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Impact of Temperature and Air Pollution on the Mortality of Children in Madrid

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Learning Objectives

- Appraise the effects of individual air pollutants on mortality in children less than 10 years of age who lived in Madrid, Spain.
- Describe the effects of air temperature on child mortality and how pollutant levels vary seasonally.
- Identify biological mechanisms that may underlie the adverse effects of high pollutant levels and temperature extremes on child mortality.

Abstract

This work analyzes the impact of temperature and air pollution on infant mortality in Madrid. Daily values of mortality of children younger than 10 years, maximum and minimum temperatures, and air pollutants were considered for an 11-year period. In winter, mortality was mostly associated with very low temperatures and high total suspended particles (TSP) concentrations, whereas summer mortality depended crucially on the occurrence of high TSP and nitrogen oxides concentrations. In winter, the temperature effect increases dramatically for daily maximum temperature values lower than 6°C. This pattern is rather different from the one obtained for older age groups in the same location, which show the well-known V relationship between temperature and mortality. The association with TSP shows 2 linear branches without threshold and a strong increase in mortality for concentrations more than 100 μg/m³. (J Occup Environ Med. *2004; 46:000–000*)

The year 2003 was characterized by the occurrence of exceptionally abnormal weather episodes on both sides of the Atlantic Ocean. In fact, during the period of January to March 2003, most of the eastern North American continent experienced one of the coldest and longest winters in recent decades. However, a large sector of Western Europe suffered a remarkable intense and persistent heat wave throughout the summer 2003. These 2 independent events have led to a considerable increase in public attention on the occurrence of extreme temperature events and also their health and environmental impacts. This public awareness is reinforced by the fact that the Intergovernmental Panel on Climate Change[1,2] predicts an increase on the occurrence of extreme climatic events, especially heat waves over continental areas. However, it is not yet clear how this global trend will be reflected locally. Recent studies show that in North America, the observations do not necessarily fit the predictions from general circulation model simulations on the extreme events frequencies.[3,4] Additionally, it is now accepted that there is an adaptation effect of human communities to local weather conditions, and their physiological or cultural protection to the outdoor extreme temperatures.[5] Thus, possible shifts on the occurrence of future extreme events and their potential impact on humans are still an open topic, with ample uncertainties. In particular, it is not yet clear how the frequency will evolve

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locally and how the populations will adapt to those possible changes.

Current studies on the impact of temperature extremes on urban populations[6–10] show a strong connection between mortality and morbidity and these events. Generally, these studies tend to focus on the elderly (eg, older than 65 years of age), with few of them analyzing the impact on children.[11] This is particularly relevant, because the typical seasonal mortality pattern of the elderly is poorly associated with that of the population younger than 10 years of age. Nevertheless, it is now widely accepted that the susceptibility of children to environmental hazards is remarkably different than the susceptibility of adults.[12] This fact is mostly the consequence of a child's higher exposure level as a result of his or her small size and height, his or her higher physical activity, and his or her continual state of growth and development. Furthermore, in urban areas the effect of climatic extremes acts synergically with air pollutants, whose exposure is, in itself, a relevant risk factor.[13–15]

Some of these previous studies have shown that the behavior of extreme temperatures and their associated impact can be strongly modulated by local environmental conditions. Therefore, further analysis is required at a local scale to obtain a more reliable picture of the impact of extreme values on different population groups, in particular those groups poorly studied up to now. The aim of this work was to analyze the effects of extreme temperatures and air pollutants on daily mortality in the group of children younger than 10 years of age in Madrid (Spain) from 1986 to 1997.

Materials and Methods

Daily mortality has been computed as the number of daily deaths occurring in Madrid, between the 1st of January 1986 and the 31st of December 1997, for children younger than 10 years of age. Here, all deaths, except those classified as accidental were included (International Classification Diseases 9th Revision: ICD IX 1–799), being labeled as organic (all causes but accidents). Different age groups have been considered: from 0 to 9 years old, less than 1 year old, from 1 to 4 years old, and those from 5 to 9 years old. This analysis was performed for the whole period considered and for winter (November to March, both included) and summer (June to September, both included) separately. The Madrid Regional Department of Statistic provided mortality data.

