European Journal of Epidemiology **14**: 571–578, 1998.

Daily mortality in Madrid community 1986–1992: Relationship with meteorological variables

Juan Carlos Alberdi, Julio Díaz, Juan Carlos Montero & Isidro Mirón
Centro Universitario de Salud Pública de Madrid, Madrid, Spain

Accepted in revised form 5 May 1998

Abstract. Daily mortality displays a seasonal pattern linked to weather, air pollution, photoperiod length, influenza incidence and diet, among which temperature ranks as a leading cause. This study thus sought to assess the relationship between temperature, relative humidity, wind speed and mortality in the Madrid Autonomous Region (Spain) for the period January 1986–December 1992, controlling for the effects of air pollution and influenza incidence. Daily data on maximum, minimum and 24-hour mean temperature, relative humidity and wind speed were matched against daily mortality. Transfer function was identified using the Box–Jenkins pre-whitening method. Multivariate time series regression models were used to control for the confounding effects of air pollution and influenza incidence. Separate seasonal analyses were carried out for winter and summer periods. A J-shaped relationship between outdoor temperature, relative humidity and daily mortality was found. Mortality proved to be inversely related to cold temperature (4- to 11-day lag) and directly related to warm temperature (1-day lag). High relative humidity during summer periods was negatively related to mortality. Thermal variation ascribable to Madrid's mesothermal Mediterranean climate was strongly related to daily mortality, even where air pollution and influenza incidence were controlled for.

Key words: ARIMA, Mortality, Weather

Abbreviations: ACF = autocorrelation function; AR = autoregressive; CCF = cross-correlation function; MA = moving average; MC = Madrid community; PACF = partial autocorrelation function

Introduction

Owing to economic growth, the seasonal mortality pattern has changed [1], currently registering a winter peak [2] the size of which varies with calendar time and geographical location [3]. This peak has been associated with a decrease in photoperiod length, changes in diet, influenza epidemics and temperature [4]. Statistically significant correlations have been detected between outdoor temperature and daily deaths due to cardiovascular, cerebrovascular [5], respiratory [6–8] and coronary heart diseases [9–11]. Recent studies have reported an association between moderate air pollution levels and daily mortality [12–17].

A summer peak, smaller than its winter equivalent, has been found in certain developed countries where the appropriate climatic features prevail. This increase in summer mortality has been linked to high temperatures and different atmospheric parameters, such as relative humidity and air pollution [18]. Persons most contributing to such summer excess mortality have been described in age group and sex [19, 20].

Mention should likewise be made of the potential health-related effects of the global climate change thought to be currently affecting the world. According to the experts, such a change would be characterised by wider temperature fluctuation, and an ensuing increase in heat waves and cold spells [20, 21]. The World Health Organisation envisages these effects as constituting one of the main problems for the 21st century [22].

Previous mortality studies [20] conducted in the Madrid Autonomous Region (MAR), have shown the marked influence which extreme temperatures exert on daily deaths. With a view to its being used as a tool for prevention purposes, this paper now seeks to analyse the association between daily deaths and atmospheric variables (temperature, relative humidity and wind speed), while controlling for the confounding effects of atmospheric pollution and influenza incidence [24]. All results pertaining to the association between air pollution and mortality are presented in another paper.

Methods

Mortality

Data on daily deaths among MAR residents for the period 1 January 1986–31 December 1992 were obtained from the Madrid Regional Authority Economics Advisory Board. All deaths of residents occurring outside the Madrid Region were excluded. Daily deaths were analysed by the following causes (all references to ICD-9 codes): organic diseases (1–799), cardiovascular diseases (390–459), coronary heart diseases (410–414), cerebrovascular diseases (430–438) and respiratory diseases (460–487). Deaths were also analysed by age group (65 year and older) and sex.

Weather

The National Meteorological Institute supplied data on minimum, maximum and 24-hour mean temperature and wind speed. Temperature was measured in degrees Celsius (°C). All data were based on the Madrid-Getafe Meteorological Station, the station showing the highest correlation with the overall average for the MAR monitoring grid [25]. Dry- and wet-thermometer temperatures and barometric pressure were used to calculate relative humidity [26].

