The effect of atmospheric thermal conditions and urban thermal pollution on all-cause and cardiovascular mortality in Bangladesh

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Abstract

This study assessed the effect of temperature and thermal atmospheric conditions on all-cause and cardiovascular mortality in Bangladesh. In particular, we investigated differences in the human response to heat between rural and urban areas. Daily death counts were analysed using Poisson generalized models (GAMs), adjusting for trend, season, year, week and day of the week. Breakpoint models (hockey-stick-models) were applied in order to determine the percentage increase in mortality above and below a threshold (equivalent) temperature. Generally, a '*V*' shaped (equivalent) temperature-mortality curve with increasing mortality at low and high temperatures was observed. In particular, urban areas suffered from heat-related mortality with a steep increase in mortality above a specific threshold. This adverse heat effect may well increase with ongoing urbanisation processes and the intensification of the urban heat island due to the densification of building structures. Moreover, rising temperatures as a consequence of climate change could aggravate thermal stress.

1. Introduction

Several studies have investigated the association between temperature and human mortality. Hyperthermia and hypothermia are generally associated with all-cause and cardio-respiratory morbidity. In the majority of cases *'U'* or *'V'* -shaped temperature-mortality curves with increasing mortality levels at high and low temperatures was displayed (Kunst et al., 1993; Basu and Samet, 2002; McMichael et al., 2008; Basu, 2009). Moreover, there is evidence that the effect of temperature is influenced by non-atmospheric conditions. Apart from the prevailing burden of disease (e.g. burden of cardiovascular vs. infectious disease), different research outcomes highlighted that not only environmental but socio-economic and socio-demographic variables serve to modify the effects of temperature. Different cities in the United States exhibited differences in the relationship between heat and mortality with stronger heat effects in cities with a milder climate and higher population density (Medina-Ramón and Schwartz, 2007). Another study showed that the strongest relationship between heat and mortality in Southern Ontario occurred in cities with relatively high levels of urbanisation and high costs of living (Smoyer et al., 2000). A comparative study found that extent of short-term mortality displacement was high in London but lower in Delhi, where infectious and childhood mortality still predominate (Hajat et al., 2005). Klinenberg (2002) noted that the urban poor and those with less-developed social networks were most at risk of death during the Chicago heat wave of 1995, whilst the African-American population also displayed a higher risk of heat-related mortality (Kaiser et al., 2007).

A process to be found throughout the world and associated with profound changes in both the physical and social environment, urbanization is especially prevalent in developing countries. According to United Nations projections, the rate of urban population change from 2010 to 2025 is set to register 2.1% in less developed and 3.8% in the least developed countries, compared with 0.6% in developed countries (3.1% projected growth rate for Bangladesh) (UN, 2008; 2010). Urban populations appear to be more vulnerable to the effects of heat. Differences in socioeconomic conditions, lifestyles and pre-existing health conditions between rural and urban areas might be possible explanations for this phenomenon. Furthermore, the anthropogenic modification of the urban mesoclimate, the so-called urban heat island (UHI) is likely to increase thermal stress and have an adverse effect on human health.

The majority of research on thermal effects has been conducted in industrialized countries located in the mid-latitudes, whereas little is known about the temperature-mortality relationship in less developed and especially in tropical countries (Hashizume et al., 2007; McMichael, et al., 2008). Considering the modifying effect of non-atmospheric variables, the insights gained from studies conducted in temperate climate zones cannot be applied directly to tropical climates. Moreover, many recent studies focused on the impact of temperature with several controlling for humidity. However, in addition to temperature, the human heat budget is affected by humidity, air movement and short and long wave radiation fluxes (Steadman, 1979; Fiala et al., 1999; Höppe, 1999; Fiala et al., 2001; Jendritzky et al., 2007). In this context, reducing thermal effects to temperature effects fails to address the complexity of the question.

This study aims at reducing the research gap for tropical developing countries. We assessed the impact of thermal conditions on urban and rural mortality, considering all physiologically relevant meteorological parameters. Special focus was placed on thermal impacts in urban areas and the additional effect of urban excess temperatures. We determined the threshold values above and below which a rise in mortality occurred and the percentage increase beyond these thresholds. Furthermore, we assessed the influence of thermal conditions on previous days (lag periods) and analysed the suitability of different atmospheric indices as predictors of mortality.

2. Material and methods

2.1. Meteorological data

Meteorological data, comprising 3-hourly values of temperature, humidity, wind speed and cloud coverage was collected by the Bangladesh Meteorological Department for 22 stations across Bangladesh. Daily mean and extreme values were calculated, as far as the measurements were complete for a given day. If three-quarters of the daily values for a month were available, we calculated monthly values to perform the bioclimate and heat island assessment (approximately 17% of the data were missing). The magnitude of the urban heat island was calculated as the differences in the monthly average values between an urban station in Dhaka and a rural reference station in Mymensingh, located approximately 120 km from Dhaka. To conduct the regression analysis, the missing daily values of temperature or thermo-physiological indices were replaced by linear interpolation. As meteorological stations were highly correlated and the differences between stations were only minor, Bangladesh was taken as representing a single climatic unit. Regional meteorological variations were not considered and a single average daily mean value was calculated which was taken as representing macroclimatic conditions. Such aggregation helped to increase the statistical power and significance of the regression analysis. Nevertheless, climatic specifications on a meso- or micro scale could not be covered by such an average value.

2.2. Thermo-physiological modelling

Different thermo-physiological indices (TPIs) were calculated based on the 3-hourly values. These indices can be described as equivalent temperatures reflecting the atmospheric effect on the human energy balance accounting for different physical parameters of the surrounding atmosphere (humidity, wind movement, radiation fluxes). Human thermoregulation is basically determined by metabolic heat production and energy transfer with the surrounding environment. The human organism seeks to maintain a core body temperature of 37°C. Deviation from this temperature results in the organism triggering various counteractions. Following hypothermia, an enhanced metabolic rate increases internal heat production and mechanical heat production is effected by shivering; furthermore, heat transfer is reduced by vasoconstriction (Fiala, et al., 2001; Parsons, 2003). In the case of hyperthermia, heat conductivity to the body periphery is increased by vasodilatation and augmented disposal through an increase in the sensible and latent heat flux (e.g. sweating) (Fiala, et al., 2001; Parsons, 2003).

