Differential health effects of short-term exposure to source-specific particles in London, U.K.

Evangelia Samoli[a], Richard W Atkinson[b], Antonis Analitis[a], Gary W Fuller[c], David Beddows[d], David C Green[c], Ian S Mudway[c], Roy M Harrison[d,e], H Ross Anderson[b,c], Frank J Kelly[c].

[a]Department of Hygiene, Epidemiology and Medical Statistics, Medical school, National and Kapodistrian University of Athens, 75 Mikras Asias Str, 115 27 Athens, Greece.

[b]Population Health Research Institute and MRC-PHE Centre for Environment and Health, St George's, University of London, Cranmer Terrace, London SW17 0RE, UK.

[c]MRC-PHE Centre for Environment and Health, King's College London, 150 Stamford Street, London SE1 9NH, UK.

[d]School of Geography, Earth & Environmental Sciences, Division of Environmental Health & Risk Management, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK.

[e]Department of Environmental Sciences / Center of Excellence in Environmental Studies, King Abdulaziz University, Abdullah Sulayman St, Jeddah, Saudi Arabia.

Address for correspondence: Evangelia Samoli, Department of Hygiene, Epidemiology and Medical Statistics, Medical school, National and Kapodistrian University of Athens, 75 Mikras Asias Str, 115 27 Athens, Greece. Telephone number: +30 210 746 2085. Fax number: +30 210 746 2205. Email address: esamoli@med.uoa.gr

Abstract

Background. There is ample evidence of adverse associations between short-term exposure to ambient particle mass concentrations and health but little is known about the relative contribution from various sources.

Methods. We used air particle composition and number networks in London between 2011-2012 to derive six source-related factors for PM_{10} and four factors for size distributions of ultrafine particles (NSD). We assessed the associations of these factors, at pre-specified lags, with daily total, cardiovascular (CVD) and respiratory mortality and hospitalizations using Poisson regression. Relative risks and 95% confidence intervals (CI) were expressed as percentage change per interquartile range increment in source-factor mass or number concentration. We evaluated the sensitivity of associations to adjustment for multiple other factors and by season.

Results. We found no evidence of associations between PM_{10} or NSD source-related factors and daily mortality, as the direction of the estimates were variable with 95% CI spanning 0%. Traffic-related PM_{10} and NSD displayed consistent associations with CVD admissions aged 15-64 years (1.01% (95%CI:0.03%, 2.00%) and 1.04% (95%CI: -0.62%, 2.72%) respectively) as did particles from background urban sources (0.36% for PM_{10} and 0.81% for NSD). Most sources were positively associated with pediatric (0-14 years) respiratory hospitalizations, with stronger evidence for fuel oil PM_{10} (3.43%, 95%CI: 1.26%, 5.65%). Our results did not suggest associations with cardiovascular admissions in 65+ or respiratory admissions in 15+ age groups. Effect estimates were generally robust to adjustment for other factors and by season.

Conclusions. Our findings are broadly consistent with the growing evidence of the toxicity of traffic and combustion particles, particularly in relation to respiratory morbidity in children and cardiovascular morbidity in younger adults.

Key words: Hospital Admissions; Mortality; Particles; Source Apportionment; Time series.

1. INTRODUCTION

A number of detailed reviews of the health effects of short-term exposure to particles have been published (Adar et al., 2014; Atkinson et al., 2014; WHO, 2013). These highlight an increasing focus on better identification of specific particle components and/or sources in order to target measures for the protection of public health. Nevertheless these are two quite different approaches: the former addresses single components of ambient particulates that may have a dominant source under certain climatic conditions (for example during warm periods urban elemental carbon is dominated by vehicle exhaust) while the latter addresses clusters of components as defined by source apportionment that may better represent a specific source. The latter approach has also been proposed as a way to address multi-pollutant exposure and health associations (Lall et al., 2011; Ostro et al., 2011; Sarnat et al., 2008; Zanobetti et al., 2014).

Source apportionment may be useful in epidemiological investigation of health effects but the application of varying methodologies and interpretation of identified clusters leave uncertainties making comparison between studies difficult. The US Environmental Protection Agency (EPA) sponsored a workshop that investigated source apportionment and health effects analyses by examining the associations between daily mortality and the investigators' estimated source-apportioned $PM_{2.5}$ for Washington, DC between 1988-1997 (Ito et al., 2006; Thurston et al., 2005). This analysis demonstrated that source-related effect estimates and their lagged association patterns were similar across investigators/methods with variation in the source apportionments increasing only by 15% the mortality regression confidence intervals. The panel concluded that their results provided supportive evidence that existing $PM_{2.5}$ source apportionment methods were sufficiently robust to derive reliable insights into the source components that contribute to $PM_{2.5}$ health effects (Thurston et al., 2005).

The Clearflo project (Bohnenstengel et al., 2014) characterized, in detail, the air pollution mixture in London between 2011-2012 and provided the opportunity to conduct daily time-series analyses focusing on specific sources, using data on the chemical composition of particles, estimation of the urban increment, as well as routine and study specific pollutant measurements. While we have previously used this extensive database to investigate the health effects of selected pollutants representative of the various components of traffic related air pollution (Atkinson et al., 2015; 2016; Samoli et al., 2016), in the present paper we investigate the effects of pollution from various sources. We used the UK national particle composition and numbers networks along with Clearflo data to apply positive matrix factorization analysis and derive source-related concentrations of PM_{10} and size distributions of ultrafine particles in order to assess their associations with daily total, cardiovascular and respiratory mortality, as well as hospitalizations in London, U.K.

2. METHODS

2.1 Health data

Daily counts of deaths from all non-accidental causes (ICD-10 Chapters A-R), cardiovascular (ICD-10 Chapter I) and respiratory causes (ICD-10 Chapter J) for people resident and dying in London, U.K. between January 2011 and December 2012 were constructed from death registrations obtained from the UK Office of National Statistics. For the same time period and using the same ICD-10 codes, daily counts of the numbers of emergency, first episode, hospital admissions for cardiovascular and respiratory diseases stratified by age (0-14, 15-64 and 65+ years) were derived from records of individual admissions obtained from the English Hospital

Episode Statistics system. Hospital admissions were stratified by age group as the occurrence of both respiratory and cardiovascular diseases vary with age.

2.2 Pollutants and Meteorological variables

Using data collected from the Clearflo project, supplemented by national and local network measurements made at the North Kensington (NK) urban background site in London, U.K., we assembled a database of over 100 metrics for 2011-2012, that included daily concentrations of particle mass (for particles with aerodynamic diameter less than 10 μm (PM_{10})), particle number and size distribution (NSD) as a measure of ultrafine particles (with diameter less than 0.6 μm), as well as particle chemical composition and a wood smoke tracer derived using the aethalometer model (Fuller et al., 2014). More specifically, particle number concentrations were obtained from the Condensation Particle Counter (CPC, TSI model 3022) with an upper size limit of around 3 μm, while number concentrations associated with the source apportionment were derived from the analysis of data from the Scanning Mobility Particle Sizer (SMPS, TSI model 3080 classifier and TSI 3075 CPC) with an upper size limit of around 0.6 μm. Although the instruments used to measure the particle number count and NSD are sensitive to particles well beyond the ultrafine size range, typically around 90% of particles by number in urban air are smaller than 100 nm diameter and consequently the particle number count and NSD are good measures of the abundance of ultrafine particles. The air pollution climate of the North Kensington site has been characterised in detail previously (Bigi and Harrison, 2010).

Mean daily temperature (^{o}C) and relative humidity data were also collected for the period 2011-12 from a meteorological station close to the North Kensington monitoring site.

2.3 Source apportionment

Positive Matrix Factorisation (PMF) is a multivariate data analysis method widely applied in atmospheric aerosol science. It is a least squares formulation of factor analysis first reported by Paatero and Tapper (1994). In common with other receptor modelling methods used for source apportionment of airborne particles, it is based upon a concept of mass conservation. Thus,

$$c_{ij} = \sum f_{i,k}\, g_{j,k} + e_{ij}$$

where c_{ij} is the concentration of component i in air sample j, $f_{i,j}$ the fractional contribution of component i to the particles emitted by source k, $g_{j,k}$ is the contribution of source k to the mass of particles in air sample j, and $e_{i,j}$ is the error associated with this estimate.

