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The Effects of Particulate Matter Sources on Daily Mortality: A Case-Crossover Study of Barcelona, Spain

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Abbreviations:

Al = Aluminum
As = Arsenic
Ca = Calcium
Cd = Cadmium
Cl^- = Chloride
Cr = Chromium
Cu = Copper
EV = Explained variation
Fe = Iron
ICD-10 = International Classification of Diseases, 10^{th} Revision
IQR = Interquartile range
K = Potassium
Mg = Magnesium
Mn = Manganese
Na = Sodium
NH_4^+ = Ammonium
Ni = Nickel
NO_3^- = Nitrates
P = Phosphorus
Pb = Lead
PM2.5 = Particulate matter less than 2.5 microns in diameter
PM10 = Particulate matter less than 10 microns in diameter
OR = Odds ratio
Rb = Rubidium
S = Sulfur
Sb = Antimony
Sn = Tin
Sr = Strontium
Ti = Titanium
TC = Total carbon
V = Vanadium
Zn = Zinc
$\mu g/m^3$ = micrograms per cubic meter

ABSTRACT

Background: Dozens of studies link acute exposure to particulate matter (PM) air pollution to premature mortality and morbidity, but questions remain about which species and sources in the vast PM mixture are responsible for the observed health effects. While a few studies exist on the effects of species and sources in U.S. cities, European cities, which have a higher proportion of diesel engines and denser urban populations, have not been well characterized. Information on the effects of specific sources could aid in targeting pollution control and in articulating the biological mechanisms of PM.

Objectives: Our study examines the effects of various PM sources on daily mortality for 2003 to 2007 in Barcelona, a densely populated city in the northeast corner of Spain. Methods: Source apportionment for both PM2.5 and PM10 (PM less than 2.5 and 10 microns in diameter) using positive matrix factorization identified eight different factors. Case-crossover regression analysis was used to estimate the effects of each factor.

Results: Several sources of PM2.5, including vehicle exhaust, fuel oil combustion, secondary nitrate/organics, mineral, secondary sulfate/organics and road dust had statistically significant associations ($p < 0.05$) with all-cause and cardiovascular mortality. Also, in some cases relative risks for a respective interquartile range increase in concentration were higher for specific sources than for total PM2.5 mass.

Conclusions: These results along with those from our multi-source models suggest that traffic, sulfate and construction dust are important contributors to the adverse health effects linked to PM.

INTRODUCTION

Particulate matter (PM) air pollution is a heterogeneous mix of chemical elements and sources. While dozens of studies now link exposure to ambient PM with increases in both mortality and morbidity (Pope and Dockery 2006), considerable uncertainty remains about the relative toxicity of its different sources and constituents. The reports on future research for PM2.5 and PM10 (PM less than 2.5 and 10 microns in diameter, respectively) from both the U.S. National Academy of Science (NAS 2004), and the European Commission II Position Paper on PM (EC 2004) stressed the need for identifying the specific components and sources of the particle composition that are most harmful to the exposed population. Epidemiologic studies that examine the health impacts of specific sources of PM, therefore, are critical to addressing this uncertainty. Knowledge of the species and sources of concern would help prioritize research on the biological mechanism for PM effects and help target future pollution control strategies.

In beginning to address this issue, several epidemiological studies have examined the impact of specific components of PM on both mortality and morbidity (Bell et al. 2009; Franklin et al. 2008; Ostro et al. 2007; Peng et al. 2009; Sarnat et al. 2008; Zanobetti et al. 2009). These studies that examine the daily association between PM and adverse health over time, lend important insight into the relative toxicity of the myriad constituents of PM. However, many PM constituents are highly correlated, are unmeasured or, when measured, have many values below detection levels. In addition, the constituents of one PM source, such as vehicle exhaust, will vary greatly from those of other sources, such as residual oil combustion or road dust. Thus, analysis of sources exists as an important complement to the study of specific constituents.

To date, only a few studies have examined the effects of multiple sources of PM2.5 or PM10 (Laden et al. 2000; Mar et al. 2000; Thurston et al. 2005). Such efforts require source

apportionment techniques to determine the share of each element within a given source or factor. Several statistical techniques are available to apportion PM into different source classes (Hopke et al. 2006). As a consequence, the U.S. Environmental Protection Agency sponsored a set of studies to evaluate alternative apportionment methods produced by various investigators. Analysis of these methods indicated that they generally produced similar source categories (Thurston et al. 2005). Further, when the health impact of exposure to these sources was examined, relatively similar effect estimates were obtained (Mar et al. 2006). This suggests that it is reasonable to use these source estimates in epidemiological studies to determine their impact on various health outcomes.

While several studies have been conducted in the United States, there are few that examine sources in Europe where the PM composition and exposure patterns will be quite different. For example, most major European cities tend to be more densely populated than those in the United States, and have a much greater share of mobile sources utilizing diesel fuel. Thus, our study focuses on PM2.5 and PM10 sources in Barcelona, Spain, a city of 100 km^2 with approximately 1.6 million people (with 4.5 million in the greater metropolitan area) located in the northeast corner of Spain. Barcelona has among the highest population density in the world at approximately 16,000/km^2 -- more than three times that of the other major cities of Spain (Madrid, Valencia and Seville) and such U.S. cities as Chicago and Philadelphia (Población de España 2010; United Nations Cities Statistics 2010). Also the relatively scarce precipitation increases the accumulation and re-suspension of road and urban dust. In this study, we examined the associations between premature mortality and the various sources of both PM2.5 and PM10 in Barcelona.