The magnitude and efficiency of energy exchange between a body and its surroundings is determined by meteorological conditions; primarily through the ambient temperature but humidity, air movement and long or short wave radiation also exert influence. Thermo-physiological models are used to model the complex interactions between external energy gain, physiological reactions of the human organism and body-environment energy exchange (Steadman, 1979; Höppe, 1999; Fiala, et al., 2001; Parsons, 2003). The output variables of these models are equivalent temperatures which are temperatures resulting in the same energy gain or loss like under a reference atmospheric environment. This paper uses the terms thermo-physiological index and equivalent temperature synonymously.

The Heat Index (HI), originally developed by Steadman (Steadman, 1979) and adapted by the US National Weather Service is an index combining air temperature and humidity. HI is defined for temperatures above 26°C and a relative humidity above 40%. It is an index assessing heat (not cold) by accounting for the diminished latent energy release following higher atmospheric water vapour pressure (reference environment: temperature 25°C; humidity 50%). Conversely, the Wind Chill Index (WCI), combining air temperature and wind speed, is an index assessing cold by accounting for increased energy disposal due to air movement. It is defined for temperatures below 10°C and wind speeds above 4.8 km/h (reference environment: wind speed 1.34 m/s) (Steadman, 1971). Our analysis combined the HI and WCI in order to assess both, cold and heat. Indices were calculated whenever threshold criteria were met; whenever no index was calculated, the measured temperature was retained.

The physiological equivalent temperature (PET) is based on the Munich Energy-balance Model for Individuals. PET is defined as the air temperature at which, in a typical indoor setting (without wind and solar radiation), the heat budget of the human body is balanced with the same core and skin temperature as under the complex outdoor conditions to be assessed (reference environment: temperature 20°C, humidity 50%) (Höppe, 1999). PET requires the input parameters temperature, humidity, mean radiation temperature and wind speed.

The universal thermal climate index (UTCI) is based on the Fiala model; a thermo-physiological model which has been extensively validated using experimental data from numerous groups (Jendritzky, et al., 2007). The model accounts for heat transfer occurring inside the human body and at its surface and additionally, simulates responses of the human thermoregulatory system (Fiala, et al., 1999; Fiala, et al., 2001). A reference environment with 50% relative humidity, still air and a radiant temperature equalling air temperature is defined. The input variables are temperature, humidity, wind speed and mean radiation temperature. To determine PET and UTCI, the input variable mean radiant temperature (uniform temperature of a surrounding surface which results in the same radiation energy gain on a human body as the prevailing radiation fluxes) is modelled as a function of cloud coverage and the other input parameters using RayMan (Version 1.2) (Matzarakis et al., 2007). All models contain assumptions about the human body mass and height, clothing and the amount of physical activity undertaken.

2.3. Mortality Data

Mortality data from 2003 to 2007 based on the Vital Sample Registration System (VSRS) was provided by the Bangladesh Bureau of Statistics (BBS). The VSRS comprises 1 000 primary sample units (PSUs), from which 640 PSUs are located in rural areas, 280 in urban areas and 80 in the statistical metropolitan area. Approximately one million individuals, living in 206 552 households, fall under the scope of this monitoring program. The data is initially collected by a locally-recruited recorder. Further, data is collected by a group of officials from the BBS on a quarterly basis. Both data sets are matched by pre-designed matching criteria. For further information on the VSRS please see (BBS, 2008). Accidental deaths and maternity related deaths were excluded for the purposes of this study. Likewise, data from metropolitan areas was excluded. In total, we analyzed 25 758 deaths, accumulated over 5 years from 2003 to 2007. Major causes of death included respiratory diseases, cardiovascular diseases and infectious diseases, which accounted for almost half of all deaths. Diarrhoeal disease and vector-borne diseases did not attribute more than six percent of all deaths. The percentage distribution of causes of death varied between rural and urban areas. The Chi-square test was applied to determine whether the probability of dying from a particular disease was significantly different in urban compared to rural areas. Significant differences ($p<0.001$, significance level of 0.05) between rural and urban areas were found for mortality due to respiratory disease (19.4% vs. 16.1%), cardiovascular disease (11.5% vs. 20.8%), diarrhoeal disease (3.9% vs. 2.7%), infectious disease (13.9% vs. 9.5%), cancer (6.1% vs. 7.5%), and vector-borne diseases (1.5% vs. 0.7%). Significant differences also existed in the group of causes of death that were not specifically classified (41.5% vs. 40.9%). No significant differences could be found between malnutrition as cause of death (2.0% vs. 1.8%). An overview table of causes of death in rural and urban areas containing the exact number and percentage in each category is provided in the supplementary material.

2.4. Statistical methods

The association between daily all-cause or cardiovascular death counts and ambient temperature, HIWCI, PET or UTCI was analyzed using Poisson generalized additive models (GAMs). The R (Version 2.11.0) package 'mgcv' was used for model fitting. The degree of smoothness of model terms is estimated as part of fitting by finding a trade-off between 'wiggliness' and badness of fit. The models are fit by penalized likelihood maximization, in which the model likelihood is modified by the addition of a penalty for each smooth function (Wood, 2006). The smoothing parameter estimation was solved using Un-Biased Risk Estimator (UBRE) criterion. Smoothing parameters are chosen to minimize the UBRE scores for the model. A Bayesian approach to variance estimation was employed to calculate the confidence interval (Wood, 2006). The models were adjusted for trend, season, year, week and day of the week in order to allow for long- and short-term trends and other variations. After incorporation of these confounder variables, plots of partial autocorrelation showed no autocorrelation and an autocorrelation term was therefore not incorporated into the final models. Humidity was also not integrated into the models, as humidity and temperature are highly correlated and result in multicollinearity problems. Instead, humidity and other meteorological variables were accounted for by thermo-physiological indices. Models were fitted integrating (equivalent) temperatures of the actual and the previous day (lag 0-1) in order to identify heat and cold effects caused by recent thermal conditions. To account for more delayed thermal effects, models incorporating the average of daily (equivalent) temperatures and the recent six days (lag 0-6) and the recent 13 days (lag 0-13) were fitted.

