

Estimating Error in Using Residential Outdoor PM_{2.5} Concentrations as Proxies for Personal Exposures: a Meta-Analysis

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Abbreviations: CI, confidence interval; PM, particulate matter; PM_{2.5}, particulate matter < 2.5 ug/m³; *r*, within-participant residential outdoor-personal PM_{2.5} correlation; SLP, sea level pressure

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ABSTRACT

Background: Studies examining the health effects of particulate matter $< 2.5 \mu\text{m}$ ($\text{PM}_{2.5}$) commonly use ambient $\text{PM}_{2.5}$ concentrations measured at distal monitoring sites as proxies for personal exposure assuming spatial homogeneity of ambient $\text{PM}_{2.5}$. An alternative proxy—the residential outdoor $\text{PM}_{2.5}$ concentration measured adjacent to participant homes—has few advantages under this assumption. Objectives: To systematically review residential outdoor-personal $\text{PM}_{2.5}$ correlation (r) estimates as a means of comparing the magnitude and sources of measurement error associated with their use as exposure surrogates. Methods: We searched seven electronic reference databases for studies of the within-participant, residential outdoor-personal $\text{PM}_{2.5}$ correlation. Results: The search identified 567 candidate studies, nine of which were abstracted in duplicate. The studies were published between 1996 and 2008. They represented 329 non-smoking participants aged 6-93 years in eight U.S. cities among whom r (median 0.53; range 0.25-0.79) was estimated based on a median of seven residential outdoor-personal $\text{PM}_{2.5}$ pairs per participant. There was modest evidence of publication bias (symmetric funnel plot; $P_{\text{Begg}}=0.4$; $P_{\text{Egger}}=0.2$); however, evidence of heterogeneity was identified (Cochran's Q test $P=0.05$). Of the 20 characteristics examined, earlier study midpoints, eastern longitudes, older mean age, higher outdoor temperatures, and lower personal-residential outdoor $\text{PM}_{2.5}$ differences were associated with increased r . Conclusions: These findings were similar to those from a contemporaneous meta-analysis that examined ambient-personal $\text{PM}_{2.5}$ correlations (median 0.54; range 0.09-0.83). Collectively, the meta-analyses suggest that residential outdoor-personal and ambient-personal $\text{PM}_{2.5}$ correlations merit greater consideration when evaluating the potential for bias in studies of $\text{PM}_{2.5}$ -mediated health effects.

Numerous epidemiological and toxicological studies have linked particulate matter (PM) air pollution with adverse health outcomes, including mortality (Burnett et al. 2000; Dominici et al. 2003; Katsouyanni et al. 2003), hospital admissions (Burnett et al. 1995; Linn et al. 2000; Oftedal et al. 2003), and subclinical disease (Diez Roux et al. 2008; Liao et al. 2009; Whitsel et al. 2009). A common feature of such studies is their reliance on ambient PM concentrations measured at distal monitoring sites as proxies for personal exposure to PM of ambient origin. The reliance is consistent with regulatory policies developed under Clean Air Act, which have been informed by studies of the correlation between personal exposures to PM originating outdoors and residential outdoor PM concentrations (Wallace 2000). However, ambient PM may not adequately represent total PM exposure, as human activity pattern surveys suggest that on average, individuals spend >85% of their time inside (Klepeis et al. 2001) where they are exposed to numerous sources of indoor PM, the physico-chemical properties and toxicities of which often differ from those of ambient PM (Monn and Becker 1999; Wainman et al. 2000).

Available exposure studies, although small in number, have suggested that several factors may influence the relationship between ambient and total PM exposure, including home ventilation, indoor PM sources, and time-activity patterns (Williams et al. 2003a; Sarnat et al. 2006; Rodes et al. 2001). As these factors are not well quantified (Janssen et al. 1998), we previously reviewed the literature examining the within-participant, ambient-personal PM_{2.5} correlation to determine the magnitude and sources of measurement error inherent in using ambient PM_{2.5} as a surrogate for personal exposure (Avery et al. 2009 (in press)). We found that characteristics of participants, studies and the environments in which they are conducted affect

the accuracy of ambient PM_{2.5} as a proxy for personal exposure and that the potential for exposure misclassification may be substantial.

Although the residential outdoor PM_{2.5} concentration measured adjacent to participant homes may be equally prone to misclassification under the assumption of spatial homogeneity, use of this measure as an alternative proxy for personal exposure may have some advantages if this assumption is not uniformly applicable. Studies of spatial variability in ambient PM_{2.5} concentrations among 27 U.S. urban areas (Pinto et al. 2004) suggest that this may be the case. The fact that PM_{2.5} varies at the micro-environmental level as a function of e.g. topography, proximity to PM_{2.5} point sources, adjacency to major traffic arterials, and prevailing winds (United States Environmental Protection Agency 2009; Zhu et al. 2002) also is consistent with this suggestion. It nonetheless remains unclear how spatial variability and outdoor microenvironments affect the use of ambient PM_{2.5} concentrations as a proxy for personal PM_{2.5} exposure. We therefore reviewed the literature examining the within-participant, residential outdoor-personal PM_{2.5} correlation and contrasted its meta-analytic findings with those from the review of the within-participant, ambient-personal PM_{2.5} correlation referenced above (Avery et al. 2009 (in press)). Findings from the two meta-analyses will facilitate quantification of bias resulting from the use of surrogates for personal PM_{2.5} exposure in studies relying on outdoor PM_{2.5} measurements.

