8} and with other morbidity outcomes. Climate change is expected to increase the frequency and intensity of hot weather.⁹ This has led to the recognition of extreme heat as a public health hazard; the 2003

heat wave across Europe, for example, led to the development of heat-health warning systems (HHWS) across European countries.^{10,11} The HHWS in England raises public awareness and triggers actions in health and social care to limit the adverse effects when average temperatures exceed daytime and nighttime regional thresholds; the London region thresholds are 32°C and 18°C, respectively.^{11,12} Elevated nighttime temperatures are of specific concern because of the lack of relief from high temperatures at night.^{9,13} There is some evidence to suggest that the increased probability of death during the 2003 heat wave in France was worsened by higher elevated nighttime temperatures.¹³ However, most of the existing evidence is based on daily minimum temperatures^{14–16} and may be criticized because of the confounding effect of daytime temperature exposures.¹⁷ This leaves scope for more detailed assessments of the thermal characteristics of the night and their impact on health.¹⁸

Heat-related mortality is higher in urban areas than in rural and surrounding areas^{9,13,19,20} because of the urban heat islands effect.⁹ The temperature difference between urban and rural areas shows a distinct hourly variation, where the greatest differences are observed in the evenings and early morning hours.^{21,22} Cardiac and cerebrovascular events also exhibit a similar diurnal

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The mortality data was requested from the Office of National Statistics (ONS) for specific use in this project and is not in the public domain. The environmental was downloaded with permission from the Medical & Environmental Data Mash-up Infrastructure (MEDMI) database. Researchers can request this data from this link <https://www.data-mashup.org.uk/>. All coding was conducted using the distributed lag nonlinear model (dlm) package on R; more information about how to use this package can be found at <http://www.ag-myresearch.com/package-dlm.html>.

Sponsorships or competing interests that may be relevant to content are disclosed at the end of the article.

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What this study adds

Exposure to high daytime temperatures is known to increase the risk of death in vulnerable individuals. This study shows that high nighttime temperatures carry an additional risk of heat-related death, which may be highest in patients with heart diseases, particularly those most likely to suffer from stroke and chronic ischemic diseases. In addition, nighttime temperatures make an important contribution to mortality risk in people below the age of 65 years. The study also demonstrated that warm nights preceded by a hot day have the greatest health impacts, indicating that nighttime temperatures are an important consideration when developing heat-health warning systems.

pattern whereby the occurrence of deaths from cardiovascular and cerebrovascular conditions peaks in the morning hours.^{23–27} This distinct pattern in cardiac events has been linked to the mammalian circadian rhythm,^{23–27} and it has been suggested that sleep patterns and temperature are important exogenous factors for the circadian rhythm in humans.^{23,28–30} However, there is no evidence that explicitly links the diurnal pattern of cardiovascular events with diurnal changes in temperature.

In this study, we examine the heat effect of summer nighttime temperatures on mortality in London. London is an appropriate case study because it is a predominantly urban area; the higher density of buildings and lower density of vegetation compared with rural and suburban areas suggests that nighttime temperatures are likely to be elevated during the summer months,²² which could have a measurable impact on health. Additionally, a social survey of London found that the high levels of transient communities has increased social isolation amongst some long-term residents, and this has increased their vulnerability during extreme weather.³¹

We hypothesize that nighttime temperatures make a substantial contribution to the heat-related mortality burden over and above exposures experienced during the daytime. Climate change and a growing urban population will likely increase minimum temperatures and make the heat-island effect more prominent; the need to characterize the risk from nighttime exposures is, therefore, becoming increasingly important. In addition to examining the effect of high nighttime temperatures, we also compare the difference in risk between warm nights preceded by a hot day and cooler nights that were also preceded by a hot day. This will help ascertain the validity of the English HHWS system that issues warnings based on a hot day followed by a hot night.¹¹ Finally, we examine differences in age and disease-specific mortality to understand the underlying mechanisms that may explain any effect on mortality from nighttime heat stress.

