



*APPENDIX D:
PITTSBURGH STUDY
SUMMARIES*

The Health Impacts of Pittsburgh
Air Quality: A Review of the
Scientific Literature, 1970–2012

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PITTSBURGH STUDY SUMMARIES

A Retrospective Investigation of PM₁₀ in Ambient Air and Cardiopulmonary Hospital Admissions in Allegheny County, Pennsylvania: 1995–2000

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Publication: Journal of Occupational and Environmental Medicine, Vol. 48, No. 1, 2006

Data: Cardiopulmonary hospital admissions for people age ≥ 65 ; mean daily PM₁₀ averaged from eight monitors; daily mean temperature, dew point, barometric pressure, relative humidity; Allegheny County, Pennsylvania; 1995–2000

Analytic Method: A generalized additive model and constrained (distributed) and unconstrained lag models were used to analyze the data.

Findings and Conclusions: Though PM₁₀ levels decreased by 17 percent over the study period, overall cardiopulmonary hospital admissions for the target population remained relatively constant during the same period. Cardiopulmonary hospital admissions in age 65–74 decreased, while admissions for those ≥ 75 increased during the study period. Cardiopulmonary hospital admissions were statistically significant for same day PM₁₀ exposures, with a 1 $\mu\text{g}/\text{m}^3$ increase in PM₁₀ associated with a 0.0609 percent increase in cardiopulmonary hospital admissions. For the 20 $\mu\text{g}/\text{m}^3$ interquartile increase in PM₁₀ levels experienced during the study period, hospital admissions were estimated to increase by 1.2256 percent.

Ozone and Short-term Mortality in 95 U.S. Urban Communities: 1987–2000

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Publication: Journal of the American Medical Society, Vol. 292, No. 4, 2004

Data: Daily total non-accidental deaths and cardiopulmonary deaths in 95 cities for 1987 to 2000; 24-hr average, maximum 8-hour average, and 1-hour maximum ozone values, 24-hour average PM₁₀ values; 24-hour average dew point and temperature

Analytic Method: A 2-stage statistical model was used for analysis. The first stage analysis used a distributed-lag Poisson regression model for city-specific relationships between mortality and ozone exposure, using various ozone exposure metrics in the last week with adjustments for seasonality and long-term trends and accounting for temperature and dew point. PM₁₀ levels were included and high temperature days excluded in a sensitivity analysis to assess potential confounding. The second stage involved both combining the community-specific relative rates from the first stage analysis to develop a national estimate, as well as community-specific estimates using a Bayesian model that integrated the community-specific risk estimates from the first stage with the national average estimate to account for within-community and across-community variability (i.e., heterogeneity). Effect of ozone on mortality was estimated for three age groups: <65, 65–74, ≥75.

Findings and Conclusions: National average estimate for the effect of a 10 ppb increase in ozone exposure during the week preceding the event was associated with a statistically significant 0.52 percent increase in the risk of premature death from non-accidental causes. Ozone exposure on the same day and the day preceding the event produced the largest risk estimates. Risk estimate was largest for the 65-74 age group, though statistically significant risks were estimated for all three age groups. The authors note that these risk estimates likely underestimate the total mortality burden from ozone exposure as they account for only short-term effects.

A 10 ppb increase in ozone was associated with a 0.15 percent statistically significant increase in the risk of mortality even when limiting ozone exposure to days with average ozone levels less than 60 ppb, indicating that mortality risks for older adults remain even for exposure to daily ozone levels well below the current ozone NAAQS of 75 ppb. Estimated risks remained robust when adjusted for daily PM₁₀ levels and with exclusion of days with temperatures >84°F.

The Pittsburgh risk estimate of ~0.9 percent per 10 ppb increase in ozone was 23rd highest of the 95 cities included in the study, though the risk estimate was not statistically significant.

Do Respiratory Epidemics Confound the Association Between Daily Deaths and Air Pollution?

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Publication: European Respiratory Journal, Vol. 16, 2000

Data: Non-accidental daily deaths, pneumonia hospital admissions, daily PM₁₀ levels and daily temperature, dew point and barometric pressure; five cities; 1986–1993

Analytic Method: A generalized additive Poisson regression model was used to assess the influence of daily standardized mean PM₁₀ levels on daily deaths, adjusting for time trend, temperature, dew point, barometric pressure and day of the week. As the purpose of the analysis was to investigate the potential influence of respiratory disease epidemics on the relationship between air pollution and mortality, 3-day moving average pneumonia hospital admissions above the 90th percentile were selected to identify “epidemic” days.

Findings and Conclusions: In the combined city analysis, a 10 µg/m³ increase in PM₁₀ was associated with a 0.85 percent increase in daily deaths, which was only marginally reduced (0.78 percent) after controlling for respiratory epidemics. Pittsburgh-specific results were almost identical to the combined city result, with a 10 µg/m³ increase in PM₁₀ associated with a 0.84 percent increase in daily deaths. Both the Pittsburgh and combined city results were statistically significant.

The study authors conclude that the relationship between PM₁₀ levels and daily deaths is not confounded by a failure to control for influenza or pneumonia epidemics.

A Study of the Association between Daily Mortality and Ambient Air Pollution Concentrations in Pittsburgh, Pennsylvania

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Publication: Journal of the Air and Waste Management Association, Vol. 50, 2000

Data: Non-accidental daily deaths (0–74, 75+), daily PM₁₀, PM_{2.5}, SO₂, NO₂, CO, Ozone, temperature, dew point; 1989–1991

Analytic Method: Poisson regression was applied to single, double and multi-pollutant models that included a day-of-the-week variable to filter the day-of-the-week effect, with several different data filters (degrees of freedom) selected using cubic spline functions. Daily non-accidental mortality data were stratified into ages 0–74 and 75+. Additional analyses were conducted for a variety of time lags (including positive and negative lags) and for restriction of exposure data by season.

Findings and Conclusions: PM₁₀, NO₂, and CO data were highly correlated, while SO₂ and ozone were less correlated with these three pollutants. For same-day (O-lag) 1-pollutant models, of the five pollutants assessed (PM₁₀, SO₂, NO₂, CO, ozone), only PM₁₀ was positive and statistically significant with daily mortality in the 0–74 year age group. In 2-pollutant models, the PM₁₀ coefficient was unchanged with inclusion of either ozone or SO₂, while inclusion of CO resulted in a smaller and non-significant PM₁₀ coefficient and inclusion of NO₂ resulted in an increase in the size and significance value for the PM₁₀ coefficient. Results for the 3-pollutant model using the three highly correlated pollutants (PM₁₀, NO₂, CO) resulted in an increase in the PM₁₀ coefficient, though this result was deemed a ‘marginally significant’ value. Inclusion of the remaining two pollutants (SO₂, ozone) in a 5-pollutant model did not change the results of the 3-pollutant model. The authors conclude that “despite the evidence of multicollinearity, the PM₁₀ coefficient in the different models remain stable and significant.”

Results from the various lag and seasonal models found substantial heterogeneity and instability in the pollutant-mortality associations. Results for the 75+ age group found a statistically significant PM₁₀ coefficient only for one of the four weather variable models applied to the two-pollutant model, though all other pollutant model results were positive but not statistically significant for PM₁₀. Results for PM_{2.5} and coarse particles (PM₁₀–PM_{2.5}) were positive but not statistically significant for both age categories in all reported pollutant models.

The Toll From Coal

Authors & Affiliations: Conrad Schneider¹ and Jonathan Banks¹

¹Clean Air Task Force

Publication: Clean Air Task Force, September 2010

Data: U.S. electric generating unit (EGU) emissions, non-EGU emissions for all U.S. counties

Analytic Method: The study estimates PM_{2.5} concentrations and related morbidity and mortality in U.S. counties using the Abt Associates Powerplant Impact Estimator (PIE) software. A source-receptor model provides estimates of county-level PM_{2.5} concentrations based on emissions inventory that included SO₂, NO_x, direct PM_{2.5} emissions, ammonia and volatile organic compounds. PIE integrates emissions estimates with concentration-response functions from selected peer-reviewed studies to provide health outcome estimates. Health costs associated with the estimated health outcomes were derived from various health valuation studies.