Influenza epidemics were defined on the basis of viral detection in throat smear and influenza cases reported to the regional Notifiable Disease Surveillance System. A dummy variable was included for influenza epidemic weeks.

Air pollution data were furnished by the Municipal Monitoring Network. Previous cluster analysis had detected two homogeneous zones for total suspended particulates (TSP) and for sulphur dioxide (SO2). Air pollution series were obtained from two monitoring stations representing these zones. Twenty-four-hour TSP and sulphur dioxide averages were used in the analysis, with TSP being measured by attenuation of β radiation and SO2, by ultraviolet fluorescence absorption.

Each variable was firstly analysed for normality. Descriptive statistics for each variable were obtained. Spectral density function, simple autocorrelation function (ACF) and partial autocorrelation function (PACF) [27] were used to detect the deterministic components of the series (trend, season and cycle). To remove these components, two methods were used: differentiation (simple or seasonal) or circular-function modelling. ACF and PACF were used to identify the autoregressive (AR) or moving average (MA) components of the stationary series.

Scatterplots were used to detect the functional form of the relationship between the respective atmospheric variables and mortality. Temperature, wind speed and relative humidity were sorted in ascending order. Equal intervals were introduced containing consecutive observations. Mean daily mortality was plotted for each interval. Where indicated by the plots, independent variable transforms (log, square-root) were examined in the regression.

Given that the presence of autocorrelation in the series could confound the association with mortality, the Box–Jenkins pre-whitening method was used [27] to identify the transfer functions. Weather variables were modelled using different ARIMA models. White-noise residuals were obtained. Where more than one model fitted the series, the model having the smallest Akaike Information Criteria was selected. The selected model was applied to mortality in order to obtain a series of residuals, and a cross-correlation function (CCF) between both series of residuals, then computed. Once these CCF results had been obtained, mortality regression models, including all significant lagged weather-pollution variables, were developed.

Owing to the different mortality patterns associated with cold and heat, two analyses were carried out: November–March for cold and June–August for warm temperatures. CCFs between daily deaths and maximum, minimum and 24-hour mean temperature, relative humidity and wind speed at 7 a.m. were obtained for each period.

In order to control for the confounding effects of air pollution, influenza and season, variables with significant lagged values were included – one by one – in a multivariate (ARIMA) time-series regression model, using a forward method. Once the best model had been obtained, validity was assessed by residuals analysis, using the ACF, PACF and Box Ljung Portmanteau test. AR or MA components of the residuals were included in the final model.

Statistical analysis was performed using the BMDP statistical software package (modules 1T and 2T).

Results

Descriptive statistics for causes of death, broken down by sex and age group, are shown in Table 1. For each mortality series, variance was greater than the mean, suggesting a non-random variation. The scatterplots for all mortality series showed a trend and marked seasonal pattern. The spectral density of daily deaths due to organic and cardiovascular diseases (Figure 1a) registered a trend and two statistically significant peaks, corresponding to winter and summer excess mortality. Respiratory disease mortality

Table 1. Descriptive statistics of causes of death

Mortality due to	ICD-9		All		> 65	
			Mean	Variance	Mean	Variance
Organic	(1–799)	Men	43.1	75.7	28.9	49.0
		Women	40.3	77.4	33.6	65.6
		Total	83.4	213.2	62.5	161.3
Cardiovascular	(390-459)	Men	15.2	21.2	11.1	15.2
		Women	18.2	29.2	16.7	27.0
		Total	33.4	64.0	27.8	51.8
Ischaemic	(410–414)	Men	4.5	5.3	3.1	3.6
		Women	3.2	3.6	3.0	3.2
		Total	7.7	9.6	6.0	7.3
Cerebrovascular	(430–438)	Men	3.5	4.0	2.8	2.9
		Women	5.1	5.8	4.7	5.3
		Total	8.6	10.2	7.6	9.0
Respiratory	(460–487)	Men	4.7	6.8	3.8	5.3
		Women	3.4	4.4	3.0	4.0
		Total	8.0	15.2	6.8	12.3

showed only an annual periodicity (winter). Weekly cycles were not present for mortality. Attention should be drawn to the sex-specific difference in behaviour.