Meteorological variables and air pollution concentrations were used as predictants. Meteorological variables data were provided by the Spanish National Institute of Meteorology, from the Madrid-Retiro Observatory, whereas pollution variables were provided by the City Council. The meteorological variables included in the models are: maximum daily temperature (T_{max}), minimum daily temperature (T_{min}), and relative humidity (RH) observed at 7 AM On the other side, air pollution variables used correspond to daily average mean concentrations of nitrogen oxides (NO_x), sulfur dioxide (SO_2), total suspended particles (TSP), and ozone (O_3).

A standard methodology based on Fast Fourier Transform[16] was used to identify trends and periodicities through spectral density function analysis. This approach necessarily leads to the introduction of dummy variables to control periodicities; they are labeled as *s365* and *co365* for the annual seasonality, *s180* and *co 180* for the semiannual seasonality, *s90* and *co 90* for the 3-month seasonality and *n1* for trends. Additionally, the potentially conflicting effect associated to the influenza epidemics was taken into account through (g1), a covariable equal to 1 if it was an epidemic day and 0 otherwise.

To eliminate analogous periodicities and autocorrelations not caused by causal relationships, a prewhitening procedure[17] was performed. This procedure eliminates spurious time covariability from the cross-correlation coefficients, thus isolating the correlation really associated to causality. A more detailed description of this procedure can be found in Díaz et al.[18] The cross-correlation function between the residuals of the prewhitened series was then computed. This procedure allows the identification of lags with significant cross-correlation values and the evaluation of the corresponding lagged associations. Finally, scatter-plot diagrams and "lowest" fits functions were used to identify the relationships between dependent (mortality) and predictants.

Because daily infant mortality obeys to a Poisson distribution, Poisson regression models were used to describe the association between mortality and the predictants through a step-by-step procedure. In the first step, the individual effect of all predictants was assessed taking into account the control covariables. Because the predictants exhibit a significant degree of interdependence, a model describing the joint predictants effect was obtained. This procedure was applied to the following mutually exclusive child age groups: 0 to 1, 1 to 4, 5 to 9, as well as to the entire sample 0 to 9. AQ: 1

Goodness-of-fit was evaluated through simple (ACF) and partial autocorrelation functions (PACF) of the residuals, using additionally the Akaike′s information criteria.[19] The influence of predictants on mortality was assessed through the attributable risk (AR), assuming that the whole population was exposed to its effects. In this way, attributable risk can be easily computed as follows: $AR = (RR - 1)/RR$,[20] where RR is the relative risk obtained by the previously described Poisson models. Our analysis was performed using S-Plus 2000 statistics pack.

Results

Table 1 shows simultaneously the descriptive statistics for the mortality, meteorological, and pollutant T1

TABLE 1
Descriptive Statistics for Children Mortality, Air Pollution, and Meteorological Variables Series in Madrid, 1986 to 1997

	Maximum	Minimum	Mean	SD	Trend	Periodicities
All causes 0–9	6	0	0.68	0.86	Decreasing	Annual, 3 weeks, 7 days, 3–4 days
All causes 0–1	5	0	0.54	0.77	Decreasing	Annual, 3 weeks, 7 days, 3–4 days
All causes 1–4	3	0	0.09	0.30	Decreasing	Annual, 3 weeks, 7 days, 3–4 days
Organic 5–9	2	0	0.05	0.23	White noise	White noise
T_{max}	40.0	0.6	19.7	8.6	No	Annual
T_{min}	25.4	−4.3	10.3	6.4	No	Annual
TSP	195	12.5	43.6	19.5	Decreasing	Annual, 7 days, 3 days
SO_2	401	6	52	43.2	Decreasing	Annual, 7 days, 3 days
O_3	78	0	22.8	14.3	Increasing	Annual, 7 days, 3 days
NOx	688	44	189.5	97.4	Decreasing	Annual, 7 days, 3 days
Humidity	100	10	78.3	16.1	No	Annual, 3 days

T_{max} indicates Maximum temperature; T_{min}, Minimum temperature; TSP, total suspended particles; SO_2, sulfur dioxide; O_3, ozone; NO_x, nitrogen oxides.

Units: Temperatures in °C; pollutants in ($\mu g/m^3$); humidity (%).