MATERIALS AND METHODS

Systematic Review Strategy

A search strategy was devised to identify studies of the within-participant, residential outdoor-personal PM_{2.5} correlation. No document type, language, or publication starting-date limitations were used. Searches were conducted in PubMed (1950 to date), ISI Web of Science (1955 to date), ISI BIOSIS Previews (1969 to date), CSA Environmental Sciences and Pollution Management (1967 to date), Toxline (1965 to date), and Proquest Dissertations & Theses (1861 to date) on November 12, 2007. STN EMBASE (1974 to date) was searched on December 14, 2007.

The following strategy was used to search PubMed: (PM 2.5 OR PM2.5 OR PM25 OR PM 25 OR fine particle*) AND (ambient OR outdoor OR outdoors OR outside OR exterior OR external OR background OR fixed site*) AND (individual OR personal) AND (correlat* OR associat* OR relat* OR compar* OR pearson OR spearman). The same four sets of keywords were adapted for input into Web of Science, BIOSIS, Environmental Sciences, Toxline, and EMBASE. The Dissertations & Theses search only required the first three sets of keywords to create a small enough result set for review.

Citations were downloaded to an electronic reference manager (EndNote X1®, Thomson Reuters), de-duplicated, and supplemented with secondary references cited by articles identified in the primary search. The citations were independently reviewed with respect to three inclusion criteria: measurement of residential outdoor PM_{2.5}, measurement of personal PM_{2.5}, and estimation of the within-participant, residential outdoor-personal PM_{2.5} correlation. Study,

participant and environment characteristics were extracted from all articles meeting inclusion criteria. Study characteristics included journal of publication, publication date, setting, study dates, sample size, duration, timing (consecutive; non-consecutive), lower limit of PM_{2.5} detection, number (minimum; mean) of paired PM_{2.5} measures, and correlation metric (Pearson; Spearman). Participant characteristics included age (mean; minimum; maximum), % female, and the presence of comorbidities (pulmonary; cardiovascular; multiple; neither). Environmental characteristics included the mean, median and standard deviation of PM_{2.5} concentrations (residential outdoor; personal), the within-participant, residential outdoor-personal PM_{2.5} correlation coefficients and corresponding number of paired measurements, season, distance to monitor, monitor type, air exchange rate, % of time using air conditioning, and % of time with windows open. Discrepant exclusions and extractions were adjudicated by consensus. Supplemental data were requested from authors by electronic mail as needed. City-specific longitudes and latitudes were obtained from the GEOnet Names Server (GEOnet Names Server 2009). Meteorological data were obtained from the National Climatic Data Center (National Climatic Data Center 2009).

Statistical analysis

Summary correlation and variance estimates for the j^{th} study were estimated from the personal-ambient PM_{2.5} correlations measured within each of the i^{th} participants. Each within-participant correlation coefficient (r_i) was converted to its variance-stabilizing, Fisher's z-

transform (Z_{r_i}) = $\frac{1}{2} \log_e \left(\frac{1+r_i}{1-r_i} \right)$ (Fisher 1925). Estimates of the within-participant variance (v_i) =

$\frac{1}{n_i - 3}$ and between-participant variance (τ_j^2) = $\frac{Q_j - (k_j - 1)}{c}$ for the j^{th} study were estimated

from the number of paired personal-residential outdoor PM_{2.5} measurements for each participant (n_i), the number of participants per study (k_j), the weighted sum of squared errors (Q_j) =

$$\sum_{i=1}^k (n_i - 3)(Z_{r_i} - \bar{Z}_{r_i})^2, \text{ and a constant } (c) = \sum_{i=1}^k (n_i - 3) - \frac{\sum_{i=1}^k (n_i - 3)^2}{\sum_{i=1}^k (n_i - 3)}. \text{ The transformed effect size}$$

$$\text{for the } j^{th} \text{ study is given by } \bar{Z}_j = \frac{\sum_{i=1}^k w_i Z_{r_i}}{\sum_{i=1}^k w_i} \text{ with participant-specific weights } (w_i) =$$

$$\left(\frac{1}{n_i - 3} + \tau_j^2 \right)^{-1}, \text{ study-specific standard errors } (S_j) = \sqrt{\frac{1}{\sum_{i=1}^k w_i}}, \text{ and study-specific weights}$$

$$W_j = \left(\frac{1}{S_j} \right)^2. \text{ Negative } \tau^2 \text{ estimates were set to 0 (Field 2001).}$$

Publication bias, present when study results influence the chance or timing of publication (Begg and Berlin 1989), was assessed using a “funnel plot” of W_j versus \bar{Z}_j . In the absence of publication bias, plots usually resemble a symmetrical funnel with the more precise estimates forming the spout and the less precise estimates forming the cone. We also evaluated the adjusted rank correlation (Begg and Mazumdar 1994) and regression asymmetry tests (Egger et al. 1997) as well as a non-parametric “trim and fill” method that imputes hypothetically missing results due to publication bias (Duval and Tweedie 2000). Low P values associated with the former tests (P_{Begg} ; P_{Egger}) give evidence of asymmetry.

