Original article

Title: The effect of temperature on mortality in rural Bangladesh − a population based time-series study.

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ABSTRACT

Background: Studies in urban cities have consistently shown evidence of increased mortality in association with hot and cold weather. However, few studies have examined temperature-mortality relationship in the rural areas of developing countries. In this study we therefore aimed to characterize the daily temperature-mortality relationships in rural Bangladesh.

Methods: A generalized linear Poisson regression model was used to regress a time-series of daily mortality for all cause and selected causes against temperature, controlling for seasonal and interannual variations, day of week and public holidays. A total of 13,270 all-cause deaths excluding external causes for residents under demographic surveillance in Matlab, Bangladesh were available between January 1994 and December 2002.

Results: There was a marked increase in all-cause deaths and deaths due to cardiovascular, respiratory and perinatal causes at low temperatures over a lag of 0−13 days. Every 1°C decrease in mean temperature was associated with a 3.2% (95% CI: 0.9, 5.5) increase in all-cause mortality. However, there was no clear heat effect on all-cause mortality for any of the lags examined.

Conclusions: This study found that daily mortality increased with low temperatures in the preceding weeks, while there was no association found between high temperatures and daily mortality in rural Bangladesh. Preventive measures during low temperatures should be considered especially for young infants.

Key words: Bangladesh, climate, mortality, rural, temperature, time-series, weather

Introduction

Studies in urban cities have consistently shown evidence of increased mortality in association with hot and cold weather[1-3]. However, few studies have examined temperature-mortality relationship in rural areas, especially in less-developed countries, despite the fact that 57% of the population in less-developed regions is rural, compared with 26% in more-developed regions.[4] One reason for this lack of research lies in the availability of data: large cities contain greater mortality sample sizes and often have a greater availability of meteorological data. Nevertheless, much of the existing literature also suggests that urban residents are more affected than rural residents by oppressive heat.[5] The so-called "urban heat island"[6], building types[7, 8] and greater social isolation[7, 8] are often cited as the main reasons for this differential. Some modeling studies of the impacts of future climate change ignore the possibility that rural residents may be vulnerable as well.[9] Further, despite the indications of adaptation/acclimatization to heat effects in warm cities, it has been suggested that urban populations in tropical climates are vulnerable to high temperatures.[3, 10, 11] Populations living in tropical rural areas may be less vulnerable to heat effects, although the patterns of mortality can be influenced by the underlying prevalence of temperature-sensitive diseases, the level of socio-economic development and population age structure. We therefore undertook time-series analyses to characterize the daily temperature-mortality relationships in rural Bangladesh.

Methods

The primary outcome for this study was daily mortality in Matlab, a field research station of the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR,B). Matlab is situated approximately 50 km southeast of Dhaka, the capital city of Bangladesh. The area is typical of many rural and riverine delta areas of Bangladesh. The most common livelihoods are rice cultivation and fishing. The ICDDR,B has maintained a registration system of birth, death and migrations in Matlab Health and Demographic Surveillance Site (HDSS) since 1966. In 2002, 142 villages with a population over 220 000 were under demographic surveillance.[12] From this demographic surveillance database, we retrieved information on date of death, age at death and cause of death during a 9-year period (January 1994 to December 2002). The field procedures and methods for detecting deaths and assessing their causes are described elsewhere in detail.[13] Briefly, the ICDDR,B field staff recorded demographic events during their monthly visits to the households and determined the causes of deaths by interviewing families of the deceased within 2−10 weeks of the deaths. The signs and symptoms preceding death were described on a separate blank form by the health workers as an unstructured verbal autopsy. Deaths were assigned using a simplified version of the Ninth Revision of the International Classification of Diseases (ICD-9). We categorized cause-of-death; cardiovascular (corresponding to ICD-9: 390−459), respiratory (ICD-9: 460−519), conditions arising during the perinatal period (ICD-9: 760−779), infectious and parasitic (ICD-9: 001−139) and other non-violent death. The age-group categories were children (0−14 years), adults (15−64 years), and elderly (≥65 years).

Meteorological data (daily maximum and minimum temperature, relative humidity and rainfall) for the 9-year period in Chandpur, located 10 kms from central Matlab, were provided by the Bangladesh Meteorological Department. Daily average temperatures were computed as the mean of the daily maximum and minimum value.

