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ORIGINAL PAPER

Excess deaths during the 2004 heatwave in Brisbane, Australia

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Abstract The paper examines whether there was an excess of deaths and the relative role of temperature and ozone in a heatwave during 7–26 February 2004 in Brisbane, Australia, a subtropical city accustomed to warm weather. The data on daily counts of deaths from cardiovascular disease and non-external causes, meteorological conditions, and air pollution in Brisbane from 1 January 2001 to 31 October 2004 were supplied by the Australian Bureau of Statistics, Australian Bureau of Meteorology, and Queensland Environmental Protection Agency, respectively. The relationship between temperature and mortality was analysed using a Poisson time series regression model with smoothing splines to control for nonlinear effects of confounding factors. The highest temperature recorded in the 2004 heatwave was 42°C compared with the highest recorded temperature of 34°C during the same periods of 2001–2003. There was a significant relationship between exposure to heat and excess deaths in the 2004 heatwave [estimated increase in non-external deaths: 75 ([95% confidence interval, CI: 11–138; cardiovascular deaths: 41 (95% CI: −2 to 84)]. There was no apparent evidence of substantial short-term mortality displacement. The excess deaths were mainly attributed to temperature but exposure to ozone also contributed to these deaths.

Keywords Air pollution · Climate change · Harvesting · Mortality displacement · Ozone

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Introduction

The impact of weather on people has long been a matter of public health interest (Martens 1998; Curriero et al. 2000; McMichael et al. 2003; Vandentorren et al. 2004; Gosling et al. 2009). Extreme events such as heatwaves and cold spells are known to affect health. Historically, researchers have observed that episodes of extreme hot or cold temperatures increase mortality, and contemporary time series analyses show an association between temperature and mortality across a range of climate zones (Martens 1998; Vandentorren et al. 2004).

Global warming and other climate phenomena, such as El Nino events, have generated new interest in the temperature–mortality relationship (McMichael et al. 2003). According to the Intergovernmental Panel on Climate Change, global average surface temperature will likely increase by 1.8–4.0°C by 2100 (IPCC 2007). Further, the frequency, intensity and duration of extreme events, such as the numbers of hot days, heatwaves, and cold days, are expected to change. For example, in a temperate climate, a 2–3°C increase in the average summer temperature—even without allowing for any future increases in

variability—doubles the frequency of extremely hot days. Thus, the warming expected through the next decades is likely to substantially affect temperature-related mortality. Therefore, understanding the relationships between temperature and ill-health is becoming increasingly important.

Heatwaves are associated with significant short-term excess mortality, particularly in vulnerable populations (Rooney et al. 1998; Curriero et al. 2000; Laaidi et al. 2006; Basu et al. 2008; Zanobetti and Schwartz 2008). For example, it is estimated that the 2003 European heatwave caused nearly 15,000 excess deaths during the period from 1 to 20 August in France alone (Poumadere et al. 2005). The elderly and young children may not be able to cope with heat stress through efficient thermoregulation because of their higher sweating thresholds, thus increasing the risk of life-threatening consequences when their body temperatures rise (Drinkwater and Horvath 1979). A majority of deaths in heatwaves appear to be due to pre-existing chronic diseases, especially cardiovascular disease (McGeehin and Mirabelli 2001).

In Australia, not only are there considerable natural temporal variations in temperature, but there is also evidence that climatic conditions are changing. For example, eastern Australia experienced record temperatures during the period 1–22 February 2004 (almost exactly 6 months after the European heatwave of August 2003). About two-thirds of continental Australia recorded maximum temperatures over 39°C during the period 1–22 February, 2004. Brisbane recorded 41.7°C on the weekend of 21–22 February, exceeding the previous February record by nearly 1°C (National Climate Centre 2004). The 2004 heatwave occurred against a background of a long-term increase in the frequency of hot days and nights in Australia (Collins 2000), and more generally across the western Pacific–eastern Asia region (Manton et al. 2001). Over 1957–2004, the Australian average number of hot days (35°C or more) has increased by 0.10 days per year, the number of hot nights (20°C or more) by 0.18 nights per year, while the number of cold days (15°C or less) has decreased by 0.14 days per year and cold nights (5°C or less) by 0.15 nights per year (Collins 2000).

