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# Socioeconomic differentials in the temperature–mortality relationship in São Paulo, Brazil

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**Background** We investigated the impact of environmental temperature on mortality in São Paulo, Brazil, and examined differences in the temperature–mortality relationship with respect to cause, age group, and socioeconomic position (SEP).

**Methods** Generalized additive Poisson regression models adjusted for non-temperature related seasonal factors (including air pollution) were used to analyse daily mortality counts for selected causes from 1991 to 1994. Individuals were classified by the aggregate SEP of their area of residency. These were analysed as potential modifiers of the temperature–mortality relationship.

**Results** Among the elderly we observed a 2.6% increase in all-cause mortality per degree increase in temperature above 20°C, and a 5.5% increase per degree drop in temperature below 20°C, after adjustment for confounding. Relationships were similar in children, but somewhat weaker in adults. Cold effects were present for deaths due to cardiovascular disease (CVD), respiratory, and other causes, with effects being greatest in the respiratory group. Heat effects were not found for CVD deaths in adults, but otherwise varied little by cause of mortality. There was little evidence for a modification of the mortality effects of cold or heat by SEP.

**Conclusions** These findings show that the U-shaped pattern of the temperature–mortality relationship found in cooler northern countries occurs also in a sub-tropical city. In addition, the relative effects of temperature were similar in each socioeconomic grouping.

**Keywords** Temperature, heat, cold, mortality, socioeconomic position, epidemiology

It is now increasingly recognised that our planet is getting warmer and that human activity is at least partially responsible for this.[1–3] The scientific evidence for global climate change has allowed climatologists to forecast future changes in ambient temperatures.[4,5] Thus, concerns about longer-term indirect effects of global warming are now accompanied by concerns on more immediate impacts of ambient temperature on health.

Many studies have reported that days of low and high ambient temperatures are associated with increases in mortality. Episode analysis and time-series analysis have been used to determine the acute effects of very hot weather on populations, and to describe the daily temperature–mortality relationships.[6–13] Analyses of daily meteorological and mortality data in cities in Europe and the US show that, during summer, the overall mortality rises as temperatures increase.[13–15] Similarly, other studies from countries with colder climates have examined the relationship between exposure to severe cold weather and mortality and found significant cold-related excess mortality.[16–19]

However, to date there has been very little epidemiological research on the health impact of thermal stress on urban populations of developing countries. In addition, important issues concerning the effect of temperature on health have not yet been properly addressed. In particular, there are indications that populations can differ substantially in their vulnerability to thermal stress.[15] It is plausible that the effect of weather on mortality depends on the age structure, socio-demographic and environmental conditions of populations. Such information is important for predicting the impact of weather changes on mortality.

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We therefore carried out a study in São Paulo, Brazil, aimed at investigating the impact of ambient temperature on mortality. We sought to identify the most vulnerable population subgroups in terms of age and cause, and to examine differences in the temperature–mortality relationship with respect to socioeconomic position (SEP) gradients within the city.

## Methods

Daily counts of deaths for different causes and age groups were extracted from the city's mortality information system for the period of 1991–1994. The great majority of deaths in São Paulo are assigned by medically qualified personnel and checking procedures are routinely carried out by this system to ensure high quality of the data. All-cause mortality excluding violent deaths (International Classification of Diseases Ninth Revision [ICD-9], chapter 17) was examined for the elderly (≥65 years), for adults (15–64 years), and for children (<15 years). Neonatal deaths (up to one month old) were excluded from the children's age group since these deaths are highly associated with childbirth and perinatal conditions.

Along with all-cause mortality, cardiovascular (ICD-9: 390–459), respiratory (ICD-9: 460–519), and other non-violent deaths were examined separately for the elderly and adults. Due to the smaller number of deaths in children we decided to analyse only deaths for all causes (excluding violent deaths) for this age group. Only deaths of city residents, selected by their residential address, were included in this analysis.

Data were obtained on areas within São Paulo to allow the classification of their resident population by a range of five socioeconomic factors. These factors, all at the district level, were: mean income level, average level of education, presence of adequate sewerage system in the household, percentage of households with piped treated water services, and crowding. These variables were used to generate a composite index of SEP for each of the 58 administrative districts of São Paulo. For each socioeconomic variable a value was assigned from zero (for the district with the worst conditions) to one (for the district with best conditions). The composite index was the mean of these five values for each district. The 58 administrative districts of São Paulo were then classified into quartiles of this composite socioeconomic index. This approach, which has been used previously,[20,21] was employed in the present study to characterize the socioeconomic conditions of each subject based on their district of residence.

