



APPENDIX AVAILABLE ON THE HEI WEB SITE

Research Report 183

Development of Statistical Methods for Multipollutant Research

Part 2. Development of Enhanced Statistical Methods for Assessing Health Effects Associated with an Unknown Number of Major Sources of Multiple Air Pollutants

E.S. Park et al.

Appendix B. Summary of Studies Evaluating Health Effects Associated with Source-Apportioned Particulate Matter (PM)

Note: Appendices available only on the Web have been reviewed solely for spelling, grammar, and cross-references to the main text. They have not been formatted or fully edited by HEI.

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This document was reviewed by the HEI Health Review Committee.

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Table B.1: Summary of Studies Evaluating Health Effects Associated with Source-Appportioned Particulate Matter (PM)

Study	Location Time period	Outcome	Pollutants	Source-appportionment Method/Health Effects Model	Effect Measure	Comments
Laden et al. 2000	Watertown, MA; Kingston-Harriman, TN; St. Louis, MO; Steubenville, OH; Portage, WI; Topeka, Kansas 1979–1988	Total nonaccidental mortality (ICD-9 < 800) Ischemic heart disease (ICD-9: 410–414) Pneumonia (ICD-9: 480–486) Chronic obstructive pulmonary disease (ICD-9: 490–496)	PM _{2.5} : Si, S, Cl, K, V, Ma, Al, Ni, Zn, Se, Br, Pb, Cu, Fe (from a single monitor at a central location in each city)	Factor analysis (using Procrustes rotation) Three sources identified in all 6 cities: mobile source (lead); coal combustion source (Se); and crustal source (Si) Poisson generalized additive time-series model with LOESS functions of date, temperature and dew point temperature; indicator variables for day-of-the-week.	% increase in daily deaths per 10- $\mu\text{g}/\text{m}^3$ increase in mass concentration of PM _{2.5} (2-day mean [same and previous day]) Crustal sources (Si): -2.3 (-5.8 to 1.2) Motor sources (Pb): 3.4 (1.7–5.2) Coal sources (Se): 1.1 (0.3–2.0)	
Mar et al. 2000	Phoenix, AZ (Maricopa County) 1995–1997	Total nonaccidental mortality among individuals aged 65 years and older (ICD-9 < 800) 8.55 deaths/day Cardiovascular mortality (ICD-9: 390–448.9) 3.85 deaths/day	PM _{2.5} , PM ₁₀ , PM _{CF} (PM ₁₀ -PM _{2.5}), PM _{2.5} chemical composition data (from a single monitoring station in central Phoenix): S, Zn, Pb, soil K _S , nonsoil PM, OC, EC, TC and soil (summing oxides of Al, Si, Ca, Fe, Ti) CO (4 sites), NO ₂ (2 sites), O ₃ , SO ₂ (1 site)	Factor analysis using principal component analysis with a varimax rotation Five sources: motor vehicle exhaust and resuspended road dust; soil; vegetative burnings; local sulfate; regional sulfate Poisson Regression (adjusted for day-of-	RR per IQR increase in pollutant Total mortality: S (280.6 $\mu\text{g}/\text{m}^3$) (lag 3): 0.96 (0.93–1.00) Soil (1,767.45 $\mu\text{g}/\text{m}^3$) (lag 1): 0.97 (0.94–1.00) PM ₁₀ (24.88 $\mu\text{g}/\text{m}^3$) (lag 0): 1.03 (1.00–1.05) PM _{CF} (18.39 $\mu\text{g}/\text{m}^3$) (lag 0) 1.02 (1.00–1.05)	Only statistically significant ($P < 0.05$) or marginally significant ($P < 0.10$) results reported For total mortality, results for S (lag 4) and Soil (lags 2–4) also reported For cardiovascular mortality, results for PM ₁₀ (lag 1), PM _{2.5}

