Estimated Short-Term Effects of Coarse Particles on Daily Mortality in Stockholm, Sweden

Kadri Meister,1 Christer Johansson,2,3 and Bertil Forsberg1

1Department of Public Health and Clinical Medicine, Occupational and Environmental Medicine, Umeå University, Umeå, Sweden; 2Department of Applied Environmental Science, Stockholm University, Stockholm, Sweden; 3Environment and Health Administration, Stockholm, Sweden

BACKGROUND: Although serious health effects associated with particulate matter (PM) with aerodynamic diameter ≤ 10 μm (PM10) or ≤ 2.5 μm (PM2.5) are well documented in many studies, the effects of coarse PM (PM2.5–10) are still under debate.

OBJECTIVE: In this study, we estimated the effects of short-term exposure of PM2.5–10 on daily mortality in Stockholm, Sweden.

METHOD: We collected data on daily mortality for the years 2000 through 2008. Concentrations of PM10, PM2.5, ozone, and carbon monoxide were measured simultaneously in central Stockholm. We used additive Poisson regression models to examine the association between daily mortality and PM2.5–10 on the day of death and the day before. Effect estimates were adjusted for other pollutants (two-pollutant models) during different seasons.

RESULTS: We estimated a 1.68% increase [95% confidence interval (CI): 0.20%, 3.15%] in daily mortality per 10 μg/m3 increase in PM2.5–10 (single-pollutant model). The association with PM2.5–10 was stronger for November through May, when road dust is most important (1.69% increase; 95% CI: 0.21%, 3.17%), compared with the rest of the year (1.31% increase; 95% CI: –2.08%, 4.70%), although the difference was not statistically significant. When adjusted for other pollutants, particularly PM2.5, the effect estimates per 10 μg/m3 for PM2.5–10 decreased slightly but were still higher than corresponding effect estimates for PM2.5.

CONCLUSIONS: Our analysis shows an increase in daily mortality associated with elevated urban background levels of PM2.5–10. Regulation of PM2.5–10 should be considered, along with actions to specifically reduce PM2.5–10 emissions, especially road dust suspension, in cities.

KEY WORDS: coarse particles, health effects, mortality, PM2.5, PM10, road dust, short-term exposure.

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Particle effects on mortality. Hundreds of epidemiological studies have shown that the ambient particulate air pollution is associated with daily mortality, generally studied using the concentration of particulate matter (PM) with an aerodynamic diameter ≤ 10 μm (PM10) or fine PM with aerodynamic diameter of ≤ 2.5 μm (PM2.5) (Samoli et al. 2008). The effect of coarse PM (PM2.5–10) on mortality has been less studied. In their review article, Brunekreef and Forberg (2005) concluded that most published mortality studies that applied two-pollutant models were unable to demonstrate independent PM2.5–10 effects on mortality after adjusting for PM2.5. However, PM2.5–10 levels are expected to be more spatially heterogeneous than are PM2.5 levels, which increase exposure misclassification when one or a few monitors provide exposure data (Monn 2001). Moreover, most time-series studies that have reported significant effects on mortality associated with PM2.5–10 were conducted in arid areas, including such places as Phoenix, Arizona (Mar et al. 2000), Coachella Valley, California (Ostro et al. 2000), and Mexico City (Castillejos et al. 2000). In arid areas, particle dust often originates from the surrounding land, not from local point sources, and particle levels are therefore expected to be more spatially homogeneous.

In a more recent study, Malig and Ostro (2009) used data from 15 counties in California and found an association between PM2.5–10 and daily mortality (both all-cause and cardiovascular mortality), particularly among demographic subgroups of lower socioeconomic status. In their study, only those participants who resided close to an air pollution monitor were included in the study in order to reduce exposure misclassification. Adjusting for PM2.5 had no effect on the effect estimates for PM2.5–10, likely due to its low correlation with PM2.5–10 and daily mortality that persisted after adjusting for PM2.5 (Zanobetti and Schwartz 2009). Recent studies from southern Europe have explored the effects of windblown Saharan dust, including a study conducted in Barcelona, Spain, that found evidence of an effect of PM2.5–10 on daily mortality during Saharan dust days, despite rather moderate particle concentrations (Perez et al. 2008).