Daily meteorological parameters were obtained for the same period from the University of São Paulo. These included daily mean, maximum and minimum temperature, and relative humidity, barometric pressure, rainfall, wind speed, and wind direction, measured at one site close to the central region of the city. This site is considered to be representative of average weather levels of the city as a whole. Daily levels of sulphur dioxide ($SO_2$), fine particulate matter ($PM_{10}$), carbon monoxide (CO), ozone ($O_3$), and nitrogen dioxide ($NO_2$) were obtained from CETESB, the environmental agency in São Paulo. Additional information on the air pollution monitoring system in São Paulo can be found elsewhere.[21]

The analytical approach followed standard time-series methods that have been developed for air pollution studies. These involved the use of generalized additive Poisson regression models (GAM) with non-parametric smoothers to control for seasonal patterns.[22] The approach takes into account any smooth variations in the denominator and rates over time, and so allows for a seasonally adjusted baseline against which the measurement of the effects of temperature can be made. The aim of the seasonal adjustment was to remove the mid- to long-term seasonal cycles in the series, but not the short-term patterns, since it is these that we seek to investigate against fluctuations in temperature. Specifically, seasonal control was achieved by using smoothing splines with 7 d.f. per year of data so that little information from time-scales longer than approximately 2 months is included when estimating temperature effects.[23]

The possible confounding effects of humidity were controlled for using a broad smoothing spline (6 d.f.) because graphical inspection suggested a weak non-linear relationship with mortality. Indicator variables were added to allow for day-of-week effects, and daily measures of air pollution were also adjusted for in each model. As there was no evidence in these data that wind speed, pressure, and rainfall were associated with mortality ($P > 0.1$), these were not included in the final analysis.

In order to investigate the temperature–mortality relationship, a smooth function for temperature (again using 6 d.f.) was added to each model and the smoothed effect plotted. This was conducted for temperature measures averaged across the day of death and the previous day (lags 0–1) to reflect very prompt effects, and over 3 weeks (lags 0–20) to assess effects delayed by up to this duration. Comparison of model likelihoods suggested that mean temperature was a better predictor of both hot and cold-related mortality, compared with either maximum or minimum temperature. The plots of the smoothed effect of temperature suggested that the relationship between temperature and mortality was linear above (and below) temperature values at which mortality was at its lowest. To this end, in order to quantify the effects of temperature we used a characterization of heat (and cold) effects simpler than the spline curves, comprising linear terms for effects above (and below) temperature values below the minimum mortality point. We refer to the slopes of these lines as heat and cold effects respectively.

Goodness of fit of each statistical model was assessed from the model residuals, the dispersion-penalized AIC, and the partial auto-correlation function (PACF) to determine the degree of remaining autocorrelation (non-independence of adjacent days).[23] Poisson GAM regression allowing for overdispersion was used to determine the relative risk (RR) of death associated with a 1°C change in mean temperature. Results are discussed in terms of percentage changes, which are derived from the RR using the following formula: % change = (RR-1)*100. After adjusting for temporal variation as described above, no significant autocorrelation remained.

The analysis was repeated for models with separate mortality counts for the four socioeconomic groups (SEP) to explore the potential modification of temperature–mortality effects of area-level differences in socioeconomic index. Each model contained an indicator variable for each SEP group along with an interaction term to estimate temperature effects within each of the these four groups. More formally, the models for count $Y_{ij}$ of deaths on date i in SEP group j were:

$$E(Y_{ij}) = \exp\{\alpha_j + \beta_{1j} x_{1i} + \beta_{2j} x_{2i} + S_0(\gamma, i) + S_3(\delta, x_{3i}) + \Sigma_{k>3}(\beta_k x_{ki})\}$$

Where $\beta_{1j}$ and $\beta_{2j}$ are the coefficients (slopes) for low ($x_{1i}$) and high ($x_{2i}$) temperature effects specific to SEP group j; $S_0(\gamma, i)$ is the smooth function of date (i), $S_3(\delta, x_{3i})$ is the smooth function of relative humidity, and $\beta_k x_{ki}$ are linear terms for air pollution, holidays, and day-of week (indicators).

We also fitted completely separate models for each SEP quartile, thus allowing non-temperature effects ($S_0$, $S_3$, and $\beta_k$, $k > 3$) to differ across quartiles, but found that these did not significantly improve model fit. All statistical analyses were carried out using the statistical package S-Plus.[24]

## Results

A total of 212 577 deaths for all-causes and ages excluding external causes for city residents occurred in the city of São Paulo during 1991–1994. Descriptive statistics for mortality, and meteorological and air pollutant variables are displayed in Table 1. The characteristic temperate climate of a sub-tropical city is clear from the non-extreme meteorological parameters observed in São Paulo.

As previous studies in São Paulo have shown an association between air pollution and mortality,[21] daily levels of these pollutants were included in the analysis of the effect of temperature on mortality to control for confounding. Humidity was associated in these data (after adjustment for other variables) with a reduction in mortality at very high levels. Humidity was therefore included as a smooth spline (6 d.f.) to control for possible confounding.

