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Short-term impact of particulate matter ($PM_{2.5}$) on respiratory mortality in Madrid

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Objectives: This paper sought to quantify the particulate matter ($PM_{2.5}$) pollutant's impact on short-term daily respiratory-cause mortality in the city of Madrid.
Methods: As our dependent variable, we took daily mortality registered in Madrid from 1 January 2003 to 31 December 2005, attributed to all diseases of the respiratory system as classified under heads J00–J99 of the ICD 10 and broken down as follows: J12–J18, pneumonia; J40–J44, chronic diseases of the respiratory system except asthma; J45–J46, asthma; and J96, respiratory failure.
Results: The relative risk (RR) for daily overall respiratory mortality was RR 1.0281 (1.0043–1.0520), with a proportional attributable risk (PAR) of 2.74%. This effect occurred in lag 1; respiratory failure, RR 1.0816 (1.0119–1.1512) and PAR 7.54% at lag 5; and pneumonia, RR 1.0438 (1.0001–1.0875) and PAR 4.19% at lag 6.
Conclusions: Our results reflect the association that exists between $PM_{2.5}$ concentrations and daily respiratory-cause mortality.

Keywords: particulate matter; respiratory mortality; air pollution

Introduction

The new European Directive 2008/50/EC governing ambient air quality already acknowledges the need for restrictions to be placed on and detailed measurements to be taken of $PM_{2.5}$ concentrations. These substances have thus come to constitute yet another pollutant which poses problems in terms of complying with the statutory limits in the city of Madrid. The principal source of such emissions, i.e., road vehicle traffic, has been rising relentlessly. This increase is far more evident for diesel- than for gasoline-powered vehicles, and indeed the former have even come to outnumber the latter in recent years (Ministerio de Medio Ambiente [MMA] 2005).

The city of Madrid registers pollution levels similar to those of other European cities (Keuken et al. 2005), with road transport being responsible for 85% of all $PM_{2.5}$ concentrations (OSE 2007). Under the limits and deadlines set by European

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Directive 2008/50/EC, in cases such as Madrid (period 2003–2005), where detected $PM_{2.5}$ concentrations range from 18–22 $\mu g/m^3$, a reduction of 20% must be attained by the statutory deadline of 2020 (DE 2008). The mean annual limit value for Madrid would be around 15 $\mu g/m^3$, with the limit values that may not be exceeded being set at 25 $\mu g/m^3$ for 2015 and 20 $\mu g/m^3$ for 2020. These values are variable and depend on the initial figures presented by each country. The main aim is to achieve a gradual reduction in concentrations, with the goal of reaching particulate levels that are innocuous to health being relegated to a secondary plane. Consequently, these figures are considerably more permissive than those recommended by the World Health Organisation (WHO) for the protection of health (10 $\mu g/m^3$) (WHO 2005) and those envisaged under the United States Environmental Protection Agency (EPA) guidelines (USEPA 1997).

Suspended particulate matter has been shown to have greater impact on health than do other pollutant substances (WHO 1996; Landen et al. 2000; Ferrís et al. 2003; Sanderson et al. 2005). The WHO puts the number of Quality Adjusted Life Years (QALYs) lost as a consequence of exposure to PM at 6.4 million (Cohen et al. 2005). The composition of $PM_{2.5}$, mostly made up of organic carbon and secondary particles (sulphates, nitrates and organic aerosols), makes it a more toxic substance (Artíñano et al. 2004). Moreover, this origin, for the most part influenced by traffic emissions, means that it is regarded by the WHO as a more reliable indicator of anthropogenic activity (WHO 2005; Linares and Díaz, 2010).

From the standpoint of respiratory disease, studies, largely US-based, have associated increased risk of respiratory diseases and tumours of the respiratory system with chronic exposure to these particles (Pope et al. 2002; Brunekreef and Forsberg 2005; Chen et al. 2005; Fung et al. 2006; Girardot et al. 2006; Lagorio et al. 2006; Lee et al. 2007) Every increase of 10 $\mu g/m^3$ in mean ambient $PM_{2.5}$ values amounts to an increase of 15–21% in cases of lung cancer (Chen et al. 2008).

