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RESEARCH ARTICLE

Short-term effect of fine particulate matter ($PM_{2.5}$) and ozone on daily mortality in Lisbon, Portugal

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Abstract
Introduction Urban ambient air pollution exposures continue to be a global public health concern. Although air quality targets are often exceeded in Lisbon, the largest city in Portugal, there is currently no study that has assessed the quantitative impact of these pollutants on daily mortality.
Materials and method In this study, we conduct a time series analysis using generalized additive modeling to determine the exposure-response effect from ambient ozone (O_3) and fine particulate matter ($PM_{2.5}$) concentrations on daily mortality in Lisbon. The dataset used was limited to the Lisbon municipality and for the period 2004–2006.
Results and conclusion For $PM_{2.5}$ exposures, we found that the relative risk for cardiovascular mortality in the population group ≥65 years is 2.39% (95%C.I. 1.29%, 3.50%) for each 10 μg/m^3 increase. A statistically significant cause–effect relationship for $PM_{2.5}$ and mortality was not observed in other population groups. We also report O_3 exposures to be associated with an increase of 1.11% (95% C.I. (0.58, 1.64)) for all-cause mortality in the population group ≥65 years and an increase of 0.96% (95%C.I. (0.56, 1.35)) for the general population. When analyzing by cause of death, our results showed a stronger association between O_3 exposure and cardiovascular mortality.

Keywords Air pollution · Ozone · PM2.5 · Time series · Mortality · Lisbon · Portugal

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1 Introduction

There is a significant volume of studies providing evidence that exposure to urban air pollution is a public health concern (Katsouyanni et al. 2009). These studies typically focus on particulate matter (PM), nitrogen oxides (NOx), and ozone (O_3) exposures.

In urban atmospheres, PM can generally be separated into three major fractions on the basis of particle size: coarse particles (PM_{10}) with aerodynamic diameter of ≤10 μm, fine particles ($PM_{2.5}$) with aerodynamic diameter ≤2.5 μm, and ultrafine particles ($PM_{0.1}$) with aerodynamic diameter ≤0.1 μm. Air quality guidelines are typically set for PM_{10} and $PM_{2.5}$ ambient levels. The former are primarily produced by mechanical processes such as construction activities, road dust re-suspension, and wind-blown dust from uncovered soils, while the latter are primarily from combustion sources. The ratios and composition of particles in these two sizes vary across cities, depending on local geography, meteorology, and specific sources. In Europe, particulate emissions between 1990 and 2004 decreased in most countries except for Portugal, Greece, Iceland, and Turkey, where increases were recorded (EEA 2007).

The health effects associated with the different PM sizes also differ. PM_{10} are able to penetrate the thoracic region, while $PM_{2.5}$ particles can penetrate deeper into the lungs and have a high probability of deposition in the

smaller conducting airways and alveoli. Hence, the former is more likely to cause local respiratory effects, while the latter is more likely to affect other body systems as well (Brunekreef and Holgate 2002).

Epidemiological studies show that particulate matter (PM) exposure affect morbidity and mortality. For short-term PM exposures, the effect estimates for cardiovascular and respiratory causes of mortality are often higher than all-cause mortality (Cohen et al. 2005; Katsouyanni et al. 2009). Other studies have shown associations with morbidity indicators, including increases in hospital admissions, asthma, and cardiovascular diseases (WHO 2006). Traditionally, epidemiological studies were conducted using PM_{10} as the exposure indicator. In its recent air quality guideline report, the World Health Organization (WHO) recommends that exposure indicators based on $PM_{2.5}$ rather than PM_{10} should be encouraged (WHO 2006). However, currently, there are very few European studies that use the $PM_{2.5}$ indicator (Ballester et al. 2008).

