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Estimating personal exposures from ambient air-pollution measures: Using meta-analysis to assess measurement error

Katelyn M Holliday[1], **Christy L Avery**[1], **Charles Poole**[1], **Kathleen McGraw**[2], **Ronald Williams**[3], **Duanping Liao**[4], **Richard L Smith**[5,6], and **Eric A Whitsel**[1,7]

[1]Department of Epidemiology, University of North Carolina, Chapel Hill, North Carolina, USA

[2]Health Sciences Library, University of North Carolina, Chapel Hill, North Carolina, USA

[3]United States Environmental Protection Agency, Research Triangle Park, Durham, North Carolina, USA

[4]Department of Public Health Sciences, Pennsylvania State University, Hershey, Pennsylvania, USA

[5]Statistical and Mathematical Sciences Institute, Research Triangle Park, Durham, North Carolina, USA

[6]Department of Statistics and Operations Research, University of North Carolina, Chapel Hill, North Carolina, USA

[7]Department of Medicine, University of North Carolina, Chapel Hill, North Carolina, USA

Abstract

Background—Although ambient concentrations of particulate matter ≤10μm (PM_{10}) are often used as proxies for total personal exposure, correlation (*r*) between ambient and personal PM_{10} concentrations varies. Factors underlying this variation and its effect on health outcome-PM exposure relationships remain poorly understood.

Methods—We conducted a random-effects meta-analysis to estimate effects of study, participant and environmental factors on *r*; used the estimates to impute personal exposure from ambient PM_{10} concentrations among 4,012 non-smoking, diabetic participants in the Women's Health Initiative clinical trial; and then estimated the associations of ambient and imputed personal PM_{10} concentrations with electrocardiographic measures such as heart rate variability.

Results—We identified fifteen studies (in years 1990-2009) of 342 participants in five countries. The median *r* was 0.46 (range = 0.13 to 0.72). There was little evidence of funnel-plot asymmetry but substantial heterogeneity of *r*, which increased 0.05 (95% confidence interval [CI]= 0.01 to 0.09) per 10 $\mu g/m^3$ increase in mean ambient PM_{10} concentration. Substituting imputed personal exposure for ambient PM_{10} concentrations shifted mean percent changes in electrocardiographic measures per 10$\mu g/m^3$ increase in exposure away from the null and decreased their precision, e.g. −2.0% (95% CI= −4.6% to 0.7%) versus −7.9% (−15.9% to 0.9%) for the standard deviation of normal-to-normal RR interval duration.

Address for Correspondence Katelyn M. Holliday UNC Gillings School of Global Public Health Department of Epidemiology Cardiovascular Disease Program Bank of America, Suite 306 137 E Franklin St Chapel Hill, NC 27514 (T) 812-593-3170 (F) 919-966-9800 khausman@email.unc.edu.

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Conclusions—Analogous distributions and heterogeneity of *r* in extant meta-analyses of ambient and personal $PM_{2.5}$ concentrations suggest that observed shifts in mean percent change and decreases in precision may be generalizable across particle size.

Particulate matter (PM) exposure is associated with numerous adverse health outcomes, particularly those involving the cardiovascular and respiratory systems.[1] Although these health effects may be strongest for small particles,[2] many studies have found that large particles have independent, adverse effects on health.[3] This fact, combined with global interest in PM_{10}, suggests that focus on larger-size fractions is still merited when examining PM-disease associations. In studies of these associations, researchers have quantified PM exposure using ambient, micro-environmental, or personal sampling. Although personal concentrations may represent the most accurate assessment of total exposure, it is ambient concentrations that are federally regulated by the Environmental Protection Agency under the Clean Air Act.[4] Moreover, using ambient data is often less costly for sponsors, less burdensome for participants, and sometimes the only feasible method of retrospectively characterizing PM exposure in longitudinal cohort studies. As a result, many epidemiologic studies rely on ambient concentrations of PM, which are associated with varying degrees of measurement error.

The characteristics and determinants of such exposure measurement error have been largely unknown or ignored in analyses of PM-health outcome associations, although the body of literature on this topic is growing.[5-11] Further, researchers can examine the potential effects of measurement error if the relationship between ambient and personal exposures can be quantified. Fortunately, many studies have uniformly reported the correlation (*r*) of ambient and personal PM_{10} concentrations in a variety of geographic locations, often with an emphasis on vulnerable populations.[12-27] However, these studies have not been systematically assessed. We therefore reviewed the literature examining the longitudinal, within-person, ambient-personal PM_{10} concentration correlation to identify and characterize factors influencing the observed distribution and heterogeneity of *r*. We illustrate how results from such a review can be used to impute personal PM concentrations from ambient concentrations, and we clarify effects of exposure measurement error on the relation of PM exposure to health outcomes in epidemiologic studies.

