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ORIGINAL ARTICLE

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Heat waves in Madrid 1986–1997: effects on the health of the elderly

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Abstract ***Objective***: The objective of this paper is to analyse and quantify the effects exerted on summer mortality by extremes of heat, particularly among persons aged 65–74 and 75 years and over, groups in which mortality is higher. ***Methods***: The study included the period from 1 January 1986 to 31 December 1997, for all people aged over 65 years resident in Madrid, based on mortality due to all causes except accidents (ICD-9 codes 1–799), and circulatory (390–459) and respiratory (460–487) causes. Meteorological variables analysed were: daily maximum temperature, daily minimum temperature and relative humidity. To control the effect of air pollution on mortality we considered the daily mean values of sulphur dioxide (SO_2), total suspended particulate (TSP), nitric oxides (NO_x), nitrogen dioxide (NO_2) and tropospheric ozone (O_3). Univariate and multivariate ARIMA models were used. Box-Jenkins pre-whitening was performed. ***Results***: The results yielded by this study indicate a mortality increase up to 28.4% for every degree the temperature rises above 36.5 °C, with particular effect in women over the age of 75 years and circulatory-cause mortality. The first heat wave that leads to the greatest effects on mortality, due to the higher number of susceptible people and the duration of the heat wave, show an exponential growth in mortality. Furthermore, low relative humidity enhances the effects of high temperature, linking dryness to air pollutants, ozone in particular. ***Conclusions***: Since a warmer climate is predicted in the future, the incidence of heat wave should increase, and more comprehensive measures, both medical and social, should be adopted to prevent the effects of extreme heat on the population, particularly the elderly.

Keywords Heat waves · Mortality · Elderly

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Introduction

A number of studies conducted in the Madrid Autonomous Region have analysed seasonal daily mortality patterns with reference to a series of environmental variables, the most relevant of which proved to be those of a meteorological nature, temperature in particular [2, 26]. The above-mentioned studies found that in the case of Madrid there was a V-shaped functional relationship between the variables of temperature and mortality, akin to that observed for other places where similar climatic conditions prevail [5, 21, 31]. Analysis of the causes of mortality underlying this relationship showed that, whereas excess winter mortality was principally due to respiratory and cardiovascular diseases, excess summer mortality was fundamentally due to cardiovascular diseases [1, 29]. It would seem that the seasonal variations observed in blood pressure, fibrinogens and lipids [30, 36, 39, 40] might explain the relationship between temperature and cardiovascular-disease mortality, particularly at higher latitudes [11].

While the observed temperature-mortality relationships may not be exclusive to our region, what is different is the importance of the peak of excess summer mortality peculiar to regions with marked extremes of heat [13, 22, 23]. Of course, there may well be environmental factors at work, other than temperature, that contribute to such excess summer mortality [19, 32, 33]. Nevertheless, the importance of the phenomenon is clear, with some authors [18] ranking these so-called heat waves as the leading cause of death due to natural disasters. Moreover, there are studies which have

focused on the effects of climatic change on health, based on models that estimate the probable greenhouse effect. These studies envisage a sharp rise in summer mortality, even if people acclimatise to the increased warmth, and a slight fall in winter mortality [18, 25]. Thus, a sizable net increase in weather-related mortality is estimated if the climate warms as the models predict [18], as well as an increased frequency for such extremes of heat in the coming years [15, 37].

In another way, the Madrid community shows special population characteristics that make this study interesting, with a mean life expectancy in the analysed period of 75.68 years for men and 82.97 years for women [14]. People over 65 years of age number 234,574, of whom 93,891 are men and 140,683 are women [14].

There would seem, therefore, to be a clear need for the type of study presented here, i.e., one that principally seeks to analyse and quantify the effects exerted on summer mortality by extremes of heat, particularly among persons aged 65–74 and 75 years and over, groups in which mortality is higher [1], and the contribution made by heat could thus be expected to be proportionately greater [22, 23, 29, 36, 39, 40]. Studies suggest that physiological as well as behavioural changes contribute to the increased vulnerability of the elderly to temperature-related mortality. Diminished perception, impairment of thermoregulatory capacity, and reduced ability to detect temperature change with an associated lack of precision in adjusting the thermal environment have been reported [6, 7, 22, 34]. Detection of a threshold above which the effects on a population's health become more marked is a necessary prerequisite for the adoption of any type of preventive measures targeted at minimising such effects.