Findings and Conclusions: County-level estimates of power plant PM_{2.5} emissions and health outcomes were aggregated by metropolitan area and state for 2010, 2015 and 2020. The Pittsburgh metropolitan area ranked fourth among U.S. metropolitan areas in magnitude of power plant-related health impacts, with an estimated 340 premature deaths, 242 cardiopulmonary hospital admissions and 555 heart attack cases for 2010. Based on per capita mortality rates for 2010, Pittsburgh ranked tenth among U.S. metropolitan area with a mortality risk rate of 17.9 per 100,000 adults.

Air Toxics in Allegheny County: Sources, Airborne Concentrations, and Human Exposure

Authors & Affiliations: Carnegie Mellon University

Publication: Report submitted to the Allegheny County Health Department, March 2009

Data: Monitored data for 65 air toxics 2001–2002; monitored data for 36 volatile organic air toxics 2006–2007; archived compliance monitoring data

Analytic Method: Lifetime cancer risk for each of the air toxic compounds was assessed based on a linear, no-threshold model for 65 air toxics. The hazard quotient was calculated for each compound to assess non-cancer risks. The EPA interaction model from the EPA 2000 Supplementary Guidance for Conducting Health Risk Assessments of Chemical Mixtures was applied to assess the combined cancer and non-cancer risks from cumulative exposure to both the subset of 36 volatile organic compounds (VOC), as well as all 65 air toxic compounds including diesel particulate matter (DPM).

Findings and Conclusions: Cumulative cancer risks were highest for the Downtown Pittsburgh monitoring site, reaching 1×10^{-4} for the subset of 36 VOC air toxics and 1×10^{-3} when combined with DPM levels. Of the compounds evaluated, diesel particulate matter, benzene, formaldehyde and trichloroethene posed the highest individual cancer risks of greater than 10^{-5} . Of the four classes of air toxics compounds evaluated (VOC, metals, polycyclic organic matter and diesel particulate matter), diesel particulate matter presented the highest risk at all sites.

With respect to non-cancer risks, only acrolein presented a potential chronic risk with a hazard quotient greater than one.

Estimating Particulate Matter-Mortality Dose-Response Curves and Threshold Levels: An Analysis of Daily Time-Series for the 20 Largest U.S. Cities

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Publication: American Journal of Epidemiology, Vol. 152, No. 5, 2000

Data: Daily cause-specific, non-external deaths classified by age group (<65, 65-74, ≥75); 24-hour mean PM₁₀, temperature, dew point data; 20 cities; 1987–1994

Analytic Method: Generalized additive model using a log-linear function was used to estimate each city's air pollution/mortality relative rate, accounting for age-specific long-term trends, weather and day of the week.

Findings and Conclusions: For the overall 20-city analysis, a 10 µg/m³ increase in day 0 + day 1 mean PM₁₀ levels was associated with a statistically significant 0.69 percent increase in cardiorespiratory mortality; a statistically significant 0.54 percent increase in total external mortality; and a statistically significant 0.34 percent increase in other cause mortality.

Linear, spline and threshold dose-response models were applied to the data to test for linearity, with results based on Akaike information criterion indicating that the linear model was preferred for both city-specific and overall assessments. No PM₁₀ concentration threshold was found for total and cardiopulmonary mortality, though evidence of a threshold was found for other cause mortality. Bayesian posterior probability analysis indicated the greatest likelihood for a threshold for total and cardiopulmonary mortality was at 15 µg/m³ daily PM₁₀, while the most likely threshold for other cause mortality was at 65 µg/m³ daily PM₁₀.

Results for Pittsburgh indicate a positive and statistically significant increase in total and cardiopulmonary mortality per 10 µg/m³ increase in day 0 + day 1 mean PM₁₀ levels, while the association for other cause mortality was positive but not statistically significant.

The National Morbidity, Mortality, and Air Pollution Study Part III: PM₁₀ Concentration — Response Curves and Thresholds for the 20 Largest U.S. Cities

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Publication: Health Effects Institute Special Report Number 94, May 2004

Data: Daily cause-specific, non-external deaths classified by age group (<65, 65–74, ≥75); 24-hour mean PM₁₀, temperature, dew point data; 20 cities; 1987–1994.

Analytic Method: Generalized additive model using a log-linear function was used to estimate each city's air pollution/mortality relative rate, accounting for age-specific long-term trends, weather and day of the week.

Findings and Conclusions: Linear, spline and threshold dose-response models were applied to the data to test for linearity, with results based on Akaike information criterion indicating that the linear model was preferred for both city-specific and overall assessments. Bayesian hierarchical posterior probability model found PM₁₀ concentration thresholds for total and cardiopulmonary mortality ranging from 0–20 µg/m³, depending on the lag model selected. Evidence of a threshold was found for other cause mortality at levels between 45–75 µg/m³, depending on the exposure lag selected. For the multi-city analysis using the linear model, a 10 µg/m³ increase in day 0 + day 1 mean PM₁₀ levels was associated with a statistically significant 34 percent increased risk of cardiopulmonary-related death; a statistically significant 28 percent increase in total mortality; and a statistically significant 20 percent increase in other cause mortality.

Using the city-specific, non-Bayesian linear model, results for Pittsburgh for association between a 10 µg/m³ increase in day 0 + day 1 mean PM₁₀ levels was positive though statistically significant for cardiorespiratory mortality only.

The National Morbidity, Mortality and Air Pollution Study, Part IV: Hierarchical Bivariate Time-Series Models — A Combined Analysis of PM₁₀ Effects on Hospitalization and Mortality

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Publication: Health Effects Institute Research Report Number 94, September 2005

Data: Daily PM₁₀, temperature, humidity, barometric pressure, cardiovascular hospital admissions and cardiovascular-related mortality for people age ≥65; 10 cities; 1986–1993

Analytic Method: Poisson regression models were applied to estimate the PM₁₀ associated log-relative rates of cardiovascular-related mortality and hospital admissions for each city's bivariate time-series data taking into account the relationship between the two data sets using generalized estimating equations. In the second stage of analysis, the relative rates of all-cause mortality and cardiovascular hospital admissions were combined across the 10 cities to characterize their overall relationship.

Findings and Conclusions: A 10 µg/m³ increase in same-day plus previous-day PM₁₀ levels was associated with a 0.26 percent increase in cardiovascular-related mortality for the combined 10-city analysis, though this relationship was not statistically significant. An increase of 0.71 percent was associated with a 10 µg/m³ increase in same-day plus previous-day PM₁₀ levels, and this association was statistically significant. The log-relative rates of mortality and hospitalization were similar in heterogeneity across cities, with the results providing a basis for predicting city-specific estimates of cardiovascular hospitalization rates related to PM₁₀ exposure if only the relative rate of mortality is available for a city.

Consistent with the overall combined city results, the Pittsburgh-specific relative rate for cardiovascular mortality associated with a 10 $\mu\text{g}/\text{m}^3$ increase in same-day plus previous-day PM_{10} levels was positive (0.36 percent) but not statistically significant, and the relative rate for cardiovascular hospital admissions of 0.91 percent was statistically significant.

Revised Analyses of the National Morbidity, Mortality and Air Pollution Study: Mortality Among Residents of 90 Cities

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Publication: Journal of Environmental Health and Toxicology, Part A, Vol. 68, 2005

Data: PM_{10} , Ozone, CO, NO_2 , SO_2 , temperature, dew point, daily mortality by cause; 90 U.S. cities; 1987–1994

Analytic Method: Updated analysis of the NMMAPS study published by Samet et al. 2003b, comparing revised convergence criteria for the generalized additive model (GAM) published in S-Plus software with original model convergence criteria as well as generalized linear model (GLM) with natural cubic splines.