Table 2 sets out the mean and variance for each independent variable. Temperature and relative humidity exhibited annual periodicity without trend (Figure 1b). The relationship between temperature and mortality depicted a J-shaped distribution. Daily deaths were lowest at a minimum temperature of 15.4 °C, a maximum of 30.8 °C (Figure 2) and a 24-hour mean of 20.3 °C, with the relationship proving linear on either side of that point. The warm temperature side had a steeper slope than the cold. Minimum temperature registered the greatest correlation with mortality during the winter period, and maximum temperature with mortality during the summer period.

Relative humidity (RH) displayed a non-linear relationship with mortality. Lowest mortality corresponded to an RH of 70.4% at 7 a.m. Relative humid-

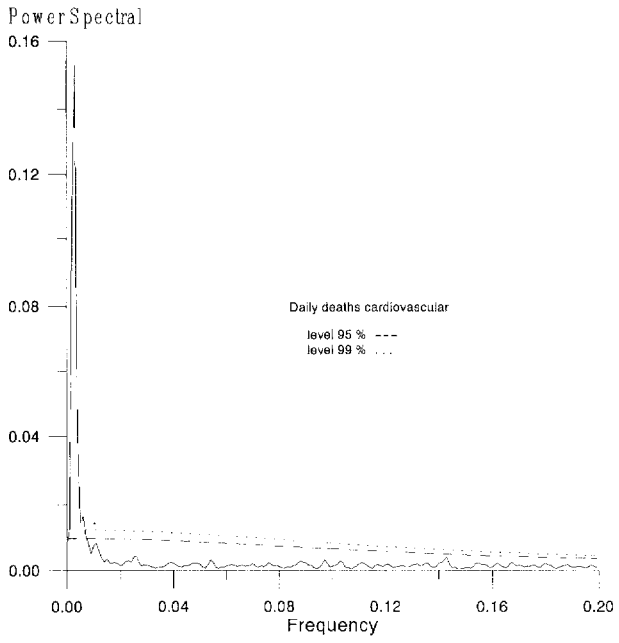

Figure 1a. The spectral density function of cardiovascular diseases.

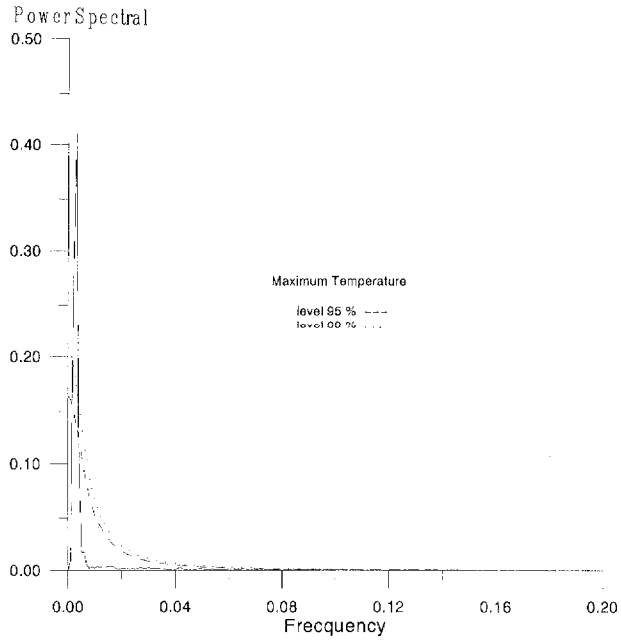

Figure 1b. The spectral density function of temperature.

