Mortality Displacement of Heat-Related Deaths: A Comparison of Delhi, São Paulo, and London

Author(s): Shakoor Hajat, Ben G. Armstrong, Nelson Gouveia and Paul Wilkinson

Source: *Epidemiology*, Vol. 16, No. 5 (Sep., 2005), pp. 613-620
Published by: Lippincott Williams & Wilkins
Stable URL: http://www.jstor.org/stable/20486112
Accessed: 10-08-2017 13:03 UTC

Mortality Displacement of Heat-Related Deaths

A Comparison of Delhi, São Paulo, and London

Shakoor Hajat, Ben G. Armstrong,* Nelson Gouveia,† and Paul Wilkinson**

Background: Mortality increases with hot weather, although the extent to which lives are shortened is rarely quantified. We compare the extent to which short-term mortality displacement can explain heat deaths in Delhi, São Paulo, and London given contrasting demographic and health profiles.

Methods: We examined time-series of daily mortality data in relation to daily ambient temperature using Poisson models and adjusting for season, relative humidity, rainfall, particulate air pollution, day of the week, and public holidays. We used unconstrained distributed lag models to identify the extent to which heat-related excesses were followed by deficits (mortality displacement).

Results: For each city, an increase in all-cause mortality was observed with same-day (lag 0) and previous day (lag 1) temperatures greater than a threshold of 20°C. At lag 0, the excess risk was greatest in Delhi and smallest in London. In Delhi, an excess was apparent up to 3 weeks after exposure, after which a deficit was observed that offset just part of the overall excess. In London, the heat excess persisted only 2 days and was followed by deficits, such that the sum of effects was 0 by day 11. The pattern in São Paulo was intermediate between these. The risk summed over the course of 28 days was 2.4% (95% confidence interval = 0.1 to 4.7%) per degree greater than the heat threshold in Delhi, 0.8% (−0.4 to 2.1%) in São Paulo and −1.6% (−3.4 to 0.3%) in London. Excess risks were sustained up to 4 weeks for respiratory deaths in São Paulo and London and for children in Delhi.

Conclusions: Heat-related short-term mortality displacement was high in London but less in Delhi, where infectious and childhood mortality still predominate.

(*Epidemiology* 2005;16: 613–620)

Submitted 12 March 2004; final version accepted 23 February 2005.

From the *London School of Hygiene & Tropical Medicine, United Kingdom; and †Universidade de São Paulo, Brazil.

Supported by a grant from the UK Medical Research Council, Mortality impact of heatwaves in low- and middle-income countries, G9900947. Paul Wilkinson is supported by a Public Health Career Scientist Award (NHS Executive, CCB/BS/PHCS031).

Correspondence: Shakoor Hajat, Public & Environmental Health Research Unit, London School of Hygiene & Tropical Medicine, Keppel Street, London WC1E 7HT, UK. E-mail: shakoor.hajat@lshtm.ac.uk.

ISSN: 1044-3983/05/1605-0613
DOI: 10.1097/01.ede.0000164559.41092.2a

Time-series studies have consistently shown evidence of increased mortality in association with hot weather (ISOTHURM Study Group, unpublished data).[1–10] However, for reasons well-described in the context of studies of air pollution and daily mortality,[11–13] past studies have not quantified the extent to which lives are shortened (displaced). This omission has obvious public health significance: most observers would consider shortening of life by days or weeks as far less important than shortening by months or years. Thus, the question is whether people susceptible to heat effects are those with advanced forms of illness (largely cardiorespiratory) who would die anyway within a short period. For these people, the added stress of heat may be merely the final physiological insult that precipitates death.

Some evidence indeed exists for such short-term displacement ("harvesting").[14] Studies of mainly high-income populations have shown apparent reductions in mortality at lags of more than a few days after a hot day, which is the pattern expected if a finite pool of highly susceptible individuals is depleted by the initial heat stress.[15–17] Harvesting was estimated to account for about half of the total number of deaths during the 1994 heatwaves in the Czech Republic.[18]

It would, however, be simplistic to assume a single pattern of mortality displacement for all populations. Theoretical considerations suggest a dependence on the epidemiologic profile of the population at risk, with variation by cause-of-death and age. It would seem unlikely, for example, that very short-term displacement could account for all temperature-related diarrheal deaths in children, many of whom might otherwise live to adulthood; or for all deaths from acute thrombotic events (heart attacks, strokes) at older ages, given that many thrombosis-related deaths occur in people without a clinical history of cardio-respiratory symptoms. Thus, it would be surprising to see the same mortality patterns in high-income countries, where deaths are dominated by chronic diseases in the elderly, as in low-income countries where a high proportion of deaths are at younger ages and where infections and acute respiratory disease are common. We undertook time-series analyses of daily mortality data to determine the extent to which short-term mortality displace-

ment can explain heat-related deaths in 3 cities of contrasting demographic and health profiles.