Thus, the study objectives are: firstly, to detect the highest temperature threshold that produces effects on the health of people over 65 years old; secondly, to analyse and quantify these effects to adopt preventives measures.

Materials and methods

Mortality

The mortality data employed represent daily deaths of registered residents in the Madrid municipal area for the period 1 January 1986–31 December 1997. These data were furnished by the Madrid Regional Inland Revenue Authority (Consejería de Economía y Hacienda). The study was based on mortality due to all causes except accidents (ICD-9 codes 1–799), which will be referred to below as organic-cause mortality; cardiovascular or circulatory disease [390–459]; and respiratory disease (460–487). The study was broken down by age group (65–74 and over 74) and gender. The mortality variables were, therefore, as follows:

- Orgh65: organic-cause mortality among men aged 65–74 years.
- Orgm65: organic-cause mortality among women aged 65–74 years.
- Circh65: circulatory-cause mortality among men aged 65–74 years.
- Circm65: circulatory-cause mortality among women aged 65–74 years.
- Resph65: respiratory-cause mortality among men aged 65–74 years.
- Respm65: respiratory-cause mortality among women aged 65–74 years.

Likewise, analogous variables were created for the over-74 age group, namely Orgh75, Orgm75, Circh75, Circm75, Resph75 and Respm75.

Environmental variables

Meteorological variables

Data on meteorological variables were supplied by the National Meteorology Institute and based on readings taken at the Madrid-Retiro Observatory, selected for its inner-city location. The variables analysed were: daily maximum temperature (T_{max}), daily minimum temperature (T_{min}) and relative humidity at 7 a.m. (RH).

Air pollution variables

To control for the effect of air pollution on mortality in Madrid [3, 9, 10], we included in the study a series of variables, consisting of the daily mean values of the following pollutants: sulphur dioxide (SO_2), total suspended particulate (TSP), nitric oxides (NO_x), nitrogen dioxide (NO_2) and tropospheric ozone (O_3). These values were obtained as the mean of the combined readings for all 24 stations making up the Madrid municipal air-pollution monitoring grid. Measurement of photochemical pollutant data (NO_x, NO_2 and O_3) began on 1 January 1990.

Other variables

Account was also taken of the existence of other variables that might have an effect on mortality, an example being ''influenza epidemic'' (g1), a dichotomic variable equalling 1 in epidemic periods and zero in periods when there was no epidemic. These data were drawn from the Statutorily Notifiable Disease registers and furnished by the Madrid Regional Public Health Authority (Consejería de Sanidad).

In order to control for possible periodicities, we created circular variables in the form of sine and cosine functions of 365-day (S_{365} and Co_{365}), 180-day (S_{180} and Co_{180}), 90-day (S_{90} and Co_{90}) and 60-day periods (S_{60} and Co_{60}).

Previous studies [3, 9, 10] targeting the city of Madrid highlighted the fact that the relationships between mortality-pollution and mortality-temperature were not linear. In order to establish this type of functional relationship, the relevant scatter-plot diagrams were drawn. In the case of temperature a scatter-plot diagram of this nature also serves to define the so-called heat wave, as corresponding to the daily temperature above which a clear upturn is observed in the mortality-temperature curve. The functional relationships so detected serve as a basis for the creation of other indirect variables.