Thus the ambient aerosol, C, represented by a matrix of observations and constituents, is explained by the product of a source composition matrix F and a contribution matrix, G. The residuals are accounted for in matrix E, and G and F are obtained by a minimisation algorithm. The program is constrained not to give negative solutions. The components entered can be either chemical constituents, or size bins from measured particle size distributions.

Full details of the data collection, the PMF analysis and the results of PMF application have been reported by Beddows et al. (2015, see also Supplementary Table S1). The version of PMF which was used was PMF2, version 4.2 (Paatero and Tapper, 1994). The best fit to the PM_{10} data was given by six factor solution. Source profiles for PM_{10} are presented in Supplementary Figure S1. The largest contribution to PM_{10} mass was a factor attributed to secondary particulate matter which explained a high proportion of the variance in nitrate, sulphate and ammonium concentrations with also a contribution to organic carbon. Almost as substantial were non-exhaust and crustal particles which show a soil-like profile making major contributions to the concentrations of aluminium,

calcium, titanium and organic carbon. Of similar magnitude was a contribution from the urban background which appeared to be comprised largely of carbonaceous particles associated with organic and inorganic markers of wood smoke and traffic emissions. There was a smaller contribution from marine aerosol (sea spray) for which sodium, magnesium and chloride were the major constituents. Two other factors also made modest contributions to PM_{10} mass. One explained a large proportion of the variance in vanadium and nickel and showed the presence of sulphate, organic carbon and elemental carbon as major constituents. Such a profile typically derives from the combustion of heavy fuel oil and may be associated largely with shipping sources. The other contribution with large concentrations of elemental carbon and organic carbon and large contributions to copper, barium, antimony and zinc concentrations had a clear signature relating to exhaust and non-exhaust particles from road vehicles and was attributed to local road traffic.

The particle number size distribution data were best fitted by a four factor solution. Source profiles for particle number size distribution are presented in Supplementary Figure S2. Two of those factors made by far the greatest contribution to particle number. The first was attributed to road traffic. It showed a mode in the size distribution at around 30 nm and a diurnal variation typical of road traffic activity. The second was an urban background contribution peaking at around 70 nm in the number size distribution and showing a marked elevation at night. Its size distribution and diurnal and seasonal variation suggested a large contribution from wood smoke accompanied by aged traffic particles. Two sources made much smaller contributions, one, peaking at around 20 nm diameter with a strong temporal peak in the early afternoon was attributed to regional nucleation (new particle formation). The other constituent whose main size mode was at around

157 0.25 μm and peaked at nighttime was attributed to secondary particles which may be inferred to
158 have arisen from long-range transport processes.

159 *2.4 Statistical Analysis*

160 We investigated the associations between short-term exposure to the source factors and health
161 outcomes using Poisson regression models allowing for overdispersion. The model was of the
162 form:

163 $$\log E\,[Y_t] = \beta_0 + b * Pol_t + s(time_t) + \sum_i s(X_{it})$$

164 where $E[Y_t]$ is the expected value of the Poisson distributed variable Y_t indicating the daily
165 outcome count on day t with $Var(Y_t)=\varphi E[Y_t]$, φ being the over-dispersion parameter, $time_t$ is a
166 continuous variable indicating the time (day) of event, Pol_t is the concentration of the source-
167 related factor on day t, X_{it} is the value of confounder X_i on day t, and s denotes smoothing
168 functions. We used penalized regression splines (Wood, 2000) as smoothing functions s to
169 capture the association between time-varying covariates, calendar time and health outcome.
170 Degrees of freedom (df) for long term trends were based on the minimization of the absolute
171 value of the sum of the partial autocorrelations of the residuals from lags 1 to 30, imposing a
172 minimum of 3 df per year. We also included dummy variables for the day of the week and public
173 holidays. For the analysis of respiratory admissions among ages 0-14 and 15-64 years we
174 included an extra dummy variable denoting the month of August, as the decrease in the
175 respiratory admissions at this period could not be sufficiently captured by the smooth term of
176 seasonality. We controlled for mean daily temperature and relative humidity to address any
177 potential confounding effects of weather. For temperature control we applied a natural spline
178 with 3 df for same day's exposure (lag 0) to capture the effect of high temperatures on health,

while to capture the health effects of lower temperatures we used the corresponding function on the average of the six previous days exposure (lags 1-6), as these terms minimized the Akaike's Information Criterion (Stafoggia et al., 2013). For relative humidity adjustment, we included a linear term for the average of the same and the two previous days, sufficient to capture any residual weather confounding.

We decided *a-priori* to include previous day's exposure for CVD outcomes and total mortality (lag1) and exposure two days before the event for respiratory outcomes (lag2), based on prior indications of longer lags for the latter (Atkinson et al., 2010).

We investigated the linearity of the associations by including a penalised spline for the exposure metric. We tested the sensitivity of our findings by mutually adjusting in the models for all source-related mass concentrations for PM or numbers for NSD. We also applied two pollutant models by including both the source-related factor and the remaining particles mass/numbers (i.e. PM – source-related PM), for each identified source category in order to estimate the source-specific impact, after adjusting for the impact of the rest (Thurston et al., 2015).

We investigated the associations by season defined as warm (April-September) and cool (October-March) period to test the hypothesis of effect modification due to differential emissions and exposure misclassification between periods. For these analyses we controlled for seasonality and long-term trends using indicator variables per month per year of the study, while the rest of the confounding control was the same as in the annual model. Effect modification between strata was assessed by applying a t-statistic and a chi square test for heterogeneity.

We tested the hypothesis of possible residual confounding for our positive results using the method proposed by Flanders et al. (2011). Briefly the associations are estimated for pollutant

concentrations on the day after the health event (lag -1) given pollutant levels on the day of interest. If this estimate indicates effects and/or affects the estimate of the main exposure metric then the presence of residual confounding is considered, due to the non causal underlying association.

All models were fit in R v.3.0.3 (R development Core Team (2011), ISBN 3-900051-07-0, URL http://www.R-project.org) using the package *mgcv (v.1.7-28)*. Results in tables and plots are presented as percent change associated with an interquartile increase (IQR) in the source-related factor.

3. RESULTS

Table 1 presents descriptive measures for the health outcomes analyzed and the source-related mass for PM_{10} or numbers for NSD, as well as the meteorological data. Health data provided large mean numbers and variability. The mean daily PM_{10} during 2011-12 in London was 18.4 $\mu g/m^3$ and the largest contribution to its mass originated from non-exhaust, secondary and urban background sources. In Table 1, the total count refers to the particle number concentrations obtained from the CPC, while the four related source categories were derived from the PMF analysis of data from the SMPS. The total of the four sources falls well short of the total number count from the CPC because the CPC covers a wider range of particle sizes, and corrections applied for internal particle losses in the SMPS may be an underestimate. The mean NSD was 12,726.5 n/cm^3 and the largest part was attributed to the traffic source closely followed by urban background. Correlation between identified factors of each pollutant (PM_{10} or NSD) was small

(Table 2, in general less than 0.3) except for the correlation between the urban background and traffic source in PM_{10} (r=0.62) and with the secondary source in NSD (r=0.69).

Table 3 presents the percent change in mortality associated with an IQR increase in the respective exposure. We found no evidence of associations between PM_{10} total mass or NSD or source-specific metrics and health, as the direction of the estimates was variable with 95% confidence intervals that spanned 0%. Nevertheless, there were consistent positive associations with mortality outcomes and PM_{10} originating from marine or fuel oil sources, while all but the secondary-related PM_{10} presented positive effect estimates with respiratory mortality. Negative statistically significant effects were estimated between secondary –related PM_{10} and total / CVD mortality or NSD (total number concentration and secondary-related one) and CVD mortality.