Breakpoint models (hockey stick models) were applied to quantify the effect of cold and heat. These are regression models assuming a piecewise linear relationship between the response and the explanatory variable (Muggeo, 2008). The lines are connected at unknown values called breakpoints which in this study represent the temperatures above and below which the temperature–mortality relationship changes. The fitting of the breakpoint regression models was carried out with R (Version 2.11.0) and the R package 'segmented'. Based on a generalized linear regression model (GLM), the 'segmented' package tries to estimate a new model having broken-line relationships for an (equivalent) temperature. A GLM incorporating all variables used in the GAMs was fitted (R package ‘mgcv’ and ‘splines’) prior to the fitting of the breakpoint model. Initial values for the breakpoints were specified over a range of possible integer values as indicated by the (equivalent) temperature–mortality plots. Where no breakpoint was evident in the (equivalent) temperature-mortality plots, the slope was determined by a GLM.

3. Results

3.1. The urban bioclimate in Bangladesh

Bangladesh is located between 21 - 26th degrees northern latitude. The Köppen system classifies it as a tropical winter dry (monsoon) climate, characterized by constant high temperatures with average temperatures of 18°C or higher for twelve months of the year and a pronounced dry season. Fig. 1 shows the monthly distribution of average mean, maximum, and minimum temperatures, TPIs (HIWCI, PET, and UTCI) and precipitation in Dhaka. The average mean temperatures remained high from April to October with equivalent temperatures surpassing measured temperature by about 3 to 4 Kelvin during this period. Differences between TPIs and measured temperature were minor for the colder period (October – March) and did not exceed 1 Kelvin. The highest average maximum temperatures were measured in April and May, and the TPI peak occurred in June (HIWCI), March (PET), and April (UTCI). The average maximum values of equivalent temperatures were surpassing those of temperature throughout the year (except HIWCI), ranging between 5 and 15 Kelvin. The average minimum values peaked during the monsoon season at the points of lowest long-wave emission. The differences between average minimum temperatures and TPIs were small. PET remained constantly below temperature whilst the UTCI remained below temperature from November to March. The lowest values for all temperatures considered were observed in December and January. Heavy rainfall occurred between June and September, and thermal levels remained high throughout this season (Fig. 1).

Urban-rural differences in temperature ranged between 0.3 and 2.1 K. The most pronounced UHI was observed in March and April during the dry summer season, whereas its magnitude was reduced during the rainy (monsoon) season (Fig. 2). In addition to higher temperatures, lower specific or relative humidity and reduced wind speed was measured at the urban station (data not shown). Differences in TPIs basically followed the seasonal distribution of temperature differences, but the magnitude of equivalent temperature differences varied heavily depending on the index considered. Although the UHI has often been described as a night-time phenomenon, this could not be observed here. Urban-rural differences in minimum (night-time) temperatures were rather smaller. A more detailed analysis of bioclimate and thermal stress in Dhaka and Bangladesh has been presented elsewhere (Burkart and Endlicher, 2010).

3.2. Thermal effects on mortality

This study demonstrated a clear association between thermal conditions and mortality. Both cold and heat effects could be observed. Generally, the diverse nature of the effects exhibited considerable differences between urban and rural areas and varied with the causes of death. Fig. 3 and 4 show smoothed plots of all-cause and cardiovascular mortality plotted against the mean (equivalent) temperature for different lag periods. Differences in the shape of the temperature-mortality curve and the equivalent temperature-mortality curves were small. As with most research we analyzed the effect of mean temperatures; the decision to focus on this relationship permitted better comparability with other studies. Plots of mortality against minimum and maximum values are included in the supplemental material. For the most part, curve progressions for mean, maximum, and minimum values were quite similar.

In terms of all-cause mortality, a '*V*' shaped (equivalent) temperature-mortality relationship could be observed (Fig. 3). The increase in mortality was roughly linear above and below a breakpoint (equivalent) temperature. With decreasing temperatures (cold effect), mortality levels were augmented regardless of the lag period. Mortality increased by approximately 2 - 3% per 1°C decrease in mean (equivalent) temperature (Table 2). In rural areas, an increase in mortality with increasing (equivalent) temperatures (heat effect) was observed for a lag period of 0–1 days. The percentage increase of rural mortality for a 1°C increase in temperature amounted to approximately 8%. The heat effect observed for a lag period of 0-6 days had already subsided (approximately 1.5% increase per 1°C increase in temperature) and could no longer be detected for a lag period of 0-13 days. On the contrary, a clear increase in heat-related mortality could be observed in urban areas for all lag periods. Depending on the predictor employed, we determined a percentage increase per 1°C temperature increase of between 7 - 20% for a lag period of 0-1 days. The mortality increase was slightly lower for the longer lag periods (0-6 and 0-13 days), ranging between 2.6 and 12.5%. Equally strong heat effects were observed using minimum values as predictor, whilst the heat slope was shallower for maximum values (Table S2 and S3). Breakpoint (equivalent) temperatures were between 29 and 30°C in rural and urban areas with no considerable difference between the two areas. Threshold equivalent temperatures were slightly surpassing threshold temperatures. Again, rural and urban areas exhibited no major differences in this respect.