A number of studies have examined the impact of high temperature on mortality (Le Tertre et al. 2006; Ren and Tong 2006; McMichael et al. 2008). However, little is known about the relative effects of heat and air pollution on excess deaths during a heatwave period. Additionally, there is an increasing interest in whether there is any impact of high temperature on mortality in a population accustomed to warm weather. This paper examined whether a heatwave was associated with excess deaths in a subtropical environment and quantified the relative role of ozone and heat during the 2004 heatwave in Brisbane, Australia.

Materials and methods

Data

Brisbane is the capital of Queensland, Australia, located at 27°29′S, 153°8′E with a typical sub-tropical climate. There were 0.97 million residents living in Brisbane City in 2005 (BCC 2008). The data set used in this study consisted of daily counts of health outcomes, meteorological conditions, and air pollution in Brisbane City from 1 January 2001 to 31 October 2004. Daily meteorological data were provided by the Australian Bureau of Meteorology. There is no universally accepted definition of a heatwave, because it is difficult to agree on an appropriate heat index with an acceptable event threshold and duration, and to relate it to the climatology of the area under investigation. Generally speaking, a heatwave can be defined as a prolonged period of excessive heat (Australian Bureau of Meteorology 2008). The heatwave of this study had above average temperatures for a period of about 20 days (i.e. 7–26 February 2004), with a particularly hot spell at the end of that period (i.e. 17–26 February 2004).

The health outcome data included total non-external cause mortality (NECM) and cardiovascular mortality (CVM) in Brisbane City, which was supplied by the Australian Bureau of Statistics. NECM refers to deaths from all causes except injury and suicide. All deaths were classified according to International Classification of Disease, 10th version (ICD-10). Both NECM (all ICD-10 codes excluding ICD-10: S00-U99) and CVM (ICD-10: I00-I79) were chosen because heat is likely to contribute to these deaths.

The air pollution data for the same period comprised the daily average concentrations of ozone (O_3), particulate matter with diameters less than 10 μm (PM_{10}), and nitrogen dioxide (NO_2). Air pollution data were recorded at the central monitoring site at the Queensland University of Technology (QUT) and were provided by the Queensland Environmental Protection Agency (QEPA). This station was chosen because it is located in the center of the City. Twenty four-hour average O_3, PM_{10}, and NO_2 concentrations were calculated from the daily records from the monitoring station.

Analytical methods

We used two Poisson regression models to examine the heatwave effect. Firstly, we fitted a flexible nonparametric model to capture the patterns between predictors and health outcomes. Specifically, we used a generalised additive model (GAM) to examine the effects of the heatwave on NECM and CVM and to calculate excess deaths related to the high temperatures (Model 1). In this model, we adjusted

for seasonal effects using calendar days for 46 months (i.e. from 1 January 2001 to 31 October 2004) using a cubic smoothing spline function with 10 degrees of freedom (*df*) per year, which removed seasonal and long-term temporal trends and minimised autocorrelation in the residuals (Peng et al. 2006; Ren et al. 2006). We also adjusted for ozone, maximum temperature, dew point temperature using the cubic smoothing function with 4 *df* to capture the expected effects of these variables on daily mortality, independent of the heatwave. We also adjusted for days of week as a factor variable. We chose ozone to represent air quality because the concentrations of other air pollutants (PM_{10} and NO_2) differed little between the heatwave (2004) and non-heatwave periods 2001–2003). Additionally, we adjusted for other air pollutants (PM_{10} and NO_2), but these adjustments made very little difference to the modelling outcomes. Thus, we did not include these pollutants in the final models.

