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**Assessing the relationship between global warming and mortality: lag effects of temperature fluctuations by age and mortality categories.**

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## Abstract

Although interests in assessing the relationship between temperature and mortality have arisen due to climate change, relatively few data are available on lag structure of temperature-mortality relationship, particularly in the Southern Hemisphere. This study identified the lag effects of mean temperature on mortality among age groups and death categories using polynomial distributed lag models in Brisbane, Australia, a subtropical city, 1996-2004. For a 1°C increase above the threshold, the highest percent increase in mortality on the current day occurred among people over 85 years (7.2% (95% CI: 4.3%, 10.2%)). The effect estimates among cardiovascular deaths were higher than those among all-cause mortality. For a 1°C decrease below the threshold, the percent increases in mortality at 21 lag days were 3.9% (95% CI: 1.9%, 6.0%) and 3.4% (95% CI: 0.9%, 6.0%) for people aged over 85 years and with cardiovascular diseases, respectively. These findings may have implications for developing intervention strategies to reduce and prevent temperature-related mortality.

**Key words:** Cardiovascular deaths; Lag structure; Mortality; Temperature; The elderly.

## Capsule:

In Brisbane, the lag effects lasted longer for cold temperatures, and shorter for hot temperatures. Elderly people and cardiovascular mortality were vulnerable to temperature stress.

## 1. Introduction

Although the association between temperature and mortality has long been known, there is an increasing interest in the assessment of the nature, magnitude and trend of such a relationship across different geographic areas due to climate change (Mirchandani et al., 1996).

There is a general agreement that the relationship between temperature and mortality is V-, U- or J-shaped, with optimum temperature corresponding to the lowest point in the temperature-mortality curve (Curriero et al., 2002; Kalkstein and Davis, 1989). The generalised additive model (GAM) is widely used to explore the temperature impact on mortality (Dominici et al., 2004). The three-piece linear spline (segmented linear) function is a simpler model that can be used to investigate the temperature-mortality relationships. This model divides temperature into three linear parts with hot and cold thresholds and the middle section is constrained to a zero slope. The “V” shaped association is the special case when cold and hot thresholds are equal (Armstrong, 2006).

It has been increasingly recognised that the current day’s mortality is often derived from exposure to the current day’s, or several previous days’, even weeks’ temperatures (Anderson and Bell, 2009; Bi et al., 2008). Recently, the distributed lag model has been applied to explore the lag structure of temperature on mortality (Analitis et al., 2008; Baccini et al., 2008; Hajat et al., 2005). Because there is substantial correlation between temperatures on days close together, investigators have implemented constrained distributed lag models (e.g. stratified, polynomial and smoothing constrained distributed lag models) to overcome this problem (Armstrong, 2006).

A number of previous studies have explored the lag effects of temperature on mortality (Anderson and Bell, 2009; Armstrong, 2006; Braga et al., 2001; Carder et al., 2005; Hajat et al., 2005). However, few data are available from the Southern Hemisphere. The vulnerability to temperature variation may differ with geographic area, and therefore, there is a need to examine the temperature-mortality patterns in different populations. This paper assessed the lag structure of temperature effects on mortality stratified into age groups and disease categories in Brisbane, Australia, a subtropical city.

## **2. Materials and methods**

### **2.1 Data collection**

Brisbane city, the capital of the state of Queensland, is situated on the east coast of Australia (27° 30' south and 153° 00' east). The area covers 1326.8 km<sup>2</sup> and the population was 1.76 million in the 2006 census year (Australian Bureau of Statistics, 2007). Brisbane has typically subtropical climate characteristics, with average temperatures around 25°C in summer (Dec-Feb), 21°C in autumn (Mar-May), 15°C in winter (Jun-Aug), and 21°C in spring (Sep-Nov) and the average annual precipitation was 908.8 mm during the study period.

Daily counts of deaths between January 1996 and December 2004 were obtained from the Office of Economic and Statistical Research of the Queensland Treasury. The causes of all deaths were classified according to the International Classification of Diseases, ninth version (ICD-9) before December 1996 and ICD-10 between December 1996 and December 2004 (ICD-9: 001–799 and ICD-10: A00–R99). Along with all-cause mortality, cardiovascular (ICD-9:390–459; ICD-10:I00-I79) (CVD) and respiratory (ICD-9: 460–519; ICD-10:J00-J99) deaths (RD) were examined separately. Influenza epidemics (ICD-9: 487.0-487.7; ICD-10: J10-J11) were considered as a potential confounder in the data analysis.