TABLE 2
Lags With Significant Prewhitened CCF Values Between Total Children Mortality (0 to 9 Years Old) Mortality and Predictants

Variables	Lags
T_{max}	7, 12
T_{min}	Without relation
TSP	0, 1
SO_2	1, 2
O_3	Without relation
NOx	1, 2

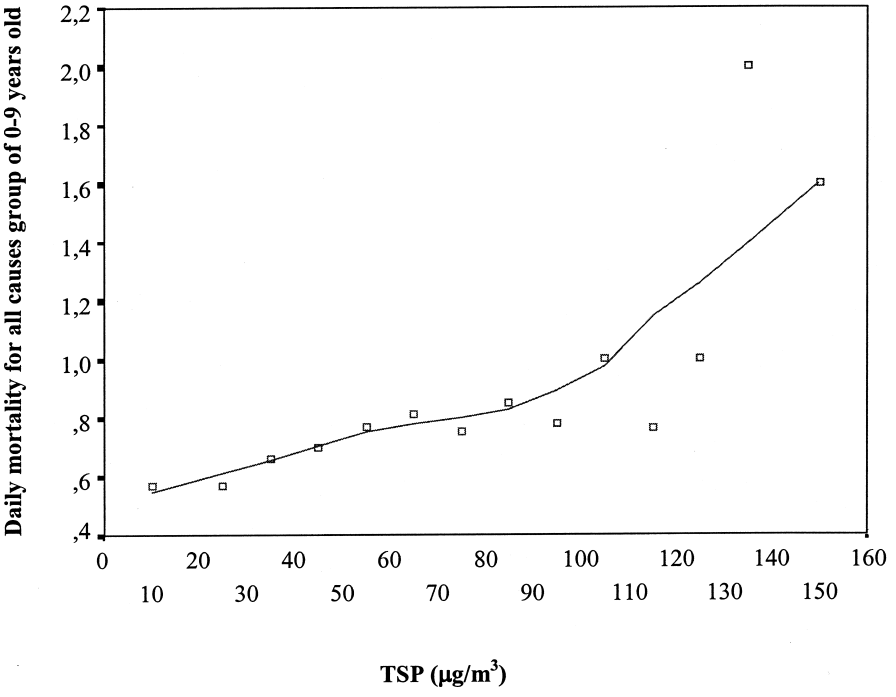

Fig. 1. Scatter plot of TSP concentrations and mortality in the group of 0 to 9 year olds.

variables. It should be noted that the global sample (0 to 9 years old), as well as the 0 to 1 and 1 to -4 year-old age groups, show significant periodicities and trends, whereas the 5 to 9 year-old group reveals a white noise behavior (ie, a purely stochastic behavior). For every mortality group and predictant, the following information is provided: maximum, minimum, average, and standard deviation values for the study period. The last 2 columns show the existence (or not) of significant trends and periodicities, as detected through the Fast Fourier Transform procedure.

T2 Table 2 shows the cross-correlation function outputs between environmental variables and mortality for the whole sample after the prewhitening process. Only statistically significant lags and variables ($P < 0.05$) are included. It can be seen that: (1) TSP presents an association with mortality in lags 0 and 1, whereas SO_2 and NO_x present the association lagged 1 and 2 days; (2) there is no statistically significant association between mortality and tropospheric ozone; and (3) among the temperature variables considered, the maximum daily temperature shows a significant relationship (up to 12 days afterward) with child mortality, whereas no such significant relationship is found for the minimum daily temperature.

Figure 1 shows the scatter-plot diagram of the relationship between TSP and global mortality in lag 0, whereas Fig. 2 shows the corresponding scatter-plot diagram for SO_2 and global mortality but lagged 1 day. Figure 1 can be reasonably well described by the superposition of 2 linear branches; however, the existence of a unique threshold value linking these 2 branches is not obvious. Nevertheless, a strong increase in mortality for TSP concentrations greater than 100 $\mu g/m^3$ is observed. In fact, when we consider TSP con-

