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Particulate Air Pollution and Acute Cardiorespiratory Hospital Admissions and Mortality Among the Elderly

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Background: It is known that particulate air pollution affects cardiorespiratory health; however, it is unclear which particle size fractions and sources of particles are responsible for the health effects.

Methods: Daily levels of nucleation (<0.03 μm), Aitken (0.03–0.1 μm), accumulation (0.1–0.29 μm), and coarse mode (2.5–10 μm) particles, particles with diameter <2.5 μm ($PM_{2.5}$), and gaseous pollutants were measured at central outdoor measurement sites in Helsinki, Finland between 1998 and 2004. We determined the associations of particles with daily cardiorespiratory mortality and acute hospital admissions among the elderly (≥65 years). For the analyses we used Poisson generalized additive models and for the source apportionment of $PM_{2.5}$ we used the EPA positive matrix factorization method.

Results: There was a suggestion of an association of hospital admissions for arrhythmia with Aitken mode particles and $PM_{2.5}$ from traffic. Otherwise few associations were observed between various sizes and types of particles and either cardiovascular admissions or mortality. In contrast, most particle fractions had positive associations with admissions for pneumonia and asthma-chronic obstructive pulmonary disease (COPD). The strongest and most consistent associations were found for accumulation mode particles (3.1%; 95% confidence interval = 0.43–5.8 for pneumonia over the 5-day mean, and 3.8%; 1.3–6.3 for asthma-COPD at lag 0, for an interquartile increase in particles). We also found a positive association of respiratory mortality mainly with accumulation mode particles (5.1%; 1.2–9.0 at lag 0).

Conclusions: All particle fractions including Aitken, accumulation, and coarse mode had especially adverse respiratory health effects among the elderly. Overall associations were stronger for respiratory than for cardiovascular outcomes.

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Associations of ambient PM_{10} (particles with diameter <10 μm) and $PM_{2.5}$ (<2.5 μm) with cardiorespiratory endpoints such as all cardiovascular and respiratory mortality and pneumonia and ischemic heart disease hospitalizations have been observed in numerous studies.[1–5] However, there are very few studies on the short-term effects on health of more accurately size-segregated or source-specific particles.[6–8] There is also a lack of studies comparing the health effects of particles on cardiovascular and respiratory outcomes.

The scarcity of studies comparing the health effects of different particle measures is mainly due to limited availability of long time-series of particle concentrations.[9] This is especially true for ultrafine particles (<0.1 μm), which have been suggested to be more harmful for health than larger particles, because of their high concentration and larger active surface area.[10,11] Moreover, ultrafine particles have the ability to inhibit phagocytosis, and they may even be able to enter the blood circulation.[11,12]

Ultrafine particles consist mainly of carbon substances and can further be divided into 2 subfractions that differ in dynamics and may have varying effects on health. Nucleation mode particles (<0.03 μm) are mainly formed via atmospheric nucleation,[10] but they are also in part directly formed from traffic emissions. These particles have high short-term peak concentrations, so they can make a substantial contribution to short-term exposure to inhalable particles. However, the lifetime of nucleation mode particles is short. The peak number concentrations of Aitken mode (0.03–0.1 μm) particles are lower, but these particles are always present. Aitken mode is formed typically via condensational growth from nucleation mode and via coagulation, but sometimes is directly derived from emissions.

Accumulation mode (0.1–0.5 μm) particles, on the other hand, are mostly transported long-range.[13] Accumulation mode particles consist mainly of carbon compounds, sulfates, and nitrates. Because of their longer lifetime, these particles are more evenly distributed over large areas than

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ultrafine particles. The largest inhalable size fraction, coarse particles ($PM_{10-2.5}$, diameter 2.5–10 μm), is formed mainly of crustal material. Authors of many studies have shown an association between coarse particles and respiratory outcomes, but fewer studies have looked at the associations between coarse particles and cardiovascular health.[9,14]

The size of a particle is related to the emission source. Studies evaluating the effects of particle exposures on cardiovascular and respiratory morbidity and mortality have suggested that particles from combustion sources, especially traffic, are the most harmful.[3,7,16,17] However, the current evidence is insufficient for accurate ranking of the health effects of particles from different sources.[17]

In this study, we determined whether daily changes in ambient particles in several different size fractions or source-specific $PM_{2.5}$ are associated with cause-specific cardiorespiratory mortality and acute hospital admissions among people aged 65 years or older in Helsinki, Finland.

METHODS

The Helsinki metropolitan area consists of 4 cities (Helsinki, Vantaa, Espoo, and Kauniainen) for which the total population is around 1 million, and the surface area is 745 km.[2] We obtained daily data on mortality counts from Statistics Finland, and daily acute hospital admission counts from the National Research and Development Center for Welfare and Health for years 1998–2004. Data were included only for those people whose permanent residence was in the study area.

For all cardiovascular disease mortality and hospital admissions we used ICD-10 (International Classification of Diseases 10th revision) codes I00–99: for coronary heart disease, codes I20–I25; for stroke, codes I60–61 and I63–64; and for arrhythmia, codes I46.0, I46.9, and I47–I49. The remaining codes from I00–I99 were considered "other" cardiovascular diseases. Among hospital admissions, cardiac failure was the dominant subgroup in "other" cardiovascular diseases, but for mortality, other causes of death were more evenly distributed. For hospital admissions, we also analyzed myocardial infarction (I20–I21) as an independent group.

For all respiratory disease hospitalizations we used codes J00–J99; for pneumonia, codes J12–J15, J16.8, and J18; and for pooled asthma–chronic obstructive pulmonary disease (COPD), codes J41, and J44–J46. The remaining codes from J00–J99 for hospital admissions were considered "other" respiratory diseases, and bronchitis was the most common disease in this subgroup. For mortality, we used the same codes for all respiratory diseases and pneumonia. However, we analyzed mortality data for COPD (J41, J44) without asthma because there were only a few deaths due to asthma. "Other" respiratory mortality was not analyzed separately, because there were only 429 deaths in this group.