Mortality and Covariate Data

Daily data on mortality for residents of the city of Barcelona who died in the city from 2003 through 2007 were obtained from the Barcelona mortality registry (based on the Catalan mortality registry and the census of Barcelona). We examined mortality from all causes (minus accidents and homicides) and cardiovascular disease (codes I00-I99 of the International Classification of Diseases, 10th Revision (ICD-10) (World Health Organization 1993). Data on daily temperature and humidity were obtained from the National Meteorological Institute which maintains a station at the airport, 8 km from the city center.

Exposure Estimates

We used source estimates developed from an analysis that has been published previously (Amato et al. 2009a). Basically, PM data were collected in Barcelona from 2003 to 2007 at an urban background monitoring station located on the roof (two stories) of the Institute of Environmental Assessment and Water Research (IDAEA-CSIC). About 150 meters away is a large traffic arterial (Diagonal Avenue) which often experiences over 50,000 vehicles/day. Twenty-four hour averages of PM2.5 and PM10 were collected using MCV high volume (30 m^3/h) samplers approximately every six days. PM data collected during clear African dust outbreaks were identified following the methodology described by Perez et al. (2008) and excluded from the analysis in order to differentiate it from the other two mineral sources (urban dust and road dust). After this exclusion plus additional exclusions due to possible contamination and errors in weighing, the frequency of PM species data was approximately every sixth day with differences in the number of days with PM2.5 versus PM10 data.

Particles were collected on quartz-fiber filters (15 cm diameter, QF20 Schleicher and Schuell) and analyzed following the procedures described by Querol et al. (2001). Concentrations of total carbon (TC) were determined by elemental analysis; Al, Ca, K, Mg,

Fe, Ti, Mn, P, S, Na and 46 trace elements by inductively coupled plasma atomic emissions spectrometry (ICP-AES) and by inductively coupled plasma mass spectrometry (ICP-MS); NO_3^- and Cl^- by ion chromatography; and NH_4^+ by specific electrode

Ultimately, only 26 chemical species were selected for the source apportionment study, based on the signal to noise criterion (Paatero and Hopke 2009) and percentage of data above detection limit. These species included: Al, As, Ca, Cd, Cl^-, Cr, Cu, Fe, K, Mg, Mn, Na, NH_4^+, Ni, NO_3^-, P, Pb, Rb, S, Sb, Sn, Sr, TC, Ti, V and Zn.

In addition to the periodic sampling of PM mass and species, PM10 and PM2.5 mass were also measured every day using optical counters (GRIMM, versions 1107, 1108) corrected by inter-comparison with MCV high volume samplers.

Estimates of source contribution were developed from receptor models based on the mass conservation principle:

$$x_{ij} = \sum_{k=1}^{p} g_{ik} f_{jk} \quad i=1,2,\ldots,m \quad j=1,2,\ldots,n \quad [1]$$

where x_{ij} is the i^{th} concentration of the species j, g_{ik} is the i^{th} contribution of the source k and f_{jk} is the concentration of the species j in source k. When both g_{ik} and f_{jk} are unknown, factor analysis techniques such as Principal Components Analysis (PCA) (Henry and Hidy 1979; Thurston and Spengler 1985) and Positive Matrix Factorization (PMF) (Paatero and Tapper 1994) are used for solving equation [1]. PMF is a weighted least squares method that can be solved using the Multilinear Engine (ME-2) developed by Paatero (1999). ME-2 is a flexible program that permits the incorporation of any *a priori* information such as chemical properties or linear constraints into the model as a target to be fit to some specified precision. Therefore, ME-2 is especially suitable for source apportionment studies where some knowledge (chemical ratios, profiles, mass conservation etc.) of involved sources is available and was used for our analysis. Additional details on the technique used have been previously

described (Amato et al. 2009a). Identification of factors was also aided by information on their seasonal patterns.

Besides the effects of specific sources, we also examined the effects of total mass concentrations of PM2.5 and PM10. Our PM mass analysis was performed on two different data sets: a limited data set that only included PM mass measurements for days when species data also were available, and a separate data set that included daily PM measurements during the study period (except for the Saharan dust days), including measurements taken on days when species data were not collected.

Study design and data analysis

We used a time-stratified case-crossover study design described by Levy et al. (2001). In this method, the exposure on the date of an event (case) is compared to several non-event control days (referent periods) occurring on the same month and year. Since all referent periods are selected within the same month as the mortality, seasonal or long-term effects are generally eliminated by design. Variables for temperature, humidity, day of the week and flu epidemics were also included in the regression model. Temperature and humidity were each modeled using an average of values on the same day of the case (or control) and those of the previous day (i.e., lag01). We also examined other forms of temperature including 2-, 3-, and 4- day moving averages, quadratic terms and smoothing splines. Day of week was modeled using 6 dichotomous variables and was necessary since we did not have enough data to match case and controls by day. A flu epidemic week was designated as a dichotomous variable for a week with incidence rates above baseline levels based on local information (Perez et al. 2008). Each source was then entered separately into the model.

Besides examining the effect of same-day mortality (lag0), we also considered the effects of exposures on one to three previous days (lag1 to lag3). However, since data on PM

species were not collected every day, a cumulative average could not be investigated. After the basic analysis, we conducted forward stepwise analysis to determine which sources were the best predictors and whether multiple sources were concurrently associated with mortality. We used an inclusion criterion for variable entry of $p < 0.10$. We also created an additional source labeled ¨traffic¨ that was the sum of several other sources (described below) and examined this variable in the single- and multi-source model. All analyses were conducted using conditional logistic regression in STATA 11 (Statacorp, College Station, TX, 2011). We calculated the excess risk of mortality, defined as (OR - 1) x 100% where OR is the odds ratio from the logistic regression, and 95% confidence intervals (CIs) for an interquartile range increase in each source.