Ozone is a secondary air pollutant formed primarily through a complex series of photochemical reactions between nitrogen oxides and reactive hydrocarbons. The key precursor pollutants for O_3 formation are NOx and volatile organic compounds, both of which have increased in Portugal in recent years (EEA 2007). The principal sources of these precursors in urban areas are emissions from motor vehicles, although vegetation can also contribute significant amounts (Fuentes et al. 2000). A recent European Environmental Agency report concluded that while in most European countries, no exceedances above health and environmental protective O_3 thresholds are reported, the situation in Portugal is different (EEA 2010). Short-term exposure to O_3 has shown a positive association with all-cause and respiratory mortality and, to a lesser extent, cardiovascular mortality (Gryparis et al. 2004).

While there is general agreement that urban air pollution is hazardous to human health, the exposure-response relationships differ between studies and locations (Gryparis et al. 2004). Exposure-response relationships determined within a particular country/region may thus not be readily transferable to others.

Studies in the Lisbon metropolitan area show frequent exceedances of EU directive targets for air quality (Almeida et al. 2007). However, studies focusing on the health impacts of air quality in Lisbon are surprisingly very few and none have quantitatively assessed the impacts of short-term exposure on mortality (Casimiro et al. 2006; Alves et al. 2010). The most comprehensive study conducted in the region demonstrated significant associations between hospital admission of cases of circulatory and cardiac diseases for all age groups and proxy markers (NO_2 and CO) of road traffic pollution (Alves et al. 2010). The goal of our study was to conduct a time series analysis using generalized additive modeling to determine the exposure-response effect from ambient O_3 and $PM_{2.5}$ concentrations on daily mortality in the municipality of Lisbon.

2 Materials and methods

2.1 Study area

The study area is the municipality of Lisbon (hereafter referred to as Lisbon), capital of Portugal and the westernmost capital in mainland Europe. Lisbon is set on Seven Hills, north of the Tagus estuary and in close proximity to the Atlantic Ocean. It is the largest urban area in Portugal with a population of about 0.5 million.

Lisbon has specific social characteristics such as large proportions (24%) of the residential population are over 65 years, and it has one of the highest population densities (6,134 inhabitants per square kilometers) in the country. It is, thus, not surprising that it has one of the highest mortality rates (14.1‰), significantly higher than the national rate of 9.7‰.

Services are the main economic sectors in the city, although there is some industrial activity. The latter includes textiles, chemicals, steel, oil and sugar refining, shipbuilding, and waste incineration units. However, traffic is the main source of local atmospheric emission. Apart from road traffic, the international airport located in the north of the city is also a significant contributor to local emissions.

2.2 Environmental and health data

Hourly data of O_3 and $PM_{2.5}$ for the period starting January 17, 2004 and ending December 31, 2006 was obtained from the Portuguese Environmental Agency. Data from the urban background monitoring station of Olivais was used as this is the only background station monitoring for both parameters in Lisbon. Previous studies in Lisbon have demonstrated that this station represents the air quality in the municipality well (Ferreira et al. 2007; Tente 2005). Hourly O_3 and $PM_{2.5}$ readings were transformed to daily mean, daily 95th percentile, daily 98th percentile, and daily maximum. Only days with more than 75% of the hourly measurements for that particular day were used. This station had an hourly efficacy of 96.4% and 97.6% for $PM_{2.5}$ and O_3, respectively.

Mortality groups were aggregated in accordance to the tenth revision codes of the World Health Organization International Statistical Classification of Diseases and Related Health Problems (ICD-10). Daily death counts for all-cause mortality (except external causes) (ICD-10 codes A00-R99), and for cardiovascular mortality (ICD-10 codes I00-I99) were obtained from the Portuguese Statistics Institute for the same study period and study area. Mortality

Table 1 Summary statistics for daily health and environmental indicators in Lisbon (2004–2006)

	Mean	Max	Min	SD	Variance
All-cause mortality (cases)	20	49	6	6	34
Cardiovascular mortality (cases)	8	23	3	3	11
All-cause mortality ≥65 years (cases)	16	41	3	5	28
Cardiovascular mortality ≥65 years (cases)	8	22	3	3	10
PM 2.5 ($\mu g/m^3$)	14.7	67.6	1.4	10.8	116.4
Ozone ($\mu g/m^3$)	48.3	115.2	4.4	22.4	499.9
Temperature (°C)	17.7	32.3	5.5	5.2	27.3

data were further classified into two groups: one group for all ages and another group ≥65 years.