Methods

We obtained daily counts of cause- and age-specific mortality for the Greater London region for the period beginning 1 January 1993 to 31 December 2015. Hourly observed air temperature and relative humidity data was obtained from the Medical & Environmental Data Mash-up Infrastructure (MEDMI) data set.³² The London region was specified as the area covered by a 30 km radius from a central London location of 51.5° N, 0.13° W, which is the location of the British Museum (Fig. 1). Hourly air temperature and relative humidity data was obtained from 31 weather stations located within this area, and region-wide and temporal means were estimated. Hourly specific humidity was thereafter computed by calculating water vapor pressure using air temperature and relative humidity data and applying a mixing ratio³³ where standard air pressure was assumed to be 1013.25 hPa.³³

Primary outcomes were defined as age-specific mortality for 0–64, 65–74, and 75 plus years and cause-specific mortality for the following causes: cardiovascular (ICD10 I00–I99), respiratory diseases (J00–J99), diseases of the nervous system (G00–G99), endocrine, nutritional and metabolic disease (E00–E99), and mental and behavioral disorders (F00–F99). Disease-specific mortality for the following diseases was also examined: stroke (I60–I69), chronic ischemia (I25), myocardial infarction (I22–I23), heart failure (I50), chronic obstructive pulmonary disease and chronic bronchitis (J40–J44), diabetes (E10–E14), and infectious respiratory diseases (J10–J22).

Daily mean temperatures were estimated as average temperatures between 9 am and 9 pm. Nighttime temperatures were estimated as the average temperature between 4 am and 8 am, to correlate with the hours that may be most sensitive to physiological processes related to cardiovascular and cerebrovascular events described in the Introduction. Hot nights were identified

as nights where the daily minimum and daily maximum temperatures were above 16°C and 25°C, respectively. Cool nights were identified as nights where the daily maximum was also above 25°C, but daily minimum was below 16°C. These temperature thresholds were used to provide the maximum number of hot and cool nights in the time series. It was not possible to use the HHWS London alert thresholds of a daily maximum of 32°C and a daily minimum of 18°C¹¹ because very few days met this threshold, and this would not have provided sufficient statistical power.

A time-series quasi-Poisson regression analysis was used to examine the association between the temperature variables and the primary outcomes. The analysis was restricted to the summer months between June and September and was conducted on R version 3.3.2 using the *dlnm* (distributed lag nonlinear model) package that simultaneously models the nonlinear and delayed effects between temperature and mortality.³⁴ The delayed effect represents temporal change in mortality after heat exposure and estimates the distribution of immediate and delayed effects that cumulate across a specified time period (lags)³⁵; a lag of period of 7 days was specified in this study.

In the models estimating the effect of night-time heat exposure on mortality, the association was estimated using a cross basis defined by a quadratic B spline for temperature variables, centered at the 80th percentile, and with two internal knots placed on equally spaced values along the temperature distribution. The lag-response was modeled with a natural cubic B spline, with an intercept and three internal knots placed at equally spaced values in the log scale. All models were adjusted for the effect of daytime heat exposure by adding a second cross basis that defined this exposure, using the methodology described above. Detailed information of how the cross basis operate within the *dlnm* package has been previously published.^{34–36} Given the difference in the temperature range between the daytime and nighttime exposures and to allow comparisons between these exposures, the relative risks were estimated by comparing the mortality risk between the 80th and 99th percentile of the summer temperatures for the specific exposure. The 80th percentile roughly corresponds to the point of minimum mortality in the temperature distribution.

The heat effect of hot and cool nights on mortality was also estimated using a cross basis. All nights where the daily minimum exceeded 16°C during the summer period, preceded by a daily maximum temperature of 25°C and above, were binary coded as “hot nights” versus “all other nights.” The models were adjusted for any effect of daytime temperatures below 25°C and nighttime temperatures below 16°C. The lag response was modeled with a natural cubic spline in a similar way to the nighttime models. This methodology was also applied in estimating the effect of cool nights on mortality, adjusting for days where the daytime temperatures were below 25°C and nighttime temperatures were above 16°C. Relative risks were calculated by comparing mortality risk of hot or cool nights with all the other days that did not meet the hot/cool night criteria.