The data on particulate size fractions for years 1998–2004 were measured continuously with a differential mobility particle sizer at an urban background site. The measurement site location was changed once, in March 2001 as described earlier.[18] Particle counts at these 2 sites (eFigure 1 in the online version of this article) were closely correlated, although the number counts were slightly lower at the second site.[19,20] In the analyses, we controlled for the measurement site change by using a dummy variable in the models. Particles with diameters ranging from 0.01–0.29 μm were segregated into 10 size classes. For the ultrafine particles, we used the sum of fractions below 0.1 μm. Two subgroups of ultrafine particles, namely nucleation mode ($<$0.03 μm) and Aitken mode (0.03–0.1 μm), were separately analyzed. In addition, the effects of accumulation mode particles (0.1–0.29 μm) were studied. The particle modes were defined based on particle number distribution of the present data. These calculations and data handling are described in detail elsewhere.[19]

Particulate mass was measured daily by using a β-attenuation method (FH 62 I-R, Eberline instruments, GmbH, Erlangen, Germany). We obtained the coarse particulate mass by subtracting $PM_{2.5}$ from PM_{10}. The measurements of $PM_{2.5}$ and PM_{10} were performed at 2 closely situated urban background measurement sites (location changed in the beginning of 2003) (eFigure 1). The measurement site change was controlled for in the models with a dummy variable.

In the analyses, we used the 24-hour median for the nucleation, Aitken, and accumulation mode, and ultrafine particle counts because of their rightly skewed distribution. For the particle mass, we used 24-hour average concentrations. Lag 0 is defined as the concentration measured during the 24-hour period from midnight to midnight on the day of death or hospitalization, lag 1 as the previous 24-hour period, and so on. We analyzed single-day lags from 0–5 days, and the average of days 0–4 (5-day mean). All the results are provided as a percentage of change in mortality or hospital admissions for an interquartile increase in the pollutant level.

We determined the sources of $PM_{2.5}$ by using the EPA positive matrix factorization 1.1 model. Positive matrix factorization is an advanced multivariate receptor modeling technique that calculates site-specific source profiles and source contributions.[21] We used daily averages of $PM_{2.5}$, PM_{10}, $PM_{10-2.5}$, SO_2, NO_2, CO, NC_{33-45} (number concentration of particles 33–45 nm in diameter), and NC_{84-114} from the previously described urban background stations in the model. In addition, we included particulate SO_4^{2-} concentration measured at the Co-operative Programme for monitoring and evaluation of the long-range transmissions of air pollutants in Europe background station at Virolahti, 170 km east of Helsinki, to estimate the contribution of long-range transported particles to $PM_{2.5}$ in Helsinki. A more detailed description of the source apportionment is published elsewhere.[22]

We conducted the analyses of the associations between air pollutants and health outcomes with the Poisson generalized additive model. Modeling was implemented using R 2.1.1 software[23] and the mgcv 1.3–7 package.[24] We first built models without the air pollutants. We used penalized spline smoothing to model the variable for the time trend. Time trend and a dummy variable for weekday were always in the model, as were variables for the current day mean temperature, relative humidity, and barometric pressure. The significance of other confounders; 3 previous days' mean temperature and relative humidity; and the significance of variables for influenza epidemics, high pollen episodes, and general holidays were always checked when building the model, but these variables were dropped from the model if they were of low importance ($P > 0.25$).

We had weekly influenza counts. Because the distribution of influenza counts was skewed, we created a 3-level variable for influenza epidemics by using <35 (cumulative 79%), 35–179 (80%–94%), and ≥180 (≥95%) cases per week as the cutpoints. Data were entered into the models as 2 dummy variables. Similarly, a 2-level variable for pollen episodes was created using the daily sum (≥100) of the 4 most allergenic species (birch, mugwort, alder, and grass).

Pollen data were obtained from the Aerobiology Unit of the University of Turku. Daily pollen counts were measured from the beginning of March to the end of August, which is the annual pollen period in Finland. There was 1 pollen measurement site in Helsinki located approximately 4 km from the particulate measurement sites (eFigure 1).

We also performed analyses for cause-specific hospital admissions by warm and cold season (May–September and October–April, respectively).

As sensitivity analyses, we excluded days with pollution levels above the 98th percentile to evaluate the effect of extreme pollution levels. In the case of the 5-day average results, the exclusion was based on the 5-day mean and not on single-day averages. In 2-pollutant analyses, we adjusted pollutants that had significant associations with the outcomes for other pollutants. To avoid problems with collinearity, these analyses were only conducted when the pollutants' intercorrelation was <0.7. We also ran the analyses using the same lag for temperature and the pollutant in the model. In addition, in some analyses we included only those people living closer to the measurement sites, that is, in the city of Helsinki, to assess the effect of exposure misclassification on the results. To avoid oversmoothing and to define the amount of autocorrelation in the residuals we did partial autocorrelogram of the residuals, and visual inspection of the smoothed curves. To examine the effect of the chosen smooth function we also ran some of the analyses using the generalized linear model with natural cubic splines with 40 degrees of freedom (*df*). In the generalized additive model analyses, we also tested 20, 30, 35, 40, and 45 *df* for the time trend. These analyses had minor effect on the results.

RESULTS

Table 1 provides a summary of the daily mortality and hospital admission counts, levels of air pollutants, and weather variables. Spearman rank correlations between different indicators of particulate pollution are shown in Table 2. All correlations between pollutants, weather variables, and source-specific $PM_{2.5}$ are given in eTable 1.

We identified 4 sources for $PM_{2.5}$. The first factor was formed of secondary sulfate and other long-range transported particles, and the factor's average source contribution was 5.5 $\mu g/m^3$ or 57% (eFigure 2). The factor describing traffic emissions was characterized by NO_2, CO, NC_{33-45} (number concentration of particles with diameter 33–45 nm), and NC_{84-114}. It explained, on average, 19% of the $PM_{2.5}$ mass. The average source contribution of the third factor, soil and road dust fraction, was 1.0 $\mu g/m^3$ (10%). This factor described road dust resuspended by traffic or wind, and other sources of soil-related or coarse particles. The fourth factor was characterized by SO_2 that, in Helsinki, describes mostly coal combustion emissions and emissions from ship engines that burn residual oil. The average source contribution of the fourth factor was 0.5 $\mu g/m^3$ (6%). The sum of source-specific $PM_{2.5}$ concentrations explained fairly well the measured daily $PM_{2.5}$ ($R^2 = 0.79$).

