Supplement

Scand J Work Environ Health 2004;30(2):19-27

Mortality and air pollution in metropolitan Helsinki, 1988-1996

by Penttinen P, Tiittanen P, Pekkanen J

Affiliation: Unit of Environmental Epidemiology, National Public Health Institute, PO Box 95, FI-70701 Kuopio, Finland. Pase.Penttinen@ktl.fi

The following article refers to this text: 2004;30 suppl 2:1-98

Key terms: air pollution; carbon monoxide; cardiovascular mortality; Helsinki; mortality; nitrogen dioxide; ozone; particulate matter; particulate matter with an aerodynamic diameter of <10 µm; respiratory mortality; sulfur dioxide

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/15487682

Original article

Scand J Work Environ Health 2004;30 suppl 2:19–27

Mortality and air pollution in metropolitan Helsinki, 1988–1996

by Pasi Penttinen, MD,[1, 2] Pekka Tiittanen, MSc,[1] Juha Pekkanen, MD[1]

Penttinen P, Tiittanen P, Pekkanen J. Mortality and air pollution in metropolitan Helsinki, 1988–1996. *Scand J Work Environ Health* 2004;30 suppl 2:19–27.

Objectives The daily respiratory, cardiovascular, and total mortality of the population in the subarctic climate of the Helsinki metropolitan area was analyzed for associations with daily variations in concentrations of common ambient-air pollutants.

Methods The associations between daily mortality and the ambient-air concentrations of ozone (O_3), nitrogen dioxide (NO_2), carbon monoxide (CO), sulfur dioxide (SO_2), total suspended particulate matter (TSP), and particulate matter with an aerodynamic diameter of <10 μm (PM_{10}) in 1988–1996 were analyzed with Poisson regression in a generalized linear model. Specifically, the differences for combustion and noncombustion particulate matter were determined using the blackness of the TSP filters as a surrogate for mostly traffic-derived combustion particles. Total mortality was analyzed according to three age groups (15–64, 65–74, ≥75 years) of the population.

Results Significant associations consistent across lag times and age groups were found between spring and summer O_3 levels and respiratory (4.30% increase per 10 μg/m^3 of the 4-day mean O_3 level) and total (2.42% increase per 10 μg/m^3 of the 4-day mean O_3 level) mortality. PM_{10} was consistently associated with respiratory mortality across the lag times and age groups (3.96% increase per 10 μg/m^3 of PM_{10} at lag-time 1). When adjusted for TSP concentration, the blackness of the TSP was significantly associated with total mortality (2.06% per 10^{-5} units, 95% confidence interval 0.09–4.06) at lag time 1, but not at the other lag times.

Conclusions These results provide additional evidence of an association between summertime O_3 concentrations and mortality. They also support the findings of previous studies indicating that coarse mineral particles are not as strongly associated with mortality as fine, combustion-derived particles are.

Key terms carbon monoxide, cardiovascular mortality, nitrogen dioxide, ozone, respiratory mortality, particulate matter, sulfur dioxide, thoracic particles.

Ambient-air particulate matter is associated with mortality from respiratory and cardiovascular causes (1–4). This association has been demonstrated for both day-to-day variations in mortality (1, 2) and long-term mortality (3, 4). Previous studies have suggested that the fine particulate fractions, derived from combustion sources, are probably responsible for the associations between particles and mortality (5, 6). In subarctic weather conditions, as in Finland, a large portion of urban-air particles are derived from resuspended coarse road dust (7). Therefore, it is important to try to separate the health effects associated with coarse soil particles from those associated with locally produced fine, combustion-derived particles. Unfortunately, fine particulate matter ($PM_{2.5}$, aerodynamic diameter <2.5μm) has been monitored in Finland for only a few years, and, therefore, only short time-series are available for studies on their health effects. We have used a simple optical measurement of the blackness of archived filters of total suspended particulate matter (TSP) as a surrogate measure for fine, combustion-derived particle concentrations. We have shown earlier that this measure of blackness is highly correlated with the daily concentrations of black smoke and traffic-related gaseous pollutants (8).

The independent health effects of individual ambient-air pollutants are difficult to separate from the complex mixture in which they appear. The results of the European multicenter study Air Pollution and Health: a European Approach (APHEA) have implied an independent effect of sulfur dioxide (SO_2) and particulate matter on mortality (1). However, a recent analysis of the APHEA-2 data has shown differences between

1 Unit of Environmental Epidemiology, National Public Health Institute, Kuopio, Finland.
2 European Programme on Intervention Epidemiology Training (EPIET), Swedish Institute for Infectious Disease Control, Solna, Sweden.

Reprint requests to: Dr Juha Pekkanen, Unit of Environmental Epidemiology, National Public Health Institute, PO Box 95, FI-70701 Kuopio, Finland. [E-mail: Juha.Pekkanen@ktl.fi]

cities in the effect estimates for particulate matter (9). Ozone (O_3) has also been shown to be positively associated with mortality in some (6, 10), but not all (11), studies, and it has been suggested that O_3 could serve as a surrogate for SO_2 or particles (12).

The objective of this study was to analyze the relationship between daily variations in the gaseous and particulate pollutant measures and mortality in the subarctic climate of the Helsinki metropolitan area. The particulate measures especially focused on the blackness of archived TSP filters.