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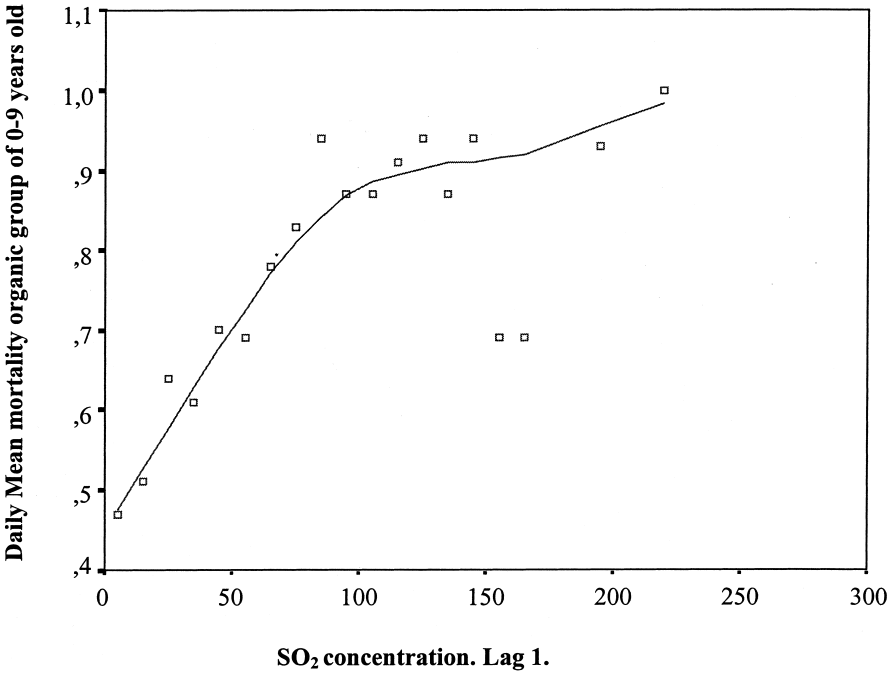

Fig. 2. Scatter plot diagram of SO_2 and mortality in the group of 0 to 9 year olds.

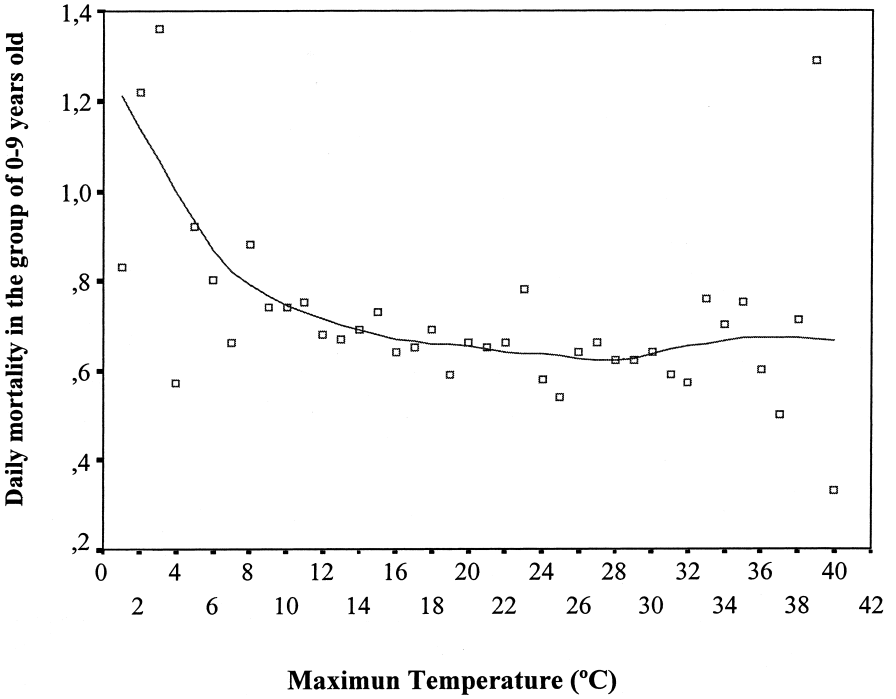

Fig. 3. Scatter plot diagram of T_{max} and mortality.

centrations lower than 100 $\mu g/m^3$, daily mean mortality in the group 0 to 9 years old is 0.67 deaths/day. However, for days with TSP concentrations greater than 100 $\mu g/m^3$, mortality increases to 1.03 deaths/day, with this difference being significant at $P < 0.05$. The SO_2relationship with mortality is better represented by a logarithmic curve as shown in Fig. 2, whereas NO_x are linearly related with mortality (not shown).