Table 2. Mean and variance for each independent variables

		Maximum temperature (°C)	Minimum temperature (°C)	Relative humidity
Annual	Mean	20.7	8.8	86.8
	Variance	81.0	46.2	124.8
	Maximum	40.2	24.4	100
	Minimum	1.0	− 6.6	44.4
Winter	Mean	13.0	3.0	94.3
	Variance	16.8	15.2	26.8
	Maximum	26.4	13.0	100.0
	Minimum	1.0	− 6.6	59.1
Summer	Mean	31.5	16.8	73.8
	Variance	23.9	11.6	88.4
	Maximum	40.2	24.4	98.7
	Minimum	17.2	8.0	44.4

ity was inversely linked to temperature ($r = -0.78$), leading to a high collinearity problem in the regression analysis. Wind speed at 7 a.m. ($r = 0.004$) failed to attain statistical significance with respect to daily mortality.

During the summer period, the CCF between maximum temperature and death by organic causes (Figure 3a) presented a positive correlation up to 4 days later, but was only significant at lag 1. Deaths due to circulatory diseases plotted a similar shape yet no cross-correlation coefficient with temperature proved significant. While both sexes presented similar CCF patterns, the correlation coefficient at lag 1 was greater for women than for men. Those aged 65 years or older showed a similar behaviour pattern to that observed for all-age mortality. In this age group, circulatory disease mortality registered a positive and statistically significant coefficient at lag 1.

Maximum temperature was a significant predictor of mortality with a slope in lag 1 $\beta = 0.97$ ($p < 0.001$). Despite the high degree of collinearity between temperature and relative humidity, in joint regression RH remained a significant predictor of mortality. Relative humidity registered a negative relationship up to lag 3, indicating that a decrease in RH was accompanied by increased mortality. The slope in lag 2 $\beta = -0.11$ reached statistical significance ($p < 0.05$) (Table 3). The control variable (month) showed a decrease in mortality from June to August. The ACF and PACF of the residuals displayed a white-noise pattern. The Box–Ljung statistical value corresponded to $p = 0.848$ at lag 90.

During the winter period, the cross-correlation function between minimum temperature and organic-disease mortality registered a positive coefficient for the same day, and negative values in lags thereafter, with highest significant cross-correlation coefficients 7–11 days later (Figure 3b). Cardiovascular deaths exhibited a dual behaviour pattern. There was an immediate effect due to ischaemic heart diseases, followed by a delayed effect starting 10 days later. The CCF between temperature and respiratory diseases yielded a negative significant coefficient 9 days later.

The above applied across the sexes, yet the effect of minimum temperature on mortality was observed earlier in women than in men. This effect was more marked in those aged 65 and older. For males, temperature/mortality cross-correlation coefficients reached statistical significance 7 days later. The CCF with relative humidity did not prove significant in any lag.

In the regression analysis involving winter period,

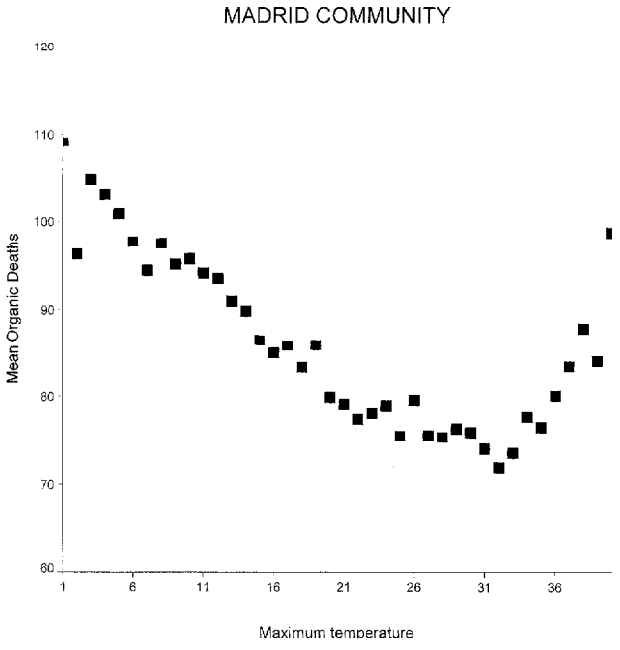

Figure 2. Relation between maximum temperature and mortality.

