

Daily respiratory mortality and PM₁₀ pollution in Mexico City: importance of considering place of death

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Daily respiratory mortality and PM₁₀ pollution in Mexico City: importance of considering place of death. M.M. Téllez-Rojo, I. Romieu, S. Ruiz-Velasco, M-A. Lezana, M. Hernández-Avila. ©ERS Journals Ltd 2000.

ABSTRACT: Significant associations have been reported between particles with a 50% cut-off aerodynamic diameter of 10 μm (PM₁₀) and ozone ambient concentrations, and daily number of deaths from respiratory causes. The aim of the present study was to assess such associations among elderly (≥ 65 yrs) residents of Mexico City.

Ambient air pollution data were provided by the Metropolitan Monitoring Network. During the study period, the average daily PM₁₀ ranged 23.4–175.3 $\mu\text{g}\cdot\text{m}^{-3}$, and ozone 1 h daily maximums ranged 39.4–216.7 ppb. Information was compiled on the primary and underlying causes of death. The analyses were conducted separately according to place of death (within or out of a hospital unit) using time-series methodology.

The total number of deaths from all respiratory causes and mortality for chronic obstructive pulmonary diseases (COPD) were significantly related to PM₁₀ over different lags: an increase of 10 $\mu\text{g}\cdot\text{m}^{-3}$ was related to a 2.9% (95% CI: 0.9–4.9%) increase and to a 4.1% (95% CI: 1.3%–6.9%) increase with a 3-day lag when death occurred out of medical units, respectively. For deaths occurring in medical units, a longer lag and smaller risk estimate was observed. An interactive effect between PM₁₀ and ozone was detected.

This study confirms that there is an important impact of PM₁₀ on respiratory morbidity among elderly subjects. It also indicates that accounting for primary and underlying causes of death, and considering place of death may reduce misclassification and provide more accurate estimates of the adverse impact of PM₁₀ on mortality. *Eur Respir J 2000; 16: 391–396.*

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A series of recent studies have reported significant associations between daily number of deaths and levels of ambient air pollutants, especially levels of particulate matter. These associations have been found across a wide range of air pollutant levels and weather patterns in western countries [1–6] as well as in some developing countries [7]. However, many factors, such as particulate composition, exposure pattern, simultaneous exposure to other pollutants, and underlying health status may affect the association and explain variations in the effects observed [1, 4].

Although these reports indicate that exposure to particulate air pollution is related to short-term increases in mortality and morbidity, in general, they only include primary cause of death which provides limited information on the underlying associated causes. Such an omission will lead to an underestimation of the daily counts of specific causes of death and subsequently could lead to an underestimation of the effect of air pollution. In addition, it is likely that the place of death (within or out of a medical unit) reflects the exposure to air pollutants on the days preceding death. Therefore, it would be expected that the acute effect of air pollution on specific causes of death have a different pattern depending on the place where death occurred, particularly with regards to lag structure.

In this study, the results of a time-series study conducted in Mexico City, Mexico are reported, in which the association between mortality, due to respiratory causes, and air pollution was evaluated in an elderly population (≥ 65 yrs). Information on primary and underlying causes of death as well as on the place of death was obtained. Because Mexico City experiences high levels of both PM₁₀ and ozone, it was possible to study the interactive effect of these pollutants on death from respiratory causes.

Material and methods

This study was carried out in Mexico City, a city of ~8,600,00 people, from January 1–December 31, 1994. The study population comprised of people age ≥ 65 yrs who died during the study period and who lived in the city at the time of death.

Air pollution and meteorological data were provided by the automatic network of ambient monitoring of Mexico City, which comprised of 33 stations, 22 of which were located within the Distrito Federal, and 11 in surrounding areas of the city. The stations recorded daily concentrations of ozone (O₃), nitrogen dioxide (NO₂), sulphur dioxide (SO₂), and PM₁₀, as well as meteorological variables such

as temperature, relative humidity, and wind speed. These concentrations were measured in adherence with the USA Environmental Protection Agency (EPA) [4] standard methods. The study refocused on the association of the daily variations of O_3 and PM_{10} and deaths due to respiratory causes, because these pollutants reach high levels frequently exceeding the Mexican standard and constitute the main health concern for residents of Mexico City.