Findings and Conclusions: Results of the combined 90-city analysis using the revised GAM convergence criteria for total mortality were reduced in comparison to the original NMMAPS analysis (0.27 percent v. 41 percent per 10 $\mu\text{g}/\text{m}^3$ PM_{10}), but remain larger than the GLM analysis (0.21 percent per 10 $\mu\text{g}/\text{m}^3$ PM_{10}). These results were insensitive to the inclusion of ozone alone or ozone plus either nitrogen oxides and sulfur dioxide in two and three pollutant models. Heterogeneity of across-city results was examined using three different models (two-stage hierarchical, three-stage regional, spatial correlation) applied to seven regions of the U.S., with results of the national average three models insensitive to model selection.

Results for the Pittsburgh metropolitan area were positive though not statistically significant for the association of PM_{10} with total and cardiopulmonary mortality.

National Maps of the Effect of Particulate Matter on Mortality: Exploring Geographic Variation

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Publication: Environmental Health Perspectives, Vol. 111, No. 1, 2003

Data: 24-hour average PM_{10} , temperature, dew point, and daily cause-specific mortality; 88 U.S. cities; 1987–1994

Analytic Method: Data were analyzed using a two-stage Bayesian hierarchical model, in which a maximum likelihood estimate of the effect of a $10 \mu\text{g}/\text{m}^3$ increase in 24-hour average PM_{10} was obtained in the first stage for each city using a generalized linear model with natural cubic splines. In the second stage, a Bayesian estimate of the mortality relative rate was obtained, which takes into account between-city spatial correlations.

Findings and Conclusions: City-specific results for both the maximum likelihood estimates and Bayesian estimates were plotted for seven U.S. geographical regions. Results of the 88-city analysis indicate that PM_{10} -related mortality rates were greatest for cardiopulmonary mortality (0.31 percent/ $10 \mu\text{g}/\text{m}^3 \text{PM}_{10}$) compared with total mortality (0.22 percent/ $10 \mu\text{g}/\text{m}^3 \text{PM}_{10}$) and other cause mortality (0.13 percent/ $10 \mu\text{g}/\text{m}^3 \text{PM}_{10}$), with only cardiopulmonary and total mortality results statistically significant. Modest variation in the city-specific mortality rates was found across results for the 88 cities.

Pittsburgh region Bayesian estimates for the relative risks of mortality associated with a $10 \mu\text{g}/\text{m}^3$ increase in 24-hour average PM_{10} was statistically significant, though the maximum likelihood estimate was positive but not statistically significant.

Airborne Particulate Matter and Mortality: Timescale Effects in Four U.S. Cities

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Publication: American Journal of Epidemiology, Vol. 157, No. 12, 2003

Data: Daily mortality by cause of death for three age groups (<65, 65–75, and >75 years), 24-hour average PM_{10} , and temperature and dew point (from hourly values); Four cities; 1987–1994

Analytic Method: A Fourier series analysis was conducted to decompose the timescale for association of PM_{10} with daily mortality. A Poisson regression model was used to estimate city-specific and pooled estimates of log relative rates of mortality according to six timescales of PM_{10} exposure: ≥ 60 days, 30–59 days, 14–29 days, 7–13 days, 3.5–6 days, and <3.5 days.

Findings and Conclusions: Results from pooled data across all four cities found overall exposure was associated with positive but not statistically significant associations between PM_{10} with daily mortality, with the largest effect associated with cardiopulmonary mortality (0.22 percent) followed by total mortality (0.17 percent) and other cause mortality (0.13 percent). However, results for the longest timescale (≥ 60 days) were substantially greater for each category of death (cardiopulmonary = 1.87 percent, total = 1.35 percent, other = 0.72 percent), and results at this timescale of exposure were statistically significant for cardiopulmonary and total mortality. The authors interpret the results of this analysis to indicate that the association of particulate matter pollution with premature death is greatest at longer timescales, and thus does not reflect very short-term displacement of death due to “harvesting” of very frail individuals. They suggest that these findings may reflect a greater biologic impact from chronic rather than acute exposures.

Results for Pittsburgh were positive for all three mortality classes and largest at the ≥ 60 day time scale (cardiopulmonary = 1.19 percent, total = 1.34 percent, other = 1.45 percent), though only the result for total mortality was statistically significant. Unlike the results for the other three cities and the pooled data, as well as results from numerous other analyses, the Pittsburgh relative risk was largest for total mortality rather than cardiopulmonary mortality.

Association between PM_{2.5} and All-cause and Specific-cause Mortality in 27 U.S. Communities

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Publication: Journal of Exposure Science and Environmental Epidemiology, Vol. 17, 2007

Data: Daily PM_{2.5}, all (non-accidental) cause, cardiovascular, respiratory and stroke mortality, dry bulb and dew point temperatures; prevalence of central air conditioning; 27 U.S. cities; 1999–2002

Analytic Method: Conditional logistic regression was applied to a time-stratified case-crossover design methodology for the association of PM_{2.5} exposure with daily death in each city. Potential confounding by temperature and day of the week were addressed through use of a quadratic spline for apparent temperature (integration of dry bulb and dew point temperatures) and a variable for day of the week. Effect modification of age and gender were investigated through use of interaction terms in the regression model. In a second stage of analysis, a meta-analysis was conducted involving data from all 27 cities, and to investigate between-city heterogeneity, a meta-regression was applied to the full data set related to east versus west geographic location, whether the annual PM_{2.5} concentration exceeded the 15 $\mu\text{g}/\text{m}^3$ PM_{2.5} NAAQS and the prevalence of central air-conditioning.

Findings and Conclusions: For the combined city analysis, a 10 $\mu\text{g}/\text{m}^3$ increase in previous-day PM_{2.5} was associated with a 1.21 percent increase in all-cause mortality, a 1.78 percent increase in respiratory mortality, a 1.03 percent increase in stroke mortality and a 0.94 percent increase in cardiovascular mortality. Results for all mortality increases were statistically significant except for the estimate for cardiovascular mortality. Effect of PM_{2.5} exposure was greater in subjects ≥ 75 years of age (statistically significant for all-cause and stroke mortality), and there was suggestive (non-statistically significant) evidence of a greater effect in women than in men for all-cause and cardiovascular mortality and in the eastern versus western regions of the U.S. Increased prevalence of central air-conditioning was associated with a decreased effect of PM_{2.5}. Of particular interest was a finding (non-statistically significant) that mortality effects were greater in cities with annual PM_{2.5} levels $\leq 15 \mu\text{g}/\text{m}^3$ compared to cities with annual PM_{2.5} levels $\geq 15 \mu\text{g}/\text{m}^3$, suggesting that excess mortality associated with PM_{2.5} exposure is found at annual PM_{2.5} levels below the current PM_{2.5} annual NAAQS.

Results for Pittsburgh were provided only for all-cause mortality and were positive but not statistically significant.

The Impact of Secondary Particles on the Association between Ambient Ozone and Mortality

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Publication: Environmental Health Perspectives, Vol. 116, No. 4, 2008

Data: Daily 1-hour average ozone, 24-hour average PM_{2.5}, sulfate, nitrate and organic carbon, daily mortality, daily temperature and dew point; 18 U.S. cities; 2000–2005

Analytic Method: Time-series modeling using Poisson regression was applied to each city to assess the relationship of ozone with daily deaths, both for ozone alone as well as adjusting for PM_{2.5}, sulfate, nitrate and organic carbon as a linear term in two-pollutant models. Analyses were restricted to the May–September period. Temperature and dew point were controlled as potential confounders using 3-day mean data as a linear term in the regression model. The potential non-linear effect of including PM_{2.5} was also examined using a cubic regression spline model. In a second-stage analysis, the results of the individual city mortality effects analyses were pooled using a random effects meta-analysis model to obtain a multi-city average. Ozone exposures of lag 0, lag 1 and 2-day averaged lag 0 & 1 were examined in the analysis.