Daily meteorological data including daily maximum temperature, minimum temperature and mean relative humidity (RH) were obtained from the Australian Bureau of Meteorology. Air pollution data including daily mean ozone (O<sub>3</sub>), nitrogen dioxide (NO<sub>2</sub>) and particulate matter with aerodynamic diameter less than 10µm (PM<sub>10</sub>), recorded in a central monitoring site in Brisbane, were obtained from the Queensland Environmental Protection Agency.

In general, the quality of datasets used in this study is good. For example, the causes of deaths in Australia are usually assigned by medically qualified personnel and the checking procedures are routinely carried out to ensure high quality of the data. Climate and air pollution monitoring data are recorded according to the international standards. Additionally, the datasets in this study are comprehensive including climate, air pollution and mortality with very few missing values (<0.01%).

## **2.2 Data analysis**

In our previous research, we evaluated the performance of different temperature indicators through a comparison of Akaike's Information Criteria (AIC) across different models (Yu et al., 2010). Mean temperature was found to be a better predictor and was thus used as the temperature indicator in the study.

We plotted the graphs of the relationship between mean temperature and all-cause mortality based on the multivariable model with a thin plate regression spline function of temperature with 3 degrees of freedom. There was a "V" shaped association between temperature and mortality. AIC values were iteratively calculated for generalised additive models (GAMs) using one degree increment in mean temperature from 20 °C to 30 °C which was selected based on the visual inspection of the plots. The temperature corresponding to the model with

the lowest AIC value was chosen as the threshold temperature. A similar method has been adopted in many previous studies (Chung et al., 2009; Kim et al., 2006; Yu et al., 2010).

The polynomial distributed lag models were used to analyse the lag effects of mean temperature on mortality for all ages ( $D_{all}$ ), aged 15–64 ( $D_{15-64}$ ), 65–84 ( $D_{65-84}$ ) and over 85 years old ( $D_{85+}$ ), as well as mortalities for CVD and RD.

We applied the GAM model as follows (Dominici et al., 2004):

$$\begin{aligned} \text{Log}(Y_t) = & \alpha + \beta_C (T_t - \tau_C) + \beta_H (T_t - \tau_H) + \sum_{j=1}^p s(x_{jt}, 3) + \\ & \sum_{d=1}^q \delta_d x_d + s(\text{time}, 7 * \text{year}) + \varepsilon_t \end{aligned} \quad 1$$

Where  $t$  refers to the day of the observation;  $Y_t$  denotes the observed daily death counts on day  $t$ ;  $T_t$  denotes daily mean temperature on day  $t$ ;  $\tau_C$  and  $\tau_H$  represent cold and hot thresholds;  $s(.)$  denotes thin plate smoothing splines available in R software. Three degrees of freedom were used to smooth the covariates according to previous studies (Anderson and Bell, 2009; Filleul et al., 2006; Hajat et al., 2007; McMichael et al., 2008; Stafoggia et al., 2008b) and the time trend was controlled by smoothing calendar time with 7 degrees of freedom/year;  $x_j$  denotes the covariates of RH,  $PM_{10}$ ,  $NO_2$ , and  $O_3$ .  $x_d$  represents categorical factors for day of the week (DOW), holidays and influenza outbreak;  $\alpha$  is the intercept term;  $\beta$  and  $\delta$  are coefficients; and  $\varepsilon_t$  is the residual.

Polynomial distributed lag non-linear models were used to examine the lag effect of temperature on morality in this study to overcome the problem of the collinearity

(Armstrong, 2006; Schwartz, 2000). The polynomial distributed lag model is the subject to the restriction of equation 1 as follows:

$$\beta_C (T_t - \tau_C) / \beta_H (T_t - \tau_H) = \alpha + \beta_0 T_t + \beta_1 T_{t-1} + \dots \beta_n T_{t-n}$$

$$\beta_n = \sum_{k=0}^d a_k l_n^k, \quad l \in [0, m] \quad 2$$

where d is the degrees of freedom, l is the lag days and q is the maximum number of lag days (Schwartz, 2000). To obtain flexibility in modeling the distribution of lags, we constrained the shape of the distributed lag curve to fit a fourth-order polynomial (Armstrong, 2006). Sensitivity analysis showed no substantial changes in results when fifth- or sixth-order polynomial was used. Lags were chosen up to 30 days because longer-term effects might involve longer lags (Pattenden et al., 2003).