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Study	Location Time period	Outcome	Pollutants	Source- apportionment Method/Health Effects Model	Effect Measure	Comments
				<p>the-week with indicator variables and time trends, temperature, and relative humidity with smoothing functions</p> <p>Lag days from 0 to 4</p>	<p>Pb (6.00 $\mu\text{g}/\text{m}^3$) (lag 3): 0.98 (0.97–1.00)</p> <p>Cardiovascular mortality:</p> <p>K_s (55.62 $\mu\text{g}/\text{m}^3$) (lag 3): 1.03 (1.00–1.07)</p> <p>PM_{10} (24.88 $\mu\text{g}/\text{m}^3$) (lag 0): 1.05 (1.01–1.09)</p> <p>$\text{PM}_{2.5}$ (8.52 $\mu\text{g}/\text{m}^3$) (lag 0): 1.03 (0.99–1.08)</p> <p>PM_{CF} (18.39 $\mu\text{g}/\text{m}^3$) (lag 0) 1.05 (1.01–1.09)</p> <p>Nonsoil $\text{PM}_{2.5}$ (6601.06 $\mu\text{g}/\text{m}^3$) (lag 1): 1.04 (1.00–1.08)</p> <p>OC (2976.50 $\mu\text{g}/\text{m}^3$) (lag 1): 1.04 (1.00–1.09)</p> <p>EC (1165.50 $\mu\text{g}/\text{m}^3$) (lag 1): 1.05(1.01–1.10)</p> <p>TC (4169.00 $\mu\text{g}/\text{m}^3$) (lag 1): 1.05(1.01–1.09)</p> <p>Total mortality:</p> <p>Factor 2 (soil with high loadings on Al, Si, Fe) (lag 1) (IQR = 1.26 $\mu\text{g}/\text{m}^3$): 0.96 (0.93–1.00)</p>	<p>(lags 1, 3, 4), OC (lag 3) and TC (lag 3) also reported</p> <p>For total mortality, results for Factor 2 also reported for lags 2, 3, and 4; for Factor 4, for lag 4, and Factor 5, for lag 3</p>

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Study	Location Time period	Outcome	Pollutants	Source- apportionment Method/Health Effects Model	Effect Measure	Comments
					<p>Factor 4 (local source of SO₂ with a high loading on SO₂) (lag 2) (IQR = 1.09 µg/m³): 0.97 (0.94–1.00)</p> <p>Factor 5 (regional sulfate with a high loading on S) (IQR = 1.38 µg/m³): 1.04 (1.01–1.08)</p> <p>CVD mortality:</p> <p>Factor 1 (motor vehicle exhaust and resuspended road dust) (Mn, Fe, Zn, Pb, OC, EC, CO, NO₂) (lag 1) (IQR = 1.11 µg/m³): 1.06 (1.01–1.10)</p> <p>Factor 3 (vegetative burning with high loadings on OC and K_s) (lag 3) (IQR = 1.02 µg/m³): 1.05 (1.01–1.09)</p> <p>Factor 5 (regional sulfate with a high loading on S) (lag 0) (IQR = 1.38 µg/m³): 1.06 (1.00–1.12)</p>	
Thurston et al. 2005	Washington D.C. and surrounding 6 counties (8/1988–12/1997) and Phoenix, AZ (3/1995–6/1998)	Washington, D.C. Total nonaccidental Cardiovascular Cardiovascular plus respiratory	PM _{2.5} samples (Phoenix): Na, Mg, Al, Si, P, S, Cl, K, Ca, scandium (Sc), Ti, V, Cr, Mn, Fe, Co, Ni, Cu, Zn, Ga, Ge, Ar, Se, Br, Rb, Sr, Y, Zr, Mo, Rh, Pd, Ag, Cd, Sn, Sb, Te, I, Cs, Ba, La, W, Au, Hg, Pb, organic carbon, elemental carbon	7 groups evaluated the same set of data using different source-apportionment analyses including: Unmix, positive matrix factorization (PMF2) and expanded model	Washington: soil, secondary sulfate and nitrate, oil burning, and incineration were highly correlated across analyses; wood burning, salt, and traffic were less correlated across analyses. Phoenix: soil, traffic, secondary sulfate, and sea spray were most highly correlated; wood and	Results from a U.S. EPA-sponsored workshop to investigate source-apportionment and health effects analyses

