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Background. Increased mortality is associated with both very low and very high ambient temperatures. This study assesses the relationship between daily numbers of deaths and variations in ambient temperature within the city of Valencia.

Methods. The daily number of deaths from all causes (total deaths and only those occurring in people aged over 70), as well as those deaths from specific causes (e.g. cardiovascular and respiratory diseases, malignant tumours and all causes except external ones) occurring within the city of Valencia were related to the average daily temperature using autoregressive Poisson regression controlling for seasonality, day of the week, holidays, air pollution, influenza incidence, and humidity. Temperature was measured within the regression model as two complementary variables: ‘Heat’ and ‘Cold’; also taken into account were their delayed effects up to 2 weeks after measurement.

Results. Graphical analysis revealed a relationship between temperature and mortality according to the time of year. For the cooler months (November–April), the temperature at which mortality was lowest was the ‘minimum’ (i.e. around 15°C), while for the warmer months (May–October), it occurred at around 24°C. Because of this, a stratified analysis was undertaken with different values for the ‘Heat’ and ‘Cold’ variables according to which of the two seasons was involved. During the colder months of the year, higher temperatures tended to exert a rapid influence on mortality and the lower temperatures a more delayed relation. During the hot season it is the heat variable which more clearly manifests an effect, and this is prolonged over the two following weeks. Variations also occur according to age and cause of death. The effect of temperature is greater in persons aged over 70 years of age, and it is also greater in cases of circulatory and respiratory diseases.

Conclusions. A statistically significant association has been found between temperature and mortality. This relationship is not monotonic, but mortality increases in proportion to the variance in ambient temperature from a range of temperatures that varies from winter to summer.

Keywords: mortality, temperature, lagged effects, Spain.
to country according to the usual climate of each zone.\textsuperscript{19–21}

As well as temperature, the possible effects of other meteorological phenomena on mortality (e.g. humidity or wind) have also been mentioned.\textsuperscript{21} These factors are usually treated as individual variables within the model. On other occasions, however, an attempt has been made to use variables or indices which combine the most important element—temperature—with other meteorological variables (i.e. indices combining relative humidity or wind speed with temperature have been used)\textsuperscript{22,23} without, on the whole, yielding significantly better results compared to the separate use of meteorological variables.

Research on the association between temperature and mortality has relevance to the formulation of health policy, given that preventive policies could be developed based on the results of these studies. In recent years there has also been great concern about ‘global climatic change’.\textsuperscript{24–32} This adds new relevance to the work because of the effects that any climatic change may have on the health of the population worldwide.

The objective of this study is to examine the form and relationship, as well as to assess the magnitude of the association, between general and specific causes of mortality and temperature variations within the city of Valencia during the period 1991–1993.

\section*{MATERIAL AND METHODS}

\subsection*{Study Area}

The study was carried out in the city of Valencia, Spain. Its population, according to the census of 1991, is over 750,000 inhabitants. It is situated on the shores of the Mediterranean, and is largely urban, although it conserves several zones dedicated to agriculture. The climate in the city, given its location, is that known as mesothermal (temperate), with mild, humid winters and warm, hot summers. Given the low level of rainfall it is classified as arid-semiarid.\textsuperscript{33}

\subsection*{Data}

The daily number of deaths in Valencia was obtained from the Valencian Community’s Mortality Register and was restricted to city residents only. The completeness of the register, the quality of patient diagnosis and certificated cause of death have been examined and it has been demonstrated that the register is both complete and reliable.\textsuperscript{34,35} Specifically, the high level of accuracy of the results obtained for all cause mortality, cancer and circulatory diseases has been verified. In the case of respiratory illnesses the results are less satisfactory.\textsuperscript{36} The causal groups considered in this study are total mortality, total mortality for subjects over 70 years of age, total mortality figures excluding external causes (ICD-9 codes 001–799), deaths from diseases of the circulatory system (ICD-9 codes 390–459), deaths from respiratory diseases (ICD-9 codes 460–519), and from malignant tumours (ICD-9 codes 140–208).

The mean daily temperature and daily relative humidity figures were obtained from the National Institute of Meteorology at the city’s weather station in the Meteorological Centre of Valencia. The values recorded at this location were considered more representative of the temperature exposure of the population as a whole than those recorded at another weather station situated at the airport (10 km from the city centre), given that various studies have shown the effects of the ‘thermal island’ which is produced in the city of Valencia.\textsuperscript{37,38} The temperature of a locality is modified by a large urban agglomeration. For Valencia the magnitude varies from almost nil (in situations of strong winds), to over 6°C (in anticyclonic situations with clear skies and slight winds).

