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Effects of Temperature and Ozone on Daily Mortality During the August 2003 Heat Wave in France

Keith Dear, PhD; Geetha Ranmuthugala, PhD; Tord Kjellström, PhD;
Carol Skinner, BSC, DipMet; Ivan Hanigan, BA (Hons)

ABSTRACT. During the summer of 2003, Europe experienced a heat wave that lasted almost 2 weeks in which high temperatures were accompanied by exceptionally high ozone levels. Unfortunately, few studies have examined the effects of temperature and ozone simultaneously. The authors use constrained distributed lag models to estimate the effects of daily temperature and peak ozone on mortality in 12 French cities during the heat wave and to estimate the deaths attributable to each component. Elevated minimum and maximum temperature and peak ozone all increase mortality, with substantial interaction effects between temperature and ozone. Researchers expect extreme weather events, along with elevated ozone levels and temperatures, to become more common. Our results suggest that ozone will be an important contributor to the adverse health effects of such events.

KEY WORDS: air pollution, epidemiology, exposure assessment, mortality, ozone

In August 2003, Europe experienced a summer heat wave lasting almost 2 weeks. The high temperatures were accompanied by "exceptionally long-lasting and spatially extensive episodes of high ozone concentrations."[1] France experienced the greatest impact in terms of mortality, with more than 14,800 excess deaths (a 60% increase) reported for the period between August 1 and August 20.[2] Following the heat wave, several studies estimating the number of cases attributable to the high levels of air pollution were published. For example, it was estimated that of the 1000 to 1400 excess deaths that occurred in the Netherlands during the August 2003 heat wave, 400 to 600 may have been caused by air pollution,[3] and between 21% and 38% of the 2045 excess deaths that occurred during the first 2 weeks of August in the United Kingdom were attributable to increased ozone levels.[4] None of these early studies examined the effects of temperature and ozone simultaneously.

A time-series study released recently by the French Institute for Health Surveillance (Institut de Veille Sanitaire—InVS) found no interaction effects between minimum or maximum temperature and 24-hour average ozone level.[5] This finding, based on 8 years of data from 9 cities in France, is contrary to the expectation that temperature and air pollutants interact synergistically to increase mortality.[6,7] We believe that such time-series analyses, which include winters and summers and use 24-hour average ozone levels, may dilute and underestimate the effects of an extreme event occurring over a relatively short period of time.

In this study, we focus specifically on the 2 weeks of the heat wave in August 2003, using the preceding month's observations as a baseline, to examine the impact of extreme temperature and ozone pollution on daily deaths reported in France. In particular, we identify how the available weather variables (daily maximum temperature, minimum temperature, and peak ozone) affected daily deaths over that lag period, and we also estimate the fraction of deaths attributable to each variable and to interactions between them.

Keith Dear, Tord Kjellström, *and* ***Ivan Hanigan*** *are with the National Centre for Epidemiology and Population Health, Australian National University, Canberra.* ***Geetha Ranmuthugala*** *is with the School of Rural Health, Monash University, Bendigo.* ***Carol Skinner*** *is with the Bureau of Meteorology, National Climate Centre, Melbourne.*

METHODS

In this study, we examine temperature and ozone data for the period from June 25 through August 19, 2003, for 12 cities in France: Paris, Marseille, Lyon, Toulouse, Nice, Strasbourg, Bordeaux, Renne, Lille, Dijon, Le Mans, and Poitiers. This 56-day period includes the 2-week period between August 1 and August 14, 2003, when temperatures rose and remained high. There were no missing data for the 56-day study period in any of the variables we examined.

Outcome Measure

We gathered the number of deaths in each city, for each day of the 56-day study period, from graphs presented in a report released by the InVS in France.[8]

Exposure Measures

We investigated the effects on daily mortality of 3 atmospheric variables: maximum daily temperature (max), minimum daily temperature (min), and daily peak ozone level. Other atmospheric variables (such as humidity, rainfall, wind speed, and other pollutant measures) were not available. We gathered daily maximum and minimum temperatures for the study period from graphs presented in the InVS report.[8] We estimated daily peak levels of photochemical ozone by using PREV'AIR models, which generate, for Europe, daily forecasts and maps of air quality from digital simulations on various spatial scales.[9] We downloaded daily charts into ArcMap,[10] and we estimated ozone levels at the location of each city by interpolation. Predictions made by these models have been verified against observed data, and the correlation between the predicted and observed peak ozone levels for suburban stations is between 0.63 and 0.69, depending on the lag period between day of observation and day of prediction.[11]

Statistical Modeling

The 3 exposure measures were positively correlated, but not to such a degree that estimation of their individual effects was compromised by multicollinearity. The partial correlations, with adjustment for differences between cities, were as follows: max:min, $r = 0.65$; max:ozone, $r = 0.83$; and min:ozone, $r = 0.60$.