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Study	Location Time period	Outcome	Pollutants	Source- apportionment Method/Health Effects Model	Effect Measure	Comments
		Phoenix: Total nonaccidental (CD-9 < 800.00) Cardiovascular (ICD-9: 390.00–448.99)	Washington, D.C. samples; Na, Mg, Al, Si, P, S, Cl, K, Ca, Sc, Ti, V, Cr, Mn; Fe, Co, Ni, Cu, Zn, Ga, Ge, As, Se, Br, Rb, Sr, Y, Zr, Mo, Rh, Pd, Ag, Cd, Sn, Sb, Te, I, Cs, Ba, La, W, Au, Hg, Pb	(ME), target rotated PCA, PMF, absolute principal-component analysis (APCA), iterated, confirmatory factor analysis (FA), single-elemental multiple regression Poisson generalized linear model with natural splines of time to adjust for seasonal trends and unmeasured seasonal confounders. Natural splines of the same-day temperature, natural splines of the average of lags 1–3 of daily temperature to fit “cold” temperature effects; an indicator variable for “hot” and “humid” days.	vegetative burning, metals industry particles, and coal fly ash were less well correlated. Between-source variation in daily mortality RR was larger than the between-research group variation in estimated RRs. Most source categories were nonsignificant. The most strongly associated source category was sulfate-associated mass: % increase in total mortality per 10 µg/m ³ increase was 5.2% in Phoenix and 3.8% in Washington Motor vehicle exhaust approached significance. Per 10 µg/m ³ , total mortality increased 0.9% in Phoenix and 4.2% in Washington.	
Ito et al. 2006	Washington D.C. and surrounding counties 8/1988–12/1997	Total nonaccidental Cardiovascular Cardiovascular plus respiratory	PM _{2.5} mass, 24 trace elements, anions (sulfate, nitrate) and cations (particulate ammonium) PM ₁₀ , O ₃ , SO ₂ , NO ₂ , CO	Absolute principal components analysis; positive matrix factorization; Unmix; target-rotation (or specific-rotation) factor analysis; confirmatory factor analysis	Variance-weighted mean percent excess deaths per 5–95 th percentile increment of apportioned PM _{2.5} for total mortality Secondary sulfate: 6.7% (95% CI: 1.7–11.7) (lag 3) (largest effect)	Results from a U.S. EPA-sponsored workshop to investigate source-apportionment and health effects analyses

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Study	Location Time period	Outcome	Pollutants	Source- apportionment Method/Health Effects Model	Effect Measure	Comments
					<p>Primary coal-related PM_{2.5} (3 teams): 5.0% (95% CI: 1.0–9.1) (lag 3)</p> <p>Residual oil factor: 2.7% (95% CI: –1.1 to 6.5) (lag 2) (most consistent positive estimate across teams)</p> <p>Traffic-related PM_{2.5}: 2.6% (95% CI: –1.6 to 6.9)</p> <p>Soil-related PM_{2.5}: 2.1% (95% CI: –0.8 to 4.9)</p>	
Seagrave et al. 2006	Southeastern U.S. Birmingham, AL site; Atlanta, GA site; Pensacola, FL site; Centreville, AL site	Animal (rat) model: General toxicity, acute cytotoxicity and inflammation (instillation of samples into rat lungs)	PM _{2.5} samples (X-ray fluorescence for speciation)	<p>Chemical Mass Balance Receptor model</p> <p>Sources considered: emissions from diesel and gasoline engines, wood combustion, paved road dust, meat cooking, vegetative detritus, natural gas combustion, and emissions from coke facilities</p> <p>OC, EC, ammonium, NO₃⁻, SO₄⁻, Ar, Br, Cu, Mn, Pb, Se, Titanium dioxide, Zn, composite of metal oxides</p>	<p>PLS analysis using chemical predictors: OC, Pb and hopanes/steranes were the most important predictors with NO₃⁻ and As influencing the 1st component and MMOs influencing the 2nd component.</p> <p>The 1st component more strongly affected the cytotoxic responses, whereas the second component more strongly affected the inflammatory responses</p> <p>PLS analysis using the source-apportionment results: gasoline emissions were the most important predictor for 1st and 2nd component, whereas diesel more strongly influenced the 2nd component and secondary NO₃⁻ influenced the 1st components. Greater influence of</p>	

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Study	Location Time period	Outcome	Pollutants	Source- apportionment Method/Health Effects Model	Effect Measure	Comments
				<p>(MMOs) were considered as separated predictors</p> <p>Separate analysis used the CMB-attributed sources as predictors</p> <p>Relative toxicity rankings by site and projection-to-latent-surfaces (PLS) analysis</p>	<p>the 1st component on cytotoxic responses and of the 2nd component on inflammatory responses.</p>	
Mar et al. 2006	Phoenix, AZ 3/1995–6/1998	<p>Total nonaccidental (ICD-9 < 800.00)</p> <p>Cardiovascular (ICD-9: 390.00–448.99) 2/9/1995–12/31/97</p>	PM _{2.5} speciation data	Poison generalized linear models with indicator variables for extreme temperatures, day-of-the-week, and smoothing terms for time trends, temperature, and relative humidity	<p>Median percent excess deaths per 5–95th percentile increment of apportioned PM_{2.5} for total mortality</p> <p>Cardiovascular mortality</p> <p>Secondary sulfate factor: 16.0% (lag 0) (8 investigators)</p> <p>Traffic: 13.2% (lag 1) (9 investigators)</p> <p>Cu smelter factor: 12.0% (lag 0) (5 investigators)</p> <p>Sea salt factor: 10.2% (lag 5) 6 investigators</p> <p>Biomass/wood combustion factor: 8.6% (lag 3) 8 investigators</p>	Results from a U.S. EPA-sponsored workshop to investigate source-apportionment and health effects analyses