Air pollution values were obtained from the Valencian Community’s Air Pollution Monitoring Network. The basic parameters measured are 24-hour sulphur dioxide (\text{SO}_2) and suspended particulates (black smoke). We have used ‘suspended particulates’ as an appropriate indicator for pollution levels, given that previous studies have uncovered a greater association between this measure and mortality compared with \text{SO}_2.\textsuperscript{39} Monitoring is carried out according to European Union standards.\textsuperscript{37,40}

The number of influenza cases declared weekly was obtained from the epidemiological services of the city of Valencia. The quality of information in this register has also been evaluated, revealing a good level of coverage in the Valencia Region.\textsuperscript{41}

\section*{METHOD}

Epidemiological evaluation of the data was undertaken using both simple analysis and multivariant models.\textsuperscript{42} In each, analysis was carried out for the entire period of study and for two semestral periods. Specifically, it was decided to group the relatively colder months (November–April), and the hotter months (May–October). In this article we will present only the results of the stratified analysis since this more reflects the dynamic relationship between temperature and mortality.

\subsection*{Identification of the Models}

A scatter diagram was produced showing the values for mortality and temperature. The regression line was adjusted by means of a locally weighted running line
smoother, Loess; a method employing weighted least-squared fits in a series of data, predefined in each case (window). A non-monotonic relationship between mortality and temperature emerged in this diagram with mortality reaching its minima in the mean temperature range 22–25°C.

The nature of the relationship between temperature and mortality was also analysed by season. Scatter charts for mortality residuals data were produced for each cause and temperature. The corresponding mortality minima for each season were determined based on the previous graphs (taking the value 15°C in the colder months, and 24°C in the relatively hotter months). This allowed the formation of two pairs of complementary variables, one for the colder months, labelled ‘Heat-C’ and ‘Cold-C’, ‘Heat-C’ was equal to the daily mean temperature (MT) – 15 if MT > 15°C, being equal to zero otherwise. ‘Cold-C’ was equal to 15 – MT, when MT < 15°C, being equal to zero otherwise. For the relatively warm months, these variables were labelled ‘Heat-H’ and ‘Cold-H’, with ‘Heat-H’ being equal to MT – 24 if MT > 24°C, it being equal to zero otherwise. ‘Cold-H’ = 24 – MT if MT < 24°C, it being equal to zero otherwise.

Using the information obtained in the simple analysis and the previously mentioned temperature variables, a model was created for each cause of death including all variables to be studied using the linear regression method as well as graphical representation. Mortality figures were redrawn on a logarithmic scale with the intention of obtaining a series with stationary variance. The variables controlled for were seasonality, the year in question, weekly periodicity, public holidays, air pollution (suspended particulates), the incidence of influenza among the population and humidity.

As part of the construction of the appropriate model for each cause of death the criterion used was the ‘best fit’, as determined by the correlation coefficient of each one of the variables kept within the model i.e. those with $P \leq 0.1$.

The effect of temperature on mortality can either be immediate or can occur after some delay. Because of this, separate indicators were created for each one of these two temperature variables. Following other authors, we calculated different lag intervals: 1–2, 3–6, and 7–14 days. In our opinion, exploring an excessive number of lag terms runs the risk of finding non-causal relationships which have occurred simply by chance or by seasonal autocorrelation. Bearing this in mind, it was decided not to include any lag time beyond the 14th order. These variables were introduced into the model in alternate pairs, corresponding in all cases to the variables ‘Heat’ and ‘Cold’ over the same chronological period.

**Estimation of the Degree of Association between Temperature and Mortality**

Using autoregressive Poisson regression and starting from the foundation of the models emerging from the previous stage, the final models were created in order to estimate the parameters of each one of the explicative variables. In this instance, the best adjustment of the model was evaluated based on the deviance. This procedure was followed for each of the causes of death being studied and for each of the three study periods, in other words, all year round, the cooler seasons as well as the hotter ones.

In each of the previous models, possible autocorrelation of the mortality residuals was examined by means of introduction into the model of the mortality lags up to the term of the third order. These terms were retained within the model as long as they or their subsequent terms were significant at $P \leq 0.05$.