Cardiovascular mortality had little association with any of the indicators of particulate air pollution (Table 3). When coronary heart diseases, stroke, and other cardiovascular mortality were analyzed separately, we found a positive association between other cardiovascular diseases and nucleation mode particles (4.2%; 95% confidence interval = 0.49–8.1 at lag 3 for an interquartile increase). A positive association was also observed between Aitken mode particles and current-day stroke mortality (2.8%; –1.3 to 6.9) (eTable 2). As we have earlier reported, $PM_{2.5}$ is associated with stroke mortality in Helsinki during the warm season.[18] Stroke mortality was associated also with traffic-related $PM_{2.5}$ (7.8%; 0.67–15.5 over the 5-day mean, data not shown). We found no other notable associations for $PM_{2.5}$ sources and cardiovascular mortality outcomes.

Hospital admission for all cardiovascular diseases was associated with coarse particles and with $PM_{2.5}$ related to soil and road dust at lag 0 (eTable 3). We also found an association between all cardiovascular admissions and $PM_{2.5}$ from coal and oil combustion (1.1%; 0.22–1.9, at lag 1).

In the cause-specific admission analyses, there was a positive association between arrhythmia and Aitken mode particles over the 5-day mean (Table 4). In the source-specific $PM_{2.5}$ analyses, arrhythmia was positively associated with $PM_{2.5}$ from traffic. During the warm season, all except coarse particles had positive associations with arrhythmia admissions (eTable 4).

TABLE 1. Daily Mortality and Hospital Admission Counts, Air Pollution Concentrations, and Weather Variables: 1998–2004

	Min.	25%	50%	75%	Max.
Deaths					
All cardiovascular disease	0	5	6	8	17
Coronary heart disease	0	2	4	5	12
Stroke	0	0	1	2	6
Other cardiovascular disease	0	1	1	2	7
All respiratory disease	0	0	1	2	8
Pneumonia	0	0	1	1	6
COPD	0	0	0	1	4
Hospital admissions					
All cardiovascular disease	7	19	24	29	64
Coronary heart disease	1	6	8	10	21
Stroke	0	3	4	5	16
Arrhythmia	0	2	4	6	15
Other cardiovascular disease	1	6	8	10	22
All respiratory disease	1	7	10	12	58
Pneumonia	0	3	4	5	28
Asthma-COPD	0	2	3	5	22
Other respiratory disease	0	1	2	3	17
Pollutants					
Nucleation mode (<0.03 μm) (1 cm^{-3})	379	2,673	4,187	6,256	22,790
Aitken mode (0.03–0.1 μm) (1 cm^{-3})	400	2,470	3,628	4,937	27,990
Ultrafine particles (<0.1 μm) (1 cm^{-3})	914	5,780	8,203	11,540	50,990
Accumulation mode (0.1–0.29 μm) (1 cm^{-3})	57	238	359	525	2,680
$PM_{2.5}$ ($\mu g\ m^{-3}$)	1.1	5.5	9.5	11.7	69.5
$PM_{2.5-10}$ ($\mu g\ m^{-3}$)	0.0	4.9	7.5	12.1	101.4
CO (mg m^{-3} 8-h max moving average)	0.1	0.3	0.5	0.6	2.4
NO_2 ($\mu g\ m^{-3}$)	3.4	20.1	28.2	34.3	96.4
Source-specific $PM_{2.5}$					
Traffic	0	1.2	1.8	2.3	7.4
Long-range transport	0	2.5	5.5	7.3	30.8
Soil and road dust	0	0.5	1.0	1.2	10.7
Coal/oil combustion	0	0.2	0.5	0.7	11.3
Weather variables					
Temperature (°C)	−23.2	0.0	6.2	13.6	25.4
Relative humidity (%)	38.0	73.0	80.2	89.0	99.0
Barometric pressure (mbar)	957	1,005	1,012	1,019	1,052

Coronary heart disease admissions were negatively associated with Aitken mode particles at lag 0, but positively associated with coarse particles and with soil and road dust particles at the same lag (Table 4). In more detailed analysis, myocardial infarctions also had negative association with current-day Aitken mode and ultrafine particles (–2.1%; –4.8 to 0.66, and –3.1%; –6.4 to 0.2, respectively, data not shown). However, there was also a positive association between myocardial infarction admissions and accumulation mode particles at 2-day lag (2.8%; 0.17–5.4). In the warm season, we found no important associations between accumulation mode particles and myocardial infarctions (data not shown). We also found no associations between $PM_{2.5}$ sources and myocardial infarction (data not shown).

Stroke admissions were negatively associated with particles larger than 0.1 μm and with $PM_{2.5}$ from traffic and long-range transport, but only at lag 2 (Table 4). However, there was a positive association between stroke admissions and $PM_{2.5}$ from coal and oil combustion also at lag 2. In the analyses restricted to the warm season, negative associations became less apparent (eTable 4). We found no associations between particles and "other" cardiovascular disease admissions (data not shown).

All respiratory disease mortality was associated with accumulation mode particles on the current day (Table 3). Positive associations were also observed for other particle measures especially at 1-day lag. In the analyses of source-specific $PM_{2.5}$, the highest effect estimates were observed for

TABLE 2. Spearman Rank Correlation Coefficients for Air Pollutants and Source-Specific $PM_{2.5}$: 1998–2004

	Nuc[a]	Ait[b]	UFP[c]	Acc[d]	$PM_{2.5-10}$[e]	Traffic	LRT	Soil	Coal
$PM_{2.5}$	0.14	0.48	0.35	0.88	0.25	0.21	0.28	0.27	0.43
Nuc		0.64	0.92	0.11	0.14	0.63	−0.02	0.12	0.30
Ait			0.48	0.45	0.28	0.73	0.26	0.26	0.51
UFP				0.35	0.24	0.74	0.13	0.22	0.44
Acc					0.20	0.26	0.77	0.22	0.38
$PM_{2.5-10}$						0.16	0.06	0.99	0.17
Traffic							−0.06	0.16	0.11
LRT								0.06	0.32
Soil									0.14

LRT indicates long-range transport.
[a]Nucleation mode <0.03 μm.
[b]Aitken mode 0.03–0.1 μm.
[c]Ultrafine particles <0.1 μm.
[d]Accumulation mode 0.1–0.29 μm.
[e]Coarse particles 10–2.5 μm.

long-range transported (lags 0–5) and traffic-originating (lag 1) particles (Table 3).

Of the cause-specific mortality outcomes, pneumonia had the strongest association with accumulation mode particles (eTable 5). In the case of COPD mortality, most particle fractions had strong associations.