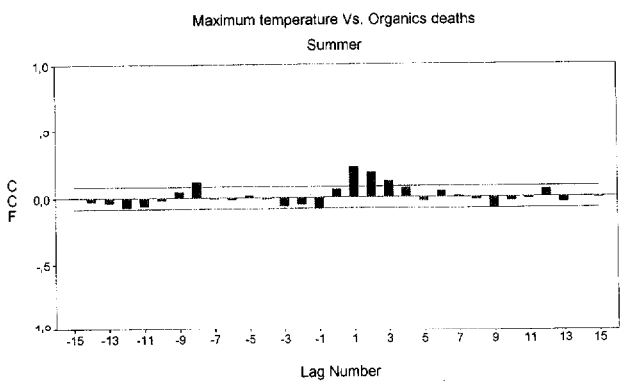

Figure 3a. CCF maximum temperature versus organics deaths.

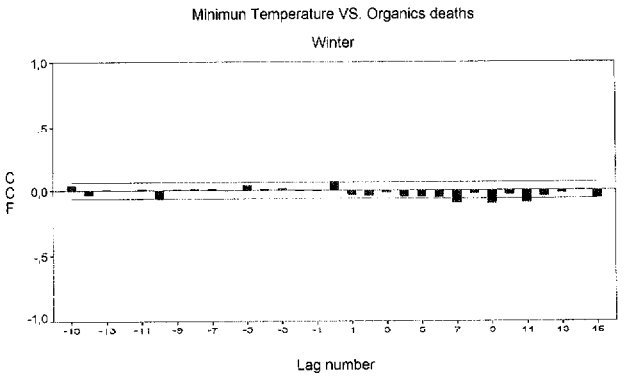

Figure 3b. CCF minimum temperature versus organics deaths.

the regression coefficient of influenza epidemic was $\beta = 5.48$ ($p < 0.001$). Temperature had an independent effect on mortality: a drop of 1°C during this period was associated with an increase of 1.07 deaths between lags 6 and 11 (Table 3). The residuals were autocorrelated and it was necessary to fit an ARIMA(1,1) model to achieve a white-noise structure.

Discussion

As regards the winter peak, daily mortality in the Madrid region (Figure 1a) presents a behaviour similar to that found elsewhere [4–6, 16, 22]. What seems to be specific to the MAR's rigorous climate is the existence of a second, summer peak. A similar study conducted in another Spanish city [28] with a more moderate climate, failed to find this type of periodicity.

The functional form of the relationship between temperature and mortality is consistent with previous analyses [5, 8, 24]. There is some disparity however as to the temperature that produces lowest daily mortality. Kunst [5], in the Netherlands, observed the lowest

Table 3. Association between weather variables and daily mortality by organic diseases in all winters and summers from 1986 to 1992. Madrid community, Spain

	Explanatory variables (deaths by organic diseases)			
	Winter (lags)[a]	Aggregate effect[b]	Summer (lags)[a]	Aggregate effect[b]
Total	Temp. (4): – 0.30** Temp. (7): – 0.39*** Temp. (9): – 0.23* Temp. (11): – 0.28*	Temp: – 1.20 TSP (1): 0.23*	Temp. (1): 0.97*** R.h. (2): – 0.11*	Temp: 0.97 R.h.: – 0.11 ln SO2 (1): 1.93*
Men	Temp. (4): – 0.20** Temp. (7): – 0.17* Temp. (9): – 0.19*	Temp: – 0.56 TSP (1): 0.11**	Temp. (1): 0.22*	Temp: 0.22 ln SO2 (1): 1.17*
Women	Temp. (5): – 0.18* Temp. (7): – 0.23** Temp. (11): – 0.22**	Temp: – 0.63 R.h.: 0.13	Temp. (0): 0.48*** Temp. (1): 0.43*** R.h. (1): – 0.08*	Temp: 0.91 R.h.: – 0.08
R.h. (0): 0.13**	TSP (1): 0.13***			ln SO2 (1): 1.23**