Findings and Conclusions: For the combined 18-city analysis, a 10 ppb increase in same day summertime 24-hour average ozone levels was associated with a 0.89 percent increase in non-accidental mortality. Adjustment for PM_{2.5} levels resulted in a slight reduction in the effect estimate. Due to data limitations, the percentage of days for which secondary pollutant data was available significantly reduced analysis of secondary pollutant impacts to 17–18 percent of total days. Results of two-pollutant models showed a substantial reduction and no statistical significance in the ozone mortality effect (0.54–0.62 percent) in two-pollutant models with secondary pollutants but not with PM_{2.5} (0.79–0.82 percent, depending on model selection). The authors conclude the relationship between summertime ozone exposure and mortality is confounded by secondary PM sulfate exposure, suggesting that secondary particle pollutants could be responsible for part of the observed ozone mortality effect.

Results for Pittsburgh were positive but not statistically significant for the ozone only model, but consistent with the multi-city analysis, they became negative after adjustment for sulfate pollution exposure.

The Relationship of Ambient Ozone and PM_{2.5} Levels and Asthma Emergency Department Visits: Possible Influence of Gender and Ethnicity

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Publication: Archives of Environmental and Occupational Health, Vol. 67, No. 2, 2012

Data: Daily 1-hour maximum ozone and mean PM_{2.5}, mean temperature and humidity, emergency department (ED) visits for asthma; metropolitan Pittsburgh, Pennsylvania (Allegheny County); 2002–2005

Analytic Method: A case-control analysis was conducted with controls defined as all days within a 28-day interval using the same day-of-the-week as the case period. A 28-day “washout” period was used to avoid counting repeat visits that were a continuation of the same event. The effect of ozone, PM_{2.5} and the combination of both pollutants together was evaluated using a conditional logistic regression model. The effect of temperature as well as lagged exposures up to five days and a 6-day average lag were also examined in the analysis.

Findings and Conclusions: A 10 parts per billion increase in one-hour daily maximum ozone was associated with 2.5 percent increase in asthma ED visits two days later. A previous day 10 µg/m³ increase in PM_{2.5} was associated with a 3.6 percent increase in asthma ED visits. In a two-pollutant model in which increases in both ozone and PM_{2.5} two days prior to the event were analyzed, asthma ED visits increased by 2.1 percent. The authors note that the unit of ambient air PM_{2.5} increase is proportionally substantially larger than for the ozone unit of exposure, and thus find a stronger physiological effect of ozone on asthma exacerbations than for PM_{2.5}.

Results of analysis of ED visits by race found larger effects of PM_{2.5} on ED visits for African American populations in comparison to effects in Caucasian Americans. No significant increase in risks was found for either race in the two-pollutant model combining increases in ozone and PM_{2.5} exposures.

Particulate Air Pollution Exposure and C-reactive Protein During Early Pregnancy

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Publication: Epidemiology, Vol. 22, No. 4, 2011

Data: Questionnaire-obtained maternal demographic characteristics, socioeconomic status, active and passive smoking, consumption of alcohol, other lifestyle factors, and medical history from 1696 pregnant women in Allegheny County, PA; maternal residence ZIP code; C-reactive protein concentrations from

blood samples; CO, NO₂, SO₂, ozone, PM₁₀, 1996–2001; PM_{2.5} 1999–2001; 1-hour temperature and daily relative humidity

Analytic Method: Space-time ordinary kriging interpolation was used to estimate daily air pollution concentrations at the ZIP code level for each subject. A logistic regression model was used to assess the association between air pollution and C-reactive protein levels in early (<22 weeks) pregnancy. C-reactive protein levels were assessed based on a dichotomous classification using a level of 8 ng/mL as the threshold associated with increased risk of preterm birth. C-reactive protein levels were also evaluated as a continuous measure using linear regression models after log transformation. Potential effect modification related to obesity using body mass index ≥ 30 kg/m² was also assessed.

Findings and Conclusions: Adjusted odds ratio for association of PM₁₀ with high (≥ 8 ng/mL) C-reactive protein was positive but not statistically significant for the entire population and was positive (1.47) and statistically significant for non-smoking women exposed lag 0-21. Adjusted odds ratio for the association of PM_{2.5} exposure with high C-reactive protein was positive and statistically significant for the entire population for lag 0-7 (1.21) and lag 0-21 (1.32) and positive and statistically significant for lag 0-21 (1.55) and lag 0-28 (1.47) in non-smoking women. Results for the adjusted association of ozone with high C-reactive protein was positive but not statistically significant for both the entire population and non-smoking women. No association was found for the relationship of the other measured air pollutants and high C-reactive protein. Obesity did not modify the relationship between air pollution exposure and high C-reactive protein.

First Trimester Exposure to Ambient Air Pollution, Pregnancy Complications and Adverse Birth Outcomes in Allegheny County, PA

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Data: 34,705 births at one Pittsburgh, Pa. hospital, maternal age, race/ethnicity, education level, mother's marital status, year and date of birth, infant gender, gestational age at delivery, cigarette smoking during pregnancy, parity, insurance type, preeclampsia, gestational hypertension, small for gestational age (SGA, defined as birth weight below 10th percentile for gestational age according to growth curves based on California normograms) and maternal residential ZIP code at delivery. Gestational hypertension (defined as systolic blood pressure (SBP) ≥ 140 mmHg or diastolic blood pressure (DBP) ≥ 90 mmHg) during the second half of pregnancy, preeclampsia (defined as gestational hypertension accompanied by proteinuria after 20 weeks of gestation). Gestational age at delivery estimated based on the last menstrual period (LMP) and other measurements, including uterine size, detection of fetal heartbeat, and first or second trimester ultrasonography if available, 1997–2002; Allegheny County and neighboring county PM₁₀, PM_{2.5}, ozone, 1996–2002.

Analytic Method: Exposure to air pollutants was assessed at the ZIP code level using space-time ordinary kriging interpolation based on daily average concentrations for the three air pollutants considered. Daily concentrations of air pollutants were averaged over the first trimester of pregnancy according to estimated gestational age.

A multiple logistic regression model was employed to assess the association between first-trimester air pollutant exposures and preeclampsia, gestational hypertension, preterm delivery and small for gestational age.

Findings and Conclusions: Adjusted association of PM₁₀ exposure was positive though not statistically significant for preeclampsia, gestational hypertension, preterm delivery and SGA. Adjusted association of PM_{2.5} was positive though not statistically significant for preeclampsia, and SGA, as well as positive and statistically significant for gestational hypertension and preterm delivery. Adjusted association of ozone with preeclampsia, gestational hypertension and preterm delivery was positive but not statistically significant and was not associated with SGA.

The association between PM₁₀ with gestational hypertension was higher in non-smokers than smokers, though effect estimates were similar for preterm delivery and SGA. The association between PM_{2.5} and preterm delivery was greater in non-smokers than in smokers, though similar for gestational hypertension and SGA. For ozone, the effect estimates for preterm delivery were greater in smokers, but associations for gestational hypertension and SGA were greater in non-smokers.

The adjusted association between PM_{2.5} and preterm delivery was positive and statistically significant in Caucasian women, but was not evident for African-American women. Adjusted association between PM₁₀ and gestational hypertension was positive in both Caucasian and African-American women, but was statistically significant only for African-American women.

