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Different health effects of particulate pollution from different sources. TRAPART 2
Abstract

The size and composition of PM vary with source, and the health effects from PM10 have been reported to depend on these properties. Usually fine combustion related particles are judged to be more toxic, and crustal and coarse particles to be less harmful. In Swedish cities the coarse fraction comes to a great deal from road dust in spring and winter. The regional background is mainly from long distance transported PM, originating from combustion processes and thus has a large proportion of secondary particles and soot.

We have studied the effect of daily concentrations of regional PM10, urban background road dust PM10 and motor vehicle exhaust indicated by NOx. We have studied morbidity in terms of daily number of cardiovascular and respiratory hospital admissions and deaths in Greater Stockholm. The association between the mean of lag 0-1 (same day and yesterday) pollution concentration and daily number of cases was evaluated using Poisson regression (software R) with adjustments for meteorology, calendar variables, influenza and time trends.

Cardiovascular hospital admissions were significantly associated with NOx (vehicle exhaust), regional background PM10 and ozone, but not with PM10 from road dust. For road dust we found a significant association with respiratory hospital admissions. The respiratory admissions were not associated with NOx, but with regional background PM10 and ozone. For daily mortality there were no significant association observed. Primary and secondary particles from combustions sources do not seem to show the same relations to health as road dust dominated by crustal particles.
Introduction

There is a great body of evidence that ambient particles have short-term effects on mortality, hospital admissions and emergency visits. These positive associations have been found rather consistently in studies using particles up to 10 µm in aerodynamic diameter (PM10) and other particle measures such as PM2.5, black smoke and soot. There is also human experimental support for respiratory and cardiovascular effects of particle exposure, even for the coarse fraction 2.5-10 µm (Graff et al, 2009). Thus, these relations are usually considered to be causal.

A literature review (Brunekreef and Forsberg 2005) concluded that the majority of published mortality studies were unable to demonstrate independent coarse particle effects on mortality after adjustment for fine particles. However, most time-series studies that found significant effects on mortality associated with coarse particles, come from arid areas where the particles likely were much out of sand and soil. These studies were from places like Phoenix (Mar et al. 2000); Coachella Valley, California (Ostro et al. 2000); and Mexico City (Castillejos et al. 2000).

Also recent studies from southern Europe have explored the effects of windblown desert sand on health. An interesting study conducted in Barcelona, Spain found an effect of coarse particles on daily mortality during Saharan dust days despite rather moderate particle concentrations (Perez et al. 2008). It has also been suggested that also particles of crustal origin are associated with markers of inflammation and acute toxicity (Steerenberg et al. 2006), and how the mineral composition and surface reactivity has been important for the inflammatory response (Schwarze et al. 2007).

The major sources of coarse particles are often windblown dust from soil, roads, building and construction activities. During winter and spring PM10 in Swedish cities could to a large proportion be coarse road dust particles. Particles may also have a biological origin, as pollen, mould spores and parts of plants. Fine mode particle mass is largely a secondary particles (i.e. sulphates and nitrates) originating from combustion and formed by atmospheric conversion of gases into particles and coagulation of small particles to larger.
Both fine and coarse particles are generated by road traffic. The major part of the fine particles are primary particles formed due to incomplete combustion of diesel and gasoline and secondary particles formed due to gaseous emissions (organic compounds, NOx, and SO2). Primary non-exhaust particles are formed due to mechanical wear of roads, brake linings and tyres. Non-exhaust particle emissions along roads are often called re-suspension. This is somewhat misleading since it is not mainly a suspension of existing particles on the surface and also not a resuspension of formerly airborne particles that have been emitted in the same urban airshed and deposited on the road surface due to settling.

Non-exhaust emission processes may depend strongly on the road surface conditions; whether it is wet or dry, and also on the type of vehicle, its weight and speed. The build-up of particles on the road surface also depend on meteorological processes; rain events clean the surface, drying/freezing of a wet surface may increase erosion of the surface. A wet surface cause more efficient wear due to tyres causing an accumulation of dust during wet periods that may become airborne during subsequent dry periods. Other important factors include type of asphalt and especially in northern Europe the frequency and amount of sanding and salting. Sand on a road surface may increase the wear due to a “sandpaper” effect. Salt may also increase erosion of road material. The wear of the road surface is much larger when studded tyres are used due to ice and ground frost on the roads. The cleaning regime is important for the amount of particles on the road surface. Street sweepers may effectively capture large material (e.g. sand) but be ineffective at removing finer dust or even increase dust emissions. The general recirculation of urban dust also depends on the humidity and wind speed.

Due to the many important determinants, it is not possibly to give general emission factors for road dust. The amount of road dust varies substantially between locations and over time. A Swedish study found that vehicles emit ten times more particle mass of non-exhaust particles than exhaust particles, while other studies found road dust to important but be less dominating.