**Sensitivity Analysis**

Two separate data analyses were performed. Firstly, we regressed using least squares fit, the logarithm of mortality for each of the causal groups along with the corresponding final model previously used in the Poisson regression. Secondly, so as to evaluate whether the substantial number of parameters forming part of each model affected the conclusions or the stability of the calculated parameters, a sensitivity analysis was undertaken, consisting of observing the changes produced in the parameters while at the same time feeding remaining variables into the model. In both cases, the parameter predictors (unpublished observations) were consistent with those presented here.

Analysis of the data was carried using the SPSS statistical package for the descriptives, graphs and multiple linear regressions, whilst the EGRET package was employed in the Poisson regression.43–46

**RESULTS**

The mean daily number of deaths among the citizens of the city of Valencia for 1991–1993 was 17.49, with a standard deviation of 5. Mortality within the city of Valencia displayed a marked seasonality, with peaks in the winter and relative troughs in the summer. There was also a notable weekly pattern, with more deaths occurring on work days than at weekends. The mortality data reflect the previously mentioned seasonality, as for all the cause of death groups, the mean number of
daily deaths occurring during the cold months is greater than that occurring during the hot months (Table 1). Furthermore, a sizable difference in atmospheric levels of suspended particulates can be seen between the two seasonal periods.

From a graphical analysis of the whole period, a relationship emerges between the number of deaths from all causes and mean temperature—the lowest mortality rate being reached at temperatures within a range between 22–25°C (Figure 1). Taking the two periods separately (Figure 2), it can be seen that, as well as a seasonal effect (i.e. more deaths occurring during the ‘colder’ months than in the ‘hotter’ months), the non-monotonic nature of the data previously seen in the scatter chart for the whole study period is now also being seen in each seasonal stratification. The mortality minima can be found within a wide temperature range centred around 15°C during the colder months and around 24°C during the hotter months.

Tables 2 and 3 show the results of the estimated association between mortality and the two variables ‘Cold’ and ‘Heat’, for each season, causal group and lag period studied. The RR of dying due to a rise or fall in mean temperature in the order of 1°C below or above the appropriate temperature cutoff point were estimated.

Cold Months
The results for the cooler months (Table 2) demonstrate that, apart from cases involving neoplasms, the RR corresponding to the ‘Cold’ variable is higher than 1 and has, or at least approaches, statistical significance for every causal group and lag period studied. Taking the ‘all causes’ mortality group as an example, on average, during this 6-month period, each 1°C decrease in daily temperature below 15°C alters the mortality index by approximately 1.6% during the first week and double this value (3.2%) during the lag period of the second week. The greatest effect on mortality of temperature is found in the case of respiratory and cardiovascular diseases. The 70-plus age group showed higher RR, particularly in lag periods 1–2, and 3–6. The estimate for the ‘Heat’ variable shows an immediate and positive effect that reveals itself more clearly in the case of cardiovascular mortality. The direction of this shift in relationship from lags 3–6 is such that there appears a negative effect which is especially marked in cases of respiratory diseases. As for ‘Cold’, changes in the ‘Heat’ variable do not have any noticeable effect on mortality due to malignant tumours.

Hot Months
In the case of the ‘Cold’ variable we found a significant (or almost significant) degree of association for some causes of death (Table 3). Particularly noteworthy is the degree of association between ‘Cold’ and mortality caused by respiratory disease, which remains constant throughout all the studied lag periods. Furthermore, a significant association was discovered with cardiovascular diseases, especially during the lag period 3–6. An immediate effect (same day) was seen on mortality owing to neoplasms.

The association of higher temperatures (‘Heat’) is, on the whole, the strongest of all those revealed, although,
in some cases (e.g. mortality from diseases of the circulatory system and malignant tumours) no significance was found. Taking the 'all-cause' mortality group as an example, for each 1°C increase in temperature over and above 24°C during the six warmest months of the year, there is a change in mortality of between 1% and 4.2%. ‘Heat’ displays its most marked effect on mortality during the first week, except in the case of mortality due to respiratory diseases, in which case the greatest effect was found at the 7–14th lag. This mortality group exhibits the greatest susceptibility to the ‘Heat’ variable.

Humidity was discovered to have an inverse relationship to mortality, although generally not a significant one.

**Figure 1** Scatter diagram for all-cause mortality and mean temperature. Valencia, 1991–1993.
DISCUSSION
An association has been found between temperature and mortality within the city of Valencia. It is non-monotonic, resembling a ‘V’ shape. If one carries out a stratified analysis for two periods (‘cold’ months and ‘hot’ months), the values of temperature at which mortality figures reach their respective minima are different for each season of the year.