To enable quantitative analysis of the effect of maximum temperature on heat wave days, the following two new temperature variables were defined:

$$T_{hwave} = T_{max} - 36.5 \text{ if } T_{max} > 36.5\,^{\circ}C$$

$$T_{cwave} = 36.5 - T_{max} \text{ if } T_{max} < 36.5\,^{\circ}C$$

Following a similar procedure to that outlined above, scatter-plot diagrams were also drawn for mortality due to different causes and pollutants, proving linear for NO_2 and NO_x, logarithmic for

SO_2, and quadratic, with the minimum point at 35 $\mu g/m^3$, for the concentration of daily mean ozone. This led to the creation of the variables, L_{SO2}, defined as the Neperian logarithm of SO_2, and

$$O_3a = 35 - O_3 \text{ if } O_3 > 35\,\mu g/m^3$$

$$O_3b = O_3 - 35 \text{ if } O_3 < 35\,\mu g/m^3$$

To detect the effect of possible acclimatisation to heat, in other words, whether the effects of any heat wave might be greater depending upon the chronological order in which it occurred during the year (i.e., whether the wave in question was the first heat wave of the summer, etc.), we created the variable, ''wave number''. Similarly, to observe the effect of the duration of such waves, we created the variable, ''dur'', to measure the number of days during which the temperature exceeded the designated threshold.

For analysis of the deterministic components of the series of the respective study variables, the pertinent basic descriptive statistics were ascertained. Trend and periodicities were analysed using the relevant frequency spectra yielded by the Fast Fourier Transform method [16].

Univariate ARIMA modelling [24] was used to ascertain the non-deterministic components of the series, i.e., the autoregressive (AR) part and moving average (MA). Using the Box-Ljung Portmanteau test, we selected those models where their partial (PACFs) and simple autocorrelation functions (ACFs) indicated white-noise structure.

Box-Jenkins pre-whitening was performed to eliminate analogous periodicities and autocorrelations as between the mortality and temperature series [24]. When the series had thus been pre-whitened, the cross-correlation functions (CCFs) between their sets of residuals were then calculated, thereby establishing the lags at which significant relationships between the variables had occurred. This procedure led to lag variables being created for all the above environmental variables.

Once the mortality/environmental-variable association had been established, in order to eliminate the effects of possible collinearities existing between environmental variables we constructed ARIMA models of the mortality variables, with the environmental variables being included as exogenous inputs [24]. Goodness-of-fit was evaluated by the running of the Box-Ljung Portmanteau test on the PACF and ACF residuals.

In the process of multivariate modelling, and given that the goal of the study was to analyse the effect of high temperatures on mortality, selection was restricted to the months June–September, a period when maximum temperatures are registered in Madrid.

The final models were submitted to a sensitivity analysis to test its time stability. This model examines the whole series, removing 1 or 2 years and determines whether the estimator values suffer statistically significant variations.

For calculation purposes we used the statistical computer software package, SPSS for Windows, version 9.0.

Results

The deterministic components of the mortality series and environmental variables are shown in Tables 1 and 2, respectively.

Scatter-plot diagrams were drawn, depicting maximum temperature and the various specific causes of mortality, broken down by age group and gender, for the summer months and for those days in Madrid on which all-cause mortality for all groups surpassed the 95th percentile of the series (100 deaths/day). The graphs showed a marked inflection point, with a rise in mortality at or around 36.5 °C, regardless of cause, age group or gender, with this effect proving more pronounced for women versus men; persons over the age of 75 years; and circulatory causes. By way of example, Fig. 1 shows the scatter-plot diagram for daily circulatory-cause mortality in women over the age of 75 and

