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Effects of road dust PM on acute morbidity. TRAPART 1

Abstract

Health effects from PM have been reported to depend much on the fine fraction, PM2.5, while the coarse fraction, including road dust, has been seen as less harmful. In Swedish cities the proportion of cars with studded tyres is often 70-90 percent in winter, producing large emissions of road wear particles and in some cases violations of the Swedish air quality limits for PM10. Thus there has been a discussion whether these particles should be in focus or not.

We have studied the effect of daily concentrations of PM10 from road dust on morbidity in terms of hospital admissions i Greater Stockholm. The concentration of road dust PM10 has been calculated from urban background PM10 from a TEOM instrument by subtracting the rural PM10 concentration and exhaust particles as PM10 estimated from the urban NOx contribution.

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.

We found a significant association between road dust PM10 and all respiratory hospital admissions, RR = 2.6% per 10 µg/m3 (95% CI= 3.2-4.9%), while the effect on cardiovascular hospital admissions was non-significant and negative. Also other studies have found cardiovascular effects to be more associated with fine PM, and coarse PM to affect more respiratory outcomes.

Introduction

Hundreds of reports and papers constitute a strong body of evidence that particles in ambient air have short-term effects on morbidity and mortality. Acute effects on morbidity are shown using register data on daily number of hospital admissions and emergency visits, as well as panel studies. Positive associations have been found rather consistently in studies using the regulated size fractions PM10 (particles less than 10 µm in aerodynamic diameter) and PM2.5. These epidemiological associations are usually considered to reflect causal links (Donaldsson et al, 2005). Experimental studies both in humans and in animals suggest that particles have pathogenic effects largely through inflammation. Particles worsen asthma symptoms and trigger exacerbations of chronic obstructive pulmonary disease (COPD). Studies of fluctuations in concentrations have also shown variation in peak flow and declines in lung function. Pulmonary inflammation is also believed to induce cardiovascular effects via several different pathways, including systemic inflammation, imbalance in coagulation factors resulting in thrombogenesis and interference in the control of heart rhythm that could lead to fatal dysrhythmia.

Fine (<2.5) and coarse fraction PM10 differ in sources and formation mechanisms. The major sources of coarse particles are windblown dust from soil, roads, building and construction activities. Coarse particles may also have a biological origin, as pollen, mould spores and parts of plants. Since many studies did not find an effect of coarse particles on mortality, particles from combustion are assumed to be more harmful (Brunekreef & Forsberg, 2005). 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).

Both fine and coarse PM10 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. One 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.

Population and Methods

Environmental data

Data on daily urban background concentrations of PM_{10}, nitrogen oxides (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 the same monitoring station have been used to represent fluctuations in particle and ozone levels in Stockholm in previous studies such as APHEA-2 (Kasouyanni et al, 2001; Gryparis et al, 2004). The rural (regional) background concentration of PM10 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. Ozone was measured using UV-absorption.

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 PM10 from road dust (PM_{resusp}) was calculated as:

$$PM_{resusp} = PM_{10urban} - PM_{10ruraladj} - 24/1000*(NO_{Xurban} - NO_{Xrural})$$

We needed to adjust the zero for $PM_{10rural}$ since there was a shift in the baseline for parts of the study period:

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

Health data

This analysis of 1998-2005 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.

Statistical Analysis

We modelled the data with an additative 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.

The regression models included smooth adjustment for time trend and season, relative humidity and temperature. In addition, day of the week, holidays and influenza (admissions) were adjusted for.

For road dust PM10 we used the approach used in APHEA2 and many other studies, and evaluated the association with the mean value of the same and previous day (lag01). In the final model we adjusted for rural PM10 and NOx and ozone from the urban station at Torkel Knutssonsgatan.

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, respectively.

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. Figure 1 shows the daily mean values of PM10 from road dust and the rural (regional background) PM10 concentration at Aspvreten.

Road dust PM10 had a very low correlation with 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).