We modelled counts of daily deaths by using a modification of the polynomial distributed lag (PDL) model, which has previously been used to examine the effect of air pollution and temperature on mortality.[12–14] These models allow a daily outcome to be dependent on covariate values measured on the same day and on previous days. Most epidemiological studies assessing the effect of air pollution and temperature have used moving averages and short lag periods, which restrict the influence of exposure. Limiting the lag period to a short period such as 3 days assumes that, beyond that period, there is no impact on mortality. In contrast, using longer period of lag would require a large number of highly correlated covariates to be fitted in the model, making it difficult to assess the lag effect because of multicollinearity. A distributed lag approach uses parsimonious models that nevertheless allow the outcome to be influenced by exposure for many days before. Researchers have recently used this approach to examine the health effects of temperature and air pollution.[12,13] Schwartz compared the use of constrained and unconstrained PDL models. He recommended that PDL models become standard practice in air pollution epidemiology because limiting the lag period resulted in an underestimated effect.[13]

We considered quartic polynomials with up to 15 lags, allowing daily mortality to be influenced by temperature and ozone over the previous 2 weeks. We allowed the maximum lag to vary, and we estimated it by using a maximum likelihood method. A previous study using distributed lag models to examine the effect of weather on respiratory and cardiovascular deaths in 12 US cities found that 3 weeks (21 days) was more than sufficient to capture the effects of temperature.[15] Most air pollution studies have used shorter lag periods in the assessment of short-term impacts. Given the short study period (56 days), we found that controlling for long-term trends and seasonality was not required.

Considering only a single lagged daily covariate, X, we assume that the number of deaths, Y_t, on day t follows a Poisson distribution, with mean $E[Y_t]$ modeled as

$$\log E[Y_t] = \alpha + \beta_0 X_t + \beta_1 X_{t-1} + \cdots + \beta_q X_{t-q} + \log(\text{population}). \quad (1)$$

In the standard quartic PDL model, the βs follow a polynomial curve in the lag time, j:

$$\beta_j = \eta_0 + \eta_1 j + \eta_2 j^2 + \eta_3 j^3 + \eta_4 j^4, \quad j = 0, \ldots, j. \quad (2)$$

For given maximum lag J, the model assumes that conditions beyond J days ago do not affect today's outcome; that is, $\beta_j = 0, j > J$. The standard model makes no particular assumptions about the shape of the curve at lag J, and thus it allows a discontinuity in the curve between J and $J + 1$ that seems undesirable. To avoid this discontinuity, we impose 2 additional constraints on the polynomial. Regarding β_j as a continuous function $\beta(j)$, we require $\beta(J) = 0$ and $\beta'(J) = 0$, where $\beta'(j)$ is the first derivative with respect to j. Thus the curve smoothly meets its implied zero continuation beyond J. Given sufficient data, and if long-term lag effects are in fact absent, a fitted curve would tend to have these properties; but here, with data for only 56 days, we found it necessary to impose them explicitly as constraints on the model.

The 2 constraints reduce the number of parameters to be estimated for the quartic polynomial from 5 to 3 and can be achieved by replacing Eq. (2) with

$$\beta_j = \eta_1 (J - j)^2 + \eta_2 (J - j)^3 + \eta_3 (J - j)^4, \quad j = 0, \ldots, J \quad (3)$$

Thus we fit a linear model, with no intercept, to 3 constructed covariates calculated as the second, third, and fourth powers of $(J - j)$. Because each of these covariates is itself

zero at $j = J$ and also has zero slope there, any linear combination of them will also have these properties, which achieves the required constraints.

It is not clear that the effect of today's conditions can affect today's mortality in the same way as previous days (clearly, tomorrow's conditions cannot). We therefore included additional parameters to allow deviation of today's effects from the polynomial curve. We achieved this by adding today's conditions (max, min, and ozone) directly as covariates in the models.