Table 1 Descriptive statistics for the mortality series

Variable	Maximum	Minimum	Mean	SD	Trend	Periodicities
Organic: men 65–74	27	1	11.2	3.6	Yes	Annual, 6, 3–4 months, 3 days
Organic: women 65–74	19	0	6.6	2.6	No	Annual, 6, 3–4 months 10, 7, 3–4 days
Circulatory: Men 65–74	11	0	3.5	1.9	No	Annual, 6months, 15, 3–4 days
Circulatory: women 65–74	10	0	2.3	1.6	Yes	Annual, 4–5 months, 13, 3–4 days
Respiratory: men 65–74	8	0	1.1	1.1	Yes	Annual, 6, 4–5 months, 12, 3–4 days
Respiratory: women 65–74	0	4	0.4	0.6	Yes	Annual, 4–5 months, 10, 7, 3–4 days
Organic: men > 75	45	5	19.8	5.5	Yes	Annual, 6, 4 months, 3–4 days
Organic: women > 75	72	6	28.6	7.3	Yes	Annual, 6, 3–4 months, 3–4 days
Circulatory: men > 75	20	0	7.6	3.0	Yes	Annual, 6 months, 3–4 days
Circulatory: women > 75	41	2	14.2	4.6	Yes	Annual, 6, 3–4 months, 3–4 days
Respiratory: men > 75	13	0	3.1	2.0	Yes	Annual, 6, 3–4 months, 3–4 days
Respiratory: women > 75	17	0	2.9	2.0	Yes	Annual, 6, 3–4 months, 3–4 days

Table 2 Descriptive statistics for the environmental variables (T_{max} daily maximum temperature, T_{min} daily minimum temperature, RH relative humidity at 7 a.m., TSP total suspended particulate, SO_2 sulphur dioxide, NO_x nitric oxides, NO_2 nitrogen dioxide, O_3 tropospheric ozone)

Variable	Maximum	Minimum	Mean	SD	Trend	Periodicities
T_{max} (°C)	40.9	0.4	19.9	8.7	No	Annual
T_{min} (°C)	27.2	–7.6	10.3	6.5	No	Annual
RH (%)	100	10	78.3	16.1	Yes	Annual, 3–4 days
TSP ($\mu g/m^3$)	195	12.5	43.6	19.5	Yes	Annual, 6 months, 7, 3–4 days
SO_2 ($\mu g/m^3$)	401	6	52.0	43.2	Yes	Annual, 6 months, 7, 3–4days
NO_2 ($\mu g/m^3$)	172	26	72.4	21.6	Yes	Annual, 6 months, 7, 3–4 days
NO_x ($\mu g/m^3$)	688	44	189.5	97.4	Yes	Annual, 6 months, 7, 3–4 days
O_3 ($\mu g/m^3$)	78	0	22.8	14.3	Yes	Annual, 6 months, 7, 3–4 days

maximum temperature. Analogous results were obtained for T_{min}, with an inflection point in the region of 23.5 °C. In the case of RH, the relationship proved to be linear, without any marked inflection points.

The above scatter-plot diagrams served to define a heat wave in Madrid as any day on which the maximum temperature exceeded 36.5 °C, as measured at the Madrid-Retiro Observatory.

In the period June–September for the years under review, the daily maximum temperature equalled or exceeded 36.5 °C on 74 days, corresponding to 5% of all cases, with the longest heat wave being that of July 1995, which lasted for 11 consecutive days and registered a maximum temperature of 40.9 °C. The daily mean organic-cause mortality in the over-75 age group during this heat wave was 71 deaths/day versus a mean of 42 on summer days on which the temperature failed to rise above the 36 .5°C threshold.

Figures 2 and 3, respectively, show the bar charts corresponding to the effect of the chronologically numbered heat waves in the year, the variable ''wave number'', and its duration in days, the variable ''dur''.

Figure 4 depicts the CCF between circulatory-cause mortality in women over the age of 75 and maximum temperature.

On establishing the lags at which significant associations had occurred between temperature and the different causes of mortality, we then constructed ARIMA models, with all the environmental, circular and other control variables mentioned in the Methods section above being included as exogenous variables. We thus analysed the effect of temperature on mortality, controlling for the different confounding variables. Tables 3 and 4 list the estimators of the variables that proved significant for organic-, circulatory- and respiratory-cause mortality, broken down by age group and gender. The value of the estimator indicates the rise in deaths/day due to this cause, induced by a unit-rise in the value of the variable. For instance, in the case of organic-cause mortality among women over the age of 75 years, a value of 8.15 for the T_{hwave} estimator would indicate that for every degree by which the daily maximum temperature exceeded 36.5 °C, daily mortality would rise by 8.15 persons, and a TSP estimator of 0.15 would similarly indicate that for every $\mu g/m^3$ rise in particulate concentration, daily mortality would rise by 0.15 persons/day.