In order to examine the complete pattern of the heatwave effect and the possible mortality displacement, the "heatwave" term was defined, in Model 1, as a continuous variable assigned as 1–60 for days of the period between 1 February and 31 March 2004, covering the heatwave spell. All other days were assigned as 0, and a similar approach was used by Le Tertre et al (2006).The "heatwave" term was smoothed because we aimed to achieve the above two goals: (1) examining the pattern of the relationship between exposure to heatwave and mortality, and (2) investigating the possibility of short-term mortality displacement. The cubic smoothing function therefore captured the specific effects of the heatwave episode, after adjustment for a range of confounding factors including the common effects of air pollution and temperature in the model. We used that period because it would be long enough to allowed us to examine the potential short-term mortality displacement from the heatwave effect. Model 1 is described as follows:

$$\begin{aligned}\mathrm{Log}[\mathrm{E}(Y_t)] = {} & Dow_t + s(ozone_t) + s(temp_t) \\ & + s(season_t) + s(dptemp_t) \\ & + s(heatwave_t) \end{aligned} \quad (1)$$

where the subscript t denotes the time (day) of the observation; $\mathrm{E}(Y_t)]$ denotes expected NECM or CVM count on day t; Dow_t is a factor variable for the day of week at time t; $s(\cdot)$ is a cubic smoothing spline; $ozone_t$ and $temp_t$ refer to daily concentrations of ozone and maximum temperature at time t, respectively; $season_t$ means seasonality using day of calendar time of the year at time t and 10 *df* per year were used to adjust for seasonality. We used 10 *df* per year for season so that each degree was not over one and half months. $dptemp_t$ refers to dew point temperature at time t; $heatwave_t$ refers to heatwave at time t (df=12, 10, 8, or 6).

Because previous studies have shown that using the default convergence criterion might result in bias in GAM with S-plus software (Dominici et al. 2002; Ramsay et al. 2003), we adopted a stringent convergence criterion (1.0×10^{-10}).

Then, we estimated the excess number of deaths for NECM and CVM attributable to temperature and ozone during the heatwave period. Model 1 uses the variable 'day of the heatwave period' as a vehicle to capture the impact of the heatwave. However, using 'day of the heatwave period'' does not (1) capture the magnitudes of the excess temperature and ozone effects, nor (2) separate the temperature and ozone effects. To explore the relative role of ozone and temperature in excess mortality during the heatwave period, we analysed the data using a slightly different model. Model 2 was constructed by replacing the heatwave variable in Model 1 by two new variables "heattemp" and "heatozone". The values assigned to these new variables were average maximum temperatures and ozone concentrations, respectively, for each day. This enabled us to estimate the relative risk of temperature and ozone for NECM and CVM in the heatwave period, compared to the non-heatwave periods. We used 12 *df* for heattemp and heatozone to quantify the role of temperature and ozone during the heatwave period because the large degree freedom could sufficiently explore the variation during the short-term heatwave period. We obtained the predicted values of temperature (heattemp) and ozone (heatozone) by fitting Model 2 to calculate the relative contributions of temperature and ozone to NECM and CVM and the excess number of deaths during the heatwave period by the comparison of predicted values from these spline functions minus the usual seasonal patterns (Dominici et al. 2006; Le Tertre et al. 2006). Model 2 is described as follows:

$$\begin{aligned}\mathrm{Log}[\mathrm{E}(Y_t)] = {} & Dow_t + s(ozone_t) + s(temp_t) \\ & + s(season_t) + s(dptemp_t) \\ & + s(heattemp_t) + s(heatozone_t) \end{aligned} \quad (2)$$

Results

Table 1 shows the statistical summary of climate variables, concentrations of air pollutants and mortality in Brisbane between the 2004 heatwave and the same periods of 2001–2003. The highest temperature recorded in the 2004 heatwave was 42°C compared with the highest recorded temperature of 34°C during the same periods of 2001–2003. There was a statistically significant increase in both daily maximum and minimum temperatures in 2004

