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

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Background: Although serious health effects associated with particulate matter (PM) with aerodynamic diameter ≤ 10 μm (PM_{10}) and ≤ 2.5 μm ($PM_{2.5}$; fine fraction) are documented in many studies, the effects of coarse PM ($PM_{2.5–10}$) are still under debate.

Objective: In this study, we estimated the effects of short-term exposure of $PM_{2.5–10}$ on daily mortality in Stockholm, Sweden.

Method: We collected data on daily mortality for the years 2000 through 2008. Concentrations of PM_{10}, $PM_{2.5}$, ozone, and carbon monoxide were measured simultaneously in central Stockholm. We used additive Poisson regression models to examine the association between daily mortality and $PM_{2.5–10}$ 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/m^3 increase in $PM_{2.5–10}$ (single-pollutant model). The association with $PM_{2.5–10}$ 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 $PM_{2.5}$, the effect estimates per 10 μg/m^3 for $PM_{2.5–10}$ decreased slightly but were still higher than corresponding effect estimates for $PM_{2.5}$.

Conclusions: Our analysis shows an increase in daily mortality associated with elevated urban background levels of $PM_{2.5–10}$. Regulation of $PM_{2.5–10}$ should be considered, along with actions to specifically reduce $PM_{2.5–10}$ emissions, especially road dust suspension, in cities.

Key words: coarse particles, health effects, mortality, $PM_{2.5}$, PM_{10}, road dust, short-term exposure. *Environ Health Perspect* 120:431–436 (2012). http://dx.doi.org/10.1289/ehp.1103995 [Online 19 December 2011]

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 (PM_{10}) or fine PM with aerodynamic diameter of ≤ 2.5 μm ($PM_{2.5}$) (Samoli et al. 2008). The effect of coarse PM ($PM_{2.5–10}$) on mortality has been less studied. In their review article, Brunekreef and Forsberg (2005) concluded that most published mortality studies that applied two-pollutant models were unable to demonstrate independent $PM_{2.5–10}$ effects on mortality after adjusting for $PM_{2.5}$. However, $PM_{2.5–10}$ levels are expected to be more spatially heterogeneous than are $PM_{2.5}$ 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 $PM_{2.5–10}$ 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 $PM_{2.5–10}$ 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 $PM_{2.5}$ had no effect on the effect estimates for $PM_{2.5–10}$, likely due to its low correlation with $PM_{2.5–10}$ in these California counties. An even larger study of U.S. cities found an association between $PM_{2.5–10}$ and daily mortality that persisted after adjusting for $PM_{2.5}$ (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 $PM_{2.5–10}$ on daily mortality during Saharan dust days, despite rather moderate particle concentrations (Perez et al. 2008).

European toxicological studies have indicated that $PM_{2.5–10}$ has the same toxicological potential as $PM_{2.5}$ 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).

$PM_{2.5–10}$ sources and its importance for PM_{10}. A directive from the European Union (EU; European Commission 2008) regulates the total mass of all PM_{10} irrespective of size, morphology, chemistry, and health effects. In the urban environment, different sources contribute differently to total PM_{10} 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 PM_{10} 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 PM_{10} during summer months in Birmingham, United Kingdom, was due to $PM_{2.5–10}$ (Harrison et al. 1997). In northern Europe, $PM_{2.5–10}$ 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 PM_{10} during the winter may be due to road abrasion (Johansson et al. 2007). Suspension of road dust is a major contributor to $PM_{2.5–10}$ and to the exceedances of the EU limit values for PM_{10} in Stockholm (Norman and Johansson 2006).

The aim of this study was to assess the effect of $PM_{2.5–10}$ on daily mortality in Stockholm.

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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 PM_{10}, $PM_{2.5}$, ozone (O_3), and carbon monoxide (CO) were obtained from the Environment and Health Administration of Stockholm (2011). PM_{10}, $PM_{2.5}$, and O_3 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 O_3 levels in Stockholm in previous studies, such as APHEA 2 (Air Pollution and Health: A European Approach; Gryparis et al. 2004; Le Tertre et al. 2002).