All statistical analyses were performed in SAS 9.1 (SAS Institute Inc., Cary, NC, USA) and using “mgcv” and “dlnm” functions of R packages (The R Foundation for Statistical Computing, version 2.11.1, 2010 <http://cran.r-project.org>).

### 3. Results

During the study period, the average values of the mean temperature and RH were 20.1°C and 72.5%, respectively. The mean daily concentrations of PM<sub>10</sub>, NO<sub>2</sub> and O<sub>3</sub> were 16.6 µg/m<sup>3</sup>, 12.1 ppb and 11.3 ppb, respectively (Table 1). In a total of 3,274 days, there were 53,316 deaths (including 22,805 CVD and 4,625 RD) registered in Brisbane. The percentages of total deaths by age group were 17.9%, 48.7%, 33.3% for 15-64 years, 65-84 years and over



85 years, respectively. As only 0.1% of deaths were from children aged less than 15 years, we did not include this age group.

The 3-dimesion plots of the temperature, lag and relative risk demonstrated a pattern with a lag of approximately 21 days for a range of cold temperatures and 3 days for a range of hot temperatures (Figure 1). In general, the estimates of hot effects on current days were higher than the overall lag effects of 3 days. However, the cold effects significantly increased when lags were considered in the groups of  $D_{all}$ ,  $D_{85+}$  and CVD (Table 2).

Compared to  $D_{all}$ , the groups of  $D_{85+}$  and CVD had higher percent increases for the hot effects. The percent increases in mortality associated with one degree increment of temperature were 7.2% (95% CI: 4.3%, 10.2%) and 5.8% (95% CI: 3.2%, 8.5%) on current days, and 5.4% (95% CI: 1.4%, 9.5%) and 4.1% (95% CI: 0.3%, 8.1%) on 3-day lags, respectively. The current day's hot temperature was significantly associated with mortality in the groups of  $D_{15-64}$  and RD (5.0%, 95% CI, 1.5%, 8.6% and 8.4%, 95% CI, 2.2%, 14.9%, respectively). The overall 3-day lag effects became insignificant for both groups (Table 2). However, for CVD mortality, the positive risks observed at lag 0-1 were followed by a period of lower-than-average deaths for lags between 2 and 6 days, with a return to the base line after 6 days (Figure 1).

Compared to  $D_{all}$ , the groups of  $D_{85+}$  and CVD mortality had higher percent increases associated with the overall 0-20 days' lag cold temperature. There were 3.9% (95% CI: 1.9%, 6.0%) and 3.4% (95% CI: 0.9%, 6.0%) increases, respectively, in mortality associated with one degree decrease of temperature below the threshold.

#### 4. Discussion

This is the first attempt to quantify the lag effects of temperature on mortality in a subtropical city in the Southern Hemisphere. This study contributes to the literature because the climate, adaptation and socio-demographic characteristics in Brisbane may differ from those countries in the Northern Hemisphere. We investigated lag effects up to 30 days on total deaths, deaths from different age groups (i.e.  $D_{15-64}$ ,  $D_{65-84}$  and  $D_{85+}$ ), and from cardiovascular and respiratory diseases for both hot and cold effects. Approximately up to three-week lag effects were observed for cold effects and the strongest hot effects were generally identified as acute with short-term lags (i.e. up to 3 days). The temperature effects resulted in more stress among the elderly aged over 85 years and people with cardiovascular diseases.

Heat seems to induce an acute event in the people with advanced forms of illness (e.g. myocardial infarction and stroke) and those who may find it difficult to deal with heat (e.g. the elderly) (Muggeo and Hajat, 2009). The mechanisms of heat-related deaths may result from failure in the thermoregulation which may be impaired by dehydration, salt depletion and increased surface blood circulation (Basu, 2009; Bouchama and Knochel, 2002). Elevated blood viscosity, cholesterol levels and sweating thresholds may also trigger heat-related mortality (Basu, 2009; McGeehin and Mirabelli, 2001).

However, there was a mortality deficit among CVD mortality after 2 lag days, which returned to the baseline after one week on hot days. This pattern suggests some compensatory risk reduction and is sometimes referred to as the harvesting phenomenon-viz, the bringing forward imminent deaths. This pattern was not seen for the effect of cold weather. The findings are consistent with previous studies (Braga et al., 2001; Pattenden et al., 2003).