Table 4 presents corresponding model results for hospital admissions. Both PM_{10} and NSD effect estimates were positive only for adult (15-64 years) cardiovascular hospitalizations (0.17% increase, 95% confidence interval (CI):-0.86%, 1.21% for an IQR increase in PM_{10} and 0.81%, 95%CI: -0.78%, 2.42% in NSD) and pediatric respiratory (0-14 years) hospitalizations (0.69%, 95%CI: -0.85%, 2.25% increase associated with PM_{10} and 1.86%, 95%CI: -0.28%, 4.05% with NSD). Our results do not support associations with cardiovascular admissions among those aged 65+years or respiratory admissions among those over 15 years old. Regarding adult cardiovascular admissions traffic-related PM_{10} and NSD displayed the higher effect estimates (1.01%, 95%CI: 0.03%, 2.00% and 1.04%, 95%CI: -0.62%, 2.72% respectively) as well as particles associated with background urban sources (0.36% for PM_{10} and 0.81% for NSD). Non-exhaust-related PM_{10} also displayed a positive increase in adult CVD hospitalizations. Most sources were positively associated with pediatric respiratory hospitalizations. In particular fuel oil-related PM_{10} displayed the highest and statistically significant effect estimate (3.43%, 95%CI:

245 1.26%, 5.65%), while nucleation sources drove the association with NSD (0.97%, 95%CI: -
246 1.31%, 3.30%). While the CIs between different age strata greatly overlap indicating absence of
247 heterogeneity, the effect of fuel oil PM_{10} on pediatric respiratory hospitalizations was
248 significantly different to the corresponding estimates for the other age groups as was also the
249 traffic related PM for both pediatric respiratory hospitalizations and CVD for those 15-64 years.
250 Finally, as negative findings make also positive effect estimates dubious, we tested the
251 hypothesis of possible residual confounding for our positive results using the method proposed
252 by Flanders et al. (2011). For all estimates that were found to be positive and statistically
253 significant and there was no indication of residual confounding, as they were robust to
254 adjustment of future pollution levels.

255 The investigation of the concentration response associations supported linearity with indications
256 of deviations mainly at higher levels for the non-exhaust-related PM with CVD and respiratory
257 mortality or respiratory admissions for those 15-64 years old, and for the associations between
258 urban background NSD and CVD and respiratory mortality. Nevertheless when we tested these
259 associations excluding the upper fifth percentile of the exposure distribution our conclusions
260 were stable in direction and significance.

261 Figure 1 and Supplementary Tables S2 and S3 present results from models including two or all
262 sources. Effect estimates were generally robust to co-source adjustment, although mutual
263 adjustment for all sources generally exerted greater influence on the estimates compared with
264 estimates from two sources models. Effect estimates for the association of mortality outcomes
265 with fuel oil-related PM_{10} (but not with marine-related) remained positive after control for other
266 sources, with non-exhaust and traffic-related PM_{10} also remaining positively associated with
267 respiratory mortality. Traffic-related PM_{10} effect estimate remained robust for adult CVD (1.24%

increase from the two sources model, 95%CI: 0.11%, 2.39%) and pediatric respiratory (0.84%corresponding increase, 95%CI: -0.68%, 2.38%) hospitalizations. Similarly fuel oil-related PM_{10} retained the strong association with pediatric respiratory admissions (3.53% increase, 95%CI: 1.34%, 5.76%). Effect estimates of background urban NSD with either adult CVD or pediatric hospitalizations remained robust as did the estimates between nucleation NSD and pediatric hospital admissions.

Figure 2 and Supplementary Tables S4 and S5 present results from stratified analysis by the warm and cool periods of the year. Most effect estimates were not significantly different between seasons. Fuel oil and traffic-related PM_{10} effects on mortality outcomes differed by season, as did nucleation NSD with adult CVD and pediatric respiratory hospitalization. Fuel oil PM_{10} displayed higher effect estimates with total mortality in the cool period, 2.87% increase (95%CI: 1.01%, 4.76%) vs 0.91% increase (95%CI:-1.02%, 2.88%) in the warmer months and traffic-related PM_{10} with CVD mortality in the warm, 1.07% (95%CI: -0.71%, 2.88%) vs -1.94% (95%CI: -4.12%, 0.29%) in the cool period. Most notably all source specific particles (except urban background and fuel oil PM_{10} and traffic-related NSD) displayed statistically significantly higher effects on elderly respiratory hospitalizations (65+ years) during the warm period of the year, except for marine-related PM_{10} that displayed significantly greater effects during the cold period (2.04% in the cool period vs -0.36% in the warm).

4. DISCUSSION

Associations between daily health metrics and six source-realed factors for PM_{10} (reflecting urban background, marine, secondary, non-exhaust traffic/crustal, fuel oil and traffic sources) and four for NSD (secondary, urban background, traffic and nucleation) were investigated for

London,U.K. during 2011-12. This study has used results from the receptor modelling (source apportionment) of both particle mass and particle number. The mass of particles in urban air is typically dominated by fine particles in the accumulation mode (0.1-1 μm diameter) and in the coarse particle mode (2.5-10 μm diameter). In contrast, the particle number is dominated by very small particles which are predominantly less than 100 nm diameter and hence ultrafine, but contribute little to mass. The two source apportionment studies are therefore complementary in that one apportions mass, reflecting particles of greater than 100 nm diameter, while the other apportions number which is dominated by the ultrafine particles of less than 100 nm diameter. Specifically, the analysis of the PM_{10} chemical composition data is able to distinguish components contributing largely to particle mass, whereas the number particle size distribution data set – although limited to detecting sources of particles below the diameter upper limit of the SMPS (604 nm) – is more effective for identifying components making an appreciable contribution to particle number. Consequently, even though particles in the mass-based and number-based studies may be attributed to the same source, they represent different populations of particles which might have different effects upon.We found weak evidence for associations between mortality and short-term exposure to fuel oil PM_{10}, while the traffic-related part displayed positive asssociations with respiratory mortality. No associations emerged for source-specific NSD and mortality. PM_{10} effect estimates on adult cardiovascular hospitalizations were driven by the traffic factor, while NSD positive associations were driven by the urban background one. Pediatric respiratory hospitalizations displayed the greatest and most consistent positive associations with particles derived from most sources, with the strongest findings estimated for fuel oil related PM_{10} and nucleation NSD.

313 Our positive results for the traffic related source are consistent with our previous reports using
314 measurements of specific traffic-related elemental components of PM_{10} for the same time period
315 in London. Atkinson et al. (2016) reported robust associations between short term exposure to
316 elemental carbon, an indicator of diesel exhaust, and respiratory mortality, while Samoli et al.
317 (2016) reported associations with adult cardiovascular and pediatric respiratory hospitalizations
318 for carbon in PM as a diesel exhaust marker and carbon monoxide, as an indicator of petrol
319 vehicle exhaust. Although previously there were indications that aluminum as an indicator of
320 dust re-suspension and non-exhaust traffic was associated with adult hospitalizations, this was
321 not replicated in the present analysis that incorporated aluminum in the non exhaust factor along
322 with other related elements. Nevertheless, previous studies in London during 2000-2005 have
323 reported associations between particle number concentration and cardiovascular mortality and
324 admissions (Atkinson et al., 2010) as well as secondary particles and respiratory mortality (Pirani
325 et al., 2015) that we did not find in our study. These discrepancies may be attributed to the
326 different health and exposure metrics used (for example Atkinson et al. (2010) analysed number
327 concentration and all-ages CVD admissions), different statistical approaches (Bayesian approach
328 in Pirani et al. (2015)) and the longer period (6 and 4 years in the previous analyses over 2 in our
329 case). Changes in the urban pollution mixture and concentrations over the period 2000 to 2012
330 can also not be ruled out, but these possible temporal issues cannot be assessed under the settings
331 of this study. One notable change which occurred between the periods of data used by Atkinson
332 et al. (2010), the years 2000-2005, and the current study, 2011-2012, is a reduction in the sulfur
333 content of motor fuels which caused a major reduction in the concentration, size distribution and
334 chemical composition of ultrafine particles (<100 nm diameter) (Jones et al., 2012).

335 The main strengths of our study are the range, the quality and the completeness of the pollution
336 metrics assembled from routine and augmented monitoring at a central urban background site
337 and the use of the large London population that provides variability in the health outcomes for
338 time-series analyses. Limitations of the present study include the relatively small sample size
339 (two years) and the exposure misclassification associated with the time–series design, induced
340 due to the use of a single fixed background monitoring site to estimate the population's exposure.
341 This may have a greater impact in the identification of source–related associations as different
342 source-related concentrations' will be affected in different ways by the misclassification, as
343 larger measurement error is expected for more spatially heterogeneous factors (e.g. traffic) than
344 homogeneous ones (e.g. secondary) (Sarnat et al., 2010). Whilst the urban background site
345 employed in this study has previously been shown to be representative of the city-wide
346 background, and the sensitivity analysis supported our positive results, residual confounding
347 cannot be completely ruled out, as also partly supported by the modification of the magnitude of
348 the effect estimates in the multi source models. Although associations that display consistent
349 patterns may suggest causation, a longer time-series with more monitoring sites is needed to help
350 confirm or reject the null findings of our study.