METHODS

Search and data abstraction strategy

We searched seven electronic databases using the strategy described in eAppendix 1. We downloaded articles to Endnote (EndNote X1; Thomson Reuters, New York, NY), de-duplicated, and examined the list for potential omissions. We reviewed each article, excluding those without PM_{10} concentrations measured in both ambient (central site or outside participant home) and personal environments, and those lacking an ambient-personal Pearson or Spearman correlation coefficient (*r*) or at least four paired ambient-personal concentrations. We then abstracted the following data: individual participant *r* (study mean or median if individual participant unavailable) or paired ambient-personal concentrations; number of paired concentrations; and selected characteristics of the study, participants, and environment (eTables 1-3). Article review, exclusion, and abstraction were conducted in duplicate by two authors who resolved discrepancies by consensus. We requested additional data from primary authors as needed. Coordinates were assigned to cities in which studies were conducted using the United States Geological Survey Geographic Names Information System.[28] We then linked additional weather variables (eTable 3) from the National Climatic Data Center[29] to the coordinates by downloading data from the three nearest monitors and calculating inverse distance-weighted means across study dates.

Meta-analysis statistical procedures

When possible, we calculated a study-level mean *r* (r_j) that weighted each participant's contribution by the number of that participant's paired ambient-personal PM_{10} concentrations. To do this, we "*r*-to-z transformed" participant-level measures of *r*[30] and then calculated a study-level mean z ($\overline{z}_r$) using the Hedges-Olkin and Rosenthal-Rubin method under a random-effects model.[10,11,31] In this method, $\overline{z}_r = \left[\Sigma_{i=1}^{k}(w_i)(z_{r_i})\right] / \left[\Sigma_{i=1}^{k} w_i\right]$, where k denotes the number of participants in the study *i* identifies the participant z_{r_i} the participant's *r*-to-z transformed correlation coefficient, and w_i the corresponding weight.[31] The weight is composed of within- and between-participant variances: $1 / (n_i - 3)$ and τ^2. It is calculated as $[(1 / (n_i - 3)) + \tau^2]^{-1}$, where n_i is the participant-level number of paired ambient-personal PM_{10} concentrations, $\tau^2 = [Q - (k - 1)] / c$, $Q = \Sigma_{i=1}^{k}(n_i - 3)(z_{r_i} - \overline{z}_r)^2$, and $c = \Sigma_{i=1}^{k}(n_i - 3)\left[\Sigma_{i=1}^{k}(n_i - 3)^2\right] / \left[\Sigma_{i=1}^{k}(n_i - 3)\right]$.[31] Negative values of τ^2 were set to zero.[31] When participant-level data were unavailable, $\overline{z}_r$ was calculated under a fixed-effects model as follows: $\overline{z}_r = \left[\Sigma_{i=1}^{k}(w_i)(z_r)\right] / \left[\Sigma_{i=1}^{k} w_i\right]$, where z_r is the study-level median *r*-to-z transformed correlation coefficient, w_i is (*n* - 3), and *n* is the study-level mean number of paired ambient-personal PM_{10} concentrations per participant.[31] The standard errors of the study-level random-effects and fixed-effects $\overline{z}_r$ were calculated as $\mathrm{SE}(\overline{z}_r) = \left(1/\Sigma_{i=1}^{k} w_i\right)^{1/2}$.[31] Funnel-plot asymmetry was examined by plotting the study-level $\overline{z}_r$ versus its weight $(w_j) = 1/\mathrm{SE}(\overline{z}_r)^2\big)$, computing Begg and Egger test statistics[32,33] and completing a trim-and-fill analysis.[34] We evaluated homogeneity of *r* using Cochran's Q[35] and explored potential sources of heterogeneity by first assembling study, participant, and environmental characteristics with putative effects on *r*, then dichotomizing interval-scale characteristics at their medians, and computing summary random-effects correlation coefficients within strata defined by the characteristics. We also conducted univariate, random-effects meta-regressions to examine differences in *r* among strata, estimated changes in *r* per one-unit increase in interval-scale measures,[36] and examined their sensitivity to exclusion of outlying observations identified using an extreme studentized deviate multiple-outlier procedure.[37] Potential sources of heterogeneity identified in univariate random-effects meta-regressions were dichotomized at their median values (for continuous variables) and included in bivariable random-effects meta-regressions when cross-classification cell size was ≥ 2 to examine the possibility that one variable might explain all or part of the relationship observed between *r* and the other variables.