A heat effect on cardiovascular mortality could not be detected in rural areas. Indeed, a negative (equivalent) temperature relationship was observed over the whole range of values (Fig. 4). For the linear model, rural cardiovascular mortality increased by approximately 1.8% per 1°C decrease in mean (equivalent) temperature. Similar negative slopes were returned for the minimum (equivalent) temperature (Table S4), whilst the corresponding slope for maximum (equivalent) temperature was smaller (Table S5). As in rural areas, no heat effect was detected in urban areas for a lag period of 0-1 days. After a minimum of 4 days with continuing high temperatures, a rise in urban cardiovascular mortality could be observed. For a lag period of 0-6 days a clear heat effect, particularly for temperature and PET, was visible. This urban heat effect continued until a lag period of 0-13 days. An increase in cardiovascular mortality of 25 to 30% per1°C increase in temperature was observed, and an increase of 1°C PET produced an increase in mortality of 13.8 to 42.6 %. The rise in heat-related mortality for HIWCI and UTCI was rather moderate, ranging between 2.4 and 6.3%. Lying between 28 and 32.5°C, breakpoint (equivalent) temperatures for cardiovascular mortality were similar to those for all-cause mortality. In the case of minimum (equivalent) temperatures, an adverse heat effect on urban cardiovascular mortality was observed for a lag period of 0-1 days (Fig. S3, Table S4).

Predictive advantage

Judging by the (minimization) of the UBRE criterion, minimum values of temperature and TPIs showed a slight predictive advantage in general and in urban areas in particular. Maximum values were stronger predictors of all-cause mortality in rural areas, whilst minimum values were better predictors for cardiovascular mortality in rural areas. Minimum values were good predictors for cardiovascular mortality. Comparing the predictive power of temperature and TPIs in the 12 different confounder models, temperature produced the best results four times and HIWCI five times respectively. PET had the highest predictive advantage in two models and UTCI was strongest in one model (Table 1). Nevertheless, differences were only minor.

4. Discussion

This study observed an increase in mortality at low and high temperatures. Despite the tropical climate, a cold effect was discovered over a wide range of values for all lag periods. Considering the elevated temperatures characteristic to this region, these finding are certainly surprising. Other studies conducted in Delhi (India), Bangkok (Thailand) and Salvador (Brazil), or Matlab (a rural area in Bangladesh) found no cold effect until a lag period of 0-13 days (Hashizume, et al., 2007; McMichael, et al., 2008). The percentage increase of mortality per 1°C (equivalent) temperature recorded in this study ranged between 2 and 3% for all-cause mortality (Table 2), and 2 and 4% for cardiovascular mortality (Table 3). The slopes determined are comparable with those found for a lag-period of 0-13 days in Delhi (2.8%), Bangkok (4.1%), or Sao Paulo (2.5%) (McMichael, et al., 2008). More shallow slopes were observed for mid-latitude cities like Ljubljana (0.4%), Bucharest (0.9%), or Sofia (0.9%) (McMichael, et al., 2008).

This pronounced cold effect suggests that the present degree of adaptation or acclimatization to (relative) cold in Bangladeshi society is inadequate. Adaptation involves physiological, cultural and behavioural factors. Physiological adaptation relates to the time spent living in an area (long-tem adaptation) but also to the thermal conditions prevelant in the previous weeks and months (short-term adaptation). Cultural and behavioural strategies refer to building structures, clothing, outdoor activities etc. While aligned to the high temperatures prevailing most of the year protection against cold is small. Insufficient protection might also be due to the perception of the relatively low temperatures as comfortable. Moreover, the period of relative cold is restricted to few months of the year. This line of argumentation is supported by research showing that people living in colder climates protect themselves better against the cold than those living in moderate climates with the same outdoor temperatures (The Eurowinter Group, 1997; Donaldson et al., 2001). A low socio-economic status could well serve to aggravate the adverse effects of cold and provides a possible an explanation for the cold effect observed after a short lag period.

Although the study population appeared well adapted to hot weather, heat effects occurred above a specific threshold (equivalent) temperature. Heat effects depended on the cause of death, location (urban vs. rural), and lag period. Urban areas exhibited stronger heat effects. Although the impact on all-cause mortality was equally pronounced in rural and urban areas for the lag period of the current and previous day (lag 0-1), it quickly subsided in rural areas over longer lag periods whilst remaining high in urban areas. The mortality increase due to elevated temperature observed in this study (~7-10%) was more pronounced than the effect observed for other low-latitude cities like Delhi (3.9%), Bangkok (4.1%) or Sao Paulo (3.5%). Particularly, a tremendous increase (33.6%) in heat-related deaths was observed for cardiovascular causes in the urban areas of Bangladesh. This strong impact of heat might be due to the low socioeconomic status of the overriding majority of Bangladeshi society. Nevertheless, judging by the width of the confidence intervals, slope estimation is rather imprecise. Threshold values were comparable for rural and urban areas (Table 2, Table 3). Given that our analysis is based on synoptic measurements capturing macroclimatic conditions, this implies that the effective mesoclimatic urban threshold temperature is higher than the rural threshold temperature. This would indicate that urban populations are better adapted to heat. However, the continuing heat effect over several lag periods demonstrates the high vulnerability of urban populations.

Extreme values and particularly minimum values displayed a higher predictive advantage compared to mean values. This finding underlines the importance of night-time temperature for the regeneration of the human organism. No predictive advantage was observed for TPIs. Nevertheless, the physical mechanisms triggered by humidity, air movement or radiation are indisputable. The crucial research question is the extent to which human health outcomes are connected to or determined by the human heat balance. In addition to the effect of meteorological conditions on the prevalence of certain pathogens, biochemical reactions triggered or influenced by temperature have an effect on health outcomes. Several studies have demonstrated that changes in blood composition are influenced by temperature (van Beaumont et al., 1974 ; van Beaumont et al., 1981; Keatinge et al., 1984; Keatinge et al., 1986; Neild et al., 1994; Keatinge et al., 1997).