**Table 1** Descriptive statistics for daily levels of meteorological variables, air pollutants, and mortality in São Paulo, Brazil, 1991–1994

| | | Percentile | | | | |
|---|---|---|---|---|---|---|
| Variable (unit) | Mean (SD) | min. | 25 | 50 | 75 | max. |
| **Temperature (°C)** | | | | | | |
| Mean | 19.3 (3.3) | 7.0 | 17.1 | 19.6 | 21.9 | 26.3 |
| Maximum | 25.4 (4.6) | 10.6 | 22.4 | 26.0 | 28.8 | 35.4 |
| Minimum | 15.2 (3.3) | 0.6 | 13.0 | 15.5 | 17.8 | 21.4 |
| **Relative humidity (%)** | 78.3 (9.9) | 42.0 | 72.9 | 79.4 | 85.4 | 98.0 |
| **Rainfall (mm)** | 4.1 (10.0) | 0 | 0 | 0 | 2.6 | 121 |
| **Wind speed (km/h)** | 6.1 (1.8) | 1.6 | 4.9 | 6.0 | 7.2 | 13.8 |
| **Pressure (mmHg)** | 95.8 (2.8) | 88.4 | 93.9 | 95.7 | 97.6 | 106.0 |
| **Air pollutants** | | | | | | |
| $PM_{10}$ ($\mu g/m^3$) | 65.0 (30.4) | 18.4 | 44.3 | 56.6 | 77.5 | 231.8 |
| $SO_2$ ($\mu g/m^3$) | 17.7 (8.6) | 2.9 | 11.6 | 16.2 | 21.9 | 61.1 |
| $NO_2$ ($\mu g/m^3$) | 165.3 (89.1) | 26.0 | 104.4 | 144.0 | 204.1 | 692.9 |
| CO (p.p.m.) | 5.6 (2.3) | 0.9 | 4.0 | 5.4 | 6.9 | 22.8 |
| $O_3$ ($\mu g/m^3$) | 63.4 (41.9) | 2.7 | 30.4 | 53.6 | 85.5 | 272.0 |
| **Mortality** | | | | | | |
| All causes | 145.5 (20.1) | 76 | 131 | 144 | 157 | 234 |
| Cardiovascular disease | 55.0 (10.3) | 21 | 48 | 54 | 61 | 94 |
| Respiratory disease | 18.2 (6.0) | 4 | 14 | 17 | 21 | 50 |
| **>65 years old** | | | | | | |
| All causes | 69.4 (13.3) | 28 | 60 | 68 | 77 | 121 |
| Cardiovascular disease | 34.0 (8.0) | 11 | 28 | 33 | 39 | 67 |
| Respiratory disease | 11.3 (4.5) | 1 | 8 | 11 | 14 | 35 |
| **15–64 years old** | | | | | | |
| All causes | 60.7 (9.5) | 31 | 54 | 60 | 67 | 106 |
| Cardiovascular disease | 20.6 (5.0) | 7 | 17 | 20 | 24 | 42 |
| Respiratory disease | 6.4 (2.8) | 1 | 4 | 6 | 8 | 17 |
| **0–14 years old** | | | | | | |
| All causes | 6.8 (2.9) | 1 | 5 | 7 | 9 | 20 |
| Respiratory disease | 2.8 (1.6) | 1 | 2 | 2 | 4 | 10 |

As expected, temperature and daily deaths exhibit a marked temporal pattern (Figure 1), with daily counts of deaths higher during colder months. In addition, a slight increase in the mean number of deaths was observed over time, which probably reflects the population growth during the period of the study. Figure 2 shows the relationship of temperature with mortality, both before and after adjustment for season, day-of-week, pollution, holidays, and humidity levels. The top graphs on the Figure are for mean temperatures over the current and previous day (lags 0–1), and the bottom graphs are for mean temperatures over 3 weeks (lags 0–20). At both lags there is a marked increase in deaths at low temperatures, though the adjusted relationship is stronger for the longer lag period (0–20). Only the 0–1 lag values show a clear relationship with heat. In subsequent analysis we therefore used 0–1 lags in linear heat effect models, and 0–20 lags in linear cold effect models.

To refine the estimate of the cold effect we calculated a weighted mean temperature, weighting each of the three included weeks (days 0–6, 7–13, 14–20) with weights proportional to coefficients obtained when fitting each of the three weeks separately—a crude distributed lag model.[25] This gave most weight to days 0–6 (w = 50%), less to 7–13 (w = 29%) and least to 14–20 (w = 21%). With these lags, we used maximum likelihood to identify the 'change-points'—temperatures at which linear cold and heat effects started. Both change-points were 20°C. Analysis by age and cause-specific deaths and by separate SEP groups also suggested change-points close to 20°C (results not shown). To simplify comparisons, a change-point of 20°C was therefore adopted in all models. In no model was a significant modification of the effect of temperature by humidity observed.

We observed a heat effect on all-cause mortality in children (<15 years), in adults (15–64 years), and in the elderly (≥65 years) (Table 2). Effects for adults and the elderly were observed for all specific causes of death investigated except CVD in adults. In summary, for a 1°C increase in mean temperatures above 20°C for the average of the same and the previous day we observed a 2.6% (95% CI: 1.6–3.6%) increase in deaths for all causes in children and a 2.5% (95% CI: 2.1–2.8%) increase in the elderly. For adults proportional increases were smaller but still statistically significant: 1.5% (95% CI: 1.1–1.8%).