European toxicological studies have indicated that PM2.5–10 has the same toxicological potential as PM2.5 on a mass basis (Gerlofs-Nijland et al. 2007; Sandström et al. 2005). It also has been suggested that particles of crustal origin are associated with markers of inflammation and acute toxicity in bioassays (Steerenberg et al. 2006). A cluster of European in vitro studies have shown that for mineral particles the composition and surface reactivity appeared to be most important for the proinflammatory potential of the particles (Schwarze et al. 2007).

PM2.5–10 sources and its importance for PM10. A directive from the European Union (EU; European Commission 2008) regulates the total mass of all PM10 irrespective of size, morphology, chemistry, and health effects. In the urban environment, different sources contribute differently to total PM10 because of variation in the size distribution of the emitted particles (Johansson et al. 2007). At roadside locations, most traffic exhaust particles are 10–30 nm in diameter, which is too small to result in a large aerosol mass, even when number concentrations are high (Gidhagen et al. 2004). Samples collected in Berlin showed that about 45% of local traffic contributions to roadside PM10 concentrations were due to suspended soil material, and the remaining traffic contribution was due to vehicle exhaust and tire abrasion (Lenschow et al. 2001). Likewise, about 50% of PM10 during summer months in Birmingham, United Kingdom, was due to PM2.5–10 (Harrison et al. 1997). In northern Europe, PM2.5–10 concentrations are generally elevated during winter and spring because of the use of studded tires, road salt, and traction sand. In Stockholm, road wear increases drastically because of the use of studded winter tires and traction sand on streets, such that up to 90% of the locally emitted PM10 during the winter may be due to road abrasion (Johansson et al. 2007). Suspension of road dust is a major contributor to PM2.5–10 and to the exceedances of the EU limit values for PM10 in Stockholm (Norman and Johansson 2006).

The aim of this study was to assess the effect of PM2.5–10 on daily mortality in Stockholm.

Address correspondence to K. Meister, Department of Public Health and Clinical Medicine, Occupational and Environmental Medicine, Umeå University, Umeå, Sweden. Telephone: 46 90 785 2363. Fax: 46 90 785 2456. E-mail: kadri.meister@enmed.umu.se

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Material and Methods

Health data. This study of the greater Stockholm area (population ~1.3 million) was based on daily counts of deaths excluding external causes [International Classification of Diseases, 10th Revision (ICD-10) codes A00 through R99; World Health Organization 2007], for the years 2000 through 2008 from the Cause of Death Register at the Swedish National Board of Health and Welfare (Stockholm, Sweden).

Environmental data. Data on daily urban background concentrations of PM10, PM2.5, ozone (O3), and carbon monoxide (CO) were obtained from the Environment and Health Administration of Stockholm (2011). PM10, PM2.5, and O3 were measured in central Stockholm at Torkel Knutssonsgatan, a monitoring station located at rooftop level (at a height of 25 m) not directly affected by nearby emissions (Johansson et al. 2007). Measurements from the same monitoring station have been used to represent fluctuations in particle and O3 levels in Stockholm in previous studies, such as APHEA 2 (Air Pollution and Health: A European Approach; Gryparis et al. 2004; Le Terre et al. 2002).

The mass concentrations of PM10 and PM2.5 were measured using tapered element oscillating microbalance (TEOM 1400i; Thermo Fisher Scientific, East Greenbush, NY, USA). To account for losses of volatile material in the PM, all data were corrected following Areskoug (2007). Continuous measurement of O3 was based on its absorption of ultraviolet light (UV Absorption Ozone Analyzer, model 42M; Environnement S.A., Poissy, France). The urban background CO concentrations were based on continuous measurements of two rooftop stations (Hornsagan and Sveavagen, both at a height of 25 m) in central Stockholm. The instruments were based on a nondispersive infrared technique (Carbon Monoxide Analyzer, model 48; Thermo Environmental Instrument Inc., Franklin, MA, USA). The coarse fraction of PM10 (PM2.5–10) is based on the difference between PM10 and PM2.5.

The contribution of road dust to the particle concentrations varies with the wetness of the road surfaces (Norman and Johansson 2006) and is not correlated with exhaust particles (Johansson et al. 2007). The number of days with high PM2.5–10 levels therefore depends on the meteorological conditions, especially during the late winter and spring. Therefore, we adjusted for meteorological data that was collected from the Swedish Meteorological and Hydrological Institute. Daily temperature and relative humidity were measured at Bromma Airport, a city airport, 9 km from Stockholm city center.