The sensibility analysis carried out shows that the obtained models are stable in time.

To compare the effect of maximum heat-wave temperature on cause-specific mortality by age group and gender, we calculated the percentage increase in mortality for every degree that daily maximum temperature exceeded 36.5°C, with the daily mean values in the period under review being taken as reference. The results are set out in Table 5.

Discussion

The temperature of 36.5 °C, above which there is a sharp rise in daily mortality in Madrid, is slightly lower than that reported by other authors for Japan [28], 38 °C, or for Chicago, 37.8 °C, [38] and considerably lower than that cited by Smoyer in the case of St Louis, Missouri, 40.6 °C [35], thereby implying that the phenomenon of acclimatisation to heat by the local population in any given place [20] plays an important role. Moreover, the results yielded by this study indicate that

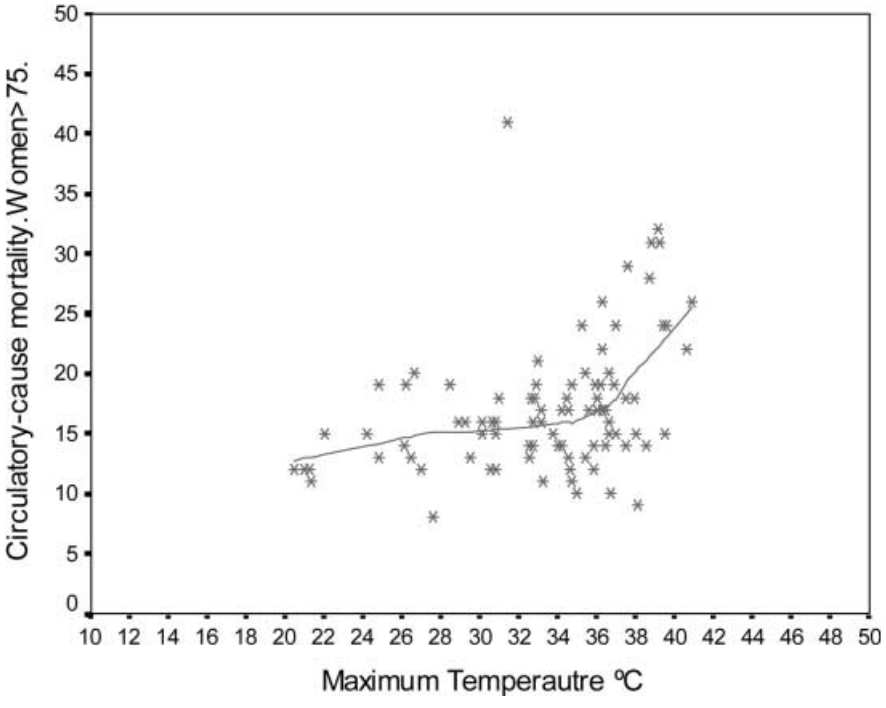

Fig. 1 Scatter-plot diagram for daily circulatory-cause mortality in women over the age of 75 and maximum temperature