Furthermore, these exposures also produce short-term effects on respiratory mortality (Simpson et al. 2005). Although the physiopathologic mechanism has not been completely clarified yet, many more studies are appearing that develop new knowledge (Laing et al. 2010). $PM_{2.5}$ concentrations cause oxidative stress reactions in the interior of alveolar epithelial cells (Xia et al. 2006), triggering an immediate inflammatory response which is responsible for the exacerbation of episodes of asthma, chronic obstructive pulmonary disease (COPD) and respiratory failure. On the other hand, these same particles are also able to depress pulmonary defence mechanisms through the production of defence-immunoglobulin-specific antigens (Zanobetti et al. 2000; Svendsen et al. 2007). This state of ''immunodepression'' causes an increase in the incidence of upper and lower tract respiratory infections (pneumonias, bronchitis).

Yet, despite the many US studies that consistently show an association between exposure to $PM_{2.5}$ and respiratory-cause mortality (Ostro et al. 2006; Franklin et al. 2007), there are still very few European cities in which these types of studies have been conducted. It is the promulgation of the new law that has heightened European countries' interest in measuring this pollutant and undertaking epidemiological studies (Cassano et al. 2009; Belleudi et al. 2010; López-Villarrubia et al. 2010).

Insofar as the city of Madrid is concerned, European Directive 2008/50/EC has not yet been transposed to this country, and the currently prevailing Royal Decree 1073/2002 which merges European ambient air quality Directives 96/62/EC, 1999/30/EC and 2000/69/EC does not envisage this type of particulate matter (B.O.E. 2002)

Nevertheless, the Madrid City Council has an Air Quality Monitoring Grid made up of 27 monitoring stations, seven of which have been measuring $PM_{2.5}$ levels since 2003 (MMA 2008).

The aim of our study was thus to analyse the short-term effect of daily mean $PM_{2.5}$ concentrations on respiratory mortality across all age groups in a city such as Madrid, where 85% of pollution comes from traffic and the local climatic conditions (e.g., low precipitation) do not contribute to the cleaning of the atmosphere.

Methods

We conducted an ecological time series study in which the study population was made up of Madrid city residents of all ages, over a three-year period dating from 1 January 2003 to 31 December 2005.

Dependent variables

As our dependent variable, we took daily mortality due to respiratory causes in the city of Madrid, as classified under heads J00–J99 of the International Classification of Diseases-10th revision (ICD 10), namely: J96, respiratory failure; J12–J18, pneumonia; J40–J44, chronic diseases of the respiratory system except asthma; and J45–J46, asthma. These data were furnished by the Madrid Regional Revenue Authority (*Consejería de Hacienda de la Comunidad de Madrid*).

Independent variables

The following types of air pollution variables were used as independent variables: chemical (chemical pollutants); physical (noise levels); and biotic (pollens). In addition, the variable, ''temperature'', was also included.

For chemical pollution, we used daily concentrations, measured in $\mu g/m^3$, of sulphur dioxide (SO_2), nitrogen dioxide (NO_2), tropospheric ozone (O_3), airborne particles having an aerodynamic diameter <10 micra (PM_{10}) and those having an aerodynamic diameter of less than 2.5 micra ($PM_{2.5}$).

For physical pollution, we used equivalent daily sound levels from 8–22 h (LEQday), equivalent nocturnal sound levels from 22–8 h (LEQn) and equivalent mean daily sound levels for a 24-h period (LEQ24), measured in dB(A) and based on readings from the Madrid City Council's 30-station acoustic pollution monitoring grid.

To measure pollinic air pollution, we used readings of the daily mean concentrations of the different species of *Cupresaceae, Gramineae, Olea europaea* (Olive) and *Platanaceae* pollen, all measured in grains/m^3 and drawn from single station of the Madrid Regional Health Authority Palynology Network (*Red Palinológica de la Consejería de Sanidad de la Comunidad de Madrid – PALINO-CAM*). Maximum and mean daily temperature readings in °C were provided by the National Meteorology Institute corresponding to the Madrid-Retiro Observatory.

Other variables

Similarly, trend, seasonality, autocorrelation of the series, influenza epidemic and day of the week were also controlled for.

Statistical analysis

Prior to performing the statistical analysis, absent values were imputed by using the linear interpolation method and control of extreme values. After completing the descriptive study and plotting time-series graphs for each of the variables studied, we proceeded to establish the functional relationship between the respective independent variables and each of the causes of mortality, using scatter diagrams for the purpose. These diagrams were fitted using LOWESS smoothing and enabled the existence of threshold values and type of functional relationship to be clarified. Should the relationship between mortality and the independent variable not be linear, the latter must be transformed before being introduced into the Poisson model. This occurs in the case of respiratory mortality and O_3, in which the relationship is quadratic and V-shaped. Minimum mortality is identified at around 50 $\mu g/m^3$ of O_3. Consequently, the two arms of the variable had to be defined for subsequent analysis, with two new variables being created, namely, O_3 high and O_3 low:

$$O_{3\,\text{high}} = O_3 - 50, \quad \text{if } [O_3] > 50$$

$$O_{3\,\text{low}} = 55 - O_3, \quad \text{if } [O_3] < 50$$

This applies equally to temperature, where minimum respiratory mortality is observed to occur at around 30°C. Temperature was thus divided into two groups so that the respective effects of cold and heat could be subsequently observed.