A cold effect could also be observed for the three age groups, with greater slopes (Table 2). For a 1°C decrease in mean temperature for the 3-weekly weighted average of temperatures below 20°C we observed a 4.0% (95% CI: 3.2–4.8%) increase in deaths for all causes in children. In contrast to the heat effect, the effect of cold on mortality was slightly higher for CVD and respiratory mortality in the elderly and adults than all-cause mortality.

According to these results, the precipitation of 2.4%, 1.4%, and 2.3% of all-cause deaths in children, adults, and the elderly are attributed to temperatures above 20°C in São Paulo. A higher proportion—5.5%, 3.6%, and 7.4% of all-cause deaths for the same age groups—are attributable to temperatures below 20°C.

Next, we investigated whether there were any differences in the effect of temperature on mortality for all causes according to SEP (Figure 3). There was little evidence for trends in the heat effect by SEP. The only suggestion of a trend was in the elderly,

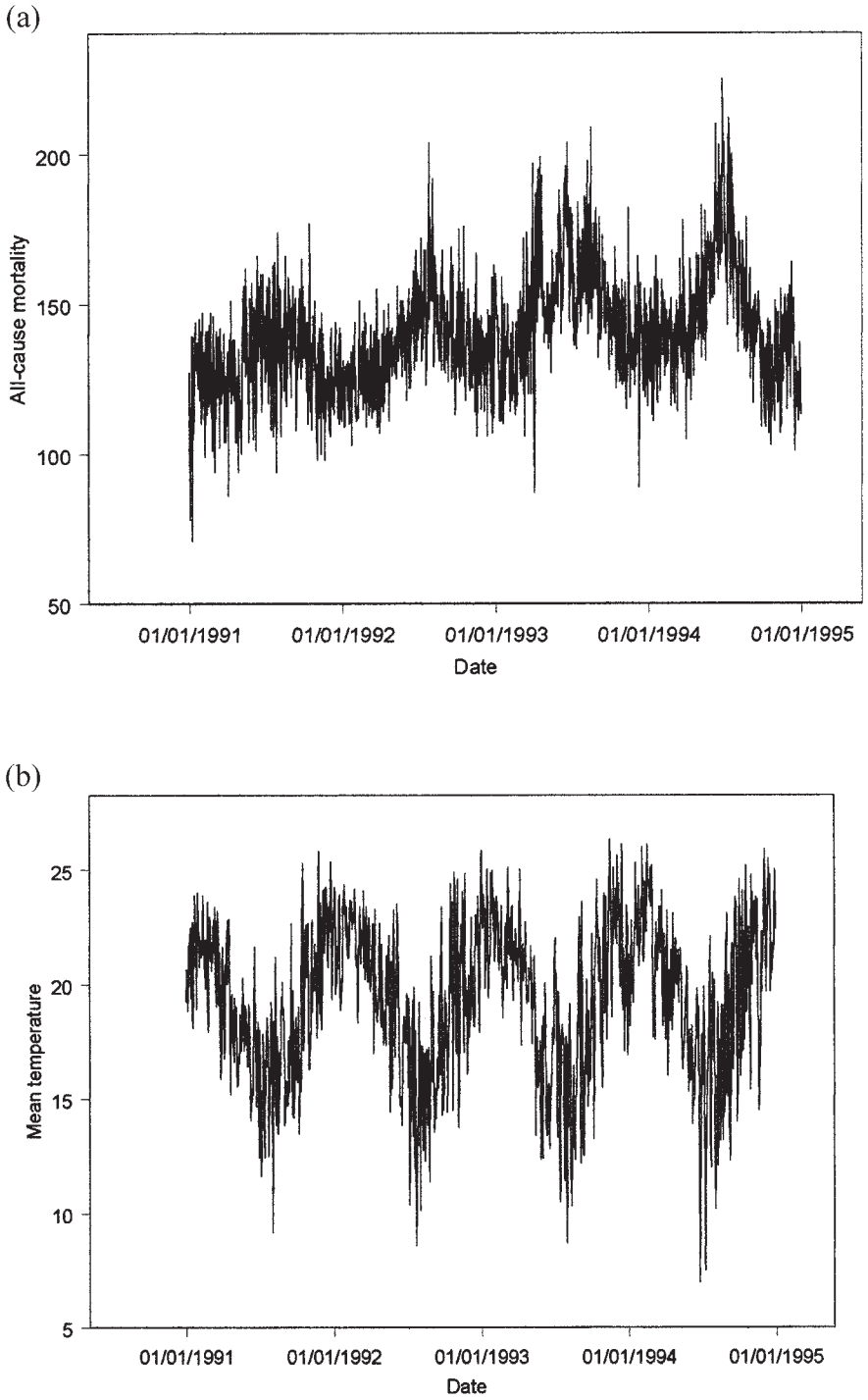


**Figure 1** Time-series of (a) daily all-cause mortality counts and (b) daily mean temperature in São Paulo, Brazil, 1991–1994

with the group in the highest SEP being affected to a smaller extent (1.4% per degree) than the other SEP groups (2.4–2.6%). However, this did not attain a conventional level of statistical significance. Using maximum rather than mean temperatures, we found a stronger trend in heat effects for the elderly ($P = 0.05$), but not for children or adults (results not shown).