RESULTS

Eight sources or factors of PM10 and PM2.5 were identified in the source apportionment model including: secondary sulfates/organics (power plants, ship emissions, long-range transport), road dust (brake/tire/road wear and re-entrained PM), minerals (urban and construction dust), fuel oil combustion (ship emissions and industrial combustion), industrial (process emissions), secondary nitrate/organics (mobile sources and other fuel combustion), vehicle exhaust and aged sea salt. Descriptive statistics on the sources, mass concentrations of PM10 and PM2.5, and mortality and are provided in Table 1. For sensitivity analysis, a "traffic¨ variable was created and set equal to the sum of concentrations of vehicle exhaust (primary PM), road dust and 70% of nitrate (secondary PM). The latter is the approximate share of secondary nitrate/organics due to mobile sources in Barcelona. For PM2.5, the mean concentration on days with species data was 26 $\mu g/m^3$, and the dominant sources were vehicle exhaust (30% of the total), secondary sulfate/organics (28%) and secondary nitrate/organics (19%). The average concentration of PM10 on days with species

data was 42 $\mu g/m^3$, with the dominant sources including minerals (23%), secondary sulfates/organics (18%), vehicle exhaust (18%) and road dust (17%). Mean concentrations of PM2.5 and PM10 were similar when based on all daily mass samples collected over the 5-year study period. There was an average of 39 and 12 deaths per day from all-cause and cardiovascular mortality, respectively, for both the species day and every day analyses.

Figure 1 provides details regarding the estimated source profiles indicating the concentration of each specific species within each estimated source. For example, for vehicle exhaust, the largest constituents were total carbon, sulfur and potassium, while the source identified as mineral consisted primarily of calcium, aluminum and iron (among the analyzed species). Figure 1 also provides information about the explained variation (EV). As described by Paatero (2000), the EV indicates the importance of each factor in explaining the variation of a given species. It measures the contribution of each source to the ambient air concentrations of each chemical species and can therefore be useful for qualitative identification of the sources. For example, a factor that explains a great proportion (i.e., high EV values) of Vanadium and Nickel would be identified as a fuel oil combustion emission source. Likewise, Figure 1 shows that the industrial factor explains most of the variation (high EV values) for Manganese, Lead and Zinc.

Table 2 summarizes correlations among the estimated sources. For both PM2.5 and PM10, correlations were fairly modest, generally with $0.1 < r < 0.4$. Among the highest correlations for PM2.5 were vehicle exhaust with road dust (0.39) and secondary sulfate (-0.40).

Figure 2 summarizes the regression results for all-cause mortality and sources in the fine fraction of PM. Since the model fit was best for a two-day lag, the figure provides results for this lag. A full set of results for all lags is provided in the Supplemental Material, Table 1. Based on single-source models, statistically significant associations ($p<0.05$) were

observed between mortality and road dust, mineral, fuel oil combustion and vehicle exhaust with a two-day lag. In general, we observed excess risks of around 2 to 4% as central estimates for a change in the respective IQRs of each of the eight original estimated sources. In contrast, for the composite traffic variable the excess risk was almost 6% for an IQR change. For PM10, significant associations were only observed for mineral, vehicle exhaust and traffic with excess risks that were fairly similar to those produced from the sources of PM2.5 (See Supplemental Material, Table 1). The source results were unchanged when we used other temperature metrics (i.e., moving averages, smoothing splines) in the regression models (data not shown).

For the stepwise regression of the eight PM2.5 sources, a 2-day lag was used for each source since this was the lag that best predicted mortality. Three PM2.5 sources met the stepwise regression inclusion criteria of $p < 0.10$: mineral, fuel oil combustion and secondary nitrate/organics (Figure 2b). The resultant excess risks were generally similar to those generated when the sources were entered separately into the model. When we performed a stepwise regression that included a factor that encompassed the full effects of traffic, two factors met the inclusion criterion: minerals and traffic with excess risks of 3 and 5%, for their respectively IQRs (Figure 2c). Since few PM10 sources were associated with mortality, a multi-source model was not examined for this pollutant.

Significant associations with all-cause mortality were not seen for PM2.5 and PM10 based on the limited (i.e., approximately every sixth day) data set that was restricted to the days when species data were collected, but significant associations were observed based on the full, daily data set of approximately 1,700 observations. Lags of 0, 1 and 2 days were all associated with mortality (Figure 2d and Supplemental Material Table 1). The excess risk for all-cause mortality using a one-day lag was 1.9% (95% CI = 0.8, 3.1) for an IQR increase in PM2.5 of 13.6 $\mu g/m^3$, which corresponds to an excess risk of 1.4% (95% CI = 0.6, 2.3) for

a 10 μg/m^3 increase in PM2.5. The excess risk of all-cause mortality for the IQR for PM10 of 20.6 μg/m^3 was 2.4% or 1.2% (95% CI = 0.6, 1.7) for a 10-μg/m^3 change (Supplemental Material, Table 1).

A fairly similar pattern of associations emerged when examining cardiovascular mortality, with a few differences (Figure 3 and Supplemental Material Table 2). Specifically, statistically significant associations with PM2.5 at lag 2 were observed for secondary sulfate/organics, but not for vehicle exhaust. As with all-cause mortality, significant associations were also observed for the sources road dust, mineral, fuel oil combustion, secondary nitrate/organics and traffic (Figure 3). For PM10, associations were again observed for mineral, vehicle exhaust and traffic (Supplemental Material, Table 2). As expected, the excess risks for cardiovascular mortality were generally higher than those observed for all-cause mortality: 5 to 7% for the associated IQRs of the eight original PM2.5 sources and 10% for the PM2.5 traffic factor (Figure 3a). In the multi-source model for PM2.5, the same factors that were included in the all-cause mortality model met the inclusion criterion for cardiovascular mortality: minerals, secondary nitrate/organics and fuel oil combustion (Figure 3b). When the "traffic" factor was added to the stepwise regression, it was included in the final model along with minerals and secondary sulfates (Figure 3c). Again, a multi-source model for PM10 was not examined since few sources were associated with cardiovascular mortality.