Figure 3 shows the scatter-plot diagram between maximum daily temperature (T_{max}) lagged 7 days. Again, 2 branches corresponding to two distinct T_{max}–mortality relationships can be observed. The first is valid for values smaller than 6°C, shows a steep negative slope, and corresponds to the impact of the coldest temperatures. Above this threshold no significant slope can be detected. Therefore, daily global mortality average is 0.68 (deaths/day) when T_{max} is higher than 6°C. This value is significantly different ($P < 0.05$) from the daily average mortality in the negative branch (0.95 deaths/day). It should be stressed that the 6°C value corresponds to the T_{max} distribution 5th percentile during the analyzed period. The effect becomes even more evident when the scatter-plot diagram is restricted to winter months, as in Fig. 4. To quantify this effect of enhanced mortality in very cold days, a new variable ($T_{c\ wave}$ = Temperature in cold wave) was created, defined as follows:

This new variable was included in the Poisson models. There is a significant association at lag 4, with RR = 1.23 (1.13, 1.32) and AR = 18.6%.

Table 3 summarizes the impact of atmospheric pollutants in global mortality. For each predictant, the statistically significant variables ($P < 0.05$), the lags, the RR, and the AR are provided. Ozone did not produce a significant impact, whereas TSP was the most relevant at lag 0 and SO_2 for lag1. The apparent impact of SO_2 in lag 0 in the individual model is attributable to the colinearity with TSP because them both share most of their sources.[18] High interdependence is also evident for some of the variables in Table 4, which shows the Pearson correlation coefficient between T_{max} and the pollution predictant. Next, the joint effect was evaluated through Poisson regression models, including all the predictants. Results are summarized in Table 5 for the entire year and in separate for the 2

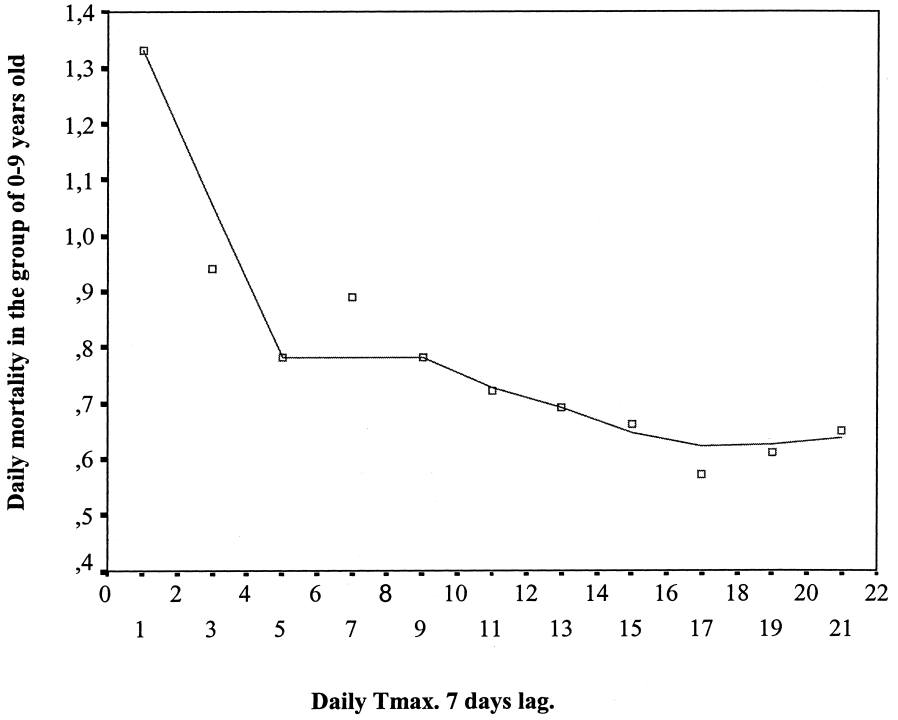

Fig. 4. Scatter plot diagram for T_{max} lagged 7 days with mortality in winter.