Ambient Air Pollution Exposure and Blood Pressure Changes During Pregnancy

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Publication: Environmental Research, Vol. 117, 2012

Data: 1,684 pregnant women who resided in Allegheny County, Pennsylvania during 1997–2001; Questionnaire-based socio-demographic characteristics, reproductive and medical histories at baseline, diet, cigarette smoking and consumption of alcohol. Hospital records provided maternal residential ZIP codes at the time of delivery, maternal history of chronic hypertension and diabetes, systolic and diastolic blood pressure, maternal weight; one-hour CO, NO₂, SO₂, ozone; 24-hour PM₁₀, temperature, 1996–2001; PM_{2.5} 1999–2001

Analytic Method: Exposure assessment was conducted using space-time ordinary kriging to estimate air pollution levels at the centroid of each grid. One-hour CO, NO₂, SO₂ and ozone data were averaged to provide daily concentrations. Daily air pollutant concentrations averaged over first, second and third

trimesters of pregnancy (first 12 weeks, 13–27 weeks, >27 weeks, respectively). Lag 0–7 and mean eight-day concentrations prior to blood pressure measurement were also calculated. Multiple linear regression was used to estimate the association between air pollution exposures and blood pressure changes between first 20 weeks of gestation and late pregnancy. The effect of short-term air pollution exposures on blood pressure patterns was assessed using a linear mixed effects model.

Findings and Conclusions: Adjusted association between interquartile change in PM₁₀ levels and an increase in average blood pressure between the first 20 weeks of gestation and late pregnancy was positive and statistically significant for systolic blood pressure only for both the entire study population as well as the non-smoker subset, with the magnitude of effect substantially greater for non-smokers than for the entire population. Systolic blood pressure increases were positively associated with interquartile change in ozone levels in non-smokers only, and the adjusted association was statistically significant. Results for association of CO, SO₂ and NO₂ with blood pressure changes were null for both the entire study population and non-smokers. Analysis of blood pressure changes associated with first trimester ozone and PM₁₀ exposures stratified by race found both diastolic and systolic blood pressure changes in both Caucasians and African-American women.

Eight-day average ozone levels were associated with a slight rise in blood pressures during pregnancy. No other short-term pollutant exposure measures were associated with blood pressure changes during pregnancy.

Identifying Priority Pollutant Sources: Apportioning Air Toxics Risks Using Positive Matrix Factorization

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Publication: Environmental Science and Technology, Vol. 43, No. 24, 2009

Data: Hourly concentrations of 35 gas-phase organic hazardous air pollutant (HAP) compounds measured at three sites (Downtown, residential, background) in Pittsburgh, Pennsylvania; cancer unit risk estimates from EPA IRIS and California EPA

Analytic Method: A linear, no-threshold model was applied to unit risk estimates for the identified HAPs used to identify which of the measured HAPs contributed significantly to cancer risks. Positive Matrix Factorization (PMF) was used to apportion compounds with significant spatial and temporal variability, and thus assumed to be related to local sources. Cancer risks were calculated by multiplying the pollutant unit risk estimate by the fraction of the study-average outdoor concentration attributed by PMF to that factor. Additive cancer risks were estimated for each of the three monitoring sites. PMF factors were linked to sources or source categories (mobile, non-mobile, secondary/background) using a combination of meteorological data and published source profiles.

Findings and Conclusions: Emissions from non-mobile sources were the dominant contributor (~69 percent) to cancer risks at all three sites, including for benzene exposure. Emissions from large industrial facilities such as coke works and chemical plants, as well as smaller point sources such as dry cleaners, contributed significantly to cancer risks at all sites.

Spatial Variation in Ambient Air Toxics Concentrations and Health Risks between Industrial-influenced, Urban, and Rural Sites

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Publication: Journal of the Air and Waste Management Association, Vol. 60, 2010

Data: 24-hour average concentrations of 38 gas-phase hazardous air pollutant compounds measures at four sites (Downtown, two residential, background) in the Pittsburgh, Pennsylvania metropolitan area, 2006–2008; 2001–2002 and archived data on metals, polycyclic aromatic hydrocarbons, diesel particulate matter and coke oven emissions; Toxicity risk estimates from EPA IRIS, California EPA, and ATSDR

Analytic Method: Mean pollutant concentrations and spatial variability were calculated using a non-parametric bootstrap method. Spatial variability was also assessed using a correlation analysis of pollutant concentrations across the four monitoring sites on a given day.

A linear, no-threshold model was applied to estimate cancer risks, and a hazard quotient was calculated to estimate non-cancer risks. Cumulative cancer risk was calculated by adding lifetime cancer risks for multiple pollutants at each site. Hazard index was calculated by adding the hazard quotients for the pollutants identified at each site. An EPA model was applied to calculate interactive cancer and non-cancer risks associated with exposure to multiple pollutants.

Findings and Conclusions: Average concentrations for six of the HAPs (bromomethane, carbon tetrachloride, dichloroethane, 1,2 chloromethane, bromoform, formaldehyde) varied by less than 25 percent across the four sites. Only differences for formaldehyde were statistically significant across the four sites. Average concentrations of 32 HAPs were more than 25 percent higher, and average concentrations for 13 HAPs were a factor of two or more higher at one or more of the urban sites, indicating influence from local sources.

Eleven of the 38 organic compounds presented cancer risks greater than 1×10^{-6} , and three HAPs (formaldehyde, benzene, TCE) presented cancer risks greater than 1×10^{-5} . Formaldehyde and carbon tetrachloride contributed more than 50 percent of the cumulative cancer risk at all four sites. Benzene contributed substantial cancer risks at all sites, whereas trichloroethene and 1,4-dichlorobenzene contributed substantial cancer risks only at the Downtown site. Diesel particulate matter was estimated to pose a much greater cancer risk than other classes of air toxics including gas-phase organic, metals, polycyclic aromatic hydrocarbons and coke oven emissions.

Compared to the HAPs-related cancer risks for 14 other U.S. cities, Pittsburgh risks were in the lower two quartiles of the multi-city risk distribution. The profile of Pittsburgh HAPs related to risks was similar to that of the other U.S. cities. Only acrolein posed a chronic non-cancer risk based on a hazard quotient greater than one, with this risk present at all four monitoring sites.

Relationships of Air Pollution to Health: Results from the Pittsburgh Study

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Publication: Archives of Environmental Health, Vol. 38, No. 1, 1983

Data: Allegheny County, Pennsylvania; Total and heart disease mortality (date of death, age, sex, race, cause of death, residence), urgent and emergency hospital admissions; daily average SO₂ and Coefficient of Haze (CoH); maximum, minimum and average temperature, relative humidity; 1972–1977

Analytic Method: Regression model with linear filtering, with mortality and air pollution data corrected to eliminate extreme values greater than three standard deviations from monthly mean values. All data corrected to eliminate seasonal variation by subtracting the 15-day moving average from the daily value and corrected for temperature by regression of same day, lag up to 13 days and nonlinear functions of same day and lag days.

Findings and Conclusions: Total and heart disease mortality was positively associated (2.15 percent and 3.14 percent, respectively) with CoH for only one (Hazelwood) of the three monitoring sites, and the association was statistically significant only for the joint CoH and SO₂ analysis. No association between SO₂ and mortality was found.

Positive and statistically significant associations were found between CoH and total morbidity at all three monitoring sites in analysis of CoH alone and jointly with SO₂. Association between CoH and respiratory disease was positive and statistically significant for only one (Bellevue) of three monitoring sites. CoH was associated with heart disease at two of the monitoring sites (Bellevue and Logans Ferry), both alone and jointly with SO₂, and with other circulatory disease, only with data from the Hazelwood site.

Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987–1994

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Publication: The New England Journal of Medicine, Vol. 343, No. 24, 2000

Data: Non-external deaths by age group (<65, 65–74, ≥75 years), cause of death (cardiovascular, respiratory, other), selected demographic data; PM₁₀, ozone, SO₂, CO, NO₂; hourly temperature and dew point averaged over 24 hours; 1987–1994; 20 largest U.S. cities

Analytic Method: A two-stage, log-linear regression model was used to assess the relationship of the air pollutants with mortality. City-specific analysis including confounders was conducted in the first stage analysis, and city-specific mortality rates were combined with model fitting using a Bayesian statistical method in the second stage of analysis. Analysis was performed for PM₁₀ and ozone separately and combined, as well as trivariate models that included SO₂, NO₂ and CO after adjustment for PM₁₀ and ozone levels.