Statistical analysis

We examined the relationship between daily mortality and mean temperature using generalised linear Poisson regression models allowing for overdispersion.[14] We used mean temperature as it was a better predictor of temperature-mortality relationships when compared with either maximum or minimum temperature by the maximum likelihoods for the models. Indicator variables for the years of the study were incorporated into the model to allow for long-term trends and other variations between years. Indicator variables for the day of the week and public holidays were incorporated in the model because the patterns of mortality and behaviour influencing exposure to ambient temperatures may vary depending on the day. To account for seasonality of mortality not directly due to temperature, Fourier terms up to the sixth harmonic per year were introduced into the model. Fourier terms can capture repeated periodic (e.g. seasonal) patterns comprising a combination of pairs of sine and cosine terms (harmonics) of varying wavelength.[15] This number of harmonics was chosen as a compromise between providing adequate control for unmeasured confounders and leaving sufficient information from which to estimate temperature effects.[16] As there was no evidence that humidity was associated with daily mortality ($P>0.1$), humidity was not included in the final analysis. To allow for the autocorrelations, autoregressive terms at order one and seven were incorporated into the models.[17]

We fitted models of the average of daily mean temperature over the same day and the day before the day of death (lags 0−1) to identify heat effects which are mainly related to recent high temperatures, and over two weeks (lags 0−13) to assess cold effects, which are generally more delayed.[3] All models for immediate effects (lags 0−1) adjusted for delayed effects by incorporating the average temperature over lags of 2 to 13 days.

In the initial analyses we fitted natural cubic splines (3 df) [18] to the average mean temperature to create graphs of the temperature-mortality relationships, where mortality is plotted as smoothed functions of temperature. As the plots of the smoothed relationships of all-cause mortality suggested log-linear associations below a threshold,

we fitted the data to linear threshold models i.e., models that assume a log-linear increase in risk below a threshold and no increase in risk above the threshold to quantify the adverse effects of low temperature.[16] The same method was applied to cause or age-specific mortality other than cardiovascular and elderly deaths. For cardiovascular and elderly deaths, we quantified both heat and cold effects by using models that assume a log-linear increase in risk above a heat threshold and below a cold threshold[3] as the smoothed relationships with temperature at lag of 0-1 days suggested a reverse "J" shape with increases in mortality at temperature lower and higher than a certain temperature. For low temperature effects, mortality was assumed to increase with delay over two weeks (lags 0−13). High temperature effects were assumed to be immediate (lags 0−1). The high and low temperature terms were fitted simultaneously in the models for cardiovascular and elderly deaths. (Model equations are summarized in the supplementary online appendix). The choice of threshold was based on maximum likelihood estimation for temperature over a grid of all possible integer values within a

range indicated on the temperature-mortality graphs. Likelihood profile confidence intervals (CIs) for each threshold were calculated as the thresholds for which deviance of the model was 3.84 more than the minimum. The increase in daily mortality that were associated with a 1°C decrease in a given measure of temperature below the threshold, estimated as coefficients from the regression model, was reported as percentage change.

We then examined the lag effects in more detail, by cross-basis functions proposed by Armstrong.[16] We illustrate in Figure 5 a cross-basis model fit to the mortality data with a 3-df natural cubic spline for the temperature-mortality curves and a 4-df natural cubic spline for the coefficient-lag curves. As a sensitivity analysis, the temperature-mortality relationships were also estimated using different degrees of seasonal control (3 and 12 harmonics).

Results

A total of 13,270 deaths for all-causes excluding external causes for residents were recorded in Matlab HDSS during 1994−2002. Descriptive statistics for the causes of death by age group are displayed in Table1. Proportions of child, adult and elderly deaths were 30, 28 and 42%, respectively. More than one third of child deaths were due to perinatal causes. The most frequent diagnoses in each category of cause of death were acute but ill-defined cardiovascular diseases (n=666, 5.0%) and complications of heart disease (619, 4.7%) for cardiovascular deaths; chronic bronchitis (911, 6.9%) and pneumonia or acute lower respiratory infections (827, 6.2%) for respiratory deaths;

immaturity (small-for-date) (569, 4.3%) and prematurity (328, 2.5%) for deaths due to perinatal causes; acute watery diarrhea (422, 3.2%) and persistent diarrhea with malnutrition (399, 3.0%) for infectious and parasitic deaths; senility (1512, 11.4%) and edema with ascitis (929, 7.0%) for other deaths. The number of deaths impossible to specify was 843 (6.4%). The mean (SD) daily number of all causes of death was 4.0 (2.2). Figure 1 shows the all-cause daily mortality and the daily mean temperature averaged across the study period. A clear increase in all-cause mortality in winter can be observed.