All respiratory disease hospital admissions were associated with Aitken and accumulation mode particles and with $PM_{2.5}$ (eTable 3). We also observed an immediate association with coarse particles and a negative association with nucleation mode particles at lag 2. All respiratory admissions had a positive association with $PM_{2.5}$ related to traffic and a weaker association with soil and road dust particles at lag 0 (eTable 3).

The observed associations of $PM_{2.5}$ and accumulation mode with all respiratory hospital admissions were explained by pneumonia and pooled asthma-COPD admissions (Table 5). Immediate effect of accumulation mode particles and $PM_{2.5}$ was observed with asthma-COPD admissions and a cumulative effect with pneumonia admissions. Coarse particles and $PM_{2.5}$ related to traffic, long-range transport, and soil and road dust were also associated with asthma-COPD admissions.

The association of all respiratory diseases with Aitken mode particles was seen in the subgroup of other respiratory diseases (Table 5). $PM_{2.5}$ and $PM_{2.5}$ from traffic were also positively associated with other respiratory diseases (at lags 2 and 3, respectively) although the confidence intervals were rather wide. The effects of all particle fractions on respiratory admissions were stronger during the warm season than in the whole-year analyses (eTable 6).

When we excluded pollutants above the 98th percentile, the association between Aitken mode and "other" respiratory diseases admissions was reduced substantially (2.3%; –3.6 to 8.5, over the 5-day mean). This analysis reduced slightly the effects of particles on pneumonia (1.9%; −0.32 to 4.2 at lag 1, $PM_{2.5}$) and on pooled asthma-COPD admissions (1.8%; −0.62 to 4.3, and 2.9%; −0.3 to 6.2, at lag 0 for $PM_{2.5}$ and accumulation mode, respectively).

The observed positive associations of $PM_{2.5}$ and accumulation mode particles with respiratory admissions remained the same or slightly reduced when adjusting for other pollutants (eTable 7).

Of the gaseous pollutants, CO was associated with asthma-COPD and "other" respiratory diseases admissions (eTable 8). NO_2 was associated with arrhythmia and asthma-COPD admissions.

DISCUSSION

In this time-series study among people aged 65 years or older most particle fractions were associated with respiratory health. The strongest and most consistent associations were observed between accumulation mode particles and acute hospital admissions for all respiratory diseases, pneumonia, and asthma-COPD. Accumulation mode particles had immediate effect also on mortality due to all respiratory causes and pneumonia. Few associations were found between particles and cardiovascular health: arrhythmia admissions were associated with Aitken mode particles and $PM_{2.5}$ from traffic. Overall, associations for particles with respiratory outcomes were stronger and more consistent than with cardiovascular outcomes.

We found consistent associations between respiratory hospital admissions and particulate air pollution. All respiratory disease and pneumonia admissions increased by 3.9% and 4.9%, respectively, for 10-$\mu m/m^3$ increase in previous-day $PM_{2.5}$ level. These estimates are higher than the 1.3% and 3.8% increases in the all respiratory and pneumonia admis-

TABLE 3. Percentage of Change in All Cardiovascular and Respiratory Mortality for an Interquartile[a] Increase in Pollutants: 1998–2004

	All Cardiovascular Mortality[b] n = 16,233 % Change (95% CI)	All Respiratory Mortality[c] n = 3,701 % Change (95% CI)
Nucleation mode (<0.03 μm)		
Lag 0[d]	−0.38 (−2.96 to 2.26)	0.77 (−4.69 to 6.55)
Lag 1	−0.52 (−3.00 to 2.03)	3.41 (−1.80 to 8.88)
Lag 2	−0.29 (−2.82 to 2.30)	−4.13 (−9.29 to 1.33)
Lag 3	2.30[f] (−0.25 to 4.92)	−2.78 (−7.88 to 2.61)
5-d mean	0.57 (−3.43 to 4.75)	−3.85 (−11.3 to 4.18)
Aitken mode (0.03–0.1 μm)		
Lag 0	0.03 (−1.80 to 1.90)	1.92 (−2.13 to 6.14)
Lag 1	−0.62 (−2.49 to 1.28)	3.25 (−0.80 to 7.46)
Lag 2	−0.29 (−2.14 to 1.60)	−0.10 (−4.05 to 4.01)
Lag 3	−0.17 (−1.98 to 1.67)	−0.75 (−4.55 to 3.21)
5-d mean	−0.08 (−2.72 to 2.95)	1.5 (−5.02 to 8.48)
Accumulation mode (0.1–0.29 μm)		
Lag 0	0.27 (−1.54 to 2.11)	5.06[e] (1.23 to 9.02)
Lag 1	−0.06 (−1.84 to 1.75)	2.34 (−1.40 to 6.22)
Lag 2	0.46 (−1.30 to 2.25)	0.25 (−3.64 to 4.30)
Lag 3	0.23 (−1.54 to 2.02)	−0.20 (−4.12 to 3.88)
5-d mean	0.6 (−1.79 to 3.05)	1.94 (−2.49 to 6.57)
$PM_{2.5}$		
Lag 0	0.73 (−0.66 to 2.13)	2.67[f] (−0.39 to 5.82)
Lag 1	0.74 (−0.63 to 2.13)	1.59 (−1.43 to 4.70)
Lag 2	0.74 (−0.62 to 2.11)	0.03 (−2.99 to 3.16)
Lag 3	0.06 (−1.29 to 1.43)	−0.11 (−3.13 to 3.01)
5-d mean	0.87 (−0.94 to 2.70)	1.39 (−2.83 to 5.81)
$PM_{2.5–10}$		
Lag 0	−0.01 (−1.52 to 1.53)	−0.66 (−4.16 to 2.97)
Lag 1	−0.26 (−1.69 to 1.18)	2.90[f] (−0.48 to 6.39)
Lag 2	−0.61 (−2.03 to 0.83)	0.35 (−3.03 to 3.84)
Lag 3	−0.57 (−1.98 to 0.85)	−0.38 (−3.67 to 3.02)
5-d mean	−0.70 (−2.56 to 1.20)	0.36 (−4.54 to 5.51)
$PM_{2.5}$ sources		
Traffic		
Lag 0	0.97 (−1.33 to 3.32)	0.39 (−4.43 to 5.45)
Lag 1	0.5 (−1.78 to 2.83)	3.23 (−1.61 to 8.30)
Lag 2	0.06 (−2.16 to 2.33)	−0.20 (−4.88 to 4.71)
Lag 3	−0.59 (−2.78 to 1.66)	−0.06 (−4.73 to 4.83)
5-d mean	0.29 (−2.79 to 3.46)	0.69 (−6.13 to 8.00)
Long-range transport		
Lag 0	0.74 (−1.31 to 2.82)	1.63 (−2.85 to 6.33)
Lag 1	1.39 (−0.56 to 3.37)	1.05 (−3.26 to 5.54)
Lag 2	0.4 (−1.48 to 2.31)	2.86 (−1.62 to 7.53)
Lag 3	−0.75 (−2.60 to 1.14)	3.3 (−1.19 to 7.99)
5-d mean	0.17 (−2.45 to 2.86)	3.46 (−2.98 to 10.3)
Soil/road dust		
Lag 0	0.25 (−1.29 to 1.82)	0.44 (−3.10 to 4.12)
Lag 1	−0.67 (−2.12 to 0.81)	2.01 (−1.45 to 5.59)
Lag 2	−0.52 (−1.95 to 0.93)	−0.92 (−4.40 to 2.69)
Lag 3	−0.32 (−1.73 to 1.11)	−1.08 (−4.48 to 2.43)
5-d mean	−0.63 (−2.30 to 1.06)	−0.43 (−4.64 to 3.98)
Coal/oil combustion		
Lag 0	−1.05 (−2.70 to 0.62)	−0.83 (−4.29 to 2.76)
Lag 1	−0.98 (−2.61 to 0.67)	0.2 (−3.25 to 3.78)
Lag 2	−0.19 (−1.80 to 1.45)	−2.40 (−5.98 to 1.31)
Lag 3	0.48 (−1.09 to 2.06)	−3.07 (−6.58 to 0.57)
5-d mean	0.66 (−0.96 to 2.31)	−2.43 (−5.99 to 1.26)