We found the optimal extent of the distributed lag functions by using a maximum likelihood method, manually testing models from $J = 3$ to $J = 15$. We assumed that the same value of J applied to all 3 variables and to the 3 interaction functions between them. Because the lagged variables are not defined at the beginning of the data set, when comparing different values of J, we deleted 1 or more days from the analysis with the smaller J, in order to compare the 2 models on a fair basis, using the same range of days.

After fitting the model, we recovered the estimated βs by replacing the ηs with their estimates in Eq. (3). We expressed the percentage increase in death associated with each independent variable at a particular lag as $100[\exp(\beta) - 1]\%$, and we found the total effect by summing these daily effects.

The models also included log-linear terms allowing death rates to differ by city (11 *df*) and by day of the week (6 *df*).

Assessing Interactions

We incorporated interactions into the model as products of deviations from the mean. For an independent variable, $X^{(12)}$, representing the interaction between two atmospheric variables, $X^{(1)}$ and $X^{(2)}$, such as maximum daily temperature and minimum daily temperature, we calculated $X^{(12)} = (X^{(1)} - \bar{X}^{(1)})\,(X^{(2)} - \bar{X}^{(2)})$. We applied this formulation to each of the 3 two-way interactions between the 3 variables in our data, and we modeled the resulting product terms by using constrained PDL models in the same way as the main effects. If such interactions are present, the effect of each atmospheric variable depends on the levels of the other 2 variables over the period of the lagged models. For example, we obtained the effect of a unit change in $X^{(1)}$ at particular values of $X^{(2)}$ and $X^{(3)}$ as

$$\frac{d \log E[Y]}{dX_j^{(1)}} = \beta_j^{(1)} + \beta_j^{(12)} X_j^{(12)} + \beta_j^{(13)} X_j^{(13)}. \qquad (4)$$

The set of these effects for $X^{(1)}$ therefore also involves the values of $X^{(2)}$ and $X^{(3)}$ over the 15 days. We can achieve a graphical presentation of the interactions with $X^{(1)}$ by fixing each other variable at a constant value for all lags (eg, $X^{(2)*}$ and $X^{(3)*}$) and graphing Eq. (4) first for a series of values of $X^{(2)*}$ and then for a series of values of $X^{(3)*}$.

Attributable Risk

It is of interest to examine what, according to the fitted model, the mortality experience in these cities would have been had the heat wave not occurred. We can examine the effect of removing components of the climatic anomaly to compare their contributions to the excess mortality. In this way, we can assess the relative importance of maximum temperature, minimum temperature, and ozone.

The heat wave can be considered to have started on the first of August. We held one or more climatic variables constant as it was on that day in each city, and we used the fitted model to estimate how many deaths might have been expected had that been the case. For each variable, we calculated 4 sets of daily predicted deaths, $P0$ to $P3$. For variable $X^{(1)}$, these are as follows.

$P0$ represents the actual fitted values, using all covariate data as actually observed on each day. These approximate the actual observed deaths day by day, although with some random error. We calculated $P1$ by holding variable $X^{(1)}$ constant at its value on August 1, with $X^{(2)}$ and $X^{(3)}$ as observed. Assuming the actual excess deaths were in part due to the effect of $X^{(1)}$, we found that these predictions will be smaller than $P0$. We calculated $P2$ by holding variables $X^{(2)}$ and $X^{(3)}$ as they were on August 1, with $X^{(1)}$ as observed. These also are expected to be smaller than $P0$, although they may be larger or smaller than $P1$, depending on the relative contributions of the variables. We calculated $P3$ by holding all 3 variables constant as they were August 1. These predictions are expected to be smaller than $P1$ and $P2$.

We can now measure the unique contribution of $X^{(1)}$ in two ways: either as the difference between predicted deaths $P0$ and $P1$, or else as the difference between predicted deaths $P2$ and $P3$. Difference $P0 - P1$ estimates how many deaths would have been avoided if the heat wave had not included $X^{(1)}$: these are attributable to $X^{(1)}$, including both its main effect and also any interaction effects between $X^{(1)}$ and the other variables. Difference $P2 - P3$ estimates how many excess deaths would have been caused if the heat wave had involved *only* $X^{(1)}$: these are attributable to the direct effect of $X^{(1)}$ only. Because of interaction effects, two somewhat different estimates can be expected for each variable.