The relationships between cause-specific daily mortality and daily mean temperature over a lag of 0−1 days adjusted for seasonal variation, between-year variation, public holidays and day of week are shown in Figure 2. There is slight increase in all-cause and respiratory deaths at low temperatures, while no heat effects are observed at high temperatures. The pattern for cardiovascular mortality shows an increase in deaths both at low temperatures and at the higher end of the temperature range. There was no clear relationship between temperature and deaths due to perinatal causes, infectious diseases, or other causes.

The relationships between cause-specific daily mortality and daily mean temperature over a lag of 0−13 days are shown in Figure 3. There is a marked increase in all-cause deaths, and deaths due to respiratory and perinatal causes at low temperatures. The relationship between infectious disease mortality and temperature was linear positive.

The relationships between all-cause daily mortality and daily mean temperature over a lag of 0−1 and 0−13 days by age group are shown in Figure 4. For the shorter lag period (0−1), there is no clear relationship in children, while there is a marked increase

in low temperature-related deaths in adults. There is an increase in elderly deaths at the higher end of the temperature range. For the longer lag period (0−13), there is increase in mortality at low temperatures for children and elderly. Increasing the degrees of freedom (4-6) for natural cubic splines did not change the patterns to any great extent (results not shown).

For the linear model, all-cause deaths increased by 3.2% (95% CI: 0.9, 5.5) for every 1°C decrease in temperature (Table 2). The effect of cold days was strongest for deaths due to perinatal causes, giving an increase of 29.6% (95% CI: 12.9, 48.7) below the threshold of 21°C (95% CI: 20, 27). The risk for deaths due to infectious diseases steeply increased with increase in average temperature over lags 0-13 days although the estimate was quite uncertain. Analyses by age group showed that the cold effect was strong in children, especially for infants (under 1 year), giving an increase of 20.7% (95% CI: 10.1, 32.3) below the threshold of 21°C (95% CI: 19, 27). The very large heat effects on cardiovascular and elderly deaths in part reflect the fact that the corresponding thresholds were very close to the edge of the observed temperature distributions. Varying the level of seasonal adjustment had little effect on the results: model for cold effect on all-cause deaths with three harmonics (3.8% (95% CI: 1.6, 6.1) increase); model with 12 harmonics (3.2% (95% CI: 1.0, 5.5) increase).

The overall relationships of all-cause mortality against lag and temperature are shown in Figure 5. The cold effect is most prominent at lags 3−4, and remained positive up to around lag 10 with no sign of mortality displacement at later lags. There was no clear heat effect on all-cause mortality at any lags.

Discussion

This study shows a marked increase in all-cause deaths, and deaths due to cardiovascular, respiratory and perinatal causes at low temperatures, while there were no clear heat effects observed in all-cause deaths in the rural Bangladeshi population.

Cold effects were very evident in our study, with a 3.2% (95% CI: 0.9, 5.5) increase in all-cause mortality for every 1°C decrease in temperature over a lag of 0−13 days. This is comparable with those observed in a study conducted in urban cities of low-latitude countries including Delhi, Monterrey, Mexico Coty, Bangkok and Sao Paulo, i.e., a 2.5-6.9% increase in all-cause mortality for every 1°C decrease in temperature below the threshold;[3] and stronger than the cold effects observed in mid- to high-latitude cities like Sofia (0.7%) and London (1.4%).[19] In England and Wales, risk in cold-related deaths was particularly high in the rural populations of deprived areas.[2]

A new finding of the present study is that deaths due to perinatal causes steeply increased with low temperatures. This might have been due to an increase in neonatal hypothermia with low ambient temperatures. Lower ambient temperature increases the oxygen consumption of infants with consequent heat production,[20] whereas heat loss usually exceeds heat production and body temperature will fall unless appropriate measures are initiated.[21] Laptook *et al.* reported that the odds of death increased by 28% for every 1°C decrease in the admission body temperature of low birth weight infants.[22] Hypothermia has been shown to be an important risk factor for neonatal death even in tropical countries.[23-25] The high prevalence of low birth weight in Bangladesh[26] may be

related to high vulnerability to low ambient temperature.