Imputation of Personal PM_{10} Concentration

We used the results of the meta-analysis to impute personal PM_{10} concentrations from ambient concentrations. Imputation was performed among 4,012 non-smoking, diabetic women who participated in the Women's Health Initiative clinical trial. Women had to be residing in the contiguous U.S. at the time of their first resting, standard, twelve-lead electrocardiogram (ECG), for which measures of RR, PR, QRS, and QT interval durations, as well as the root mean square of successive differences in and the standard deviation of normal-to-normal RR interval duration, were available.[38-40] Collectively, the ECG measures reflect the rate of atrioventricular conduction, rate of ventricular depolarization / repolarization, and variation in heart rate.[41] Each has been recommended as a candidate outcome in studies of air-pollution health effects under a mechanistic hypothesis postulating that the cardiovascular effects of air pollution depend in part on autonomic and myocardial pathophysiology.[42]

Imputation was completed in two steps. In step 1, we estimated participant-specific correlations between ambient and personal PM concentrations using the random-effects meta-regression equation, $r = \beta_0 + \beta_1 x$ (Figure 3A, solid line), where β_0 is the intercept and x is the participant-specific ambient PM_{10} concentration, a plausible, consistently identified, and important source of between-study heterogeneity in r.[10,11] In this setting, ambient PM_{10} concentrations were the geocoded address-specific daily means[43,44] averaged over the day of and two days before ($lag_{0\text{-}2}$) the ECG recording.

In step 2, we assumed that the distributions of the ambient and personal PM concentrations are bivariate normal and estimated the participant-specific mean personal PM_{10} concentration (p) at a given ambient PM_{10} concentration (x) using the equation, $\mu_{p|x} = \bar{y} + r\frac{s_y}{s_x}\left(x_i - \bar{x}\right)$, where for each participant i, r is estimated as in Step 1; $\bar{x}$ (s_x) is the mean (standard deviatio ambient PM_{10} concentration among the Women's Health Initiative participants; and $\bar{y}$ (s_y) is the mean (standard deviation) personal PM_{10} concentration estimated from the distributions of the personal concentrations observed in the studies contributing to the meta-analysis. The variance of $\mu_{p/x}$ was calculated as $\sigma^2_{y/x} = \left(1 - r^2\right) s^2_y$.

Bias Analysis

The authors assessed effects of exposure measurement error by (i) iterating participant-specific estimation of $\mu_{p/x}$ as in Step 2 using y and s_y from each of the d studies contributing both pieces of information to the meta-analysis, (ii) computing the random-effects weighted mean and variance of the d estimates of $\mu_{p/x}$ for each participant, (iii) regressing each of the ECG measures on the weighted mean $\mu_{p/x}$, and then (iv) comparing the estimated associations with conventional estimates obtained by regressing the same ECG measures on x_i. In (iii), error-in-variables regression models were implemented in SAS® Proc Calis (SAS; Cary, NC) to accommodate the random-effects weighted variance of the weighted mean $\mu_{p/x}$, averaged across all participants. A covariable adjustment strategy similar to that in Whitsel et al.[45] was adopted in both (iii-iv). This strategy involved adjusting for the previously described sociodemographic, geographic, temporal, clinical, behavioral, and environmental variables footnoted in the Table.

RESULTS

The electronic search strategy identified 698 articles, of which 14 (2.0%) met inclusion criteria. We identified an additional unpublished thesis, yielding a total of 15 studies. In addition, three studies provided results for sub-studies, totaling 21 for analysis. The studies were conducted over 20 years (1988-2007) and encompassed a large geographic area including 19 cities, 8 U.S. states, and 5 countries. The studies included 342 participants (median: 14 per sub-study) who were assessed over widely varying durations (0.3 to 21.0 months); however, samples were collected for 24-hour periods in 19 (90%) sub-studies (eTable 1).

The mean participant age ranged from 9 to 85 years and several sub-studies focused on populations with conditions commonly associated with increased susceptibility to PM health effects: chronic obstructive pulmonary disease (COPD, 24%), asthma (19%), and coronary artery disease (19%) (eTable 2).