Statistical methods. We studied the association between daily mortality and PM2.5–10 concentrations averaged over the day of death and the day before death (lag01) with a timeseries analysis. The exposure lag1 has been commonly used when effects of air pollution on mortality have been studied (Gryparis et al. 2004; Katsouyanni et al. 2001; Samoli et al. 2007). Time-series analysis allows estimation of relatively small acute effects in large study populations.

We applied additive Poisson regression models, controlling for long-term trend using a smooth function with eight degrees of freedom per year, and for day of the week and public holidays using indicator variables. We controlled for the effect of weather by adjusting for the current day’s temperature and relative humidity, together with smooth functions of mean temperature and relative humidity over the previous 2 days (each using six degrees of freedom). Influenza episodes were controlled by modeling the daily influenza hospital admissions as a smooth function. All influenza hospital admissions in Sweden were obtained from the Patient Register at the Swedish National Board of Health and Welfare (Stockholm, Sweden). Each of the smooth functions in

Table 1. Summary of daily air pollution and meteorological data.

| Variable          | Season  | Mean ± SD      | IQR     | Maximum |
|-------------------|---------|----------------|---------|---------|
| PM10 (µg/m³)      | Overall | 15.5 ± 9.6     | 9.4     | 95.2    |
|                   | Nov–May | 17.0 ± 10.8    | 11.9    | 95.2    |
|                   | Jun–Oct | 13.5 ± 7.0     | 6.4     | 67.0    |
| PM2.5 (µg/m³)     | Overall | 8.6 ± 5.7      | 4.9     | 46.2    |
|                   | Nov–May | 9.2 ± 6.1      | 5.3     | 46.1    |
|                   | Jun–Oct | 8.2 ± 5.0      | 4.5     | 43.0    |
| PM2.5–10 (µg/m³)  | Overall | 7.1 ± 6.4      | 5.4     | 61.9    |
|                   | Nov–May | 8.3 ± 7.8      | 8.0     | 61.9    |
|                   | Jun–Oct | 5.5 ± 3.2      | 3.5     | 36.7    |
| PM2.5–10/PM10²    | Overall | 0.44 ± 0.20    | 0.31    | 0.93    |
|                   | Nov–May | 0.45 ± 0.13    | 0.18    | 0.81    |
|                   | Jun–Oct | 0.25 ± 0.12    | 0.20    | 0.99    |
| CO (µg/m³)        | Overall | 281 ± 95       | 109     | 812     |
|                   | Nov–May | 300 ± 95       | 109     | 812     |
|                   | Jun–Oct | 254 ± 78       | 95      | 612     |
| O3 (µg/m³)        | Overall | 60.0 ± 22.4    | 31.7    | 142.0   |
|                   | Nov–May | 57.8 ± 24.0    | 35.9    | 142.0   |
|                   | Jun–Oct | 63.1 ± 19.5    | 26.0    | 126.6   |
| Temperature (%)   | Overall | 7.7 ± 8.0      | 12.7    | 26.2    |
|                   | Nov–May | 2.8 ± 5.9      | 7.6     | 19.0    |
|                   | Jun–Oct | 14.4 ± 5.0     | 6.6     | 26.2    |
| Relative humidity (%) | Overall | 0.75 ± 0.13 | 0.20    | 0.99    |
|                   | Nov–May | 0.77 ± 0.14    | 0.20    | 0.99    |
|                   | Jun–Oct | 0.73 ± 0.12    | 0.17    | 0.97    |

*Fraction of PM10 that is PM1.0

Figure 1. Seasonal variation in PM2.5–10 concentrations in Stockholm, Sweden, over the study period, 2000 through 2008.
the model was represented using penalized regression splines.

We modeled 24-hr average concentrations of PM$_{2.5-10}$, PM$_{2.5}$, and CO and the maximum of 8-hr moving-average (between 0600 hours and 2200 hours) concentrations of O$_3$ on the same day and the previous day (lag01). Results are reported for single-pollutant models (adjusted for time trend, day of the week, public holidays, temperature, humidity, and influenza outbreaks) and for multipollutant models (including two pollutants in the same model, in addition to the covariates listed above). Results also are reported for a 10-μg/m$^3$ increase as well as for an interquartile range (IQR) increase in each pollutant.