Inter-study heterogeneity was evaluated using a plot of $\frac{\bar{Z}_j}{S_j}$ versus $\frac{1}{S_j}$ (Galbraith 1988)

and Cochran's Q test (Cochran 1954). The plot and test are related in that the position of the j^{th} study along the vertical axis illustrates its contribution to Q test statistic. In the absence of appreciable evidence of heterogeneity, all studies fall within the 95% confidence limits and $P_{\text{Cochran}} > 0.1$.

Variation in the strength and precision of \bar{Z}_j across levels of the study, environment, and participant characteristics was first assessed by estimating a summary random-effects estimate of \bar{Z} within each study, environment and participant category (Berkey et al. 1995). A series of univariable random-effects meta-regression models were also constructed to relate each study, environment, and participant characteristic to differences in \bar{Z} . Lastly, a multivariable random-effects meta-regression model and a backwards elimination strategy were used to evaluate ten study, participant, and environment characteristics routinely available in epidemiologic studies of PM_{2.5} health effects: latitude, longitude, mean age, % female, mean residential outdoor PM_{2.5}, relative humidity, sea level pressure (SLP), and mean temperature. Interval-scale characteristics were analyzed before and after dichotomization at their medians unless noted otherwise. All analyses were performed using STATA (College Station, TX). To facilitate interpretation, estimates of \bar{Z} were back-transformed to their original metric \bar{r} after data analysis.

RESULTS

The systematic review identified 567 candidate studies for screening. Of these studies, nine (2%) met criteria for critical appraisal and were abstracted (Brown et al. 2008; Liu et al. 2003; Reid 2003; Rodes et al. 2001; Rojas-Bracho et al. 2000; Suh et al. 2003; Wallace 1996; Williams et al. 2000a; Williams et al. 2000b; Williams et al. 2003b). Abstracted studies were published between 1996 and 2008 (Table 1), were set in eight cities in six U.S. states and were conducted between 1989 and 2001. The median study duration was 1.9 months (range 0.2, 15.2), a period in which 70% of the studies collected PM_{2.5} data over consecutive days. During data collection, the studies recorded a median of seven (range 5, 20) residential outdoor and personal PM_{2.5} concentration pairs per participant on which the within-participant Pearson (63%) and Spearman (37%) correlation coefficients were based (Table 1).

The studies represented 329 non-smoking participants aged 6-93 (median 70) years, 55% of whom were female and 25% of whom did not report chronic pulmonary or cardiovascular disease (Table 2). On average, residential outdoor PM_{2.5} concentrations (range 8.6, 42.6 ug/m³) were lower than personal PM_{2.5} concentrations (range 9.3, 70.0 ug/m³), with a median residential outdoor-personal PM_{2.5} difference of -1.55 (range -27.4, 9.0) (Table 3). The estimated \bar{r} (median 0.53; range 0.25, 0.79) and its standard deviation varied widely (Figure 1), the latter reflecting variability in sample weights (median 53.6; range 9.4, 548.1). Temperature (range 2.0, 24.0 °C) and relative humidity (range 27.3%, 78.9%) were also variable.

Figure 2, a funnel plot of \bar{Z}_j , suggested little evidence of asymmetry. This was consistent with $P_{\text{Begg}} = 0.4$, $P_{\text{Egger}} = 0.2$, although the “trim and fill” analysis imputed seven

hypothetically missing studies. Figure 3, a Galbraith plot in which three observations fell outside the 95% confidence limits, provided evidence of heterogeneity. This evidence was consistent with $P_{\text{Cochran}} = 0.05$.

Several study, participant, and environment characteristics were suggestively associated with moderate increases in the within-participant residential outdoor-personal $\text{PM}_{2.5}$ correlation coefficient (Figure 4), including earlier study midpoints, eastern longitudes, older mean age, lower personal-residential outdoor $\text{PM}_{2.5}$ differences (and ratios), and higher mean temperatures (Figure 5). When evaluating multivariable meta-regression models, only higher mean ages and eastern longitudes were associated with an increased within-participant residential outdoor-personal $\text{PM}_{2.5}$ correlation coefficient ($P < 0.05$).

DISCUSSION

Epidemiologic studies of PM_{2.5} health effects typically estimate PM_{2.5} exposures using daily mean concentrations obtained from either a single ambient PM_{2.5} monitoring site or averaged across several sites (United States Environmental Protection Agency 1996). Although rapid dispersion and secondary formation of atmospheric PM_{2.5} via chemical reaction of gases like SO₂, NO_x and NH₃ ensure some geographic uniformity of the monitored concentrations, primary sources of anthropogenic PM_{2.5} including traffic, construction, and industry (Samet and Krewski 2007) can increase the spatial variability of PM_{2.5}. Additional factors that influence the relationship between ambient PM_{2.5} concentrations and PM_{2.5} exposures include home ventilation, indoor activities associated with generation or resuspension of PM_{2.5} like cooking or cleaning, and time-activity patterns (Liu et al. 2003; Williams et al. 2000b). Thus, estimates of PM_{2.5} exposure based on ambient PM_{2.5} concentrations are associated with an acknowledged degree of uncertainty (Janssen et al. 1998).