Regarding the effects of PM mass on cardiovascular mortality, in the limited species data set, associations were observed for lag1 PM2.5 and PM10. For the daily data set, associations with lags of 0, 1 and 2 days were observed for both PM2.5 and PM10. The excess risk for cardiovascular mortality using a one-day lag was 3.9% (95% CI = 1.9, 6.0) for an IQR increase in PM2.5 of 13.6 μg/m^3, which corresponds to an excess risk of 2.9% (95% CI = 1.4, 4.4) for a 10-μg/m^3 increase in PM2.5. The excess risk of cardiovascular mortality

for the IQR for PM10 of 20.6 µg/m^3 was 5.7% or 2.8% (95% CI = 1.7, 3.8) for a 10-µg/m^3 change (Supplemental Material, Table 2).

DISCUSSION

In our analysis of short-term exposure to the estimated sources of PM2.5 and PM10 we observed several important associations with both all-cause and cardiovascular mortality. Specifically, for all-cause mortality and PM2.5, there were significant associations with estimated sources identified as road dust, mineral, fuel oil combustion, vehicle exhaust and traffic. For cardiovascular mortality, associations were also observed for secondary sulfate/organics. In multi-source models, both traffic and mineral sources were significantly associated with all-cause mortality while traffic, minerals and sulfate were significantly associated with cardiovascular mortality.

For the limited data set where mass was collected concurrently on the days of speciation collection (n = 279), no association was observed between PM2.5 mass and all-cause mortality. This indicates the importance of the specific sources (and species) since, in contrast, several of them were associated with mortality. For the full five-year period of daily PM2.5 measurements (n = 1,656), which also included days when chemical speciation data were not collected, lags of 0, 1 and 2 days were associated with both all-cause and cardiovascular mortality. For this full sample of PM2.5 data, the estimated excess risk of mortality per 10 µg/m^3 (lag1) was 1.4% (95% CI = 0.6, 2.3), which is within the upper range of those reported in previous multi-city studies in the United States (Franklin et al. 2007; Ostro et al. 2006; Schwartz et al. 1996; Zanobetti and Schwartz 2009.

It should be noted that for the analysis using daily PM2.5, a one-day lag provided a slightly better fit, based on t-statistics, than either lag0 or lag2. With the less frequent PM2.5 data collected every 3 to 6 days, lag2 provided the best fit. This difference in results for lags

is likely due to chance since with the periodic data set, each lag corresponds to a different mortality day. That is, with PM collected on day t or PM(t), a lag0 corresponds to mortality on day t or M(t). A one-day lag relates PM(t) to M(t+1) while a two-day lag relates PM(t) to M(t+2). Thus, lags in the source data set correspond to non-overlapping mortality data sets.

In contrast to PM2.5, fewer sources of PM10 were significantly associated with mortality. However, significant associations with all-cause and cardiovascular mortality were detected for the sources identified as mineral and vehicle exhaust. For the full sample of days with PM10 data, associations were also observed between it and both all-cause and cardiovascular mortality.

In considering the eight single source categories of PM2.5, the highest effect estimates for all-cause mortality with excess risks of 4% per their respective interquartile range were observed for the sources vehicle exhaust, road dust and mineral. The vehicle exhaust particles represent primary emissions from vehicle engines, since secondary inorganic species where not included in this source. While dominated by total carbon, this factor also includes S, Fe, Cu and Sn (Amato et al. 2009a). The estimated mortality effect of short-term exposure to this source amounts to approximately 8% per 10 $\mu g/m^3$ (lag1), much larger that the estimated effects of traffic reported by Laden et al. (2000) (3.4%) for six eastern U.S. cities and 4.2% for Washington D.C. (Thurston et al. 2005). The larger estimated effect for Barcelona may be due to greater population density and subsequent exposure to traffic, differences in the mobile source mix especially the high proportion of diesel vehicles and/or the source apportionment method itself. When the traffic factor was created and included in the model, it had the smallest p-value and largest effect estimate for both all-cause and cardiovascular mortality. This provides additional evidence of the importance of mobile sources.

The second estimated factor, identified as road dust, has significant shares of TC, Fe, Cu and Sb and likely reflects both brake wear and re-entrained particles (Amato et al. 2009a,

2009b). The heavy metals may be serving simply as general markers for this factor or may have independent toxic properties of their own. The importance of this factor in Barcelona may be especially due to the generally infrequent rainfall resulting in significant re-entrainment of PM in the streets, concurrent with subsequent high exposures for the relatively dense urban population. Several toxicological studies of PM components have documented the role of these transition metals in enhancing oxidative stress and increasing the production of reactive oxygen species. (Maciejczyk et al. 2010; Sangani et al. 2010) Such effects are likely to play a significant mechanistic role in the pathology of air pollution (Kelly, 2003).

The third estimated major factor, identified as "mineral" contains large shares of Ca, Al and Fe. A previous analysis suggested several sources for this factor including urban dust, such as construction dust and re-entrained particles from unpaved areas including parking lots and gardens (Amato et al. 2009a). Thus, toxicity might result from both the transition metals described above and from other anthropogenic mineral materials. Days with African dust outbreaks were deleted from the data. However, previous studies have documented evidence of mortality effects in Barcelona from coarse Saharan dust (Perez et al. 2008) and it is likely that some of these particles will be deposited and re-suspended even a few days after dust outbreaks, thereby likely impacting the PM concentrations in the intermodal and fine particle size range.