[a] = change in daily mortality by one unit increase in explanatory variable (k-lags distributed).
[b] = the sum of the effect associated with the lag periods.
* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.
Temp. = maximum temperature (°C).
R.h. = relative humidity (%).

number of deaths at a 24-hour mean daily temperature of around 16.5 °C, versus the temperature of 20.3 °C recorded for the MAR. Other studies [5, 28] pinpointed lowest mortality for the maximum temperature as lying in a range between 20–25 °C, as opposed to 30.8 °C for Madrid, a finding comparable to the 29 °C and 30 °C reported by Wen-Harn [8] for Taiwan and Katsouyanni et al. [24] for Greece, respectively. These differences appear to be related to geographical latitude [1, 2, 7, 8, 22, 29]. In the USA, this point changes from north to south, ranging between 15–26 °C in cold and 26–32 °C in warm zones [22].

Warm temperatures higher than this point lead to an increase in mortality. The way in which people react to heat is related to where they are, with the maximum effect in areas with moderate climates [22]. High-temperature periods in such areas cause a large increase in mortality, with a lower effect being observed in zones enjoying higher mean temperatures [22]. There is no summer peak in Kuwait for instance, even though it is one of the hottest places on Earth during this season [29]. These findings seem to point to physiological adaptation to climate and would support the hypothesis: that ideal climatic niches are located in the tropics [30]; and that the remaining populations around the world have achieved their current status thanks to adaptation and progressive insulation from the outdoors. The Madrid Region's annual temperature range (– 6 °C to 41 °C) and the marked daily thermal fluctuation render acclimatisation difficult, thereby giving rise to the heat-related excess observed in daily mortality. Nonetheless, as the control variables indicate, acclimatisation to heat during the summer period does take place, with heat-wave effect on mortality being highest in June and subsequently decreasing until August.

The effect of heat is direct and immediate, as the high positive CCF value in the first lag shows. In part, heat acts through a 'harvester' effect, by advancing the death of those who would have died in the short term [22]. This is confirmed by the negative correlations observed after this lag, indicating a decline in the number of daily deaths.

The fact that this behaviour is related to cardiovascular, and more specifically to cerebrovascular, deaths is consistent with the underlying biological mechanism [31, 32]. Heat places stress on the thermoregulatory system, which is intimately tied in with the circulatory system. Similar results were obtained by other authors, demonstrating the selfsame relationship between high temperatures and cerebrovascular disease mortality [8], as well as a similar response period [5]. The different male-female behaviour patterns are largely due to the greater number of women (aged 65 years and older) who are susceptible to high temperatures, which would in turn explain the higher value at lag 1 for women versus men. Temperature-related mortality is particularly important among older people, due to reduced thermo-regulatory response and less sensitive thermal perception [1, 8, 10, 31].

The inverse association between mortality and relative humidity in this period agrees with Kunst's Netherlands [5] and Kalkstein's US findings [22]. Low relative humidity would be associated with marked thermal fluctuation, causing death in those individuals less capable of adapting to such changes [31].

The lack of relationship between the wind at 7 a.m. and mortality could be due to the random structure of instantaneous measurement. An average of this variable (over an interval of time long enough for the turbulence phenomenon to be filtered) could be used [33] to analyse the association.

The relationship between minimum temperature and mortality is consistent with that reported elsewhere [5, 16]. Most winter mortality is explained by an increase in infectious diseases [34]. Low temperatures cause individuals to group together in enclosed spaces, thereby improving the transmission of infectious agents and increasing influenza incidence. Our findings show that influenza is related to a 5-unit increase in daily deaths.