All models were controlled for seasonal variation and long-term trend by fitting a natural cubic spline on the day of year (4 knots per year) and time (3 knots per year) variable, respectively. The argument ‘group’ in the *dlnm* package was used to define groups of observations representing multiple and independent series based on summer months.³⁶ Models were also adjusted for the day of the week and for the effect of daily air quality. The latter reportedly has a synergistic effect with temperature that is most acute in the summer months,^{37,38} and was controlled for by using an unconstrained distributed lag of 0–7 days for ozone and particulate matter with a diameter smaller than 10 µm.

For sensitivity analysis, all models were further adjusted for specific humidity, which calculated to correspond with daytime and nighttime hours of exposure. In addition, we also considered



Figure 1. Map of London with a 30 km radius from a central London location.

models where the nighttime exposure period was derived using different hours of exposure, between 10 pm and 6 am.

Results

Summary statistics of the health and environmental variables are shown in Table 1. Daily mean temperatures ranged from a minimum of 9.0°C to a maximum of 32.4°C. Unsurprisingly,

Table 1
Summary Statistics of Mortality, Temperature, and Air Quality Variables, During the Summer Months

	Min	Median	80th Percentile	99th Percentile	Max
Environmental variables					
Daily minimum temperatures	4.9	13.3	15.5	19.4	21.3
Daily mean temperature	9.0	18.6	21.6	27.8	32.4
Daily maximum temperatures	9.5	20.3	23.6	30.3	36.4
Nighttime average temperature	5.9	14.5	16.6	20.8	23.2
Ozone	2.0	26.0	39.0	73.0	105.0
PM10	5.0	23.0	34.0	70.0	130.0
Mortality variables					
All cause	82.0	136.0	157.0	192.0	280.0
0–64 years	9.0	30.0	36.0	47.0	57.0
65–74 years	6.0	24.0	32.0	45.0	54.0
75+ years	44.0	82.0	94.0	116.0	196.0
Cardiovascular diseases	18.0	47.0	60.0	82.0	118.0
Myocardial infarction	0.0	9.0	15.0	26.0	36.0
Chronic ischemic	2.0	13.0	18.0	27.0	45.0
Stroke	2.0	11.0	15.0	23.0	33.0
Heart failure	0.0	2.0	4.0	7.0	10.0
Respiratory diseases	5.0	18.0	24.0	35.0	58.0
COPD	0.0	6.0	8.0	13.0	16.0
Infectious respiratory	0.0	9.0	15.0	25.0	37.0
Endocrine diseases	0.0	2.0	3.0	6.0	8.0
Diabetes	0.0	1.0	3.0	5.0	7.0
Nervous system diseases	0.0	3.0	5.0	9.0	15.0
Mental health	0.0	3.0	6.0	14.0	19.0

COPD, chronic obstructive pulmonary disease; PM10, particulate matter with a diameter smaller than 10 µm.

daily mean temperatures were higher than nighttime average temperatures: median of 18.6°C versus 14.5°C, respectively. The temperatures at the 80th and 90th percentile used to estimate the RRs in this study are 21.6°C and 27.8°C for the daily mean models and 16.6°C and 20.8°C for the nighttime models (Table 1).

The median daily mortality from all causes across the summer months was 136, and the largest burden from mortality was from the eldest age group (75 years plus) and from cardiovascular diseases (Table 1).

Table 2 shows the distribution of variables used in the hot and cool nights analyses. There were 2806 days in the time series; of these, 225 were categorized as hot nights that preceded a hot day and 143 as cool nights that preceded a hot day.

Daytime exposure was associated with an increase in mortality in all the models examined (Fig. 2A). Mortality risk at the 99th temperature percentile after daytime exposure was highest for heart failure (relative risk (RR) 1.75; 95% confidence interval [CI] = 1.37, 2.25), infectious respiratory diseases (RR 1.67; 95% CI = 1.47, 1.89), and diseases of the nervous system (RR 1.66; 95% CI = 1.34, 2.04; Fig. 3A).