A 24-h average, and maximum 1-h and 8-h moving averages were used to reflect daily ozone levels. Particulate samplers recorded 24-h average concentrations during the study.

On the basis of statistical criteria including goodness of fit and maximum correlation, as well as on the units in which norms are stated, it was decided to use 1-h ozone daily maximum and 24-h average for PM_{10} in the final regression model.

The mean population exposure was estimated by averaging the measurements provided by all monitors that recorded the corresponding pollutants. The mean correlation coefficients assessed between monitoring stations were 0.43 for PM_{10} and 0.48 for O_3 .

The Ministry of Health in Mexico provided the death certificates, which included individual information such as age, sex, county of residence, place of death (in or out of a medical unit), and primary and underlying causes of death. Trained health professionals reviewed and extracted data from these certificates and categorized them by date, causes, and place of death.

To date, most of the studies relating mortality to ambient air pollution have considered only the primary cause of death [4]; however, incorporating the information on underlying causes, may help in giving a better understanding of the relationship between air pollution and death due to respiratory causes. The causes of death were classified into two groups according to International Classification of Diseases, ninth revision (ICD-9): 1) total respiratory diseases, which includes chronic obstructive pulmonary disease (COPD) (ICD-9 490-496), and lower respiratory infections (ICD-9 466, 480-487) and 2) the specific group for COPD. The outcome variables were defined as the daily number of deaths for each of these groups.

Statistical Analysis

The daily number of total respiratory deaths and the number of deaths due to COPD, and their relationship with the daily levels of O_3 and PM_{10} were modelled using a time series analysis with Poisson regression [8].

The outcome variables were modelled controlling for long- and short-term trends that might have caused confounding [9-10], following the methodological issues proposed by SCHWARTZ *et al.* [11]. Long-term trends were adjusted for by controlling for cold or warm months (October-January/February-September). Short-term trends were controlled for by including in the model the minimum temperature on the day preceding death; in addition, adjustment for place of death was performed and no remaining seasonal effect was observed. When variables such as, time, month, relative humidity, day of the week and holidays were included in the model, the results remained similar; therefore, the most parsimonious model was reported.

Single pollutant models exploring different lag periods ranging 1-7 days were tested and the cumulative effect was examined by averaging levels over 3, 5 and 7 days. The effects of each pollutant were examined separately, stratifying for place of death and compared using an unpaired t-test.

To account for potential serial autocorrelation due to longitudinal data and overdispersion, the final models were estimated using the Iteratively Weighted and Filtered Least-Squares method (IWFLS), which can be considered as a generalization of the Poisson regression that takes into account these issues [12]. Since the results were very similar to those found using Poisson regression, it was decided to present those obtained with the standard technique. An outlier was detected on March 13, 1994 that showed a strong influence in the models. On that day, only one death from respiratory causes occurred, a stark contrast to the observed mean of 13.5 deaths per day; therefore, this outlier was excluded from the data.

The goodness of fit of the final model was checked using a quantile-quantile normal plot of the deviance residuals [8]. The relationship between the total mortality from respiratory causes occurring within a medical unit with a 3-day lag for PM_{10} adjusted for minimum temperature and cold/warm months is shown in fig. 1. The probability distribution for the deviance residuals is close enough to a normal distribution to validate the model; the residuals of the remaining models exhibited a similar pattern. The proposed core models for deaths occurring inside and outside a medical unit reduced the total deviance by 12.23% and 14.47%, respectively.

Results

Summary statistics for pollutants and meteorological variables are shown in table 1. Of the 20,669 deaths that occurred during the study period, 4,919 (23.8%) were attributed to respiratory causes; of these 2,294 (11.1% of total) were due to COPD. The average number of daily respiratory deaths was 13.5 and were similarly distributed in and out of medical facilities (7.2 and 6.3 respectively). For COPD, the daily average was 6.3 and was also similarly distributed in and out of medical units (3.1 and 3.2, respectively).