We made all analyses by using Stata version 8.2.[16]

RESULTS

Approximately 11,211 deaths occurred in the 12 cities in France between the dates of June 25 and August 19, 2003. Table 1 shows the daily death rates and a summary of atmospheric conditions in each of the cities during the 56-day period. The cities are ordered in this table according to the proportional increase in deaths during the heat wave relative to the immediately preceding 3 weeks. We performed the same calculation for the 3 atmospheric variables (data not shown) and indicated which cities showed the greatest increase in each. There is a tendency for the cities that experience the most extreme weather to also suffer the greatest increase in deaths, although Lyon and Bordeaux were exceptions: Lyon had a high increase in death rate despite a modest heat wave, and Bordeaux had the opposite.

Table 1.—Daily Deaths and Atmospheric Conditions for 12 French Cities: June 25–August 19, 2003

City	No. of deaths/day		Heat wave increase*	24-h temperature (°C)				24-h peak ozone (μg/m³)	
				Min.		Max.			
	Mdn	Range		*M*	Range	*M*	Range	*M*	Range
Paris	48	29–322	2.02	18.5	13–26†	28.8	20–39†	139.2	40–360†
Le Mans	6	1–26	1.89	16.5	12–23†	28.7	20–40†	116.3	60–290†
Lyon	15	6–55	1.74	19.0	13–25	31.5	21–40	133.1	80–210
Poitiers	6	2–19	1.66	15.5	10–22†	29.4	20–40†	117.6	60–290
Rennes	6	0–12	1.50	16.1	9–24†	27.5	20–40†	105.9	60–290†
Strasbourg	11	2–20	1.41	16.1	12–21†	29.7	21–39	141.9	60–290†
Toulouse	13	5–24	1.34	19.2	14–24	32.1	24–41	128.9	80–185
Dijon	7	1–17	1.33	17.3	12–23	30.5	21–39†	128.3	60–290†
Bordeaux	13	4–30	1.28	17.9	12–23†	30.2	21–41†	121.2	60–210
Nice	16	5–32	1.19	22.6	18–28	30.0	26–35	152.5	95–205
Lille	9	2–16	1.10	15.2	11–21	26.0	19–37	106.5	40–290†
Marseille	28	16–45	1.08	21.5	16–25	33.3	27–38	156.3	95–290

*Heat wave increase shows the ratio of mean daily deaths during the heat wave (July 30–August 19) to before it (June 25–July 29).
†For each weather variable, these 6 cities showed the greatest proportional increase during the heat wave.

The minimum temperature during this 56-day period ranged from 9°C to 28°C, whereas the maximum temperature ranged from 19°C to 41°C. According to the PREV'AIR charts, of the 12 chosen cities in France, Paris experienced the highest level of daily peak ozone (360 μg/m³). We are unable to examine the estimated ozone levels in relation to World Health Organization (WHO) air quality guidelines for Europe, because of the different time frames used: the 120 μg/m³ guideline value specified in the WHO guidelines for ozone is a time-weighted average for an 8-hour period, whereas the PREV'AIR model predicts the expected peak level for the day. Regulations in the UK are based on hourly averages in recognition of the different exposure times required to cause adverse health effects at the various concentrations of air pollution. A 1-hour average of 90–179 μg/m³ is considered to be above the "information threshold," whereas 180 μg/m³ and above is classified as being over the "alert threshold."[17] Even though we do not have 1-hour average levels of ozone for the French cities, it is likely that the information threshold and possibly the alert threshold were exceeded on the days estimated to have peaked at levels approaching 300 μg/m³.

The lagged effects on mortality are evident from the time-series plots in Figure 1. The plots for Paris, Poitiers, Bordeaux, Lyon, Le Mans, Strasbourg, and Dijon, in particular, show that the transient increase in temperature and ozone that occurred in early July was not associated with an obvious increase in mortality. During the August heat wave, however, when much higher temperatures and ozone levels were experienced from about August 1, the increase in mortality lagged by a few days. This effect has already been demonstrated in US cities.[13]

The optimum extent of the distributed lag models, assessed by maximum likelihood, was 9 days. The distributed lag curves in Figure 2 illustrate the effects of each atmospheric variable and the pattern of its interactions with the other two variables in turn. In each graph, a second variable takes 4 equally spaced values approximating its quintiles. The third variable in each case is held constant at its mean. The Max × Min interaction was highly significant ($p < .0001$), as was the Min × Ozone interaction ($p < .0001$). All 3 main effect terms are therefore also required. The Max × Ozone interaction was not significant ($p = .19$).