$$T_{\text{cold}} = 30^{\circ}\text{C} - T_{\max}, \quad \text{if } T_{\max} < 30$$

$$T_{\text{heat}} = T_{\max} - 30^{\circ}\text{C}, \quad \text{if } T_{\max} > 30$$

The cross-correlation functions (CCFs) then qualitatively determine the exact moment in time at which the statistical relationship between the independent and dependent variable arises, without the remaining variables being taken into account.

The series must be previously prewhitened. To this end, the residuals of the independent variable must be obtained by means of univariate ARIMA models, based on visual inspection of the simple autocorrelation function (ACF) and partial autocorrelation function (PACF) (Box et al. 1994), and minimisation of Akaike's information criterion (Akaike 1974). Once the residuals for both variables have been obtained and the prewhitening process completed, Box-Jenkins methodology is used (Makridakis et al. 1983). In this methodology, it is the method of the independent variable that is applied to the dependent variable, with the correlation between the residuals being established.

As a final step, Poisson models were created for the purpose of quantifying the association between dependent and independent variables, with a regression model being fitted for each of the variables of total respiratory mortality and for the different specific causes. The variable "mortality due to all respiratory causes" was analysed on a stratified basis by considering two seasonal periods, one for winter, corresponding to the months of November to March (both inclusive), and the other for summer (May-September). The model was constructed using the S-Plus 2000 statistical package (Insightful Corporation, Seattle, WA, USA).

The corresponding relative risks (RRs) with their confidence intervals (95% CI) and proportional attributable risks (PARs) were calculated for 10 $\mu g/m^3$ increases in each pollutant, using the SPSS computer software programme 15.0 (SPSS Inc.,[TM] The Apache Software Foundation).

Results

Mortality across all age groups registered in Madrid over the study period showed a total of 10,351 deaths due to respiratory disease. Of these, 2770 deaths were due to pneumonia, 952 due to respiratory failure, 2246 due to chronic diseases of the respiratory system except asthma, and 78 due to asthma. Table 1 shows the descriptive statistics of the dependent variables considered.

Shown below are the time-series graphs for daily mortality due to all diseases of the respiratory system and to each of the various specific causes (Figure 1). Whereas mortality due to all respiratory causes showed no variations in the overall trend, it did nevertheless display seasonal variations, with mortality being highest in the

Table 1. Descriptive statistics of the dependent variables from 2003–2005.

Mortality cause	*n*	Minimum	Maximum	Mean	SD
All respiratory cause	1096	0	32	9.4	4.4
Pneumonia	1096	0	12	2.5	1.8
Respiratory failure	1096	0	5	0.9	1
Chronic diseases of the airways	1096	0	10	2	1.6
Asthma	1096	0	2	0.1	0.3