1 By contrast, cold effects appeared to increase after several days, persisting for approximately  
2 three weeks. Other researchers reported similar findings (Braga et al., 2002; Goodman et al.,  
3 2004; Gouveia et al., 2003; Pattenden et al., 2003), but Curriero et al. (2002) only found a  
4 weak association between cold temperature and mortality at lag 4 days. The results suggest  
5 that cold temperature can exhibit a direct and indirect impact on mortality from days to  
6 weeks. It is plausible that the lags for the effects of low temperature may vary on mortality in  
7 length for different death categories (Carder et al., 2005). Deaths such as myocardial  
8 ischaemia from increased work demands on the heart muscle or from increased formation of  
9 thrombus can occur in hours or days (Mercer et al., 1999; Neild et al., 1994). For respiratory  
10 deaths, on the other hand, the timescale for manifestations of an adverse health effect may be  
11 longer—running into days or weeks. The progression to a full blown exacerbation of chronic  
12 obstructive pulmonary disease may take much longer (Carder et al., 2005).

13  
14 The results show that the relationship between temperature and mortality was stronger among  
15 the very old people (85+). The ability of older adults to maintain core temperature is usually  
16 compromised. The reduced sweat gland output, reduced skin blood flow, smaller increase in  
17 cardiac output and less redistribution of blood flow from renal and splanchnic circulations  
18 can consequently aggravate the injury to thermoregulation (Basu, 2009; Bouchama and  
19 Knochel, 2002; McGeehin and Mirabelli, 2001). Other factors such as living conditions  
20 including air conditioning, family and/or social support as well as medical cares can be  
21 modifiers of high temperature-related effects on the elderly mortality (Stafoggia et al., 2008a).

22  
23 Both cold and hot temperatures can affect the CVD deaths. A number of risk factors for  
24 increased cardiovascular deaths associated with the stress of low temperature include the  
25 incremented heart rate, blood pressure, peripheral vasoconstriction, plasma fibrinogen

1 concentrations, blood cholesterol levels and platelet viscosity (Ballester et al., 1997; Barnett  
2 et al., 2008; Carder et al., 2005). Whereas in hot weather, the heat balance of the body is  
3 sustained by enlarging skin vessels and increased sweating which in turn increases the  
4 cardiac work and loss of fluid and salt. This may lead to haemoconcentration, increased blood  
5 viscosity and the risk of thrombosis (Keatinge et al., 1986; Näyhä, 2005). In addition, the  
6 different types of CVD deaths may have different sensitivity to higher and lower  
7 temperatures. For example, cold temperature can induce deaths from myocardial ischemia  
8 and acute myocardial infarction (Hong et al., 2003; Stewart et al., 2002). In people with  
9 congestive heart failure, the extra heat load may lead to fatal consequences (Keatinge et al.,  
10 1986; Näyhä, 2005).

11  
12 We used daily mean temperature as exposure indicator for both heat and cold temperatures  
13 because 1) it may best reflect daily thermal stress and has been widely used in  
14 epidemiological studies (Anderson and Bell, 2009; Hajat and Haines, 2002; Kim et al., 2006);  
15 2) average temperature experienced throughout the whole day and night usually provides  
16 more easily interpreted results within a policy context (Anderson and Bell, 2009); 3) some  
17 researchers compared mean temperature with other indicators using statistical methods (e.g.  
18 comparison of AIC) and found that mean temperature was a better predictor of the death  
19 counts (Anderson and Bell, 2009; Hajat and Haines, 2002; Yu et al., 2010).

20  
21 Three major strengths are worth to mentioning. Firstly, to our knowledge, this is the first  
22 research on lag effects of temperature on mortality in Southern Hemisphere. Secondly, this  
23 study examined both cold and hot lag effects on mortality stratified by different age and death  
24 categories. Thirdly, the datasets in this study are comprehensive including climate, air  
25 pollution and mortality with very few missing data (<0.01%).

Three limitations of this study should also be acknowledged. Firstly, longer lag structure may introduce more measurement error due to increased time between the exposure and event (Anderson and Bell, 2009). A pattern of elevated mortality risk at the longer lag period would be evidence for the need to consider longer lags (Braga et al., 2001). The results suggest that 30 days may be more than sufficient, and that a 21-day lag would probably suffice for this particular dataset (Figure 1). Secondly, the data were only from one city and it is hard to be generalised. Finally, misclassification may occur to both exposure and outcome as this study used an ecological design. However, non-differential misclassification is likely to lead to an under-estimation of the effect.