To further characterize this uncertainty, the present study extended a prior meta-analysis of the within-participant ambient-personal PM_{2.5} correlation (Avery et al. 2009 (in press)) by examining the within-participant residential outdoor-personal PM_{2.5} correlation using analogous meta-analytic methods. In both cases, the examination generated little evidence for publication bias of Fisher's z-transformed \bar{r} , but strong evidence of heterogeneity. Several study, participant, and environment characteristics were associated with an increased \bar{r} , including earlier study midpoints, eastern longitudes, lower personal-residential outdoor PM_{2.5} differences (and ratios), higher mean ages, and higher mean temperatures. Moreover, the direct association

between eastern longitudes and increased r was consistent with the prior meta-analysis of the within-participant ambient-personal PM_{2.5} correlation.

The direct association between eastern longitudes and increased r may reflect several regional factors including higher urban PM_{2.5} concentrations (Rom and Markowitz 2006) or a greater influence of secondary PM_{2.5} sources in eastern locales (Pinto et al. 2004). The associations between lower residential outdoor-personal PM_{2.5} differences (and ratios) and higher mean temperatures and \bar{r} may also suggest an increased contribution of outdoor PM_{2.5} to personal exposure, either through time-activity patterns or increased air exchange. We were unable to fully evaluate the influence of these factors given the limited number of published studies and their inconsistent reporting of other geographic, household, and personal factors potentially responsible for the above associations. However, higher mean ages and eastern longitudes were associated with increased \bar{r} in the multivariable prediction model that included study, participant, and environment characteristics routinely available in epidemiologic studies of PM_{2.5} health effects.

While the meta-analyses of the ambient-personal and residential outdoor-personal PM_{2.5} correlations summarized a wide range of published correlation coefficients, both of them estimated a median $\bar{r} \approx 0.5$, which suggested that attempting to account for spatial variability and outdoor microenvironments did not appreciably affect the use of outdoor PM_{2.5} concentrations as proxies for personal PM_{2.5} exposure in the settings examined by the source studies. Nonetheless, these simple measures of central tendency have potentially important implications for studies using PM_{2.5} concentrations measured at distal or proximal monitoring

sites. For example, a value of $\bar{r} = 0.5$ implies that, on average, only \bar{r}^2 or one-fourth of the variation in personal $PM_{2.5}$ is explained by ambient or residential outdoor $PM_{2.5}$ concentrations. Under a simple measurement error model, it also implies that the variances of ambient or residential outdoor $PM_{2.5}$ concentrations are $1/\bar{r}^2$ or four times as large as the variance of the true, but often unmeasured, personal $PM_{2.5}$ exposure. Moreover, values of $\bar{r} = 0.5$ in diseased and non-diseased subpopulations (i.e. non-differential exposure measurement error) imply that [1] sample sizes needed to detect between-group differences in mean ambient or residential outdoor $PM_{2.5}$ concentrations are $1/r^2$ or four-fold as large as those needed to detect the same differences in personal $PM_{2.5}$ exposures, and [2] effect estimates expressed per $\mu g/m^3$ increases in ambient or residential outdoor $PM_{2.5}$ concentrations are equal to those associated with the same $\mu g/m^3$ increases in personal $PM_{2.5}$ exposure, albeit attenuated toward the null by the power, r^2 or 0.25. The latter form of attenuation is capable of obscuring weak to modest health effects of $PM_{2.5}$ (Armstrong et al. 2003), yet it cannot be adequately controlled by methods commonly used to account for confounding (Greenland and Robins 1985).

Given the above considerations, it is tempting to assume that all health effect estimates based on ambient or residential outdoor $PM_{2.5}$ concentrations would be considerably larger if they were instead based on personal $PM_{2.5}$ exposures, but to do so would yield more biased estimates if the original $PM_{2.5}$ -disease associations were spurious due to chance or confounding (Armstrong 1998). This justifies the application of the present findings to the $PM_{2.5}$ -disease associations that are the most precise and least biased according to criteria used to judge epidemiologic evidence (Hill 1965; Poole 2001; United States Environmental Protection Agency 2009). Furthermore, factors associated with \bar{r} , such as mean age and eastern longitudes, may

differ among participants and the studies in which they are enrolled. It is therefore difficult to predict the degree to which PM_{2.5} health effects estimates may be biased by exposure measurement error. Nonetheless, the above examples clearly illustrate that the impact of \bar{r} on the interpretation of findings from studies of PM_{2.5} health effects may be substantial.

Although the present study attempted to quantify the error associated with using residential outdoor and ambient PM_{2.5} concentrations as proxies for total personal exposure, the approach adopted here has several limitations. First, residential outdoor and ambient PM_{2.5} concentrations are likely to be poor proxies for exposure to non-ambient particles because particles originating indoors have different compositions and biologic properties (Long et al. 2001). Although the relative toxicity of outdoor and indoor particles remains under investigation, results from a panel study of sixteen chronic obstructive pulmonary disease patients in Vancouver, British Columbia reported that only those particles originating outdoors were associated with adverse cardiopulmonary effects (Ebelt et al. 2005). Moreover, the present study did not evaluate the correlation between concentrations of particles originating almost exclusively outdoors (e.g. sulfate or elemental carbon) and personal PM_{2.5} exposure, despite reports that their associations with ambient PM_{2.5} are particularly strong (Sarnat et al. 2006; Ebelt et al. 2000). Further work examining the relative contributions of PM_{2.5} constituents to PM-mediated health effects is clearly needed.