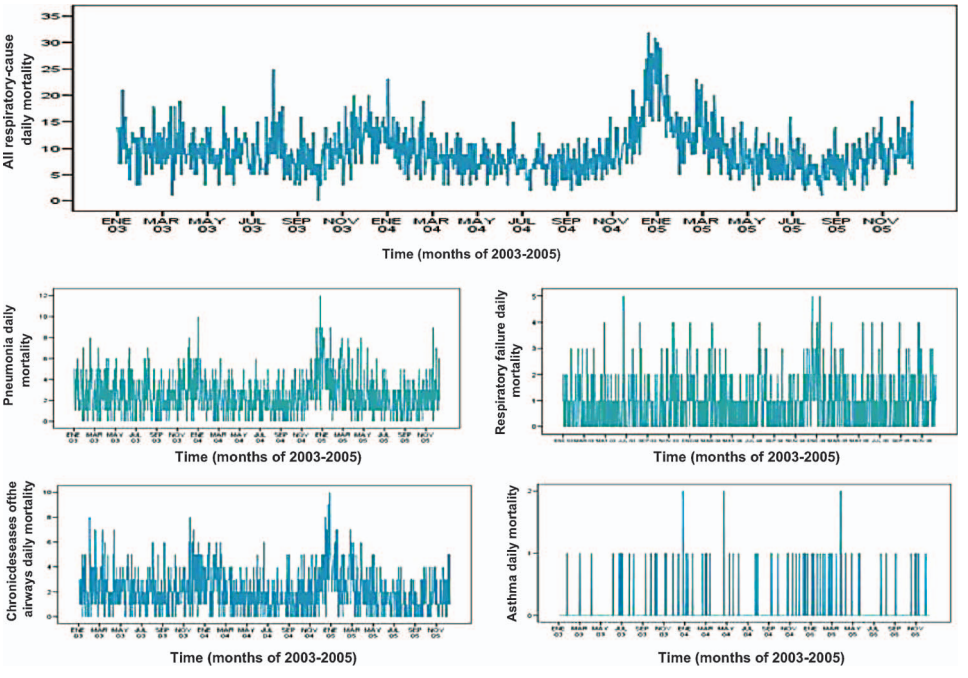

Figure 1. Temporal distribution of cause specific daily mortality.

Table 2. Descriptive statistics of the independent variables from 2003–2005.

	n	Minimum	Maximum	Mean	SD
Chemical pollution ($\mu g/m^3$)					
SO_2	1095	5	36	11.8	5.5
NO_2	1095	19	133	59.9	17.7
NO_x	1094	22	495	118.1	65.3
O_3	1095	5	89	34.8	18
PM_{10}	1094	8	150	34.4	17.5
$PM_{2.5}$	1038	5	71	19.2	8.6
Biotic pollution(grains/m^3)					
Cupresacea	1082	0	811	7.6	38
Gramineae	1082	0	124	2.8	8.3
Oleaceae	1082	0	480	2.8	23.4
Platanus	1082	0	1126	8.4	57.3
Physical pollution (dBA)					
Leq 24	1096	61.5	66.4	64.1	0.7
Leq day	1090	62.1	67.3	65.1	0.8
Leq n	1096	58.7	71	60.5	0.9
Temperature (°C)					
Temperature max	1095	2.0	38.6	20.4	9
Temperature min	1095	−6.1	27.8	10.5	7

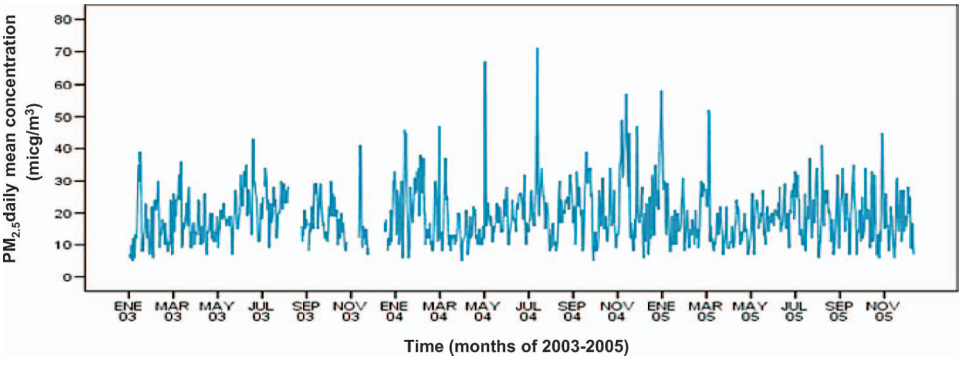

Figure 2. Temporal distribution of mean concentrations of $PM_{2.5}$.

winter and minimal in the summer months. Even so, there were peaks in summer mortality that corresponded to peaks in maximum temperature (heat waves). In addition, a more gradual transition from the hot to the cold than from the cold to the hot season was in evidence.

Table 2 lists the descriptive statistics for each of the independent variables that formed part of the study. Focusing on $PM_{2.5}$ values, it will be seen that daily mean concentrations during the study period ranged from 5–71 $\mu g/m^3$, with a mean of 19.2 $\mu g/m^3$, without any clear seasonal component being discernible in the time-series graph (Figure 2). These concentrations rose to almost double those recommended by the WHO for the protection of health. With respect to the mean 24-h limit value set at 25 $\mu g/m^3$, this was exceeded by the city of Madrid on 23.9% of days during the study period.