During our study, the point in temperature at which mortality reached its minimum was around 24°C. This value is similar to that found in other studies carried out in zones with latitudes and climate similar to Valencia.

Figure 2. Scatter diagram for distribution of all-cause mortality and mean temperature according to seasonal period. Valencia, 1991–1993.
### Table 2  

<table>
<thead>
<tr>
<th>Temperature and causes of death</th>
<th>Relative risks(^a) per lag period</th>
<th>0</th>
<th>1–2</th>
<th>3–6</th>
<th>7–14</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>RR</td>
<td>95% CI</td>
<td>RR</td>
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<td><strong>Cold</strong></td>
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<tr>
<td>All causes</td>
<td>1.015 (1.005–1.024)</td>
<td>1.016 (1.005–1.026)</td>
<td>1.016 (1.004–1.028)</td>
<td>1.032 (1.018–1.046)</td>
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<tr>
<td>All causes in subjects &gt;70 years</td>
<td>1.016 (1.005–1.028)</td>
<td>1.024 (1.011–1.037)</td>
<td>1.023 (1.009–1.037)</td>
<td>1.037 (1.021–1.054)</td>
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</tr>
<tr>
<td>All causes—external causes</td>
<td>1.016 (1.006–1.026)</td>
<td>1.016 (1.006–1.027)</td>
<td>1.017 (1.005–1.029)</td>
<td>1.031 (1.017–1.045)</td>
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<tr>
<td>Cardiovascular diseases</td>
<td>1.021 (1.006–1.036)</td>
<td>1.026 (1.010–1.043)</td>
<td>1.015 (0.997–1.033)</td>
<td>1.043 (1.021–1.064)</td>
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<tr>
<td>Respiratory diseases</td>
<td>1.023 (0.994–1.054)</td>
<td>1.046 (1.013–1.080)</td>
<td>1.022 (0.987–1.059)</td>
<td>1.017 (0.976–1.060)</td>
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<td>Neoplasms</td>
<td>1.006 (0.987–1.026)</td>
<td>1.000 (0.979–1.022)</td>
<td>1.018 (0.995–1.042)</td>
<td>1.015 (0.988–1.042)</td>
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<tr>
<td><strong>Heat</strong></td>
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</tr>
<tr>
<td>All causes</td>
<td>1.024 (1.002–1.046)</td>
<td>1.012 (0.987–1.038)</td>
<td>0.973 (0.947–0.999)</td>
<td>0.999 (0.970–1.029)</td>
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<tr>
<td>All causes in subjects &gt;70 years</td>
<td>1.019 (0.993–1.046)</td>
<td>1.020 (0.989–1.051)</td>
<td>0.970 (0.939–1.003)</td>
<td>0.986 (0.951–1.022)</td>
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<tr>
<td>All causes—external causes</td>
<td>1.028 (1.006–1.051)</td>
<td>1.013 (0.988–1.040)</td>
<td>0.973 (0.946–1.000)</td>
<td>0.995 (0.966–1.026)</td>
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<tr>
<td>Cardiovascular diseases</td>
<td>1.037 (1.003–1.071)</td>
<td>1.030 (0.991–1.071)</td>
<td>0.966 (0.926–1.008)</td>
<td>1.005 (0.960–1.052)</td>
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<tr>
<td>Respiratory diseases</td>
<td>0.985 (0.915–1.061)</td>
<td>1.054 (0.973–1.142)</td>
<td>0.876 (0.795–0.966)</td>
<td>0.847 (0.758–0.946)</td>
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<td>Neoplasms</td>
<td>1.027 (0.983–1.073)</td>
<td>0.994 (0.943–1.047)</td>
<td>0.997 (0.944–1.053)</td>
<td>1.000 (0.943–1.061)</td>
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</table>

\(^a\) Estimated relative risk (RR) and confidence interval of 95% (95% CI) per change in 1°C from autoregressive Poisson regression analysis of mortality on the average values of both ‘Cold’ and ‘Heat’ variables in the four lag periods, controlling for influenza incidence, black smoke, humidity, day of the week and holidays.

Cold months: November–April.

Cutoff point = 15°C (see Methods).