[a]Interquartile range for nucleation mode = 3583; Aitken mode = 2467; ultrafine particles = 5760; accumulation mode = 287; $PM_{2.5}$ = 6.2; coarse particles = 7.2.

[b]Model adjusted for trend, weekday, influenza episodes, temperature (lag 0 and 1–3-d mean), relative humidity (lag 0), and barometric pressure.

[c]Model adjusted for trend, weekday, influenza episodes, temperature (lag 0 and 1–3-d mean), relative humidity (lag 0 and 1–3-d mean), and barometric pressure.

[d]Lag 0 defined as the concentration measured during the 24-h period from midnight to midnight at the day of death or hospitalization, lag 1 as the previous 24-h period, and so on.

[e]$P < 0.05$.

[f]$P < 0.10$.

TABLE 4. Percentage of Change in Cause-Specific Cardiovascular Hospital Admissions for an Interquartile[a] Increase in Pollutants: 1998–2004

Pollutant	Coronary Heart Disease[b] n = 20,007 % Change (95% CI)	Stroke[c] n = 10,383 % Change (95% CI)	Arrhythmia[d] n = 10,423 % Change (95% CI)
Nucleation mode (<0.03 μm)			
Lag 0[e]	−1.64 (−3.96 to 0.74)	2.41 (−0.84 to 5.75)	2.77[g] (−0.47 to 6.11)
Lag 1	−0.85 (−3.11 to 1.46)	−0.62 (−3.72 to 2.59)	−2.93[g] (−5.96 to 0.19)
Lag 2	2.12[g] (−0.25 to 4.55)	0.42 (−2.79 to 3.74)	−1.54 (−4.71 to 1.74)
Lag 3	−0.13 (−2.42 to 2.22)	0.75 (−2.42 to 4.02)	2.52 (−0.73 to 5.87)
5-d mean	−0.81 (−4.47 to 2.99)	2.06 (−3.09 to 7.49)	2.48 (−2.66 to 7.88)
Aitken mode (0.03–0.1 μm)			
Lag 0	−1.78[g] (−3.53 to 0.01)	0.55 (−1.81 to 2.96)	1.10 (−1.32 to 3.57)
Lag 1	0.32 (−1.46 to 2.12)	0.97 (−1.41 to 3.40)	0.08 (−2.32 to 2.53)
Lag 2	0.43 (−1.32 to 2.20)	0.78 (−1.56 to 3.17)	0.33 (−2.02 to 2.74)
Lag 3	−0.02 (−1.67 to 1.66)	0.25 (−2.00 to 2.56)	1.66 (−0.64 to 4.02)
5-d mean	−0.81 (−3.53 to 1.98)	2.59 (−1.03 to 6.35)	4.09[f] (0.32 to 8.01)
Accumulation mode (0.1–0.29 μm)			
Lag 0	0.21 (−1.50 to 1.96)	−1.03 (−3.34 to 1.33)	0.09 (−2.28 to 2.52)
Lag 1	0.45 (−1.18 to 2.12)	−0.46 (−2.70 to 1.84)	0.57 (−1.70 to 2.89)
Lag 2	0.71 (−0.91 to 2.34)	−2.37[f] (−4.59 to −0.11)	0.58 (−1.64 to 2.85)
Lag 3	0.02 (−1.59 to 1.65)	0.11 (−2.12 to 2.39)	0.07 (−2.17 to 2.36)
5-d mean	0.66 (−1.56 to 2.94)	−0.86 (−3.86 to 2.24)	0.87 (−2.20 to 4.04)
$PM_{2.5}$			
Lag 0	−0.17 (−1.50 to 1.18)	−0.99 (−2.78 to 0.84)	0.82 (−1.03 to 2.68)
Lag 1	−0.03 (−1.31 to 1.26)	0.02 (−1.74 to 1.82)	0.18 (−1.58 to 1.97)
Lag 2	−0.63 (−1.87 to 0.62)	−1.38 (−3.13 to 0.40)	−0.09 (−1.82 to 1.67)
Lag 3	0.48 (−0.78 to 1.76)	−0.17 (−1.92 to 1.61)	−0.48 (−2.22 to 1.29)
5-d mean	0.80 (−0.94 to 2.58)	−0.78 (−3.10 to 1.60)	0.16 (−2.16 to 2.54)
PM_{10}–$PM_{2.5}$			
Lag 0	1.12 (−0.28 to 2.55)	−1.33 (−3.26 to 0.63)	0.57 (−1.33 to 2.49)
Lag 1	−0.38 (−1.68 to 0.94)	−1.90[g] (−3.82 to 0.07)	−0.65 (−2.55 to 1.29)
Lag 2	0.01 (−1.33 to 1.37)	−1.09 (−3.04 to 0.89)	0.02 (−1.93 to 2.00)
Lag 3	−0.53 (−1.82 to 0.78)	−0.51 (−2.40 to 1.43)	−1.34 (−3.26 to 0.62)
5-d mean	0.23 (−0.29 to 0.75)	−2.21 (−4.75 to 0.39)	−1.11 (−3.68 to 1.53)
$PM_{2.5}$ source			
Traffic			
Lag 0	−0.25 (−2.32 to 1.86)	0.38 (−2.47 to 3.31)	3.05[f] (0.09 to 6.09)
Lag 1	0.32 (−1.73 to 2.42)	−0.10 (−2.90 to 2.78)	−0.69 (−3.49 to 2.19)
Lag 2	0.71 (−1.32 to 2.79)	−2.88[f] (−5.60 to −0.09)	−0.34 (−3.14 to 2.54)
Lag 3	1.80 (−0.24 to 3.87)	−0.45 (−3.21 to 2.40)	1.73 (−1.09 to 4.64)
5-d mean	2.39 (−0.44 to 5.30)	−0.70 (−4.47 to 3.22)	3.45[g] (−0.47 to 7.53)
Long-range transport			
Lag 0	−0.74 (−2.66 to 1.22)	0.27 (−2.31 to 2.92)	0.40 (−2.29 to 3.16)
Lag 1	−0.53 (−2.32 to 1.29)	−0.10 (−2.65 to 2.32)	−1.02 (−3.49 to 1.51)
Lag 2	−0.70 (−2.44 to 1.06)	−2.81[f] (−5.23 to −0.34)	−0.16 (−2.62 to 2.37)
Lag 3	0.13 (−1.61 to 1.90)	0.05 (−2.43 to 2.59)	0.38 (−2.09 to 2.92)
5-d mean	−0.09 (−2.57 to 2.45)	−0.89 (−4.31 to 2.65)	0.13 (−3.36 to 3.74)
Soil/road dust			
Lag 0	1.12 (−0.30 to 2.57)	−1.62 (−3.58 to 0.38)	0.64 (−1.32 to 2.65)
Lag 1	0.21 (−1.12 to 1.56)	−1.81[g] (−3.75 to 0.18)	0.20 (−1.73 to 2.16)
Lag 2	0.02 (−1.36 to 1.33)	−0.57 (−2.56 to 1.45)	−0.40 (−2.38 to 1.63)
Lag 3	−0.30 (−1.63 to 1.04)	−0.74 (−2.68 to 1.25)	−1.01 (−2.97 to 0.99)
5-d mean	0.15 (−1.41 to 1.74)	−1.86 (−4.11 to 0.45)	−0.71 (−2.98 to 1.62)
Coal/oil combustion			
Lag 0	−0.59 (−2.13 to 0.98)	0.20 (−1.77 to 2.22)	0.22 (−1.78 to 2.27)
Lag 1	0.98 (−0.49 to 2.42)	1.39 (−0.53 to 3.36)	0.02 (−1.97 to 2.05)
Lag 2	0.27 (−1.23 to 1.79)	2.27[f] (0.39 to 4.18)	1.13 (−0.82 to 3.13)
Lag 3	−0.50 (−1.99 to 1.01)	0.93 (−1.02 to 2.93)	0.90 (−1.05 to 2.89)
5-d mean	0.07 (−1.60 to 1.47)	1.19 (−0.90 to 3.33)	0.82 (−1.28 to 2.96)