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Study	Location Time period	Outcome	Pollutants	Source- apportionment Method/Health Effects Model	Effect Measure	Comments
					Soil factor: no association Total nonaccidental mortality Fewer statistically significant associations (Cu smelter and sea salt factors showed some significant associations with lag structures ... similar to those for cardiovascular mortality but with smaller effect sizes)	
Andersen et al. 2007	Copenhagen, Denmark, 1999–2004	Hospital admissions in elderly (≥ 65 years) and children (5–18 years (asthma)) Angina pectoris (ICD-10: I20) Acute and subsequent myocardial infarction (ICD-10: I21–22) Acute Ischemic heart diseases (ICD-10: I24) Chronic Ischemic heart disease (ICD-10: I25)	CO, NO ₂ , O ₃ , PM ₁₀ (one station) and Total suspended particulates (TSP) (forest rural station 35 km from Copenhagen) Chemical composition data on PM (May 2002–Dec 2003); Al, Si, S, Cl, K, Ca, Ti, V, Cr, Mn, Fe, Ni, Cu, Zn, Ga, As, Se, Br, Rb, Sr, Zr, Mo, Sn, Sb, Ba, Pb, Na ⁺ , NH ₄ ⁺ , Cl ⁻ , SO ₄ ²⁻	Principal Component Analysis and the constrained physical receptor model (COPREM) Six main PM ₁₀ sources: biomass combustion, oil (fuel oil combustion), vehicle (exhaust emissions and brake wear emissions) sea salt; crustal (road concrete and tire wear and igneous rock and limestone); secondary inorganic compounds Poisson generalized additive time-series model (adjusted for season, day-of-the-week, public	RR per IQR of pollutant Jan 1999–Dec 2004 PM ₁₀ (14 $\mu\text{g}/\text{m}^3$) CVD admissions (4-day moving avg): 1.027 (1.013–1.042) RD admissions (5-day moving avg): 1.037 (1.014–1.060) Asthma admissions (ages 5–18): 1.077 (1.004–1.155) May 2002–Dec 2003: 4-day moving avg CVD admissions PM ₁₀ (13 $\mu\text{g}/\text{m}^3$): 1.045 (1.016–1.074) Biomass (5.4 $\mu\text{g}/\text{m}^3$): 1.040 (1.009–1.072)	Results for two pollutant models are also reported

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Study	Location Time period	Outcome	Pollutants	Source- apportionment Method/Health Effects Model	Effect Measure	Comments
		Pulmonary embolism (ICD-10: I26) Cardiac arrest (ICD-10: I46) Cardiac arrhythmias (ICD-10: I48–I49) Heart failure (ICD-10: I50) Chronic bronchitis (ICD-10: J41–J42) Emphysema (ICD-10: J43) Other chronic obstructive pulmonary disease (ICD-10: J44) Asthma (ICD-10: J45) Status asthmaticus (ICD-10: J46)		holidays, influenza epidemics, grass pollen, school holidays, and meteorology Lag days from 0 to 5	PM ₁₀ secondary inorganic compounds (NH ₄ NO ₃ +(NH ₄) ₂ SO ₄ +(NH ₄)HSO ₄) (6.1 µg/m ³): 1.050 (1.021–1.081) Fuel oil combustion (Oil) (2.8 µg/m ³): 1.035 (1.006–1.065) Road concrete and tyre wear+igneous rock+limestone (Crustal) (1.8 µg/m ³): 1.054 (1.028–1.081) Salt +NANO ₃ (Sea Salt) (2.2 µg/m ³): 0.980 (0.947–1.017) Vehicle exhaust emissions and brake wear emission (Vehicle) (0.6 µg/m ³): 0.989 (0.949–1.032) RD admissions PM ₁₀ (13 µg/m ³): 1.072 (1.023–1.122) Biomass (5.4 µg/m ³): 1.084 (1.034–1.136) PM ₁₀ secondary (6.1 µg/m ³): 1.060 (1.012–1.110) Oil (2.8 µg/m ³): 1.042 (0.996–1.090)	