The findings of this study may have implications for policymakers and future research. The findings are particularly important for estimating temperature-related effects on mortality for different subpopulations. The identified susceptible subpopulations signify the need to target high risk groups to effectively reduce temperature-related mortality. Future research might consider exploring the lag structure of temperature effects from different geographic areas and populations. As evidence on temperature-related health effects accumulates, it will facilitate the development of weather/health warning systems within a wide public health context.

## **5. Conclusions**

This research focused on the lag effects from a city with subtropical climate in the Australian continent, little research of such kind has been conducted in the Southern Hemisphere, while most studies were carried out in the US and Europe. The results of this study were consistent with those conducted in the Northern Hemisphere that cold temperature seems to have longer

lag effects, while hot temperature appears to exhibit short-term, acute effects. The very old people (ie, aged 85 or over) were most vulnerable to temperature stress. The cardiovascular mortality was also sensitive to the temperature variation.

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Table 1. Characteristics of mortality, weather condition, and air pollutants in Brisbane between 1996 and 2004.

			Percentile			
	Mean(SD)	Minimum	25%	50%	75%	Maximum
No. of daily deaths						
Total (non-external)	16.6 (4.5)	1	14	16	20	43
15-64	2.9 (1.7)	0	2	3	4	12
65-84	8.1 (3.1)	0	6	8	10	22
≥85	5.6 (2.6)	1	4	5	7	23
CVD	6.9 (3.0)	0	5	7	9	31
RD	1.4 (1.3)	0	0	1	2	8
Meteorological measures						
24-h mean temperature (°C)	20.1 (4.0)	9.8	16.8	20.5	23.4	31.9
Daily relative humidity (%)	72.5 (10.8)	23.8	67.1	73.6	79.6	98.4
Air pollutant concentration						
PM <sub>10</sub> (µg/m <sup>3</sup> )	16.6 (7.9)	2.1	12.1	15.3	19.3	162.1
NO <sub>2</sub> (ppb)	12.1 (5.8)	2.0	8	11	16	35.8
O <sub>3</sub> (ppb)	11.3 (4.8)	0	8	11	14	45

Abbreviations: PM<sub>10</sub>: particulate matter with an aerodynamic diameter less than 10 µm; NO<sub>2</sub>: nitrogen dioxide; O<sub>3</sub>: ozone; SD: standard deviation; RD respiratory deaths; CVD: cardiovascular deaths.

Table 2. The current and overall lag effect of temperature on mortality among all ages, 15-64, 65-84, over 85 years, and cardiovascular and respiratory deaths in Brisbane, 1996-2004.

Categories	Hot effects ( $\geq 24^{\circ}\text{C}$ ) % increase <sup>a</sup> (95% CI)		Cold effects ( $< 24^{\circ}\text{C}$ ) % increase <sup>b</sup> (95% CI)	
	0 day	0-2 days	0 day	0-20 days
All deaths	4.7 (3.1, 6.4)*	3.2 (0.9, 5.6) *	0.1 (-0.5, 0.8)	2.0 (0.7, 3.3)*
15-64	5.4 (1.8, 9.0)*	3.0 (-2.2, 8.4)	0.6 (-0.5, 1.7)	-1.2 (-3.6, 1.3)
65-84	3.4 (1.0, 5.8)*	2.0 (-1.5, 5.6)	0.4 (-0.6, 1.3)	0.3 (-2.2, 2.8)
$\geq 85$	7.2 (4.3, 10.2)*	5.4 (1.4, 9.5)*	0.2 (-0.9, 1.4)	3.9 (1.9, 6.0)*
CVD	5.8 (3.2, 8.5)*	4.1 (0.3, 8.1)*	0.4 (-0.6, 1.5)	3.4 (0.9, 6.0)*
RD	8.4 (2.2, 14.9)*	7.6 (-1.4, 17.4)	-0.5 (-2.8, 1.8)	4.1 (-1.6, 10.1)

Abbreviations: CVD: deaths from cardiovascular diseases; RD: deaths from respiratory diseases. <sup>a</sup> the percent increase with 1°C of temperature increase above 24 °C; <sup>b</sup>the percent increase with 1°C of temperature decrease below 24°C;\* p<0.05.