[a]Interquartile range for nucleation mode = 3583; Aitken mode = 2467; accumulation mode = 287; $PM_{2.5}$ = 6.2; coarse particles = 7.2.
[b]Model adjusted for time trend, weekday, influenza and pollen episodes, temperature (lag 0 and 1–3-d mean), relative humidity (lag 0), and barometric pressure.
[c]Model adjusted for time trend, weekday, holiday, temperature (lag 0 and 1–3-d mean), relative humidity (lag 0 and 1–3-d mean), and barometric pressure.
[d]Model adjusted for time trend, weekday, holiday, influenza epidemics, temperature (lag 0 and 1–3-d mean), relative humidity (lag 0 and 1–3-d mean), and barometric pressure.
[e]Lag 0 defined as the concentration measured during the 24-h period from midnight to midnight at the day of death or hospitalization, lag 1 as the previous 24-h period, and so on.
[f]$P < 0.05$.
[g]$P < 0.10$.

TABLE 5. Percentage of Change in Cause-Specific Respiratory Hospital Admissions for Interquartile[a] Increases in Pollutants: 1998–2004

Pollutant	Pneumonia[b] n = 10,733 % Change (95% CI)	Asthma + COPD[c] n = 9,242 % Change (95% CI)	Other[d] n = 6,120 % Change (95% CI)
Nucleation mode (<0.03 μm)			
Lag 0[e]	−0.31 (−3.59 to 3.07)	−2.3 (−5.60 to 1.11)	−0.58 (−4.70 to 3.72)
Lag 1	−1.55 (−4.61 to 1.60)	−1.1 (−4.30 to 2.21)	−1.39 (−5.22 to 2.60)
Lag 2	−1.03 (−4.00 to 2.03)	−1.82 (−4.94 to 1.40)	−2.96 (−6.64 to 0.87)
Lag 3	−0.55 (−3.48 to 2.46)	0.8 (−2.28 to 3.97)	2.42 (−1.33 to 6.31)
5-d mean	−0.73 (−4.95 to 3.68)	−2.19 (−7.24 to 3.14)	2.41 (−3.02 to 8.15)
Aitken mode (0.03–0.1 μm)			
Lag 0	0.79 (−1.57 to 3.21)	−0.01 (−2.43 to 2.48)	0.69 (−2.35 to 3.82)
Lag 1	1.55 (−0.69 to 3.85)	1.69 (−0.66 to 4.09)	1.24 (−1.56 to 4.12)
Lag 2	0.72 (−1.50 to 2.99)	−0.6 (−2.93 to 1.79)	1.51 (−1.21 to 4.31)
Lag 3	1.5 (−0.72 to 3.77)	0.82 (−1.51 to 3.20)	4.34[f] (1.60 to 7.16)
5-d mean	3.86[f] (0.17 to 7.69)	0.99 (−2.71 to 4.84)	7.65[f] (2.98 to 12.5)
Accumulation mode (0.1–0.29 μm)			
Lag 0	0.84 (−1.48 to 3.20)	3.75[f] (1.29 to 6.27)	0.65 (−2.53 to 3.94)
Lag 1	1.98[g] (−0.31 to 4.31)	3.30[f] (0.90 to 5.76)	0.62 (−2.44 to 3.78)
Lag 2	1.81 (−0.44 to 4.11)	0.11 (−2.26 to 2.55)	0.65 (−2.36 to 3.75)
Lag 3	1.96[g] (−0.30 to 4.27)	0.24 (−2.17 to 2.70)	1.02 (−2.01 to 4.15)
5-d mean	3.06[f] (0.43 to 5.75)	2.93[g] (−0.45 to 6.43)	1.24 (−2.31 to 4.91)
$PM_{2.5}$			
Lag 0	0.93 (−0.85 to 2.75)	2.48[f] (0.60 to 4.39)	0.05 (−2.38 to 2.54)
Lag 1	2.41[f] (0.64 to 4.21)	2.62[f] (0.78 to 4.49)	0.2 (−2.17 to 2.62)
Lag 2	1.48g (−0.27 to 3.26)	1.22 (−0.62 to 3.10)	2.03[g] (−0.29 to 4.41)
Lag 3	1.91[f] (0.14 to 3.70)	0.59 (−1.28 to 2.49)	1.72 (−0.63 to 4.12)
5-d mean	3.10[f] (0.60 to 5.65)	2.49[g] (−0.08 to 5.12)	1.88 (−1.50 to 5.36)
PM_{10}–$PM_{2.5}$			