As several studies spanned multiple seasons, mean weather variables should be viewed cautiously; however, the ranges of mean temperature (−4° to 30°C) and wind speed (1 to 7 m/s) were large. The ranges of mean personal and ambient PM_{10} concentrations also were

large among studies: 12 to 115 μg/m^3 and 14 to 131 μg/m^3, respectively. Despite these ranges, personal concentrations were typically greater than ambient concentrations (eTable 3), and only one value was identified as an outlier: the mean ambient PM_{10} concentration in the paper by Watchalayann et al.[23]

The median of r_j was 0.46 (range = 0.13 to 0.72) (Figure 1; eTable 3), with no outlying *r* values. Although the funnel plot symmetry test P-values were high (P_{Egger}=0.6, P_{Begg}=0.9), the visual impression of the plot suggested asymmetry and the trim-and-fill analysis imputed five hypothetically missing results, all with r_j near zero (Figure 2). In addition, there was substantial evidence of heterogeneity ($P_{Cochran's\ Q}$ <0.001). Consequently, an overall summary *r* was not estimated.

The magnitude and precision of stratum-specific, random-effects correlation coefficients suggested that participants without COPD or asthma, and those exposed to higher ambient PM_{10} concentrations, higher ambient-to-personal concentration ratios, and lower wind speeds, had more strongly correlated ambient and personal PM_{10} concentrations (Figure 3). Random-effects meta-regression results were consistent with these suggestions (Figures 3-4), as was the strengthened association between the ambient PM_{10} concentration and *r* after excluding an outlying ambient PM_{10} concentration (Figure 4). Although study location appeared to influence *r*, 76% of studies were located in the U.S., and *r* was similar among north-south and east-west dichotomization of coordinates (Figure 3). In addition, *r* was comparable among studies relying on PM_{10} measured at a central site versus outside home (0.54 [95% CI= 0.41 to 0.65] versus 0.47 [0.31 to 0.61]) and over the range of ambient and personal concentrations (Figure 3). Between-group differences were slightly attenuated in bivariable meta-regressions including combinations of mean ambient PM_{10} concentration, ambient-to-personal PM_{10} concentration ratio, and wind speed; however overall conclusions did not change (eTable 4).

The median ambient PM_{10} concentration measured among Women's Health Initiative participants was 25.7 (range = 7.3 to 109.6) μg/m^3. Before and after excluding the outlying results,[23] the median imputed *r* was 0.46 (range = 0.38 to 0.71) and 0.44 (0.30 to 0.84), respectively, while the corresponding median imputed personal PM_{10} concentration was 34.1 (23.8 to 135.7) μg/m^3 and 28.6 (20.6 to 152.8) μg/m^3.

The relationships between PM_{10} concentration, the root mean square of successive differences in normal-to-normal RR interval duration, and the standard deviation of normal-to-normal RR interval duration were notable in the bias analysis (Table). When the ambient PM_{10} concentration was used as the exposure, percent changes in the root mean square of successive differences in normal-to-normal RR interval duration and the standard deviation of normal-to-normal RR interval duration per 10 μg/m^3 increase were −1.5% (95% CI= −4.3 to 1.3) and −2.0% (−4.6 to 0.7), but when the imputed personal concentration was substituted for the ambient PM_{10} concentration, corresponding estimates shifted away from the null and their precision decreased: −6.7% (95% CI= −15.3 to 2.8) and −7.9% (−15.9 to 0.9). The posterior probabilities of a positive percent change also decreased, from 0.14 to 0.08 and 0.07 to 0.04, respectively. Similar changes were observed across ECG measures in sensitivity analyses excluding (1) Watchalayann et al,[23] (2) child studies[14, 19, 22], and (3) both 1 and 2. For example, percent changes in the root mean square of successive differences in normal-to-normal RR interval duration and the standard deviation of normal-to-normal RR interval duration per 10 μg/m^3 increase were −5.5% (95% CI= −12.6 to 2.2) and −6.5% (−13.1 to 0.7) when Watchalayann et al.[23] and child studies[14,19, 22] were excluded from the bias analysis.

DISCUSSION

The use of ambient PM_{10} concentrations in health association studies remains commonplace. Although potentially important sources of measurement error in this surrogate of true personal exposure have been suggested by many investigators, [5-11] no systematic review or application of results from studies examining the correlation between ambient and personal PM_{10} concentrations has been carried out to date. We therefore summarized these studies, characterized factors influencing the among-study heterogeneity of *r*, and then described an accessible framework for using quantitative information about the sources of heterogeneity to impute personal from ambient PM concentrations and clarify the effects of exposure measurement error on health outcome-PM exposure relationships.