Daily maximum temperature and daily minimum temperature for Lisbon (2004–2006) were obtained from the European Climate Assessment & Dataset and processed according to Klein Tank et al. (2002). Data for a single meteorological station (Geofísica) located in Lisbon city center was used.

2.3 Statistical analysis

Generalized additive models (GAMs) were used to link air pollution with mortality in the municipality of Lisbon. We assumed that the independent response variables followed a Poisson distribution with a log link function. Thin plate regression spline was chosen as low-rank isotropic smoother with fixed degrees of freedom (Woods 2003, 2004, 2008). The R software (R Development Core Team 2007) version 2.6.0 was used for statistical analysis with the "mgcv" package version 1.4–1. The mathematical formulation of the GAM model is given by:

$$M_i \sim P(\mu_i) \text{ and } E[M_i] = \mathrm{Var}(M_i) = \mu_i$$

$$\mathrm{Log}(\mu_i) = g(x_i), \text{ where } g(x_i) = \alpha + \beta x_1 + f(x_2) + \ldots + f(x_i)$$

In the GAM formula above, M is the mortality counts for each mortality group (i.e., independent variable), α is the intercept, β is the slope and $f(x_n)$ is the smooth function for each covariate.

An independent model was built for each mortality type/group and with each of the following explanatory variables individually: (1) temperature, (2) O_3, and (3) $PM_{2.5}$. For each explanatory variable, we calculated the daily mean, maximum, 95th percentile, and 98th percentile from the hourly dataset. In addition, for each of these variables, we used a lag from 1 to 4 days and a moving average from 1 to 4 days giving a total of 64 explanatory variables. By a lag of 1 day, we mean that the response variable today is influenced by the values of the explanatory variable measured the day before, whereas by a moving average of 1 day, we mean that the response variable is influenced by the explanatory variable average of the current and the day before.

To simulate natural seasonality of mortality, the following variables were created:

(a). cumulative number of days from 2004 to 2006 (TIME);
(b). discrete variable representing each month (MONTH);

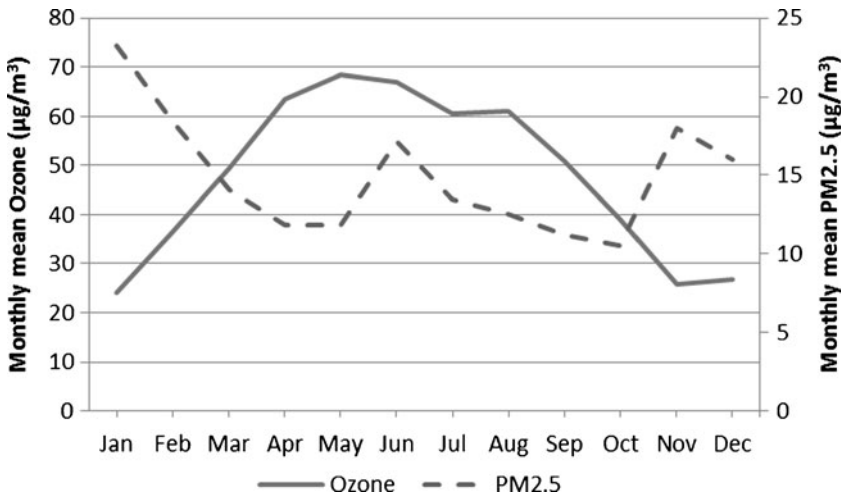

Fig. 1 Monthly average of ozone and PM 2.5 between 2004 and 2006 in Olivais monitoring station