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Study	Location Time period	Outcome	Pollutants	Source- apportionment Method/Health Effects Model	Effect Measure	Comments
		Pediatric asthma (ICD-10: J45) Pediatric status asthmaticus ICD-10: J46)			Crustal (1.8 µg/m ³): 1.040 (0.995–1.088) Sea Salt (2.2 µg/m ³): 1.014 (0.962–1.069) Vehicle (0.6 µg/m ³): 0.949 (0.886–1.017) Asthma admissions (5–18 yrs) PM ₁₀ (13 µg/m ³): 1.004 (0.866–1.164) Biomass (5.4 µg/m ³): 0.979 (0.848–1.131) PM ₁₀ secondary (6.1 µg/m ³): 0.936 (0.815–1.075) Oil (2.8 µg/m ³): 1.004 (0.862–1.170) Crustal (1.8 µg/m ³): 0.942 (0.800–1.108) Sea Salt (2.2 µg/m ³): 0.930 (0.793–1.091) Vehicle (0.6 µg/m ³): 1.203 (0.983–1.473)	

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<p>Nikolov et al. 2007</p>	<p>Boston, MA</p>	<p>Myocardial ischemia in dogs (Canine ST-segment)</p>	<p>Concentrated air particles – Sulfate, black carbon, elemental carbon, organic carbon, Aluminum (Al), Arsenic (Ar), Barium (Ba), Bromine (Br), Calcium (Ca), Cadmium (Cd), Chlorine (Cl), Chromium (Cr), Copper (Cu), (Iron) Fe, Potassium (K), Manganese (Mn), (Nickel) Ni, (Sodium (Na), (Lead) Pb, (Sulfur) S, Selenium (Se), Silicon (Si), Titanium (Ti), Vanadium (V), Zinc (Zn)</p>	<p>Bayesian Structural Equation Model (SEM) compared to a tracer approach and a 2-stage approach</p>	<p>Informative Bayesian SEM</p> <p>Change in log peak ST-segment associated with an increase in the contribution of each pollution source (source tracer) on the scale of 1 SD increase in the concentration of Si</p> <p>Road dust (Si): 0.154 (95% CI = 0.030–0.276)</p> <p>Power plants (S): –0.071 (95% CI = –0.217 to 0.072)</p> <p>Oil combustion (Ni): –0.034 (95% CI = –0.167 to 0.115)</p> <p>Motor vehicles (BC): 0.062 (95% CI = –0.217 to 0.341)</p> <p>Source Tracer Approach</p> <p>For each factor, change in log peak ST-segment associated with 1 SD increase in concentration of source tracer</p> <p>Road dust (Si): 0.137 (95% CI = 0.037–0.237)</p> <p>Power plants (S): –0.063 (95% CI = –0.227 to 0.100)</p> <p>Oil combustion (Ni): –0.063 (95% CI = –0.227 to 0.110)</p> <p>Motor vehicles (BC): 0.062 (95% CI = –0.217 to 0.341)</p>	
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<p>Nikolov et al. 2008</p>	<p>Boston, MA</p>	<p>Canine cardiorespiratory function: respiratory frequency, time for inspiration, time for expiration, peak inspiratory flow, peak expiratory flow, tidal volume, minute ventilation, end inspiratory pause, end expiratory pause (<i>eep</i>), pause, enhanced pause</p>	<p>Concentrated air particles – Sulphate, black carbon, elemental carbon, organic carbon, AL, Ar, Ba, Br, Ca, Cd, Cl, Cr, Cu, Fe, K, Ma, Ni, Na, Pb, S, Se, Si, Ti, V, Zn-collected in 1997–1998 and 2001–2002</p>	<p>Structural equation approach – three different receptor models (standard additive Factor Analysis (FA); multiplicative FA, mixed multiplicative FA)</p> <p>Major sources identified as: Road dust (Si, Al); Power plants (S and sulphate); Oil combustion (Ni and V); Motor vehicles (BC, OC, EC)</p>	<p>Estimated regression coefficients from mixed multiplicative SEM:</p> <p>Log of respiratory frequency</p> <p>Road dust (Al): -0.1709 (-0.3317 to -0.0248)</p> <p>Power plants (S): -0.0470 (-0.4571 to 0.3582)</p> <p>Oil combustion (Ni): -0.2580 (-0.5044 to -0.0557)</p> <p>Motor vehicles (OC): 0.8599 (0.2420 to 1.5861)</p> <p>Log of peak expiratory flow</p> <p>Road dust (Al): -0.1232 (-0.2799 to 0.0004)</p> <p>Power plants (S): -0.1797 (-0.6169 to 0.2157)</p> <p>Oil combustion (Ni): -0.0990 (-0.3365 to 0.1135)</p> <p>Motor vehicles (OC): 0.8283 (0.1598 to 1.5810)</p> <p>Log of enhanced pause</p> <p>Road dust (Al): -0.3014 (-0.7088 to -0.0097)</p> <p>Power plants (S): -0.8176 (-1.7353 to 0.0368)</p> <p>Oil combustion (Ni): 0.0516 (-0.5181 to 0.5321)</p> <p>Motor vehicles (OC): 1.8720 (0.5166 to 3.6915)</p>	<p>Estimated regression coefficients and 95% CIs also reported for the multivariate analysis</p> <p>Estimated regression coefficients also reported for the univariate analyses from the following models: Additive SEM₁, Multiplicative SEM₂, Mixed multiplicative SEM₃, Tracer analysis and Multivariate mixed multiplicative SEM</p>