### Table 3  

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<th>Temperature and causes of death</th>
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<td><strong>Cold</strong></td>
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<tr>
<td>All causes</td>
<td>0.999 (0.987–1.010)</td>
<td>1.001 (0.988–1.014)</td>
<td>1.008 (0.994–1.023)</td>
<td>1.004 (0.996–1.039)</td>
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<tr>
<td>All causes in subjects &gt;70 years</td>
<td>1.008 (0.994–1.023)</td>
<td>1.008 (0.993–1.024)</td>
<td>1.017 (0.999–1.036)</td>
<td>1.011 (0.993–1.030)</td>
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<tr>
<td>All causes—external causes</td>
<td>0.999 (0.988–1.011)</td>
<td>1.002 (0.989–1.015)</td>
<td>1.010 (0.995–1.025)</td>
<td>1.005 (0.991–1.020)</td>
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<tr>
<td>Cardiovascular diseases</td>
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<td>Respiratory diseases</td>
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<td>1.033 (0.988–1.078)</td>
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<td>1.015 (1.000–1.031)</td>
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<td>1.008 (0.991–1.025)</td>
<td>0.998 (0.980–1.016)</td>
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<td><strong>Heat</strong></td>
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</tr>
<tr>
<td>All causes</td>
<td>1.024 (1.003–1.046)</td>
<td>1.036 (1.012–1.060)</td>
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<td>All causes in subjects &gt;70 years</td>
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<td>1.009 (0.973–1.045)</td>
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<td>1.009 (0.994–1.039)</td>
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<td>Cardiovascular diseases</td>
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<td>Respiratory diseases</td>
<td>1.098 (1.024–1.179)</td>
<td>1.057 (0.975–1.145)</td>
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\(^a\) Estimated relative risk (RR) and confidence interval of 95% (95% CI) per change in 1°C from autoregressive Poisson regression analysis of mortality on the average values of both ‘Cold’ and ‘Heat’ variables in the four lag periods, controlling for influenza incidence, black smoke, humidity, day of the week and holidays.

Hot months: May–October.

Cutoff point = 24°C (See Methods).
The extent of association between the ‘Heat’ and ‘Cold’ variables with the figures for total mortality is different according to the season of the year and the lag period being examined. During the relatively colder months the variable exerting the greatest effect is the one involving cold temperatures. Furthermore, this variability represents a significant positive effect over the 2 months following exposure. The ‘Heat’ variable has an immediate effect which is not prolonged any further than the second day post-exposure. However, during the warmer months it is the heat variable that exhibits the greatest effect—one which, moreover, is sustained during the two following weeks.

**Relationship According to Cause of Death Groups**

The association of mortality with low temperature is greater for deaths due to respiratory and cardiovascular causes. In England and Wales, low temperatures occurring 5 days prior to death were the most important for respiratory infections, the temperature over the previous 3–4 days was relevant in deaths caused by cerebrovascular accident and temperature over the previous 2 days is of most critical relevance for deaths due to myocardial infarction. Kunst et al. found a stronger association for deaths due to diseases of the circulatory system.

Various hypotheses have been advanced to explain the increased mortality due to diseases of the circulatory system following exposure to low temperatures or during cold months. Of these, the most plausible would seem to be an increase in arterial blood pressure, cardiac workload and an increase in platelet viscosity. Respiratory infections may be responsible for deaths whose basic cause has been classified as cardiovascular. An indirect mechanism which may help to account for this process would be the increase in platelet aggregation during infections. Kunst et al. argue that this hypotheses is contradictory, given that the majority of mortality associated with Cold occurs during the first few days following exposure. As part of our investigation, an attempt was made to control for the confounding effects of influenza epidemics by introducing an incidence variable for this disease. In the case of cardiovascular mortality owing to Cold, the degree of association was found not to be great enough for it possibly to constitute a significant confounding influence on the statistics for deaths owing to respiratory infections. Nevertheless, we cannot rule out the possibility of respiratory infections having a certain influence on cardiovascular mortality.

The relative effect of the ‘Cold’ variable on mortality due to respiratory diseases is considerable during the two seasons studied. This raises the question of the involvement of another related factor such as an increase in respiratory infections other than influenza e.g. pneumonia. One possible mechanism could be that breathing fresh air causes a bronchoconstriction which could in turn increase the susceptibility to, or the risk of contracting, a pulmonary infection. Respiratory diseases also have a greater association with the ‘Heat’ variable, especially during the warmer months. The fact that these effects occur within a short space of time indicates a fast-acting mechanism. The discovery that damp weather is a protective factor suggests that the inhalation of dry and hot air is detrimental to the respiratory system due, perhaps, to the presence of irritant substances. It is possible that there may exist some other confounding factor not controlled for in this study, possibly respiratory infections other than influenza.