METHODS

The 3 study cities were Delhi (India), São Paulo (Brazil), and London (England). For each city, we obtained daily counts of all-cause mortality excluding violent deaths (*International Classification of Diseases*, Ninth Revision ICD-9), as well as counts by age- and cause-of-death group, during a 4-year period (January 1991 to December 1994) from mortality registries. Mortality data for Delhi were supplied by the World Bank, which had previously assembled them for studies of air pollution and health. The Delhi data are for 1 of 3 districts in the National Capital Territory and include approximately 25% of the city's total deaths. The age-group categories were children (<15 years), adults(15–64 years), and elderly (≥65 years). The separate cause-of-death groups were cardiovascular (ICD-9: 390–459), respiratory (ICD-9: 460–519), and other nonviolent deaths.

Meteorological and Air Pollution Variables

We obtained daily maximum and minimum temperature, relative humidity, and precipitation for the study period in each of the 3 locations. Daily average temperature was computed as the mean of the daily maximum and minimum value. Daily mean concentrations of particulate pollution—PM_{10}, or total suspended particles (TSP) in the case of Delhi—also were obtained for each city. Missing data for pollution were imputed using linear regression models. More information on site locations and handling of missing data are provided elsewhere (ISOTHURM Study Group, unpublished data).[19]

Statistical Methods

For each city, we examined daily mortality in relation to hot weather using Poisson generalized linear models, allowing for over-dispersion.[20] Daily levels of humidity and air pollution and weekly averages of rainfall up to 2 weeks before the day of death were incorporated into each regression model as possible confounding variables, regardless of statistical significance. Weekly reports of laboratory-based influenza A activity also were adjusted for in the case of London. We used indicator variables to allow for any day-of-week or public-holiday effects. Cubic smoothing splines of time with equally spaced knots were used to control for secular trends (eg, demographic shifts) and any additional confounding by seasonally-varying factors other than temperature. Seven degrees of freedom (*df*) per year for these smoothing splines (roughly equivalent to a 2-month moving average) were used.[21] This number of degrees of freedom was chosen as a compromise between providing adequate control for unmeasured confounders and leaving sufficient information from which to estimate temperature effects. Plots of partial autocorrelation suggested that this left approximately uncorrelated residuals. The same confounder control (including the same amount of seasonal control) was conducted on each series, although sensitivity of the estimates to the degree of seasonal control was also examined.

To quantify any effect of heat on death, we assumed simple linear threshold models, that is, models that assume a log-linear increase in risk above a heat threshold. Also, all models adjusted for cold effects by assuming a log-linear increase in risk below a cold threshold. Cold variables thus constructed were summed over lags 0 to 13 days (based on previous work; ISOTHURM Study Group, unpublished data). In exploratory analyses, we calculated model likelihoods for each city over a grid of all possible integer-values for heat and cold thresholds, and obtained maximum-likelihood estimates and likelihood-profile confidence intervals for thresholds. The values for both the heat and the cold threshold common to all cities that maximized the combined likelihood were then assumed in all models. These common thresholds were 20°C for the heat effect and also 20°C for the cold, both of which fell within the individual likelihood profile confidence intervals for each city. This approach allowed focus on a single parameter, the heat slope, in comparisons between cities.

To examine the pattern of mortality displacement, we adopted a modification of the method described by Schwartz, using distributed lag models.[22] These models simultaneously fit multiple lags of the exposure variable:

$$\log(E(Y_i) = \beta_0 Z_i + \beta_1 Z_{i-1} + \cdots \beta_k Z_{i-k} + \text{ns(time,7}df\text{/year)} + \text{other covariates}$$

where Y_i is the mortality count on day i, Z_i is the heat exposure on the same day (lag 0), Z_{i-1} on the previous day (lag 1), etc. This approach has been primarily used to model adverse effects spread over several days, with different potency of effect at different lags, but it has also been used to detect presence of short term displacement of mortality by temperature effects[14] and air pollution.[23] Displacement of deaths by k days or less is characterized by positive coefficients of exposures at lower lags (an excess due to exposure) and negative coefficients at higher lags (a deficit due to the deaths that have been displaced forward). Net effects up to k days (ie, discounting displacement of k days or less) was estimated by summing coefficients $\beta_0 - \beta_k$.[13,24–26] We found this approach to be a transparent way of quantifying effects of mortality displacement.

We placed no constraints on the coefficients (β_0, $\beta_1, \ldots, \beta_k$) but for plots of independent effects at different lags, we summed coefficients over lags 4–5, 6–8, 9–13, 14–21, and 22–34 (lags 0–3 plotted separately). This grouping, based on the Fibonacci series, was chosen to allow distinction of effects at short lags, but to reduce standard

errors in the otherwise imprecisely estimated longer lag effects where sharp differences are unlikely. We did not consider models with lags beyond 34 days ($k = 34$), because estimates became imprecise, presumably due to colinearity with the time smooth.