The summary included a funnel plot suggesting that the historically high costs and burdens associated with personal PM monitoring may have resulted in more studies of *r* enrolling few participants (scattered near the bottom of the plot) and few studies enrolling many participants (near the top). The results imputed by the accompanying trim-and-fill analysis could represent those that remain unpublished for a variety of reasons, such as implausibility (correlations near or below zero) or discordance with the extant literature. Had such low correlations actually been withheld from publication, the observed among-study heterogeneity of *r* would have been even greater. Despite this possibility, the tests of funnel-plot asymmetry support the ability of the included studies to represent the literature and their suitability for meta-analysis.

Because the meta-analysis provided substantial evidence of among-study heterogeneity of *r*, presentation of an overall fixed- or random-effects summary correlation coefficient was not warranted. Instead, we characterized the potential sources of heterogeneity. As *r* changed little with the range of the study-specific ambient or personal PM_{10} concentration, its association with other variables was anticipated. That expectation was substantiated by the observed increase in the ambient-personal PM_{10} concentration correlation with increasing ambient PM_{10} concentration, increasing ambient-to-personal PM_{10} concentration ratio, and decreasing wind speed. Additionally, we observed higher correlations in participants without versus with COPD or asthma.

The observed patterns appear plausible. In areas where ambient concentrations or ambient-to-personal concentration ratios are high, ambient PM may contribute more to total personal exposure than in areas where ambient concentrations are low. Direct increases in exposure to ambient concentrations, changes in ventilation, or altered activity patterns may account for this. Wind speed also may influence the ambient-personal PM_{10} concentration correlation, as it affects the distribution of PM in the environment. Lower wind speed impedes dispersion of PM_{10} from its sources, thus allowing central site monitors to better predict an individual participant's exposure to ambient PM.[46] Persons with and without COPD (or asthma) may also have different activity patterns, such as time spent outdoors,[47] which could influence the relationship between their personal and ambient concentrations of PM.

We used bivariable meta-regression models to address the possibility that one of the aforementioned factors could explain part or all of the association of another with *r* (eTable 4). However, too few studies included participants with COPD, thereby preventing examination of this characteristic in bivariable meta-regression. Estimates of *r* did not differ substantially among the uni- and bi-variable meta-regression models, suggesting that meta-confounding of the univariable association of *r* with ambient PM_{10} concentration, ambient-to-personal PM_{10} concentration ratio, and wind speed may be less of a concern in this

context. Nevertheless, all bivariable meta-regressions should be interpreted cautiously, given sample size constraints.

The observed pattern of ambient-personal PM_{10} correlation coefficients ($r > 0$; low median; high range) is similar to those previously reported in meta-analyses of $PM_{2.5}$.[10,11] Further, the meta-analyses of both PM_{10} and $PM_{2.5}$ suggest that ambient PM concentrations are an important source of heterogeneity in r.[10,11] Although $PM_{2.5}$ concentrations comprise a large portion of PM_{10} concentrations, the extent of the similarity was unexpected. The differing distributive properties of the two size fractions[48] suggest that r would be somewhat higher for $PM_{2.5}$ than PM_{10}. While the ambient-personal PM_{10} correlation may have been driven by $PM_{2.5}$, data availability and methodological constraints limit ability of the present study to determine the extent to which this is true. Nonetheless, the similarity suggests that a variable and non-negligible degree of measurement error is incurred when using ambient PM concentrations as proxies for personal exposures in studies of PM-health associations, regardless of particle size.

The direction of PM effects on heart-rate variability in this setting is consistent with that described by a recent review of the topic. Ambient PM was inversely associated with the root mean square of successive differences in (and the standard deviation of) normal-to-normal RR interval duration, overall and among a variety of sub-groups.[49] However, the review did not address the error inherent in substituting ambient for personal exposures, which has several components.[8] In the present study, we addressed the component most likely to produce bias (the difference between average personal and true ambient exposure), because the remaining components are largely Berksonian and therefore less likely to produce bias. The results suggest that this non-Berksonian component behaves like classical exposure measurement error to the extent that it biases PM_{10} health-effect estimates toward the null when r depends on the ambient PM_{10} concentration, as in the current meta-analysis. This observation may well generalize across particle size, given the analogous dependence of r on centrally and proximally measured ambient $PM_{2.5}$ concentrations in prior meta-analyses.[10,11] As such, the true magnitude of PM effects on heart-rate variability may be larger than previously anticipated by Pieters and colleagues.[49]