Only a few studies have examined the relationship between hot temperature and daily mortality in a rural area. Hajat *et al*. observed that heat-related deaths in rural settings were fewer than in urban areas in England and Wales by demonstrating a time-series analysis.[2] All cause mortality following a July 1980 heat wave increased by 57 and 64% in the major metropolitan areas of St. Louis and Kansas City, respectively, but only by 10% in predominantly rural areas in other parts of Missouri.[27] In contrast, the level of urbanisation was not found to be a significant predictor for percentage increase in heat-related mortality in Ohio.[28]

There is also limited evidence for heat-related mortality in tropical and sub-tropical regions. For example, in Delhi, where climate and cause of deaths are similar to those of Bangladesh, a clear heat effect was observed in children, adult and the elderly.[11] The evidence for heat-related deaths was also shown in Monterrey, Bangkok, Salvador and Sao Paulo.[3] These studies were, however, conducted in urban cities and therefore may not pertain to rural areas.

The lack of heat effects on daily mortality in rural areas may be partly due to the absence of the "heat-island effect" in rural environments. Temperatures are usually higher and heat is more efficiently retained throughout the night-time in urban centres than in surrounding non-urban areas.[5] Thus, inhabitants of rural areas often obtain some relief from thermal stress during nocturnal hours even during periods of hot weather.[29] In Matlab, large number of water bodies, trees, greenery fields and lower population density may make the relation weak. Another possible explanation is

acclimatization/adaptation to hot weather in tropical conditions; communities naturally adapt − physiologically, culturally and behaviourally − to living in warmer climates. Some evidence of acclimatization has been provided by previous studies. For example, minimum mortality temperature, defined as the temperature of the lowest temperature-associated mortality observed in a city, was higher in warmer southern cities than in cooler northern cities in the United States.[30] In 50 US cities, heat effects were found to be greater in cities with lower temperatures during warmer months.[31] Alternatively, lower proportions of elderly and cardiovascular deaths in this study population than in high-income urban cities may have resulted in the lack of heat effects on all-cause mortality; elderly and cardiovascular deaths have been generally sensitive to high temperatures in previous studies in urban cities,[1] as was observed in this study.

In previous studies, cold effects on daily mortality, which are generally more delayed than heat effects, were identified by fitting models of the average of daily temperature over the previous 2 to 3 weeks.[3, 10, 19] However, a positive relationship between infectious disease mortality and temperature at longer lag periods, which was observed in this study, suggests that the estimation of cold effects on total mortality using temperature at longer lag periods should be done with caution when performed in countries with high infectious disease mortality; in such cases, the positive relationship of temperature with infectious disease mortality can be canceled out with the negative relationship of temperature with cardiovascular and respiratory mortality.

Studies of high-income urban populations have shown reductions in mortality at lags more than a few days or weeks after a hot day,[11, 19] while the mortality displacement is

less obvious in the cities in less developed countries.[11] In contrast, the effect of cold temperatures on mortality is persistent, and there is no clear evidence of short-term mortality displacement.[32] Similarly, there was no evidence of short-term mortality displacement by lag structure analysis in this study.

In this study, information on air pollution and influenza epidemic, which could confound the relationship between temperature and daily mortality,[33] were not available. However, there is little traffic in Matlab, and the air pollution level is likely to be low. In addition, there were no reports of the occurrence of an influenza epidemic in Matlab during the study period. Thus, the impact of not controlling for these factors is likely to be small.

Potential misclassification of cause of death as defined by verbal autopsy may be another concern. The cause of death data were assessed by Fauveau et al.[13]; 35% of the neonatal deaths and 22% of the child deaths older than 1 year of age were disagreed with the diagnosis of independent physicians. However, as misclassification of cause of death is not likely to vary quickly with time, there is no reason to believe that this would obscure the short-term dependence of daily mortality on temperature.

In this study we analyzed five different causes of death and three age groups. Stratified analysis by causes of death and age groups should have provided more information on the temperature effect on each stratum, however, due to the limitation of statistical power, we have not planned to conduct stratified analysis by age group and specific cause of death.

The results of this study should not be simply interpreted to mean that expected increases in temperature with future climate change would not pose adverse effects on

mortality in tropical rural areas. The short-term associations reported here cannot be directly extrapolated to changes in climate over decades. In addition, the patterns of mortality we observed are influenced not just by the temperature distributions but also by social and demographic factors – the level of socioeconomic development and population age structure. While it remains uncertain how increases in hot temperatures could affect heat-related mortality, the results of this study suggest that cold-related mortality would likely decrease with climate change in rural Bangladesh.