Material and methods

Study area

The Helsinki metropolitan area consists of four cities, Helsinki, Espoo, Vantaa, and Kauniainen. At the end of 1988, the total population of this area was 816 733 (905 545 in 1996).

Mortality data

We obtained individual mortality data for the residents of the Helsinki metropolitan area from Statistics Finland. The causes of death were coded according to the 9th revision of the International Classification of Diseases (ICD-9) for 1988–1995 and according to the 10th revision (ICD-10) for 1996. Deaths that occurred abroad and deaths from external causes (E800–E990 in ICD-9 and V01–Y89 in ICD-10) were excluded. We calculated the daily counts of deaths for the following four age groups: all ages, 15–64 years, 65–74 years, and ≥75 years. The daily counts were calculated also separately for all-cause, respiratory (460 ≤ICD-9 ≤519, J00 ≤ICD-10 ≤J64, J66 ≤ICD-10 ≤J99) and cardiovascular (390 ≤ICD-9 ≤459, I00 ≤ICD-10 ≤I99, ICD-10=F01.1 and ICD-10=G45) deaths.

Air pollution data

We obtained the air pollution data from the Helsinki Metropolitan Area Council (YTV), which monitored air quality at several measurement sites in 1988–1996. A station measuring gaseous pollutants with continuous monitoring methods was excluded if more than 25% of its data was missing for the study period. This completeness criterion was not applied for the 24-hour sampling of particulate matter because particles with an aerodynamic diameter of <10 μm (PM_{10}) were measured only every fourth day and TSP was measured only every second day. Thus three stations were available for nitrogen dioxide (NO_2) and SO_2, and two for O_3, carbon monoxide (CO), PM_{10}, and TSP. The correlations between the measurements from the selected sites ranged from 0.66 to 0.71 for SO_2 and from 0.54 to 0.73 for NO_2, and they were 0.63 for O_3, 0.54 for CO, 0.79 for PM_{10}, and 0.76 for TSP. The blackness of the TSP filters (8) was measured only for one station (Vallila), and, therefore, all the analyses for TSP were limited to this station.

Several calculations were performed for each air pollutant at each measurement site. Twenty-four-hour average concentrations (from midnight to midnight) were calculated for NO_2, SO_2, PM_{10}, and TSP. The 8-hour moving average concentrations were calculated for O_3 and CO, and the maximum value on each day was chosen. The maximum 1-hour average values of NO_2 and O_3 were also obtained on each day. Missing values in the time-series of air pollutants (except for PM_{10} and TSP) at each measurement site were imputed using the APHEA protocol (6). The missing value on day i at measurement site j in year k (X_{ijk}) was replaced after two-phase calculations. First, the arithmetic daily mean from the other available sites ($X_{i.k}$), the annual mean of the pollutant at site j in year k ($X_{.jk}$), and the annual mean of the pollutant at all sites in year k ($X_{..k}$) were calculated. Then the missing value of the air pollutant at site j was replaced by a weighted mean of the corresponding 24-hour, 8-hour, or 1-hour values from the other available measurement sites, multiplied by the annual mean at site j and divided by the annual mean of all the sites:

$$X_{ijk} = X_{i.k} * (X_{.jk} / X_{..k}).$$

Finally, the mean 24-hour, 8-hour, or 1-hour averages of the air pollutants over all stations were calculated for all days of the study period and used in the analysis. Possible missing values in these final time series (ie, days with missing values at all used sites) were replaced with the mean of the previous and next day, if they were available. However, this procedure was not carried out for PM_{10} and TSP, as the yearly proportion of missing data exceeded the APHEA criterion (12). In the final analyses, 0–3 days of lagged values and the 4-day averages of the pollutant concentrations were used. The decision to choose these lag times was based on the health associations found in previous studies.

Other data

We obtained the meteorological data from the Finnish Meteorological Institute. The daily mean, minimum, and maximum temperatures and relative humidity were measured at a meteorological station in the center of Helsinki (Kaisaniemi). Dummy variables for hot and cold days were created using 1% (–13.6 C°) and 99% (+21.6 C°) percentiles of the daily mean temperature as cut points.

The data on weekly counts of influenza, diagnosed at the municipal health care centers, was obtained from

the Health Bureau of the City of Helsinki. We created a dummy variable for an influenza epidemic by using 40 cases per week as the cut point, and gave the value 1 for all days in that week.

Statistical methods

The association between the daily counts of death and air pollution was examined with a Poisson regression. We applied generalized additive models (GAM) to explore and allow nonlinear relations between mortality and covariates. The model was created stepwise. In the first step, to control for long-term trends and seasonal variations in mortality, smooth functions of time [locally weighted least squares regression (LOESS)] were introduced into the model. An appropriate amount of smoothing was chosen on the basis of visual exploration of the smoothed curves. A partial autocorrelogram of the residuals was also checked to avoid over-smoothing and to define the amount of autocorrelation in the residuals. In the second step, the weather terms (ie, daily mean temperature and relative humidity) were incorporated into the model using LOESS functions. The appropriate span of the smoothing window was chosen on the basis of Akaike's Information Criteria. The shape of the association between mortality and temperature was a mirror image of the letter J. The association with relative humidity was linear. In the third step, dummy variables for influenza epidemics and weekdays were entered into the model. Finally, single pollutant data were incorporated into the model and the lags from 0 to 3 days and the 4-day means were examined. The shapes of the associations between mortality and pollutant were investigated using LOESS. As the associations were linear, the pollutants were treated as linear predictors.