RESULTS

The 3 study cities have approximately similar size populations but, as Table 1 shows, there is wide contrast among them in wealth (gross national income per capita) and in the causes of deaths. During the period of analysis, a high proportion of Delhi deaths (48%) occurred in children younger than 15 years. This incidence was much greater than the proportion in São Paulo (10%), which in turn was substantially greater than the proportion in London (1%). This difference also was reflected in the causes of death. More than half of all deaths in London and São Paulo were from cardiorespiratory causes, whereas in Delhi the proportion was one-quarter. The category of "other causes" (which includes deaths from infectious disease) was the main category of cause-of-death for Delhi. The cities also varied in their climates, with Delhi having the highest mean temperatures, and London the lowest.

All-Cause Mortality

For each city, all-cause mortality increased with same-day temperatures greater than the heat threshold of 20°C (Table 2). The highest point estimate at lag zero was observed in Delhi, followed by São Paulo, and then London. Figure 1 shows the cumulative excess risks (with deficits deducted) for all individual days from zero lag up to the lag shown on the x-axis, eg, the excess risk for lag 4 is formed from the addition of the excess risks for day 0, day 1, day 2, day 3, and day 4, with all terms fitted simultaneously in the model. In Delhi, the estimate of the summed excess risk per degree above the threshold temperature continued to increase with longer periods of summation up to approximately 3 weeks of lag. Thereafter, the summed excess risk showed evidence of decline.

The independent excess risks for other specified periods of lag are shown in Figure 2. Excess risk for each lag range is plotted against the midpoint of the range. In Figure 2, the negative excess risk in Delhi after the third week of lag is clearly shown, explaining the attenuation of the summed excess after lag 21 of Figure 1.

The profile of excess risk in São Paulo was flatter than for Delhi, and there was some evidence of a dip around week 3 (Fig. 1). Point estimates of net effects consistently remained above zero and included a small increase at day 28, although the lower confidence interval included zero from the third week.

In London, there was a much steeper decline in summed excess risk, driven by negative point estimate excess

TABLE 1. City Characteristics

	Delhi	São Paulo	London
Approximate population (millions)	9.9	9.7	7.4
Gross National Income per capita (2001/US$)*	470	3060	25,120
Mean temperature (°C)			
Mean (5th to 95th percentile)	25 (14–35)	20 (14–25)	12 (3–22)
Relative humidity (%)			
Mean (5th to 95th percentile)	76 (56–93)	78 (59–92)	79 (59–96)
Particulate concentrations ($\mu g.m^{-3}$)			
Mean (5th to 95th percentile)	TSP: 375 (220–568)	PM_{10}: 65 (32–129)	PM_{10}: 11 (5–23)
Daily number of deaths			
Mean (5th to 95th percentile)	25† (14–37)	170 (137–208)	177 (142–223)
Age at death (years), %			
0–14	48	10	1
15–64	39	42	19
65+	13	48	80
Cause of death; %			
Cardiovascular	16	38	42
Respiratory	9	14	15
Other	76	48	43

*Source: World Bank. http://www.worldbank.org/data/countrydata/countrydata.html.
†Deaths for Delhi are for one of 3 districts in the National Capitol Territory and include approximately 25% of the total for the city.
TSP, total suspended particles.

TABLE 2. Percent Change in Deaths (95% Confidence Interval) per Degree Celcius Increase in Temperature Greater Than 20°C: Excess Risks Summed Over Varying Time Lags*

	Lag 0	Lag 0 to 1 Week	Lag 0 to 4 Weeks
Delhi			
Cause			
All causes	2.2 (1.3 to 3.2)	3.2 (1.8 to 4.5)	2.4 (0.1 to 4.7)
Cardiovascular	1.9 (−0.3 to 4.2)	4.3 (1.1 to 7.6)	4.6 (−0.9 to 10.4)
Respiratory	3.7 (0.5 to 7.0)	4.5 (0.0 to 9.2)	−1.2 (−8.4 to 6.5)
Other	2.1 (1.0 to 3.2)	2.8 (1.3 to 4.3)	2.4 (−0.1 to 4.9)
Age (years)			
0 to 14	3.2 (1.8 to 4.5)	4.2 (2.3 to 6.1)	3.9 (0.8 to 7.1)
15 to 64	1.0 (−0.5 to 2.4)	1.6 (−0.4 to 3.6)	2.0 (−1.4 to 5.6)
65+	2.5 (0.0 to 5.0)	4.0 (0.5 to 7.6)	−1.3 (−6.9 to 4.7)
São Paulo			
Cause			
All causes	1.6 (1.2 to 2.0)	1.4 (0.8 to 2.0)	0.8 (−0.4 to 2.1)
Cardiovascular	1.2 (0.6 to 1.8)	0.4 (−0.6 to 1.4)	−1.1 (−3.1 to 0.9)
Respiratory	1.9 (0.9 to 3.0)	3.8 (2.1 to 5.5)	4.0 (0.6 to 7.5)
Other	1.8 (1.2 to 2.3)	1.5 (0.6 to 2.4)	1.6 (−0.2 to 3.5)
Age (years)			
0 to 14	0.6 (−0.5 to 1.7)	1.1 (−0.7 to 2.9)	0.0 (−3.4 to 3.6)
15 to 64	1.5 (0.9 to 2.1)	0.9 (−0.0 to 1.8)	0.7 (−1.1 to 2.6)
65+	1.9 (1.3 to 2.4)	2.0 (1.1 to 2.8)	1.1 (−0.7 to 2.9)
London			
Cause			
All causes	1.4 (0.8 to 2.0)	0.9 (−0.2 to 2.0)	−1.6 (−3.4 to 0.3)
Cardiovascular	0.9 (0.0 to 1.8)	0.5 (−1.1 to 2.1)	−2.8 (−5.5 to −0.1)
Respiratory	2.4 (0.8 to 4.0)	5.6 (2.6 to 8.6)	5.9 (1.0 to 11.0)
Other	1.6 (0.7 to 2.5)	−0.1 (−1.6 to 1.4)	−3.0 (−5.6 to −0.3)
Age (years)			
0 to 14	−2.2 (−6.7 to 2.6)	−3.0 (−11.0 to 5.3)	−6.4 (−19.1 to 8.2)
15 to 64	0.8 (−0.5 to 2.1)	1.3 (−1.0 to 3.6)	−0.2 (−4.1 to 3.8)
65+	1.7 (1.0 to 2.3)	0.9 (−0.3 to 2.1)	−1.7 (−3.7 to 0.4)