Fitted Values

Figure 3 shows the daily deaths and model fit for each city. It also illustrates the severity of the heat wave in Paris. From June 25 to July 29, the 5 weeks before the heat wave, the Paris mean death rate was 45.9 deaths per day. On August 12, 322 deaths were recorded, which is 7 times this background rate.

Attributable Risk

Figure 4 illustrates the result for Paris. Four models are shown: the full model, which closely follows the trace of actual daily deaths; a model in which ozone is held constant at its August 1 value so as to remove its estimated effect ("MaxMin"); a model in which both temperature variables are held constant ("Ozone"); and a base model in which all 3 are constant ("None"). The predicted total deaths from August 1 to August 19 were as follows: Full model, 1864; MaxMin model, 1313; Ozone model, 1418; and None model, 827 deaths. Thus the estimated effect of all variables in Paris is 1864–827 = 1037 deaths. The effect of ozone alone can be estimated either as 1864–1313 = 551 deaths (the effect of high ozone over and above the effect of high temperatures), or as 1418–827 = 591 deaths (the effect of ozone in a hypothetical context of normal temperatures). Table 2 shows the corresponding differences, for each city and in total, for all 3 variables.

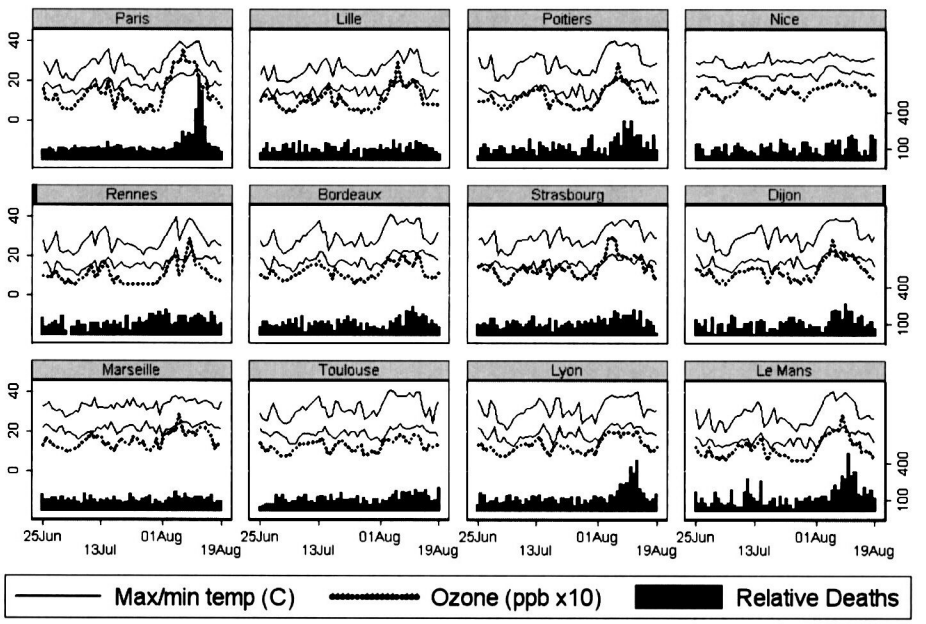

Fig. 1. Daily time series of mortality (right axis) and maximum temperature, minimum temperature, and ozone levels (left axis) for the period from June 25 through August 19, 2003. Mortality is expressed as a percentage of the average daily rate before the heat wave (June 25–July 29).