Table 1 Summary statistics of temperatures, air pollutants and health outcomes between the 2004 heatwave and the same periods of 2001–2003[a]

Variable	2004 heatwave		2001–2003 non-heatwave periods	
	Mean (SD)	Range	Mean (SD)	Range
Tmax (°C)	32.28 (3.45)	26–42	29.64 (2.46)	22–34
Tmin (°C)	22.17 (2.99)	16–28	21.05 (1.36)	18–24
NECM	16.76 (6.01)	7–35	13.88 (3.39)	6–23
CVM	6.76 (3.51)	1–19	5.64 (2.31)	1–12
Ozone (ppb)	12.54 (4.16)	6.00–19.63	9.16 (3.34)	4.58–23.00
PM_{10} ($\mu g/M^3$)	14.39 (2.98)	3.94–27.7	17.02 (3.31)	6.98–29.28
NO_2 (ppb)	7.77 (2.14)	2.0–18.17	9.12 (1.88)	5.79–17.92

[a] *SD* Standard deviation, *Tmax* maximum temperature, *Tmin* minimum temperature

compared with those during 2001–2003. The concentrations of ozone also increased in the 2004 heatwave, compared with the same periods of 2001–2003. In contrast, the levels of PM_{10} and NO_2 were relatively lower in the 2004 heatwave than those during the same periods of 2001–2003. Daily means of the numbers of NECM and CVM were noticeably higher in the 2004 heatwave, relative to the same periods of 2001–2003. The Person correlation coefficient between temperature and ozone was 0.164 ($p<0.01$).

Figure 1 shows that there was a remarkable increase in the counts of NECM and CVM during the hottest days in the 2004 heatwave.

In order to capture the impact of heatwave on mortality, we fitted the GAM model (Model 1) with the adjustment for seasonal effects, short-term fluctuations, ozone, maximum temperature and dew point temperature on the current day. Results illustrate that the heatwave was associated with an increase in the number of deaths from NECM and CVM (Fig. 2). The modelling estimates were similar to what we observed in Fig. 1 (i.e., relative risk for NECM and CVM increased remarkably during the heatwave period). No significant deficit of deaths existed during 4 weeks after the heatwave, regardless of which degree of freedom for heatwave (df=12, 10, 8, or 6) was used for the cubic smoothing spline function (only the results of df=12 and 6 presented here). However, the estimates for NECM and CVM counts became negative from about day 50 to day 60 (i.e., 4 weeks after the heatwave). We also fitted the GAM model using ozone, maximum temperature and dew point temperature at the lag of 1 day, but the patterns changed little.

We undertook a preliminary evaluation on the relative importance of maximum temperature and ozone concentration in excess deaths during the heatwave spell period via fitting Model 2. We extracted the predicted values of "heatozone" and "heattemp" terms on NECM and CVD from model fitting results and then plotted them to show the relative roles of ozone and temperature in excess mortality during the heatwave period. Figure 3 illustrates the relative contributions of maximum temperature and ozone during the hot spell period (17–26 February 2004) for each day. Log relative risk was used for the vertical axis because this came naturally from the fitted generalised additive Poisson model. The results show that, in general, maximum temperature contributed more than ozone to excess deaths for NECM and CVM, but their daily contributions differed during the period.