A residual heat effect on mortality was also observed following the night-time heat exposure, which persisted after controlling for daytime exposure (Fig. 2B). The mortality risk at the 99th percentile compared with 80th percentile for respiratory diseases was higher after daytime exposures, whereas mortality risk from some cardiovascular conditions and in the younger age groups was higher after nighttime exposures (Fig. 3A, B). The disease- and age-specific models reveal that the heat effect of nighttime exposures may be higher than the effect from daytime exposures in stroke (RR 1.70; 95% CI = 1.32, 2.19), chronic ischemic diseases (RR 1.46; 95% CI = 1.16, 1.84), under 64 years (RR 1.21; 95% CI = 1.03, 1.42), and in 65–74 years (RR 1.30; 95% CI = 1.10, 1.59; Fig. 3B).

Hot nights that were preceded by a hot day carried a greater mortality risk than cool nights that were also preceded by a hot day. The mortality risk of hot nights was higher across most of the disease areas examined (Fig. 3C, D); the exception was mortality risk from endocrine diseases, where exposure to cool

Table 2
Summary of Variables in the Hot and Cool Nights Analysis

	Variables	Number of Days	Average Per Year
1	All days	2806	128
2	Hot nights preceded by a hot day (hot nights); daily max above 25°C and daily min above 16°C*	225	10
3	All other days (in relation to hot nights)	2581	117
4	Cool nights preceded by a hot day (cool nights); daily max above 25°C and daily min below 16°C†	143	7
5	All other days (in relation to cool nights)	2663	121
6	Cool nights preceded by a cool day; daily max below 25°C and daily min below 16°C	2255	103
7	Hot nights preceded by a cool day; daily max below 25°C and daily min above 16°C	188	9

*Relative risk (RR) compares mortality on hot nights (2) with all other days (3) (2806–225), adjusted for cool nights preceded by cool days (6).

†RR compares mortality on cool nights (4) with all other days (5) (2806–143), adjusted for hot nights preceded by cool days (7).

nights carried a greater effect on mortality than exposure to hot nights (Fig. 3C, D). Mental illnesses carried the highest mortality risk from exposure to hot nights (RR 1.61; 95% CI = 1.28, 2.03; Fig. 3C), estimated as mortality risk from exposure to hot nights, compared to all other days.

Repeating the analysis using nighttime temperatures estimated between 10 pm and 6 am did not change the observed patterns on Fig. 2B; this, however, reduced the relative risks in most models, apart from mental health and endocrine diseases where there was marked increase in the relative risk (presented in the supplementary material: eFigure 1a and b; <http://links.lww.com/EE/A0>).

Controlling for specific humidity in some of the age- and disease-specific models had an opposite effect on the mortality risk associated with day exposure, compared to mortality risk from nighttime exposure. For example, where mental health was the outcome, adjusting for specific humidity reduced the mortality risk from night exposure from RR 1.52 (0.95–2.45) to RR 0.77 (0.44–1.34). However, adjusting for the same increased the mortality risk from day exposure from RR 1.29 (1.05–1.59) to RR 1.53 (1.21–1.93). In contrast, where stroke was the outcome, adjusting for specific humidity increased the mortality risk after both day and night exposures, from RR 1.32 (1.18–1.49) to RR 1.48 (1.31–1.68) and from RR 1.7 (1.32–2.19) to RR 1.81 (1.34–2.46), respectively. The highest change in risk when the outcome was stroke was when nighttime was defined as hours between 10 pm and 6 am, whereby adjusting for specific humidity resulted to a change in mortality risk from RR 1.73 (1.33–2.26) to RR 2.46 (1.67–3.62).

Adjusting for specific humidity, however, did not alter our main finding, that there is a residual effect on mortality risk, which remains after removing the effect of daytime exposure. Therefore, these results are not included in the main models but have been added to the supplementary material (eFigure 2a–d; <http://links.lww.com/EE/A0>).