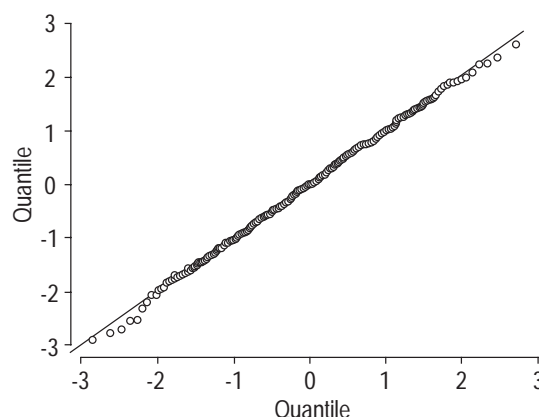


Fig. 1. – Quantile-quantile normal plot of deviance residuals for the Poisson regression model for total deaths due to respiratory causes in a medical unit, for PM_{10} lag 3, adjusting for minimum temperature and season. Mexico, Distrito Federal, 1994.

Table 1. – Summary statistics for air pollutants and meteorological variables in Mexico, Distrito Federal, 1994

	Range	Mean	SD	Percentiles		
				25%	50%	75%
PM10: daily average $\text{mg}\cdot\text{m}^{-3}$	23.4–175.3	75.1	23.7	58.6	74.4	88.3
O ₃ 1 h max ppb	39.4–216.7	134.5	33.4	116.7	135.8	156.6
NO ₂ : 1 h ppm	0.019–0.073	0.038	0.010	0.031	0.037	0.045
SO ₂ : daily average ppm	0.009–0.047	0.020	0.005	0.017	0.019	0.021
Minimum temperature daily average °C	1.5–15.8	10.8	2.2	9.4	11.0	12.4
Total daily mortality from respiratory causes						
Within medical unit	0–18	7.2	2.8	5	7	9
Out of medical unit	1–17	6.3	2.7	4	6	8
Daily mortality for COPD						
Within medical unit	0–12	3.1	1.8	2	3	4
Out of medical unit	0–11	3.2	1.9	2	3	4

PM10: particles with a 50% cut-off aerodynamic diameter of 10 μm ; O₃: Ozone; NO₂: nitrogen dioxide.

Time series for PM10 and O₃ are presented in fig. 2. During the study period the Mexican standard for O₃ (110 ppb 1-h maximum) was exceeded on 287 days (79%) and the corresponding for PM10 (150 $\mu\text{g}\cdot\text{m}^{-3}$ 24-h average) on 3 days. The 24-h average PM10 levels exceeded 50 $\mu\text{g}\cdot\text{m}^{-3}$ on 85% of the days. The observed correlation between PM10 and O₃ was 0.46 ($p < 0.01$). The highest concentrations of particulate matter, ranging 35.9–175.3 $\mu\text{g}\cdot\text{m}^{-3}$, with a mean of 84.5 $\mu\text{g}\cdot\text{m}^{-3}$ were registered during the cold months; during the warm months, these levels ranged 23.4–130.9 $\mu\text{g}\cdot\text{m}^{-3}$, with a mean of 70.3 $\mu\text{g}\cdot\text{m}^{-3}$. Ozone levels were relatively homogenous over the year. During the cold months, the levels ranged 39.4–216.7 ppb with a mean of 139.5 ppb, while during the warm months, the levels ranged 48.9–212.3 ppb with a mean of 131.9 ppb (fig. 2).