*The summation is based on an unconstrained distributed lag model. The second column of results includes the summation of individual daily excess risks for every day from 0- to 7-day lag (terms fitted simultaneously); the third column of results is the summation of excess daily risks for every day from 0- to 28-day lag.

risks after 3 days (Fig. 2). The negative excesses after day 3 reduced lower confidence intervals of summed excess to zero by 6 days, and reduced the point estimate to zero by 11 days (Fig. 1). The excess risk summed over 28 days was 2.4% (95% confidence interval = 0.1 to 4.7%) per degree above the heat threshold in Delhi, 0.8% (−0.4 to 2.1%) in São Paulo and −1.6% (−3.4 to 0.3%) in London.

Results by Cause and Age

Results by cause and age are shown in Table 2 and Figures 3 and 4. In Delhi, the pattern of excess risk for cardiovascular death was fairly well sustained beyond 3 weeks, whereas the patterns for São Paulo and London showed evidence of a decline after a lag of a few days. For respiratory deaths the estimates were less precise, but the summed excess risks were sustained up to 4 weeks in both São Paulo and London, whereas the risk appeared to attenuate after 3 to 4 weeks in Delhi, although even here the results are compatible with a continued excess at day 28. For other deaths, both Delhi and São Paulo showed evidence of continued excess risk through most of the lag period, whereas in London the risk of death appeared to fall quickly after a few days, as it did for cardiovascular disease.

As for age-specific results (Fig. 4), the evidence for sustained excess risk in Delhi was greatest in children. In

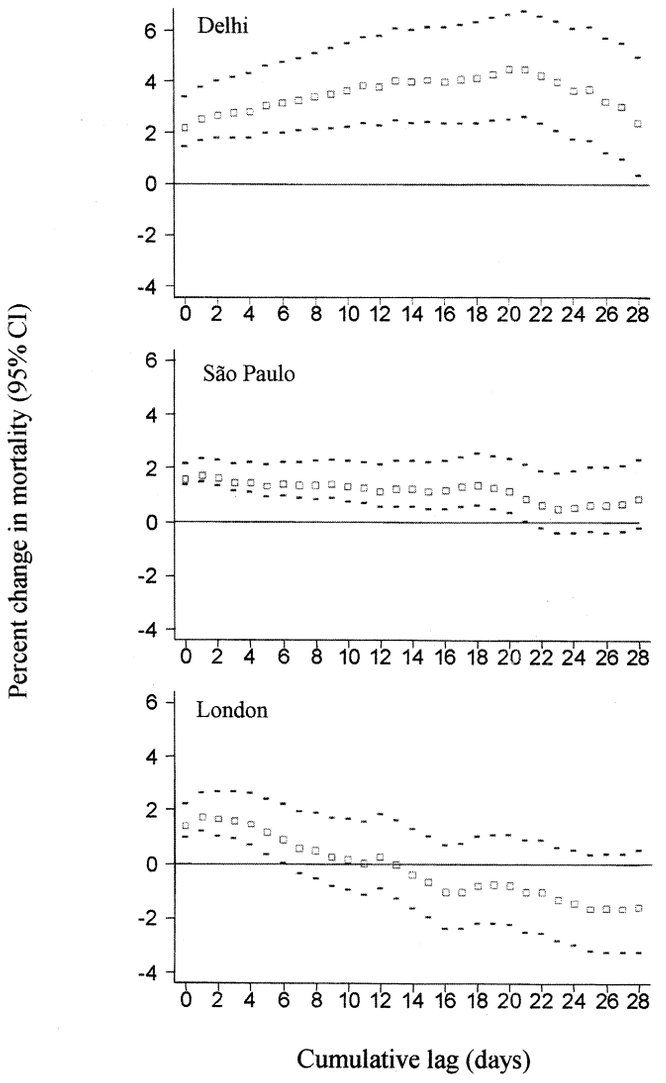

FIGURE 1. Excess risk (percent change in mortality per degree above the heat threshold of 20°C) summed over periods of increasing lag (unconstrained distributed lag models): all-cause mortality.