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<p>Gent et al. 2009</p>	<p>New Haven County, Connecticut 2000–2003</p>	<p>Symptoms (wheeze; persistent cough; shortness of breath; chest tightness and use of short-acting inhaler among 149 children (ages 4–12) with asthma from another study who lived within 30 km of the air monitoring station</p>	<p>PM_{2.5} samples from a single monitoring site (8/1/2000–2/3/2004) (Speciation for 51 elements using X-ray fluorescence)</p>	<p>Principal component analysis with an orthogonal (varimax) rotation</p> <p>Six sources of PM_{2.5} were identified: motor vehicle emissions; road dust; regional sulfur sources; biomass burning; oil combustion emissions; sea salt.</p> <p>More than half of mean PM_{2.5} was attributed to traffic-related sources motor vehicles (42%) and road dust (12%).</p> <p>Generalized estimating equation models performed for each health outcome, adjusted for season, day-of-the-week and date.</p> <p>Health effects of elements included in the factor analysis, all six sources, and PM_{2.5} were examined for same-day exposures [lag0 (L0)] as well as exposures lagged by 1 or 2 days (L1, L2) or the mean</p>	<p>ORs (95% CIs) for a 5 mg/m³ increase in same-day or lagged for same and previous 2 days</p> <p><u>Wheeze (lag 0 model)</u></p> <p>Motor vehicles: 1.05 (0.99–1.10) Road dust: 1.10 (1.01–1.19) Sulfur: 0.97 (0.94–1.00) Biomass burning: 0.80 (0.66–0.98) Oil: 1.02 (0.86–1.20) Sea salt: 0.96 (0.86–1.07)</p> <p><u>Persistent cough (lag 0 model)</u></p> <p>Motor vehicles: 1.02 (0.99–1.04) Road dust: 1.06 (1.01–1.11) Sulfur: 1.00 (0.98–1.01) Biomass burning: 0.97 (0.92–1.03) Oil: 1.02 (0.95–1.10) Sea salt: 0.99 (0.92–1.07)</p> <p><u>Shortness of breath (lag 0 model)</u></p> <p>Motor vehicles: 1.06 (1.01–1.11) Road dust: 1.12 (1.02–1.22) Sulfur: 0.98 (0.94–1.02) Biomass burning: 1.05 (0.95–1.17) Oil: 1.07 (0.92–1.26) Sea salt: 1.01 (0.92–1.12)</p> <p><u>Chest tightness (lag 0 model)</u></p> <p>Motor vehicles: 1.02 (0.97–1.08) Road dust: 1.04 (0.95–1.15) Sulfur: 0.99 (0.94–1.03) Biomass burning: 1.06 (0.95–1.18) Oil: 0.99 (0.82–1.18) Sea salt: 0.95 (0.84–1.08)</p>	<p>Results also reported for wheeze and inhaler use with a gaseous copollutant (NO₂, CO, SO₂, O₃) added to the models (no significant associations were detected in copollutant models between exposure and odds of persistent cough or chest tightness – results not shown)</p>
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				of lagged days 0–2 (L02).	<p><u>Inhaler use (lag 0 model)</u></p> <p>Motor vehicles: 1.02 (1.00–1.05) Road dust: 1.06 (1.02–1.11) Sulfur: 0.98 (0.97–1.00) Biomass burning: 1.00 (0.96–1.03) Oil: 0.98 (0.91–1.05) Sea salt: 0.99 (0.94–1.04)</p> <p><u>Wheeze (lag 02 model)</u></p> <p>Motor vehicles: 1.10 (1.01–1.19) Road dust: 1.26 (1.05–1.51) Sulfur: 0.98 (0.92–1.04) Biomass burning: 0.64 (0.46–0.88) Oil: 0.80 (0.56–1.08) Sea salt: 0.91 (0.82–1.16)</p> <p><u>Persistent cough (lag 02 model)</u></p> <p>Motor vehicles: 1.03 (0.98–1.09) Road dust: 1.16 (1.02–1.32) Sulfur: 1.01 (0.98–1.05) Biomass burning: 0.93 (0.81–1.06) Oil: 0.84 (0.71–1.00) Sea salt: 0.88 (0.77–1.01)</p> <p><u>Shortness of breath (lag 02 model)</u></p> <p>Motor vehicles: 1.12 (1.01–1.24) Road dust: 1.28 (1.05–1.55) Sulfur: 0.97 (0.90–1.04) Biomass burning: 0.78 (0.52–1.18) Oil: 0.94 (0.69–1.29) Sea salt: 1.01 (0.79–1.29)</p> <p><u>Chest tightness (lag 02 model)</u></p> <p>Motor vehicles: 1.08 (0.98–1.20) Road dust: 1.20 (0.97–1.49)</p>	
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					<p>Sulfur: 1.00 (0.92–1.08) Biomass burning: 0.87 (0.62–1.22) Oil: 0.80 (0.58–1.10) Sea salt: 0.95 (0.71–1.27)</p> <p><u>Inhaler use (lag 0 model)</u></p> <p>Motor vehicles: 1.03 (0.98–1.08) Road dust: 1.09 (1.00–1.19) Sulfur: 1.00 (0.97–1.03) Biomass burning: 0.95 (0.87–1.04) Oil: 0.92 (0.81–1.05) Sea salt: 0.97 (0.88–1.07)</p> <p>Increased likelihood of symptoms and inhaler use was largest for 3-day averaged exposures to traffic-related sources or their elemental constituents and ranged from a 10% increased likelihood of wheeze for each 5-$\mu\text{g}/\text{m}^3$ increase in particles from motor vehicles to a 28% increased likelihood of shortness of breath for increases in road dust.</p> <p>Neither the other sources identified nor $\text{PM}_{2.5}$ alone was associated with increased health outcome risks.</p>	
Bell et al. 2010	3 Connecticut counties and 1 Massachusetts county August 2000–February 2004	Birth weight and small-at-term birth (term birth < 2500 g)	For each location, data from a single monitoring station were used. 51 chemical elements were determined by x-ray fluorescence.	Positive matrix factorization method Five sources were identified: Motor vehicles; road dust; oil combustion; salt; other regional sources (sulfur) Linear regression	Road dust and related constituents such as silicon and aluminum were associated with lower birth weight, as were the motor-vehicle-related species such as elemental carbon and zinc and the oil-combustion-associated elements vanadium and nickel. An interquartile range increase in exposure was associated with low	Models adjusted for apparent temperature by trimester; infant’s sex; parity, nature of delivery, trimester prenatal care began, length of gestation in weeks; indicator variables for year of