The excess number of deaths during the 2004 heatwave was estimated using Model 2. A substantially increased number of deaths were observed during the heatwave period. The model estimates that the excess numbers of NECM and CVM attributable to heat stress were 75 (95% CI: 11, 138) and 41 (95% CI: −2 to 84), respectively, during the 2004 heatwave. We also estimated the excess number of deaths by directly comparing the number of deaths in the 2004 heatwave with the average number of deaths in the same periods of 2001–2003. The results show that there was a substantially increased number of deaths in the 2004 heatwave [NECM: 87 (23% increase); CVM: 31 (20% increase)] compared with those in the same periods of 2001–2003. Then, we estimated the relative role of temperature and ozone in the excess deaths of NECM and CVM using Model 2 (Table 2). The model indicates that temperature contributed more than ozone to the estimated excess number of deaths. According to the fitted model, the excess numbers of NECM attributable to maximum temperature and ozone were 45 (95% CI: 14, 77) and 14 (95%CI: −14, 42), respectively, between 17 and 26 February 2004 (i.e. during the hot spell period).

Discussion

It is the first study, to the best of our knowledge, to examine the relative role of temperature and ozone in excess deaths during a heatwave in a subtropical city. The major findings of this study are: (1) it is estimated that there was a significant increase in non-external and cardiovascular deaths during the 2004 heatwave in Brisbane; (2) ozone