Exposure to cold can lead to an increase in blood and plasma viscosity, raised red blood cell, and cholesterol and fibrinogen levels. Induced haemoconcentration can result in arterial thrombosis or other cold-induced cardiovascular reflexes (Keatinge, et al., 1984; Neild, et al., 1994; Keatinge and Donaldson, 1995). Furthermore, there is evidence that cold causes physiological changes in cellular and humoral immunity or more directly, can affect the respiratory tract, for example through bronchoconstriction (Bull, 1980; Berk et al., 1987). Following exposure to heat, reduced plasma and platelet volume could be observed with increases in blood viscosity. Moreover, augmented plasma protein and cholesterol levels and higher red blood cell and platelet count were also detected (van Beaumont, et al., 1974 ; van Beaumont, et al., 1981). These changes are likely to cause coronary and cerebral thrombosis during hot weather finally resulting in cardiovascular-related death (Keatinge, et al., 1986).

These biochemical processes are accorded no consideration by (current) thermo-physiological models. However, they may well constitute tipping points in the cardiovascular system, beyond which a breakdown occurs. Nevertheless, we would argue that TPIs have the potential to improve statistical modelling and the assessment of mortality related to atmospheric thermal conditions. Indices are usually determined for a standardized individual of middle-age and average height and weight. However, those in danger of dying from heat or cold are most likely to be of an older or younger age, or to suffer from a medial condition (e.g. obesity, hypertonia, diabetes). Adapting the model setting to incorporate these factors may well improve the prediction of mortality. The significance of TPIs in investigating the health effects of cold and heat represent an important avenue of further research.

4.1. Strengths and Limitations

Few studies have explored the thermal effect on mortality in tropical countries, due to the limited data availability in these regions. This study represents a substantial contribution to a much-improved understanding of the relationship between atmospheric conditions and mortality. The analysis is based on continuous data from a sample covering Bangladesh on a nationwide level. In the context of a developing country such data availability is rather exceptional. The practice of surveying households instead of merely collating registered fatalities reduces the risk of underreporting. Moreover, the dual recording system brings quite reliable data. Most importantly, this study not only considered the effect of temperature but also the combined effect of temperature and other meteorological parameters. Nevertheless, some limitations remain. Reduced data availability made it impossible to account for regional or location-specific climatological and meteorological variations. In the absence of long-term air pollution data for Bangladesh we were unable to adjust our analysis for atmospheric pollution. A further possible limitation is the lack of information regarding the socioeconomic status of the sample population. Moreover, our study illustrated the general association between thermal conditions and human mortality without allowing conclusions about extreme events such as heat or cold waves. The nature of our data (a sample covering approximately 1% of the population) meant that we were unable to cover such excess mortalities. Nevertheless, it is highly likely that following an extreme event, mortality could increase with a steeper gradient than shown for this study.

5. Conclusions

Temperature effects are strongly pronounced in Bangladesh. The increase in mortality resulting from high or low temperature surpasses the levels observed for other low-latitude areas. We assume that socio-economic conditions are responsible for the strongly pronounced impact of weather effects. Although a cold effect occurred over a wide range of temperature values, a steep increase in heat-related mortality was observed above a particular threshold temperature. In particular, urban populations seemed to be highly vulnerable to heat effects regardless of whether an increase in mortality follows from urban excess temperatures or the higher susceptibility of urban populations to heat. This adverse heat effect may well increase with continuing urbanisation and the intensification of excess temperatures due to the densification of building structures. A climate change-induced rise in temperature could also represent an aggravating factor for which mitigation strategies are urgently required.

Capsule

Mortality in Bangladesh is strongly affected by atmospheric thermal conditions. Urban areas exhibited a particularly strong response to heat with a steep increase in excess mortality above a specific threshold temperature.

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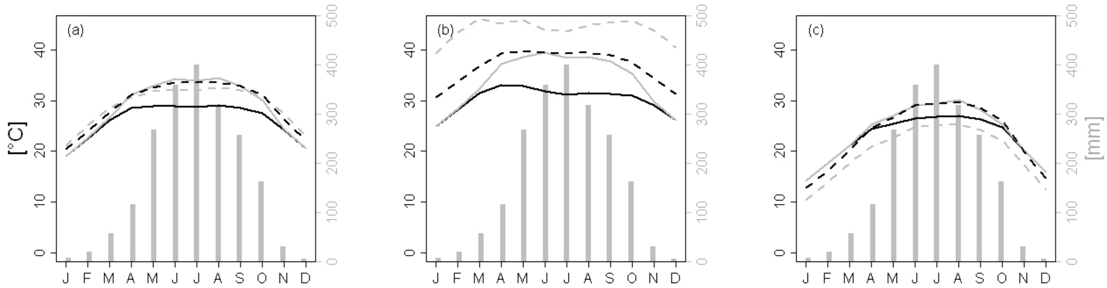

Fig. 1. Distribution of temperature (black solid line), HIWCI (gray solid line), PET (gray dashed line), and UTCI (black dashed line) of monthly average mean values (a), monthly average maximum values (b), and monthly average minimum values (c).

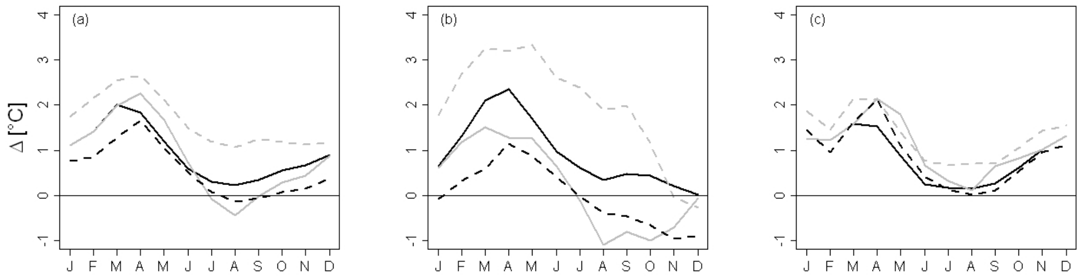

Fig.2. Seasonal distribution of urban–rural (equivalent) temperature differences between Dhaka and Mymensingh for monthly average mean values (a), monthly average maximum values (b), and monthly average minimum values (c).