Discussion

This is the first comprehensive study of night-time temperatures on mortality in a predominantly urban area. The findings confirm the strong effects of high daytime temperatures on mortality, but also show that exposure to high nighttime temperatures have a substantial effect on mortality; which persists after controlling for daytime temperature exposures. The mortality risk

of night-time exposure was most prominent in stroke patients. Furthermore, in stroke, chronic ischemic diseases, and younger age groups, the mortality risk from exposure to high nighttime temperatures may be higher than the risk of exposure from high daytime temperatures. We also found that, cooler nights that were preceded by hot days experienced lower mortality than hot nights that were also preceded by a hot day. The greatest mortality risk from a hot night was on patients with mental illnesses.

Despite anecdotal evidence of high nighttime temperature effect on health outcomes, there is minimal empirical evidence to demonstrate any associations. One previous study conducted in Paris found that exposure to high nighttime temperatures over several days during the 2003 heat wave, increased the likelihood of death among elderly urban dwellers.¹³ That study, as well as other studies^{14,15} looking at the effect of nighttime temperature on health, used daily minimum temperatures as a proxy for nighttime heat exposure. This choice of the temperature variable can be criticized because of the high correlation between daily minimum and daily maximum temperatures.¹⁷

The increased probability of death in the early morning hours is well documented; mortality from stroke and ischemic diseases is elevated in the morning hours between 6 am and 10 am.^{23,25,27,39} There is also evidence of an increase in heart failure mortality in the morning hours.⁴⁰ Mortality from myocardial infarction also peaks between 6 am and 10 am,^{24,26} although there are also accounts of a later peak between 10 am and 12 am.²³ The circadian rhythm that exhibits a diurnal pattern and regulates physiological processes may explain this morning increase in cardiovascular mortality.^{23–26,40} The rhythm is regulated by endogenous factors such as the diurnal changes in the autonomic nervous system,^{23,40,41} in blood pressure and heart rate,^{24,25} and in the aggregation of platelets.^{24,25} There is also evidence of circadian rhythm manipulation by exogenous factors such as exposure to light, drug use, shift work, stressful events, and change in temperature, a process referred to as circadian rhythm entrainment.^{28–30,42–44}

The elevated mortality in stroke and chronic ischemic diseases after nighttime heat exposure may result from an interaction between these endogenous and exogenous factors. There are several probable mechanisms; first, the circadian clock regulates blood flow to the skin, which then regulates core body temperature,^{29,30} a process associated with thermoregulation. The sleep–wake cycle is sensitive to this temperature–circadian cycle because regulation of thermosensitivity takes place in the areas of brain also involved in the regulation of sleep–wake cycle.²⁹ Feedback of skin temperature to the brain is the primary signal for the thermoregulatory system.²⁹ Furthermore, ambient temperature affects the brain temperature more markedly during a period of rest compared with periods of activity,²⁹ and thereby, sleep quality is reportedly better when the environment is mildly cool.²⁹

Second, opening windows during hot nights may also increase noise-related stress, which reduces the quality of sleep. It is also plausible that insufficient circadian entrainment because of changes in night–time temperature may also affect blood pressure in vulnerable patients. Blood pressure also exhibits diurnal patterns, whereby it is elevated during periods of sleep, and has been associated with increased morbidity and mortality from cardiovascular and cerebrovascular diseases.²⁷ Cold exposure increases blood pressure in ischemic patients, which results in end-organ damage and cardiovascular events.²⁸ Although heat exposure attenuates blood pressure,^{45,46} this may also have morbidity and mortality risk particularly on the elderly.⁴⁶ A follow-up study on participants exposed to the August 2003 heat wave reported a sharp fall in blood pressure values in August 2003 (mean systolic blood pressure, 132 mm Hg) compared to that in August 2004 (mean systolic blood pressure 138 mm Hg).⁴⁶

In the younger age groups, biological processes may be compounded with social risk factors such shift work⁴⁴ or increase