351 Finally, as source contributions are estimated and not actually measured, their estimation is
352 associated with some increased uncertainty. Following the report of the EPA workshop on
353 similar epidemiological findings using different source apportionment methods (Thurston et
354 al.,2005), Kioumourtzoglou et al. (2014) and Glass et al. (2015) showed that ignoring this
355 uncertainty may lead to significant underestimation of the epidemiological inferences, regardless
356 of the source apportionment method, and to contradicting findings between methods.

The air pollution climate and source apportionment for London is expected to be broadly similar to that of other European locations at a similar latitude. The absence of source apportionment studies using the same measurement variables and methodology makes it difficult to give exact comparisons with other cities. There are likely to be significant differences between London and cities in the extreme north of Europe due to winter sanding of road surfaces in the latter, and with southern Europe where atmospheric new particle formation through nucleation and the presence of Saharan dusts are more prevalent.

Few studies have looked into source-specific particles and health associations and direct comparison to the present study is limited due to the location specific factor identification, as well as the investigation of different outcomes and exposure periods. Laden et al. (2000) and Zanobetti et al. (2014) used k-means clustering to define clusters of similar air pollution mixture in order to address different PM toxicity and investigated the effect of short-term exposure to source-specific $PM_{2.5}$ on total mortality in U.S cities and reported associations with traffic-related particles as well as coal combustion (Laden et al., 2000) and fuel oil combustion sources (Zanobetti et al., 2014). These results, although using a different approach to identify harmful sources, are in broad agreement with the effect of fuel oil-related PM_{10} on total mortality in our study. Similarly, Ljungman et al. (2016) reported that $PM_{2.5}$ exposure from air pollution mixtures with large contributions of local ultrafine particles from traffic, heating oil, and wood combustion was associated with higher baseline pulse amplitude, but not hyperemic response in the Framingham Heart Study. Using similar source apportionment methodology Ostro et al. (2011) reported that $PM_{2.5}$ from several sources (vehicle exhaust, fuel oil combustion, secondary nitrate/organics, minerals, secondary sulfate/organics, and road dust) displayed statistically significant associations with all-cause and cardiovascular mortality in Barcelona, Spain; Sarnat et

al. (2008) reported significant, positive associations between same-day $PM_{2.5}$ attributed to mobile sources and biomass combustion in an Atlanta site and CVD-related emergency department visits, while steel industry and traffic related $PM_{2.5}$ was associated with respiratory and cardiovascular admissions respectively in New York (Lall et al., 2001). Pun et al. investigated associations between source-related PM_{10} and emergency hospitalizations either due to respiratory causes (2015) or to ischemic heart disease (IHD, 2014) in Hong Kong. Vehicle-exhaust was associated with both outcomes (2.01% and 1.87% increase correspondingly for an IQR increase in lags 2-5 and 0-1), while secondary sulphate was associated with respiratory hospital admissions (1.59% increase) and secondary nitrate (2.28%) or salt-related (1.19%) PM_{10} with IHD ones. Panel studies on susceptible population groups support the evidence of traffic-related combustion particle effects on respiratory function in adult asthmatics (Penttinen et al., 2006) or exercise-induced ischemia in patients with stable coronary heart disease (Lanki et al., 2006) and markers of systemic inflammation in IHD patients (Siponen et al., 2015). Li et al. (2016) reported that NSD of secondary origin were mostly responsible for the decrease in the respiratory function among 509 children with asthma or allergies in urban Taipei, Taiwan, which is also in accordance with our finding of a secondary NSD effect on pediatric respiratory hospitalizations, although in London background and nucleation NSD displayed stronger associations. Longitudinal studies that have investigated long-term exposure to source-related $PM_{2.5}$ and cardiovascular outcomes (Henning et al. 2014; Thurston et al., 2015) also reported traffic specific PM effects as well as with fossil fuel combustion signatures (Thurston et al., 2015).

None of the previous studies have looked into source-related effects in different periods of the year. Different source-related particles displayed slightly different patterns, although most effects

were higher during the warm period, except for most associations with marine-related particles. Such heterogeneity is partly explained by different emission patterns from the identified sources, meteorological conditions and differential exposure misclassification. The distinct pattern of secondary, non-exhaust and traffic PM_{10}, as well as urban background and secondary NSD, effects on elderly respiratory hospitalizations during the warm period may be attributed to better exposure characterization of the population that is most likely to be exposed outdoors when climatic conditions are better. Our results are in agreement with previously reported higher effects during the warm period of the year from the same data (Atkinson et al., 2016; Samoli et al., 2016).

5. CONCLUSIONS

In conclusion, our results suggest that traffic may be the dominant source for both PM_{10} and NSD driving the associations with adult CVD hospitalizations, while pediatric respiratory hospitalizations may also be driven by fuel oil PM_{10} and nucleation NSD. Our findings add to the growing evidence of the toxicity of traffic and combustion particles that call for implementation of regulation measures that would improve urban air quality.

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548

549 **Table 1.** Descriptive statistics for mortality and hospital admissions, concentrations of particles,
550 source-specific estimated concentrations and meteorological variables in London, U.K. for 2011-
551 12.

	Number of days	10^{th} percentile	Median	IQR (75^{th} -25^{th} percentile)	90^{th} percentile
Mortality (n/day)[a]					
Total	722	99	117	21	139
Cardiovascular	722	27	35	9	45
Respiratory	722	11	17	8	25
Hospital Admissions (n/day)					
Cardiovascular					
15-64 years	731	39	57	25	71
65+ years	731	76	104	37	124
Respiratory					
0-14 years	731	22	45	23	72
15-64 years	731	48	63	16	81
65+ years	731	77	91	28	125
PM_{10} ($\mu g/m^3$)					
Total concentration	729	9.0	15.0	10	32.5
Urban Background	730	0.9	3.0	3.8	9.6
Marine	730	0.4	2.1	2.5	5.6
Secondary	730	0.8	3.0	3.0	9.4
Fuel Oil	730	0.3	0.9	0.7	1.9
Non-Exhaust Traffic	730	0.9	3.2	3.9	9.5
Traffic	730	0.1	0.3	0.3	0.7
NSD (number/cm^3)					
Total number per cm^3	636	7,958.0	12,123.5	5,180.0	17,901.0

Urban Background	590	818.4	1893.2	1806.2	4442.2
Nucleation	590	43.0	279.8	519.9	991.8
Secondary	590	50.1	104.8	254.1	622.8
Traffic	590	1320.6	2355.0	1441.1	3950.4
Meteorology					
Mean Temperature (ºC)	731	5.1	11.7	7.5	18.1
Relative humidity (%)	731	61.6	78.0	14.6	88.5

552 IQR: Interquartile range; NSD: Number Size Distribution.

553 [a]01/01/20-22/12/2012

554

Table 2. Pearson correlation coefficients between source-specific particles in London for 2001-2012.

		PM_{10} Source-related						**NSD Source-related**		
		Back ground	**Marine**	**Secondary**	**Oil**	**Non-Exhaust**	**Traffic**	**Back ground**	**Nucleation**	**Secondary**
PM_{10}	**Background**	1								
	Marine	-0.25	1							
	Secondary	0.31	-0.21	1						
	Fuel Oil	-0.10	-0.07	-0.16	1					
	Non-Exhaust Traffic	0.15	-0.23	0.21	-0.15	1				
	Traffic	0.62	-0.28	0.20	-0.10	0.48	1			
NSD	**Background**	0.77	-0.35	0.30	0.02	0.41	0.72	1		
	Nucleation	-0.07	-0.09	-0.14	0.28	-0.14	-0.08	-0.08	1	
	Secondary	0.60	-0.36	0.64	-0.14	0.47	0.54	0.69	-0.13	1
	Traffic	0.41	-0.13	-0.01	-0.07	0.10	0.47	0.35	0.25	0.10

NSD: Number Size Distribution.