Besides these three factors, fuel oil combustion and secondary nitrate/organics were also associated with all-cause mortality, and secondary sulfate was associated with cardiovascular mortality. Sulfate was also one of the three factors that met the inclusion criteria for the multi-pollutant model of cardiovascular mortality. Based on the analysis of Amato et al. (2009a), the fuel oil combustion factor, which accounts for most of the explained variation of V and Ni but is mostly made up of TC and sulfur, likely reflects industrial combustion sources, but mostly shipping emissions. Their analysis also attributed secondary

nitrate/organics mostly to road traffic and the secondary sulfate factor to photochemical oxidation of sulfur oxides emitted mostly regionally, but also from a few local utilities, shipping and long range transport. Although mean levels of SO_2 in Barcelona are relatively low (i.e., in the lowest range of EU cities at around 3 $\mu g/m^3$, which is similar to Stockholm's urban background (Reche et al., 2010), previous analysis noted a clear correlation with midday sulfate nucleation processes and nanoparticle pollution episodes due to the importance of photochemical nucleation (Reche et al. 2010). The present analysis suggests an excess risk of about 10% per 10-$\mu g/m^3$ (lag2) change in sulfate. Several previous studies have reported lower estimated effects on all-cause mortality from short-term exposure to sulfates per 10 $\mu g/m^3$ for six U.S. cities (Laden et al. 2000) (2.8%), Washington, DC (3.8%) (Thurston et al. 2005) and Boston (4.8%) (Maynard et al. 2007).

Among the sources of PM10, only mineral and vehicle exhaust were significantly associated with either all-cause or cardiovascular mortality. Of note, approximately 75% of the mineral source is in the coarse particle size range, between 2.5 and 10 microns in diameter. As discussed earlier, the mineral factor, dominated by Ca, Al and Fe, likely reflects urban mineral dust other than road dust. Several previous studies have documented an association between coarse particles and all-cause and cardiovascular mortality in locations such as Palm Springs, CA, multiple counties in California, Phoenix and Mexico City (Castillejos et al. 2000; Malig and Ostro 2009; Mar et al. 2000; Ostro et al. 2003). While PM10 from road dust was generally positively associated with mortality, the estimates were not statistically significant.

Taken together, our results suggest that several sources of fine particles are likely important contributors to adverse health outcomes in Barcelona. This includes particles emanating from mobile sources either directly (i.e. vehicle exhaust, secondary nitrate/organics) or indirectly through re-entrainment of road dust, shipping and stationary

source emissions (fuel oil combustion, secondary sulfate) and mineral dust. Some additional evidence provided by the results of the multi-source regression serves to narrow the list to two or three main sources in the final model: traffic (including both primary and secondary PM and road dust), sulfates and urban dust from construction and demolition. Previous studies have observed associations with multiple sources or tracers of sources. For example, Laden et al. (2000) found evidence of effects on mortality from both motor vehicle exhaust and coal combustion in predominantly east coast U.S: cities. Ostro et al. (2007) also observed associations between mortality both traffic and biomass for multiple cities in California. Finally, Zhou et al. (2011) found evidence of traffic effects for the warm season in Detroit and the cold season in Seattle along with, in the latter, effects from biomass combustion, residual oil and metals processing.

As always, there are some caveats to the interpretation of results. First, the identification of specific sources or factors may be dependent on the analytic methods used. However, results from other epidemiological studies suggest that the associations are consistent regardless of the methods employed to identify PM sources. For example, studies undertaken in Washington, DC, Phoenix and Atlantic all considered multiple source-apportionment methods for source identification. In these studies, the subsequent analysis of health effects associated with these factors generated fairly similar results (Sarnat et al. 2008; Thurston et al. 2005). In the present study we used a hybrid factor analysis – chemical mass balance source apportionment approach to accurately characterize and subsequently quantify road dust, in addition to other common urban sources.

Due to the relative sparseness of the species data, we fitted the same source profiles for both PM2.5 and PM10. Performing positive matrix factorization analysis for PM10 and PM2.5 separately for this data set resulted in less precise results (Amato et al., 2009a). Given the small number of species data points, therefore, PM data from different size

fractions were assembled into a two-dimensional array and analyzed together in order to significantly increase the number of observations. This combined data displayed the most realistic results for factors profiles. Thus, the variability of factors profiles among different PM sizes could not be investigated. The resulting source profiles and the explained variation of the species are therefore the same for both PM10 and PM2.5.

Another caveat is that the sources that were observed to be associated with mortality may be proxy markers of exposure for unmeasured elements or sources that are the underlying causes of the associations. Third, we relied on a single monitor for our estimates of exposure. In general, the resultant biases caused by misclassification of exposure should be towards the null. However, to the extent that the different sources have different spatial exposure patterns, there may be differential misclassification, which could lead to biased results. Finally, it is possible that the results were obtained purely by chance.

In summary, our study suggests the likelihood of significant health effects in Barcelona resulting from exposure to PM2.5 and more specifically from exposure to mobile sources (both exhaust and road dust emissions). There also is evidence that exposure to other sources of fine particles, including re-entrained PM, stationary sources and other sources of urban mineral dust (such as construction dust), contribute to adverse health as well. Thus, our results lend additional support to efforts to control all sources of fine PM.

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Table 1. Descriptive statistics for mortality and particulate matter (PM) mass and sources in Barcelona, 2003- 2007. (Data for PM mass and PM sources expressed in $\mu g/m^3$).