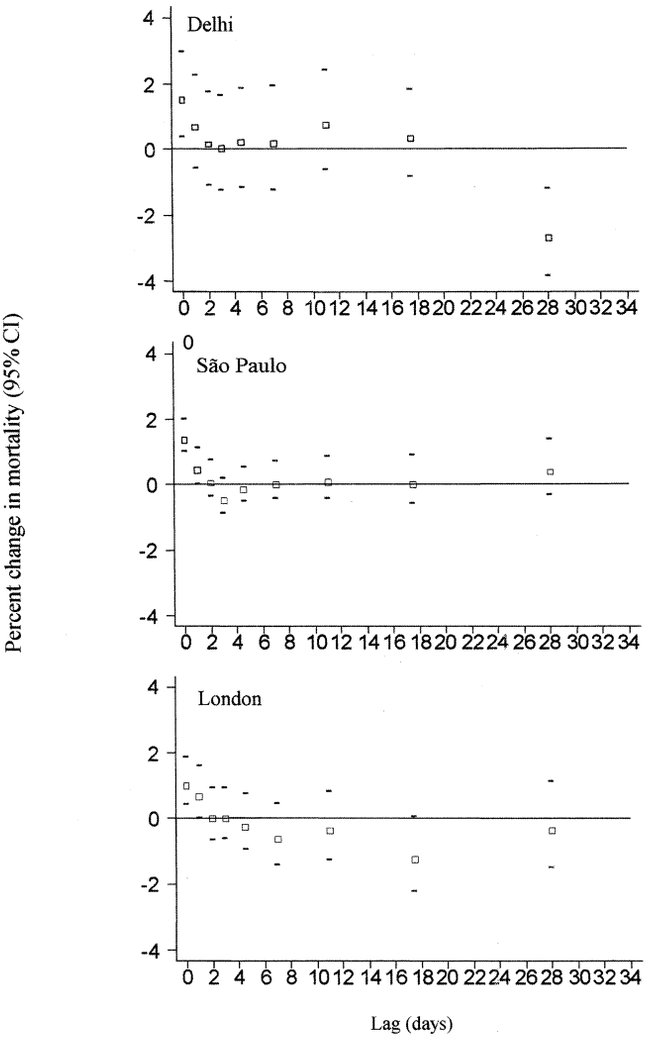

FIGURE 2. Excess risk of all-cause mortality (percent change in mortality per degree above the heat threshold of 20°C) for varying periods of summation of daily risks.

London, the results for children were too imprecise to interpret, whereas in the elderly the initial excess risk appeared to decline from a few days. The picture for São Paulo was somewhat mixed, but the excess risk among the elderly appeared to be the longest sustained of the 3 cities.

All analyses were repeated with varying levels of control for season (degrees of freedom per year) to examine any sensitivity to this modeling parameter. The results were broadly similar to those shown when more degrees of freedom (14/year) were used, but fairly different when fewer degrees of freedom were used (3.5/year). Details are available on request from the authors.

DISCUSSION

This article provides evidence that patterns of mortality displacement for heat-related deaths depend on the population at risk. We compared 3 cities with very different profiles of deaths, reflecting their stages within the demographic and epidemiologic transitions. Delhi, with the poorest population, had by far the highest proportion of deaths among children, many of whom we can assume were from infectious causes, including diarrheal deaths and acute respiratory disease. The population of London was the oldest and richest, and a high proportion of deaths were from chronic disease, specifically from cardio-respiratory causes. The profile for São Paulo was intermediate between Delhi and London.

Given these differences, we expected at the outset that heat-related deaths in London would be dominated by deaths