Controlling for the effects of PM measurement error as described herein has some general disadvantages when compared with error correction methods such as regression calibration and hierarchical Bayesian analyses. One is its dependence on relatively small, technically complex and, in some cases, incompletely documented studies of potentially low-level exposures measured with behavior-altering personal monitors. Another is that bias and precision may vary among populations with ambient or personal PM concentrations that are unlike those observed in the Women's Health Initiative or the meta-analyses, and perhaps unpredictably so among populations that smoke. Simultaneously evaluating multiple sources of heterogeneity in the ambient-personal PM_{10} correlation within a meta-analysis of 21 studies is an additional challenge. Our frequentist methods also assume bivariate normality of ambient and personal PM concentrations, which may be unrealistic. Nevertheless, the range of ambient PM concentrations is wide in both the Women's Health Initiative [44,45, 50-52] and these meta-analyses[10,11]; four of five U.S. adults aged ≥ 18 years do not smoke[53]; and robustness to modest departure from normality is well-known. Moreover, error-in-variables regression and quantitative bias assessments are familiar to epidemiologists[54] and readily accessible to a wide variety of users. In this case, they are illustrative of the meta-analytic foundation on which more comprehensive and rigorous (e.g. hierarchical Bayesian) approaches to improving estimation of air-pollution effects could be built and applied in settings where only ambient PM concentration data are available.[55]

Such application may well benefit from the fact that we relied on a systematic review encompassing a wide variety of settings and allowing for broad examination of study, participant, and environmental effects on the ambient-personal PM_{10} correlation. By focusing on total personal PM_{10} exposure instead of personal exposure to PM_{10} of ambient origin, the data collection effort also avoided complications associated with the potentially unrealistic assumption that personal exposure is best assessed by relying on a distinctly smaller and less accessible group of microenvironmentally homogenous, single-marker (e.g. sulfate) studies. In contrast, the data that were collected, quality controlled, and tabulated in eTables 1-3, readily facilitate sensitivity analyses at the discretion of future users, an option infrequently available with regression calibration factors published in isolation.

Other powerful methods for improving estimation of ambient exposures at geocoded participant addresses have been proposed.[44, 56-61] The dual benefit of improving estimation of total personal exposure to PM—a particular interest in etiologic studies—and clarifying the downstream effects of the measurement error with which it is associated, helps distinguish the meta-analytically informed interpolation method illustrated here from those alternatives. Although total personal exposure to PM is not regulated under the Clean Air Act, the effect of aggregate PM exposure on health is of no less scientific interest. The current and previously published meta-analyses[10,11] provide the necessary data and an accessible statistical framework for estimating such exposure and conducting participant-level analysis of bias in ambient PM concentration-health association studies. In combination, the data and framework detailed here can be leveraged to increase understanding of the true, but often masked, relationships underlying such associations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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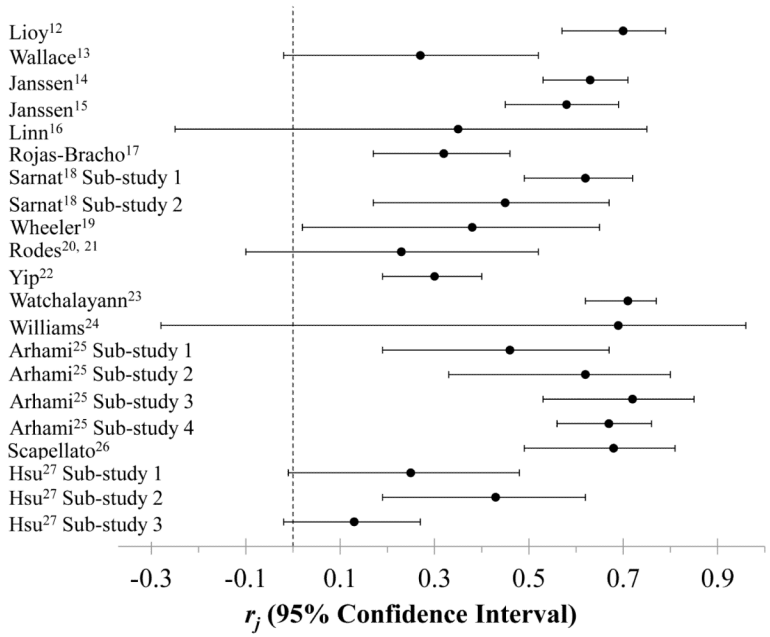

Figure 1.