Table 3. Percent change in all-cause (lag1), cardiovascular (lag1) and respiratory (lag2) mortality associated with interquartile range increases in source-related PM_{10} and NSD in London, 2011-12. Results from single source models.

	All-Cause % (95%CI)	Cardiovascular % (95%CI)	Respiratory % (95%CI)
PM_{10} ($\mu g/m^3$)			
Total concentration	-0.48 (-1.22, 0.25)	-0.87 (-2.13, 0.40)	-0.81 (-2.57, 0.97)
Urban Background	-0.03 (-0.76, 0.70)	-0.96 (-2.24, 0.34)	0.31 (-1.46, 2.11)
Marine	0.59 (-0.30, 1.49)	1.11 (-0.49, 2.73)	0.39 (-1.83, 2.67)
Secondary	**-0.95 (-1.47, -0.43)**	**-1.03 (-1.92, -0.12)**	-1.20 (-2.49. 0.10)
Fuel Oil	0.86 (-0.20, 1.93)	0.67 (-1.18, 2.56)	1.58 (-1.06, 4.29)
Non-Exhaust Traffic	-0.23 (-1.17, 0.71)	-0.63 (-2.21, 0.97)	0.63 (-1.61, 2.91)
Traffic	-0.37 (-1.08, 0.34)	-1.03 (-2.27, 0.22)	1.06 (-0.65, 2.79)
NSD (n/cm^3)			
Total number / cm^3	-0.06 (-1.16, 1.06)	**-2.04 (-3.94, -0.10)**	-1.86 (-4.50, 0.86)
Urban Background	-0.55 (-1.52, 0.43)	-1.59 (-3.29, 0.14)	1.43 (-0.97, 3.89)
Nucleation	0.21 (-0.90, 1.33)	-0.76 (-2.75, 1.26)	-0.18 (-2.93, 2.65)
Secondary	-0.84 (-1.76, 0.10)	**-1.86 (-3.45, -0.24)**	-1.19 (-3.41, 1.08)
Traffic	0.21 (-0.93, 1.37)	-0.52 (-2.57, 1.57)	-1.83 (-4.59, 1.01)

NSD: Number Size Distribution, CI: Confidence Interval. In bold: statistically significant results at $p<0.05$.

Table 4. Percent change in cardiovascular (lag1) and respiratory (lag2) hospital admissions associated with interquartile range increases in source-related PM_{10} and NSD in London, 2011-12. Results from single source models.

	Cardiovascular % (95%CI)		Respiratory % (95%CI)		
	15-64 yrs.	65 yrs.+	0-14 yrs.	15-64 yrs.	65 yrs.+
PM_{10} ($\mu g/m^3$)					
Total concentration	0.17 (-0.86, 1.21)	-0.50 (-1.27, 0.28)	0.69 (-0.85, 2.25)	-0.67 (-1.69, 0.37)	**-1.14 (-2.10, -0.16)**
Urban Background	0.36 (-0.67, 1.40)	-0.35 (-1.13, 0.43)	0.55 (-0.83, 1.94)	-0.23 (-1.25, 0.80)	-0.95 (-1.88, 0.00)
Marine	-0.50 (-1.72, 0.72)	**1.28 (0.36, 2.21)**	-0.43 (-2.15, 1.33)	0.51 (-0.72, 1.76)	0.57 (-0.56, 1.71)
Secondary	-0.14 (-0.86, 0.58)	-0.68 (-1.23, -0.13)	-0.58 (-1.64, 0.49)	**-0.87 (-1.60, -0.13)**	-0.19 (-0.89, 0.52)
Fuel Oil	-0.12 (-1.56, 1.35)	-0.14 (-1.23, 0.97)	**3.43 (1.26, 5.65)**	-1.08 (-2.52, 0.38)	-0.57 (-1.93, 0.82)
Non-Exhaust Traffic	0.18 (-1.10, 1.48)	-0.77 (-1.73, 0.19)	0.42 (-1.57, 2.44)	-0.14 (-1.42, 1.15)	-0.01 (-1.24, 1.23)
Traffic	**1.01 (0.03, 2.00)**	-0.29 (-1.03, 0.46)	0.92 (-0.40, 2.26)	-0.70 (-1.67, 0.29)	**-1.35 (-2.25, -0.45)**

NSD (n/cm^3)					
Total number /cm^3	0.81 (-0.78, 2.42)	-0.07 (-1.27, 1.15)	1.86 (-0.28, 4.05)	-1.14 (-2.66, 0.41)	-1.09 (-2.42, 0.27)
Urban Background	0.81 (-0.61, 2.26)	-0.25 (-1.34, 0.85)	0.51 (-1.39, 2.45)	-0.08 (-1.41, 1.27)	0.29 (-0.92, 1.52)
Nucleation	-0.82 (-2.35, 0.74)	-0.90 (-2.08, 0.30)	0.97 (-1.31, 3.30)	-0.48 (-1.99, 1.06)	-0.73 (-2.07, 0.63)
Secondary	0.19 (-1.14, 1.53)	-0.85 (-1.86, 0.17)	0.18 (-1.70, 2.11)	-0.57 (-1.81, 0.69)	-0.47 (-1.62, 0.69)
Traffic	1.04 (-0.62, 2.72)	-0.41 (-1.67, 0.87)	-0.20 (-2.38, 2.03)	-0.72 (-2.28, 0.87)	-1.21 (-2.57, 0.18)

NSD: Number Size Distribution, CI: Confidence Interval. In bold: statistically significant results at $p<0.05$.

Figure 1. Percent increase (and 95% confidence intervals) in respiratory admissions 0-14 years associated with an interquartile increase in the source-related PM_{10} concentrations ($\mu g/m^3$) and NSD (n/cm^3). Results from models including each source individually (triangles), adjusted for all other sources (squares) and after controlling for the total concentration in PM_{10} or numbers in NSD minus the specific source (circles).

Figure 2. Percent increase (and 95% confidence intervals) in cardiovascular (top panel) and respiratory mortality (bottom panel) associated with an interquartile increase in the source-related PM_{10} concentrations ($\mu g/m^3$) and NSD (n/cm^3) by warm (triangles) and cool (squares) period of the year.

SUPPLEMENTAL MATERIAL

Differential health effects of short-term exposure to source-specific particles in London, U.K.

Evangelia Samoli, Richard W. Atkinson, Antonis Analitis, Gary W. Fuller, David Beddows, David C. Green, Ian S. Mudway, Roy M. Harrison, H. Ross Anderson, Frank J. Kelly.

Table of Contents

particles after single day exposure (lag 1 for cardiovascular (A) and lag 2 for respiratory (B) diagnoses) in London, U.K. for 2011–12. Results from two and all sources models.

Table S4. Percent change (and 95% confidence intervals (CIs)) in mortality by season associated with season-specific interquartile range increase in source-related particles (lag 1 for total and cardiovascular and lag 2 for respiratory) in London, for 2011–12.

Table S5. Percent change (and 95% confidence intervals (CIs)) in hospital admissions by age group and season associated with season-specific interquartile range increase in source-related particles after single day exposure (lag 1 for cardiovascular (A) and lag 2 for respiratory (B) diagnoses) in London, U.K. for 2011–12.

Table S1. Measurements collected at the North Kensington site, 2011 and 2012. Source: Beddows et al. 2015, Atmos. Chem. Phys. 15:10107-10125.

Species	Brief description	PM fraction	Detailed description
TMN	Manganese	PM_{10}	Total metal concentration
TMO	Molybdenum		
TNA	Sodium		
TNI	Nickel		
TPB	Lead		
TSB	Antimony		
TSN	Tin		
TSR	Strontium		
TTI	Titanium		
TV	Vanadium		
TZN	Zinc		
TAL	Aluminium		
TBA	Barium		
TCA	Calcium		
TCD	Cadmium		
TCR	Chromium		
TCU	Copper		
TFE	Iron		
TK	Potassium		
TMG	Magnesium		
PCNT	Particle number	PM_{1}	Condensation particle counter
PM10	PM_{10}	PM_{10}	EU reference equivalent; gravimetric with gaps filled from
PM25	$PM_{2.5}$	$PM_{2.5}$	EU reference equivalent; FDMS-TEOM with gaps from
OC	Organic carbon	PM10	
CWOD	OA Wood burning	$PM_{2.5}$	OA from wood using Aethalometer
WNO3	Nitrate	PM_{10}	Water-soluble measured using near-real-time URG
WSO4	Sulfate		
WCL	Chloride		
WNH4	Ammonium		
WCA	Calcium		
WMG	Magnesium		
WK	Potassium		

Figure S1. Factors outputted from PMF2 run on PM10 mass composition data showing the contribution (grey bar) and explained variation of each metric (red bar). Source: Beddows et al. 2015, Atmos. Chem. Phys. 15:10107-10125.