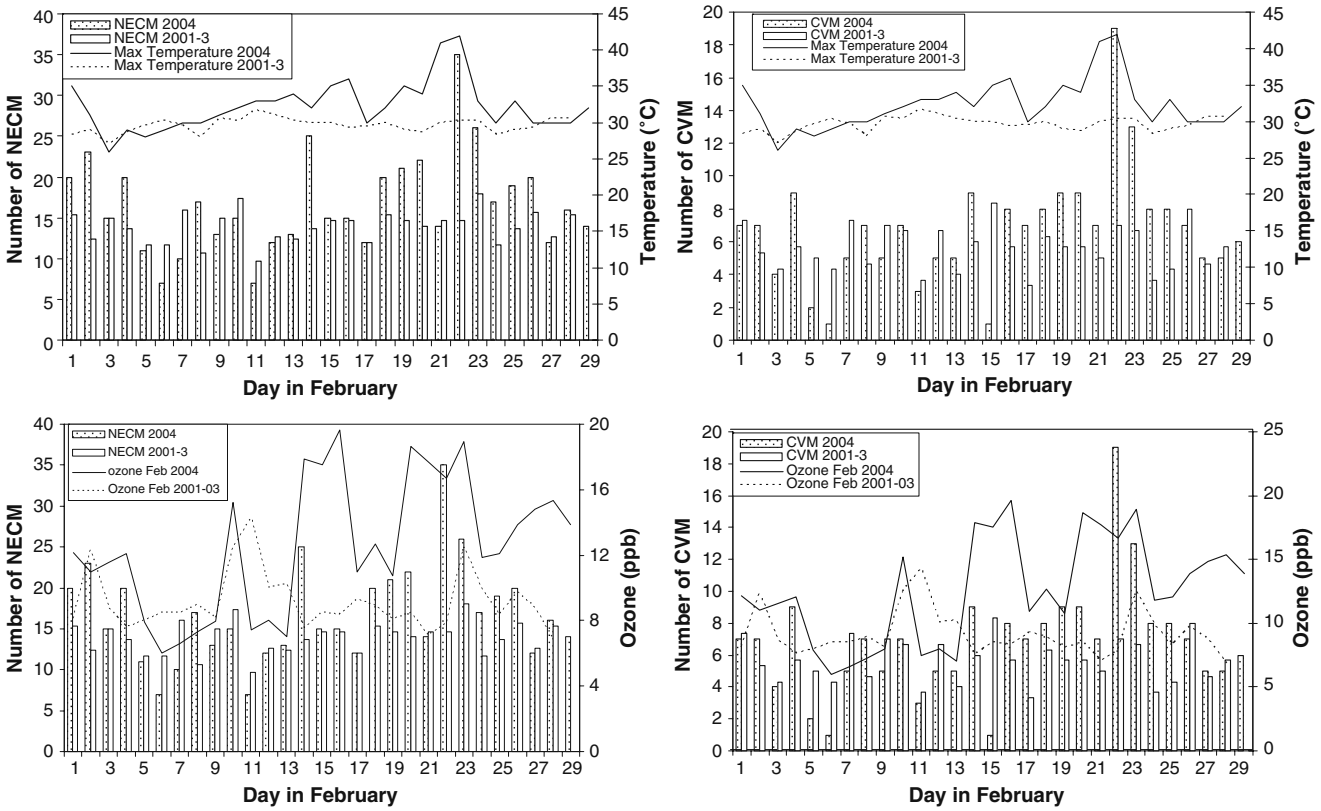

Fig. 1 Comparison of the associations of maximum temperature and daily average ozone concentrations with non-external mortality (*NECM*) and cardiovascular mortality (*CVM*) in Brisbane, Australia, between February, 2004 and the same periods of 2001–2003

contributed to the excess deaths but appeared to have played a relatively less important role than heat in the occurrence of the excess deaths; and (3) there was no significant evidence of appreciable short-term mortality displacement.

The estimated number of excess deaths identified by our analysis is relatively smaller than that observed in most French cities during their 2003 heatwave (Le Tertre et al. 2006). There are several possible reasons for this difference. Firstly, the 2004 Brisbane heatwave (8°C above season norm) was less severe than the 2003 French heatwave (e.g. 12°C above season norm in Paris), although the actual maximum temperature was much higher in the former (42°C) than the latter (32°C). Secondly, adaptability is likely to play an important role in coping with heat stress. Brisbane is subtropical and residents are accustomed to hot weather. Thirdly, population characteristics (e.g. usage of air conditioning and the prevalence of elderly living in nursing homes) might differ substantially between Brisbane and French cities. Finally, the housing structure is unique in Brisbane (so-called "Queenslander" design) and many houses are designed to allow rapid cooling in the evening/night. Additionally, people take some necessary measures to adapt to increasingly hot weather. For example, in Queensland, the proportion of households using air conditioning was 14.3% in 1980, and it is estimated that this figure will increase to 63.8% by 2010 (NAEEEC 2006). Consistent with this trend, the total number of air conditioners sold in Australia was less than 400,000 units in 1991, but rose to 1,200,000 in 2003 (NAEEEC 2006).

The inter-relationship between air pollution, heat stress and health is complex. Specifically, the question of the relative impacts of heat stress and air pollution on mortality during a heatwave is contentious, as temperature and air pollutant concentrations (e.g. ozone) are likely to increase simultaneously in such episodes and both exposures can increase deaths (Stedman 2004; Fischer et al. 2004; O'Neill et al. 2005). For example, Fischer et al (2004) reported that in the Netherlands an excess of 1,000–1,400 deaths was estimated to be due to the hot temperatures that occurred during the 2003 summer period. They argued that an excess of around 400–600 air pollution-related deaths may have occurred in the summer period June–August 2003 compared to an 'average' summer. These calculations suggest that, in the Netherlands, a significant proportion of the deaths attributed to the hot summer weather may have been caused by air pollution. Stedman (2004) also reported that temperatures peaked at a new record of 38.5°C in the UK in the first 2 weeks of August 2003. The UK Office for National Statistics reported an excess of 2,045 deaths in