Temperature has an independent effect on mortality, however. The CCF pattern is as expected, given the biological mechanisms at work in death arising from exposure to low temperatures: cardiovascular stress due to changes in blood pressure, vasoconstriction, and increase in blood viscosity, levels of plasma cholesterol and fibrinogen [3, 7, 31, 32]. A rapid effect on deaths due to cardiovascular diseases, and a longer delay in the case of respiratory diseases [5–7] are in evidence. In the latter case, inhaling cold air leads to bronchoconstriction which may aggravate previous pulmonary pathologies or enhance susceptibility to infectious agents.

The effect of temperature is constant during the winter period. There can be no acclimatisation to cold temperature akin to that observed for the summer period. Over two thirds of winter mortality can be attributed to the direct effect of cold temperatures [5], with the remaining winter mortality being related to the rise in TSP and SO2 [5].

Use of routinely-collected mortality data poses problems inherent to death certification. Misclassification of the underlying cause of death could be a source of bias, but we are of the opinion that the use of broad categories of sex, age and ICD coding reduces the likelihood of such an effect. Moreover, for such misdiagnosis to bias the estimated effect of weather, it must vary from day to day in a manner correlated with weather.

The results presented here show a marked relationship between temperature and mortality in the Madrid Region, even where air pollution, influenza epidemics, season and autocorrelation are controlled for as effectively as possible. High relative humidity during the summer period seems to protect against the effects of high temperatures. The effect of wind speed is negligible. From a Public Health perspective, the results suggest that environment-friendly improvements to home heating and insulation reduce the effect of cold on mortality [2]. Efforts should be directed at increasing home insulation, thereby ensuring comfortable temperatures while at the same time reducing fossil-fuel consumption and air pollution levels. Major heat waves pose a greater problem in Madrid given that the majority of MAR houses are not equipped with air-conditioning. Effects of warm temperatures on mortality are more pronounced due to the extreme annual thermal range, a factor which renders acclimatisation difficult.

Acknowledgments

This study was supported by the 'Consejeria de Educación y Cultura de la C.N. Acción Coordinada en Ciencias de la Salud 078'.