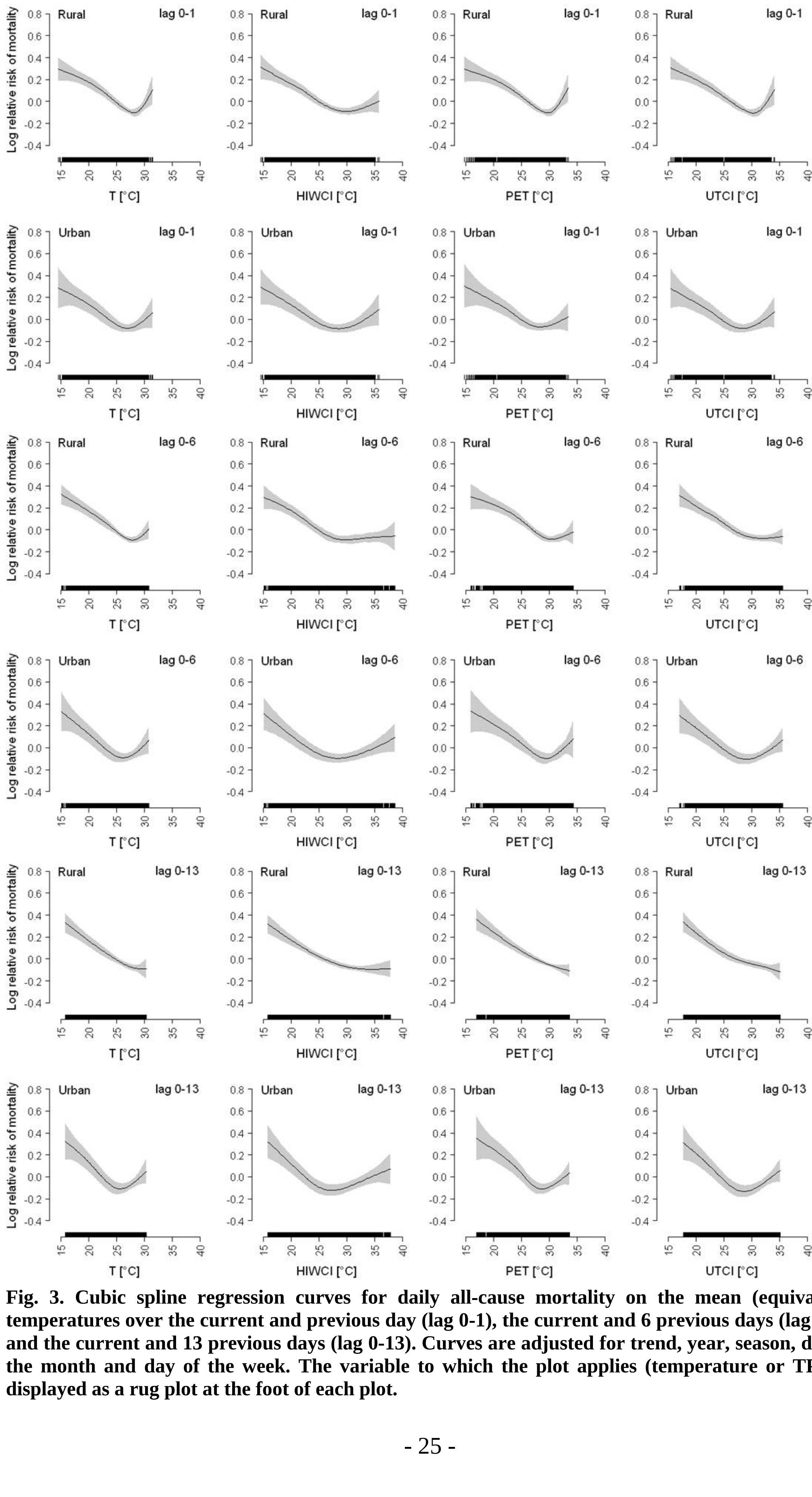

Fig. 3. Cubic spline regression curves for daily all-cause mortality on the mean (equivalent) temperatures over the current and previous day (lag 0-1), the current and 6 previous days (lag 0-6), and the current and 13 previous days (lag 0-13). Curves are adjusted for trend, year, season, day of the month and day of the week. The variable to which the plot applies (temperature or TPI) is displayed as a rug plot at the foot of each plot.