In conclusion, this study found evidence that daily mortality increases with low temperatures in the preceding weeks, while there was no association with high temperatures in rural Bangladesh. Preventive measures during low temperatures should be considered especially for young infants who might reasonably have expected decades of future life had it not been for fatal outcome of the cold-related illness. Although not conclusive as a single result, this study identifies a relevant area for future research regarding the potential difference of the impact of climate change between rural and urban populations.

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Table 1. Distribution of causes of deaths (excluding external causes) by age group in Matlab HDSS: 1994−2002

	Child (0-14 years)		Adult (15-64 years)		Elderly (65+ years)		Total	
	n	(%)	n	(%)	n	(%)	n	(%)
Cardiovascular	10	(0.3)	513	(13.7)	943	(16.9)	1,466	(11.1)
Respiratory	928	(23.5)	409	(10.9)	653	(11.7)	1,990	(15.0)
Perinatal	1,470	(37.3)	0	(0.0)	0	(0.0)	1,470	(11.1)
Infectious	595	(15.1)	520	(13.8)	629	(11.3)	1,744	(13.1)
Other	942	(23.9)	2,316	(61.6)	3,342	(60.0)	6,600	(49.7)
Total	3,945	(100.0)	3,758	(100.0)	5,567	(100.0)	13,270	(100.0)

Table 2. Thresholds and slopes of temperature-mortality relationships by selected causes and age-groups (1994-2002: Matlab HDSS)

	Cold threshold (t_l °C) (95% CI)	% increase per °C decrease below cold threshold (95% CI)[a]	Heat threshold (t_h °C) (95% CI)	% increase per °C increase above heat threshold (95% CI)[a]
All cause	-[b]	3.2 (0.9, 5.5)	-	-
Cardiovascular	30 (24, .)[c]	9.9 (2.9, 17.4)	30 (29, 32)	62.9 (23.2, 115.2)
Respiratory	25 (22, 27)	17.5 (8.1, 27.6)	-	-
Perinatal	21 (20, 27)	29.6 (12.9, 48.7)	-	-
Infectious	-	-	-[b]	83.4 (0.2, 235.8)[d]
Other	-[b]	3.3 (0.2, 6.6)	-	-
Children	21 (17, 30)	11.1 (2.4, 20.7)	-	-
Adult	-[b]	1.3 (-2.7, 5.5)	-	-
Elderly	30 (20, .)[c]	5.3 (1.8, 8.9)	31 (28, .)[c]	108.1 (32.3, 227.1)

[a]Full model adjusted for seasonal and interannual variations, day of week and public holidays.
[d]No threshold was identified.
[c]CI extends to limit of observed temperature range.
[d]The heat effect on infectious diseases was quantified with temperature at lag of 0-13 days.