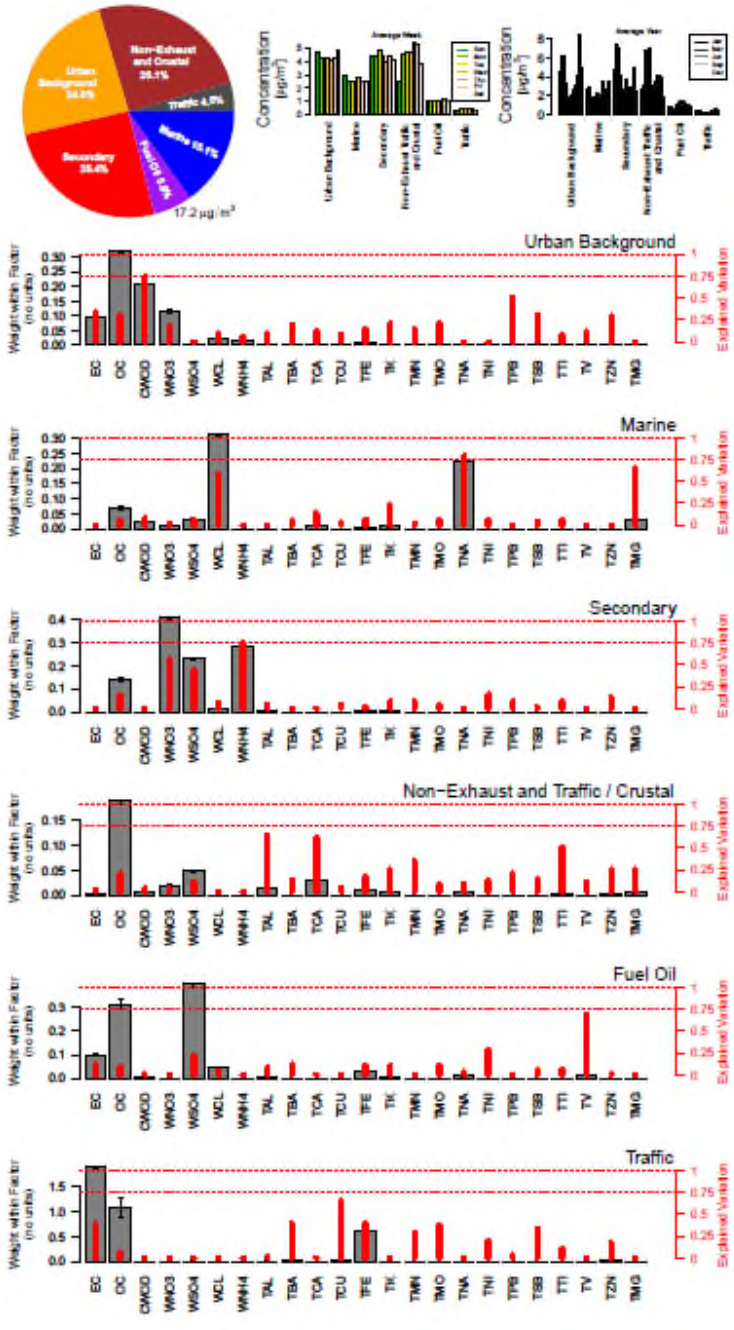

Figure S2. Factors outputted from PMF2 run on particle number size distribution showing the contribution (black line) and explained variation of each metric (red line). Source: Beddows et al. 2015, Atmos. Chem. Phys. 15:10107-10125.

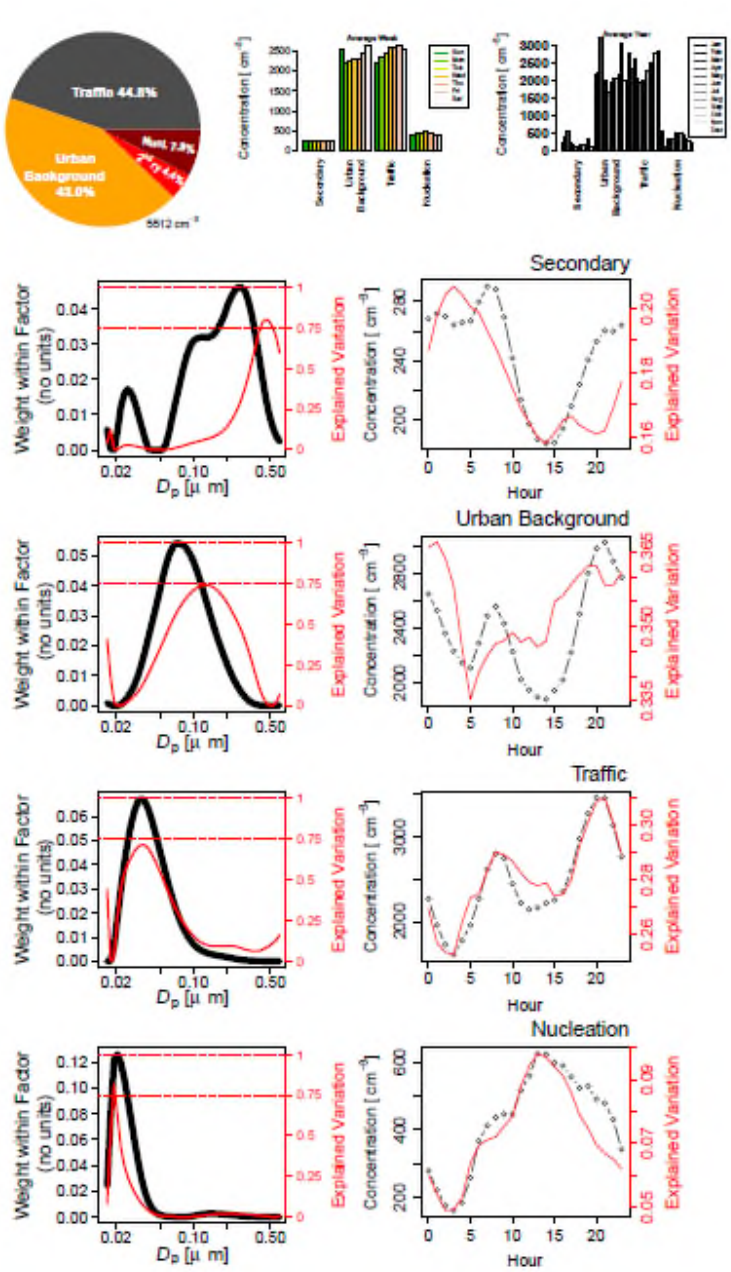

Table S2. Percent change (and 95% confidence intervals (CIs)) in mortality associated with interquartile range increase in source-related particles (lag 1 for total and cardiovascular and lag 2 for respiratory) in London, for 2011–12. Results from two and all sources' models.