In summary, the results presented here and in the previous meta-analysis of the within-participant ambient-personal PM_{2.5} correlation suggest that greater scrutiny of the effects of exposure measurement error is warranted. Further inquiry should involve quantifying the impact

of using ambient or residential-outdoor $PM_{2.5}$ concentrations as proxies for personal $PM_{2.5}$ exposure as well as the development of methodologies to apply such findings. A comprehensive understanding of the degree to which these proxies influence $PM_{2.5}$ -disease associations is especially important in air pollution epidemiology as the health effects of $PM_{2.5}$ exposure may be subtle. Such subclinical effects are particularly difficult to detect in the presence of measurement error because sensitivity of detection varies inversely with the degree of misclassification (Rom and Markowitz 2006).

REFERENCES

Armstrong BG. 1998. Effect of measurement error on epidemiological studies of environmental and occupational exposures. *Occup Environ Med* 55(10):651-656.

Armstrong BK, White E, Saracci R. 2003. *Principles of Exposure Measurement in Epidemiology*. Oxford: Oxford University Press.

Avery CL, Mills KT, Williams R, McGraw K, Poole C, Smith RL, et al. 2009 (in press). Estimating error in using ambient PM_{2.5} concentrations as proxies for personal exposures: The Environmental Epidemiology Arrhythmogenesis in the Women's Health Initiative. *Epidemiology*.

Begg CB, Berlin JA. 1989. Publication bias and dissemination of clinical research. *J Natl Cancer Inst* 81(2):107-115.

Begg CB, Mazumdar M. 1994. Operating characteristics of a rank correlation test for publication bias. *Biometrics* 50(4):1088-1101.

Berkey CS, Hoaglin DC, Mosteller F, Colditz GA. 1995. A random-effects regression model for meta-analysis. *Stat Med* 14(4):395-411.

Brown KW, Sarnat JA, Suh H, Coull BA, Spengler JD, Koutrakis P. 2008. Ambient site, home outdoor and home indoor particulate concentrations as proxies of personal exposure. *Journal of Environmental Monitoring* 10:1041-1051.

Burnett RT, Dales R, Krewski D, Vincent R, Dann T, Brook JR. 1995. Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory diseases. *Am J Epidemiol* 142(1):15-22.

Burnett RT, Brook J, Dann T, Delocla C, Philips O, Cakmak S, et al. 2000. Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. *Inhal Toxicol* 12 Suppl 4:15-39.

Cochran WG. 1954. The combination of estimates from different experiments. *Biometrics* 73:526-530.

Diez Roux AV, Auchincloss AH, Franklin TG, Raghunathan T, Barr RG, Kaufman J, et al. 2008. Long-term exposure to ambient particulate matter and prevalence of subclinical atherosclerosis in the Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol* 167(6):667-675.

Dominici F, McDermott A, Daniels M, Zeger SL, Samet J. 2003. Revised analyses of the National Morbidity, Mortality, and Air Pollution Study, Part II. Mortality among residents of 90 cities. In *Health Effects Institute. 2003. Revised analyses of time-series studies of air pollution and health*. Special report. Boston:Health Effects Institute.

Duval S, Tweedie R. 2000. Trim and fill:A simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics* 56(2):455-463.

Ebelt ST, Petkau AJ, Vedal S, Fisher TV, Brauer M. 2000. Exposure of chronic obstructive pulmonary disease patients to particulate matter:relationships between personal and ambient air concentrations. *J Air Waste Manag Assoc* 50(7):1081-1094.

Ebelt ST, Wilson WE, Brauer M. 2005. Exposure to ambient and nonambient components of particulate matter:a comparison of health effects. *Epidemiology* 16(3):396-405.

Egger M, Davey Smith G, Schneider M, Minder C. 1997. Bias in meta-analysis detected by a simple, graphical test. *BMJ* 315(7109):629-634.

Field AP. 2001. Meta-analysis of correlation coefficients:a Monte Carlo comparison of fixed- and random-effects methods. *Psychol Methods* 6(2):161-180.

Fisher RA. 1925. Statistical methods for research workers. Edinburgh:Oliver & Boyd.

Galbraith RF. 1988. A note on graphical presentation of estimated odds ratios from several clinical trials. *Stat Med* 7(8):889-894.

GEOnet Names Server. 2009. Available:<http://earth-info.nga.mil/gns/html/whatsnew.htm#C3> [accessed Nov. 18, 2009]

Greenland S, Robins JM. 1985. Confounding and misclassification. *Am J Epidemiol* 122(3):495-506.

Hill AB. 1965. The environment and disease:association or causation? *Proceedings of the Royal Society of Medicine* 58:295-300.

Janssen NA, Hoek G, Brunekreef B, Harssema H, Mensink I, Zuidhof A. 1998. Personal sampling of particles in adults:relation among personal, indoor, and outdoor air concentrations. *Am J Epidemiol* 147(6):537-547.

Katsouyanni K, Touloumi G, Samoli E, Petasakis Y, Analitis A, Le Tertre A, et al. 2003. Sensitivity analysis of various models of short-term effects of ambient particles on total mortality

in 29 cities in APHEA2. In *Health Effects Institute. 2003. Revised analysis of time-series studies of air pollution and health*. Special report. Boston:Health Effects Institute.

Klepeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, Switzer P, et al. 2001. The National Human Activity Pattern Survey (NHAPS):a resource for assessing exposure to environmental pollutants. *J Expo Anal Environ Epidemiol* 11(3):231-252.