Forest plot of 21 estimates of r_j (95% confidence interval) from twenty-one sub-studies of the within-participant correlation between ambient and personal PM_{10} concentration. (See eTable 3 for details).

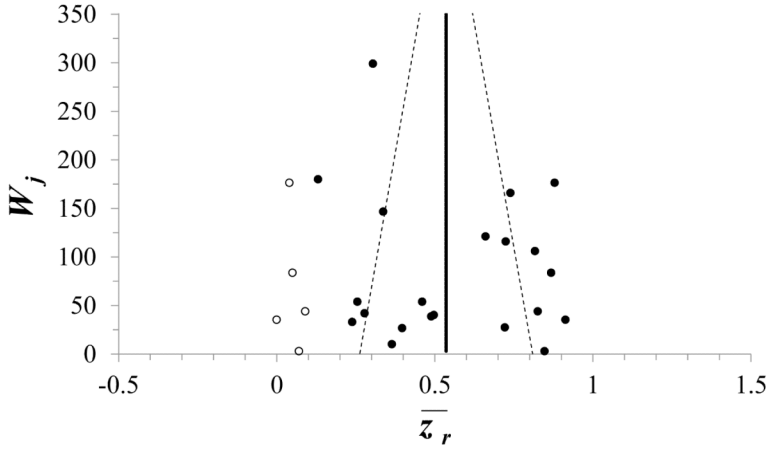

Figure 2.

Funnel plot of 21 reported (•) and five imputed (○) estimates of the z-transformed r_j from twenty-one sub-studies of the within-participant correlation coefficient between ambient and personal PM_{10} concentrations, where w_j is the inverse variance of the z-transformed r_j.

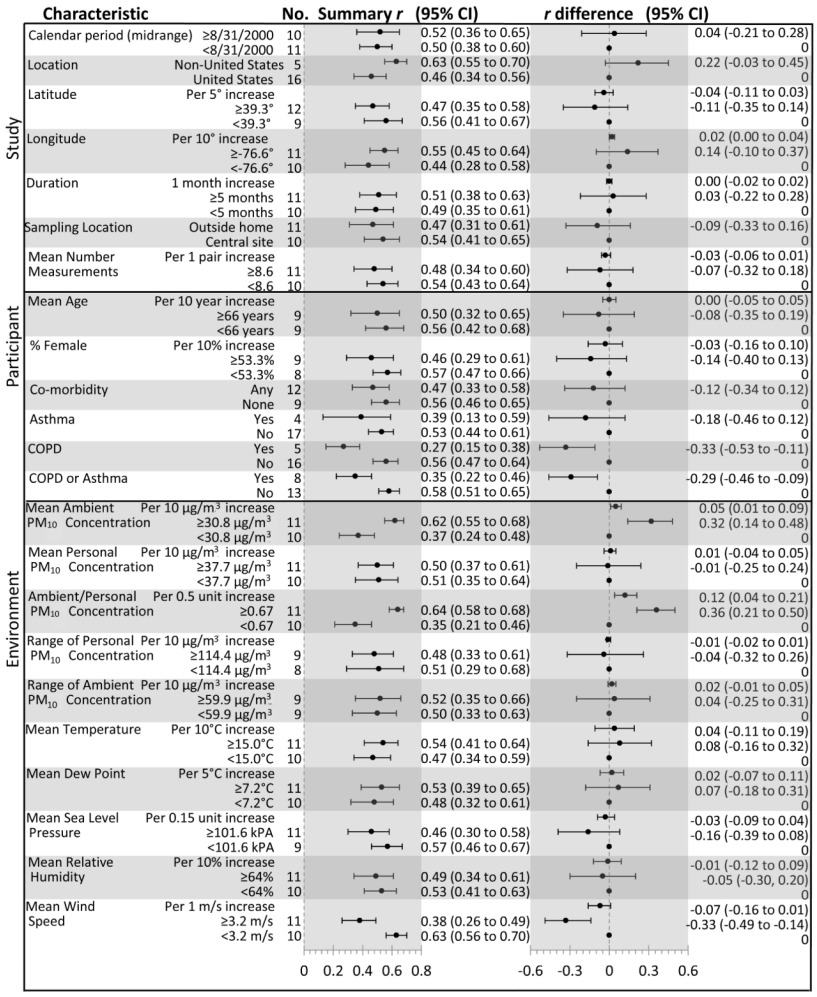

Figure 3.