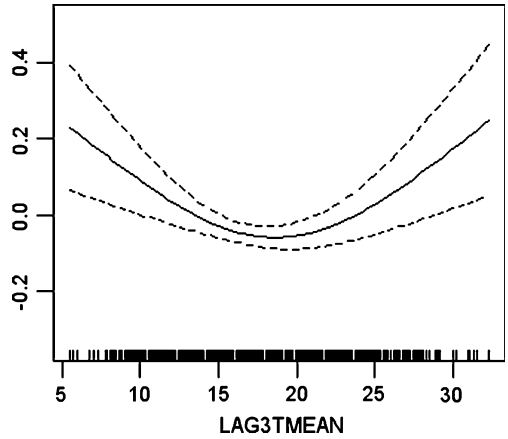

Fig. 2 Estimated effects and 95% confidence bands on cardiovascular mortality ≥65 years for 3 days lag in the daily mean temperature (°C)

(c). discrete variable representing each season (S1=December to February, S2=March to May, S3=June to August, and S4=September to November); and
(d). discrete variable representing the weekend days.

The modeling procedure was done in the following order:

a. Model adjusted to characterize the mortality seasonality using variable (a) as covariate, and (b), (c), and (d) as discrete variables;
b. After having captured the seasonality (step 1 above), each climate and air pollution variable were added separately to determine the independent effect of each one choosing the most significant degrees of freedom.
c. The most significant covariate from step 2 where chosen to form a base model, and the variables O_3 and $PM_{2.5}$ were independently added as a linear function to the base model.

Each covariate was tested with different fixed degrees of freedom, to adjust the amount of smoothness. Model and variable selection was done based on the UBRE score and the Pearson statistic, respectively.

The final outcome shows the relative risk (RR), or the risk of mortality associated with an increase of 10 μg/m^3 of $PM_{2.5}$ and O_3, calculated using the following equation:

$$RR = \exp(\beta i^{*}10) \quad (1)$$

where βi is the regression coefficients associated with a 10 μg/m^3 increment in a pollutant.

3 Results and discussion

In Table 1, we present the summary statistics for mortality, climate, and air pollution variables for Lisbon. The mortality data indicates that total cardiovascular deaths represent more than 40% of total mortality and is thus a

Table 2 Ozone exposure-response model

Covariates	Degrees of freedom	Coefficient (β)	*p* value	Pr (>−z|)
Model 1: effect of ozone on all-cause mortality (all ages)				
Time	15		<2e–16	
Mean PM2.5	3		0.268019	
P95LAG(2) O3		0.0009523		0.0181
LAG(2) TMEAN	5		0.000321	
Weekend				0.0441
Model 2: effect of ozone on all-cause mortality (≥65 years)				
TIME	15		<2e–16	
MA(3)P95 PM2.5	3		0.0686	
P95MA(2) O3		0.0011019		0.0418
LAG(2) TMAX	5		0.0068	
Model 3: effect of ozone on cardiovascular mortality (all ages)				
TIME	15		8.5e–12	
MEAN PM2.5	3		0.28681	
P95MA(3) O3		0.0019525		0.0138
LAG(3) TMEAN	3		0.00822	
Model 4: effect of ozone on cardiovascular mortality (≥65 years)				
TIME	15		3.11e–11	
MEAN PM25	3		0.1522	
P95MA(3) O3		0.0018400		0.0260
LAG(3) TMEAN	3		0.0143	

The prefix *LAG(n)* and *MA(n)* stands for lag and moving average, respectively, where *n* is the number of days. The prefix *P95* and *P98* stands for 95th and the 98th percentile, respectively