Also shown below are the scatter diagrams depicting the association existing between daily mortality due to each of the respiratory causes studied and the daily concentration of our principal study variable, $PM_{2.5}$. The results for daily mortality

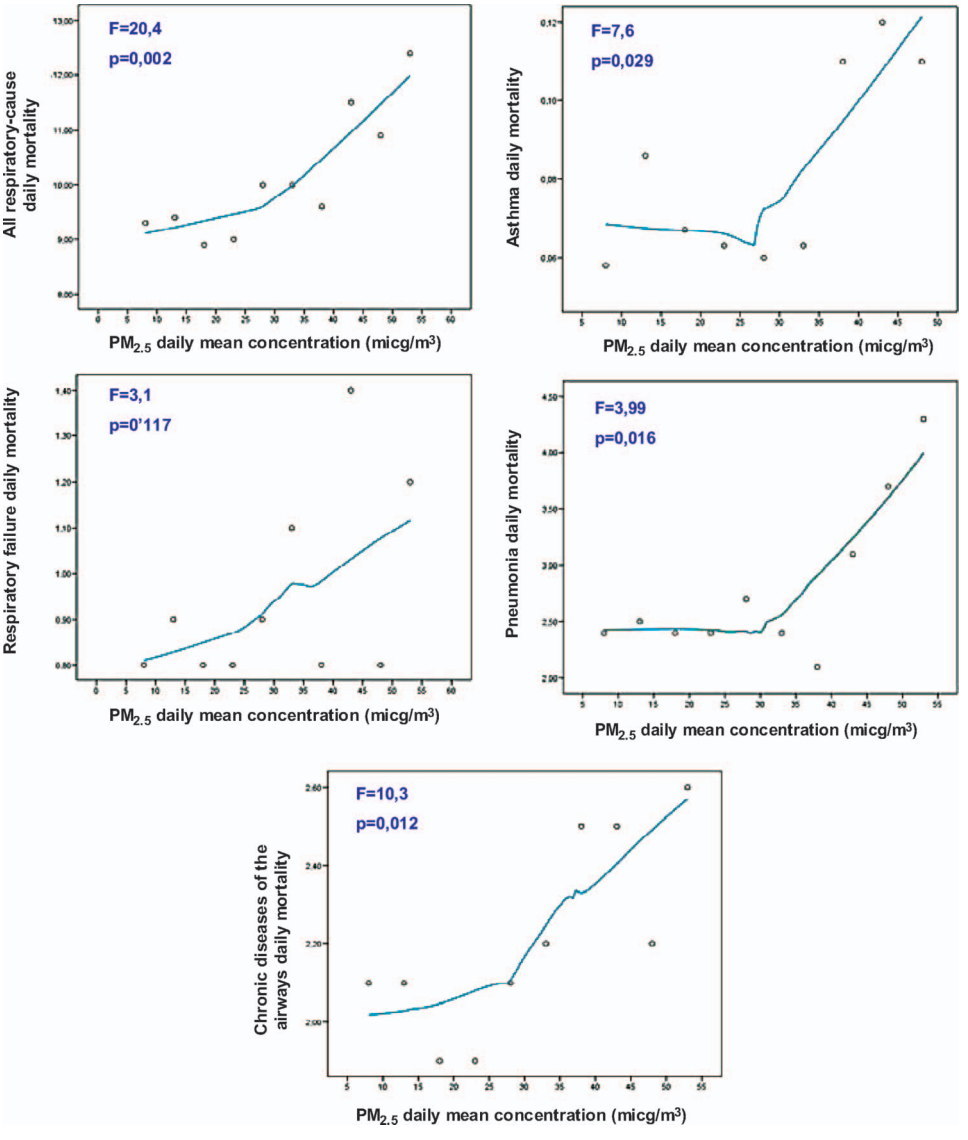

Figure 3. Scatter plot of concentrations $PM_{2.5}$ and mortality causes.

due to all respiratory causes followed a linear distribution, with a statistically significant F coefficient of 20.4 ($p = 0.002$) and a $PM_{2.5}$ concentration threshold of 29 $\mu g/m^3$, above which mortality rose steeply (Figure 3).

In respect to the CCFs, which reflect the qualitative association between each of the dependent mortality variables and daily mean $PM_{2.5}$ concentrations, once the effects of the remaining environmental variables used in the study had been controlled for, overall respiratory mortality was seen to display a relationship which proved statistically significant (90% CI) at lags 1 and 2, meaning that increases in $PM_{2.5}$ values on any given date led to an increase in respiratory-cause mortality one to two days later. Mortality due to respiratory failure was statistically significant