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

Variable	Season	Mean ± SD	IQR	Maximum
PM_{10} (μg/m^3)	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
$PM_{2.5}$ (μg/m^3)	Overall	8.6 ± 5.7	4.9	46.2
	Nov–May	8.9 ± 6.1	5.3	46.2
	Jun–Oct	8.2 ± 5.0	4.5	43.0
$PM_{2.5–10}$ (μg/m^3)	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
$PM_{2.5–10}/PM_{10}$[a]	Overall	0.42 ± 0.18	0.25	0.93
	Nov–May	0.44 ± 0.20	0.31	0.93
	Jun–Oct	0.40 ± 0.13	0.18	0.81
CO (μg/m^3)	Overall	281 ± 85	108	812
	Nov–May	300 ± 85	109	812
	Jun–Oct	254 ± 78	95	612
O_3 (μg/m^3)	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 (°C)	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

[a]Fraction of PM_{10} that is $PM_{2.5–10}$.

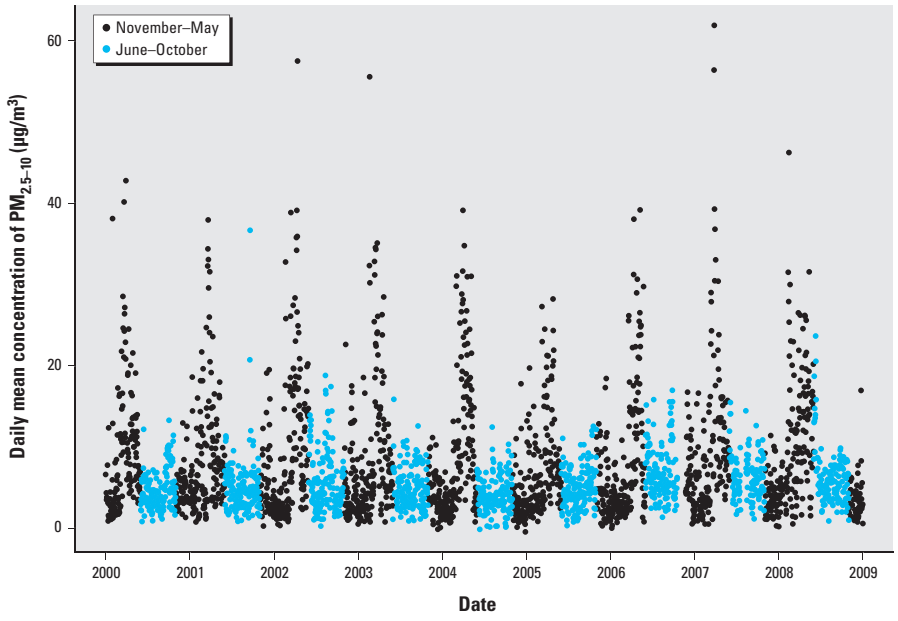

Figure 1. Seasonal variation in $PM_{2.5–10}$ concentrations in Stockholm, Sweden, over the study period, 2000 through 2008.

The mass concentrations of PM_{10} and $PM_{2.5}$ were measured using tapered element oscillating microbalance (TEOM 14001; 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 O_3 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 (Hornsgatan and Sveavägen, 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 PM_{10} ($PM_{2.5–10}$) is based on the difference between PM_{10} and $PM_{2.5}$.

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 $PM_{2.5–10}$ levels therefore depends on the meteorological conditions, especially during the late winter and spring. Therefore, we adjusted for meterological data that was collected from the Swedish Meterological 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 $PM_{2.5–10}$ concentrations averaged over the day of death and the day before death (lag01) with a time-series analysis. The exposure lag01 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

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 because of the accumulation of PM on the road surface during the winter (Ketzel et al. 2007; Norman and Johansson 2006). Therefore, we selected November through May as the period of interest for effects of studded tires ("road dust period," which results in in high concentrations of $PM_{2.5–10}$) and June through October as the reference season.

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 7 days (same day and up to 6 days earlier) for $PM_{2.5–10}$.