TABLE 3
Poisson Regression Models for Children (0 to 9 Years Old) Mortality and Air Pollutants

Factors	Statistically Significant Variables (Lag)	AR (%)	RR (95% CI)
Only TSP	TSP (0)	6.4	1.07 (1.04, 1.10)
Only NOx	NOx (0)	2.4	1.02 (1.01, 1.04)
Only SO_2	SO2 (0)	6.2	1.07 (1.03, 1.11)
	SO2 (1)	4.5	1.05 (1.01, 1.09)
Only O_3	Not significant		
All air pollutants	TSP (0)	6.3	1.07 (1.02, 1.12)
	SO_2 (1)	4.4	1.05 (1.01, 1.09)

TABLE 4
Correlation Coefficients Between the Different Air Pollutants and T_{max}

	T_{max}
TSP	−0.136*
SO_2	−0.423*
NOx	−0.313*
O_3	0.611*

* Significance: $P < 0.001$.

extreme seasons: summer (between June and September) and winter (November to March). The highly seasonal dependence of results is immediately striking, with the TSP presenting higher RR values in summer than in winter and the NOx not playing a significant role in winter.

T6 Table 6 shows the results when different age groups are considered. Overall, our results suggest that the 1 to 5 year-old group is more susceptible to the joint impacts of pollutants and extreme temperatures. The 5 to 9 year-old group has not been analyzed because it shows a purely white noise structure.

Discussion

Impact of Temperature

In the previous section we have shown that the impact of temperature on child mortality is by and large limited to those winter days with T_{max} values lower than 6°C. This behavior differs significantly from the usually accepted V-shape association between temperature and mortality for the global population.[6,21–23] This is mostly attributable to a lower exposure of children to extremely high temperature events and to the fact that, on the average, their physiological adaptation is better than that of the elderly population.[24] Nevertheless, it must be emphasized that the quantitative impact of cold temperatures in young children is higher than that in the elderly population, that is, the slope of the downward branch in Figures 3 and 4 is steeper for young children than for the older age groups.

Interestingly, the threshold of 6°C coincides with the 5th percentile of the distribution of daily T_{max}. Previous studies have shown that physiological mechanisms fail when temperatures fall below this threshold (García R, Díaz J, Trigo RM, Hernández E, Suraje D, submitted),[9,10] AQ: 2 which seems also a plausible explanation for children. The fact that daily T_{max} is the best indicator for thermal impact can be understood from the daily temperature cycle. Minimum temperature is often registered 1 or 2 h before the sunrise, when it is not likely for a child to be exposed to such low temperatures. On the contrary, maximum temperature values are usually observed during early afternoon hours, a time of the day where exposition to environmental factors is more likely.

Impact of Air Pollution

It is well known that ground-level ozone reduces lung function and can exacerbate chronic respiratory diseases in children.[25] However, the daily maximum concentration of ozone levels in Madrid (78 $\mu g/m^3$) falls under the threshold to establish any association with respiratory diseases.[26,27] Moreover, the annual mean ozone level in Madrid is 23 $\mu g/m^3$, a value that is lower than the

TABLE 5
Statistically Significant Variables in Poisson Regression for All the Variables Considered and Mortality in the Group of 0 to 9 Years Old

Period	Factor	AR (%)	RR (95% CI)
Whole year	TSP (0)*	8.1	1.09 (1.04, 1.13)
	Tcwave (4)†	17.6	1.21 (1.12, 1.31)
Winter	TSP (0)*	6.7	1.07 (1.01, 1.13)
	Tcwave (4)†	18.0	1.22 (1.12, 1.32)
Summer	TSP (2)*	33.1	1.49 (1.28, 1.71)
	NOx (0)*	5.0	1.05 (1.02, 1.08)

* RR and AR for an increase of 25 $\mu g/m^3$ in the TSP concentration (and NOx).
† RR and AR for each degree of T_{max} (Maximum temperature) less than 6°C.