	Total mortality % (95%CI)		**CVD mortality % (95%CI)**		**Respiratory mortality % (95%CI)**	
	All sources	**Two-sources**	**All sources**	**Two-sources**	**All sources**	**Two-sources**
PM_{10} ($\mu g/m^3$)						
Urban Background	0.85 (-0.18, 1.89)	0.14 (-0.62, 0.90)	-0.01 (-1.85, 1.86)	-0.81 (-2.15, 0.55)	-0.37 (-2.88, 2.20)	0.66 (-1.19, 2.55)
Marine	0.32 (-0.65, 1.30)	0.42 (-0.53, 1.38)	0.46 (-1.27, 2.23)	0.79 (-0.91, 2.51)	0.46 (-1.97, 2.95)	0.00 (-2.36, 2.42)
Secondary	**-1.01 (-1.57, -0.45)**	**-1.03 (-1.56, -0.49)**	-0.88 (-1.86, 0.11)	**-1.03 (-1.96, -0.10)**	-1.38 (-2.77, 0.02)	-1.25 (-2.58, 0.09)
Fuel Oil	0.65 (-0.40, 1.72)	0.79 (-0.27, 1.86)	0.43 (-1.43, 2.33)	0.50 (-1.37, 2.40)	1.50 (-1.16, 4.23)	1.44 (-1.22, 4.17)
Non-Exhaust Traffic	0.51 (-0.58, 1.62)	-0.03 (-1.02, 0.97)	0.45 (-1.43, 2.37)	-0.28 (-1.97, 1.44)	0.50 (-2.15, 3.22)	1.25 (-1.14, 3.70)
Traffic	-0.66 (-1.72, 0.40)	-0.17 (-0.99, 0.65)	-0.75 (-2.62, 1.16)	-0.79 (-2.23, 0.67)	1.75 (-0.90, 4.47)	1.99 (-0.02, 4.03)
NSD (n/cm^3)						
Urban Background	0.02 (-1.54, 1.60)	-0.73 (-1.83, 0.38)	-0.40 (-3.1, 2.42)	-1.68 (-3.60, 0.28)	7.54 (3.49, 11.75)	1.91 (-0.80, 4.69)
Nucleation	0.08 (-1.14, 1.31)	0.50 (-0.76, 1.79)	-0.96 (-3.12, 1.25)	-0.53 (-2.77, 1.76)	1.51 (-1.56, 4.69)	0.71 (-2.40, 3.91)
Secondary	-0.87 (-2.24, 0.52)	-0.94 (-1.97, 0.10)	-1.66 (-4.02, 0.76)	-1.66 (-3.41, 0.13)	**-5.58 (-8.72, -2.33)**	-1.25 (-3.67, 1.24)
Traffic	0.29 (-1.09, 1.68)	0.23 (-1.22, 1.71)	0.23 (-2.24, 2.77)	0.06 (-2.49, 2.68)	**-4.77 (-8.01,-1.41)**	-2.54 (-5.98, 1.03)

NSD: Number Size Distribution, CI: Confidence Interval. In bold: statistically significant results at $p<0.05$.

Table S3. Percent change (and 95% confidence intervals (CIs)) in hospital admissions by age group and season associated with season-specific interquartile range increase in source-related particles after single day exposure (lag 1 for cardiovascular (A) and lag 2 for respiratory (B) diagnoses) in London, U.K. for 2011–12. Results from two and all sources models.

(A) Cardiovascular Admissions

	15-64 years % (95%CI)		**65+ years % (95%CI)**	
	All sources	**Two-sources**	**All sources**	**Two-sources**
PM_{10} ($\mu g/m^3$)				
Urban Background	-0.75 (-2.23, 0.75)	0.35 (-0.72, 1.44)	0.13 (-0.99, 1.27)	-0.24 (-1.05, 0.58)
Marine	-0.30 (-1.63, 1.06)	-0.47 (-1.77, 0.84)	**1.15 (0.14, 2.18)**	**1.14 (0.16, 2.13)**
Secondary	-0.24 (-1.01, 0.55)	-0.21 (-0.96, 0.53)	-0.53 (-1.12, 0.07)	**-0.71 (-1.27, -0.14)**
Fuel Oil	-0.05 (-1.51, 1.43)	-0.09 (-1.55, 1.39)	-0.28 (-1.37, 0.82)	-0.22 (-1.32, 0.89)
Non-Exhaust Traffic	-0.57 (-2.07, 0.95)	0.13 (-1.24, 1.51)	-0.50 (-1.63, 0.64)	-0.70 (-1.73, 0.33)
Traffic	**1.73 (0.20, 3.28)**	**1.24 (0.11, 2.39)**	0.30 (-0.84, 1.46)	-0.06 (-0.92, 0.81)
NSD (n/cm^3)				
Urban Background	0.76 (-1.48, 3.04)	0.43 (-1.17, 2.06)	0.97 (-0.75, 2.73)	-0.36 (-1.60, 0.88)
Nucleation	-1.28 (-2.95, 0.42)	-1.05 (-2.78, 0.72)	-0.87 (-2.16, 0.44)	-0.84 (-2.19, 0.53)
Secondary	-0.47 (-2.40, 1.50)	-0.24 (-1.69, 1.23)	-1.51 (-2.98, -0.02)	-0.91 (-2.03, 0.22)
Traffic	1.31 (-0.68, 3.34)	0.39 (-1.64, 2.46)	-0.27 (-1.78, 1.26)	-1.07 (-2.62, 0.50)

NSD: Number Size Distribution, CI: Confidence Interval. In bold: statistically significant results at $p<0.05$.

(B) Respiratory Admissions

NSD: Number Size Distribution, CI: Confidence Interval. In bold: statistically significant results at $p<0.05$.

	0-14 years % (95%CI)		**15-64 years % (95%CI)**		**65+ years % (95%CI)**	
	All sources	**Two-sources**	**All sources**	**Two-sources**	**All sources**	**Two-sources**
PM_{10} ($\mu g/m^3$)						
Urban Background	-0.43 (-2.46, 1.64)	0.45 (-0.96, 1.87)	0.75 (-0.73, 2.26)	-0.04 (-1.10, 1.03)	0.20 (-1.15, 1.57)	-0.78 (-1.74, 0.20)
Marine	-0.28 (-2.19, 1.66)	-0.19 (-2.03, 1.68)	-0.05 (-1.38, 1.31)	0.17 (-1.13, 1.50)	0.01 (-1.22, 1.25)	0.12 (-1.08, 1.33)
Secondary	-0.49 (-1.63, 0.66)	-0.78 (-1.86, 0.31)	**-0.90 (-1.71, -0.08)**	**-0.87 (-1.63, -0.10)**	-0.10 (-0.85, 0.65)	0.01 (-0.71, 0.73)
Fuel Oil	**3.50 (1.28, 5.77)**	**3.53 (1.34, 5.76)**	-1.09 (-2.56, 0.40)	-1.19 (-2.64, 0.29)	-0.61 (-1.98, 0.78)	-0.73 (-2.10, 0.66)
Non-Exhaust Traffic	0.52 (-1.80, 2.91)	0.16 (-1.93, 2.29)	0.26 (-1.30, 1.85)	0.06 (-1.31, 1.44)	1.21 (-0.24, 2.68)	0.55 (-0.75, 1.86)
Traffic	1.49 (-0.62, 3.65)	0.84 (-0.68, 2.38)	-0.98 (-2.48, 0.54)	-0.50 (-1.63, 0.65)	**-1.90 (-3.28, -0.50)**	**-1.11 (-2.15, -0.07)**
NSD (n/cm^3)						
Urban Background	1.69 (-1.30, 4.77)	0.65 (-1.49, 2.83)	1.89 (-0.27, 4.09)	-0.25 (-1.77, 1.29)	2.57 (0.78, 4.39)	0.55 (-0.82, 1.94)
Nucleation	1.77 (-0.75, 4.36)	0.42 (-2.27, 3.20)	0.16 (-1.53, 1.88)	-0.38 (-2.12, 1.39)	-0.19 (-1.56, 1.20)	-0.37 (-1.92, 1.20)
Secondary	-0.63 (-3.31, 2.12)	0.28 (-1.74, 2.35)	-1.56 (-3.38, 0.30)	-0.79 (-2.16, 0.60)	**-2.16 (-3.70, -0.60)**	-0.56 (-1.82, 0.70)
Traffic	-1.70 (-4.35, 1.02)	-1.70 (-4.54, 1.23)	-1.68 (-3.57, 0.24)	-1.41 (-3.39, 0.61)	**-1.55 (-3.07, -0.01)**	**-1.80 (-3.54, -0.02)**

Table S4. Percent change (and 95% confidence intervals (CIs)) in mortality by season associated with season-specific interquartile range increase in source-related particles (lag 1 for total and cardiovascular and lag 2 for respiratory) in London, for 2011–12.