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				<p>was performed to relate birth weight to PM_{2.5} total mass, sources, and constituents in separate models.</p> <p>Each model included one of the following as the exposure variable: a single constituent, single source, or PM_{2.5} total mass.</p> <p>Logistic regressions was used to evaluate the association between pollutants and risk of small-at-term (term birth < 2500 g) compared with non-small-at-term (term birth ≥ 2500 g) births.</p>	<p>birth weight for zinc (12% increase in risk), elemental carbon (13%), silicon (10%), aluminum (11%), vanadium (8%), and nickel (11%).</p> <p>Analysis by trimester showed effects of third-trimester exposure to elemental carbon, nickel, vanadium, and oil-combustion PM_{2.5}.</p>	<p>birth; and mother's age, marital status, education, tobacco use during pregnancy, alcohol use during pregnancy (and race).</p>
Lall et al. 2011	<p>Manhattan (New York City), NY</p> <p>2001–2002</p>	<p>Respiratory and cardiovascular hospital admissions for individuals aged 65 and older from Medicare files</p> <p>Pneumonia (ICD-9: 480–486)</p> <p>Chronic obstructive</p>	<p>Single monitoring location in Manhattan (PM_{2.5} samples)</p> <p>Speciated PM concentrations obtained fusing x-ray fluorescence</p>	<p>Positive matrix factorization source apportionment</p> <p>Seven source categories were identified: long range sulfates; traffic; residual oil; steel metal works dust, soil, World Trade Center plume, chlorine</p>	<p><u>Respiratory Hospital Admissions:</u></p> <p>PM_{2.5} from steel (increment = 2.1 µg/m³) was associated with same-day lag (RR = 1.043; 95% CI, 1.007–1.080) and 3-day lag (RR = 1.048; 95% CI, 1.011–1.086)</p> <p>Positive and significant associations with pneumonia and asthma (3-day lag) (shown in a figure)</p>	<p>An unconstrained distributed-lag model of total respiratory admissions considered over a consecutive 4-day period provided cumulative risk estimates for steel PM that were higher than risk estimates for any single day:</p>