The analysis was stratified by period because the composition of particles varies seasonally. In Sweden, passenger cars, light-weight trucks, and light-weight buses are required to have winter tires from 1 December to 31 March. Heavy vehicles are not required to use winter tires. Winter tires can be studded or nonstudded, but studded winter tires were allowed from 1 October to 30 April during the study period 2000 through 2008 and were used by 70–75% of vehicles in Stockholm during those years. The share of studded winter tires usually increases from zero in September through October to its maximum in December through March and then falls back to zero in May (Norman and Johansson 2006), depending on weather and road conditions. Although studded tires are banned after 1 May, road dust remains elevated in each pollutant.

To test the hypothesis that PM$_{2.5-10}$ may affect mortality with a longer lag than lag01, we also fitted a distributed lag model for up to 30 days (interval 0.05 and 0.10 to define borderline significance (corresponding p-values = 0.06, 0.10, and 0.05, respectively).

The effect estimate for a 10-μg/m$^3$ increase in PM$_{2.5-10}$ (1.33%; 95% CI: –0.26%, 2.92%; p = 0.10) was higher than the effect estimate for a 10-μg/m$^3$ increase in PM$_{2.5}$ (0.90%; 95% CI: –0.62%, 2.41%; p = 0.25) when both pollutants were included in the same model. In addition, the estimated percent change in daily mortality for an IQR increase was larger for PM$_{2.5-10}$ (5.2 μg/m$^3$) than for PM$_{2.5}$ (4.7 μg/m$^3$; Table 3).

The smooth function of PM$_{2.5-10}$ (lag01) from the single-pollutant model, adjusted for the covariates listed above (Figure 2), suggests that the more precisely estimated part of the curve does not deviate from linearity.

We estimated a 1.69% increase (95% CI: 0.21%, 3.17%; p = 0.025) in daily mortality per 10-μg/m$^3$ increase in PM$_{2.5-10}$ for the period November through May (Table 4).

The effect estimate for the reference time period was lower (1.31%; 95% CI: –2.08%, 4.70%), but the difference between estimates was not statistically significant (p = 0.81).

After adjusting for other pollutants, there were only minor changes in the effect estimates for PM$_{2.5-10}$ for the period November through May, and the magnitudes of the changes are shown in Table 2.

### Table 2. Correlation coefficients between variables in the study.

| Pollutant | Season | PM$_{2.5}$ | PM$_{2.5-10}$ | CO | O$_3$ |
|-----------|--------|------------|---------------|----|-------|
| PM$_{2.5}$ | Overall | 0.273 | 0.229 | 0.475 |
| PM$_{2.5-10}$ | Nov–May | 0.515 | 0.031 |
| CO | Overall | 0.522 | 0.126 |
| O$_3$ | Overall | 0.209 | 0.387 |
| Temperature | Overall | 0.050 | –0.030 | –0.257 |
| Relative humidity | Overall | –0.006 | –0.418 | 0.278 |

### Table 3. Mortality and PM$_{2.5-10}$ association for lag01: overall estimates.

| Model type | Pollutant | Percent increase per 10 μg/m$^3$ (95% CI) | Percent increase per IQR (95% CI)$^a$ |
|------------|-----------|------------------------------------------|---------------------------------------|
| Single pollutant | PM$_{2.5-10}$ | 1.68 (0.20, 3.15) | 0.88 (0.11, 1.64) |
| Two pollutant | PM$_{2.5-10}$ + PM$_{2.5}$ | 1.33 (–0.26, 2.92) | 0.69 (–0.13, 1.52) |

All models adjusted for time trend, day of the week, public holidays, temperature, humidity, and influenza outbreaks.

$p$-values: PM$_{2.5-10}$, 5.2 μg/m$^3$; PM$_{2.5}$, 4.7 μg/m$^3$; O$_3$, 30.5 μg/m$^3$; CO, 100 μg/m$^3$. |
The smooth function of the relationship between PM$_{2.5-10}$ (lag01) and daily mortality from the influenza outbreaks. The shaded area represents 95% CI.