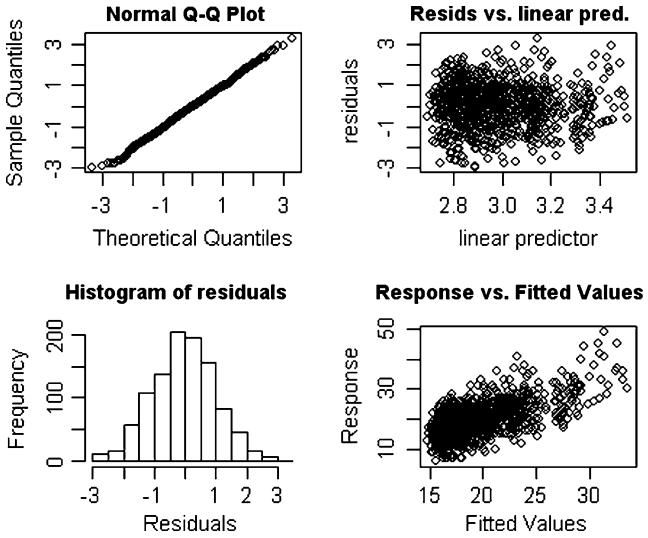

Fig. 3 Validation of normality and homogeneity of the exposure-response model 1 (ozone and all-cause mortality in all ages), described in Table 2

very important group to study in terms of vulnerability. There is a seasonal mortality pattern, with most deaths occurring in the colder months. During the study period, the mean temperature in Lisbon was 17.7°C, reaching a maximum of 32.3°C and a minimum of 5.5°C.

During the study period, the daily mean O_3 concentration was 48.3 μg/m^3 and the daily mean for $PM_{2.5}$ was 14.73 μg/m^3. Compared to other cities in Southern Europe, the O_3 levels are slightly higher, while the $PM_{2.5}$ levels lower (Maté et al. 2010). Figure 1 shows the monthly variation of O_3 and PM2.5 during the study period. A seasonal pattern is evident for both pollutants; ambient O_3 levels are highest between April and September, whereas $PM_{2.5}$ levels are highest during winter. The average PM2.5/PM10 ratio during the study period was 0.48, which is at the lower end of this ratio seen in developed countries (WHO 2006). It is assumed that a significant portion of the PM_{10} levels are due to the well-known contribution of Saharan dust that is frequently blown across Portugal (Wagner et al. 2009).

Modeling the short-term effect of air pollution has some technical statistical challenges. A common challenge is the effect of confounding variables (variables that are at the same time correlated with the dependent and independent variables). For example, O_3 formation requires radiation. The latter is highly correlated with temperature that is also correlated with health problems, so we have three explanatory variables that are correlated with each other and, at the same time, with the response variable (mortality). To determine the effect of each one of these variables separately on mortality, we adopted the following strategies: (1) splines with fixed degrees of freedom were used to determine the amount of smoothness; (2) variables to represent natural seasonality where introduced; and (3) each covariate was tested independently.

Eight base models were developed, two for each age group representing the effect of $PM_{2.5}$ and O_3 indepen-

Table 3 PM2.5 exposure-response model parameters

Covariates	Degrees of freedom	Coefficient (β)	*p* value	Pr (>−z|)
Model 5: effect of PM2.5 on all-cause mortality (all ages)				
Time	15		2e–16	
P98 PM2.5		0.0006687		0.1742[a]
P98MA(2) O3	5		0.00012	
LAG(2) TMEAN	11		0.006727	
Weekend		0.0374875		0.0173
Model 6: effect of PM2.5 on all-cause mortality (≥65 years)				
Time	15		2e–16	
P98 PM2.5		0.0005358		0.249[a]
P98MA(2) O3	6		0.0028	
LAG(2) TMAX	5		0.00969	
Model 7: Effect of PM2.5 on cardiovascular mortality (all ages)				
Time	15		2e–16	
Mean PM2.5		0.001476		0.215[a]
P98 MA(3) O3	6		0.0381	
LAG(3) TMEAN	3		0.0165	
Model 8: Effect of PM2.5 on cardiovascular mortality (≥65 years)				
Time	15		4.9e–12	
Mean PM25		0.002362		0.0326
P95LAG(3) O3	3		0.0502	
LAG(3) TMEAN	3		0.0330	

The prefix *LAG(n)* and *MA(n)* stands for lag and moving average, respectively, where *n* is the number of days. The prefix *P95* and *P98* stands for 95th and the 98th percentile, respectively