References

1. Sakamoto-Momiyama M. Changes in the seasonality at human mortality: A medico-geographical study. Soc Sci Med 1988; 12: 29–42.
2. McKee CM. Deaths in winter: Can Britain learn from Europe. Eur J Epidemiol 1989; 5: 178–182.
3. Lloyd EL. The role of cold in ischaemic heart disease: A review. Public Health 1991; 105: 205–215.
4. Mackenbach JP, Kunst AE, Looman CWN. Seasonal variation in mortality in the Netherlands. J Epidemiol Community Health 1992; 46: 261–265.
5. Kunst AE, Looman CWN, Mackenbach JP. Outdoor air temperature and mortality in the Netherlands: A time-series analysis. Am J Epidemiol 1993; 137: 331–341.
6. Enquselassie F, Dobson AJ, Alexander HM, Steele PL. Seasons, temperature and coronary disease. Int J Epidemiol 1993; 22: 632–636.
7. Khaw KT. Temperature and cardiovascular mortality. Lancet 1995; 345: 337–338.
8. Wen-Harn Pan, Luang-An L, Ming-Jan T. Temperature extremes and mortality from coronary heart disease and cerebral infarction in elderly Chinese. Lancet 1995; 345: 353–355.
9. Chou K, Thacker SB. An evaluation of influenza mortality surveillance. Am J Epidemiol 1981, 113. 215–235.
10. Chou K, Thacker SB. Mortality during influenza epidemics in the U.S. 1967–78. Am J Public Health 1982; 72: 1280–1283.
11. Marshall RJ, Scragg R, Bourke P. An analysis of the seasonal variation of coronary heart disease and respiratory disease mortality in New Zealand. Int J Epidemiol 1988; 17: 325–331.
12. Hoppenbrouwers T, Calub M, Arakawa K, Hodgman JE. Seasonal relationship of sudden infant death syndrome and environmental pollutants. Am J Epidemiol 1981; 113: 623–635.
13. Derriennick F, Richardson S, Mollie A, Lellouch J. Short term effects of sulphur dioxide pollution on mortality in two French cities. Int J Epidemiol 1989; 18: 186–197.
14. Schwartz J, Marcus A. Mortality and air pollution in London: A time series analysis. Am J Epidemiol 1990; 131: 185–194.
15. Schwartz J. Air pollution and daily mortality in Birmingham, Alabama. Am J Epidemiol 1993; 137: 1136–1147.
16. Mackenbach JP, Looman CWN, Kunst AE. Air pollution, lagged effects of temperature, and mortality: The Netherlands 1979–87. J Epidemiol Community Health 1993; 47: 121–126.
17. Momas I, Pirard D, Quenel P, Medina S, et al. Pollution atmosphérique urbaine et mortalité: Une synthèse des études épidémiologiques publiées entre 1980 et 1991. Rev Epidém et Santé Publ 1993; 41: 30–43.
18. Greenberg JH, Bromberg J, Reed CM, Gustafson TL, Beauchamp RA. The epidemiology of heat-related deaths, Texas–1950, 1970–1979, and 1980. Am J Public Health 1983; 73: 805–807.
19. Alberdi JC, Ordovás M, Quintana F. Construcción y evaluación de un sistema de detección rápido de mortalidad mediante análisis de Fourier. Rev Esp Salud Pub 1995; 69: 207–217.
20. Alberdi JC, Díaz J. Modelización de la mortalidad diaria en la Comunidad de Madrid. Gaceta Sanitaria 1997; 11: 9–15.
21. McMichel LS. Global environmental changes and human pollution health: A conceptual and scientific challenge for epidemiology. Int J Epidemiol 1993; 22: 1–8
22. Kalkstein LS. Health and climate change. Direct impacts in the cities. Lancet 1993; 342: 1397–1399.
23. World Health Organization. Potential health effects of climatic change: Report of the WHO Task Group. Geneva: WHO, 1990.
24. Katsouyanni K, Pantazopulou A, Touloumi G. Evidence for interaction between air pollution and high temperature in the causation of excess mortality. Arch Environ Health 1993; 48: 235–242.
25. Díaz J, Alberdi JC, Mirón IJ, Montero JC. Influencia de las variables atmosféricas sobre la mortalidad en la Comunidad de Madrid de 1986 a 1991. XXV Reunión Bienal de la Real Sociedad Española de Física, Santiago de Compostela, septiembre 1995; 7: 457–458.
26. Morán F. Apuntes de Termodinámica de la atmósfera. Servicio Meteorológico Nacional, Madrid, 1944.
27. Jenkins GM, Watts DG. Spectral analysis and its applications. San Francisco: Holden Day, 1968.

28. Saez M, Sunyer J, Castellsagué J, Murillo C, Antó JM. Relationship between weather temperature and mortality: A time series analysis approach in Barcelona. Int J Epidemiol 1995; 24: 576–582.
29. Douglas AS, Al-Sayer H, Rawles JM, Allan TM. Seasonality of disease in Kuwait. Lancet 1991; 337: 1393–1397.
30. Scholander PF, Waiters V, Hock R, et al. Body insulation of some arctic and tropical mammals and birds. Biol Bull 1950; 99: 225–236.
31. Woodhouse PR, Khaw KT, Plummer M, Foley A, Meade TW. Seasonal variation of plasma fibrinogen and factor VII activity in the elderly: Winter infections and death from cardiovascular disease. Lancet 1994; 343: 435–439.
32. Marshall RG, Scragg R, Bourke P. An analysis of the seasonal variation of coronary heart disease and respiratory disease mortality in New Zealand. Int J Epidemiol 1988; 117: 325–331.
33. Haltiner GJ, Martin FL. Dynamical and physical meteorology. New York: McGraw-Hill Book Company, 1977.
34. Bull GM. The weather and deaths from pneumonia. Lancet 1980; 1: 1405–1408.

Address for correspondence: D. Julio Díaz Jiménez, C/ General Oráa 39, 28006 Madrid, Spain
Phone: 34-91-564 24 99; Fax: 34-91-411 66 96