There was no evidence for modification of the mortality impact for all causes due to cold by SEP (Figure 4). Restricting attention to deaths from CVD or respiratory disease in adults and the elderly again suggested very little evidence for a trend in either the heat or cold effect by SEP.

## Discussion

This study found that both hot and cold daily temperatures were associated with daily mortality in São Paulo. The effects

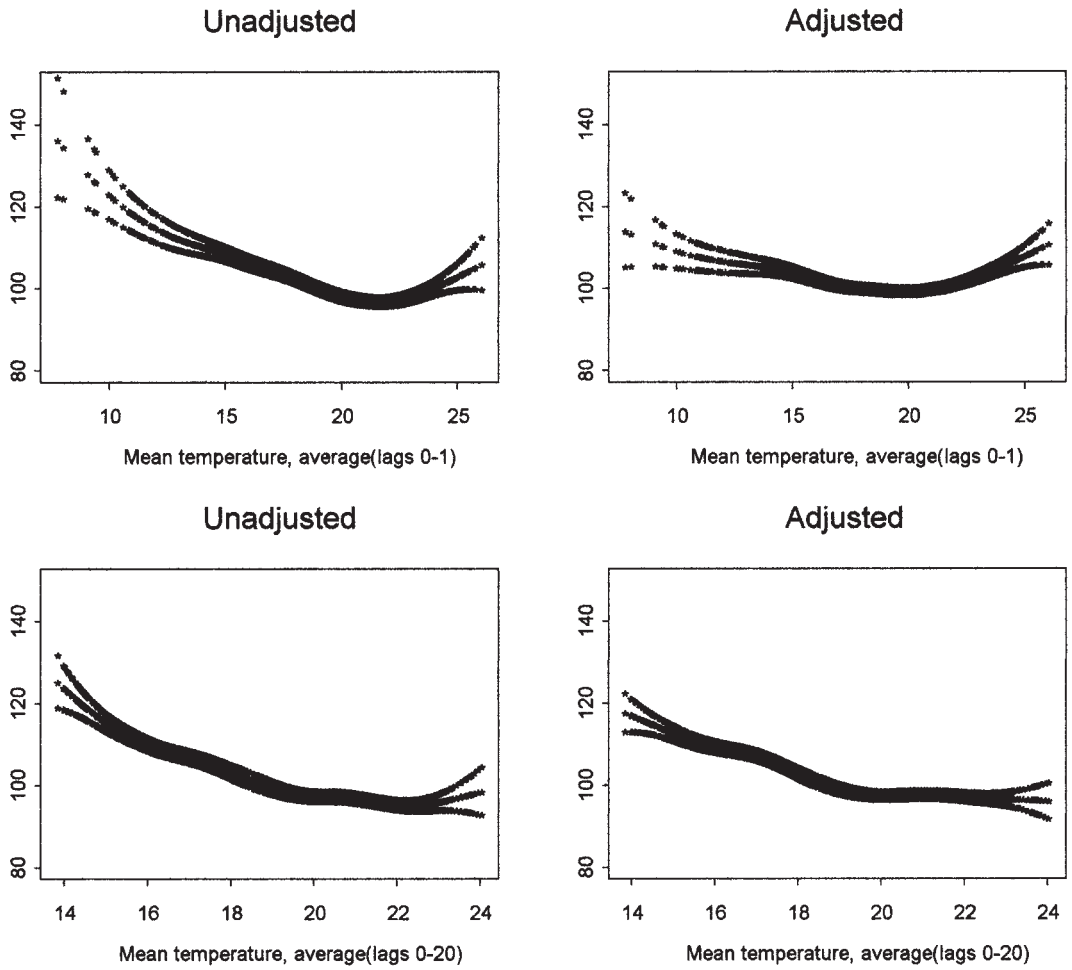


**Figure 2** Relationship of all-cause all-age mortality with mean temperature (shown as a 6 d.f. smoothing spline on both lags 0–1 and 0–20) before and after adjustment for season, day-of-week, pollution, and humidity levels. The x-axis shows the mean temperature (°C) and the y-axis represents mortality as a percentage of the average number of deaths for each group

**Table 2** Relative risks (RR) and 95% CI for heat effect of mean temperature average (lags 0–1) for a 1°C increase above 20°C, and cold effect of mean temperature weighted average (lags 0–20) for a 1°C decrease below 20°C, São Paulo, Brazil, 1991–1994

| | Heat effect | | Cold effect | |
|---|---|---|---|---|
| Mortality/Age group | RR | 95% CI | RR | 95% CI |
| **All-cause** | | | | |
| Children | 1.026 | (1.016–1.036) | 1.040 | (1.032–1.048) |
| Adults | 1.015 | (1.011–1.018) | 1.026 | (1.023–1.029) |
| Elderly | 1.025 | (1.021–1.028) | 1.055 | (1.052–1.057) |
| **Cardiovascular disease** | | | | |
| Adults | 0.998 | (0.992–1.003) | 1.026 | (1.021–1.030) |
| Elderly | 1.020 | (1.016–1.025) | 1.063 | (1.059–1.067) |
| **Respiratory disease** | | | | |
| Adults | 1.021 | (1.011–1.031) | 1.042 | (1.034–1.051) |
| Elderly | 1.023 | (1.016–1.031) | 1.064 | (1.057–1.070) |
| **Other** | | | | |
| Adults | 1.023 | (1.014–1.032) | 1.016 | (1.009–1.023) |
| Elderly | 1.029 | (1.019–1.040) | 1.030 | (1.022–1.038) |