FIGURE LEGENDS

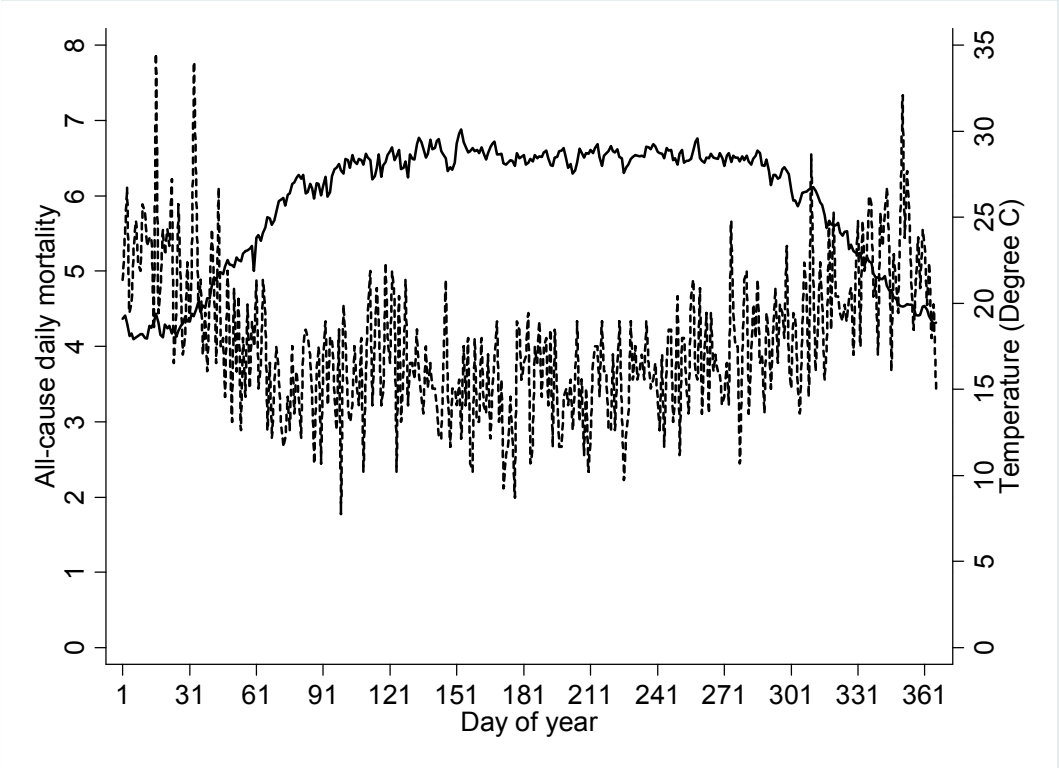

Figure 1. Seasonal distribution of all-cause daily mortality (dashed line) and daily mean temperature (line) in Matlab, Bangladesh, 1994−2002.

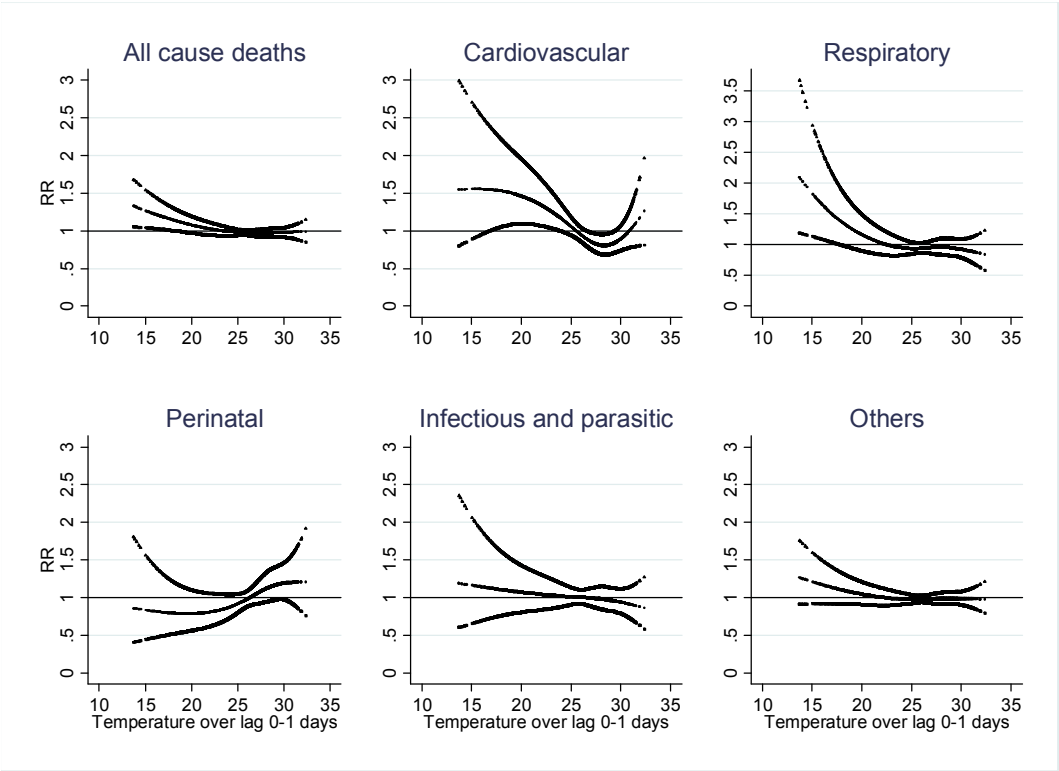

Figure 2. Relationship between all cause and cause-specific daily mortality and daily mean temperature over a lag of 0−1 days (shown as a 3 d.f. natural cubic spline) adjusted for seasonal variation, between-year variation, public holidays and day of week. RR represents the relative risk of daily mortality (scaled against the mean daily mortality). The centre line in each graph shows the estimated spline curve and the upper and lower lines represent the 95 % confidence limits.