Liao D, Whitsel EA, Duan Y, Lin HM, Quibrera PM, Smith R, et al. 2009. Ambient particulate air pollution and ectopy--the environmental epidemiology of arrhythmogenesis in Women's Health Initiative Study, 1999-2004. *J Toxicol Environ Health A* 72(1):30-38.

Linn WS, Szlachcic Y, Gong H, Jr., Kinney PL, Berhane KT. 2000. Air pollution and daily hospital admissions in metropolitan Los Angeles. *Environ Health Perspect* 108(5):427-434.

Liu LJ, Box M, Kalman D, Kaufman J, Koenig J, Larson T, et al. 2003. Exposure assessment of particulate matter for susceptible populations in Seattle. *Environ Health Perspect* 111(7):909-918.

Long CM, Suh HH, Kobzik L, Catalano PJ, Ning YY, Koutrakis P. 2001. A pilot investigation of the relative toxicity of indoor and outdoor fine particles:in vitro effects of endotoxin and other particulate properties. *Environ Health Perspect* 109(10):1019-1026.

Monn C, Becker S. 1999. Cytotoxicity and induction of proinflammatory cytokines from human monocytes exposed to fine (PM_{2.5}) and coarse particles (PM_{10-2.5}) in outdoor and indoor air. *Toxicol Appl Pharmacol* 155(3):245-252.

National Climactic Data Center. 2009.

Available:<http://www.ncdc.noaa.gov/oa/climate/research.html> [accessed Nov. 18, 2009]

Oftedal B, Nafstad P, Magnus P, Bjorkly S, Skrondal A. 2003. Traffic related air pollution and acute hospital admission for respiratory diseases in Drammen, Norway 1995-2000. *Eur J Epidemiol* 18(7):671-675.

Pinto JP, Lefohn AS, Shadwick DS. 2004. Spatial variability of PM_{2.5} in urban areas in the United States. *J Air Waste Manag Assoc* 54(4):440-449.

Poole C. 2001. Low P-values or narrow confidence intervals: which are more durable? *Epidemiology* 12(3):291-294.

Reid CM. 2003. Assessment of Exposure to Selected Criteria Pollutants for Two Sensitive Population Cohorts in Atlanta, Georgia. Atlanta: Emory University.

Rodes CE, Lawless PA, Evans GF, Sheldon LS, Williams RW, Vette AF, et al. 2001. The relationships between personal PM exposures for elderly populations and indoor and outdoor concentrations for three retirement center scenarios. *J Expo Anal Environ Epidemiol* 11(2):103-115.

Rojas-Bracho L, Suh HH, Koutrakis P. 2000. Relationships among personal, indoor, and outdoor fine and coarse particle concentrations for individuals with COPD. *J Expo Anal Environ Epidemiol* 10(3):294-306.

Rom WN, Markowitz SB. 2006. Environmental and Occupational Medicine. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins.

Samet J, Krewski D. 2007. Health effects associated with exposure to ambient air pollution. *J Toxicol Environ Health A* 70(3-4):227-242.

Sarnat SE, Coull BA, Schwartz J, Gold DR, Suh HH. 2006. Factors affecting the association between ambient concentrations and personal exposures to particles and gases. *Environ Health Perspect* 114(5):649-654.

Suh H, Koutrakis P, Chang L. 2003. Characterization of the composition of personal, indoor, and outdoor particulate exposures, Harvard School of Public Health, Environmental Science and Engineering Program, Boston, MA. Report to the California Air Resources Board. Contract No. 98-330.

United States Environmental Protection Agency. 1996. Air Quality Criteria for Particulate Matter; Report Nos. EPA/600/P-95/001aF-cF.3v; National Center for Environmental Assessment - RTP Office. Research Triangle Park, NC:U.S. Environmental Protection Agency.

United States Environmental Protection Agency. 2009. Integrated Science Assessment for Particulate Matter (Second External Review Draft). EPA/600/R-08/139-B. Washington, DC:U.S. EPA.

Wainman T, Zhang J, Weschler CJ, Liou PJ. 2000. Ozone and limonene in indoor air:a source of submicron particle exposure. *Environ Health Perspect* 108(12):1139-1145.

Wallace L. 1996. Indoor particles:a review. *J Air Waste Manag Assoc* 46(2):98-126.

Wallace L. 2000. Correlations of personal exposure to particles with outdoor air measurements:a review of recent studies. *Aerosol Science and Technology* 32:15-25.

Whitsel EA, Quibrera PM, Christ SL, Liao D, Prineas RJ, Anderson GL, et al. 2009. Heart Rate Variability, Ambient Particulate Matter Air Pollution, and Glucose Homeostasis: The Environmental Epidemiology of Arrhythmogenesis in the Women's Health Initiative. *Am J Epidemiol* 169(6):693-703.

Williams R, Creason J, Zweidinger R, Watts R, Sheldon L, Shy C. 2000a. Indoor, outdoor, and personal exposure monitoring of particulate air pollution: the Baltimore elderly epidemiology-exposure pilot study. *Atmospheric Environment* 34:4193-4204.

Williams R, Suggs J, Creason J, Rodes C, Lawless P, Kwok R, et al. 2000b. The 1998 Baltimore Particulate Matter Epidemiology-Exposure Study: part 2. Personal exposure assessment associated with an elderly study population. *J Expo Anal Environ Epidemiol* 10(6 Pt 1):533-543.