were observed for values above and below 20°C in mean temperature. The magnitude of the effects observed for heat was broadly consistent with those found in studies conducted in Europe or America.[9,13,26,27] For an increase of 1°C in temperatures above 20°C we observed between 1.5% (adults) and 2.6% (children) increases in mortality. Increments in RR per degree below 20 (the cold effect) were higher than the effects of heat and they were also higher than previous studies in cooler climates. This is consistent with a comparison of warm and cold regions of Europe where greater effects of cold temperatures were observed for warmer countries.[18]

Temperature effects were observed for all age groups, but children and the elderly were the most affected. We could only find one study that examined the effect of temperature on mortality according to age group.[13] Although the authors did not look at children, they also found an increasing temperature effect for older people.

Some studies have described stronger effects of heat on mortality due to specific causes such as CVD and respiratory diseases.[9,26,28] In our study, these specific causes exhibited similar effect estimates to those of other and all-cause mortality in the elderly, but in adults the effect was curiously absent in deaths from CVD, but present and of similar magnitude in respiratory and other causes of death. By contrast, cause-specific analysis for cold effects revealed somewhat higher

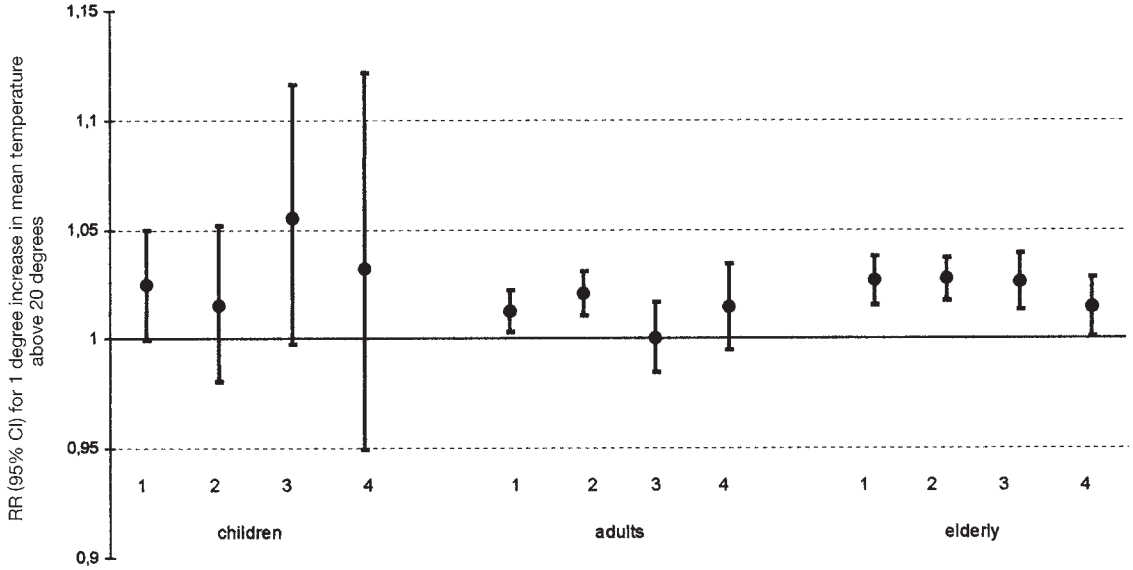


**Figure 3** Relative risk (95% CI) for all-cause mortality associated with a 1°C increase in mean temperatures above 20°C (average lags 0–1) by age group and socioeconomic position, São Paulo, Brazil, 1991–1994

Obs.: SEP groups go from 1 (the most deprived) to 4 (the most affluent)