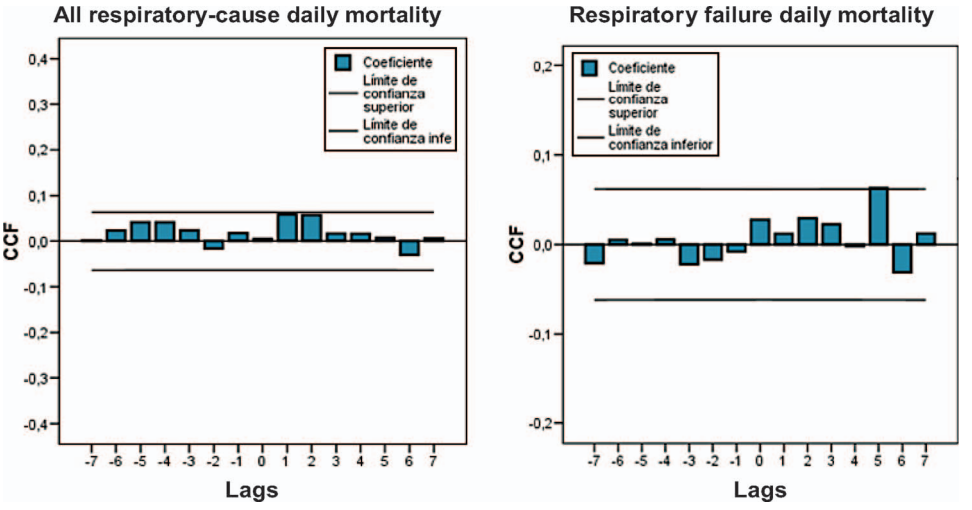

Figure 4. Cross-correlation functions between mortality and mean daily $PM_{2.5}$.

(95% CI) at lag 5 (Figure 4). For the remaining specific causes of respiratory mortality, such as pneumonia, chronic diseases of the respiratory system except asthma, and asthma, the cross-correlation functions failed to prove statistically significant (95% CI).

Lastly, Poisson regression models enabled us to quantify the associations found through the relative risks and proportional attributable risks, with both being calculated for increases of 10 $\mu g/m^3$ in daily mean $PM_{2.5}$ concentrations. The results obtained were statistically significant (95% CI) at the same lags as for the CCFs. This same analysis performed for all causes of respiratory mortality, differentiating between winter (November-March) and summer (June–September), proved statistically significant for the winter period. Table 3 shows the results for RRs and PARs.

Discussion

During the study period, daily mean $PM_{2.5}$ concentrations ranged from 5–71 $\mu g/m^3$, with a global mean of 19.2 $\mu g/m^3$, almost double the annual mean level recommended by the WHO (10 $\mu g/m^3$). The 25 $\mu g/m^3$ limit set by the WHO as the maximum daily mean concentration above which effects on short-term mortality have been observed, was exceeded on 23.9% of days. However, despite the fact that vehicle traffic constitutes the principal source of $PM_{2.5}$ emissions, the time-series graph showed no clear seasonal behaviour characterised by maximum values in winter, which would be the most logical pattern, taking into account the greater influx of vehicles accompanied by the lower dispersion of pollutants caused by higher atmospheric stability.

For our study variables, the relationship observed between daily mean $PM_{2.5}$ concentrations and mortality was linear, and, after applying the LOWESS method, displayed a threshold value above which mortality was observed to increase sharply. For overall respiratory mortality, the values above which mortality rose were somewhat higher than those set by the WHO, namely, about 29 $\mu g/m^3$. For the remaining specific respiratory causes, the thresholds detected were somewhat lower

Table 3. Relative risk and attributable risk of mortality by specific cause for each increase in the concenration of independent variables.

Mortality causes	Variables significance $P < 0.05$	Relative risk (RR)	Attributable risk (RA)
All respiratory	$PM_{2.5}$ (lag 1)	1,0281 (1,0043–1,0520)	2.74%
	Thot (lag 0)	1,2953 (1,1224–1,4681)	22.8%
	Thot (lag 3)	1,3484 (1,1724–1,5244)	25.84%
	O_3 A (lag 5)	1,1016 (1,0431–1,1601)	9.22%
	Leq24 h (lag 1)	1,7403 (1,3850–2,0955)	42.54%
	Oleaceae (lag 1)	1,0099 (1,0019–1,0179)	0.98%
	SO_2 (lag 4)	1,0721 (1,0209–1,1233)	6.72%
Pneumonia	$PM_{2.5}$ (lag 6)	1,0438 (1,0001–1,0875)	4.19%
	Thot (lag 0)	1,9870 (1,7049–2,2691)	49.67%
	NO_2 (lag 4)	1,0396 (1,0162–1,0630)	3.81%
	O_3 A (lag 3)	1,1846 (1,0797–1,2895)	15.58%
	O_3 A (lag 5)	1,1633 (1,0606–1,2660)	14.04%
	Oleaceae (lag 3)	1,0231 (1,0108–1,0354)	2.26%
	Leq day (lag 1)	2,5354 (1,9612–3,1096)	60.56%
Respiratory failure	$PM_{2.5}$ (lag 5)	1,0816 (1,0119–1,1512)	7.54%
	Oleaceae (lag 1)	1,0312 (1,0129–1,0495)	3.03%
	Tcold (lag 8)	1,2325 (1,1497–1,3154)	18.87%
All respiratory (winter)	$PM_{2.5}$ (lag 1)	1,0538 (1,0261–1,0815)	5.10%
	Platanus (lag 2)	1,0126 (1,0054–1,0198)	1.25%