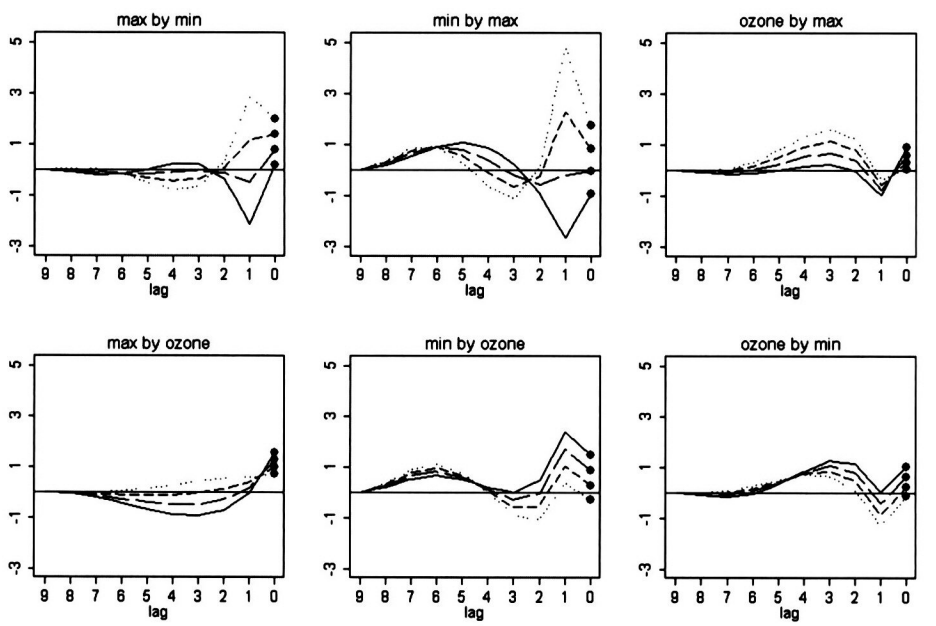

Fig. 2. Constrained polynomial distributed lag (PDL) effects, showing the impact on mortality of a unit increase in daily exposure (1°C temperature or 10 μg/m³ ozone), with mortality being influenced by exposure for up to 8 days preceding date of death (lag). Lag is plotted as increasing to the left. Each vertical pair of graphs shows the effect of one variable (max, min, or ozone) and its interaction with each of the others in turn. Values of the effect-modifying variables are equally spaced approximately at their overall quintiles: max = (25, 28, 31, 34), min = (15, 17, 19, 21), and ozone = (80, 110, 140, 170). Solid lines are for the first (smallest) quintile. Additional parameters allow lag-zero effects to deviate from the polynomial curves (dots).