We used p-values < 0.05 to define statistically significant effect estimates and p-values between 0.05 and 0.10 to define borderline significant effect estimates. Data were analyzed using the mgcv package in R (version 2.11.1; R Core Development Team 2010).

Results

There were 93,398 deaths (excluding deaths due to external causes) during the 2000 through 2008 study period (3,285 days). On average, there were 28.4 deaths per day (range, 12–52). The average number of deaths per day for the road dust period (November through May) was 29.6 (range, 13–52), and for the reference season (June through October) was 26.8 (range, 12–50). Days with missing values for environmental variables during the study period were not included, most often because of missing $PM_{2.5}$ (260 days) and PM_{10} data (106 days) that did not allow calculation of $PM_{2.5–10}$. In total, 2,789 days were included in the analysis. A statistical summary of the air pollution measurements for the whole period (2000 through 2008) is presented in Table 1.

Figure 1 shows that the daily mean concentrations of $PM_{2.5–10}$ are highest during late winter and spring. For $PM_{2.5–10}$, 148 days with a daily mean concentration of 20 μg/m^3 (95th percentile) or higher were observed within the road dust period, but only 4 days outside that season (Figure 1). On average, $PM_{2.5–10}$ is somewhat less than half (42%) of the total PM_{10} concentration. $PM_{2.5–10}$ contributes 44% on average in November through May and 40% in June through October (Table 1).

The correlations between $PM_{2.5–10}$ and the other pollutants are given in Table 2. We found a 1.68% increase [95% confidence interval (CI): 0.20%, 3.15%; p = 0.026] in daily mortality per 10-μg/m^3 increase in $PM_{2.5–10}$ (lag01) based on the single-pollutant model (Table 3). Adjusting for O_3, $PM_{2.5}$, or CO resulted in only minor decreases in effect estimates for $PM_{2.5–10}$, with 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

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				
	Nov–May				
	Jun–Oct				
$PM_{2.5–10}$	Overall	0.273			
	Nov–May	0.229			
	Jun–Oct	0.475			
CO	Overall	0.522	0.126		
	Nov–May	0.515	0.031		
	Jun–Oct	0.546	0.239		
O_3	Overall	0.209	0.387	–0.192	
	Nov–May	0.187	0.478	–0.259	
	Jun–Oct	0.287	0.293	–0.003	
Temperature	Overall	0.050	–0.003	–0.257	0.435
	Nov–May	0.079	0.244	–0.172	0.441
	Jun–Oct	0.260	0.177	0.036	0.663
Relative humidity	Overall	–0.006	–0.418	0.278	–0.729
	Nov–May	–0.055	–0.569	0.248	–0.777
	Jun–Oct	0.054	–0.212	0.251	–0.630

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			
$PM_{2.5–10}$	$PM_{2.5–10}$	1.68 (0.20, 3.15)	0.88 (0.11, 1.64)
$PM_{2.5}$	$PM_{2.5}$	1.46 (0.07, 2.84)	0.68 (0.03, 1.33)
Two pollutant			
$PM_{2.5–10}$ + $PM_{2.5}$	$PM_{2.5–10}$	1.33 (–0.26, 2.92)	0.69 (–0.13, 1.52)
	$PM_{2.5}$	0.90 (–0.62, 2.41)	0.42 (–0.29, 1.13)
$PM_{2.5–10}$ + O_3	$PM_{2.5–10}$	1.47 (–0.07, 3.00)	0.77 (–0.04, 1.57)
	O_3	0.31 (–0.32, 0.93)	0.94 (–0.98, 2.85)
$PM_{2.5–10}$ + CO	$PM_{2.5–10}$	1.53 (–0.02, 3.09)	0.80 (–0.01, 1.61)
	CO	0.04 (–0.09, 0.16)	0.37 (–0.87, 1.60)

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

estimates were similar to the corresponding estimates for the whole year (Table 4).

Also, for the reference period June through October, changes in effect estimates were small after adjustments for CO and O_3. However, no indication of an effect of $PM_{2.5–10}$ was found when $PM_{2.5}$ was adjusted for. The larger change in the estimated effect of $PM_{2.5–10}$ may be explained by the larger effect estimate for $PM_{2.5}$ and the higher correlation between $PM_{2.5}$ and $PM_{2.5–10}$ during the reference period (correlation coefficient r = 0.47) compared with the road dust period (r = 0.23; Table 2).