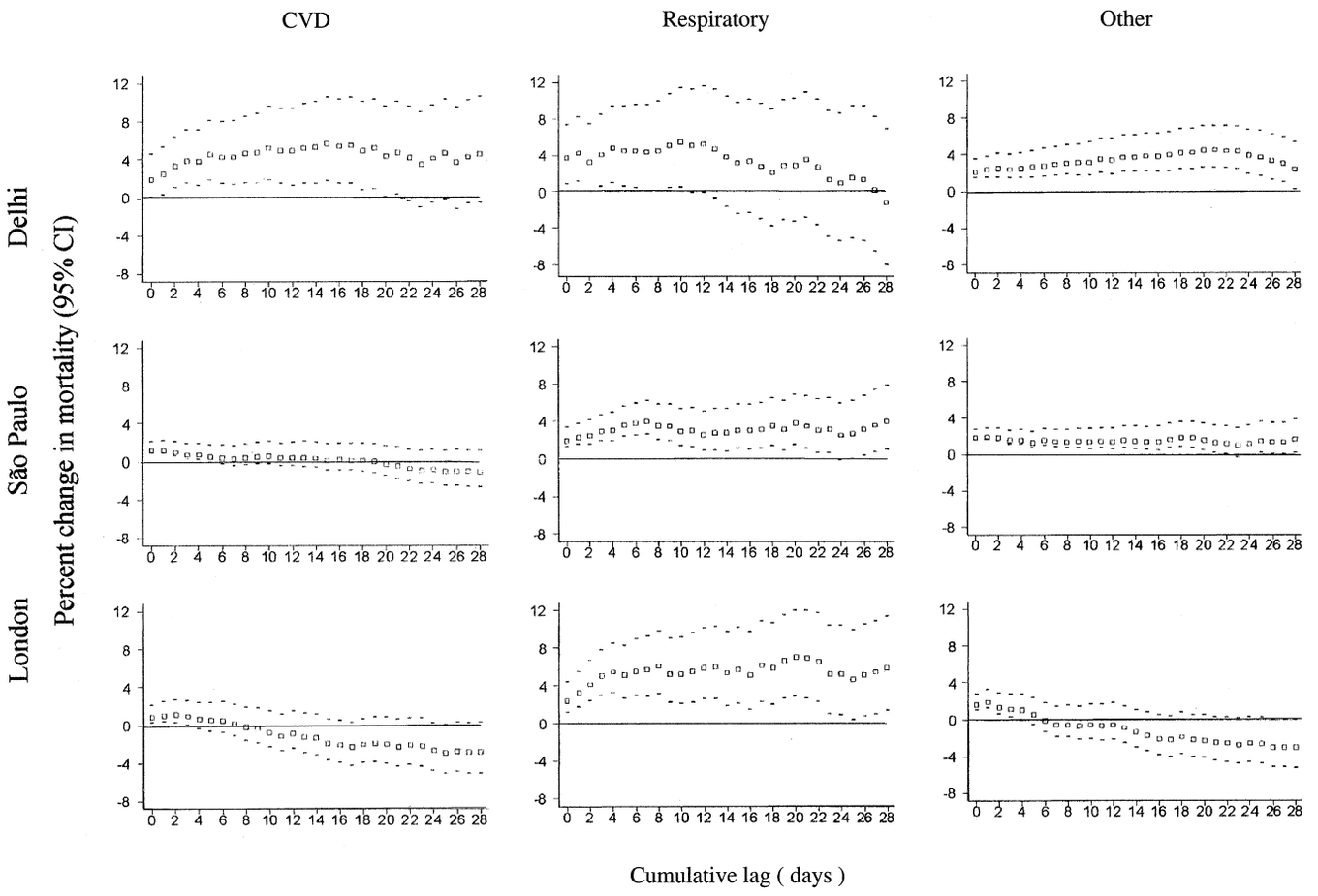

FIGURE 3. Excess risk (percent change in mortality per degree above the heat threshold of 20°C) summed over periods of increasing lag, by cause of death. Unconstrained distributed lag models.

in the elderly in persons already weakened by chronic diseases and that these would show the greatest degree of short-term mortality displacement. This hypothesis does indeed appear to be the case. The summed heat-associated excess was positive only for a few days after the heat exposure, and then was attenuated by compensatory low risks at longer lags. This is in agreement with results from studies conducted in the United States.[14,16]

However, Delhi had an excess risk for all-cause mortality that was higher than for London or São Paulo and that remained high at comparatively long lags. The estimate of the average risk at lags between 21 and 34 days was below zero, suggesting some compensatory risk reduction consistent with a harvesting phenomenon. Nonetheless, the level of risk remained highest for longest in Delhi.

The pattern for São Paulo, which has population features intermediate between those of London and Delhi, also had an intermediate pattern of mortality displacement: there is evidence of a dip after the initial excess risk but some persistence over longer time lags. The slight increase in risk at the 28-day lag measure in São Paulo is somewhat surprising, although it is not seen in specific cause or age groups and may easily be due to random fluctuation. In both London and São Paulo, risks were most sustained in the respiratory group. This is in keeping with what was observed by Goodman et al.[16] In Delhi, risks seemed most sustained in children and for nonrespiratory deaths.

We made an a priori decision to control for season using smoothing splines with 7 degrees of freedom per year in each of our models. The partial autocorrelations of residuals from our main models were free from systematic patterns and summed close to zero –an alternative criterion for optimum smoothing.[20] When, as a sensitivity analysis, we used a less flexible curve (halving the degrees of freedom) there was clear residual seasonal variation. However, doubling the seasonal control greatly increased our imprecision but without introducing bias into our estimates. This suggested our original choice of smoothing had adequately adjusted for seasonal trends.