Table 2 shows the association between air pollutant levels and the number of daily deaths caused by any respiratory disease that occurred in and out of a medical unit. Relative risks were calculated for an increase of 10 $\mu\text{g}\cdot\text{m}^{-3}$ for PM10 and 40 ppb for ozone. The number of daily deaths occurring out of a medical unit was strongly associated with particulate levels; it was observed that an increase of 10 $\mu\text{g}\cdot\text{m}^{-3}$ of PM10 levels was related to an in-

crease of 2.4% (95% confidence interval (CI): 0.4%, 4.5%) in the number of daily deaths occurring outside a medical unit from respiratory causes ($p < 0.05$) with a 1-day lag. For lags of 2, 3, 4 and 5 days, an increase in the number of daily deaths from respiratory causes of 2.7% (95% CI: 0.7%, 4.7%), 2.9% (95% CI: 0.9%, 4.9%), 2.6% (95% CI: 0.6%, 4.5%) and 2.0% (95% CI: 0.06%, 4.0%) respectively were observed. For an increase of 10 $\mu\text{g}\cdot\text{m}^{-3}$ in the 5-day average PM10 levels, a significant increase of 4.2% (95% CI: 1.7%, 6.8%) in the number of deaths from respiratory causes was observed.

The mortality pattern occurring within compared to outside a medical unit differed significantly, where lower effects and longer lag structures were observed. Increases of a 24-h average of PM10 were associated with a significant increase in the daily respiratory mortality with 5 and 7-day lags. Cumulative exposure to PM10 over 5 and 7 days was also related to mortality from respiratory causes.

A weak association between O₃ levels and deaths occurring out of medical units were observed. An increase of 40 ppb in the maximum daily 1-h ozone level was associated with a 5.6% increase in daily mortality with a 2-days lag. A significant association for cumulative exposure over 3 and 5 days was also noted, but observed no association between deaths for respiratory causes that occurred within medical units and O₃ levels.

Figure 3 presents the smoothing relationship between the estimated total number of daily deaths due to respiratory illnesses occurring outside a medical unit and the 3-day lag exposure of PM10. The smooth curve was constructed using the Lowess technique [13], and suggests a linear relationship between daily mortality and PM10 over the range of ambient levels observed in this study [13].

Daily mortality for COPD presented similar patterns in the lag structure, cumulative exposure, and differences between deaths occurring in and out of medical unit than those observed for the total number of deaths from respiratory causes. However, as shown in table 3, the estimated regression coefficients were larger for deaths from COPD than those obtained for all deaths from respiratory causes. An increase of 10 $\mu\text{g}\cdot\text{m}^{-3}$ in the daily PM10 average was related to 3.0% (95% CI: 0.1%, 5.9%) increase in daily deaths from COPD occurring out of medical units with 1-day lag. This estimate reached 6.1% (95% CI: 2.4%, 9.9%) when a cumulative exposure over a period of 5 days was considered. For deaths due to COPD that

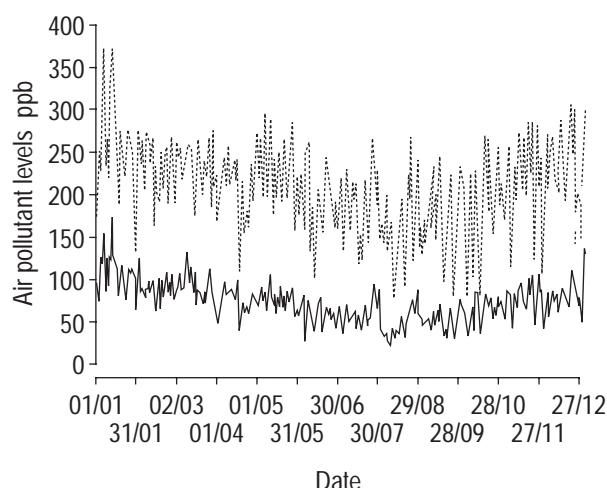


Fig. 2. – Time series of ozone and PM10 in Mexico, Distrito Federal, 1994. The x-axis represents the day and the month within 1994. - - -: ozone, parts per billion; —: particles with a 50% cut-off aerodynamic diameter of 10 μm , $\mu\text{g}\cdot\text{m}^{-3}$.