in the case of asthma and COPD, i.e., around 27 $\mu g/m^3$, and slightly higher for pneumonia, i.e., 30 $\mu g/m^3$. Mortality due to respiratory failure was found to be linear and without a threshold, in line with the findings reported by many other studies for concentrations of fine particulate matter of under 100 $\mu g/m^3$ (Schwela 2000; Crosignani 2010).

Insofar as the CCFs were concerned, it will be seen that the short-term impact of daily mean $PM_{2.5}$ concentration on overall respiratory mortality was observed at lags 1 and 2, slightly later than that observed in the elderly population, among whom it appears at lag 0, a logical development, bearing in that the general population possesses defence mechanisms or baseline conditions that enable it to combat the harmful effect of $PM_{2.5}$ for longer.

Mortality in the case of respiratory failure became evident five days after the increase in $PM_{2.5}$ concentrations because, in this case, the inflammatory mechanism triggered in the respiratory cells requires a longer period to produce a thickening of the alveolar walls and fibrosis, which may entail gas exchange impairment.

In terms of lags, the associations obtained using Poisson regression models were similar to those obtained using CCFs, with the advantage that the former models enable one to quantify these associations by means of measures of association and impact, i.e., RRs and PARs, both of which were calculated in our study for an increase of 10 $\mu g/m^3$ in daily mean $PM_{2.5}$ concentrations.

The association for overall respiratory mortality remained significant at lag 1, with an RR of 1.0281 (1.0043–1.0520) and a PAR of 2.74%. These results are somewhat lower than those yielded by the Madrid population study for subjects aged over 75 years (Jiménez et al. 2009), which is only logical in view of this latter population group's higher susceptibility. In terms of lags, our results are in line with those obtained in the EMECAM study for the Madrid population (Galán et al. 1999).

Table 4. Pearson coefficient correlation of the air pollutants.

Pearson coefficient correlation	SO_2	NO_2	NO_x	PM_{10}	$PM_{2.5}$	O_3	Leq 24
SO_2		0.686	0.850	0.390	0.434	−0.651	0.370
		0.000	0.000	0.000	0.000	0.000	0.000
		1095	1095	1095	1038	1095	1095
NO_2			0.880	0.674	0.706	−0.496	0.277
			0.000	0.000	0.000	0.000	0.000
			1095	1095	1038	1095	1095
NO_x				0.614	0.668	−0.689	0.370
				0.000	0.000	0.000	0.000
				1095	1038	1095	1095
PM_{10}					0.888	−0.184	−0.074
					0.000	0.000	0.014
					1095	1825	1094
$PM_{2.5}$						−0.272	−0.029
						0.000	0.355
						1038	1038
O_3							−0.577
							0.000
							1095

After eliminating SO_2 from the model, the relationship in the case of respiratory failure proved significant at lag 5, with an RR of 1.0816 (1.0119–1.1512) and a PAR of 7.54%. SO_2 is a pollutant that acts synergically with particles and can come to constitute up to 20% of the latter's composition (Ferrís et al. 2003); on many occasions researchers have adopted the course of studying both pollutants jointly (Ballester et al. 2005) or, as in our case, of eliminating SO_2 from the model in view of its high correlation index (Escamilla et al. 2008) (see Table 4).

For mortality due to pneumonia, the relationship was statistically positive at lag 6, with an RR of 1.0438 (1.0001–1.0875) and a PAR of 4.19%. Studies were found which associated $PM_{2.5}$ with admissions due to pneumonia at lag 5 (Halonen et al. 2009) but there are fewer mortality-related data. The EMECAM study reported a relationship between black smoke and pneumonia-related mortality at lag 5 (Arribas 1999). This result, similar to ours, is probably due to the fact that, rather than being associated with an acute inflammatory process capable of developing in the space of minutes, the physiopathological mechanism which leads to death due to pneumonia may instead be associated with a slower process, such as the weakening of defence mechanisms against bacteria, in which the decrease in phagocyte activity (decline in the production of macrophages) may already be in play. No association was found for the remaining specific causes of mortality, such as asthma and COPD.