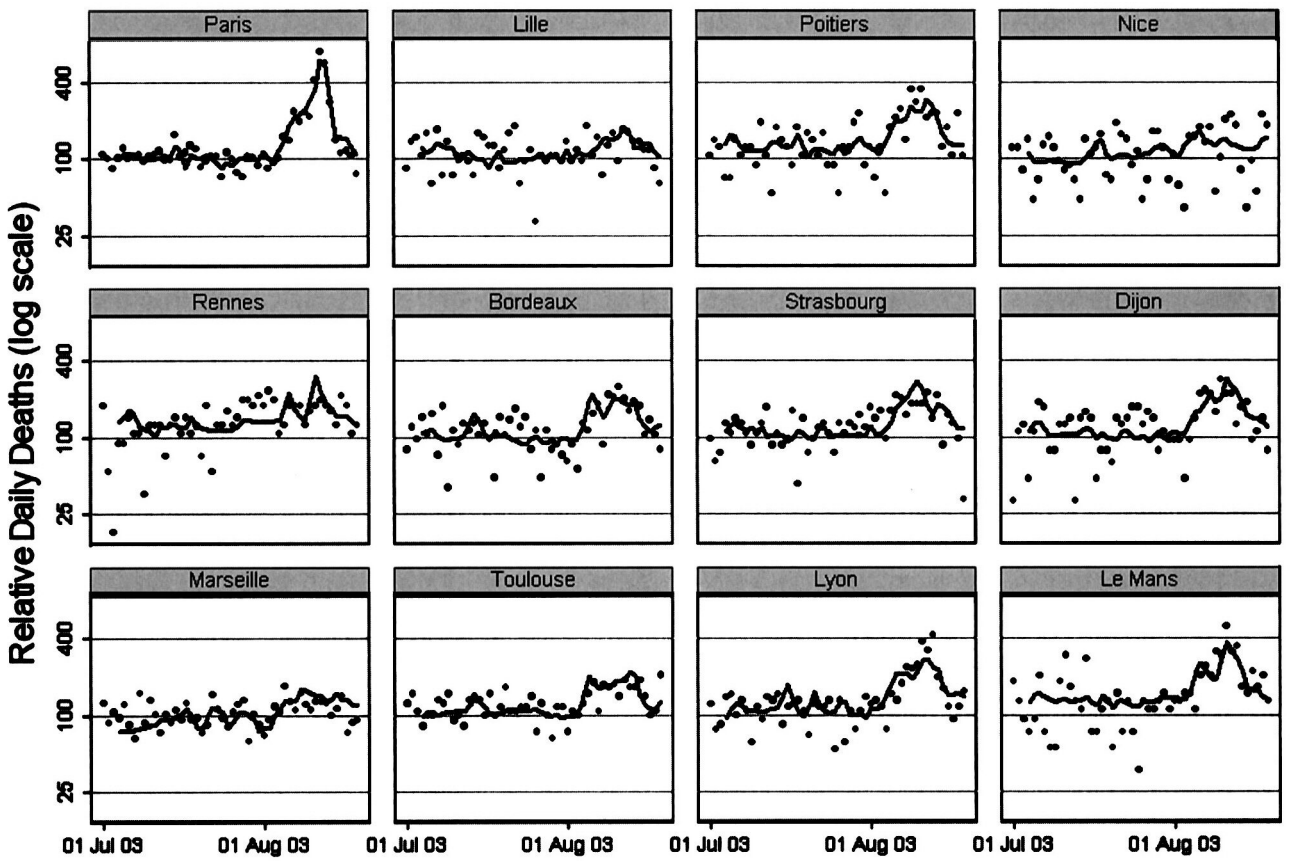

Fig. 3. Daily deaths (dots) and model predictions (lines) of 12 cities in France during August 2003. The values for each city are expressed as a percentage of mean daily deaths over the 5 weeks before the heat wave.

DISCUSSION

Most epidemiological studies published to date make no allowance for the interaction that occurs between temperature and air pollution in their impact on health. The synergistic effect between air pollution and temperature was examined by Katsouyanni et al, who used linear regression models to find a statistically significant interaction between temperature and sulphur dioxide, but only a suggestive trend for the interaction between temperature and ozone.[18] In our study, by using distributed lag models that include temperature extremes, peak ozone, and their interaction terms, we allow mortality to be influenced by extremes of temperature and ozone, and their interactions, over several days. The result is that, although high minimum temperature had the greatest impact on all-cause mortality during this heat wave, there is a significant and substantial interaction between minimum temperature and ozone in increasing mortality. This is especially important in view of other studies that have reported the effects of ozone on all-cause mortality to be negligible.[19]

Our results contrast with those reported by the InVS,[5] which found no interaction effects between temperature and ozone. There are many differences between the studies. In particular, the InVS used data accumulated over 8 years in 9 cities, whereas we focused on the critical 56 days, but in 12 cities. The long time period of their study and the use of 24-hour ozone values in both winter and summer raise questions about how well that model can identify the effects of an extreme short-term event that has occurred only once. The InVS modeled the temperature-response relationships by using penalized splines, in contrast to our simpler linear responses, reasonable here given that we consider only 56 days in the warm season. Linear effects have previously been found adequate for modeling the effects of particulate matter and mortality.[20] Finally, the InVS found lag effects extending only to 3 days past, in contrast to our estimate of 9 days. It is a limitation of our analysis that our small data set required the assumption of the same maximum lag for all variables, although a tentative exploration relaxing this assumption (not reported in detail) suggested that ozone effects may extend beyond the 15 days that we considered.

A harvesting effect ("mortality displacement") is evident from our analysis when one examines mortality in relation to maximum temperature, in that the lag curve becomes negative for a few days. Such an effect is not evident for