The final model was used as a base model for two- and three-pollutant models if no significant interaction between the pollutants was present.

We carried out all the analyses using the statistical computing package S-PLUS 2000 (Mathsoft Inc, Cambridge, MA, USA).

Sensitivity analyses

We examined several variations in the models and variables to assess the sensitivity of the results to the chosen specifications. We excluded the spring season (February 15–April 30) from the two-pollutant model for TSP and TSP blackness in order to study the effect of excluding the "resuspended dust periods" (13) and included a dummy for extreme hot and cold days. These changes had little effect on the results. We also excluded the high pollution days, using the 98th percentile as the cut point separately for each pollutant, and we analyzed O_3 data stratified by season. Because of recently found problems in the convergence of the GAM, the data were partly reanalyzed with stricter convergence criteria (14). The changes in the results were small.

Results

Table 1 summarizes the basic data on mortality, air pollution, and meteorology during the study period. The daily mortality ranged from 4 to 35 deaths/day. A large proportion of the total mortality was due to cardiovascular deaths in the two age groups of elderly persons. The mean 24-hour concentrations of CO, SO_2, PM_{10}, and TSP declined steadily during 1988–1996, while the corresponding O_3 concentrations had an increasing trend and the NO_2 concentrations remained steady. The clearest decline in these long-term trends was observed in the annual average SO_2 concentration, which decreased from 21.0 to 5.6 μg/m^3.

The correlations between the 24-hour average pollutant concentrations and meteorological variables were generally lower than 0.6, as shown in table 2. Correlations higher than 0.6 were observed only between TSP and PM_{10}, PM_{10} and SO_2, and blackness and CO.

Table 1. Distribution of daily data on mortality, air pollution, and meteorology in the Helsinki metropolitan area in 1988–1996. (Min = minimum, Max = maximum, 95% CI = 95% confidence interval, O_3 = ozone, NO_2 = nitrogen dioxide, CO = carbon monoxide, SO_2 = sulfur dioxide, PM_{10} = particles with aerodynamic diameter of <10 μm, TSP = total suspended particulate matter)

	Days (N)	Min	25%	50%	75%	Max
Mortality (deaths/day)						
Total	3288	4	15	17	31	35
15- to -64-year age group	·	0	2	4	5	12
65- to -74-year age group	·	0	2	4	5	13
≥75-year age group	·	1	8	10	12	23
Cardiovascular	3288	1	7	9	11	20
15- to -64-year age group	·	0	1	1	2	7
65- to -74-year age group	·	0	1	2	3	9
≥75-year age group	·	0	4	5	7	15
Respiratory	3288	0	1	1	2	9
15- to -64-year age group	·	0	0	0	0	3
65- to -74-year age group	·	0	0	0	0	4
≥75-year age group	·	0	0	1	2	7
Air pollutants						
O_3 (μg/m^3)	3271	2	37	50	62	116
NO_2 (μg/m^3)	3288	6	26	33	42	176
CO (mg/m^3)	3269	0	0.9	1.2	1.6	12.4
SO_2 (μg/m^3)	3288	1	4	7	13	88
PM_{10} (μg/m^3)	745	0.2	15	21	30	213
TSP (μg/m^3)	1523	1	26	39	60	493
TSP blackness ($\times 10^{-6}$ units)	1519	0.5	2.3	2.8	3.2	6.1
Weather						
Temperature (°C)	3288	–21	0	5	13	25
Relative humidity (%)	3288	29	71	81	88	99

Table 2. Spearman rank correlation coefficients for the air pollutants and meteorological parameters. (O_3 = ozone, NO_2 = nitrogen dioxide, CO = carbon monoxide, SO_2 = sulfur dioxide, PM_{10} = particles with aerodynamic diameter of <10 µm, TSP = total suspended particulate matter)

	O_3	NO_2	CO	SO_2	PM_{10}	TSP	Blackness	Temperature	Relative humidity
O_3	1.00	–0.11	–0.46	–0.30	–0.09	0.15	–0.34	0.30	–0.45
NO_2		1.00	0.59	0.46	0.50	0.44	0.58	–0.07	0.02
CO			1.00	0.55	0.45	0.26	0.64	0.24	0.31
SO_2				1.00	0.61	0.48	0.55	–0.31	0.16
PM_{10}					1.00	0.72	0.56	–0.02	0.03
TSP						1.00	0.55	0.01	–0.21
Blackness							1.00	–0.11	0.13
Temperature								1.00	–0.37
Relative humidity									1.00

Table 3. Adjusted associations [a] between daily mortality and urban air pollutants at concentrations calculated for commonly used short-term averaging times. (95% CI = 95% confidence interval, O_3 = ozone, NO_2 = nitrogen dioxide, CO = carbon monoxide, SO_2 = sulfur dioxide, PM_{10} = particles with aerodynamic diameter of <10 µm, TSP = total suspended particulate matter)