When we examined the distributed lag model with 6 lag days for PM$_{2.5-10}$, we found the largest coefficient for a 1-day lag and little evidence of mortality effects at longer lags (Figure 3). The association between mortality and the sum of the distributed lag (1.12%; 95% CI: –0.32%, 3.11%) was somewhat lower than the results for lag01 (1.68%; 95% CI: 0.20%, 3.15%).

**Discussion**

The daily mean concentrations of PM$_{2.5-10}$ are highest during late winter and spring, presumably due to increased suspension of road dust particles during dry road conditions (Ketzel et al. 2007; Omstedt et al. 2005). This is mainly because of the wear of stone materials in the asphalt by studded winter tires (Hussein et al. 2008; Omstedt et al. 2005).

We estimated a 1.7% increase in daily mortality per 10-µg/m$^3$ increase in lag01 PM$_{2.5-10}$ both for the whole year and during November through May, the high road dust period. This is a larger estimated effect than typically reported for PM$_{10}$, for example, 0.6% (95% CI: 0.4%, 0.8%) per 10-µg/m$^3$ increase in PM$_{10}$ in the European APHEA 2 study (Katsouyanni et al. 2001). The effect estimates for O$_3$ and CO were similar to those reported for APHEA 2 (Gryparis et al. 2004; Samoli et al. 2007).

When data were split into two different time periods, the estimated effect associated with PM$_{2.5-10}$ was higher during November through May, the road dust period, consistent with recent findings reported for PM$_{2.5-10}$ and PM$_{10}$ that indicated stronger associations during springtime (Zanobetti and Schwartz 2009; Zeka et al. 2006). Seasonal variation in associations could reflect greater indoor penetration during months when windows are open (Zeka et al. 2006) or seasonal variation in the composition of PM from different sources. In our case, the seasonal difference is not likely explained by indoor penetration, because associations were stronger during the winter, when windows are likely to be closed.

Detailed chemical analyses of sampled PM$_{2.5-10}$ during that period have shown that the PM is dominated by quartzite, which was the most common stone mineral in the pavements in Stockholm (Furusjö et al. 2007; Sjödin et al. 2010).

We did not directly monitor PM$_{2.5-10}$ but estimated its concentration as the difference between PM$_{10}$ and PM$_{2.5}$. This means that part of the variability in the concentration of PM$_{2.5-10}$ is due to the measurement errors in both PM$_{10}$ and PM$_{2.5}$. Comparison with a gravimetric method has shown that the relative uncertainty of the determination of PM$_{10}$ (according to the EU guidance for demonstrating equivalence of ambient air monitoring methods [European Commission Working Group on Particulate Matter 2002]) in Stockholm using the TEOM instrument is between 11% and 27% at the daily limit value.
Road dust is an important traffic-related pollutant, because road wear particles contribute substantially to local particle emissions in cities. In cities where studded tires are used, road dust may cause violations of limit values for PM$_{10}$. The effect of PM$_{2.5–10}$ on mortality has been questioned because of many inconsistent findings when controlling for PM$_{2.5}$ (Brennkeft and Forsberg 2005). This has influenced discussions on limit values and abatement strategies. Several recent studies (Malig and Ostro 2009; Perez et al. 2008; Samoli et al. 2011; Zanobetti and Schwartz 2009) have, like the present one, produced evidence of a short-term effect of PM$_{2.5–10}$ and crustal PM$_{10}$ (not originating from combustion processes) on mortality. Results regarding the effect modification of Saharan dust days on a PM$_{10}$-mortality relationship are inconsistent, despite positive associations, with negative effects and no interaction effects also reported. These inconsistent findings could reflect differences in the composition of other PM$_{10}$ fractions, but also differences in correlations with other pollutants.

### Conclusions

Given our results on road dust and other recent findings showing an impact of PM$_{2.5–10}$ on daily mortality in studies of U.S. cities (Malig and Ostro 2009; Zanobetti and Schwartz 2009) and desert dust on daily mortality in Barcelona (Perez et al. 2008), it seems appropriate to separately regulate and control PM$_{2.5–10}$. One must keep in mind that a large proportion of PM$_{2.5}$ in many cities is transported over long distances and is difficult to avoid, whereas PM$_{2.5–10}$ as in Stockholm, may be largely of local origin. Therefore, it also may be easier to improve health by reducing exposures to PM$_{2.5–10}$.

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