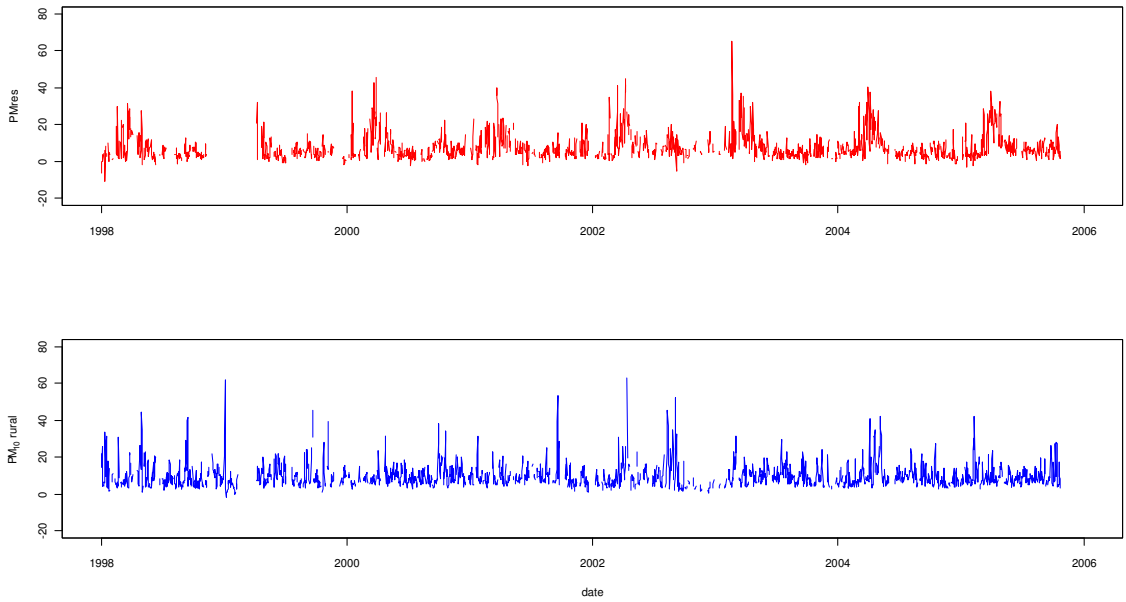

Figure 1. Daily mean values of PM10 (µg/m3) from road dust (upper curve) and the rural (regional background) PM10 concentration at Aspvreten (bottom curve).

Effects of road dust PM10 on admissions

The fluctuations in the concentration of road dust PM10 had a significant association with respiratory admissions. An increase of 10 µg/m3 of road dust PM10 (lag01) resulted in approximately 2.6 percent increase in admissions (95% CI: 0.3-4.6%).

In a sensitivity analysis road dust PM10 was studied as a smooth function, and the association appeared quite linear with no obvious threshold.

For cardiovascular hospital admissions there was no significant association with road dust PM10 concentrations. The estimated 95% CI for an increase of 10 µg/m3 of road dust PM10 (lag01) was -0.2 – 1.2%.

A sensitivity analysis of cardiovascular hospital admissions where road dust PM10 was studied as a smooth function did not support any non-linear dose-response relation.

The effects of road dust PM10 on hospital admissions were not sensitive for the inclusion of the other pollutants.

Discussion

A literature review (Brunekreef and Forsberg, 2005) concluded that the respiratory morbidity, such as admissions for asthma and COPD, was more strongly related to coarse particles that more often are of crustal origin. Cardiovascular effects were more often associated with fine particles, mainly originating from combustion. Experimental studies have shown 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). Interestingly, a recent study from Barcelona, Spain found an effect of coarse particles on daily mortality during Saharan dust days despite rather moderate concentrations of PM10 and PM2.5 (Perez et al, 2008).

Our findings are in line with most of the previous observations. We observed how an increase in the concentration of road dust PM10 is followed by a significant increase in respiratory admissions. An increase of 10 µg/m3 of road dust PM10 is estimated to result in 2.6 percent more acute hospital admissions for respiratory causes (95% CI: 0.3-4.6%).

At the same there was no significant effect of road dust PM10 on cardiovascular hospital admissions in our study. The lack of a significant association did not seem to be explained by non-linear relations.

Road dust has an effect on respiratory health, in this study represented by an increase in respiratory hospital admissions. It is very likely that other respiratory effects also occur.

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References

Brunekeef B, Forsberg B. 2005. Epidemiological evidence of effects of coarse airborne particles on health. Eur Respir J 26:309–318.

Castillejos M, Borja-Aburto VH, Dockery DW, Gold DR, Loomis, D. 2000. Airborne coarse particles and mortality. Inhal Toxicol 12:61–72.

Donaldson K, Mills N, MacNee W, Robinson S, Newby D. Role of inflammation in cardiopulmonary health effects of PM. Toxicol Appl Pharmacol. 2005;207(suppl 2):483–488.

Graff DW, Cascio WE, Rappold A, Zhou H, Huang YC, Devlin RB. 2009. Exposure to concentrated coarse air pollution particles causes mild cardiopulmonary effects in healthy young adults. Environ Health Perspect 117:1089-1094.

Gryparis A, Forsberg B, Katsouyanni K, Analitis A, Touloumi G, Schwartz J, et al. 2004. Acute effects of ozone on mortality from the "air pollution and health: a European approach" project. Am J Respir Crit Care Med 170:1080-1087.

Katsouyanni K, Touloumi G, Samoli E, Gryparis A, Le Tertre A, Monopolis Y, et al. 2001. Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 project. Epidemiology 12:521-531.

Mar TF, Norris GA, Koenig JQ, Larson TV. 2000. Associations between air pollution and mortality in Phoenix, 1995–1997. Environ Health Perspect 108:347–353.

Ostro BD, Broadwin R, Lipsett MJ. 2000. Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study. J Expo Anal Environ Epidemiol 10:412–419.

Perez L, Tobias A, Querol X, Künzli N, Pey J, Alastuey A, et al. 2008. Coarse particles from Saharan dust and daily mortality. Epidemiology 19: 800-807.

Schwarze PE, Øvrevik J, Hetland RB, Becher R, Cassee FR, Låg M, et al. 2007. Importance of size and composition of particles for effects on cells in vitro. Inhal Toxicol 19(Suppl 1): 17-22.

Steerenberg PA, van Amelsvoort L, Lovik M, Hetland RB, Alberg T, Halatek T, et al. 2006. Relation between sources of particulate air pollution and biological effect parameters in samples from four European cities: an exploratory study. Inhal Toxicol 18:333-346.