Variable	Days	Daily Mean	Standard deviation	IQR
Mortality				
Total Mortality (days with daily PM2.5 data)	1656	38.1	8.8	10
Cardio Mortality (days with daily PM2.5 data)	1656	12.3	4.3	6
Total Mortality (days with daily PM10 data)	1725	38.3	8.8	11
Cardio Mortality (days with daily PM10 data)	1725	12.3	4.3	6
Total Mortality (days with PM2.5 species data)	279	38.1	7.7	10
Cardio Mortality (days with PM2.5 species data)	279	12.3	4.0	5
Total Mortality (days with PM10 species data)	243	38.1	8.0	10
Cardio Mortality (days with PM10 species data)	243	12.1	4.1	5
PM2.5 mass, species data	279	26.1	11.1	13.0
PM2.5 mass, daily data	1656	26.2	11.7	13.6
PM10 mass, species data	243	41.6	15.7	20.8
PM10 mass, daily data	1725	39.6	16.2	20.6
PM2.5 Sources				
Secondary sulfate/organics	279	7.3	5.2	7.4
Road Dust	279	2.3	1.5	1.8
Mineral	279	3.2	3.1	3.1
Fuel Oil Combustion	279	1.7	1.5	1.6
Industrial	279	0.7	0.6	0.5
Secondary nitrate/organics	279	4.9	6.8	5.5
Vehicle Exhaust	279	7.7	4.2	5.2
Aged Sea Salt	279	0.9	0.9	0.8
Traffic	279	13.4	7.4	9.7
PM10 Sources				
Secondary sulfate/organics	243	7.3	5.5	7.5
Road Dust	243	7.0	4.8	5.9
Mineral	243	9.6	6.2	8.2
Fuel Oil Combustion	243	2.1	1.6	1.7
Industrial	243	0.7	0.7	0.7
Secondary nitrate/organics	243	5.1	5.9	6.5
Vehicle Exhaust	243	7.3	4.3	5.2
Aged Sea Salt	243	3.8	3.1	4.0
Traffic	243	17.8	9.1	11.0

Table 2. Correlation among the estimated sources of PM2.5 and PM10.

Particle Size and Source								
PM2.5	SS	RD	MI	FO	IN	SN	VE	AS
Secondary Sulfate/Organics	1							
Road Dust	-0.07	1						
Mineral	0.02	0.20	1					
Fuel Oil Combustion	0.30	0.14	0.07	1				
Industrial	0.21	0.20	0.08	0.04	1			
Secondary Nitrate/Organics	0.16	0.09	-0.11	0.16	0.37	1		
Vehicle Exhaust	-0.40	0.39	0.04	-0.13	0.12	0.16	1	
Aged Sea Salt	0.08	0.00	0.07	-0.07	-0.06	-0.22	-0.27	1
PM10								AS
Secondary Sulfate/Organics	1							
Road Dust	-0.11	1						
Mineral	0.06	0.13	1					
Fuel Oil Combustion	0.26	0.24	0.23	1				
Industrial	0.24	0.19	0.20	0.16	1			
Secondary Nitrate/Organics	0.28	0.36	0.06	0.33	0.33	1		
Vehicle Exhaust	-0.34	0.36	0.24	-0.15	0.00	-0.12	1	
Aged Sea Salt	-0.02	-0.36	-0.07	-0.12	-0.19	-0.27	-0.13	1

NOTE: SS = secondary sulfates/organics, RD = road dust, MI = mineral, FO = fuel oil combustion, IN = industry, SN = secondary nitrate/organics, VE= vehicle exhaust, AS= aged sea salt.

Figure Legends

Figure 1. Sources profiles (species concentrations within the source) and explained variation (EV) of each species. Error bars represent two times the standard deviation of bootstrap repetitions). EV value indicates the proportional distribution of each species among the different sources (i.e. sum of all EVs for one species among all sources and residuals must equal one).

Figure 2. All-cause mortality risks associated with sources of PM2.5 (Excess risk and 95% CI for IQR for lag2); a= single source models, b = multi-source models, c = multi-source models with traffic, d=PM mass models; PM2.5 = mass from periodic sampling; PM2.5all = mass from daily sampling.

Figure 3. Cardiovascular mortality risks associated with sources of PM2.5 (Excess risk and 95% CI for IQR for lag2); a= single source models, b = multi-source models, c = multi-source models with traffic, d=PM mass models; PM2.5 = mass from periodic sampling; PM2.5all = mass from daily sampling.