	Total mortality		Cardiovascular mortality		Respiratory mortality	
	% change	95% CI	% change	95% CI	% change	95% CI
O_3 (maximum 8-hour average, µg/m^3)						
Lag time 0	0.66	0.16–1.16 [b]	0.26	–0.45–0.98	3.04	1.28–4.79 [b]
Lag time 1	0.37	–0.12–0.85	0.10	–0.59–0.79	1.22	–0.48–2.92
4-day mean	0.84	0.27–1.42 [b]	0.36	–0.46–1.19	3.27	1.25–5.29 [b]
NO_2 (24-hour average, µg/m^3)						
Lag time 0	–0.23	–0.88–0.41	–0.20	–1.12–0.72	1.43	–0.74–3.61
Lag time 1	0.23	–0.42–0.87	–0.35	–1.28–0.57	1.39	–0.78–3.55
4-day mean	0.02	–0.82–0.85	–0.04	–1.23–1.15	1.22	–1.59–4.04
CO (maximum 8-hour average, mg/m^3)						
Lag time 0	–1.50	–2.78––0.22 [b]	–2. 48	–4.30––0.66 [b]	–0.48	–4.84–3.87
Lag time 1	0.15	–1.09–1.39	–0.84	–2.61–0.93	–0.14	–4.43–4.15
4-day mean	–1.00	–2.80–0.81	–1.87	–4.43–0.69	–1.49	–7.73–4.74
SO_2 (24-hour average, µg/m^3)						
Lag time 0	–0.03	–1.15–1.09	0.78	–0.78–2.34	1.38	–2.48–5.25
Lag time 1	0.08	–1.01–1.17	0.70	–0.82–2.22	1.30	–2.46–5.06
4-day mean	0.02	–1.34–1.38	1.23	–0.68–3.13	–0.18	–4.93–4.58
PM_{10} (24-hour average, µg/m^3)						
Lag time 0	–0.23	–1.47–1.01	–1.22	–3.00–0.56	3.94	0.01–7.87 [b]
Lag time 1	0.88	–0.32–2.08	0.63	–1.09–2.35	3.96	0.11–7.81 [b]
4-day mean	0.11	–0.51–0.73	–0.08	–0.96–0.81	2.13	0.03–4.22 [b]
TSP (24-hour average, µg/m^3)						
Lag time 0	0.01	–0.38–0.40	0.01	–0.53–0.56	–0.54	–1.93–0.85
Lag time 1	–0.25	–0.62–0.13	–0.46	–1.00–0.08	0.61	–0.66–1.88
4-day mean	–0.17	–0.48–0.14	–0.39	–0.83–0.05	0.03	–1.05–1.11
TSP blackness (24-hour average, units)						
Lag time 0	–2.07	–3.88––0.27 [b]	–1. 35	–3.90–1.21	–4. 74	–11.02–1.54
Lag time 1	1.24	–0.54–3.02	0.60	–1.93–3.14	2.11	–4.01–8.23
4-day mean	–0.64	–2.13–0.85	–0.81	–2.92–1.30	–0.36	–5.53–4.81

[a] The associations were calculated per 10-unit increase in pollutant concentration, except for CO (per 1 unit) and TSP blackness (per 0.00001 unit). All the models were adjusted for long-term time trend, temperature, relative humidity, weekday, and influenza episodes.
[b] $P<0.05$.

Single pollutant models

Table 3 shows the adjusted associations between the daily variations in mortality and urban air pollutant concentrations. Ozone was positively associated with both respiratory mortality and total mortality. SO_2 was rather consistently, but not significantly, associated with cardiovascular mortality across the 0-to-3-day lag times and the 4-day mean. This association appeared to be mainly due to mortality in the 65- to 74-year age group (data not shown). CO and NO_2 were not consistently associated with any mortality measure, although some statistically significant associations were observed.

From the measures of particulate air pollution, only PM_{10} was consistently associated with respiratory

mortality for the 0–1 lag times and the 4-day mean, but the associations with total and cardiovascular mortality were not consistent (table 3).

There were no consistent associations between TSP and mortality in the single-pollutant models (table 3). A significant negative association between total mortality and TSP blackness was observed at lag time 0, but the association became nonsignificant and the estimate was reduced by about 20% when days with blackness above the 98th percentile were excluded or the model was adjusted for the TSP concentration. The blackness of the TSP was positively, but not significantly, associated with total, cardiovascular, and respiratory mortality at lag time 1. However, no association was observed at lag times 2 and 3 (data not shown), or for the 4-day mean. It should be noted that lag times 0 and 1 cannot be directly compared, since, due to missing data, there were only 2 days during the study period with data on TSP or TSP blackness for both lag time 0 and lag time 1.

Seasonal analysis of ozone and mortality

Table 4 shows the results of the seasonal analysis of the association between mortality and the O_3 concentration, and also the effect of adjustment for co-pollutants. There were significant positive associations between total mortality and the O_3 concentration for lag times 0–3 and for the 4-day mean during the period from April to July. The age-group-specific analysis revealed that the associations in the age groups 15–64 and ≥75 years were consistent, whereas few associations were observed in the 65- to 74-year age group (data not shown). Adjusting for NO_2, TSP, or TSP blackness (data not shown) did not essentially change the whole-year effect estimates presented in table 3.

Multipollutant models for total suspended particulates

Table 5 shows the results from two-pollutant models including both TSP measures. TSP was negatively (–0.41% per 10 μg/m^3, 95% CI –0.82—0.01) and TSP blackness positively (2.06% per 10^{-5} units, 95% CI 0.09–4.06) associated with total mortality at lag time 1. Similar associations were observed for cardiovascular mortality.