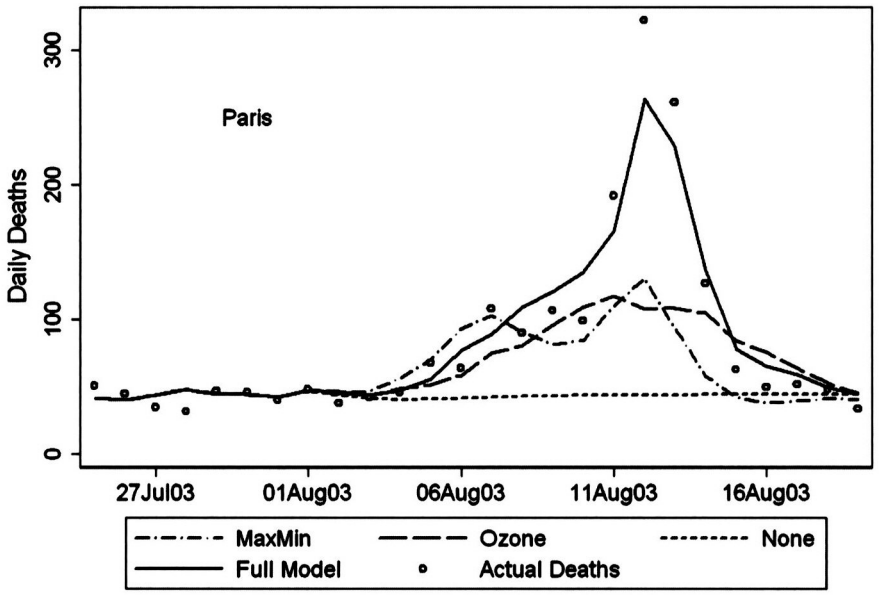

Fig. 4. Model predictions of daily deaths from Paris, France, during an August 2003 heat wave, with climatic variables held from August 1. The full model predicts 1864 deaths in Paris from August 1 until August 19 (in fact, there were 1859). MaxMin = predicted deaths with ozone held (1313); Ozone = predicted deaths with max and min held (1418); None = predicted deaths with all variables held (827).

Table 2.—Attributable Deaths: Difference in Estimated Deaths Between 2 Models

City	Effect of max.temp		Effect of min.temp		Effect of ozone	
	All–Min Ozone	Max–None	All–Max Ozone	Min–None	All–MaxMin	Ozone–None
Paris	861	–146	301	135	551	591
Marseille	88	–17	196	150	40	73
Lyon	147	–64	213	73	53	76
Toulouse	51	–35	178	118	52	53
Nice	25	0	55	48	–6	12
Strasbourg	26	–54	49	10	59	70
Bordeaux	55	–44	155	91	69	63
Renne	19	–32	47	33	43	64
Lille	24	–21	23	22	44	69
Dijon	39	–31	63	31	50	61
Le Mans	46	–51	88	31	50	84
Poitiers	16	–38	97	52	65	89
Total	1397	–533	1465	794	1070	1305

minimum temperature. In contrast to the harvesting effect of maximum temperature, the initial 2% increase in mortality (for every 1°C increase in exposure) that is observed within a couple of days of raised minimum temperature drops to a 1% increase, but then it increases gradually again over the next couple days, surpassing 2% by about the seventh day. The explanation for this is not clear, but it is possible to hypothesize that the immediate increase in mortality is the impact on the most frail and vulnerable group, whereas the lagged response possibly applies to a less frail but still susceptible group. It is also possible that the immediate and lagged responses are due to different disease categories. For example, there is evidence to suggest that cardiovascular disease presents as an acute response, whereas respiratory diseases are much slower to progress and therefore are more lagged.[21] We do not have the breakdown of mortality by disease group or age, unfortunately, with which to examine this hypothesis. It is also unfortunate that we were not able to obtain access to the original data as recorded at meteorologic stations, rather than being reduced to reading data from published graphs and interpolating from surface models. (We approached the guardians of the

original data, but without success.) Including additional atmospheric variables might have revealed how humidity and wind speed, in particular, modify the effects of temperature through their contribution to perceived thermal stress. It may also be that errors arising from our use of secondary data have attenuated the true effects, causing us to underestimate the attributable risk.

The interaction between minimum temperature and peak ozone suggests a short time frame (approximately 1 day) during which the interaction effect impacts differently on mortality. The "min by ozone" section of Figure 2 shows that the acute effect of minimum temperature varies according to peak ozone level. Mortality one day later increases by about 2.5% for every 1°C increase in minimum temperature when the peak ozone level is 80 $\mu g/m^3$, but by only approximately 1% when the peak ozone level is 110 $\mu g/m^3$. It is close to zero when peak ozone is 170 $\mu g/m^3$. More detailed studies are required to elucidate the meaning of this interaction in physiological terms.

These findings are important in light of predictions that higher maximum and minimum temperatures, and more hot days over nearly all land areas, are likely to occur as a result of global climate change.[22] The nature of the photochemical reaction that produces ozone is such that hot sunny days, in the presence of precursors such as volatile organic carbons, carbon monoxide, and methane, will be associated with high levels of ozone in the lower atmosphere. It is therefore likely that the extreme heat events predicted to occur more frequently in the future will be accompanied by high levels of ozone. Our finding of a strong ozone effect during the European heat wave of 2003 highlights the need for air pollution control policies to be included in prevention programs for heat exposure.

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Requests for reprints should be sent to Dr. Keith Dear, National Centre for Epidemiology and Population Health, Australian National University, NCEPH, Acton Australian Capital Territory, ACT 0200, Australia.
E-mail: Keith.dear@anu.edu.au

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