Williams R, Suggs J, Rea A, Sheldon L, Rodes C, Thornburg J. 2003a. The Research Triangle Park particulate matter panel study: modeling ambient source contribution to personal and residential PM mass concentrations. *Atmospheric Environment* 37:5365-5378.

Williams R, Suggs J, Rea A, Leovic K, Vette A, Croghan C, et al. 2003b. The Research Triangle Park particulate matter panel study: PM mass concentration relationships. *Atmospheric Environment* 37(38):5349-5363.

Zhu YF, Hinds WC, Kim S, Sioutas C. 2002. Concentration and size distribution of ultrafine particles near a major highway. *J Air Waste Manag Assoc* 52(9):1032-1042.

TABLE 1. Characteristics of nine US studies examining the within-participant, residential outdoor-personal PM_{2.5} correlation.

| Study ^a | Sub-study | Setting | | Study Dates | | Duration (months) | PM _{2.5} Measures | | |
|---------------------------|-----------|-------------|----------------|-------------|------------|-------------------|----------------------------|-------|----------|
| | | City | State | Start | End | | Timing | Pairs | <i>r</i> |
| Wallace 1996 | | Azusa | California | 03/06/1989 | 03/13/1989 | 0.2 | N | 7 | P |
| Rojas-Bracho et al. 2000 | | Boston | Massachusetts | 02/05/1996 | 02/02/1997 | 11.7 | C | 13 | P |
| Williams et al. 2000 | | Towson | Maryland | 07/26/1998 | 08/23/1998 | 0.9 | C | 16 | P |
| Rodes et al. 2001 | 1 | Fresno | California | 02/01/1999 | 02/28/1999 | 0.9 | C | 8 | P |
| | 2 | | | 04/19/1999 | 05/16/1999 | 0.9 | N | 7 | P |
| Suh et al. 2003 | 1 | Los Angeles | California | 06/12/2000 | 07/24/2000 | 1.4 | C | 6 | S |
| | 2 | | | 02/11/2000 | 03/22/2000 | 1.3 | C | 6 | S |
| Liu et al. 2003 | 1 | Seattle | Washington | 10/26/1999 | 08/10/2000 | 9.3 | C | 7 | P |
| | 2 | | | 10/26/1999 | 10/26/2000 | 11.8 | C | 7 | P |
| | 3 | | | 02/07/2000 | 05/24/2001 | 15.2 | C | 7 | P |
| | 4 | | | 11/27/2000 | 02/24/2001 | 2.9 | C | 7 | P |
| Reid, 2003 | 1 | Atlanta | Georgia | 09/21/1999 | 11/23/1999 | 2.0 | C | 6 | S |
| | 2 | | | 04/01/2000 | 05/13/2000 | 1.4 | C | 6 | S |
| Williams et al. 2003b | | Raleigh | North Carolina | 06/09/2000 | 05/21/2001 | 11.2 | N | 20 | P |
| Brown et al. 2008 | 1 | Boston | Massachusetts | 11/15/1999 | 01/29/2000 | 2.4 | C | 6 | S |
| | 2 | | | 06/06/2000 | 07/25/2000 | 1.6 | C | 5 | S |
| Nine studies, 1996 – 2008 | 16 | 8 | 6 | 1989 - 2001 | | 1.9 | 70% C | 7 | 63% P |

^aSummary statistics reported as counts, range, proportion, or median. C= consecutive. N = non-consecutive. Pairs = average number of outdoor-personal paired measurements for estimation of within-participant correlations. P = Pearson product-moment correlation coefficient. PM_{2.5} = particulate matter < 2.5 μ m in diameter (μ g/m³). *r* = within-participant residential outdoor-personal PM_{2.5} correlation estimation method. S = Spearman's rank correlation coefficient.

TABLE 2. Characteristics of participants in nine studies examining the within-participant residential outdoor-personal PM_{2.5} correlation.

| Study ^a | Sub-study | N | Participant Age | | | Percent Female | Comorbidity ^d |
|---------------------------|-----------|-----|-----------------|--------------|--------------|----------------|--------------------------|
| | | | Mean | Min | Max | | |
| Wallace 1996 | | 10 | 34.1 | 11 | 52 | 30 | N |
| Rojas-Bracho et al. 2000 | | 17 | ^b | ^b | ^b | ^b | P |
| Williams et al. 2000 | | 19 | 81 | 72 | 93 | 81 | N, C, P |
| Rodes et al. 2001 | 1 | 5 | 85 | 55 | ^b | 68 | N |
| | 2 | 14 | 85 | 55 | ^b | 68 | N |
| Suh et al. 2003 | 1 | 14 | 68.1 | 55 | 84 | 87 | P |
| | 2 | 13 | 70 | 60 | 84 | 93 | P |
| Liu et al. 2003 | 1 | 30 | 76.3 | 66 | 88 | 61 | N |
| | 2 | 48 | 77.3 | 65 | 89 | 55 | P |
| | 3 | 33 | 76.6 | 57 | 86 | 35 | C |
| | 4 | 22 | 9 | 6 | 13 | 24 | P |
| Reid 2003 | 1 | 23 | 64 | 33 | 88 | 33 | C, P |
| | 2 | 22 | 63 | 33 | 84 | 50 | C, P |
| Williams et al. 2003b | | 36 | 70 | 55 | 85 | 74 | C |
| Brown et al. 2008 | 1 | 12 | ^c | 40 | ^c | 20 | C, P |
| | 2 | 11 | ^c | 40 | ^c | 27 | C, P |
| Nine studies, 1996 - 2008 | 16 | 329 | 70 | 6 | 93 | 55% | 25% N |