Although there was no significant interaction between TSP and TSP blackness (P=0.18) at lag time 1, there was a suggestion that blackness was associated with total mortality especially at low TSP concentrations. When the two TSP measures were divided into tertiles, the strongest association with total mortality was observed for days with a low TSP concentration and high TSP blackness (figure 1). There were 51 such days in 1988–1996, occurring mostly during late fall and winter (figure 2). The maximum 8-hour average CO concentration (0.90 mg/m^3) was increased on these days when compared with the levels on all other days (0.50 mg/m^3).

Finally, we analyzed three-pollutant models, including TSP, TSP blackness, and one of the gaseous pollutants. In these models, the positive association of TSP blackness with total mortality was further increased (data not shown). Likewise, cardiovascular and respiratory mortality was consistently, but not significantly, associated with TSP blackness at lag time 1. In these models, there were no significant associations between daily mortality and the O_3 or TSP concentration.

Table 4. Association [a] between daily mortality and daily maximum 8-hour average ozone (O_3) concentration as stratified by season or adjusted for co-pollutants in 1988–1996. (95% CI = 95% confidence interval, NO_2 = nitrogen dioxide, TSP = total suspended particulate matter)

	Restricted (April-July)		Restricted (August-March)		Adjusted for NO_2		Adjusted for TSP and TSP blackness	
	Change (%)	95% CI	Change (%)	95% CI	Change (%)	95% CI	Change (%)	95% CI
Total mortality								
Lag time 0	1.07 [b]	0.15–2.00	0.28	–0.38–0.94	0.66 [b]	0.15–1.16	0.43	–0.40–1.26
Lag time 1	1.19 [b]	0.28–2.11	–0.24	–0.88–0.40	0.40	–0.09–0.89	0.80	–0.01–1.61
Lag time 2	1.10 [b]	0.19–2.02	–0.02	–0.65–0.62	0.47	–0.01–0.95	0.20	–0.60–1.00
Lag time 3	1.49 [b]	0.58–2.41	–0.08	–0.71–0.56	0.57 [b]	0.09–1.05	1.22 [b]	0.42–2.02
4-day mean	2.42 [b]	1.27–3.58	–0.03	–0.81–0.76	0.86 [b]	0.27–1.45	1.04 [b]	0.37–1.71
Respiratory mortality								
Lag time 0	3.06	–0.18–6.41	0.76	–1.54–3.11	3.22 [b]	1.43–5.05	3.35 [b]	0.43–6.35
Lag time 1	1.36	–1.83–4.64	–1.28	–3.47–0.96	1.37	–0.34–3.11	1.11	–1.67–3.98
Lag time 2	1.51	–1.68–4.80	–0.93	–3.09–1.27	1.44	–0.23–3.14	0.28	–2.45–3.09
Lag time 3	2.58	–0.64–5.91	–0.06	–2.23–2.16	2.19	0.51–3.90	3.64 [b]	0.82–6.54
4-day mean	4.30 [b]	0.21–8.56	–0.80	–3.47–1.95	3.33 [b]	1.25–5.45	3.25 [b]	0.90–5.65

[a] All the models were adjusted for long-term time trend, temperature, relative humidity, weekday, and influenza episodes. The associations were calculated per 10 μg/m^3 increase in O_3 concentration.
[b] P<0.05.

Discussion

We did a retrospective analysis of the associations between urban air-pollutant concentrations and daily mortality in the Helsinki metropolitan area during 1988–1996. We found consistent associations between O_3 and respiratory and total mortality. PM_{10} was consistently associated with respiratory mortality, but TSP or TSP blackness was not associated with mortality in single pollutant models. However, in two-pollutant models including TSP mass concentration, TSP blackness was associated with total mortality at lag time 1.

The total population of the Helsinki metropolitan area increased steadily from 816 733 to 905 545 inhabitants during the 9-year study period. The number of

Table 5. Adjusted associations [a] between daily mortality and the 24-hour average concentrations of total suspended particulates (TSP) and TSP blackness. (95% CI = 95% confidence interval)

	Total mortality		Respiratory mortality	
	Change (%)	95% CI	Change (%)	95% CI
Lag time 0				
TSP (24-hour)	0.22	–0.21– 0.65	–0.01	–1.51–1.52
TSP blackness (24-hour)	–2.52 [b]	–4.45––0.55	–5.26	–11.67–1.60
Lag time 1				
TSP (24-hour)	–0.41	–0.82–0.01	0.37	–1.03–1.79
TSP blackness (24-hour)	2.06 [b]	0.09–4.06	1.38	–5.21–8.42
Lag time 2				
TSP (24-hour)	–0.13	–0.55–0.29	–0.28	–1.71–1.17
TSP blackness (24-hour)	–0.57	–2.50–1.40	–1.60	–8.07–5.33
Lag time 3				
TSP (24-hour)	–0.27	–0.68–0.15	–0.24	–1.69–1.22
TSP blackness (24-hour)	0.23	–1.70–2.19	–0.36	–6.86–6.59
4-day average				
TSP (24-hour)	–0.13	–0.46–0.21	0.10	–1.06–1.27
TSP blackness (24-hour)	–0.55	–2.14–1.07	–1.06	–6.46–4.65

[a] The associations were calculated per 10 µg/m³ increase in TSP concentration and per 10^{-5} unit increase in TSP blackness. All the models were adjusted for long-term time trend, temperature, relative humidity, weekday, and influenza episodes.
[b] P<0.05.