Our results suggest that populations in low-income countries where life-threatening infections are still common

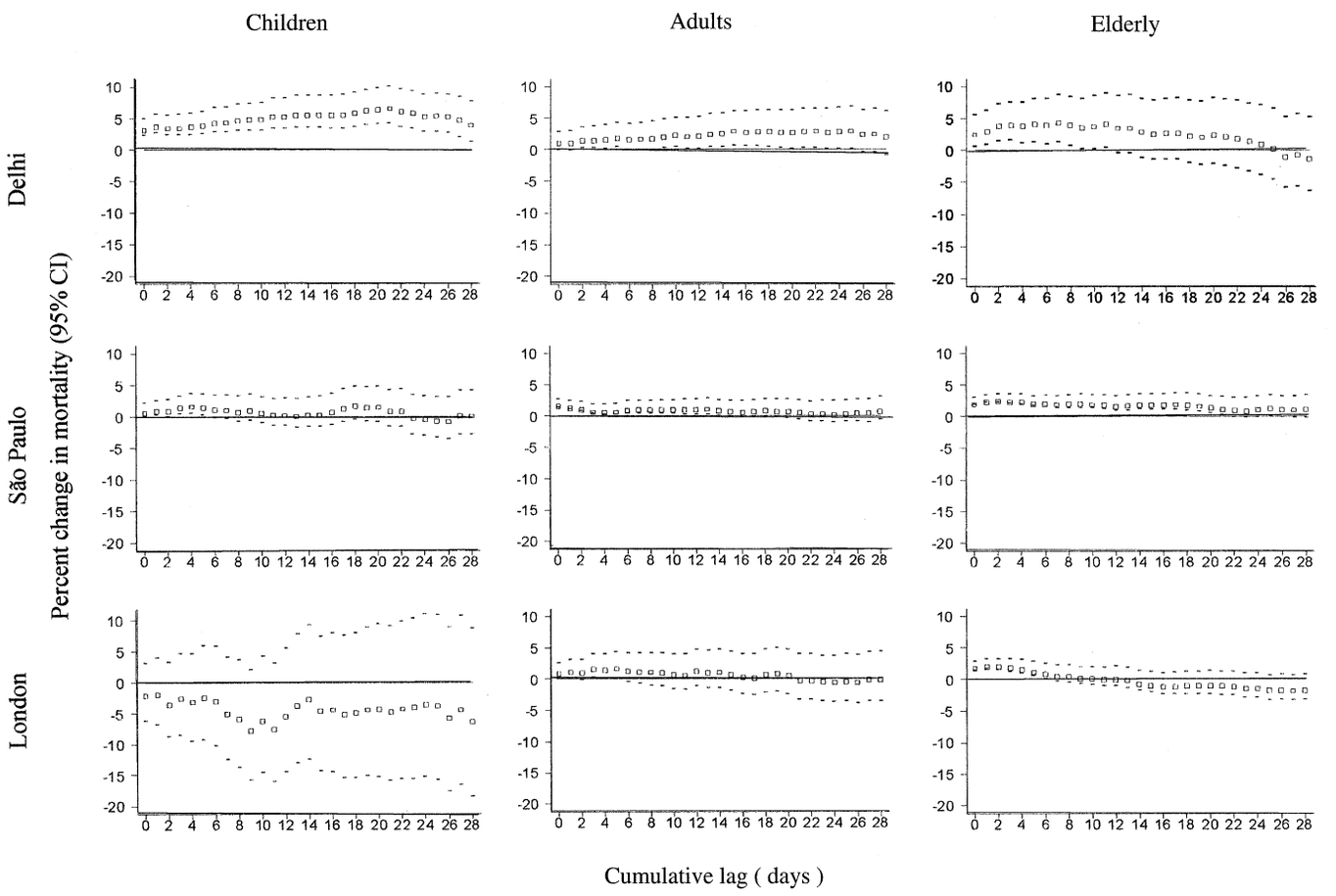

FIGURE 4. Excess risk (percent change in mortality per degree above the heat threshold of 20°C) summed over periods of increasing lag, by age-group (age 0–14 years, children; 15–64 years, adults; 65+ years, elderly). Unconstrained distributed lag models.

may have the greatest vulnerability to the effects of heat and global warming. In these populations, increases in mortality at high temperatures probably represent substantial shortening of life. Although our analyses can not establish the degree of life-shortening beyond a few weeks, the fact that deaths in children younger than 15 years of age make a large contribution to heat-related mortality in Delhi suggests that high temperatures may be leading to the deaths of many children who might reasonably have expected decades of future life had it not been for the fatal outcome of the heat-related illness. Some studies have suggested people may adapt to warmer weather,[27,28] Even so, those most susceptible to heat are likely to remain susceptible if there is not due attention paid to infectious disease, diarrheal illness, and other major causes of early mortality in these poor populations.