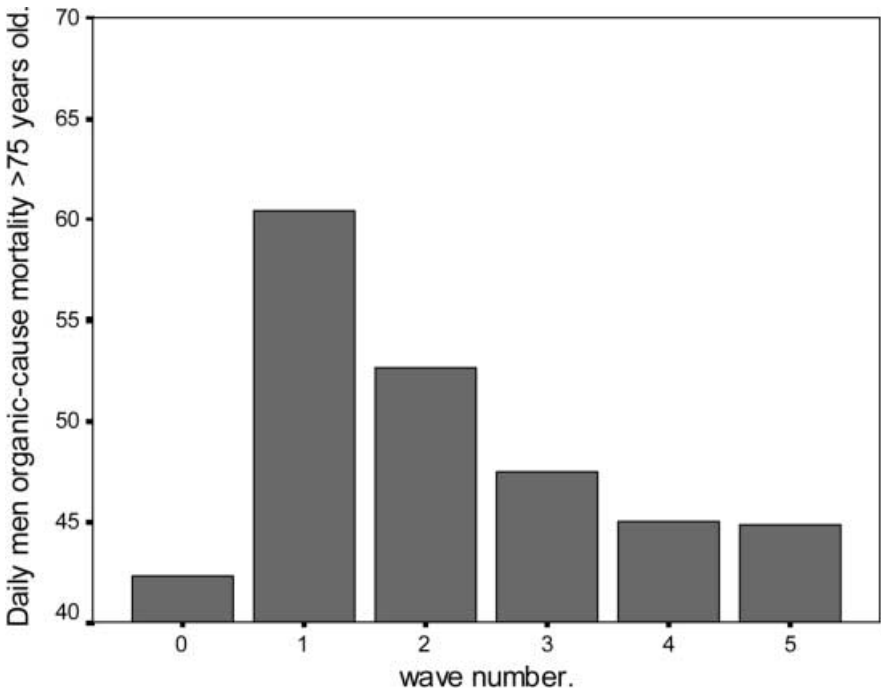

Fig. 2 Bar chart corresponding to the effect of the chronologically numbered heat waves in the year

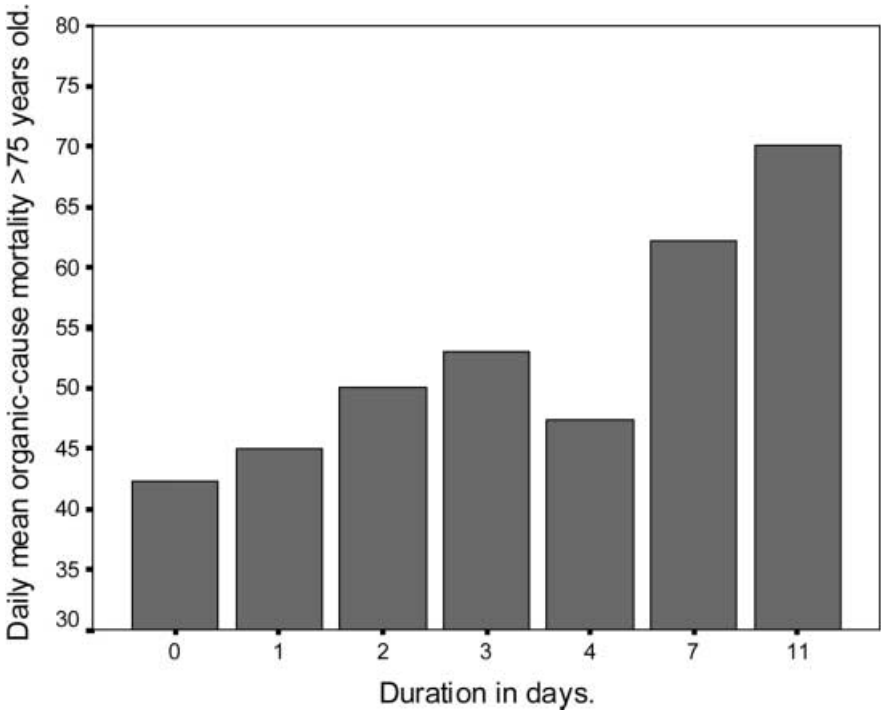

Fig. 3 Bar chart corresponding to the effect of the duration in days of heat wave

during heat waves, the over-75 age group registered an excess mortality of 26%, a figure that climbed to 78% in the case of the heat wave in 1995, results similar to those reported for the 1995 Chicago heat wave by Whitman et al. [38].

The results shown in Fig. 2 highlight the fact that it is the first heat wave that leads to the greatest effects on mortality, due to the higher number of susceptible people. However, whether the first heat wave occurs in June or August makes no difference insofar as mortality-related effects are concerned, meaning that the effect of there being a higher number of susceptible people predominates over any possible acclimatisation to heat during the course of the summer. What Fig. 3 clearly shows is the exponential growth of mortality attributable to the duration of the heat wave, a finding similar to that reported for Japan by Nakai et al. [28].