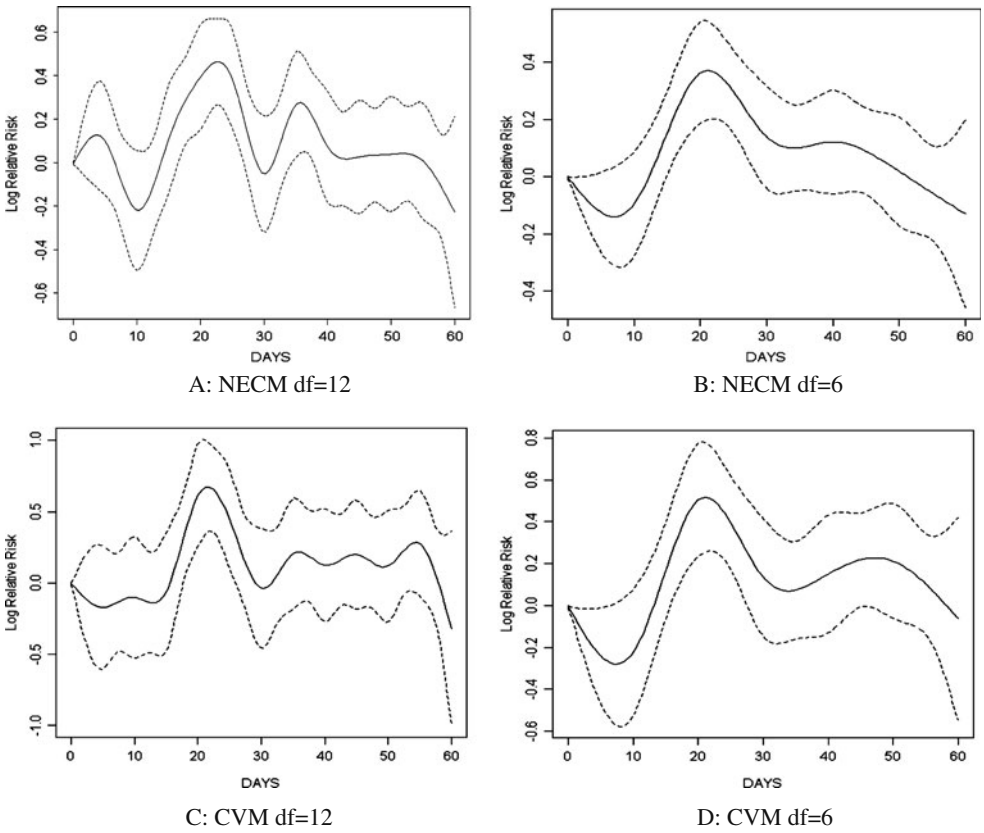

Fig. 2 Relative risks of death from NECM (**a** *df*=12, **b** *df*=6) and CVM (**c** *df*=12, **d** *df*=6) during the 2004 heatwave, compared with the same period between 2001 and 2003 in Brisbane (Days 1–60 refer to 1 February to 31 March 2004)

England and Wales for the period from 4 to 13 August 2003 above the 1998–2002 average for this time of year. It was estimated that there were between 423 and 769 excess deaths (i.e. 21–38% of the total excess deaths) in England and Wales during the first 2 weeks of August 2003 associated with the elevated ambient ozone and PM_{10} concentrations. However, it may be inappropriate to apply previously established dose-response functions to estimate attributable risks for heatwave because the conditions of both weather and air pollution (e.g. levels and components) might have changed. In this study, we found that air pollution (i.e. ozone) played a lesser role than heat in the occurrence of excess deaths because its association with deaths was relatively weaker in the model (Fig. 3). Two previous studies in warm places also reported that the heatwave effect cannot be completely explained by air pollution (Katsouyanni et al. 1993; O'Neill et al. 2005). Similar to our findings, the 2003 heatwave in France was associated with a large increase in the number of deaths. Air pollution and long-term and seasonal trends are unlikely to substantially confound these estimates (Le Tertre et al. 2006).

Increases in air pollutant concentrations (e.g. particulates) have been found to be associated with increases in daily mortality (Samet et al. 2000; Pope et al. 2002; Ritz et