No comparable studies to ours exist for high temperatures. The majority of investigations have centred on the effect of extreme phenomena (heatwaves) on mortality. It is worth mentioning that the ‘Heat’ variable as defined in our study cannot be used as an indicator of extremes of temperature, as temperatures only ranged between 24°C and 30.4°C and only one day had a mean temperature over 30°C. Our results concur with those of Kunst et al. in Holland. The causal group having the strongest association with ‘Heat’ is respiratory diseases. Furthermore, the effects of heat have a more rapid onset than those of cold. During the investigation carried out in Barcelona by Sáez et al. a greater association was found with cardiovascular and respiratory mortality, especially in this latter group. In the Dutch study and similarly in ours, the effect of Heat on cardiovascular mortality is not so evident. In the investigation carried out by Pan in Taiwan, the relationship between heat and mortality was noticeable above 26–29°C, and was only found to be statistically significant for average temperatures above 32°C.

The relationship between mortality and temperature (as much for the ‘Heat’ variable as for the ‘Cold’ variable) was particularly apparent among the elderly. This
could be due to the low tolerance of variations in temperature found in this group. As Pan et al. suggest, lower thermoregulatory capacity and reduced temperature sensitivity in older subjects could lessen their ability to maintain normal body temperature and therefore expose them to a greater risk of developing hyperthermia or hypothermia, which could in turn reinforce the risk factors and trigger mechanisms of cardiovascular disease.

On examination of the relationship between temperature and mortality we find that once other variables such as seasonality, air pollution, incidence of influenza, weekly periodicity, holidays and humidity have been controlled for, and, taking into account the autocorrelation (including the autoregressive terms) the 'V-shape' persists during the cooler months as much as during the warmer ones. In other words, even in colder months, warmer days are associated with increased mortality. This minimum, unlike in the case of the whole study period, is no longer closer to the higher temperatures, rather it is nearer to the mean temperature corresponding to each semester.

The finding of the association between high winter temperatures and mortality has only been reported on a few occasions. The Barcelona study described the link between occasional periods of at least 3 consecutive days of raised ambient temperature with raised mortality. This positive association occurs independently of the negative association with mortality exhibited during the winter months. We have not uncovered any other hypothesis for a physiopathogenic mechanism apart from the part played by the high temperatures during the cold months and the cold temperatures during the warmer months other than the previously mentioned acclimatization process. In this respect, Kalkstein argues that if one assumes that acclimatization is possible within the period of one summer, it is probable that people could acclimatize to those resulting from global warming. If so, this could lessen the health effects of such a global temperature increase. In any case, this is a complex topic and, as such, it is difficult to make any predictions in this area based only on a limited number of studies. As the Barcelona study sets out, one could speculate on two of the health consequences of global climatic change. On the one hand, the relationship between mortality and raised atmospheric temperatures, even during the cold months suggests that an increase in temperatures will result in increased mortality. On the other hand, the relationship with cooler temperatures, as reflected by the marked seasonality of the mortality series could compensate for the earlier increase. In any case, predictions in this domain are a complicated matter when the base of knowledge rests on a bedrock of only a few studies.

Finally, it seems worth mentioning that measures necessary to prevent the harmful effects of temperature fluctuations should include those aimed at the entire population as well as those more specifically aimed at vulnerable groups. The former could include adapting homes (including the installation of air-conditioning), urban design which avoids the formation of ‘thermal islands’ and initiation of meteorological warning systems such as those which presently exist for the forecasting of significant quantities of wind and rainfall. The cooperation of the authorities responsible for meteorology, civil defence and public health could lead to the implementation of measures to reduce the impact of extremes of temperatures on the population and improvement in the general population’s health education. More specifically, necessary actions to be taken by high-risk groups such as the elderly, young children, those with chronic diseases e.g. cardiovascular and respiratory, or those who may be taking certain prescribed drugs e.g. anticholinergics, diuretics etc., could be identified and implemented. Given the impact on the mortality figures of low temperatures, measures have been put forward that would focus on those patients at risk of suffering from ischaemic heart disease or stroke. For example, Green et al. propose the holding of clinical trials on the modification of antihypertensive treatment according to season. Interventions relating to risks originating from acute variations in temperature could have a positive impact in the field of public health.

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