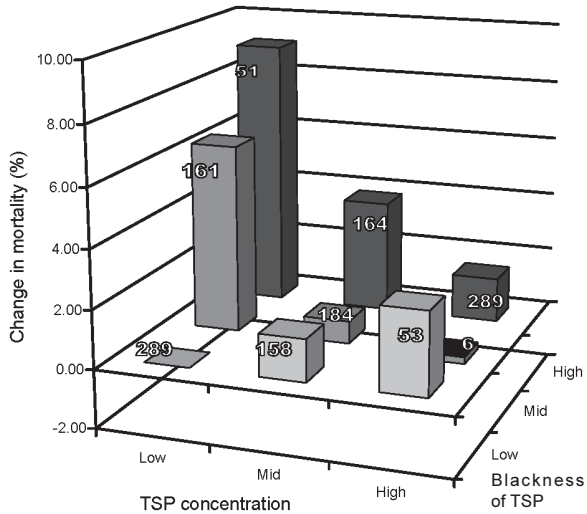

Figure 1. Adjusted percentage change in total daily mortality, shown in tertiles of total suspended particulates (TSP) and TSP blackness at lag time 1. The tertile cut points of TSP were 30.1 and 50.9 µg/ m³. The number of days in each tertile is given on the bars.

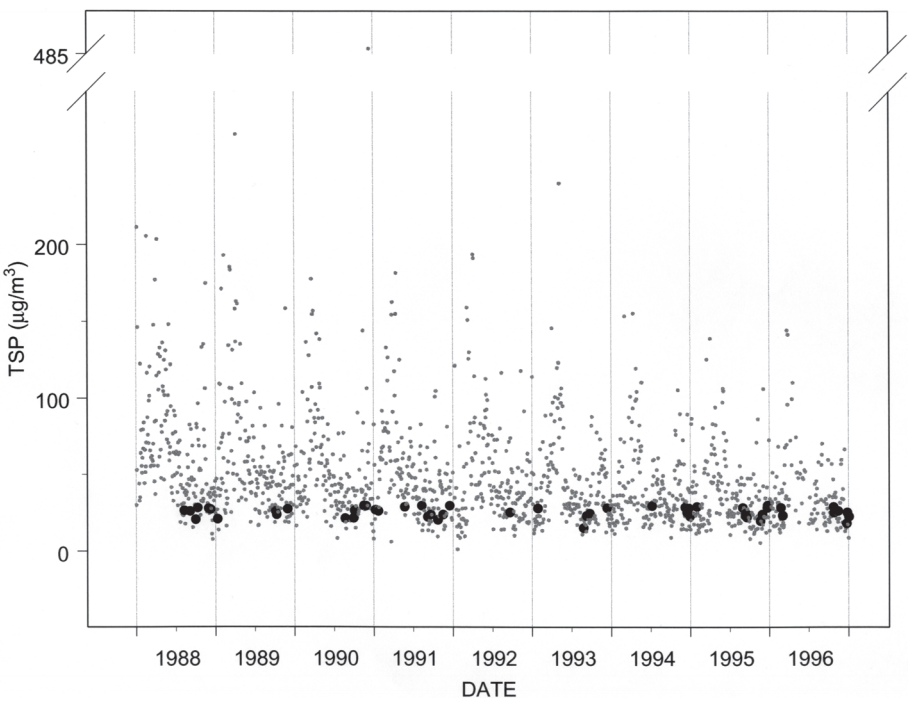

Figure 2. Twenty-four-hour average concentrations of total suspended particles (TSP) by date. Data points for days when TSP were in the lowest tertile (<30 µg/m³) and TSP blackness was in the highest tertile (blackness >3×10^{-6}) are highlighted with black color.

daily deaths ranged from 4 to 35 deaths/day, being equivalent to a daily mortality of $4.4/10^6$–$38.7/10^6$. The cardiovascular mortality accounted for 1–20 deaths per day and respiratory mortality for 0–9 deaths/day.

Helsinki is situated by the Baltic Sea, with prevailing southwesterly winds. The ambient temperature ranges from –21°C in winter to 25°C in summer, and the relative humidity is generally highest during winter days. In these climatic conditions, the highest O_3 concentrations were observed during the spring and summer months, and, to a large extent, they reflected long-range transport. The SO_2 concentrations reflected declining emissions from stationary sources (energy production, industry), while the CO and NO_2 concentrations mainly derived from local automotive engine emissions. Particle sources in the Helsinki area include long-range transport, energy production, traffic emissions, and resuspended road dust occurring especially during late fall and early spring (15–16). In our data, the PM_{10} levels were relatively highly correlated with the SO_2 and TSP concentrations, partly due to similar declining, long-term trends. The TSP blackness had the highest correlation with CO; this finding suggests that combustion-derived soot particles made a major contribution to this measure.