The EU directive regulates PM10, the total mass of all particles less than 10 µm irrespective of size, morphology and chemistry and also irrespective of their health effects. However, for pollution control it is important to know if some types of particles cause more severe effects, or if some sources, as road wear, involve mostly harmless particles.
Population and Methods

Health outcomes
This analysis of 1998 through 2005 was based on daily counts of hospital admissions for respiratory and cardiovascular causes in the Greater Stockholm area (population approx. 1.3 mil.) from the Patient Register at the Swedish National Board of Health and Welfare. Only admissions at the seven hospitals in this area by residents from the area were included. The daily number of deaths in the same population, excluding external causes, was obtained from the national Cause of death register.

Air pollution
Data on daily urban background concentrations of PM$_{10}$, NOx and ozone were obtained from the Environment and Health Administration of Stockholm, measured in central Stockholm at Torkel Knutssonsgatan, a monitoring station located at roof-top level (25 m high) not directly affected by nearby emissions. Measurements from this monitoring station have been used to represent fluctuations in particle levels in Stockholm in previous studies such as APHEA-2. The rural (regional) background concentration of PM$_{10}$ and ozone was measured at Aspvreten south-east of Stockholm, and NOx at Norr Malma north-east of Stockholm. The mass concentration of PM$_{10}$ was measured using Tapered Element Oscillating Microbalance (TEOM 14001, Rupprecht & Patashnick Inc., USA). To account for losses of volatile material of the particles, all data were corrected following Areskoug. Continuous measurement of NOx was done using chemiluminescence monitors. Continuous measurement of ozone at Torkel Knutssonsgatan and at Aspvreten was based on UV-absorption.

The urban NOx concentration was used as an indicator for motor vehicle exhaust, since no long series with specific exhaust particles were available.

Meteorological observations for temperature and relative humidity were collected from the Environment and Health Administration of Stockholm, measured at the station Torkel Knutssonsgatan.

The concentration of PM$_{10}$ from road dust (PM$_{\text{resusp}}$) was calculated as:

\[ \text{PM}_{\text{resusp}} = \text{PM}_{10}\text{urban}-\text{PM}_{10}\text{ruraladj} - \frac{24}{1000}(\text{NOx}_{\text{urban}}-\text{NOx}_{\text{rural}}) \]
We needed to adjust the zero for $PM_{10rural}$ since there was a shift in the baseline for parts of the study period:

$$PM_{10ruraladj} = PM_{10rural} - \text{annual mean } PM_{10rural} + \text{study mean } PM_{10rural}$$

**Statistical Analysis**

We modelled the data with an additive Poisson regression. The strategy follows that of the APHEA2 Study, Katsouyanni et al. (2001), i.e. a sum of nonparametric smooth functions are used to model potential nonlinear explanatory variables. Also control of overdispersion and adjustment for autoregressive components was included. The smoothing technique used in this study is penalized splines as implemented in R 2.0 using the MGCV package (Wood, 2002). Different degrees of smoothing span were evaluated and the partial autocorrelation (PACF) and visual inspection of residuals were used to select AR-term.

For air pollutants we followed the approach used in APHEA2 and many other studies, and evaluated the association with the mean value of the same and previous day (lag01).

The Poisson models included smooth adjustment for time trend and season, relative humidity and temperature. Day of the week, holidays and influenza (admissions) were also included in the regression analysis.
Results

Descriptives
The daily minimum, mean and maximum numbers of hospital admissions during the study period were 3, 22 and 62 for respiratory causes. For cardiovascular admissions these numbers were 5, 52 and 101 admissions, respectively, and for mortality (all ages, non-external) 14, 29 and 52 deaths.

The mean daily rural background concentration of PM10 was 9.7 µg/m3 and the range was 65 µg/m3. The mean daily concentration of the calculated concentration of road dust PM10 (PM10resusp) was 7.6 µg/m3, the range 76 µg/m3 and the interquartile range (IQR) for the exposure variable used (lag01) was 5.9 µg/m3. The daily mean of NOx was 23 µg/m3 (IQR= 12.6 µg/m3) and of urban background ozone 59 µg/m3 (IQR=29.7 µg/m3).

The correlations between different pollutants were in general low, as shown in Table 1. Road dust PM10 (resusp) was not correlated with regional background (rural) PM10. The correlation between road dust PM10 and urban background ozone was rather low (r=0.26), as with urban background NOx (r=0.24).

Table 1. Correlation coefficients [r] for the pollution variables.

