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Is long-term exposure to traffic pollution associated with mortality? A small-area study in London

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ABSTRACT

Long-term exposure to primary traffic pollutants may be harmful for health but few studies have investigated effects on mortality. We examined associations for six primary traffic pollutants with all-cause and cause-specific mortality in 2003–2010 at small-area level using linear and piecewise linear Poisson regression models. In linear models most pollutants showed negative or null association with all-cause, cardiovascular or respiratory mortality. In the piecewise models we observed positive associations in the lowest exposure range (e.g. relative risk (RR) for all-cause mortality 1.07 (95% credible interval (CI) = 1.00–1.15) per 0.15 μg/m^3 increase in exhaust related primary particulate matter ≤2.5 μm ($PM_{2.5}$)) whereas associations in the highest exposure range were negative (corresponding RR 0.93, 95% CI: 0.91–0.96). Overall, there was only weak evidence of positive associations with mortality. That we found the strongest positive associations in the lowest exposure group may reflect residual confounding by unmeasured confounders that varies by exposure group.

1. Introduction

There is concern over the effect of traffic pollution on health (HEI, 2010; WHO, 2013). This relates both to the increased concentrations present near roads and to the possibility that primary traffic pollutants might be more toxic than those comprising the regional/urban background such as total particulate mass with aerodynamic diameter <2.5 μm ($PM_{2.5}$). Investigating this question using epidemiological methods is challenging because variations in air pollution concentrations due to primary traffic emissions occur in addition to considerable regional/urban background pollutant concentrations (Kunzli, 2014). Further, there is considerable potential for confounding of pollution effects by socioeconomic factors when conducting small-area analyses within cities. Although the association between $PM_{2.5}$ or PM_{10} and mortality is well established (Beelen et al., 2014a; Carey et al., 2013; Cesaroni et al., 2013; Jerrett et al., 2013; Krewski et al., 2005, 2009; Maheswaran et al., 2005), these tend to be more influenced by urban/regional background concentrations and to date, there is less evidence associating mortality with long-term exposure to the primary traffic pollutants such as nitrogen oxides (NO_x) or particles related specifically to vehicle emissions (Beelen et al., 2008, 2014b; Cesaroni et al., 2013; Dimakopoulou et al., 2014; Jerrett et al., 2013; Maheswaran et al., 2005). The majority of epidemiological studies of air pollution and mortality have been based on comparisons between cities, which vary in regional/urban background pollutants as well as in traffic related pollution. In contrast, the investigation of health effects of near-traffic pollution requires analyses at a fine spatial scale within a city.

Reviews of epidemiological studies indicate that socioeconomic deprivation may, in addition to being associated with greater exposure, also adversely modify the effect of air pollution exposure on health (Deguen and Zmirou-Navier, 2010; Sacks et al., 2011). However, few long-term exposure studies have investigated whether deprivation might modify associations for primary traffic

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pollutants (Atkinson et al., 2013; Carey et al., 2013).

As part of a research programme into the health effects of traffic pollution (TRAFFIC study) (King's College London, 2014) we investigated the association between primary traffic pollution and mortality in London, a city of approximately nine million inhabitants. We conducted a small-area ecological study of mortality using spatially resolved estimates of exposure to six primary traffic pollutants as well as five pollutants, such as $PM_{2.5}$, which tend to be more influenced by regional/urban background concentrations. Traffic noise was treated as a potential confounder (Tetreault et al., 2013); a substantive paper on road traffic noise, mortality and hospital admissions is published elsewhere (Halonen et al., 2015). In this paper we address two questions: 1) is long-term exposure to primary traffic pollutants associated with increased risk of all-cause or cause-specific mortality? and 2) are these associations modified by socioeconomic deprivation?

2. Materials and methods

2.1. Study area and population

Our study area comprised all of London within the M25 motorway (~2,156 km^2) (Supplemental Fig. 1), with a population of over eight million. The spatial unit for our analysis was the census geographical unit Lower Layer Super Output Area (LSOA, n = 5482) with a mean population of 1500 (range 1000–3000) (Office for National Statistics, 2014). The analysis comprised 5358 LSOAs with complete information for the exposures, health outcomes and area-level confounders.