The daily maximum 8-hour average O_3 concentration was significantly associated with respiratory and total mortality in our data. The magnitude of the observed effect estimates (table 3) resembled the recent estimates made in other European cities (9). A seasonal analysis of our data (table 4) indicated that mortality was consistently associated with the O_3 concentration only during the spring and summer months. This finding agrees with the results of a recent analysis of data on 20 cities in the United States, in which a 10 $\mu g/m^3$ increase in the O_3 concentration was associated with a 0.41% increase in daily mortality during the hot summer months, but not throughout the year (2). However, not all studies have observed associations between mortality and O_3 (11). It is likely that the differences in the climate and, thereby in the atmospheric processes leading to O_3 formation, explain the differences between the studies. The urban air O_3 concentrations in Europe are determined to a large extent by long-range transport, the amount of ultraviolet radiation (season), and the concentrations of locally produced O_3-scavenging nitric oxides (17).

In two recent multicity studies, the increases, by 10 $\mu g/m^3$, in PM_{10} have been associated with 0.42% and 0.51% increases in total mortality in western Europe and in the United States, respectively (1, 2). In our study, PM_{10} was significantly associated with respiratory mortality in all age groups (consistent across lag times 0–3 in the age groups 64–75 and ≥75) and total mortality in the 64-to-75-year age group (lag times 1 and 2). Other particulate measures were not associated with mortality in the single-pollutant analysis. In the Helsinki area, as in other cities of Finland, TSP and PM_{10} measurements are greatly influenced by the resuspended, coarse road dust caused by sand spread on streets and studded tires used on cars during wintertime to prevent slipperiness (7, 13, 15–16). According to recent analyses, coarse particles might not be associated with mortality to the same extent as finer, combustion-related particles (5, 6). This difference could explain the lack of a consistent association between total mortality and PM_{10} in our data. It is also important to note that we had much fewer data on PM_{10} than on the other pollutants.

Because of the effect of resuspended road dust and the lack of data on fine particle concentrations in the Helsinki area, we retrieved archived TSP filters and used a simple optical method, analogous to black smoke measurements, to determine the blackness of the TSP filters (8). The measured TSP blackness was used as a surrogate for fine elemental carbon particles mostly derived from local traffic emissions. However, the blackness was not associated with mortality in the single pollutant models (table 3).

The measurement of TSP blackness is affected also by noncombustion particles (8) and, therefore, two- and three-pollutant models were also used in our analyses. In the two-pollutant models, there was a suggestion that, at low TSP concentrations, TSP blackness was associated with total mortality at lag time 1 (figure 1). In the three-pollutant models, which included also one gaseous pollutant in addition to TSP and TSP blackness, the association of TSP blackness with total mortality was even strengthened. This finding is supported by recent time-series studies in which fine, combustion-derived particles, rather than coarse mineral particles, have been associated with mortality (5, 6, 18). Previous Finnish studies on the associations of particulate air pollution with respiratory symptoms, lung function, and the medication of asthmatic adults and children have also suggested that the adverse effects are specific to the fine or ultrafine fraction of particulate matter (19–23). In addition, a recent case–referent study from Stockholm has identified organic combustion products as a risk factor for myocardial infarction (24).

In a previous analysis of the associations between daily variations in air pollution and mortality in the city of Helsinki, Pönkä and his co-workers (25) reported that a 10 $\mu g/m^3$ increase in the PM_{10} concentration was associated with a 3.5% increase in daily total mortality and a 4.1% increase in daily cardiovascular mortality. In addition, respiratory mortality was positively associated with the PM_{10} concentration. All of these associations were observed only for the <65-year age group and only with a lag time of 5 days between the exposure and date of death. We decided to analyze our data using lags of

0–3 days, mainly because most of the significant associations between air pollution and mortality in previous studies have been reported for lag times shorter than 3 days. We observed positive associations between PM_{10} and total mortality only for the 65- to 74-year age group at lag times 1 and 2. In both of these studies, the number of days with available PM_{10} data from the same monitoring site was low. In the study of Pönkä et al (25) the missing values were imputed, while only days with measured air pollution data were used in our study. In addition we had data on mortality from the Helsinki metropolitan area in 1988–1996 (population 905 545 in 1996), whereas Pönkä et al (25) studied mortality only in the city of Helsinki (population 525 031 in 1996). These differences in the methods and database probably explain the differences in the results.

In the analysis of 63 models, as shown in table 3, one can expect three statistically significant estimates at the $P<0.05$ level even by chance. Therefore, a cautious interpretation should be made of the observed significant associations in this table. Another reason for caution is that the median numbers of daily deaths due to respiratory mortality were very low. Indeed it was necessary to conduct several sensitivity analyses on the positive associations of mortality with O_3 (table 4) and combustion-derived particles (figures 1 and 2 and table 5).

We conclude from this retrospective analysis of mortality and air pollution in the Helsinki metropolitan area in 1988–1996 that the O_3 and PM_{10} concentrations were positively associated with daily total or respiratory mortality. The concentrations of other gaseous pollutants or TSP were not consistently associated with mortality. Our results from the use of the blackness of TSP filters as a surrogate measure of combustion particles supported the findings of previous studies, which indicated that coarse mineral particles are not as strongly associated with mortality as fine, combustion-derived particles are.

Acknowledgments

This study has been supported by the Academy of Finland, Finnish Research Programme on Environmental Health (SYTTY); the Centre of Excellence Programmes 2002–2007 of the Academy of Finland; and the National Technology Fund (TEKES). We thank the Helsinki Metropolitan Area Council (YTV) for providing the TSP filters and the data on air quality, Statistics Finland for providing mortality data, and the Finnish Meteorological Institute for providing meteorological data.