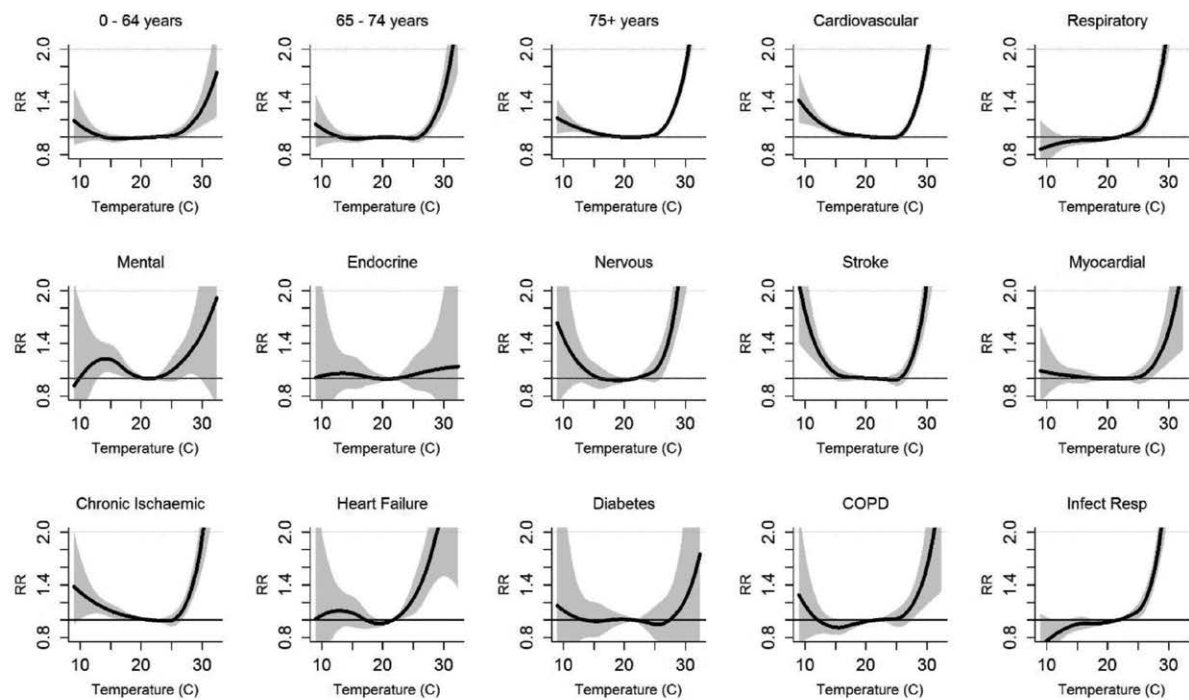
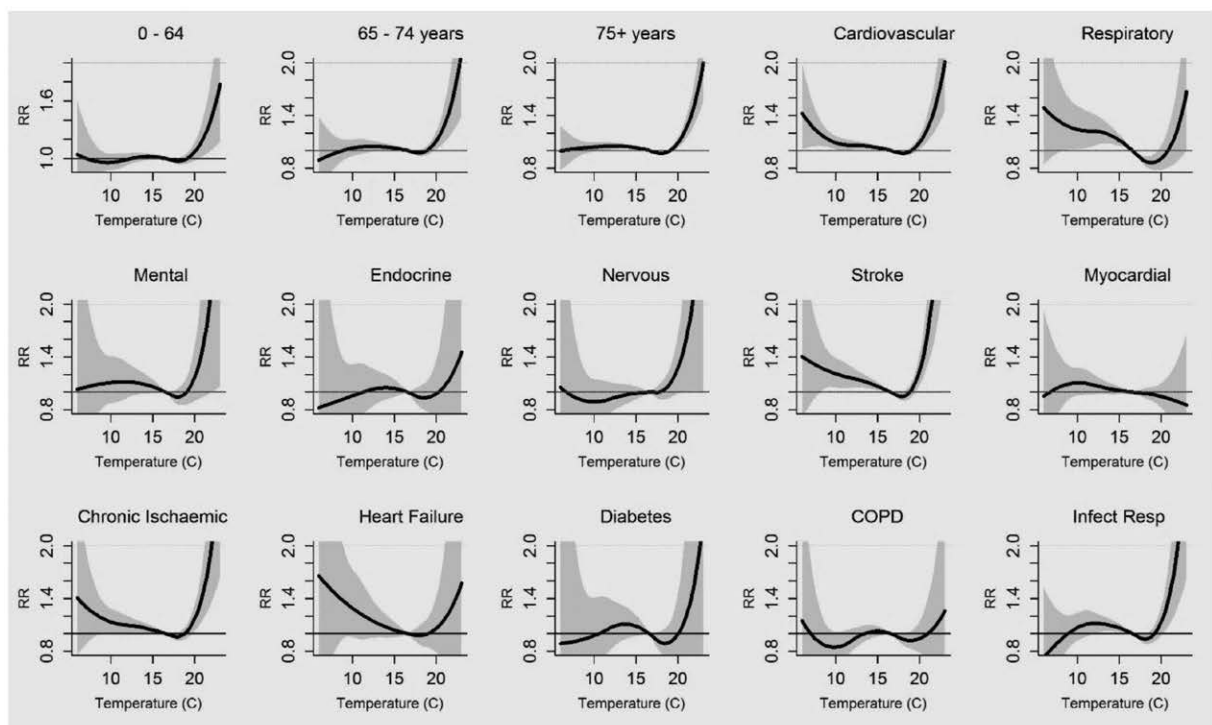
A Exposure to daily mean temperatures**B Exposure to night time average temperatures**