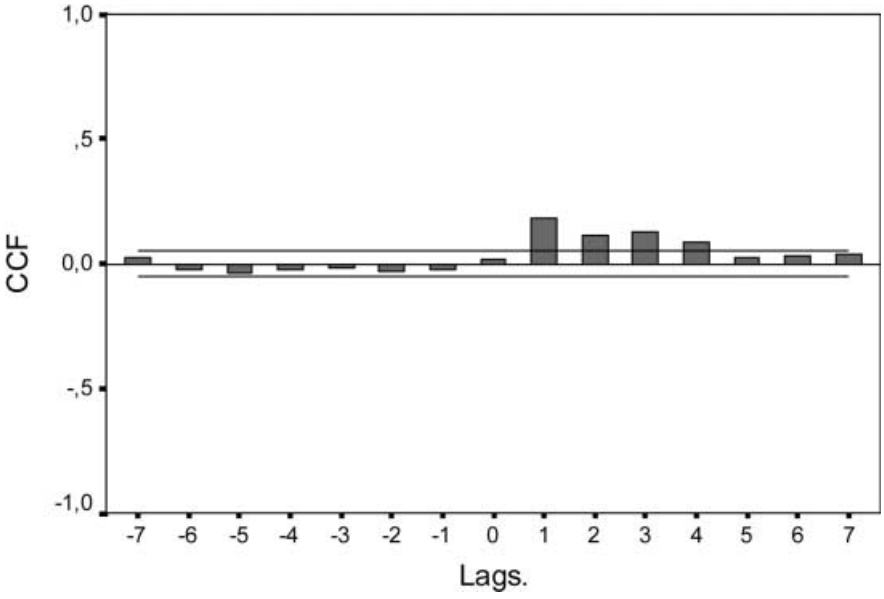

Fig. 4 Cross-correlation function (*CCF*) between circulatory-cause mortality in women over the age of 75 and maximum temperature

The functional relationships observed for relative humidity and air pollutants are comparable to those described in other Madrid-based studies [3, 9, 10]. A factor that may, perhaps, differentiate the behaviour of environmental variables vis-à-vis mortality in Madrid is the role played by relative humidity. Some authors [8] have found high relative humidity to be a factor which contributes to the increase in mortality from heat, due to the fact that it hinders evaporation of perspiration and so prevents the body from cooling. In a similar vein, research undertaken in the US with the aim of implementing preventive measures [17], defines a temperature modified by relative humidity, in the sense that, when relative humidity rises ever higher, it is as if ambient temperature were also rising at the same time. Other authors [21] contend, however, that the role of perspiration in the lowering of body temperature has been exaggerated, and indeed report results in which low relative humidity enhances the effects of high temperature. Sartor et al. [33] arrive at similar conclusions, linking dryness to air pollutants, ozone in particular, exerting a much greater effect. In the case of Madrid, the contribution made by relative humidity would tend in this direction, namely, the lower the relative humidity,

Table 3 Significant coefficients and lags (in parentheses) for every model. Mortality for the 65–74 age group (O_{3h} O_3 values higher than 35 μg/m^3, T_{hwave} T_{max} values higher than 36.5 °C, T_{cwave} T_{max} values lower than 36.5 °C, *TSP* total suspended particulate, L_{SO2} logarithmic sulphur dioxide, *RH* relative humidity)

Gender	Organic	Circulatory	Respiratory
Men	T_{hwave} (1, 3) = 1.65*** T_{cwave} (4) = 0.08*** O_{3h} (5) = 0.04*	T_{hwave} (3) = 0.33*	T_{hwave} (4) = 0.19* T_{cwave} (12) = 0.01* RH (4) = –0.01*
Women	T_{hwave} (1, 2) = 1.07** T_{cwave} (13) = 0.05** TSP (0) = 0.01*	T_{hwave} (2) = 0.27** L_{SO2} (1) = 0.29***	T_{hwave} (1) = 0.14**

