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

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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 and 2012 to derive six source-related factors for PM10 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 PM10 or NSD source-related factors and daily mortality, as the direction of the estimates were variable with 95% CI spanning 0%. Traffic-related PM10 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 PM10 and 0.81% for NSD). Most sources were positively associated with pediatric (0−14 years) respiratory hospitalizations, with stronger evidence for fuel oil PM10 (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.

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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 (Lal 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 PM2.5 for Washington, DC between 1988 and 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 PM2.5 source apportionment methods were sufficiently robust to derive reliable insights into the source components that contribute to PM2.5 health effects (Thurston et al., 2005).

The Clearflo project (Böhenstengel et al., 2015) characterized, in detail, the air pollution mixture in London between 2011 and 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., 2016a,b; 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 PM10 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 <10 μm (PM10)), particle number and size distribution (NSD) as a measure of ultrafine particles (with diameter <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 characterized in detail previously (Bigi and Harrison, 2010).

Mean daily temperature (°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,

\[ c_{ij} = \sum f_{ik} g_{jk} + e_{ij} \]

where \( c_{ij} \) is the concentration of component \( i \) in air sample \( j \), \( f_{ik} \) the fractional contribution of component \( i \) to the particles emitted by source \( k \), \( g_{jk} \) is the contribution of source \( k \) to the mass of particles in air sample \( j \), and \( e_{ij} \) 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 \( E \) 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 PM10 data was given by six factor solution. Source profiles for PM10 are presented in Supplementary Fig. S1. The largest contribution to PM10 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 aluminum, 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 PM10 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 Fig. 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 0.25 μm and peaked at nighttime was attributed to secondary particles which may be inferred to have arisen from long-range transport processes.
2.4. Statistical analysis

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

$$\log E[Y_t] = \beta_0 + b \cdot Pol_t + s(\text{time}_t) + \sum s(X_{it})$$

where $E[Y_t]$ is the expected value of the Poisson distributed variable $Y_t$, indicating the daily outcome count on day $t$ with $\text{Var}(Y_t) = \phi E[Y_t]$. $\phi$ being the over-dispersion parameter, $\text{time}_t$ is a continuous variable indicating the time (day) of event, $Pol_t$ is the concentration of the source-related factor on day $t$, $X_{it}$ is the value of confounder $X_i$ on day $t$, and $s$ denotes smoothing functions. We used penalized regression splines (Wood, 2000) as smoothing functions $s$ to capture the association between time-varying covariates, calendar time and health outcome. Degrees of freedom (df) for long term trends were based on the minimization of the absolute value of the sum of the partial autocorrelations of the residuals from lags 1 to 30, imposing a minimum of 3 df per year. We also included dummy variables for the day of the week and public holidays. For the analysis of respiratory admissions among ages 0–14 and 15–64 years we included an extra dummy variable denoting the month of August, as the decrease in the respiratory admissions at this period could not be sufficiently captured by the smooth term of seasonality. We controlled for mean daily temperature and relative humidity to address any potential confounding effects of weather. For temperature control we applied a natural spline 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 penalized 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., 2016).

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.

Table 1

Descriptive statistics for mortality and hospital admissions, concentrations of particles, source-specific estimated concentrations and meteorological variables in London, U.K. for 2011–12.

| Source | Number of days | 10th percentile | Median | IQR (75th–25th percentile) | 90th percentile |
|--------|----------------|-----------------|--------|----------------------------|----------------|
| **Mortality** (n/day) | 246/12/2012 | | | | |
| Total | 722 | 99 | 117 | 21 | 139 |
| Cardiovascular | 722 | 27 | 35 | 9 | 45 |
| Respiratory | 722 | 11 | 17 | 8 | 25 |
| **Hospital admissions** (n/day) | 2012 | | | | |
| Cardiovascular | 731 | 39 | 57 | 25 | 71 |
| Respiratory | 731 | 22 | 45 | 9 | 72 |
| 15–64 years | 731 | 48 | 63 | 16 | 81 |
| 65 + years | 731 | 77 | 91 | 28 | 125 |
| **PM_{2.5}** (µg/m³) | | | | | |
| 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 |
| Freight | 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/cm³) | | | | | |
| Total number per cm³ | 636 | 7958.0 | 12,123.5 | 5180.0 | 17,901.0 |
| Urban background | 590 | 818.4 | 1893.2 | 1806.2 | 4442.2 |
| Nuclear | 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 |

IQR: Interquartile range; NSD: Number size distribution.

* 01/01/20–22/12/2012.
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 µg/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 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 association 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 2, in general <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.

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

| 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 | | | | | | |
| | Secondary | 0.31 | -0.21 | | | | | |
| | Fuel oil | -0.10 | -0.07 | -0.16 | 1 | | | |
| | Non-exhaust traffic | 0.15 | -0.23 | 0.21 | -0.15 | 1 | | |
| | Traffic | 0.02 | -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. |
| PM$_{10}$ (µg/m$^3$) | All-cause % (95%CI) | Cardiovascular % (95%CI) | Respiratory % (95%CI) |
|-----------|------------------|---------------------|---------------------|
| 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$^3$ | All-cause % (95%CI) | Cardiovascular % (95%CI) | Respiratory % (95%CI) |
|-----------|---------------------|---------------------|---------------------|---------------------|
| Total | -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.
oil-related PM10 displayed the highest and statistically significant effect estimate (3.43%, 95%CI: 1.26%, 5.65%), while nucleation sources drove the association with NSD (0.97%, 95%CI: -1.31%, 3.30%). While the CI’s between different age strata greatly overlap indicating absence of heterogeneity, the effect of fuel oil PM10 on pediatric respiratory hospitalizations was significantly different to the corresponding estimates for the other age groups as was also the traffic related PM for both pediatric respiratory hospitalizations and CVD for those 15–64 years. Finally, as negative findings make also positive effect estimates dubious, we tested the hypothesis of possible residual confounding for our positive results using the method proposed by Flanders et al. (2011). For all estimates that were found to be positive and statistically significant and there was no indication of residual confounding, as they were robust to adjustment of future pollution levels.