^aSummary statistics reported as counts, range, proportion, or median; ^bRequested, but not provided as of 11/18/2009;

^cNot collected; ^dNo (N), chronic pulmonary (P), or chronic cardiovascular (C) disease

TABLE 3. Environmental characteristics for nine studies examining the within-participant correlation between residential outdoor and personal PM_{2.5}.

| Study ^a | Sub-study | Residential outdoor PM _{2.5} (μg/m ³) | | Personal PM _{2.5} (μg/m ³) | | <i>r</i> | | Meteorological data, mean over study dates | | | |
|---------------------------|-----------|--|------|---|------|-----------|------|--|---------|-----------|--------|
| | | Mean | SD | Mean | SD | \bar{r} | SD | T (°C) | DP (°C) | SLP (kPa) | RH (%) |
| Wallace 1996 | | 42.6 | NR | 70 | NR | 0.41 | 0.16 | 11.7 | 52.0 | 101.81 | 27.3 |
| Rojas-Bracho et al. 2000 | | 14.2 | 11.2 | 21.6 | 13.6 | 0.64 | 0.11 | 13.2 | 45.4 | 101.56 | 68.0 |
| Williams et al. 2000 | | 22.0 | 12.0 | 13.0 | 3.2 | 0.79 | 0.08 | 24.0 | 64.0 | 101.85 | 68.3 |
| Rodes et al. 2000 | 1 | 20.5 | 13.4 | 13.1 | 5.9 | 0.58 | 0.18 | 9.6 | 41.8 | 102.27 | 75.2 |
| | 2 | 10.1 | 3.2 | 11.1 | 2.8 | 0.65 | 0.20 | 17.5 | 41.2 | 101.42 | 43.9 |
| Suh et al. 2003 | 1 | 19.3 | 9.0 | 25.1 | 20.8 | 0.32 | 0.14 | 21.1 | 60.3 | 101.34 | 71.3 |
| | 2 | 13.5 | 8.5 | 19.6 | 14.5 | 0.59 | 0.16 | 13.7 | 46.8 | 101.70 | 69.7 |
| Liu et al. 2003 | 1 | 9.0 | 4.6 | 9.3 | 8.4 | 0.47 | 0.10 | 9.9 | 43.6 | 101.78 | 78.9 |
| | 2 | 9.2 | 5.1 | 10.5 | 7.2 | 0.51 | 0.09 | 10.8 | 44.8 | 101.78 | 77.8 |
| | 3 | 12.6 | 7.9 | 10.8 | 8.4 | 0.55 | 0.13 | 10.0 | 42.8 | 101.82 | 76.0 |
| | 4 | 11.3 | 6.4 | 13.3 | 8.2 | 0.41 | 0.11 | 6.9 | 37.8 | 101.90 | 77.1 |
| Reid 2003 | 1 | 14.5 | 7.3 | 16.3 | 8.4 | 0.76 | 0.18 | 15.7 | 49.7 | 102.01 | 68.3 |
| | 2 | 22.7 | 10.6 | 15.0 | 7.5 | 0.48 | 0.12 | 17.2 | 49.8 | 101.64 | 62.0 |
| Williams et al. 2003b | | 19.3 | 8.43 | 23.0 | 16.1 | 0.35 | 0.04 | 17.2 | 51.9 | 101.92 | 67.4 |
| Brown et al. 2008 | 1 | 8.6 | 5.2 | 12.0 | 6.0 | 0.25 | 0.22 | 2.0 | 22.7 | 101.67 | 59.0 |
| | 2 | 12.5 | 7.6 | 10.0 | 6.2 | 0.75 | 0.35 | 20.4 | 58.6 | 101.43 | 70.3 |
| Nine studies, 1996 – 2008 | 16 | 13.9 | 7.9 | 13.2 | 8.2 | 0.53 | 0.14 | 13.4 | 46.1 | 101.78 | 69.0 |

^aSummary statistics reported as counts or median. DP = dew point. \bar{r} = mean within-participant residential outdoor PM_{2.5}-personal PM_{2.5} correlation coefficient. Pairs = average number of outdoor-personal paired measurements for estimation of within-participant correlations. RH = relative humidity. SD = standard deviation. SLP = sea level pressure. T = temperature.

Figure 1. Forest plot for sixteen estimates of \bar{r} (95% confidence intervals) from nine studies of the within-participant, residential outdoor-personal PM_{2.5} correlation.

Figure 2. Funnel plot for sixteen estimates of the within-participant, residential outdoor-personal PM_{2.5} correlation.

Figure 3. Galbraith plot with 95% confidence limits for sixteen estimates of the within-participant, residential outdoor-personal PM_{2.5} correlation.

Figure 4. Summary correlations (95% confidence intervals) and correlation differences (95% CI) by study, participant, and environment characteristics for nine studies examining the within-participant, residential outdoor-personal PM_{2.5} correlation.

Figure 5. Plot for sixteen estimates of the within-participant, residential outdoor-personal PM_{2.5} correlation (95% confidence interval) versus mean outdoor temperature, including the random-effects meta-regression line.