Findings and Conclusions: In the univariate analysis, PM₁₀ was positively associated with total and cause-specific death in most of the 20 cities, with an approximately 0.51 percent increase in the rate of mortality per 10 µg/m³ increase in PM₁₀. The effect of PM₁₀ exposure on cardiovascular and respiratory deaths was somewhat larger (0.68 percent). Inclusion of ozone in the bivariate analysis did not substantially affect the PM₁₀-mortality relationship. Results for the association of ozone with mortality were more variable across cities, both in the univariate and bivariate analysis with PM₁₀. Ozone levels were associated positively with increased mortality rates (0.41 percent/10 ppb) only during summer months. Analysis of the relationship of CO, NO₂ and SO₂ with mortality found little evidence of an association.

Results for the association of PM₁₀ and total mortality in Pittsburgh were positive both alone and combined with ozone, but were statistically significant in the univariate analysis only. The relationship of ozone with total mortality was positive but not statistically significant either alone or in the bivariate analysis with PM₁₀.

The National Morbidity, Mortality, and Air Pollution Study Part II: Morbidity and Mortality from Air Pollution in the United States

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Publication: Health Effects Institute Research Report 94, Part II, 2000

Data: Daily non-external cause deaths, cardiac, respiratory, pneumonia, influenza, other diseases deaths, classified by age (< 65 years, 65–74 years, and ≥75 years); PM₁₀, ozone, SO₂, NO₂, CO; temperature and dew point; 20 and 90 cities; 1987-1994. Daily Medicare hospital admissions for cardiovascular disease, chronic obstructive pulmonary disease, and pneumonia; PM₁₀, SO₂, NO₂, CO, ozone; temperature and relative humidity; city demographic characteristics; 14 cities; 1985-1994

Analytic Method: The 20-city analysis method is described in Samet et al. 2000a. For the 90-city analysis, a 3-stage regional model was used to estimate PM₁₀ effects for multiple U.S. regions and a weighted linear regression approach to identify determinants of heterogeneity of PM₁₀ coefficients across locations. In the first stage, a semiparametric log-linear regression model was applied to each city, while the second and third stages of the model examined between-city variation within each region and between-region variation in the log relative rates of mortality, respectively. A set of 33 county-specific variables organized into five groups was distilled to a set of nine variables that were used in the second-stage analysis using weighted linear regression. The 90-city analysis explored factors modifying the effect of PM₁₀ on mortality rates using a univariate linear regression models. Analysis of the impact of various exposure lag structures was conducted for 10 cities.

Findings and Conclusions: Results of the 20-city analysis with respect to the relationship of pollutants to mortality rates are reported in Samet et al. 2000a. Results of the analysis of the effect of mean level of pollutants, as well as socioeconomic factors as effect modifiers on the 90-city PM₁₀-mortality relationship, were weak and none were statistically significant. With respect to the pooled regional effects analysis, results were greatest for the Northeast.

Pittsburgh results not reported in Samet et al. 2000a include a positive (0.38 percent per 10 $\mu\text{g}/\text{m}^3$ PM_{10}) and statistically significant effect of a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} on total mortality with adjustment for ozone; a positive (0.57 percent per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10}) and statistically significant effect of on cardiopulmonary mortality without adjustment for ozone exposure; and a positive (0.19 percent per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10}) but not statistically significant effect for other cause mortality also unadjusted for ozone exposure. Results for Pittsburgh trivariate air pollution analyses indicated that the inclusion of ozone with NO_2 , SO_2 and CO with respect to PM_{10} risk of mortality remained positive, but only the model with ozone and SO_2 remained statistically significant.

Results for exploration of the effect of various lag structures indicated that results were largest for distributed 7 and 14 day lags, and results for lag 1, lag 2 and distributed 7 day lags were all statistically significant. An additional analysis of the effect of PM_{10} lag structure on the risk of mortality in those age ≥ 65 years found that the largest effect was for the mean of the same day and subsequent day of exposure (day 0 + day 1) (also reported in Schwartz 2000 study).

Analysis of the relationship between a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} exposure and hospitalization for cardiovascular disease (CVD), chronic obstructive pulmonary disease (COPD) and pneumonia for the combined 14 cities indicated positive and statistically significant results for the various lag structures examined. The largest effects for CVD and pneumonia hospitalizations were related to the day 0 + day 1 lag for PM_{10} exposures of $< 50 \mu\text{g}/\text{m}^3$, or below the daily PM_{10} NAAQS then in effect, which suggested that relatively low levels of PM_{10} exposures were of significant health concern. Socio-demographic factors, such as percent of population in poverty or those non-white, as well as weather factors, did not significantly modify the effects estimates. Pittsburgh-specific results for the effect of PM_{10} on CVD, COPD and pneumonia hospital admissions were positive and statistically significant for all lag structures.

The Distributed Lag Between Air Pollution and Death

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Publication: Epidemiology, Vol. 11, No. 3, 2000

Data: Daily non-external deaths, age ≥ 65 years; daily average PM_{10} , temperature, barometric pressure, relative humidity; 10 cities; 1986–1993

Analytic Method: A generalized additive Poisson regression model with nonparametric smooth functions was used to assess the effect of daily PM_{10} exposure with levels below 150 $\mu\text{g}/\text{m}^3$ on mortality under various lag structures (lag 0, lag 0+1, quadratic and unconstrained distributed lag).

Findings and Conclusions: The distributed lag models provided consistently larger effects than the single, same-day lag for nine of the 10 cities. Analysis of the various lag models indicated that the effect of PM_{10} exposure was spread over several days and did not reach 0 until lag day 5. Higher barometric pressure was found to be associated with a decreased risk of mortality.

With respect to Pittsburgh-specific results, the largest effect of PM_{10} on mortality (1.02 percent per 10 $\mu\text{g}/\text{m}^3$) was found with the 2-day mean (day 0+1 lag).

Particulate Air Pollution and Daily Deaths: A Multicity Case-Crossover Analysis

Authors & Affiliations: Joel Schwartz¹, Antonella Zanobetti¹

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Publication: Special Report: Revised Analysis of Time-Series Studies of Air Pollution and Health, Health Effects Institute, May 2003

Data: Daily non-external deaths, age ≥ 65 years; daily average PM₁₀, temperature, barometric pressure, relative humidity; 10 cities; 1986–1993

Analytic Method: Conditional logistic regression was used in a case-crossover design applied to the relationship of PM₁₀ to daily deaths in 10 cities previously analyzed in Schwartz et al. 2003. Day of death was matched to 18 control days selected seven to 15 days before and seven to 14 days after the death event. Day of the week, temperature and relative humidity were controlled in all analyses. A two-stage design involved city-specific regression analysis, followed by a second stage analysis combining the log odds ratios for all 10 cities. In an alternative approach, the strata from all 10 cities were combined in a single model.

Findings and Conclusions: Results of the combined log odds ratio 10-city analysis indicated a 0.35 percent increase in daily deaths per 10 $\mu\text{g}/\text{m}^3$ increment of PM₁₀, with nearly identical findings from the model that combined the strata from the individual cities into a single stage analysis. The results from the alternative case-crossover models were generally consistent with those found from use of the revised GAM model in Schwartz et al. 2003. Results of an analysis of the dose-response relationship between PM₁₀ and daily deaths, in which days with PM₁₀ concentrations below 15 $\mu\text{g}/\text{m}^3$ were compared to days with PM₁₀ levels in three higher PM₁₀ concentration ranges, support a monotonic, linear dose-response relationship.

Pittsburgh-specific findings were positive (0.30 percent for a 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀) but were not statistically significant.