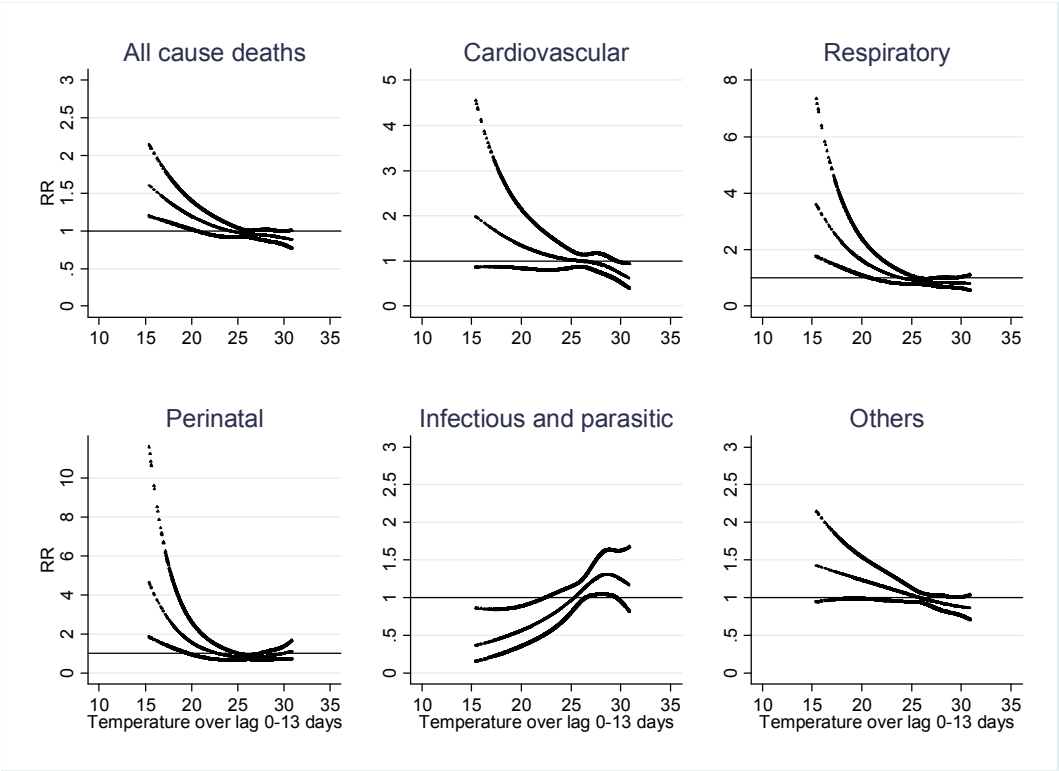

Figure 3. Relationship between all cause and cause-specific daily mortality and daily mean temperature over a lag of 0−13 days (shown as a 3 d.f. natural cubic spline) adjusted for seasonal variation, between-year variation, public holidays and day of week. RR represents the relative risk of daily mortality (scaled against the mean daily mortality). The centre line in each graph shows the estimated spline curve and the upper and lower lines represent the 95 % confidence limits.