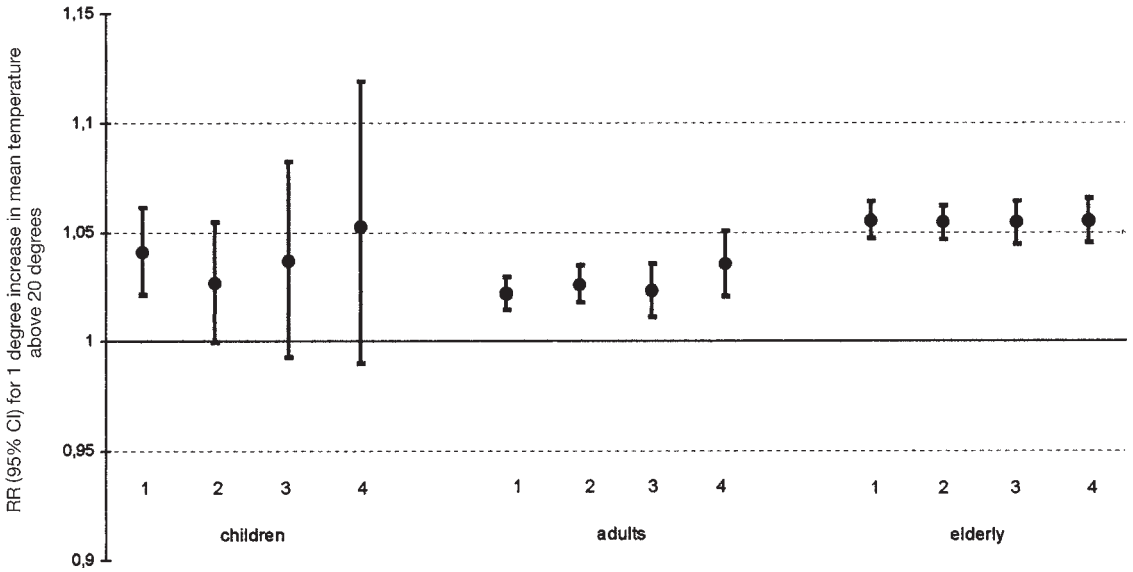


**Figure 4** Relative risk (95% CI) for all-cause mortality associated with a 1°C decrease in mean temperatures below 20°C (weighted average lags 0–20) by age group and socioeconomic position, São Paulo, Brazil, 1991–1994

Obs.: SEP groups go from 1 (the most deprived) to 4 (the most affluent)

estimates for CVD and respiratory diseases than for all-cause mortality or other causes.

We found little evidence that the effect of thermal stress on mortality was different according to the SEP of the population. Such evidence as existed was confined to the elderly, with the most deprived individuals exhibiting higher effects of heat on mortality. Because of ample evidence that mortality in general is associated with SEP, it has been argued that the seasonal pattern depends on SEP.[29] However, very few previous studies have attempted to examine the association of temperature–mortality relationships with SEP. Of these studies, all but one[13] examined the effects of cold rather than heat. Like our study, they found no evidence that measures of SEP were associated with cold- or heat-related mortality.[13,29–31] However, our study and others used *relative* risk as the measure of effect of heat and cold. If we assume (as supported by other studies) that absolute

levels of mortality are higher in those with lower SEP, an RR due to temperature that is constant across SEP groups implies a higher *absolute* effect in lower SEP groups.

An effect of SEP may have been obscured by the fact that the SEP indicator used in this study depended on each death being assigned to one of the four socioeconomic strata based on their district of residence. Such an ecological approach does not take into account the fact that districts are large and socially quite heterogeneous. A more powerful assessment of the role of socioeconomic conditions in the risk of health effects due to changes in temperature could be undertaken with individual measures of SEP, not available in this study. If temporal patterns of mortality were spatially clustered, the ecological nature of the SEP data and spatial auto-correlation between areas may also have biased standard errors downwards. However, for our largely negative results this seems an academic consideration.

Our results depend on the validity of our analytical methods, which may be obscure to those unfamiliar with air pollution epidemiology. In that field such time series methods have been widely used in recent years, although weather is considered a confounder rather than an effect of main interest. Adjustment for specific measured potential confounders—in our study relative humidity and air pollution levels—follows methods long-established in epidemiology. Allowance for other factors causing long- and medium-term temporal patterns, including seasonality, is newer, although much discussed in the air pollution literature.[32]

The method we used to control for temporal variation—fitting a smooth curve with time—is standard for air pollution epidemiology, although there is some debate as to how to choose the best level of smoothness in this curve. We followed Dominici[32] in choosing *a priori* a smoothing spline with 7 d.f. per year, on the grounds that the factors we wished to control for fluctuated over months or years, while we wished to leave fluctuation over days or weeks from which to identify immediate effects of factors varying over this time scale. Plots of residuals of daily mortality and their partial autocorrelation (PAC) function from our model showed no patterns indicative of unmodelled temporal variation. In particular the sum of the first twenty PAC was close to zero—an alternative criterion for choice of smoothing.[33] When, as a sensitivity analysis we used a less flexible curve (fewer degrees of freedom), there was clear residual seasonal variation.

Nevertheless, choice of extent of smoothing over time did influence magnitude of estimated temperature effects. For example, increases per degree above 20 in all-cause mortality in the elderly were 1.8% and 2.6% when we used 3 or 11 d.f./year, compared with the 2.5% we found with 7 d.f./year which indicates that less smoothing underestimates the heat effect. Results for cold effects were slightly less sensitive to varying degrees of temporal smoothing but still indicated under-estimation for both lower and higher degrees of smoothing. This should be considered when comparing our results with those from other studies.

Confounding by influenza epidemics, which generally occur in winter and might therefore affect the cold estimate, was not addressed in this study. Some time series mortality studies have included data on deaths from influenza as a way to control for this factor. However, at the time of this study there were no reports of influenza epidemics in São Paulo and mortality counts for influenza were extremely low during the period and did not exhibit any epidemic pattern. Furthermore, the temporal smooth curve would control for such epidemics to some extent. Thus confounding by influenza epidemics should be slight.