TABLE 6
Poisson Regression Models for the Different Age Groups and All the Environmental Variables Considered. Only Significant Variables are Included

Period	Factor	AR (%)	RR (95% CI)
0–1 year group			
Whole year	TSP (0)*	5.2	1.06 (1.05, 1.06)
	Tcwave (4)†	16.3	1.19 (1.11, 1.28)
Winter	TSP (0)*	9.6	1.11 (1.05, 1.16)
	Tcwave (4)†	17.4	1.21 (1.10, 1.32)
Summer	TSP (2)*	31.5	1.46 (1.24, 1.68)
	NOx (0)*	6.3	1.07 (1.04, 1.09)
1–5 year group			
Whole year	TSP (0)*	20.0	1.25 (1.15, 1.35)
	Tcwave (1)†	23.1	1.30 (1.07, 1.53)
Winter	TSP (0)*	20.2	1.25 (1.12, 1.38)
	Tcwave (1)†	23.1	1.30 (1.07, 1.53)
Summer	TSP (3)*	34.7	1.53 (1.29, 1.77)

* RR for an increase of 25 $\mu g/m^3$ in the TSP concentration (and NOx).
† RR for each degree of T_{max} (Maximum temperature) is less than 6°C.

critical value considered for nonexposed population in other studies on the association of ozone levels with respiratory diseases in children.[28] We believe that this single fact is the main reason for the lack of association with mortality, which is also detected by other authors when considering emergency admissions for respiratory causes.[29]

There is a strong increase in mortality for TSP concentration levels greater than 100 $\mu g/m^3$, (values that usually occur in winter months); this pattern was not detected for the general population.[18] Poisson models calculated exclusively for TSP levels higher than 100 $\mu g/m^3$ give a RR of 1.35 (1.13, 1.58) for each 25 $\mu g/m^3$ of increase, corresponding to an AR of 26.1%.

It is known that the acid nature of many pollutants can induce alveolar inflammation, which can provoke major changes in: (1) blood coagulation,[30] (2) risk of increase blood plasma viscosity,[31] and (3) an increment of fibrinogen, leukocytes, and platelets serum concentrations.[32,33] This fact seems to explain the relatively strong association between the presence of acidic substances in the atmosphere and mortality. In the case of TSP, a mechanic effect must be added,[34] as a consequence of their characteristic big size, a fact that could explain the nature of their short-term impact. The significant short-term lags found (Table 2) and for Poisson regression models (Table 3) are coherent with this fact.

Previous studies[18] showed an increase of roughly 2% in mortality associated to an increase of 25 $\mu g/m^3$ in TSP or SO_2 concentrations. The present work shows that, for children younger than 10 years of age, this risk doubles for SO_2 or increase threefold for TSP. The reason of this higher effect in children lies in the above-mentioned higher exposure to pollutants per mass unit. Compared with adults, their narrower airways suffer higher tissue exposure per unit of inhaled air, increasing the inflammation risk[35] and, finally, producing stronger health effects.

Compared with the winter season, TSP impact shows a strong increase in relative risk during summer, a circumstance that happens jointly with a delay toward lag 2. This may be the result of the synergic effects established between air pollution and high temperatures,[36] which probably enhance the TSP impact on mortality. From this perspective, the 2-day lag is probably linked with the summer mortality causes (more cardiovascular than respiratory diseases), which delay the effect. Table 6 shows that the TSP RR is higher in the group of 1 to 4 year-old children than in the group of 0 to 1 year-old children. This fact can be the result of the greater outdoor exposure of the 1 to 4 year-old to air pollution, together with more physical exercise that not only increases the metabolic rate: "Exercise increases breathing through the mouth rather than the nose, which filters approximately half of pollutants".[35]

It has been proved that SO_2 and NOx induce similar health impacts.[37] The stronger acidity of SO_2 makes its effect prevailing on NOx at high SO_2 concentrations, as is the case in winter in Madrid. However, the low summer SO_2 concentrations (daily average of 31 $\mu g/m^3$ versus 77 $\mu g/m^3$ in winter) explain the NOx significant role in summer, when the daily average value is 146 $\mu g/m^3$.

Conclusions

Our results provide a clear picture of seasonal infant mortality when associated with relevant environmental variables. In summary, winter mortality is customarily associated

with very low temperatures and high TSP concentrations, whereas summer mortality is mostly associated with high TSP and NOx concentrations. This pattern is different from other age groups, even when measured in the same locations.[9,18,21] These differences should be accounted for when official health authorities intend to implement strategies with the aim of minimizing the negative effects of the environmental factors on infant mortality. In particular, their approach should not reproduce exactly the strategy adopted for the general population.

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