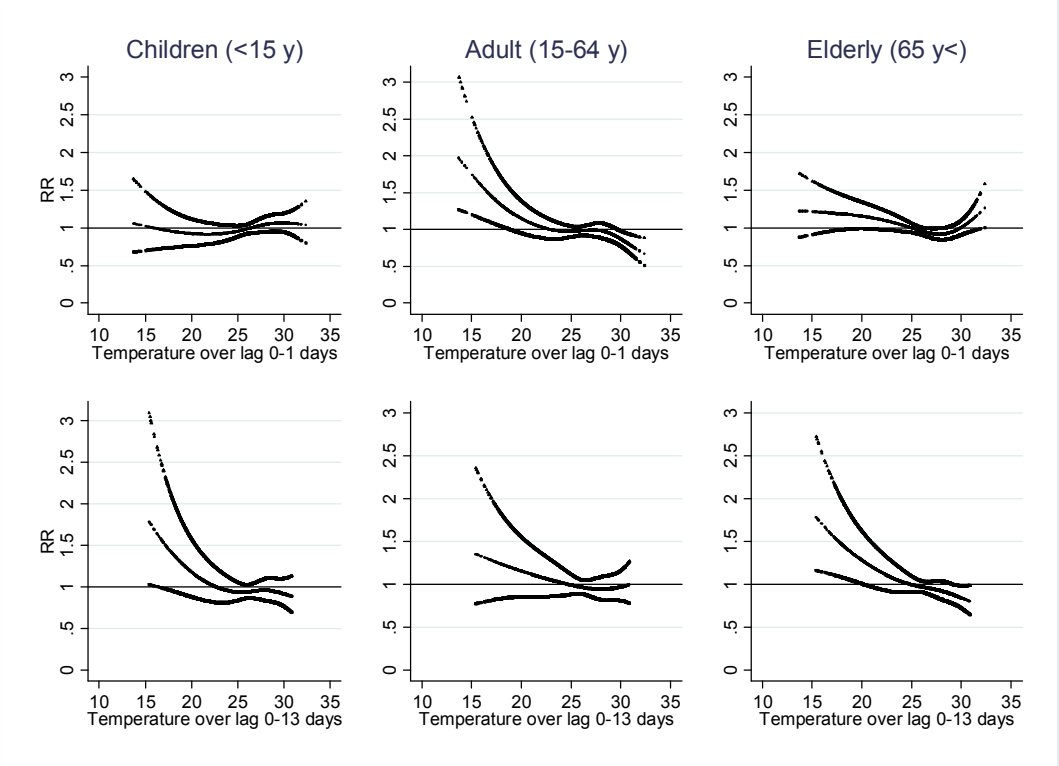

Figure 4. Relationship between all-cause daily mortality and daily mean temperature over a lag of 0−1 (upper row) and 0−13 (lower row) days by age group (shown as a 3 d.f. natural cubic spline) adjusted for seasonal variation, between-year variation, public holidays and day of week. RR represents the relative risk of daily mortality (scaled against the mean daily mortality). The centre line in each graph shows the estimated spline curve and the upper and lower lines represent the 95 % confidence limits.

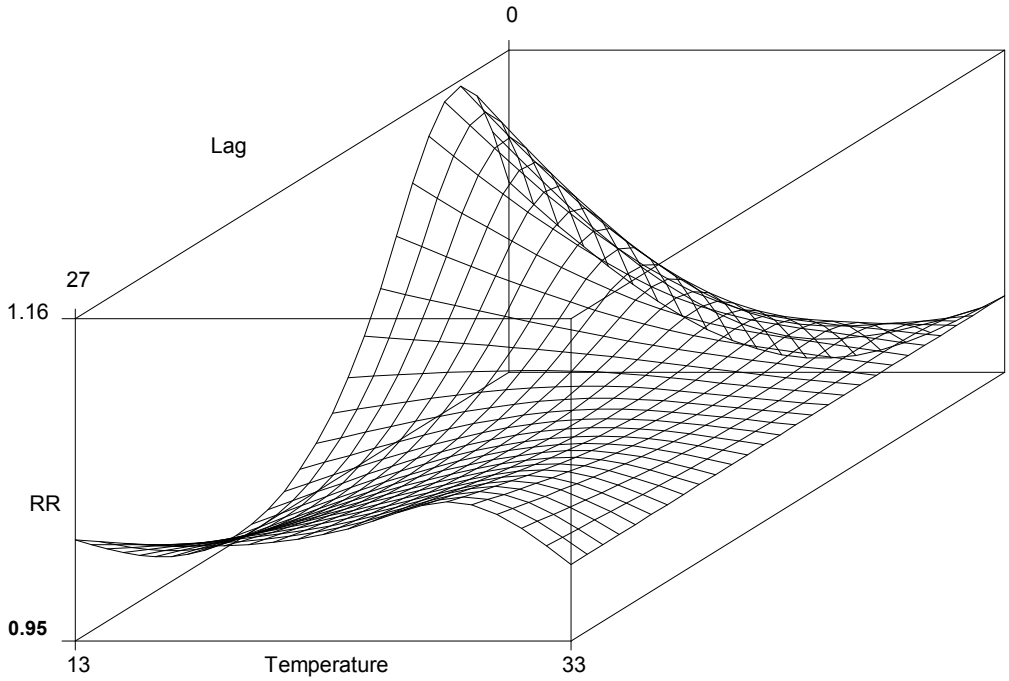

Figure 5. Relative risk of mortality by temperature (ºC) and lag using cross-basis smoothing by natural cubic splines of 3 df for temperature-mortality curves and 4 df for coefficient-lag curves.