As a final caveat we note that this study has not distinguished between deaths brought forward by years, months, or days. It is possible that temperature extremes hasten the death of people who would in any case have died a few days or weeks later.

In conclusion, our study strengthens the argument that the associations reported in northern and cooler countries of an effect of high and low temperatures on mortality is also present in a sub-tropical city with moderate climatic conditions. The magnitude of the effects, although not large in RR terms, is of importance due to the ubiquity of the exposure and the large population exposed. Relative risks were largest in children and the elderly, but did not differ by SEP group.

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**KEY MESSAGES**

- The U-shaped pattern of temperature–mortality relationship found in European and North-American cities also occurred in a developing sub-tropical city with moderate climatic conditions.
- Increments in relative risk per degree below 20°C (the cold effect) were higher than increments per degree above 20°C (the heat effect).
- The cold effect was higher than in previous studies in cooler climates.
- Both heat and cold effects were largest for children and the elderly.
- There was little evidence for a modification of the mortality effects of cold or heat by socioeconomic status.

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# Commentary: Mortality from environmental factors, but which ones?

**WR Keatinge**

The paper by Gouveia *et al.*[1] reaches two main conclusions, that in this sub-tropical city the relationship of mortality to temperature was U-shaped, and that it did not differ significantly between high and low socioeconomic groups. The first conclusion is in line with many reports from other, usually colder, countries. The second conclusion, that the relationship did not differ between socioeconomic classes, is at first sight surprising, but it too is in line with work in other countries, at least in respect to cold-related mortality.[2,3] Regional differences in cold-related mortality depend on the protective measures taken locally against cold stress, and those in turn depend mainly on the different perceived needs for protection in different climates.[4,5] The main questions relate to methodology. This paper has been written with attention to some possible pitfalls, and to methodology used in previous work. Its conclusions are likely to be broadly correct, but since the questions apply to a great many papers in this area, they are worth close inspection.

Multiple regressions to assess the effect of environmental factors on mortality contain collinear variables that readily attribute effects of one variable incorrectly to another. If measurement error differs between two closely collinear explanatory variables, the multiple regression tends to attribute mortality change to the one that has least measurement error, rather than to one which is causing the mortality change. Two-stage regressions tend to attribute effects to variables in the first stage, and suppress effects of those in the second stage. Misspecification of the true relationship tends to transfer the apparent effect from the misspecified variable. For example, if a log-linear relationship is assumed when the real relationship is linear or exponential, the effect tends to be transferred to another variable whose crude relationship to mortality happens to be closer to log-linear. Generalized additive Poisson regression models (GAM) modelling, used in the present paper, reduces that problem by seeking the best-fit relationship for each explanatory variable, but it requires subjective judgement of the minimum degree of smoothing needed to produce a convincing relationship for each explanatory variable. This can place strain on the judgement of a researcher who finds that a particular degree of smoothing will generate the result he is seeking, while a neighbouring degree of smoothing will not.

Such problems are well documented[6–8] but are frequently ignored. The existence of numerous prior papers that used similar methods is of little help in establishing validity unless those papers include a rigorous search for possible errors in their approach. Current targeting of large-scale funding on chosen research topics tends to generate numerous papers over a short period, all using a similar approach and generating the same systematic error. Substantial short-term mortality has, for example, frequently been attributed in the past to pollution by sulphur dioxide ($SO_2$), but $SO_2$ is absorbed in the upper airways and does not reach the lungs in measurable amounts.[9] The apparent mortality ascribed to it seems to have been due to collinear factors, such as unusual patterns of cold weather associated with air pollution.[10]

There is no absolute solution to these problems. This makes it important for authors, editors, and referees routinely to make a systematic search for any factors that could have spuriously generated the relationship described, and to assess frankly any such possibilities of error that they cannot eliminate. Application of a few simple principles can greatly facilitate this.

The reader must be able to see exactly what was done. This paper states that it used standard time series methods that have been developed for air pollution studies. Since the precise approach used in such papers has varied enormously, it is hard to see exactly what was done in this case. The paper states, for example, that daily values of a wide variety of pollutants were obtained, but it does not say whether all were included in all mortality analyses, whether any or all of pollutants appeared to have significant effects, and whether any that did not were dropped from the analysis.

It can also be extremely helpful to show simple relationships between key variables as well as residual relationships after processing. The more complex the processing, the more numerous the distortions it can produce and the more difficult they are to exclude. This paper does give 'unadjusted' mortality/temperature plots for pooled population mortality, as well as processed ones. Assuming that the former were not subjected to any form of processing, these plots greatly strengthen confidence in the first conclusion, that there was a U-shaped relationship between these variables. No such mortality/temperature plots are presented for the different socioeconomic groups. These would be extremely helpful in assessing the second conclusion in the paper. They might confirm the conclusion of the complex analyses that the relationship was similar in all the groups. Without them, the possibility remains that there were real differences in the mortality/temperature relationship between different socioeconomic groups, and that the subsequent analysis falsely attributed these to some other variable, such as pollution. That could happen if people in the low socioeconomic groups lived in areas of relatively high pollution, which is likely, and if such pollution increased in cold weather, as it usually does elsewhere.

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