2.2. Outcomes

We included mortality data for the population aged 25 years or over. The underlying cause of death was classified using the 10th revision of the International Classification of Diseases (ICD10): all natural (A00-R99); cardiovascular (I00–I99) and respiratory (J00-J99) causes. In addition to the cause of death, these data included the person's age, sex and postcode of residential address at registration of death. We also obtained annual mid-year population estimates at Census Output Area (COA) level (with a mean population of 300 (range 100–1200) by age and sex for 2003–2010. Annual death counts and population estimates were aggregated to LSOAs, the smallest unit with sufficient deaths for the cross-sectional analysis, by sex and 5-year age bands to calculate mortality rates. The mortality and population data were supplied by the Office for National Statistics (ONS), derived from the national mortality registrations and the Census. Data are held by the UK Small Area Health Statistics Unit (SAHSU) at Imperial College London. Data use was covered by approval from the National Research Ethics Service – reference 12/LO/0566 and 12/LO/0567 – and by National Information Governance Board and Ethics and Confidentiality Committee approval for section 251 support (NIGB – ECC 2–06(a)/2009).

2.3. Pollution concentrations

We used a dispersion model to estimate average annual concentrations of six primary traffic pollutants; nitrogen oxides (NO_x), nitrogen dioxide (NO_2), as well as exhaust (tailpipe emissions) and non-exhaust (brake and tyre wear and re-suspension) related primary $PM_{2.5}$ and PM_{10}, for ~190,000 postcode address centroids in London for each year between 2003 and 2010. This model provided corresponding estimates for regional/urban background pollutants $PM_{2.5}$, PM_{10} (aerodynamic diameter less than 2.5 μm and 10 μm, respectively) and ozone (O_3), and from these we calculated coarse fraction of PM_{10} ($PM_{10\text{-}2.5}$) and oxidative gases (O_x, i.e. combined oxidant $NO_2 + O_3$) (Clapp and Jenkin, 2001; Williams et al., 2014). The model includes regional/urban background $PM_{2.5}$, PM_{10}, and O_3 from outside London, which is predominantly secondary in nature, having longer atmospheric lifetimes and being more homogenously distributed. All "background" emission sources from London are represented as volume sources of dimension 1 × 1 km and between 2 m and 50 m high. Very close to the address centroids, then, we represent traffic emissions road by road, which results in model predictions that are highly detailed close to road sources where concentration gradients are steepest. By using dispersion models and detailed emissions we were able to estimate the contribution to pollutant concentrations from London's road traffic alone. We used the KCLurban dispersion modelling system based on Atmospheric Dispersion Modelling System (ADMS) v.4 and road source model v.2.3, which incorporates hourly meteorological measurements, empirically derived $NO–NO_2–O_3$ and PM relationships, and information on source emissions from the London Atmospheric Emissions Inventory (LAEI) (Greater London Authority, 2008). The model was not adjusted in any way and performed well when validated against measurements: a comparison of observed vs. modelled concentrations provided high spearman correlation coefficients (r). The values of r for monthly NO_x and NO_2 varied across a relatively small range giving an average (and standard deviation) of 0.82 (0.049) and 0.84 (0.033), respectively. More detailed information about the modelling procedure and model validation can be found elsewhere (Beevers and Dajnak, 2015; Beevers et al., 2013). Whilst industrial sources in the UK and Europe are important contributors to regional scale particulate matter concentrations and locally can contribute to short periods of high pollutant concentrations, in London emission sources are dominated by road traffic, whose effects on human exposure are multiplied by being released at ground level. The combination of the small scale of industrial emissions in London, their emissions released at height, and the use of annual mean concentrations in this study means that London's industrial sources play a minor role in human exposure. We aggregated all exposure data to LSOAs by 1) calculating the mean of all postcode address centroid annual averages within a LSOA, and 2) calculating the mean across all study years within a LSOA.

2.4. Confounders

Exposure to traffic-related air pollution (Goodman et al., 2011) and risk of mortality (Halonen et al., 2013; Meijer et al., 2012) have been shown to vary by area-level socioeconomic deprivation. We therefore used the Carstairs deprivation index (a composite measure based on unemployment, overcrowding, car ownership and low social class) (Morgan and Baker, 2006) standardized to the study area as a marker of LSOA-level deprivation with higher values indicating more deprived areas. We adjusted for ethnic differences between LSOAs using the percentage of black ethnicity and South Asian ethnicity. The Carstairs and ethnicity data were derived from the UK Census 2011 provided by the Office for National Statistics.

Because no individual- or area-level smoking data were available, we used annual smoothed age and sex standardised relative risk of lung cancer mortality (ICD-10: C33–C34) at the LSOA level as a proxy for smoking as in a previous study in London (Hansell et al., 2013). Because of variations in population structure and higher pollution levels in the London city centre we constructed a dummy variable for inner (n = 13) and outer n = (20) London boroughs (London Councils, 2014) for inclusion in sensitivity analyses.

To control for possible confounding by road traffic noise (Tetreault et al., 2013) we included daytime A-weighted equivalent continuous sound pressure level ($L_{Aeq16,\ road}$) in all models. We

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