Table 2. – Regression coefficients and relative risk (RR) for total number of deaths from respiratory causes adjusted for minimum temperature and season in Mexico, Distrito Federal, 1994

Variables	Outside medical unit		Inside medical unit		p-value [#]
	RR*	95% CI	RR*	95% CI	
PM ₁₀					
Lag 1	1.0244	1.0043–1.0448	1.0157	0.9971–1.0347	0.000
Lag 2	1.0268	1.0073–1.0465	1.0112	0.9933–1.0295	0.000
Lag 3	1.0286	1.0090–1.0486	1.0156	0.9975–1.0341	0.000
Lag 4	1.0255	1.0059–1.0454	1.0091	0.9910–1.0275	0.000
Lag 5	1.0201	1.0006–1.0399	1.0236	1.0054–1.0421	0.000
Avg 3	1.0377	1.0141–1.0619	1.0200	1.0010–1.0422	0.000
Avg 5	1.0422	1.0166–1.0684	1.0245	1.0010–1.0485	0.000
Avg 7	1.0393	1.0126–1.0668	1.0278	1.0031–1.0531	0.000
O ₃					
Lag 1	1.0173	0.9673–1.0699	1.0378	0.9900–1.0879	0.000
Lag 2	1.0558	1.0048–1.1094	1.0164	0.9707–1.0642	0.000
Lag 3	1.0359	0.9863–1.0880	0.9978	0.9532–1.0444	0.001
Lag 4	1.0297	0.9802–1.0816	1.0315	0.9851–1.0801	0.321
Lag 5	1.0403	0.9898–1.0934	1.0228	0.9766–1.0712	0.000
Avg 3	1.0793	1.0036–1.1608	1.0379	0.9700–1.1105	0.000
Avg 5	1.1403	1.0410–1.2490	1.0686	0.9821–1.1628	0.000
Avg 7	1.1013	0.9902–1.2248	1.0890	0.9867–1.2020	0.004

PM₁₀: particles with a 50% cut-off aerodynamic diameter of 10 μm ; lag: estimate of the latency period; Avg: estimate of the cumulative exposure; O₃: ozone; 95% CI: 95% confidence interval; *: relative risk was computed for an increase at 10 $\mu\text{g}\cdot\text{m}^{-3}$ of PM₁₀ or 40 ppb at ozone; #: comparing coefficients from the regression models according to place of death.

occurred in medical units, the effect was significantly smaller, and the lag period was longer. The important effect of O₃ ambient levels on death from COPD was smaller than that observed for PM₁₀.

Because of the high correlation between PM₁₀ and O₃ levels, the authors studied the effect of PM₁₀, stratifying for days with high and low O₃ levels; the cut-off point used was the observed median (116.7 ppb), which is very close to the standard established by EPA [14]. No significant effect of PM₁₀ during the days when O₃ levels were low were observed (table 4); however, on days when O₃ levels exceeded the cut-off point, a significant effect of PM₁₀ on the total number of respiratory deaths and death due to COPD that occurred out of medical units was observed. This finding suggests a synergic effect of PM₁₀ and O₃

on respiratory mortality on days when ambient O₃ levels are high.

Multi-pollutant models were fitted and PM₁₀ levels remained significantly related to respiratory mortality, but the effect of O₃ was no longer significant. SO₂ levels were not significantly related to respiratory deaths. When stratifying the data based on the median of SO₂, no interactive effect between SO₂ and PM₁₀ was found.

Discussion

In this study, an important impact of PM₁₀ on respiratory mortality among elderly residents of Mexico City was observed. For deaths occurring out of medical units, a 4.1% (95% CI: 1.3–6.9%) increase in daily COPD mortality associated with a 3-day lag increase of 10 $\mu\text{g}\cdot\text{m}^{-3}$ in the daily average level of PM₁₀ was observed. When cumulative exposure over 5 days was considered this estimate reached 6.1% (95% CI: 2.4–9.9%). The relationship appeared linear over the range of ambient levels observed in the study. For deaths occurring within the medical unit, the effects were significantly smaller and less consistent. Results also suggest an interaction between PM₁₀ and O₃ effects on respiratory mortality.