[a] not statistically significant at the 5% confidence level

Table 4 Percentage increase in mortality per 10 μg/m^3 of ozone

		Observations	RR	C.I. 95%	%RR
All-cause mortality	Total	1,042	1.0096	(1.0056, 1.0135)	0.96%
	≥65 years	1,040	1.0111	(1.0058, 1.0164)	1.11%
Cardiovascular mortality	Total	993	1.0197	(1.0119, 1.0276)	1.97%
	≥65 years	978	1.0186	(1.0104, 1.0268)	1.86%

dently. For each explanatory variable, the prefix LAG(n) and MA(n) stands for lag and moving average, respectively, were *n* is the number of days. The prefix P95 and P98 stands for 95th and the 98th percentile, respectively.

During the exploratory analysis, each covariate was tested with different fixed degrees of freedom in order to obtain the most suitable model. After having captured the seasonality pattern, each remaining covariate was added separately to the model and tested with fixed degrees of freedom. Figure 2 shows the effect of temperature (LAG3TMEAN) in cardiovascular mortality +65. The effects of temperature in each mortality group showed a "U" shape similar to the one in Fig. 2. In this figure, we show the effect of 3 days lag daily mean temperature in daily cardiovascular mortality over 65 years to be significant for temperatures above 25°C and below 14°C. The most significant variables that had the lowest UBRE score in the model were introduced in the base model. Model comparison and selection was done using the Akaike information criterion. Likewise, the effects of O_3 and $PM_{2.5}$ in each mortality group were tested independently.

After selecting the most suitable set of explanatory variables for each mortality group, these were introduced to form a base model, and each air pollution variable was independently added as a linear function. Model validation was confirmed by checking for normality and homogeneity. We assumed that the mortality data are single independent events. These assumptions were checked by plotting a histogram of the residuals and Q-Q plots for the normality assumption and by plotting the residuals versus the predicted variable for the homogeneity assumption. Model validation results for the normality and homogeneity assumptions for model 1 (see Table 2 for model details) is presented in Fig. 3. Results for the remaining models showed similar trends and were all validated as described above.

Figure 3 shows the Q-Q plot with a relatively straight line and the histogram of the residuals with a normal distribution. These two results validate the normality assumption of our model. In Fig. 3, we also show a scatter plot of the residuals versus the predicted values. The latter has a spread uniformly distributed and without patterns, indicating that there is no violation of homogeneity. Model performance is also checked by looking at the plot with the response variable vs. the fitted values, showing an acceptable fit.

Tables 2 and 3 show the characteristics of the explanatory variables used in the exposure-response model to determine the short-term effect of O_3 and PM $_{2.5}$ in mortality.

Our results show that the short-term health effect of O_3 and PM $_{2.5}$ are different. From Table 2, we see that for model 1, there is a 2-day lag between high O3 levels and all-cause mortality (i.e., P95LAG(2)O3). While for models 2–4, mortality increases when there is an accumulative effect of high O3 levels for two and three consecutive days (i.e., P95MA(2)O3 and P95MA(3)O3). Table 3 shows that only model 4 produced statistical significant results. The latter model indicates a positive effect between cardiovascular mortality for the >65 years group and daily mean concentrations of PM2.5 (i.e., MEANPM2.5). Based on the results presented in Tables 2 and 3, we can conclude that the exposure-response for PM $_{2.5}$ has a mortality effect occurring on the same day, while for O_3, mortality increases occur following 2–3 days of high O3 levels.