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: –0.83%, 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. However, the chemical composition is likely important, because PM produced during the winter period is dominated by stone minerals from road dust.

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

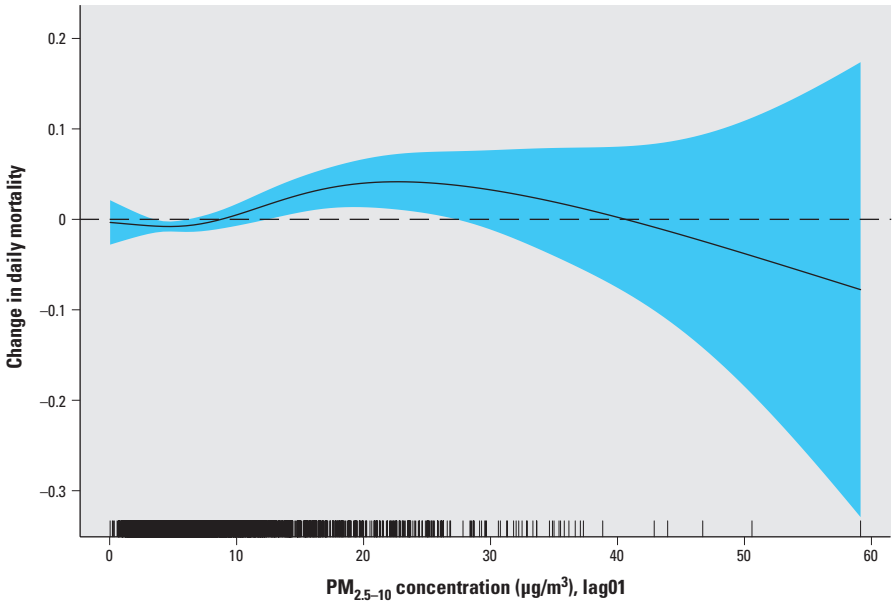

Figure 2. The smooth function of the relationship between $PM_{2.5–10}$ (lag01) and daily mortality from the single-pollutant model, adjusted for time trend, day of the week, public holidays, temperature, humidity, and influenza outbreaks. The shaded area represents 95% CI.

Table 4. Mortality and $PM_{2.5–10}$ association for lag01: by season.

Model type	Pollutant (season)	Percent increase per 10 μg/m^3 (95% CI)	Percent increase per IQR (95% CI)[a]
Single pollutant			
$PM_{2.5–10}$	$PM_{2.5–10}$ (Nov–May)	1.69 (0.21, 3.17)	1.37 (0.17, 2.57)
	$PM_{2.5–10}$ (Jun–Oct)	1.31 (–2.08, 4.70)	0.41 (–0.65, 1.46)
Two pollutant			
$PM_{2.5–10}$ + $PM_{2.5}$	$PM_{2.5–10}$ (Nov–May)	1.38 (–0.21, 2.97)	1.12 (–0.17, 2.41)
	$PM_{2.5–10}$ (Jun–Oct)	–0.28 (–5.06, 4.50)	–0.09 (–1.57, 1.40)
	$PM_{2.5}$ (Nov–May)	0.72 (–0.92, 2.37)	0.37 (–0.48, 1.22)
	$PM_{2.5}$ (Jun–Oct)	1.79 (–1.30, 4.88)	0.75 (–0.55, 2.05)
$PM_{2.5–10}$ + O_3	$PM_{2.5–10}$ (Nov–May)	1.48 (–0.07, 3.02)	1.20 (–0.05, 2.45)
	$PM_{2.5–10}$ (Jun–Oct)	1.26 (–2.13, 4.65)	0.39 (–0.66, 1.45)
	O_3	0.30 (–0.33, 0.93)	0.92 (–1.01, 2.85)
$PM_{2.5–10}$ + CO	$PM_{2.5–10}$ (Nov–May)	1.55 (–0.01, 3.11)	1.26 (–0.01, 2.52)
	$PM_{2.5–10}$ (Jun–Oct)	1.17 (–2.25, 4.59)	0.37 (–0.70, 1.43)
	CO	0.04 (–0.09, 0.16)	0.36 (–0.87, 1.59)