Several studies have investigated the association of air pollution with mortality for respiratory causes. In general, these estimates have been higher (ranging from a 1.5–3.7% per 10 $\mu\text{g}\cdot\text{m}^{-3}$ increase in PM₁₀) than for deaths from all causes occurring in the general population [3], especially among the elderly [2–5]. These estimations are similar to those observed in the study for deaths occurring within medical units. However, the impact of PM₁₀ on deaths due to respiratory causes appears larger in the presented study population when one only considers those deaths that occurred out of medical facilities. There was also a longer lag effect for those deaths that occurred within

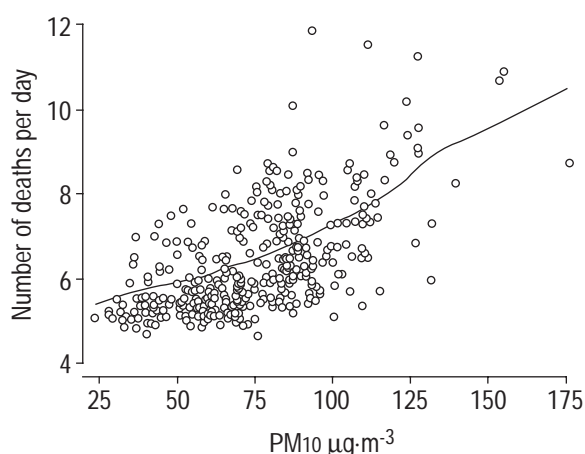


Fig. 3. – Smooth function of the number of daily death by total respiratory causes outside a medical unit and particles with a 50% cut-off aerodynamic diameter of 10 μm levels with a 3-days lag. Mexico, Distrito Federal, 1994.

Table 3. – Regression coefficients and relative risk (RR) for total number of deaths from chronic obstructive pulmonary disease adjusted for minimum temperature and season in Mexico, Distrito Federal, 1994

Variables	Outside medical unit		Inside medical unit		p-value [#]
	RR*	95% CI	RR*	95% CI	
PM10					
Lag 1	1.0295	1.0012–1.0585	1.0189	0.9907–1.0479	0.000
Lag 2	1.0403	1.0127–1.0686	1.0191	0.9917–1.0472	0.000
Lag 3	1.0407	1.0128–1.0693	1.0273	0.9995–1.0559	0.000
Lag 4	1.0350	1.0073–1.0635	1.0249	0.9971–1.0534	0.000
Lag 5	1.0339	1.0063–1.0624	1.0329	1.0051–1.0614	0.325
Avg 3	1.0525	1.0189–1.0872	1.0304	0.9973–1.0647	0.000
Avg 5	1.0609	1.0242–1.0988	1.0407	1.0046–1.0781	0.000
Avg 7	1.0556	1.0174–1.0952	1.0422	1.0044–1.0814	0.000
O ₃					
Lag 1	1.0419	0.9702–1.1189	1.0458	0.9735–1.1235	0.165
Lag 2	1.0452	0.9748–1.1207	0.9903	0.9237–1.0617	0.000
Lag 3	1.0826	1.0096–1.1609	1.0176	0.9492–1.0910	0.000
Lag 4	1.0017	0.9349–1.0733	1.0559	0.9842–1.1328	0.000
Lag 5	1.0160	0.9474–1.0897	1.0745	1.0009–1.1536	0.000
Avg 3	1.1279	1.0176–1.2503	1.0405	0.9388–1.1531	0.000
Avg 5	1.1556	1.0402–1.2838	1.1355	0.9981–1.2919	0.000
Avg 7	1.1191	0.9632–1.3003	1.1628	1.0004–1.3516	0.000

PM10: particles with a 50% cut-off aerodynamic diameter of 10 µm; lag: estimate of the latency period; Avg: estimate of the cumulative exposure; O₃: ozone; 95% CI: 95% confidence interval; *: relative risk was computed for an increase at 10 µg·m⁻³ of PM10 or 40 ppb at ozone; #: comparing coefficients from the regression models according to place of death.

medical units than for those occurring out of them. One explanation could be that those who died in medical units have received treatment and have been isolated from outdoor pollution. Additional information on the time spent in hospital before death would help to understand the different patterns observed between deaths occurring inside/outside medical unit; unfortunately, the authors did not have access to these data. Based on the results, it seems that the lack of stratification by place of death may lead to an underestimation of the impact of air pollution on mortality and may also obscure the lag structure. Another important factor to account for in estimating the impact of air pollution on health is the underlying cause of death. In this study, the impact of 10 µg·m⁻³ of PM10 on deaths for respiratory causes increased from 0.6–2.9% when underlying as well as primary causes of death were

considered. For deaths due to COPD these estimates raised from 2.0–4.1%.