Lag 0	0.72 (−1.28 to 2.77)	2.49[f] (0.47 to 4.56)	1.38 (−1.24 to 4.06)
Lag 1	0.55 (−1.34 to 2.49)	1.37 (−0.66 to 3.44)	−1.62 (−4.22 to 1.05)
Lag 2	0.65 (−1.24 to 2.58)	0.7 (−1.36 to 2.80)	−1.25 (−3.88 to 1.45)
Lag 3	0.03 (−1.86 to 1.96)	1.97[g] (−0.02 to 4.00)	0.04 (−2.52 to 2.67)
5-d mean	0.82 (−1.92 to 3.64)	2.67[g] (−0.17 to 5.58)	0.24 (−3.62 to 4.26)
$PM_{2.5}$ source			
Traffic			
Lag 0	0.47 (−2.40 to 3.43)	0.2 (−2.82 to 3.31)	1.13 (−2.53 to 4.92)
Lag 1	0.53 (−2.25 to 3.39)	3.45[f] (0.43 to 6.56)	2.23 (−1.36 to 5.96)
Lag 2	0.29 (−2.47 to 3.13)	−0.43 (−3.37 to 2.60)	1.92 (−1.65 to 5.62)
Lag 3	0.69 (−2.07 to 3.53)	0.58 (−2.38 to 3.63)	3.54[g] (−0.06 to 7.27)
5-d mean	2.13 (−1.59 to 5.99)	1.5 (−2.73 to 5.91)	3.13 (−1.77 to 8.28)
Long-range transport			
Lag 0	0.46 (−2.16 to 3.16)	3.03[f] (0.21 to 5.93)	−0.37 (−3.81 to 3.20)
Lag 1	0.5 (−2.01 to 3.07)	3.17[f] (0.43 to 5.99)	0.97 (−2.41 to 4.47)
Lag 2	0.52 (−1.92 to 3.01)	1.9 (−0.82 to 4.70)	1.14 (−2.21 to 4.62)
Lag 3	1.12 (−1.30 to 3.60)	1.44 (−1.26 to 4.20)	2.11 (−1.23 to 5.56)
5-d mean	0.75 (−2.23 to 3.82)	3.57[g] (−0.37 to 7.67)	0.13 (−3.97 to 4.40)
Soil/road dust			
Lag 0	0.71 (−1.34 to 2.80)	2.29[f] (0.24 to 4.38)	1.36 (−1.29 to 4.08)
Lag 1	0.07 (−1.86 to 2.03)	0.62 (−1.42 to 2.71)	−2.52[g] (−5.13 to 0.17)
Lag 2	0.47 (−1.46 to 2.43)	0.05 (−2.06 to 2.20)	−2.34 (−5.00 to 0.40)
Lag 3	0.03 (−1.88 to 1.97)	1.14 (−0.91 to 3.23)	−1.22 (−3.80 to 1.43)
5-d mean	1.01 (−1.38 to 3.46)	1.04 (−1.46 to 3.60)	−1.41 (−4.65 to 1.95)
Coal/oil combustion			
Lag 0	0.32 (−1.67 to 2.36)	−1.45 (−3.57 to 0.71)	0.53 (−1.94 to 3.06)
Lag 1	1.53 (−0.34 to 3.44)	−0.63 (−2.71 to 1.51)	0.38 (−1.98 to 2.79)
Lag 2	0.51 (−1.39 to 2.45)	0.18 (−2.22 to 1.90)	1.01 (−1.30 to 3.38)
Lag 3	0.55 (−1.33 to 2.47)	−0.95 (−3.03 to 1.18)	1.59 (−0.65 to 3.89)
5-d mean	0.65 (−1.39 to 2.74)	−0.44 (−2.60 to 1.77)	2.23 (−0.54 to 5.07)

[a]Interquartile range for nucleation mode = 3583; Aitken mode = 2467; ultrafine particles = 5760; accumulation mode = 287; $PM_{2.5}$ = 6.2; coarse particles = 7.2.

[b]Model adjusted for time trend, weekday, influenza epidemics, temperature (lag 0), relative humidity (lag 0), and barometric pressure.

[c]Model adjusted for time trend, weekday, influenza and high pollen epidemics, temperature (lag 0), relative humidity (lag 0 and 1–3-d mean), and barometric pressure.

[d]Model adjusted for time trend, weekday, influenza epidemics, temperature (lag 0), relative humidity (lag 0 and 1–3-d mean), and barometric pressure.

[e]Lag 0 defined as the concentration measured during the 24-h period from midnight to midnight at the day of death or hospitalization, lag 1 as the previous 24-h period, and so on.

[f]$P < 0.05$.