Summary Random-Effects Correlation Coefficients and Meta-Regression Differences by Study, Participant, and Environment Characteristics. Summary *r* computed within strata of each characteristic. Difference in *r* from meta-regression analyses predicting *r* from the characteristics.

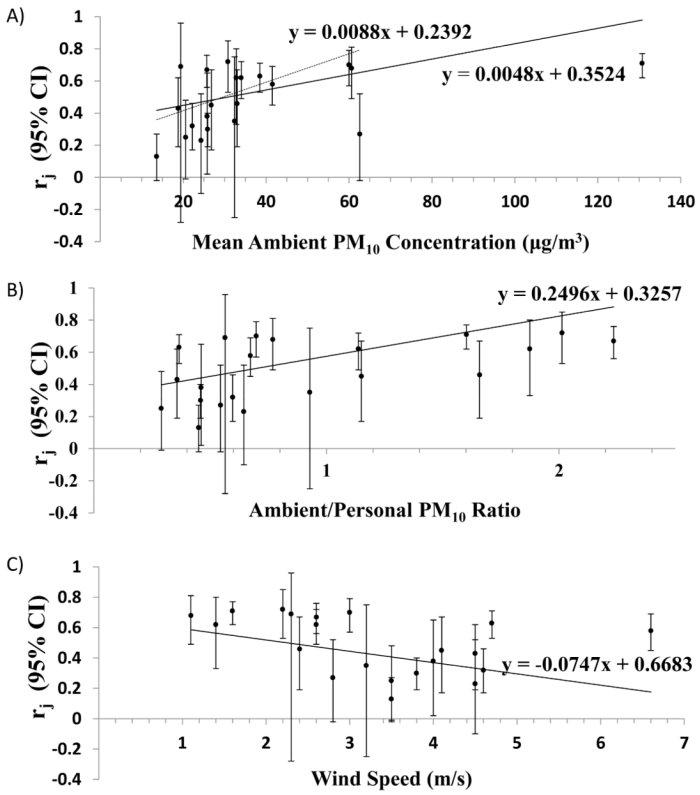

Figure 4.

Plot of 21 estimates of r_j (95% confidence interval) from twenty-one sub-studies of the within-participant correlation between ambient and personal PM_{10} concentrations versus (A) mean ambient PM_{10} concentration ($\mu g/m^3$), (B) mean ambient to personal concentration ratio, and (C) mean wind speed (m/s). Univariate random-effects regression lines (solid line). Excluding the outlying 130.7 $\mu g/m^3$ ambient PM_{10} concentration from Watchalayann et al. (dotted lines).

Table

Percent Change in Electrocardiographic Measures per 10μg/m^3 Increase in PM_{10} Concentration Among 4,012 Non-smoking, Diabetic Women's Health Initiative Clinical Trial Participants, United States, 1993-2004.

ECG Measure	Ambient PM_{10}		Imputed Personal PM_{10}	
	% (95% CI)[a]	Posterior probability of %>0	% (95% CI)[a]	Posterior probability of %>0
Root mean square of successive differences in normal-to-normal RR interval duration	−1.5 (−4.3 to 1.3)	0.14	−6.7 (−15.3 to 2.8)	0.08
Standard deviation of normal-to-normal RR interval duration	−2.0 (−4.6 to 0.7)	0.07	−7.9 (−15.9 to 0.9)	0.04
RR interval duration	−0.2 (−0.8 to 0.4)	0.25	−1.0 (−2.9 to 0.9)	0.15
PR interval duration	−0.2 (−0.7 to 0.3)	0.21	−0.5 (−2.2 to 1.3)	0.30
QRS interval duration	0.1 (−0.4 to 0.6)	0.66	0.2 (−1.5 to 1.9)	0.59
QT interval duration	0.0 (−0.3 to 0.3)	0.50	−0.2 (−1.2 to 0.8)	0.34

[a] Fully Adjusted (age, race/ethnicity, education, region, time of day (minutes), day of week, season, body mass index (kg/m^2), hypertension, systolic blood pressure (mm Hg), anti-arrhythmia medication use, total energy expenditure (kcal/kg*week), chronic lung disease, hypercholesterolemia, coronary heart disease, revascularization, congestive heart failure, lag0-1 temperature (°C), dew point (°C), and barometric pressure (kPa))