In an earlier study conducted in Mexico City, BORJA *et al.* [15] reported an excess mortality of 0.5% (95% CI: 0.3–0.7%) associated with a 10 µg·m⁻³ increase in total suspended particulates (TSPs) assuming that the PM10/TSP ratio is close to 0.50, this estimate corresponds approximately to a 1.0% (95% CI: 0.6–1.4%) per 10 µg·m⁻³ increase of PM10. For mortality from respiratory causes, (mortality that is most likely to occur among the elderly) the estimates reported would correspond to an increase of 1.9% (95% CI 0.3–3.6%) per 10 µg·m⁻³ increase in the PM10 daily mean. However, in this study, TSP was only measured by one monitor over the entire city every 6 days, therefore lag effects could not be fully explored. The risk estimates reported in the study of BORJA-ABURTO

Table 4. – Particulate matter with a 50% cut-off aerodynamic diameter of 10 µm (PM10) effect on daily mortality stratified for ozone levels in Mexico, Distrito Federal, 1994

		Upper ozone levels [#]		Lower ozone levels [#]	
		RR*	95% CI	RR*	95%CI
Total respiratory mortality					
Outside medical unit	PM10 (3 days lag)	1.133	1.054–1.218	0.973	0.858–1.103
	PM10 (avg 5 days)	1.173	1.076–1.278	0.905	0.725–1.128
Inside medical unit	PM10 (3 days lag)	1.031	0.963–1.103	1.090	0.970–1.225
	PM10 (avg 5 days)	1.058	0.977–1.146	1.117	0.912–1.367
COPD					
Outside medical unit	PM10 (3 days lag)	1.166	1.053–1.290	0.996	0.831–1.193
	PM10 (avg 5 days)	1.232	1.092–1.391	0.963	0.704–1.319
Inside medical unit	PM10 (3 days lag)	1.063	0.960–1.178	1.123	0.938–1.344
	PM10 (avg 5 days)	1.096	0.971–1.237	1.143	0.833–1.567

COPD: chronic obstructive pulmonary disease; RR: relative risk; 95% CI: 95% confidence interval; lag: estimate of the latency period; Avg: estimate of the cumulative exposure; #: using the median ozone level (1116.7 parts per billion) with the same lag of PM10 as a cut-off point; *: relative risk was computed for a 30 µg·m⁻³ increase in PM10.

et al. [15] are considerably lower than those observed in the present population. Several factors, particularly the lack of accounting for lag effect and the fact that primary and underlying causes of death were considered in the present study, may explain this difference. A more recent study, BORJA-ABURTO *et al.* [16], evaluated the impact of particles with a 50% cut-off aerodynamic diameter of 2.5 μm (PM_{2.5}) on total mortality among residents of the southwest part of Mexico City; and reported that a 10 $\mu\text{g}\cdot\text{m}^{-3}$ increase in the level of PM_{2.5} was associated with a 2.5% increase in mortality among people ≥ 65 yrs of age. Assuming a ratio of 0.7 between PM_{2.5} and PM₁₀ as observed in previous studies conducted in the southwest part of Mexico City, this estimate will correspond to an increase of 3.8% in the number of deaths. This estimate is close to the results that were observed in the present study when deaths occurring outside and inside medical units were combined (3.3% 95% CI: 1.3–5.3%).