Figure 2. Overall cumulative associations of the heat effect on mortality during summer months, estimated for lags 0–7. A, The effect of daily mean temperatures; and B, the effect of nighttime temperature after adjusting for daily mean temperatures. All models are adjusted for air quality. COPD, chronic obstructive pulmonary disease.

in risk taking activities such as alcohol consumption during the summer months.⁴⁷ Collectively, these factors may act to drive the higher cardiovascular mortality in the younger age groups. The higher risk of death from mental health illnesses that was

associated with hot nights preceded by a hot day may also be related to alcohol and other substance misuse or by use of medication. A study on temperature-related mortality on patients with a mental illness found that increases in temperature elevated

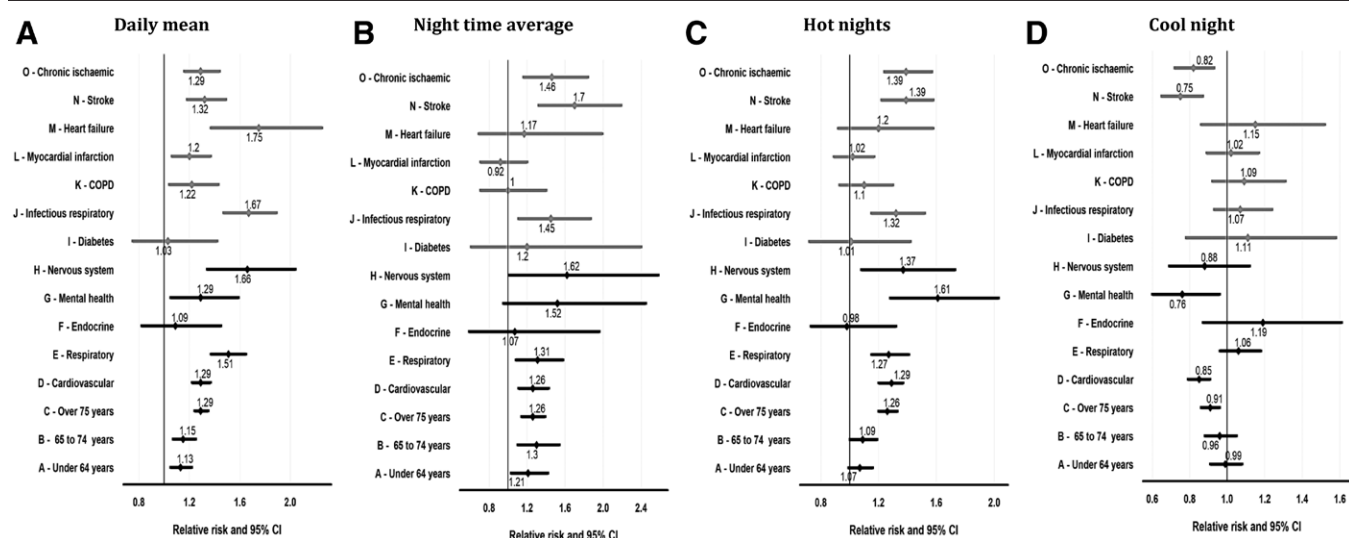


Figure 3. Relative risk (95% confidence interval [CI]) of temperature effect on mortality. A and B, The cumulative associations shown in Figure 2A, B) and is estimated as difference in mortality at the 99th temperature percentile, compared with the mortality at the 80th percentile. C, The risk associated with hot nights^a preceded by a hot day, compared to the rest of the days in the time series; and D, the risk associated with cool nights^b preceded by a hot day compared to the rest of the days in the time series. ^aThese are all days where the daily maximum exceeded 25°C and the daily minimum exceeded 16°C. ^bThese are all days where the daily maximum exceeded 25°C and the daily minimum was below 16°C. COPD, chronic obstructive pulmonary disease.

the risk of death among patients with a primary diagnosis of alcohol or substance misuse and among patients prescribed with hypnotic/anxiolytic and antipsychotic medications.⁴⁸

We did not find any association between mortality from myocardial infarction and high nighttime temperature. The seasonal variation in myocardial infarction occurrences has been documented; risk of admission increases during colder temperatures,⁴⁹ mortality also peaks during winter months²⁶ and has been linked to the reduced number of daylight hours.²⁶ The heat effect on myocardial infarctions is inconclusive; one study found no increase at higher temperatures,⁴⁹ whereas another study found that the heat effect is immediate, occurring between 1 and 6 hours of exposure and decreasing with longer lags.⁵⁰ Light exposure has a stronger entrainment on the circadian cycle than temperature,²⁹ inferring some protective qualities from heart attack mortality during summer months.