Once the βi coefficients of O_3 and PM $_{2.5}$ were calculated, we proceeded to the determination of the relative risk using Eq. 1. Table 4 shows the percentage increase in mortality (%RR) due to an O_3 increase of 10 μg/m^3. We found significant positive associations for the all-age group as well as the ≥65 years group for all-cause and for cardiovascular mortality. The effect was doubled in the cardiovascular mortalities when compared to all-cause mortality. Compared to other city-specific studies, the O_3 exposure relative risks in Lisbon are higher (Bell et al. 2004), suggesting that the Lisbon population is more

Table 5 Percentage increase in mortality per 10 μg/m^3 of PM2.5

		Observations	RR	C.I. 95%	%RR
All-cause mortality	Total	1,040	1.0067	(1.0019, 1.0116)	0.67%[a]
	≥65 years	1,040	1.0062	(1.0010, 1.0115)	0.62%[a]
Cardiovascular mortality	Total	993	1.0149	(1.0032,1.0267)	1.49%[a]
	≥65 years	981	1.0239	(1.0129, 1.0350)	2.39%

[a] not statistically significant at the 5% significance level

vulnerable to ambient O_3 levels. This can be partly explained due to city-specific cultural and socio-economic conditions. For example, in the USA and in most other Southern European cities, the use of air conditioning is common in the summer (when O_3 levels are highest). Thus, if individuals remain in the air-conditioned (indoor) areas during peak ambient O_3 concentration periods, their exposure to outdoor O_3 exposure is reduced. This situation does not happen in Lisbon. During the study period, less than 5% households in Lisbon had air conditioners; consequently, it is common practice to have the windows opened (with the blinds rolled down) to cool down the house in the afternoon when O_3 levels are typically highest. It is also common to see the older population sitting outdoors socializing in the afternoon in a shady area. Nevertheless, it is important that further investigations be conducted in order to better understand this vulnerability.

Table 5 shows the percentage increase in mortality (% RR) due to an increase of 10 μg/m^3 $PM_{2.5}$. Results show an important exposure-response risk of $PM_{2.5}$ on cardiovascular mortality in the ≥65 years group. Similar results were found in North Carolina (Holloman et al. 2004) and Madrid (Maté et al. 2010).

4 Conclusions

This study shows, for the first time, significant associations between ambient O_3 and $PM_{2.5}$ levels on mortality in Lisbon. We report that an increase in ambient O_3 of 10 μg/m^3 represents a small but significant increase in total mortality with the elderly being more vulnerable. Our results indicate an increase of 1.11% (95% C.I. (0.58, 1.64)) for all cause mortality ≥65 years and 0.96% (95% C.I. (0.56, 1.35)) for all-cause mortality in the total population. When analyzing by cause of death, our results showed a stronger association between O_3 exposure and cardiovascular mortality. We report relative risks from O_3 exposure that are almost double those reported in other cities. This clearly shows that the Lisbon population is indeed vulnerable to current ambient O_3 levels. Consequently, it is very urgent that efforts to reduce O_3 pre-courser emissions be taken seriously. It is also important to investigate how city-specific cultural and socio-economic factors contribute towards this vulnerability. Our finding that the risk of mortality by cardiovascular endpoints was higher than by all-cause mortality is expected based on previous epidemiology and toxicology studies. Due to data privacy constrains, in this study, we were not able to assess the effects on other causes (i.e., respiratory) of mortality; hence, additional vulnerable population groups could not be identified.

Our results show that the percentage increase in cardiovascular mortality ≥65 years due to a $PM_{2.5}$ increase of 10 μg/m^3 is 2.39% (95%C.I. (1.29, 3.50)). The $PM_{2.5}$ exposure-response models for the all age group, as well as all-cause mortality, were not statistically significant at the 5% significance level.

Furthermore, our models clearly show two distinct patterns of air pollution-related mortality. The exposure-response effect to $PM_{2.5}$ is statistically significant in the same day, while the exposure-response effect of O_3 is seen with a 2–3 day lag.

Taking into consideration that this is the first study in Lisbon assessing the impact of air pollution on daily mortality, and that climate change is very likely to affect local O_3 and PM concentrations, future work using longer time series, exploring different risks groups, seasonality, as well as hospital emergency room visits, is recommended to improve our understanding of the health impact of exposures to O_3 and $PM_{2.5}$ in Lisbon.

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