As observed in other studies [2–6], an exposure-response relationship between PM₁₀ and mortality from respiratory causes, that is consistent with a monotonic increase across the range of particulates, was observed in this study. A small increase in deaths due to respiratory causes associated with O₃ exposure was also observed. However, this effect was not consistent and was mostly present when exposure over several days was considered. More relevant was the interaction observed between PM₁₀ and O₃ on mortality from respiratory causes. When days of the study were classified according to O₃ levels, it was observed that the relationship between PM₁₀ and respiratory mortality was only significant on those days when O₃ levels were high (>116.7 ppb). The cut-off point to classify low or high ozone levels was close to current EPA standards [14]. Such interaction has not previously been reported, probably because few cities have concomitant high levels of PM₁₀ and O₃ pollution. This interaction could also partly explain the strong effects of PM₁₀ observed in this study.

One of the major limitations of time-series studies is that exposure is estimated at the population level and is based on ambient monitoring networks. Because of the large potential for misclassifying exposures and outcomes, it has been argued that the effects observed in this type of study is likely to underestimate the true effect [17]. Although in the present study, the estimation of exposure was based on data that was obtained from ambient monitoring stations, considering underlying as well as primary causes of death and accounting for place of death are likely to have decreased the possibility of misclassification and to have provided a more accurate estimate of the impact of air pollutants on deaths from respiratory causes in the elderly.

References

1. Thurston G. A critical review of PM₁₀ mortality time series studies. *J Expos Anal Environ Epidemiol* 1996; 6: 3–2.
2. Pope CA III, Bates D, Riazanne ME. Health effects of particulate air pollution: time for reassessment. *Env Health Perspect* 1995; 103: 472–480.
3. Dockery D, Pope A. Epidemiology of acute health effects: summary of time-series studies. In: Particles in our air. Concentration and health effects. Wilson R, Spengler J, Eds. Cambridge, MA, Harvard University Press, 1996. pp. 123–148.
4. US Environmental Protection Agency (1996). Air quality criteria for particulate matter Vols. I–III. Washington, DC, EPA, Office of Research and Development, 1996.
5. Kelsall JE, Samet JM, Zeger SL, Xu J. Air pollution and mortality in Philadelphia 1974–1988. *Am J Epidemiol* 1997; 146: 750–762.
6. Schwartz J. Air pollution and daily mortality: a review and meta analysis. *Environ Res* 1994; 64: 36–52.
7. Romieu I, Borja-Aburto VH. Particulate air pollution and daily mortality: can results be generalized to Latin American countries. *Salud Publica Mex* 1997; 39: 403–411.
8. McCullagh P, Nelder JA. Generalized linear models. London: Chapman & Hall, 1989.
9. Schwartz J, Dockery DW, Neas LN. Is daily mortality associated specifically with fine particles. *J Air Waste Manage Assoc* 1996; 46: 927–939.
10. Samet JM, Zeger SL, Berhane K. The association of mortality and particulate air pollution. Replication and validation of selected studies. Andover, MA, Health Effect Institute, 1995.
11. Schwartz J, Spix C, Touloumi G, *et al.* Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J Epidemiol Comm Health* 1996; 50: S3–S11.
12. Liang KY, Zeger SL. Regression analysis for correlated data. *Annu Rev Public Health* 1989; 8: 551–561.
13. Fan J, Gijbels I. Local polynomial modelling and its applications. London, Chapman & Hall, 1996; 24–27.
14. US Environmental Protection Agency. Air Quality Criteria for Ozone and Related Photochemical Oxidants. Vol III. Washington DC, EPA, Office of Research and Development. EPA/600/P-93/004cF. July 1996.
15. Borja-Aburto VH, Loomis DP, Bangdiwala SI, Shy CM, Rascon Pacheco RA. Ozone, suspended particulates, and daily mortality in Mexico City. *Am J Epidemiol* 1997; 145: 258–268.
16. Borja-Aburto VH, Castillejos M, Gold DR, Bierzwinski S, Loomis D. Mortality and ambient fine particles in southwest Mexico City, 1993–1995. *Env Health Perspect* 1998; 106: 849–855.
17. Hatch M, Thomas D. Measurement issues in environmental epidemiology. *Env Health Perspect* 1993; 101: 49–57.