<table>
<thead>
<tr>
<th></th>
<th>PM_{resusp}</th>
<th>PM_{rural}</th>
<th>Ozone_{urban}</th>
<th>Ozone_{rural}</th>
<th>NOx_{urban}</th>
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<tbody>
<tr>
<td>PM_{resusp}</td>
<td>0.04917</td>
<td>0.26394</td>
<td>0.37965</td>
<td>0.23745</td>
<td></td>
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<tr>
<td>PM_{rural}</td>
<td>0.04917</td>
<td>0.30934</td>
<td>0.30010</td>
<td>-0.07763</td>
<td>-0.23249</td>
</tr>
<tr>
<td>Ozone_{urban}</td>
<td>0.26394</td>
<td>0.30934</td>
<td>0.90863</td>
<td>-0.39588</td>
<td></td>
</tr>
<tr>
<td>Ozone_{rural}</td>
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<td>-0.39588</td>
<td>-0.23249</td>
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</table>

Table 2 shows the results from the analysis of dose-response relations. Cardiovascular hospital admissions were significantly associated with NOx (indicator for vehicle exhaust),
regional background PM10 and ozone, but not with PM10 from road dust. An increase in regional background (rural) PM10 of 10 µg/m3 was associated with 1.9% more hospital admissions for cardiovascular diseases. For CVD admissions the effect of an increase corresponding to the IQR was largest for ozone (2.1%).

For road dust we found a significant association with respiratory hospital admissions. An increase in road dust PM10 of 10 µg/m3 was associated with 2.6% more hospital admissions for respiratory diseases (Table 2). The respiratory admissions were even stronger associated with ozone and regional background (rural) PM10, but not with vehicle exhaust using NOx as indicator. For daily mortality there were no statistically significant associations observed. However, there was an indication that daily mortality increases with the concentration of road dust PM10.

Table 2. Percent increase in hospital admissions from multiple pollutant models.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Exposure</th>
<th>% per 10 µg/m³</th>
<th>95% CI (%)</th>
<th>% for increase = IQR</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVD admissions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM_{resusp}</td>
<td>-0.18</td>
<td>(-1.60, 1.23)</td>
<td>-0.11</td>
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<tr>
<td>PM_{rural}</td>
<td>1.89</td>
<td>(0.53, 3.25)</td>
<td>1.03</td>
<td></td>
</tr>
<tr>
<td>NO_{urban}</td>
<td>1.01</td>
<td>(0.24, 1.76)</td>
<td>1.26</td>
<td></td>
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<tr>
<td>Ozone_{urban}</td>
<td>0.71</td>
<td>(0.05, 1.37)</td>
<td>2.10</td>
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</tr>
<tr>
<td>Respiratory admissions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM_{resusp}</td>
<td>2.61</td>
<td>(0.32, 4.91)</td>
<td>1.53</td>
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<tr>
<td>PM_{rural}</td>
<td>4.18</td>
<td>(1.92, 6.45)</td>
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<tr>
<td>NO_{urban}</td>
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<td>(-1.31, 1.20)</td>
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</tr>
<tr>
<td>Ozone_{urban}</td>
<td>1.56</td>
<td>(0.45, 2.68)</td>
<td>4.64</td>
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<tr>
<td>Mortality</td>
<td></td>
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<tr>
<td>PM_{resusp}</td>
<td>1.62</td>
<td>(-0.76, 4.00)</td>
<td>0.95</td>
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<td>(-1.17, 3.37)</td>
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<tr>
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<td>-0.40</td>
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<td>Ozone_{urban}</td>
<td>-0.48</td>
<td>(-1.48, 0.53)</td>
<td>-1.42</td>
<td></td>
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</table>
Discussion

We used NOx as an indicator for vehicle exhaust, and found a statistically significant association with cardiovascular hospital admissions. This is in line with previous studies that observed an effect on cardiovascular risk from fine or ultrafine particles, soot and particle number concentration. However, there was no significant association with daily mortality and the (non-significant) coefficient was in the unexpected direction (negative). There was no sign of an effect of NOx concentrations on respiratory hospital admissions. The series of more relevant exhaust indicators than NOx still tend to be too short for this type of studies.

An increase in road dust PM10, likely dominated by particles larger than 2.5 µm, did show a significant effect on respiratory hospital admissions. This is in line with previous studies (Brunekreef & Forsberg, 2005). Respiratory admissions were also strongly associated with regional background PM10, dominated by secondary particles rather than primary combustion particles. There was also a non-significant increase in daily mortality following an increase in the concentration of road dust PM10. This observation suggests further studies with more power, meaning a longer time-series or more cities with the same road-dust problem due to studded tyres.

The effects of ground-level ozone appear clear on hospital admissions. An earlier analysis from APHEA-2 reported effects on daily mortality also in Stockholm (Gryparis et al, 2004). Ozone is not a local problem in this region, but ozone is a secondary pollutant with important precursors originating from motor vehicles, why emission reductions are important from a health point of view.
Acknowledgements

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References