	Total mortality % (95%CI)		**CVD mortality % (95%CI)**		**Respiratory mortality % (95%CI)**	
	April-September	**October-March**	**April-September**	**October-March**	**April-September**	**October-March**
PM_{10} ($\mu g/m^3$)						
Urban Background	0.24 (-1.03, 1.52)	0.04 (-1.22, 1.32)	0.62 (-1.62, 2.91)	-1.51 (-3.80, 0.83)	3.12 (-0.16, 6.50)	0.37 (-2.71, 3.55)
Marine	0.52 (-0.66, 1.71)	1.06 (-0.30, 2.43)	0.99 (-1.09, 3.11)	1.18 (-1.31, 3.73)	-1.18 (-4.20, 1.93)	2.25 (-1.06, 5.66)
Secondary	-2.13 (-4.26, 0.03)	**-1.11 (-1.78, -0.44)**	-3.14 (-6.87, 0.74)	-1.06 (-2.29, 0.19)	-1.54 (-7.28, 4.55)	-0.96 (-2.63, 0.74)
Fuel Oil	0.91 (-1.02, 2.88)	**2.87 (1.01 ,4.76)***	0.87 (-2.54, 4.41)	2.81 (-0.62, 6.35)	**5.45 (0.24, 10.94)**	3.18 (-1.36, 7.92)
Non-Exhaust Traffic	-0.25 (-1.16, 0.66)	-0.11 (-1.59, 1.38)	0.27 (-1.34, 1.91)	-1.28 (-3.95, 1.47)	0.12 (-2.17, 2.47)	1.10 (-2.51, 4.85)
Traffic	0.11 (-0.89, 1.12)	-0.20 (-1.40, 1.01)	1.07 (-0.71, 2.88)	-1.94 (-4.12, 0.29)*	1.02 (-1.74, 3.86)	2.53 (-0.34, 5.50)
NSD (n/cm^3)						
Urban Background	-0.23 (-1.83, 1.39)	-0.52 (-2.07, 1.06)	0.73 (-2.13, 3.68)	-2.52 (-5.33, 0.36)	3.22 (-0.89, 7.51)	2.11 (-1.78, 6.15)
Nucleation	0.11 (-1.97, 2.24)	-0.35 (-1.45, 0.76)	-2.15 (-5.84, 1.68)	-0.28 (-2.33, 1.81)	-3.40 (-8.74, 2.27)	-1.39 (-3.97, 1.27)
Secondary	-0.12 (-1.28, 1.06)	-1.39 (-3.43, 0.69)	-0.22 (-2.28, 1.89)	-3.74 (-7.40, 0.08)	-0.34 (-3.17, 2.58)	0.40 (-4.61, 5.68)
Traffic	1.04 (-0.61, 2.70)	-0.07 (-1.92, 1.82)	0.18 (-2.73, 3.18)	-0.45 (-3.85, 3.07)	-1.13 (-5.34, 3.27)	-2.69 (-6.94, 1.75)

NSD: Number Size Distribution, CI: Confidence Interval. In bold: statistically significant results at $p<0.05$.

*Statistically significant different effects between the two periods.

Table S5. Percent change (and 95% confidence intervals (CIs)) in hospital admissions by age group and season associated with season-specific interquartile range increase in source-related particles after single day exposure (lag 1 for cardiovascular (A) and lag 2 for respiratory (B) diagnoses) in London, U.K. for 2011–12.

(A) Cardiovascular Admissions

	15-64 years % (95%CI)		**65+ years % (95%CI)**	
	April-September	**October-March**	**April-September**	**October-March**
PM_{10} ($\mu g/m^3$)				
Urban Background	0.69 (-1.13, 2.55)	0.24 (-1.42, 1.93)	-0.91 (-2.25, 0.45)	0.12 (-1.23, 1.48)
Marine	0.30 (-1.37, 1.99)	-1.60 (-3.25, 0.09)	0.90 (-0.35, 2.17)	1.14 (-0.21, 2.51)
Secondary	-0.95 (-4.10, 2.29)	0.02 (-0.87, 0.92)	-1.01 (-3.32, 1.36)	-0.62 (-1.34, 0.10)
Fuel Oil	0.33 (-2.42, 3.16)	-0.60 (-3.00, 1.87)	0.53 (-1.51, 2.62)	0.67 (-1.29, 2.67)
Non-Exhaust Traffic	-0.48 (-1.82, 0.87)	-0.20 (-2.11, 1.75)	-0.45 (-1.45, 0.55)	-0.66 (-2.21, 0.92)
Traffic	0.73 (-0.71, 2.18)	0.57 (-0.96, 2.13)	-0.73 (-1.78, 0.34)	-0.30 (-1.54, 0.96)
NSD (n/cm^3)				
Urban Background	0.37 (-2.01, 2.80)	0.14 (-1.89, 2.20)	-0.48 (-2.25, 1.32)	0.09 (-1.62, 1.83)
Nucleation	**-5.60 (-8.45, -2.66)**	0.73 (-0.65, 2.12)*	-1.20 (-3.43, 1.08)	-0.48 (-1.68, 0.73)
Secondary	-0.65 (-2.42, 1.15)	0.40 (-2.26, 3.13)	**-1.34 (-2.64, -0.02)**	-0.24 (-2.48, 2.05)
Traffic	0.82 (-1.60, 3.29)	0.47 (-1.95, 2.95)	-0.41 (-2.21, 1.42)	0.21 (-1.83, 2.28)

NSD: Number Size Distribution, CI: Confidence Interval. In bold statistically significant results at $p<0.05$.

* Statistically significant different effects between the two periods.

(B) Respiratory Admissions

	0-14 years % (95%CI)		**15-64 years % (95%CI)**		**65+ years % (95%CI)**	
	April-September	**October-March**	**April-September**	**October-March**	**April-September**	**October-March**
PM_{10} ($\mu g/m^3$)						
Urban Background	2.43 (-0.84, 5.80)	0.56 (-1.86, 3.05)	0.40 (-1.38, 2.22)	-0.29 (-2.04, 1.48)	0.33 (-1.08, 1.76)	**-2.14 (-3.90, -0.35)**
Marine	-1.68 (-4.64, 1.36)	0.40 (-2.08, 2.93)	0.12 (-1.55, 1.81)	0.66 (-1.10, 2.46)	-0.36 (-1.68, 0.98)	**2.04 (0.24, 3.87)***
Secondary	-4.82 (-10.45, 1.17)	-0.10 (-1.45, 1.26)	0.92 (-2.33, 4.28)	**-1.06 (-1.98, -0.12)**	**4.91 (2.31, 7.58)**	-0.41 (-1.40, 0.58)*
Fuel Oil	**7.03 (2.01, 12.28)**	0.83 (-2.71, 4.50)	-2.35 (-5.02, 0.39)	-0.49 (-2.95, 2.03)	-0.54 (-2.70, 1.67)	-0.09 (-2.66, 2.54)*
Non-Exhaust Traffic	-1.63 (-3.93, 0.73)	1.45 (-1.47, 4.47)	0.57 (-0.71, 1.85)	-1.58 (-3.58, 0.45)*	**1.52 (0.53, 2.51)**	-1.40 (-3.47, 0.72)*
Traffic	-0.33 (-3.07, 2.49)	**2.54 (0.35, 4.78)**	0.44 (-1.08, 2.00)	-0.53 (-2.12, 1.08)	0.34 (-0.86, 1.57)	-1.54 (-3.17, 0.12)
NSD (n/cm^3)						
Urban Background	0.56 (-3.53, 4.81)	0.30 (-2.72, 3.42)	0.88 (-1.34, 3.15)	-0.14 (-2.20, 1.97)	**1.96 (0.15, 3.81)**	-0.04 (-2.12, 2.07)*
Nucleation	**6.33 (0.90, 12.05)**	-0.89 (-3.15, 1.42)*	0.91 (-2.03, 3.94)	-0.03 (-1.46, 1.41)	0.89 (-1.49, 3.33)	**-1.61 (-2.99, -0.21)***
Secondary	-2.60 (-5.41, 0.30)	0.12 (-3.89, 4.29)	0.39 (-1.17, 1.96)	-1.40 (-4.06, 1.33)	**1.42 (0.16, 2.69)**	-1.61 (-4.28, 1.13)*
Traffic	-0.73 (-5.00, 3.74)	1.39 (-2.19, 5.09)	0.97 (-1.38, 3.37)	-0.70 (-3.07, 1.72)	0.04 (-1.85, 1.96)	-1.60 (-3.92, 0.78)

NSD: Number Size Distribution, CI: Confidence Interval. In bold statistically significant results at $p<0.05$

*Statistically significant different effects between the two periods