References

1. Katsouyanni K, Touloumi G, Spix C, Schwartz J, Balducci F, Medina S, et al. Short-term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. BMJ 1997;314:1658–63.
2. Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL. Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. New Engl J Med 2000;343:1742–9.
3. Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 2002;287:1132–41.
4. Dockery DW, Pope CAI, Xu X, Spengler JD, Ware JH, Fay ME, et al. An association between air pollution and mortality in six U.S. cities. New Engl J Med 1993;329:1753–9.
5. Schwartz J, Dockery DW, Neas LM. Is daily mortality associated specifically with fine particles? J Air Waste Manage Assoc 1996;46:927–39.
6. Laden F, Neas LM, Dockery DW, Schwartz J. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. Environ Health Perspect 2000; 108:941–7.
7. Hämekoski K, Salonen RO. Particulate matter in Northern Climate of Helsinki metropolitan area, Finland. In: California Air Resources Board. Proceedings of the Second Colloquium on Particulate Air Pollution and Human Health. California Air Resources Board; 1996. [Available at: http://www.arb.ca.gov/research/abstracts/95-323.htm#Ch95-323 (last accessed on 2 June 2004)]
8. Penttinen P, Alm S, Ruuskanen J, Pekkanen J. Measuring reflectance of TSP-filters for retrospective health studies. Atmos Environ 2000;34:2581–6.
9. Katsouyanni K, Touloumi G, Samoli E, Gryparis A, Le Tertre A, Monopolis Y, et al. Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA-2 project. Epidemiology 2001;12:521–31.
10. Hoek G, Schwartz J, Groot B, Eilers P. Effects of ambient particulate matter and ozone on daily mortality in Rotterdam, The Netherlands. Arch Environ Health 1997;52:455–63.
11. Ostro BD, Hurley S, Lipsett MJ. Air pollution and daily mortality in the Coachella Valley, California: a study of PM_{10} dominated by coarse particles. Environ Res 1999;81:231–8.
12. Katsouyanni K, Schwartz J, Spix C, Touloumi G, Zmirou D, Zanobetti A, et al. Short-term effects of air pollution on health: a European approach using epidemiologic time series data. The APHEA protocol. J Epidemiol Community Health 1996;50:12–8.
13. Hosiokangas J, Ruuskanen J, Pekkanen J. Effects of soil dust episodes and mixed fuel sources on source apportionment of PM_{10} particles in Kuopio, Finland. Atmos Environ 1999:33:3821–9.
14. Dominici F, McDermott A, Zeger SL, Samet JM. On the use of generalized additive models in time series studies of air pollution and health [commentary]. Am J Epidemiol 2002;156:193–203.
15. Vallius MJ, Ruuskanen J, Mirme A, Pekkanen J. Concentrations and estimated soot content of PM_1, $PM_{2.5}$, and PM_{10} in a subarctic urban atmosphere. Environ Sci Technol 2000: 34:1919–25.
16. Pakkanen TA, Loukkola K, Korhonen CH, Aurela M, Mäkelä T, Hillamo R, et al. Sources and chemical composition of

atmospheric fine and coarse particles in the Helsinki area. Atmos Environ 2001;35:5381–91.
17. Syri S, Amann M, Schöpp W, Heyes C. Estimating long-term population exposure to ozone in urban areas of Europe. Environ Poll 2001;113:59–69.
18. Andersson HR, Bremner SA, Atkinson RW, Harrison RM, Walters S. Particulate matter and daily mortality and hospital admissions in the West Midlands conurbation of the United Kingdom: associations with fine and coarse particles, black smoke and sulfate. Occup Environ Med 2001;68:504–10.
19. Penttinen P, Timonen KL, Tiittanen P, Mirme A, Ruuskanen J, Pekkanen J. Number concentrations and size of particles in urban air: effects on spirometric lung function in adult asthmatic subjects. Environ Health Perspect 2001;109:319–23.
20. Penttinen P, Timonen KL, Tiittanen P, Mirme A, Ruuskanen J, Pekkanen J. Ultrafine particles in urban air and respiratory health in adult asthmatics. Eur Respir J 2001;17:428–35.
21. Tiittanen P, Timonen KL, Ruuskanen J, Mirme A, Pekkanen J. Fine particulate air pollution, resuspended road dust and respiratory health among symptomatic children. Eur Respir J 1999;13:266–73.
22. Timonen KL, Pekkanen J. Air pollution and respiratory health among children with asthmatic or cough symptoms. Am J Respir Crit Care Med 1997;156:546–52.
23. Zanobetti A, Schwartz J, Gold D. Are there sensitive subgroups for the effects of airborne particles? Environ Health Perspect 2000;108:841–5.
24. Gustavsson P, Plato N, Hallqvist J, Hogstedt C, Lewné M, Reuterwall C, et. al. A population-based case-referent study of myocardial infarction and occupational exposure to motor exhaust, other combustion products, organic solvents, lead, and dynamite. Epidemiology 2001;12:222–8.
25. Pönkä A, Savela M, Virtanen M. Mortality and air pollution in Helsinki. Arch Environ Health 1998:53;281–6.