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$

Table 4 Significant coefficients and lags (in parentheses) for every model. Mortality for the higher than 75 age group (T_{hwave} T_{max} values higher than 36.5 °C, T_{cwave} T_{max} values lower than 36.5 °C, *TSP* total suspended particulate, L_{SO2} logarithmic sulphur dioxide, *RH* relative humidity, NO_2 nitrogen dioxide)

Gender	Organic	Circulatory	Respiratory
Men	T_{hwave} (1, 2) = 2.51** T_{cwave} (13) = 0.09** RH (4) = –0.02* TSP (0) = 0.02* L_{SO2} (1) = 0.96	T_{hwave} (1) = 0.71*** T_{cwave} (13) = 0.05** RH (2) = –0.01** TSP (0) = 0.01*	T_{hwave} (1, 4) = 0.81** RH (9) = 0.01* NO_2 (0) = 0.01***
Women	T_{hwave} (1, 3, 4) = 8.15*** TSP (0) = 0.15*** L_{SO2} (1) = 1.52* NO_2 (3) = 0.03**	T_{hwave} (1, 3, 4) = 4.85*** TSP (0) = 0.06*** NO_2 (2) = 0.02**	T_{hwave} (1) = 0.51*** RH (3) = –0.01* TSP(0) = 0.01***

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$

Table 5 The percentage contribution of the T_{hwave} effect on daily mean mortality during the study period

Gender	Organic (%)	Circulatory (%)	Respiratory (%)
Men 65–74 years	14.7	9.4	17.2
Women 65–74 years	16.2	11.7	23
Men > 75 years	12.6	9.3	26.1
Women > 75 years	28.4	34.1	17.6

the higher the summer mortality. Apart from the arguments put forward by Sartor et al. above [33], the reason for this might perhaps lie in the use of the relative humidity parameter, which not only takes into account the vapour content of the atmosphere but also measures the degree of saturation of the air, something that varies with temperature [27]. Furthermore, studies targeting Madrid [12] indicate that the highest temperatures are caused by the advection of air from the SE, meaning hot dry air proceeding from Africa and so having a low humidity content. In contrast, rain-bearing winds tend to come from the west and are associated with temperatures that are not quite as high.

The results shown in Tables 3 and 4 clearly highlight the effect of heat on mortality in the short term. From the perspective of the estimators, and thus in terms of the number of daily deaths, this effect is more pronounced among men in the 65- to 74-year age range, for all cause as well as for circulatory- and respiratory-cause mortality. Within this same age group the effect is greater, in both genders, in terms of circulatory- versus respiratory-cause mortality. In the over-75 age group however, the effect is especially pronounced: in women and in circulatory-cause mortality. Both these results are in line with other studies conducted both in Madrid [2, 26] and in other Spanish cities [4, 31].

A further finding warranting mention is the effect on mortality of temperatures below 36.5 °C (T_{cwave}). This effect, which in quantitative terms is far smaller than that of heat, could be explained by two factors: firstly, there is the fact that in summer low temperatures do indeed have an effect upon mortality through a variety of physiopathological mechanisms; and secondly, there is the presence of a harvester effect, whereby the decrease in the number of susceptible people after the first episode of extreme heat means that mortality inevitably falls, despite subsequent rises in temperature. Similar results have been obtained on other occasions by different authors [2, 21, 26].

The percentage contribution of the T_{hwave} effect on daily mean mortality during the study period is shown in Table 5, which establishes that the effect is greater among women than among men, and that in the 65-year age group the greatest percentage increase is registered by respiratory causes. Among men over the age of 75 years, it is again respiratory-cause mortality that registers the greatest increase. In the case of women in this age group however, the effect of temperature on circulatory-cause mortality is double that for respiratory-cause mortality.

The results yielded by this study, indicating an increase in mortality of up to 28.4% for every degree that the temperature rises above 36.5 °C, clearly highlights the need to implement preventive measures designed to minimise the effects of extreme heat on the population, with particular attention to persons over the age of 75 years.

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