All models adjusted for time trend, day of the week, public holidays, temperature, humidity, and influenza outbreaks.
[a]IQR values: $PM_{2.5–10}$, 8.1 μg/m^3 in November through May, 3.1 μg/m^3 in June through October; $PM_{2.5}$, 5.2 μg/m^3 in November through May, 4.2 μg/m^3 in June through October; O_3, 30.5 μg/m^3; CO, 100 μg/m^3.

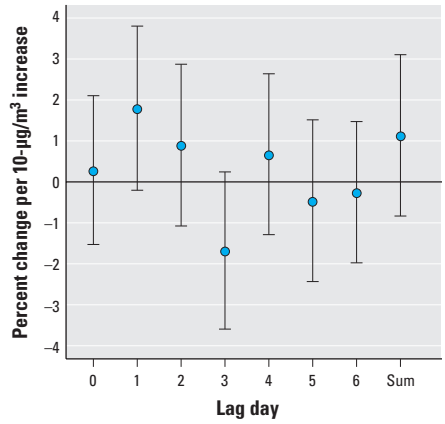

Figure 3. Percent change (95% CI) in daily mortality per 10-μg/m^3 increase in $PM_{2.5–10}$ for the 7-day distributed lag model.

(50 μg/m^3). The uncertainty of $PM_{2.5–10}$ has not been determined, but most of the uncertainty in PM_{10} is likely due to the correction for volatile PM, which is mainly present in $PM_{2.5}$ fraction and not in the $PM_{2.5–10}$ fraction.

A strength of the present study is that $PM_{2.5–10}$ during the road dust period originates from the road network covering the whole city, not from a few point sources. Thus, in Stockholm, where the main contributor to $PM_{2.5–10}$ is road traffic, the urban background daily mean concentrations in different parts of the city should fluctuate in a similar way.

The present study and other recent studies for which exposure can be expected to be spatially homogeneous support the hypothesis that there is a short-term effect on mortality, and it is at least as strong as typically found for PM_{10}. Other recent studies also have estimated large effects on daily mortality of crustal $PM_{2.5–10}$ (Malig and Ostro 2009; Perez et al. 2008; Zanobetti and Schwartz 2009).

There is support for cardiopulmonary effects of $PM_{2.5–10}$ from recent human experimental studies. When 14 healthy young volunteers were exposed to concentrated ambient $PM_{2.5–10}$ (2 hr; mean, 89 μg/m^3) and filtered air in a single-blind, crossover study, exposure produced a mild pulmonary inflammation (Graff et al. 2009) 20 hr after exposure; a decrease in blood tissue plasminogen activator, which is involved in fibrinolysis; and a decrease in heart rate variability, which indicates an effect on the autonomic nervous system. Reduced heart rate variability is a prognostic marker for the development of cardiac arrhythmias (van Boven et al. 1998).

In a Swedish experimental study, Gustafsson et al. (2008) generated particles from the wear of studded tires on two pavements and traction sanding using a road simulator. A chemical analysis showed that the generated wear particles consisted almost entirely of minerals from the pavement stone material. It is well known that silica, which is part of this stone material, is capable of producing reactive oxygen species, either directly on the particle surface or by cells in response to exposure (Hamilton et al. 2008). In a recent study, Karlsson et al. (2011) examined the toxicoproteomic effects on human monocyte-derived macrophages after exposure to wear particles generated from the interface of studded tires and a granite-containing pavement. They showed that overall, proteins associated with inflammatory response were increased and proteins involved in cellular functions such as redox balance, antiinflammatory response, and glycolysis were decreased. Activation of the inflammatory pathway is one potential explanation for cardiopulmonary effects associated with exposure to mineral particles.

Road dust is an important traffic-related pollutant, because road wear particles contributes 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 $PM_{2.5}$ (Brunekreef 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; Zauli Sajani et al. 2010) 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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