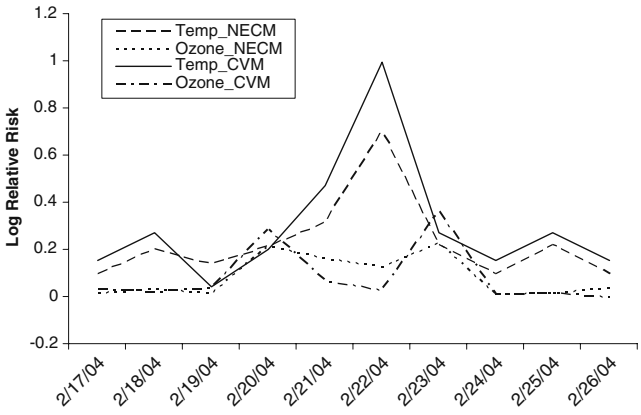

Fig. 3 The relative contribution of maximum temperature and ozone to NECM and CVM during the hot spell period (17–26 February 2004) on the current day in Brisbane (*Temp_CVM*, *Temp_NECM*, *Ozone_CVM* and *Ozone_NECM* refer to the log relative risks of maximum temperature to CVM, maximum temperature to NECM, ozone to CVM and ozone to NECM, respectively)

Table 2 Estimated excess numbers of deaths attributable to maximum temperature and ozone during the 2004 hot spell[a]

	NECM	95% CI	CVM	95% CI
Tmax	45	14~77	22	8~46
Ozone	14	–14~42	5	–14~25

[a] Using Model 2 with 12 *df* for heattemp and heatozone

95% CI: 95% Confidence Interval

al. 2006; Ren et al. 2008a, b). However, it may be that increases in air pollution only hasten the deaths of individuals in a small, frail subset of the population whose longevity is likely to be short even in the absence of particulate air pollution. This hypothesis has been termed "harvesting" or mortality displacement (Roberts and Switzer 2004). A number of studies have been conducted to investigate whether an increase in mortality associated with air pollution is caused by mortality displacement, and there are mixed results across these studies (Zanobetti et al. 2002; Bell et al. 2004; Roberts and Switzer 2004). Compared with air pollution research, relatively few data are available on whether there is mortality displacement or harvesting associated with heatwaves. Some studies reported short-term mortality displacement for heat deaths (Braga et al. 2001; Hajat et al. 2005). However, recently, Le Tertre et al. (2006) investigated the impact of the 2003 heatwave on all-cause mortality in nine French cities, and found that there was little evidence of mortality displacement in a few weeks after the heatwave. In this study, we found the negative risk around 50–60 days (Fig. 2) but it seems likely that this arises from chance fluctuations rather than displacement because it occurred more than 4 weeks after the heatwave and a similar fluctuation occurred before the heatwave, i.e. around 0–10 days in Fig. 2. The results of our study tend to corroborate the findings of the French study.

There are two major strengths in this study. Firstly, this study systematically examined the impact of heat on mortality during a heatwave period in a population accustomed to warm weather. Secondly, the relative importance of heat stress and ozone in excess deaths was assessed in relation to the impact of the 2004 Brisbane heatwave. These issues are vitally important in the quantification of the effects of a heatwave on population health.

There are also several limitations. Firstly, only one geographic location (i.e. Brisbane) was considered, and thus the findings from this study need to be interpreted cautiously. Secondly, we did not include external causes of deaths (e.g. injuries and suicides) in this study. It is possible that heatwave may affect the occurrence of such events, although the number of deaths due to these causes might be small. Additionally, we only examined the impact of heatwave on broad categories of mortality but did not distinguish associations with specific causes of death or age groups, which will be the subject of future research. Finally, we only assessed the possible health impact of heat stress on the current day but did not look into its combined lag and/or cumulative effects. However, we did fit the GAM model using ozone, maximum temperature and dew point temperature at the lag of 1 day, and the patterns changed little.

In conclusion, the heatwave in February 2004 was associated with a significant increase in non-external and cardiovascular deaths in Brisbane, Australia. a subtropical city. Although adaptive behaviour to hot weather conditions may reduce excess mortality, this study shows that a heatwave can result in an increase in mortality even in a population accustomed to warm weather. As global climate change continues, it is likely to lead to more frequent and intense heatwaves in many cities around the world. Thus, it is essential to develop and evaluate effective strategies to protect population health and community well-being from the impact of heat stress.

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