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		<p>pulmonary disease (COPD) (ICD-9: 490–492 and 496)</p> <p>Acute bronchitis and bronchiolitis (ICD-9: 466)</p> <p>Asthma (ICD-9: 493)</p> <p>Dysrhythmia (ICD-9: 427)</p> <p>Ischemic heart disease (ICD-9: 410–414)</p> <p>Heart failure (ICD-9: 428)</p> <p>Stroke (ICD-9: 431–437)</p>		<p>Daily and distributed-lag generalized linear models of Medicare respiratory and cardiovascular hospital admissions during 2001–2002 considered PM_{2.5} from five sources: transported sulfate, residual oil, traffic, steel metal works, and soil.</p>	<p><u>Cardiovascular Hospital Admissions:</u></p> <p>Same-day traffic-related PM_{2.5} exposures were positively associated with total cardiovascular admissions (RR = 1.041; 95% CI, 1.005–1.077; for a 2.8 µg/m³ increment in PM_{2.5} from traffic).</p> <p>Associations between traffic sources and specific categories of cardiovascular disease (stroke and heart failure were also observed) (results shown in a figure)</p>	<p>RR = 1.069 (95% CI, 1.013–1.127) versus RR = 1.043 (95% CI, 1.007–1.080). Similarly, the cumulative traffic-related cardiovascular effects over 0–3 days (RR = 1.078; 95% CI, 1.008–1.152) were much higher than those observed in the single maximum same-day lag model (RR = 1.041; 95% CI, 1.005–1.077)</p>
Ostro et al. 2011	<p>Barcelona, Spain</p> <p>2003–2007</p>	<p>All-cause mortality (minus accidents and homicides)</p> <p>Cardiovascular mortality (ICD 10 codes I00-I99)</p>	<p>A single monitoring station</p> <p>PM_{2.5} and PM₁₀ were collected using MCV high-volume sampler, African dust separated from urban and road dust. Particles were collected on quartz-fiber filters and analyzed by ICP-MS spectrometry. In addition to the periodic sampling of PM mass and species, PM₁₀ and PM_{2.5} mass were also measured every day using optical counters.</p>	<p>Positive matrix factorization</p> <p>Several sources of PM_{2.5}, secondary nitrate/organics, minerals (Ca, Al, and Fe), secondary sulfate/organics, Road dust (TC, Fe, Cu, and Sb)</p> <p>Time-stratified case-crossover model with a forward stepwise analysis</p>	<p>All-cause mortality excess risks (95% CIs) associated with IQR increases in sources of PM_{2.5} (lag 2):</p> <p><u>Single source models</u></p> <p>Sulphate: 1.3 (–2.5 to 5.3) Road dust: 4.2 (1.5–7.0) Minerals: 4.1 (1.5–6.7) Fuel oil: 2.1 (–0.1 to 4.2) Industry: 1.3 (–1.2 to 3.8) Nitrate: 2.0 (–0.1 to 4.1) Vehicle exhaust: 3.3 (0.4–6.3) Sea salt: –0.8 (–3.6 to 2.2) Traffic: 5.6 (1.8–9.5)</p>	<p>Source profiles (species concentrations within the source) and explained variation (EV) of each species are also reported along with the results for all the models.</p>

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				Lag days from 0 to 2.	<u>Multisource models</u> Minerals: 3.7(1.38–6.2) Fuel oil: 2.0 (–0.4 to 4.5) Nitrate: 2.0 (–0.3 to 4.3) <u>Multisource models with traffic</u> Mineral: 3.4 (1.0–5.8) Traffic: 5.0 (1.3–8.8)	
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