Lastly, in respect to overall respiratory mortality, seasonal exposure to $PM_{2.5}$ proved significant, not for the summer months, but for the winter months at lag 1, with an RR of 1.0538 (1.0261–1.0815) and a PAR of 5.10%. This pattern does not correspond to what was expected, bearing in mind that both epidemiological and experimental studies have observed a closer association between air pollutants and respiratory mortality in the summer months ?(Hu et al. 2008; Stafoggia et al. 2008; Qian et al. 2008; Stieb et al. 2009), a phenomenon explained, in part, by the higher

concentrations of ozone that serve to increase pulmonary permeability and bronchial hypersensitivity (WHO 2005).

Notwithstanding this, other studies were located in which mortality was likewise higher in the winter months (Ostro et al. 2006), with this being attributed to the different compositions displayed by particles in these two seasons. Greater particle-induced effects on mortality have been associated with areas having high NO_2 concentrations, such as Madrid, not only due to the interaction between the two pollutants, but also due to their being compounds which form part of the most harmful particulate matter in areas where NO_2 levels are also high (WHO 2004). The fact that a Madrid-based study on admissions due to respiratory disease has shown a more marked association for admissions in winter (Díaz et al. in press), supports the hypothesis that $PM_{2.5}$ composition may influence mortality differently during winter and summer (Artíñano et al. 2003). During the winter months in Madrid, carbon increases to the point where it constitutes 70–80% of the total mass. This finding, if taken together with the study conducted by various authors which links higher $PM_{2.5}$ mortality to a higher percentage of organic and elemental carbon, could account for our results (Burnett et al. 2000).

Conclusion

In general, the results obtained indicate a short-term association between overall respiratory mortality and $PM_{2.5}$ concentrations. This association was also statistically significant for total respiratory mortality in winter months and for specific causes, such as pneumonia and respiratory failure. No association was found for the remaining specific causes of respiratory mortality. The remaining chemical air pollutants showed a statistically positive association with mortality due to diseases of the respiratory system, but this association was not consistently maintained across all models.

Particles having a diameter of under 10 micra were not statistically significant for any of the models studied, which confirms that $PM_{2.5}$ is a better indicator of anthropogenic pollution.

Lastly, comparison between the results obtained for respiratory mortality by us and those obtained by other similar-type studies for respiratory-disease admissions (Linares et al. 2008) indicates that increases in $PM_{2.5}$ concentrations have a greater short-term effect on admissions than on mortality, owing to the fact that the physiopathological mechanism triggered, rather than being immediate, is instead preceded by a symptomatology that allows for medical care to be sought and, by extension, for part of the related mortality to be prevented.

$PM_{2.5}$ composition is a very important factor to take into account when studying its damaging effects, since the results suggest that particles from industrial combustion sources and traffic may, on average, have greater toxicity (Zanobetti et al. 2009).

For $PM_{2.5}$ levels above 15 $\mu g/m^3$, the results obtained by the APHEIS-3 study for the city of Madrid establish a reduction in life expectancy of 0.22 years. This same study concludes that a reduction in $PM_{2.5}$ values to levels under 10 $\mu g/m^3$ could prevent anywhere from 37,342–6,061 all-cause deaths in respect of the 23 European cities analysed (Alonso et al. 2005).

Since this is a problem that is largely modifiable and reducible, the application of specific measures aimed at reducing traffic and its emissions would result in a

great benefit, which would have an impact on the real health of citizens, as well as on their perception of more agreeable environmental surroundings (Sinclair et al. 2010).

Insofar as study limitations are concerned, it has to be said that the most important is the ecological fallacy, which bars estimates obtained for the total population being transferred to an individual level.

Furthermore, the study population group covers too wide an age range and is thus too heterogeneous to enable specific conclusions to be established.

It must also be borne in mind that the study focused on analysing particles and using their size for this purpose, without taking their chemical composition – the variability of the different components – into account.

Another possible source of error lies in the quality of the data on $PM_{2.5}$ concentrations detected by the Surveillance Network and the correct validation and classification of the mortality data furnished by the Madrid Regional Revenue Authority.

Moreover, similar $PM_{2.5}$ absorption levels were assumed for all individuals, without regard being had to individual variations in exposure or personal characteristics that determine lower or higher susceptibility. Finally, working with a longer time series would be advisable in order to improve the conclusions of this study.

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