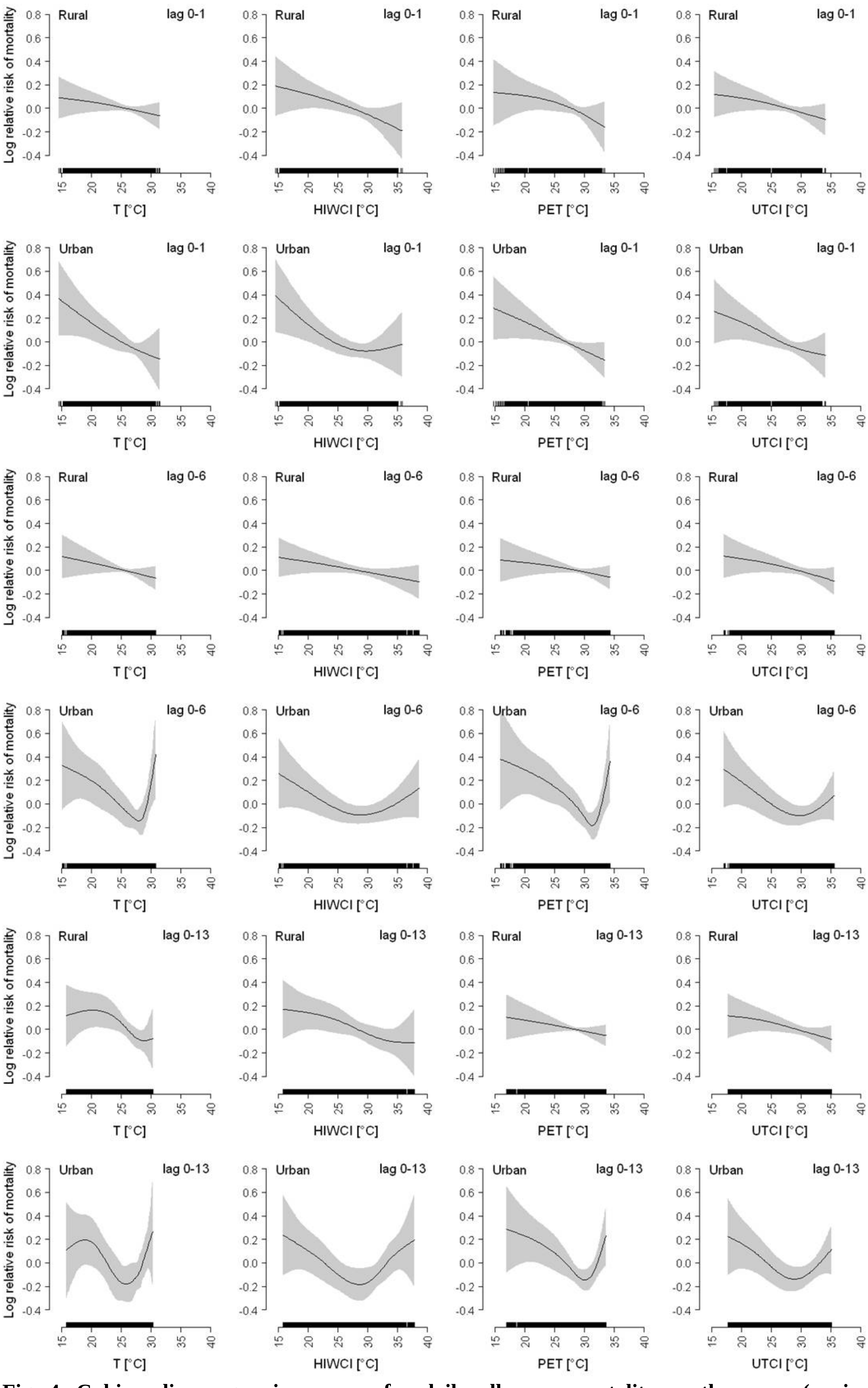

Fig. 4. Cubic spline regression curves for daily all-cause mortality on the <u>mean</u> (equivalent) temperatures over the current and previous day (lag 0-1), the current and 6 previous days (lag 0-6), and the current and 13 previous days (lag 0-13). Curves are adjusted for trend, year, season, day of the month and day of the week. The variable to which the plot applies (temperature or TPI) is displayed as a rug plot at the foot of each plot.

Table 1: UBRE scores for different predictors (mean, maximum and minimum for temperature (T), Heat Index/Wind Chill Index (HIWCI), Physiological Equivalent Temperature (PET), and Universal Thermal Climate Index (UTCI))

			Rural			Urban		
			lag 0-1	lag 0-6	lag 0-13	lag 0-1	lag 0-6	lag 0-13
All-cause	**T**							
		Mean	0.23308$^{*)}$	0.23531	0.24152	0.24999	0.24647	0.24460
		Max	0.23863	**0.23216**$^{**)}$	**0.23723**$^{**)}$	0.25315	0.25075	0.24734
		Min	0.24102	0.24141	0.24445	0.24884$^{*)}$	0.24590$^{*)}$	0.24456$^{*)}$
	HIWCI							
		Mean	0.23423	0.23730	0.24169	0.24928	0.24549	0.24398
		Max	0.23316$^{*)}$	0.23411$^{*)}$	0.23990$^{*)}$	0.25054	0.24830	0.24548
		Min	0.24128	0.24215	0.24325	**0.24762**$^{**)}$	**0.24397**$^{**)}$	**0.24324**$^{**)}$
	PET							
		Mean	**0.23269**$^{**)}$	0.23750$^{*)}$	0.24176$^{*)}$	0.25243	0.25085$^{*)}$	0.24583
		Max	0.26222	0.25706	0.25136	0.26191	0.25924	0.25368
		Min	0.24368	0.24226	0.24569	0.24906$^{*)}$	0.24555	0.24424$^{*)}$
	UTCI							
		Mean	0.23428	0.23690	0.24265	0.25100$^{*)}$	0.24702$^{*)}$	0.24378$^{*)}$
		Max	0.23347$^{*)}$	0.23266$^{*)}$	0.23826$^{*)}$	0.25396	0.25027	0.24628
		Min	0.24435	0.24364	0.24474	0.25226	0.24830	0.24479
CVD	**T**							
		Mean	0.22396	0.22391$^{*)}$	0.22509	0.18197	0.17922	0.18210
		Max	0.22640	0.22707	0.22608	0.18183	0.17953	**0.17763**$^{**)}$
		Min	0.22298$^{*)}$	0.22534	**0.22316**$^{**)}$	0.17999$^{*)}$	0.17902$^{*)}$	0.18026
	HIWCI							
		Mean	0.22458	0.22355$^{*)}$	0.22564	0.18172	0.17986	0.18116
		Max	0.22509	0.22367	0.22392$^{*)}$	0.18248	0.18186	0.18150
		Min	0.22438$^{*)}$	0.22531	0.22527	**0.17968**$^{**)}$	**0.17813**$^{**)}$	0.17937$^{*)}$
	PET							
		Mean	0.22512	0.22396	0.22403$^{*)}$	0.18041	0.17911$^{*)}$	0.18024$^{*)}$
		Max	0.22605	0.22640	0.22660	0.17988$^{*)}$	0.18026	0.18188
		Min	0.22270$^{*)}$	**0.22323**$^{**)}$	0.22476	0.18020	0.17926	0.18058
	UTCI							
		Mean	0.22355	0.22343	0.22347$^{*)}$	0.18088	0.18007	0.17974
		Max	0.22407	0.22379	0.22402	0.18046	0.17922$^{*)}$	0.17886$^{*)}$
		Min	**0.22245**$^{**)}$	0.22343$^{*)}$	0.22510	0.18023$^{*)}$	0.18065	0.18137