Morbidity and Mortality Among Elderly Residents of Cities with Daily PM Measurements

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Publication: Special Report: Revised Analysis of Time-Series Studies of Air Pollution and Health, Health Effects Institute, May 2003

Data: Daily Medicare hospital admissions for cardiovascular disease, chronic obstructive pulmonary disease and pneumonia; PM₁₀, SO₂, NO₂, CO, ozone; temperature and relative humidity; city demographic characteristics; 14 cities; 1985–1994. Daily non-external deaths, age ≥ 65 years; daily average PM₁₀, temperature, barometric pressure, relative humidity; 10 cities; 1986–1993

Analytic Method: Generalized additive model (GAM) with revised convergence criteria, penalized and natural spline models were used to reanalyze the results reported in Samet 2000b.

Findings and Conclusions: The revised GAM model resulted in reductions of 7.6 percent for cardiovascular hospital admissions, 8.9 percent for chronic obstructive pulmonary disease admissions and 9.6 percent for pneumonia admissions. The reduction in effects using the natural splines model was larger. Results for the combined city analysis indicated positive and statistically significant results for all three categories of hospital admissions.

Findings for Pittsburgh of the relationship of mean lag 0 + lag 1 PM₁₀ exposures with pneumonia hospital admissions using a natural spline model found increasing risk with increasing PM₁₀ exposure at PM₁₀ levels below 30 µg/m³, though risks fell at higher PM₁₀ levels. The study authors note that highest PM₁₀ levels occurred in Pittsburgh during July and August, though the majority of pneumonia hospital admissions occur during winter months, suggesting that future analyses of pneumonia hospitalizations should be restricted to winter months.

PM₁₀-related premature death results were reduced compared to original NMMAPS results, though distributed lag model results were larger than those from single day exposure models. Pittsburgh results for the relationship of PM₁₀ and deaths using the three alternative models with distributed lags (GAM, natural spline, penalized spline) were all positive, though relatively small effects were found.

The Effect of Particulate Pollution on Daily Deaths: A Multi-City Case Crossover Analysis

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Publication: Occupational and Environmental Medicine, Vol. 61, No. 12, 2004

Data: Daily non-accidental deaths; daily average PM₁₀, temperature, relative humidity; 14 cities; 1986–1993

Analytic Method: Conditional logistic regression was used in a case-crossover design applied to the relationship of PM₁₀ to daily deaths in 14 cities. Day of death was matched to 18 control days selected seven to 15 days before and seven to 15 days after the death event. Day of the week, temperature and relative humidity were controlled in all analyses. A two-stage design involved city-specific regression analysis, results of which were then combined to estimate the log odds ratios for all 14 cities. In a second stage analysis, all the strata were combined into a single model. In a third analysis, a stratified approach was applied based on selection of a control day for each death that was matched not only on season, but temperature as well. For comparison purposes, analysis for each city was also conducted using a Poisson regression model. To test the shape of the dose-response curve, the linear term for PM₁₀ was replaced by indicator variables for days with PM₁₀ concentrations between 15 and 25 µg/m³; 25 to 34 µg/m³; 35 to 44 µg/m³; and 45 µg/m³ and above.

Findings and Conclusions: Case-crossover analysis of the combined 14-city data using multiple analytical models found an increased risk of daily death ranging from 0.32 to 0.53 percent per 10 µg/m³ increment of PM₁₀. Results from the case-crossover analysis were consistent with those from the Poisson regression analysis (0.40 percent increase per 10 µg/m³ increment of PM₁₀). The dose-response analysis results “shows little evidence for a deviation from non-linearity.” Results for Pittsburgh were positive (0.30 increase per 10 µg/m³ increment of PM₁₀), though not statistically significant.

Effect of the Pittsburgh Air Pollution Episode Upon Pulmonary Function in Schoolchildren

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Publication: Journal of the Air Pollution Control Association, Vol. 26, No. 6, 1976

Data: 24-hour average Coefficient of Haze (CoH), SO₂, total suspended particulates, sulfate, nitrate, November 17–26, 1976; Forced expiratory volume in three-quarters of a second (FEV_{.75}), forced vital capacity (FVC), November 20–26, 1976; Allegheny County, Pennsylvania

Analytic Method: Lung function tests were conducted on 272 students in the fourth, fifth and sixth grades of parochial schools located near air pollution monitors in Allegheny County, Pennsylvania. Results were recorded for each student for seven successive days and analyzed for each school separately using a general linear model. Analysis results were presented as deviation from school pulmonary function means for each day.

Findings and Conclusions: Study was conducted in conjunction with an “air pollution emergency” reflecting high levels of air pollution from November 17–21, 1976. No indication of a consistent increase in pulmonary function with time was found in the more polluted areas, and a slight downward trend in lung function was observed. There was much larger variability in lung function results on weekend days compared to weekdays. The authors conclude that “gross impairment of lung function, sufficiently severe for that lung function to require several days to recover, did not occur, at least not in a sufficient number of children that the effect could be detected in these primary school study groups as a whole.”

Identifying a Susceptible Subgroup: Effect of the Pittsburgh Air Pollution Episode Upon Schoolchildren

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Publication: American Journal of Epidemiology, Vol. 110, No. 1, 1979

Data: 24-hour average Coefficient of Haze (CoH), SO₂, total suspended particulates, sulfate, nitrate, November 17–26, 1976; Forced expiratory volume in three-quarters of a second (FEV_{.75}), forced vital capacity (FVC), November 20–26, 1976; Allegheny County, Pennsylvania

Analytic Method: Re-analysis was done on the lung function data collected during and subsequent to the air pollution episode in Allegheny County, Pennsylvania from November 17–21, 1976. A chi-square analysis was used to assess homogeneity of the distribution of the regression coefficients for the pulmonary function tests over time for the 244 children included from the original set of 272 (see Stebbings et al. 1976). Lung function results from non-white, asthmatic and children with insufficient

data were eliminated from the original data set. Linear least squares regression of lung function results on study day number was used to analyze results from each student.

Findings and Conclusions: Lung function results from a subset of 10–15 percent of study subjects indicated that a recovery of FVC occurred, suggesting impairment of approximately 20 percent of average FVC. Results were consistent by sex and by school within sex. Analysis of lung function results for subgroups of children with lower than expected baseline lung function or who reported acute respiratory symptoms did not indicate that these subgroups responded with impairment greater than the whole group.

Particulate Air Pollution and the Rate of Hospitalization for Congestive Heart Failure Among Medicare Beneficiaries in Pittsburgh, Pennsylvania

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Publication: American Journal of Epidemiology, Vol. 161, No. 11, 2005

Data: Hospital admissions for congestive heart failure (CHF) in people age ≥ 65 years in Allegheny County, Pennsylvania; 24-hour average PM₁₀; hourly ozone, CO, SO₂, NO₂; daily mean temperature, barometric pressure, dew point; 1987–1999

Analytic Method: Hourly air pollution data were averaged to provide daily mean concentrations. A case-crossover design was used to assess the effect of changes in daily mean air pollution levels, with PM₁₀ as the exposure of interest, using either day of event (lag 0) or one to three days prior to the event (lags 1-3) as the exposure timeframe. Conditional logistic regression was used for each hospitalization to assess the effect of an interquartile-range increase in the daily mean level for each pollutant. Effect modification was examined considering age (≥ 80 v. 65-79), gender and the presence of several health-related secondary diagnoses.

Findings and Conclusions: In single pollutant models, four of five pollutants examined (PM₁₀, CO, NO₂, SO₂) were positive and statistically significant for an interquartile-range increase in ambient concentrations. Effect of pollutant exposure was greatest for CO and NO₂. In two-pollutant models, the association between CO and NO₂ were the most robust. The association of PM₁₀ with CHF hospitalization became negative in two-pollutant models with CO and NO₂. However, the authors note that given the high level of correlation between PM₁₀, CO and NO₂, the two-pollutant model is of limited value for disentangling the effect of each pollutant.