This study benefitted from a long-time series of 22 years, which enabled us to consider very hot days and nights with sufficient power. It also benefitted from use of temperature data from the MEDMI data set³² that holds hourly temperature from weather stations across England; use of MEDMI improved data accuracy by estimating a spatial and temporal mean on the environmental variables. Additionally, access to daily air quality data enabled adjustment for important covariates such as ozone and particulate matter with a diameter smaller than 10 µm. One limitation to the analysis is that our estimate of air quality was derived from only one Automatic Urban and Rural Network monitoring site in central London, which may not be representative of greater London localities. Another limitation is in the definition of nighttime hours; we chose the hours where cardiovascular events are more likely to occur, and this choice can be criticized as arbitrary, although repeated analysis using alternative nighttime hours did not alter the observed patterns. Finally, the analysis looking at the effect of exposure to nighttime temperatures might suffer from collinearity because daily mean and nighttime average temperatures maybe highly correlated. Our test for collinearity using the Generalized Variance Inflation Factor (GVIF), the recommended test for generalized linear models,⁵¹ gave an acceptable $GVIF^{(1/(2 \times df))}$ of 2 units. These results are reported in the supplementary material (eTable 1; <http://links.lww.com/EE/A0>).

In conclusion, this study shows that the increase in mortality associated with high nighttime temperatures was highest in patients susceptible to stroke; also, the heat effect of nighttime exposure may be higher than the heat effect of daytime exposure in patients with stroke and chronic ischemic diseases and in younger patients. Heat effect on mortality from most diseases was also higher during hot nights that were preceded by a hot day than in cool nights preceded by a hot day. The exact mechanisms to explain the higher risk of cardiovascular mortality from nighttime heat exposure in comparison to daytime heat exposure are not entirely clear; it is plausible that high nighttime temperatures interfere with physiological processes that regulate sleep-wake cycle and thermoregulation. These findings have some implications on the delivery of health and social care; first, improved understanding of thermal sensitivity at nighttime may inform efforts on improving sleep and thermoregulation in the most vulnerable groups. Second, the findings provide further justification of Public Health England's HHWS that is triggered by a hot day followed by a hot night.¹¹ There is scope for follow-up research to assess the spatial variation in risk from nighttime temperature exposure at a more granular level, and this would include examining how risk in mortality may be mitigated by proximity to green or blue space or may be impacted by type of dwelling.

Conflict of interest statement

The authors declares that they have no conflicts of interest with regard to the content of this report.

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