REFERENCES

1. Semenza JC, Rubin CH, Falter KH, et al. Heat-related deaths during the July 1995 heat wave in Chicago. *N Engl J Med.* 1996;335:84–90.
2. Curriero FC, Heiner KS, Samet JM, et al. Temperature and mortality in 11 cities of the eastern United States. *Am J Epidemiol.* 2002;155:80–87.
3. Curriero FC, Samet JM, Zeger SL. Re: "On the use of generalized additive models in time-series studies of air pollution and health" and "Temperature and mortality in 11 cities of the eastern United States." *Am J Epidemiol.* 2003;158:93–94.
4. Keatinge WR, Donaldson GC, Cordioli E, et al. Heat related mortality in warm and cold regions of Europe: observational study. *BMJ.* 2000;321:670–673.
5. Huynen MM, Martens P, Schram D, et al. The impact of heat waves and cold spells on mortality rates in the Dutch population. *Environ Health Perspect.* 2001;109:463–470.
6. Hajat S, Kovats R, Atkinson R, et al. Impact of hot temperatures on death in London: a time series approach. *J Epidemiol Community Health.* 2002;56:367–372.
7. Rooney C, McMichael AJ, Kovats RS, et al. Excess mortality in England and Wales, and in Greater London, during the 1995 heatwave. *J Epidemiol Community Health.* 1998;52:482–486.
8. Díaz J, Jordán A, García R, et al. Heat waves in Madrid 1986–1997: effects on the health of the elderly. *Internatl J Occup Environ Health.* 2002;75:163–170.
9. Saez M, Sunyer J, Castellsague J, et al. Relationship between weather temperature and mortality: a time series analysis approach in Barcelona. *Int J Epidemiol.* 1995;24:576–582.
10. Giles BD, Balafoutis C, Maheras P. Too hot for comfort: the heatwaves in Greece in 1987 and 1988. *Int J Biometeorol.* 1990;34:98–104.

11. McMichael AJ, Anderson HR, Brunekreef B, et al. Inappropriate use of daily mortality analyses to estimate longer-term mortality effects of air pollution. *Int J Epidemiol*. 1998;27:450–453.
12. Burnett RT, Dewanji A, Dominici F, et al. On the relationship between time-series studies, dynamic population studies, and estimating loss of life due to short-term exposure to environmental risks. *Environ Health Perspect*. 2003;111:1170–1174.
12a. Zeger SL, Dominici F, Samet J. Harvesting-resistant estimates of air pollution effects on mortality. *Epidemiology*. 1999;10:171–175.
13. Braga AL, Zanobetti A, Schwartz J. The time course of weather-related deaths. *Epidemiology*. 2001;12:662–667.
14. Pattenden S, Nikiforov B, Armstrong BG. Mortality and temperature in Sofia and London. *J Epidemiol Community Health*. 2003;57:628–633.
15. Braga AL, Zanobetti A, Schwartz J. The effect of weather on respiratory and cardiovascular deaths in 12 U.S. cities. *Environ Health Perspect*. 2002;110:859–863.
16. Goodman PG, Dockery DW, Clancy L. Cause-specific mortality and the extended effects of particulate pollution and temperature exposure. *Environ Health Perspect*. 2004;112:179–185.
17. Kysely J, Kriz B. Vysoke letni teploty a umrtnost v CR v letech 1982–2000. (High summer temperatures and mortality in the Czech Republic 1982–2000). *Epidemiol Mikrobiol Imunol*. 2003;52:105–116.
18. Gouveia N, Fletcher T. Time series analysis of air pollution and mortality: effects by cause, age and socioeconomic status. *J Epidemiol Community Health*. 2000;55:750–755.
19. Schwartz J, Spix C, Touloumi G, et al. Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J Epidemiol Community Health* 1996;50(Suppl 1):S3–S11.
20. Dominici F, Samet JM, Zeger SL. Combining evidence on air pollution and daily mortality from the twenty largest US cities: a hierarchical modelling strategy (with discussion). *J R Stat Soc Ser A*. 2000;163:263–302.
21. Schwartz J. The distributed lag between air pollution and daily deaths. *Epidemiology*. 2000;11:320–326.
22. Zanobetti A, Schwartz J, Samoli E, et al. The temporal pattern of mortality responses to air pollution: a multicity assessment of mortality displacement. *Epidemiology*. 2002;13:87–93.
23. Kelsall JE, Samet JM, Zeger SL, et al. Air pollution and mortality in Philadelphia, 1974–1988. *Am J Epidemiol*. 1997;146:750–762.
24. Deleted in proof.
25. Schwartz J. Harvesting and long term exposure effects in the relation between air pollution and mortality. *Am J Epidemiol*. 2000;151:440–448.
26. Schwartz J. Is there harvesting in the association of airborne particles with daily deaths and hospital admissions? *Epidemiology*. 2001;12:55–61.
27. Davis RE, Knappenberger PC, Michaels PJ, et al. Changing heat-related mortality in the United States. *Environ Health Perspect*. 2003;111:1712–1718.
28. McGeehin MA, Mirabelli M. The potential impacts of climate variability and change on temperature-related morbidity and mortality in the United States. *Environ Health Perspect*. 2001;109(Suppl 2):185–189.