***) best predictor comparing mean, minimum and maximum values of temperature and TPIs**
****) best predictor comparing temperature, HIWCI, PET, and UTCI**

Table 2: Thresholds and slopes of the mean (equivalent) temperature-all-cause mortality relationship in rural and urban areas for different lag periods

	Rural			**Urban**		
	Threshold (equivalent) temperature [°C]	Percentage increase in mortality for each °C decrease in (equivalent) temperature below threshold (95% CI)	Percentage increase in mortality for each °C increase in (equivalent) temperature above threshold (95% CI)	Threshold (equivalent) temperature [°C]	Percentage increase in mortality for each °C decrease in (equivalent) temperature below threshold (95% CI)	Percentage increase in mortality for each °C increase in (equivalent) temperature above threshold (95% CI)
T						
Lag 0-1	29.3 (+/-0.3)	3.0 (+/-0.7)	8.2 (+/-6.9)	28.9 (+/-0.5)	3.2 (+/-1.2)	7.0 (+/-14.8)
Lag 0-6	30.3 (+/-0.2)	2.9 (+/-0.8)	1.6 (+/-3.8)	28.8 (+/-0.4)	3.5 (+/-1.3)	11.1 (+/-12.9)
Lag 0-13		2.5 (+/-0.8)		28.5 (+/-0.7)	3.6 (+/-1.4)	10.6 (+/-12.1)
HIWCI						
Lag 0-1	30.5 (+/-0.9)	2.9 (+/-0.7)	8.2 (+/-6.9)	31.8 (+/-0.7)	2.6 (+/-1.1)	7.9 (+/-6.6)
Lag 0-6	31.4 (+/-2.4)	2.6 (+/-0.6)	1.6 (+/-3.8)	33.0 (+/-1.5)	2.2 (+/-1.0)	2.7 (+/-4.2)
Lag 0-13		1.9 (+/-0.6)		31.5 (+/-3.9)	2.7 (+/-1.1)	1.6 (+/-3.0)
PET						
Lag 0-1	30.4 (+/-0.5)	2.9 (+/-0.7)	5.9 (+/-5.2)	30.5 (+/-0.5)	2.8 (+/-1.1)	6.2 (+/-9.5)
Lag 0-6	31.5 (+/-0.8)	2.9 (+/-0.7)	1.0 (+/-5.2)	31.7 (+/-0.6)	2.8 (+/-1.1)	6.4 (+/-9.5)
Lag 0-13		2.3 (+/-0.7)		31.4 (+/-0.9)	3.2 (+/-1.2)	7.2 (+/-8.7)
UTCI						
Lag 0-1	31.3 (+/-0.4)	2.8 (+/-0.7)	8.2 (+/-6.9)	31.6 (+/-0.4)	2.6 (+/-1.1)	19.9 (+/-12.5)
Lag 0-6	33.2 (+/-1.3)	2.7 (+/-0.7)	1.6 (+/-3.8)	33.5 (+/-0.6)	2.3 (+/-1.1)	12.5 (+/-11.3)
Lag 0-13		2.3 (+/-0.7)		31.8 (+/-3.3)	2.7 (+/-1.1)	2.6 (+/- 8.2)

Table 3: Thresholds and slopes of the <u>mean</u> (equivalent) temperature- <u>cardiovascular mortality</u> relationship in rural and urban areas for different lag periods

	Rural			Urban		
	$T_{threshold}$ [°C]	Percentage increase in mortality for each °C decrease in (equivalent) temperature below threshold (95% CI)	Percentage increase in mortality for each °C increase in (equivalent) temperature above threshold (95% CI)	$T_{threshold}$ [°C]	Percentage increase in mortality for each °C decrease in (equivalent) temperature below threshold (95% CI)	Percentage increase in mortality for each °C increase in (equivalent) temperature above threshold (95% CI)
T						
Lag 0-1		1.8 (+/-2.1)			3.1 (+/-3.1)	
Lag 0-6		1.8 (+/-2.2)		28.8 (+/-0.6)	4.0 (+/-2.9)	29.4 (+/-22.4)
Lag 0-13		1.8 (+/-2.4)		28.3 (+/-0.9)	3.5 (+/-3.1)	24.4 (+/-23.0)
HIWCI						
Lag 0-1		1.7 (+/-1.7)			2.1 (+/-2.1)	
Lag 0-6		1.7 (+/-1.6)		28.4 (+/-5.5)	4.2 (+/-2.8)	2.4 (+/-3.5)
Lag 0-13		1.7 (+/-1.6)		29.9 (+/-5.5)	3.1 (+/-2.7)	4.2 (+/- 5.4)
PET						
Lag 0-1		1.6 (+/-1.9)			3.1 (+/-2.3)	
Lag 0-6		1.6 (+/-2.0)		32.5 (+/-0.51)	3.9 (+/-2.5)	42.6 (+/-31.1)
Lag 0-13		1.6 (+/-2.1)		31.2 (+/-1.15)	3.5 (+/-2.9)	13.8 (+/-15.9)
UTCI						
Lag 0-1		1.9 (+/-1.9)			2.8 (+/-2.3)	
Lag 0-6		1.9 (+/-1.9)		28.5 (+/-4.9)	4.7 (+/-3.2)	4.1 (+/-5.5)
Lag 0-13		1.9 (+/-1.9)		31.3 (+/-4.5)	4.2 (+/-3.1)	6.3 (+/-8.0)