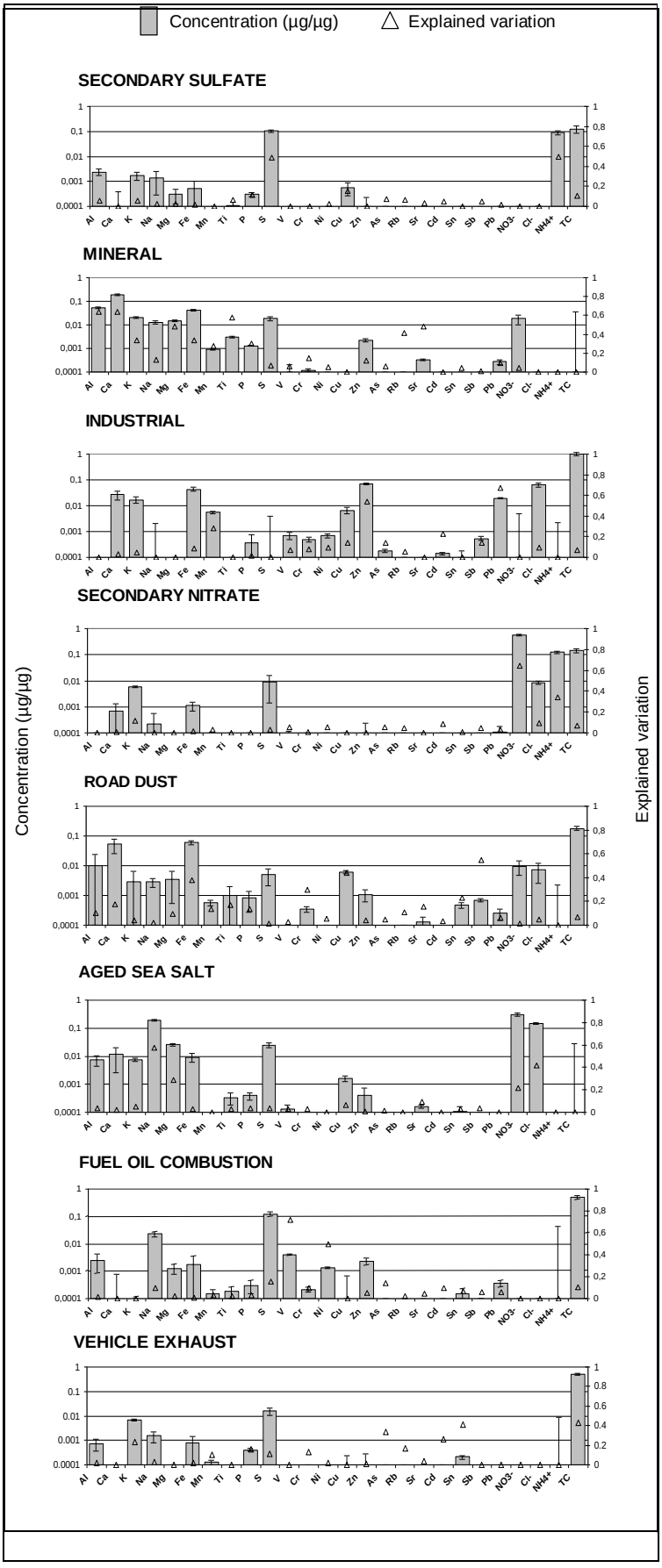
Concentration (µg/µg)
Explained variation
SECONDARY SULFATE
MINERAL
INDUSTRIAL
SECONDARY NITRATE
ROAD DUST
AGED SEA SALT
FUEL OIL COMBUSTION
VEHICLE EXHAUST
Al Ca K Na Mg Fe Mn Ti P S V Cr Ni Cu Zn As Rb Sr Cd Sn Sb Pb NO3- Cl- NH4+ TC

Figure 1.

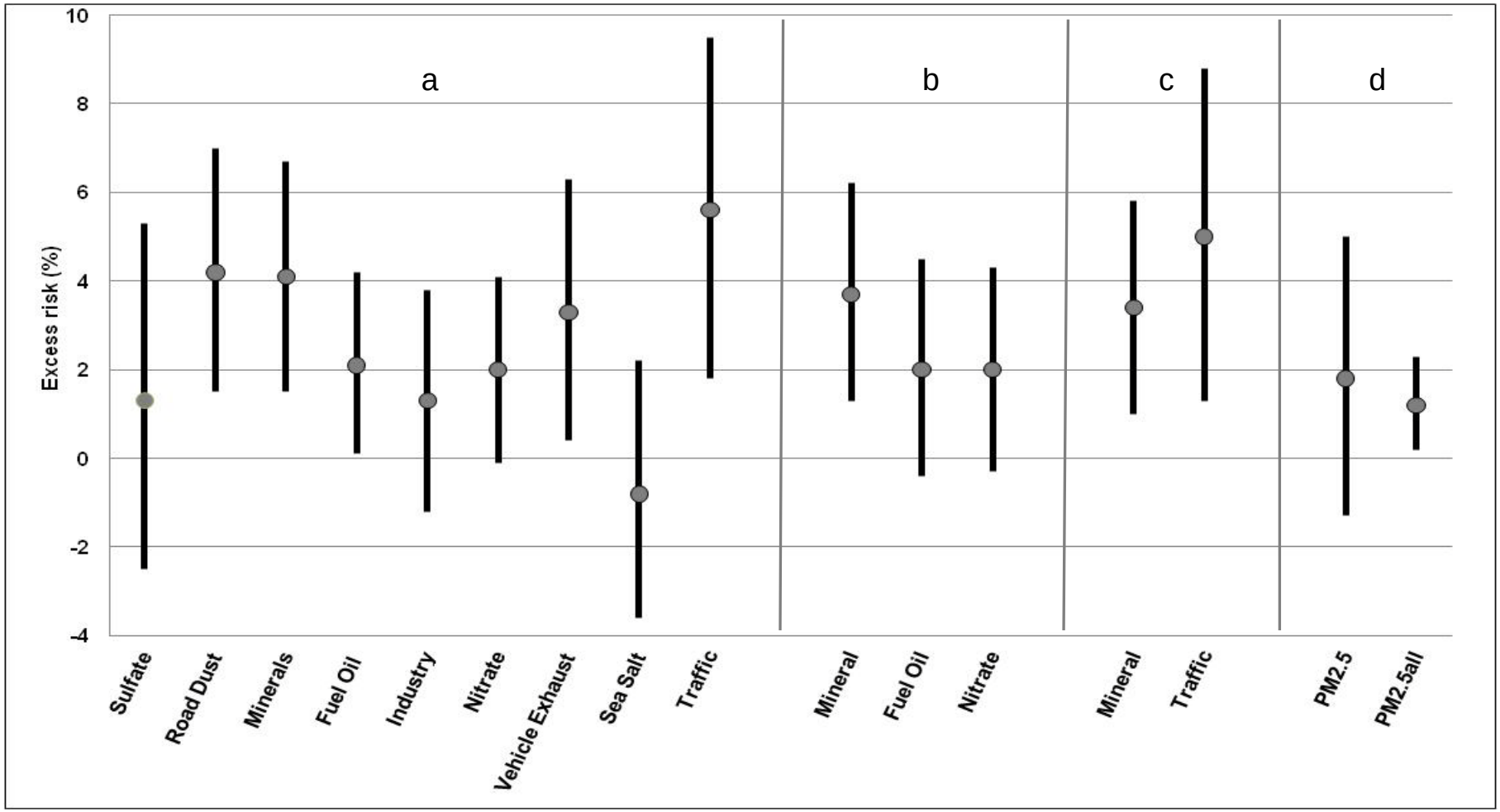
10
8
6
4
2
0
-2
-4
Excess risk (%)
a
b
c
d
Sulfate
Road Dust
Minerals
Fuel Oil
Industry
Nitrate
Vehicle Exhaust
Sea Salt
Traffic
Mineral
Fuel Oil
Nitrate
Mineral
Traffic
PM2.5
PM2.5all