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

**Table 4**

Percent change in cardiovascular (lag1) and respiratory (lag2) hospital admissions associated with interquartile range increases in source-related PM10 and NSD in London, 2011–12. Results from single source models.

| Source         | Cardiovascular % (95%CI) 15–64 years | 65 years+ | Respiratory % (95%CI) 0–14 years | 15–64 years | 65 years+ |
|----------------|--------------------------------------|-----------|----------------------------------|-------------|-----------|
| PM10 (µg/m³)   |                                      |           |                                  |             |           |
| Total concentration | 0.17 (–0.86, 1.21)                  | –0.50 (–1.27, 0.28) | 0.69 (–0.85, 2.25)               | –0.67 (–1.69, 0.17) | –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 (–1.66, –0.13) | –0.19 (–0.89, 0.52) |
| Fuel oil       | –0.12 (–1.56, 1.35)                 | 0.14 (–1.23, 0.97)  | 3.41 (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 (µ/cm³)    |                                      |           |                                  |             |           |
| Total number/cm³ | 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 (–2.26, 0.87) | 1.21 (–2.57, 0.18) |

NSD: Number size distribution, CI: Confidence interval. In bold: statistically significant results at p < 0.05.

4. Discussion

Associations between daily health metrics and six source-realted factors for PM10 (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

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**Figure 1.** Percent increase (and 95% confidence intervals) in respiratory admissions 0–14 years associated with an interquartile range increase in the source-related PM10 concentrations (µg/m³) and NSD (µ/cm³). Results from models including each source individually (triangles), adjusted for all other sources (squares) and after controlling for the total concentration in PM10 or numbers in NSD minus the specific source (circles).
the analysis of the PM10 chemical composition data is able to distinguish 
carbon in PM as a diesel exhaust marker and carbon monoxide, as an 
indicator of petrol vehicle exhaust. Although previously there were 
indications that aluminum as an indicator of dust re-suspension and 
on-exhaust traffic was associated with adult hospitalizations, this was not 
replicated in the present analysis that incorporated aluminum in the 
non-exhaust factor along with other related elements. Nevertheless, 
previous studies in London during 2000–2005 have reported associations 
between particle number concentration and cardiovascular mortality and 
admissions (Atkinson et al., 2010) as well as secondary particles and respiratory mortality (Pirani et al., 2015) that we did not find in our study. These discrepancies may be attributed to the different 
and exposure metrics used (for example Atkinson et al. (2010) 
analyzed number concentration and all-ages CVD admissions), different 
statistical approaches (Bayesian approach in Pirani et al. (2015)) and 
the longer period (6 and 4 years in the previous analyses over 2 in our 
case). Changes in the urban pollution mixture and concentrations over 
the period 2000 to 2012 can also not be ruled out, but these possible 
temporal issues cannot be assessed under the settings of this study. 
One notable change which occurred between the periods of data used 
by Atkinson et al. (2010), the years 2000–2005, and the current study, 
2011–2012, is a reduction in the sulfur content of motor fuels which 
causd a major reduction in the concentration, size distribution and 
chemical composition of ultrafine particles (<100 nm diameter) 
(Jones et al., 2012). 

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

Finally, as source contributions are estimated and not actually measured, their estimation is associated with some increased uncertainty. 
Following the report of the EPA workshop on similar epidemiological 
findings using different source apportionment methods (Thurston et al., 2005), Kioumourtzoglou et al. (2014) and Gass et al. (2015) showed that ignoring this uncertainty may lead to significant understimation of the epidemiological inferences, regardless 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 PM2.5 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 PM10 on total mortality in our study. Similarly, Lijngman et al. (2016) reported that PM2.5 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 Zanobetti et al. (2014) reported significant, positive associations between same-day PM2.5 attributed to mobile sources and biomass combustion in an Atlanta site and CVD-related emergency department visits, while steel industry and traffic related PM2.5 was associated with respiratory and cardiovascular admissions respectively in New York (Lall et al., 2011). Pun et al. investigated associations between source-related PM10 and emergency hospitalizations either due to respiratory causes (Pun et al., 2015) or to ischemic heart disease (IHD) (Pun et al., 2014) in Hong Kong. Vehicle-exhaust was associated with both outcomes (2.0% 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 (1.55% increase) and secondary nitrate (2.28%) or salt-related (1.19%) PM10 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 (Lantki 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 PM2.5 and cardiovascular outcomes (Hennig et al., 2014; Thurston et al., 2016) also reported traffic specific PM effects as well as with fossil fuel combustion signatures (Thurston et al., 2016).

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 PM10 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., 2016a; Samoli et al., 2016).

5. Conclusions

In conclusion, our results suggest that traffic may be the dominant source for both PM10 and NSD driving the associations with adult CVD hospitalizations, while pediatric respiratory hospitalizations may also be driven by fuel oil PM10 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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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.envint.2016.09.017.

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