Air Pollution and Hospital Admissions for Ischemic and Hemorrhagic Stroke Among Medicare Beneficiaries

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Publication: Stroke, Vol. 36, 2005

Data: Daily hospital admissions for cerebrovascular disease or occlusion of cerebral arteries, excluding those without cerebral infarction (ischemic stroke), intracerebral hemorrhage (hemorrhagic stroke); daily PM₁₀; hourly CO, NO₂, SO₂; temperature, relative humidity; 9 cities; health, air pollution, and climatologic data vary by city (Pittsburgh: 1987–1999)

Analytic Method: A two-stage hierarchical model was used to assess the relationship between air pollution and hospital admissions. A time-stratified case-crossover design was employed in the first stage analysis, using conditional logistic regression. Results of the analysis were reported as a percentage change in the rate of hospitalization for an interquartile-range increase in same-day daily mean air pollution levels. The second stage analysis combined the random-effects estimates from the first stage city-specific effect estimate using a random-effects meta-analysis method.

Findings and Conclusions: A positive association was found for the relationship of an interquartile-range change in PM₁₀ and ischemic stroke for seven of the nine cities, and the overall association was positive and statistically significant for all pollutants. Largest effects were for exposure to NO₂ and CO. In the combined city analysis, an interquartile-range increase in PM₁₀ or other pollutants was not associated with increased risk of hemorrhagic stroke.

Pittsburgh-specific association was positive but not statistically significant for an interquartile-range increase in PM₁₀ with both ischemic and hemorrhagic stroke admissions.

Case-Crossover Analysis of Air Pollution and Cardiorespiratory Hospitalizations: Using Routinely Collected Health and Environmental Data for Tracking: Science and Data

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Publication: Journal of Public Health Management and Practice, Vol. 14, No. 6, 2008

Data: Cardiorespiratory hospital admissions for age ≥ 65 years; daily PM₁₀, SO₂; daily mean temperature, relative humidity; Pittsburgh, Pennsylvania; 1996–2000

Analytic Method: A case-crossover approach was used to assess the relationship of air pollution with cardiorespiratory hospitalization related to the operation and then closing of a steel coke oven in Pittsburgh, Pennsylvania. Both a bidirectional and time-stratified method were used for selection of the control period.

The bidirectional control analysis was conducted for two periods: 1) January 1, 1996– February 28, 1998 (closing of plant), and 2) March 1, 1998–December 31, 2000. Bidirectional control analysis was conducted for cardiovascular and respiratory hospital admissions separately, as well as jointly. Conditional logistic regression was used to assess the relationship between pollutant exposure and hospital admissions, with air pollutants fitted into the model as either continuous or categorical (quartile) variables. Odds ratios were calculated for continuous variables and the quartiles of each pollutant for each time period adjusted for current day mean temperature and relative humidity and using the first quartile as the referent. For the time-stratified control approach, the air pollutants were fitted into the model only as continuous variables.

Findings and Conclusions: With PM₁₀ fitted as a categorical variable, significant associations were found between the highest quartile of PM₁₀ levels and cardiovascular and cardiopulmonary hospitalizations for the period prior to the plant closing. A trend of increased risk of cardiovascular and cardiopulmonary hospitalizations was found for the period prior to the plant closing, however these associations were no longer statistically significant for the period after the plant closing. Similar results were found for the approach in which PM₁₀ was fitted as a continuous variable. An association between SO₂ and hospitalizations was not found either before or after the plant closing for the categorical analysis.

PM₁₀ Air Pollution Exposure During Pregnancy and Term Low Birth Weight in Allegheny County, PA, 1994–2000

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Data: Live births; hourly PM₁₀; maternal age, education, race, tobacco use, level of prenatal care, history of low birth weight (LBW) or preterm infant, parity, birth season, infant sex, gestational age, maternal weight gained; Allegheny County, Pennsylvania; 1994–2000

Analytic Method: Hourly PM₁₀ data was aggregated into daily and quarterly averages, with quarterly averages used to estimate levels at centroids of census tracts. Maternal PM₁₀ exposure levels were calculated based on estimates using inverse distance-weighting approaches for the distance from the residence census tract to the centroid monitoring site. Trimester-specific and nine-month exposures were calculated using quarterly weighted PM₁₀ measures, month, quarter and year of birth conception.

A logistic regression model was used to assess the relationship between PM₁₀ exposure and term LBW. Odds ratios were calculated by trimester, as well as for the nine-month gestation period, for the risk of term LBW with an interquartile increase in PM₁₀ levels, controlling for maternal age, race, education, smoking, weight gain, parity, previous LBW or preterm birth, level of prenatal care, birth season, gender of infant and gestation age.

Findings and Conclusions: The association between an interquartile increase in PM₁₀ levels and term LBW was positive for all trimester and the nine-month exposure periods, but was statistically significant only for the first and second trimesters (odds ratios of 1.13 and 1.10, respectively). The potential effect of other air pollutants was not evaluated for this study.

Cardiovascular Damage by Airborne Particles: Are Diabetics More Susceptible?

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Data: Cardiovascular (CV) hospital admissions for persons age ≥ 65 years, diabetes as secondary admission factor; daily PM₁₀; day-of-event and prior-day temperature, relative humidity, barometric pressure; day of week; four U.S. cities; 1988–1994

Analytic Method: A two-stage hierarchical model was used to assess whether there were differences in the risk of CV hospital admissions for those with and without diabetes. Admissions were also stratified by two age categories (65–74, 75+). A generalized additive Poisson regression model was applied to develop the city-specific effect of PM₁₀ on hospital admissions, stratified by age group and diabetes diagnosis. The mean of PM₁₀ levels on the day of the admission and prior day was used as the exposure variable. The percentage increase in CV hospital admissions was estimated for a 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀. In the second stage analysis, the estimated effect size for PM₁₀ for each city and strata was regressed against the stratification variables in a meta-regression model.

Findings and Conclusions: Higher effects for the association of a 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀ on CV hospital admissions was found in all four cities for those with a secondary diagnosis of diabetes compared to those without diabetes. In the meta-regression analysis for all four cities, the effects estimate for those with diabetes was nearly double that of those without diabetes, with suggestion of a somewhat smaller effect in the older age group category.

Results for Pittsburgh found that the effect PM₁₀ on CV hospitalizations was three times greater for those with diabetes than those without in the younger age category. However, the effect for diabetics was smaller and not statistically significant for those with diabetes in the older group.

Mortality Displacement in the Association of Ozone with Mortality: An Analysis of 48 Cities in the United States

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Data: Daily non-external deaths, cardiovascular, stroke, respiratory death; eight-hour mean ozone; temperature, dew point; 48 cities; 1989–2000

Analytic Method: A two-stage approach was used for this study. In the first step, a generalized linear model was used to assess the relationship of eight-hour mean ozone levels during the summer months (June–August) on daily deaths for each city. Exposure was assessed using ozone levels on day of death, as well as up to 20 days prior to death in a distributed lag model, and the two approaches were then

compared to assess whether there is evidence of “harvesting” (i.e. short-term mortality displacement). A penalized quasi likelihood was used to estimate the coefficient of the smooth distributed lag. In the second stage analysis, the city-specific results were combined using a meta-regression approach.

Findings and Conclusions: There was heterogeneity in the results of ozone exposure on mortality across the 48 cities. In the combined city analysis, an increase of 10 parts per billion in day of death (lag 0) ozone exposure was associated with a 0.3 percent increase in total mortality; a 0.5 percent increase in cardiovascular mortality; a 0.5 percent increase in respiratory mortality; and a 0.4 percent increase in stroke mortality. When an unconstrained distributed lag was used, effects estimates increased to 0.5 percent for total mortality; a 0.5 percent increase in cardiovascular mortality; a 0.6 percent increase in respiratory mortality; and a 2.2 percent increase in stroke mortality. A larger effect was found in cities with a lower percentage of central air-conditioning. There was no evidence of short-term mortality displacement from an analysis of 21 days of ozone exposure simultaneously in an unconstrained distributed lag model.

Pittsburgh-specific results were negative for the relationship of lag 0 ozone exposure with total mortality.