Figure 2.

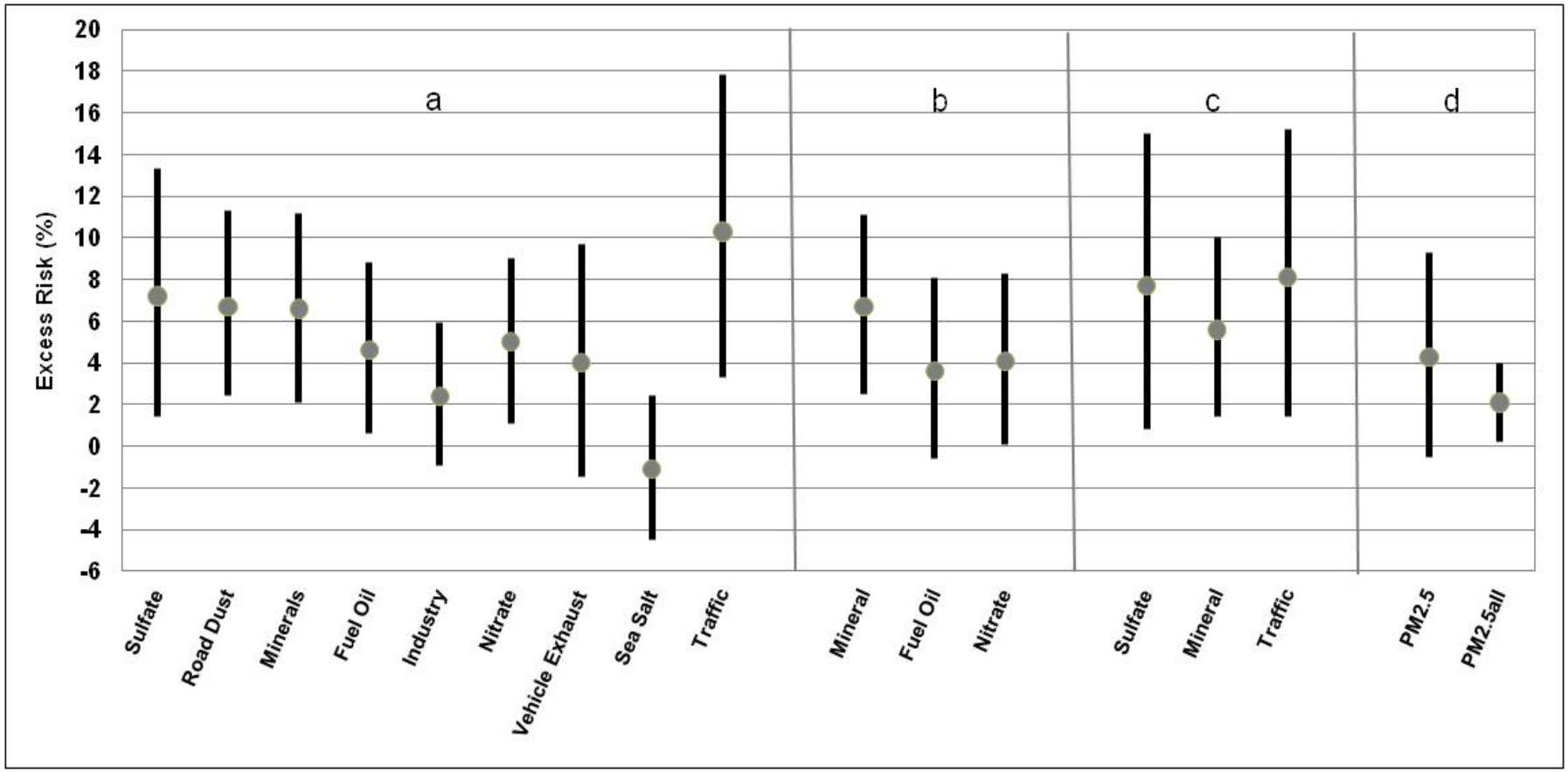
Excess Risk (%)
20
18
16
14
12
10
8
6
4
2
0
-2
-4
-6
a
b
c
d
Sulfate
Road Dust
Minerals
Fuel Oil
Industry
Nitrate
Vehicle Exhaust
Sea Salt
Traffic
Mineral
Fuel Oil
Nitrate
Sulfate
Mineral
Traffic
PM2.5
PM2.5all

Figure 3.