[g]$P < 0.10$.

sions, respectively, reported earlier in North America.[4,25] Our asthma-COPD results (5.0% and 5.3% for 10-μg/m^3 increase in $PM_{2.5}$ at lags 0 and 1, respectively) are similar to our study results on asthma-COPD emergency room visits (6.3% and 4.6% for 10 μg/m^3 $PM_{2.5}$ at lags 0 and 1, respectively).[22] These findings are also similar to earlier COPD findings although a large variation in the effects of $PM_{2.5}$ (0.9%-19.7% increase for 10 μg/m^3) has been reported.[2,6,26]

The associations of $PM_{2.5}$ and accumulation mode particles (0.1–0.29 μm) with respiratory diseases were comparable, whereas effect estimates for accumulation mode particles were slightly stronger and more consistent. Similarity of the effect estimates was expected due to the high correlation between these particle fractions. Accumulation mode particles have also been associated with hospital admissions for respiratory diseases in the elderly in a recently published Danish study.[7]

The more clear association between accumulation mode particles and respiratory health compared with other particle fractions in the current study may be related to better exposure assessment of these particles compared with particles in the ultrafine or coarse size fraction. There are 2 reasons for this. First, there is less spatial variation in the levels of accumulation mode particles (and $PM_{2.5}$) than ultrafine or coarse particles. Second, the penetration of ultrafine and coarse particles is worse indoors, where the elderly spend most of their time, than $PM_{2.5}$ and accumulation mode.[11,27] There are no studies measuring personal exposure to ultrafine particles in Helsinki, but central outdoor measurements have been shown to reflect better the indoor levels of $PM_{2.5}$ than the levels of ultrafine or coarse particles.[28,29] However, the spatial correlations for aerosol number concentrations and night-time correlations between indoor and central site particle counts are fairly good in the study area.[20,29]

In general, we observed equally large associations but with wider confidence intervals for particles with respiratory mortality than with respiratory hospital admissions, which can be related to the rather low number of daily deaths. Respiratory mortality was associated with current-day accumulation mode particles and with previous-day levels of other particle fractions such as nucleation and Aitken mode particles. These findings are in good accordance with previous findings in which fine particles and black smoke (surrogate for primary combustion particles) have been linked with increased cardiorespiratory mortality among the elderly.[1,3,30]

The associations between pollutants and mortality were acute, occurring mainly at 0- to 1-day lags, whereas the effects on admissions were more delayed. This is probably at least partly explained by the typical delay before seeking medical attention. It could also be related to the increased frailty of people vulnerable to experiencing lethal effects of air pollution.

In the current study, we observed a suggestion of positive associations for traffic-related and long-range transported $PM_{2.5}$ with all respiratory hospital admission outcomes. Long-range transported particles explain most of the association found for accumulation mode particles; particles in the accumulation mode fraction in Helsinki are dominated by pollutants transported from southwest Russia and central Europe.[13] The observed association between asthma-COPD admissions and $PM_{2.5}$ from long-range transport is similar to our previous findings on hospital emergency room visits,[22] and our findings are consistent with previous studies in which secondary fraction of PM_{10}[8] and sulfate-rich secondary $PM_{2.5}$[31] were linked to respiratory admissions.

Association for all cardiovascular mortality and morbidity with air pollution has been shown in several studies.[32] Increase in cardiovascular mortality has been estimated to be 1% for 10-μg m^{-3} increase in $PM_{2.5}$.[32] However, we found little evidence of positive associations between ischemic cardiovascular outcomes and particulate measures in Helsinki, which is consistent with earlier analysis.[33] In the Air Pollution and Health: A European Approach (APHEA) study, the largest PM effects were observed in cities with higher NO_2 levels and mean temperature.[33] These factors, together with relatively lower particulate matter levels, and the fact that the majority of $PM_{2.5}$ is long-range transported in Helsinki[34] may explain the lower effect estimates compared with studies performed elsewhere.

In the current study, arrhythmia admissions were associated with Aitken mode particles and traffic-related $PM_{2.5}$. A previous time-series study found no associations between air pollutants and arrhythmia[5]; however, arrhythmia has been associated with particulate air pollution in other studies.[35,36]

Several studies have shown association between stroke and particulate matter,[32] as in our previous study.[18] We found a positive association between stroke mortality and fine particles but only during the warm season. No such association was observed in the present study for stroke admissions. As we discussed earlier,[18] a possible explanation for this inconsistency could be that PM increases the risk of death due to other causes, such as pneumonia,[37] especially among stroke patients, but may not increase hospital admissions due to stroke.

We found some support for earlier suggestions[9,14] of an association of coarse particles with coronary heart disease admissions. Consistent with this, $PM_{2.5}$ from soil and road dust was also associated with coronary heart disease in our study. In contrast to our previous results on first acute myocardial infarctions[38] in Helsinki, we found a negative association between ultrafine particles and myocardial infarction admissions. However, in the previous study,[38] different years and a larger age group ($\geq$35 years) were included, and particle number concentrations were modeled.

Our source apportionment via positive matrix factorization analysis provided information on the same sources as earlier principal component analysis performed for Helsinki

$PM_{2.5}$ data,[34,39] with the exception that sea salt could not be separated as its own factor because of the lack of suitable marker elements. However, the average source contributions were in the range of earlier results.[34,39] Due to the long time period (2557 days) the model was stable, and 20 runs from random starting points produced the same factor compositions and contributions.

A limitation of our study is that we were not able to combine hospital admission data and mortality data on the individual level. Due to separate registers, there is overlap especially for mortality and hospital admissions due to coronary heart disease. About one-fourth of the myocardial infarction patients die before reaching the hospital, and the case fatality in hospital is also high.[40] Of pneumonia cases, 5%–8% die in the hospital within 5 days,[41] which is the longest lag used in this study. Furthermore, mortality for COPD within 30 days from hospitalization is low; about 5%.[42] Therefore, the overlap of mortality and hospital admission data is rather small for respiratory outcomes.

This study found associations between all particle fractions and cardiorespiratory health among people aged 65 years or older. The strongest associations were observed for accumulation mode (0.1–0.29 μm) particles with all respiratory mortality, and with hospital admissions for pneumonia and asthma-COPD. Of the $PM_{2.5}$ sources, long-range transported and traffic-related particles seem to be responsible for the harmful respiratory effects. Few associations were found between cardiovascular outcomes and particulate air pollutants, and overall, associations for respiratory outcomes were stronger than for cardiovascular health.

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