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Incorporating exposure models in probabilistic assessment of the risks of premature mortality from particulate matter

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This paper examines the link between the ambient level of particulate pollution and subsequent human health effects and various sources of uncertainty when total exposure is taken into consideration. The exposure simulation model statistically simulates daily personal total exposure to ambient PM and nonambient PM generated from indoor sources. It incorporates outdoor–indoor penetration of PM, contributions of PM from indoor sources, and time–activity patterns for target groups of the population. The model is illustrated for Los Angeles County using recent 1997 monitoring data for both PM_{10} and $PM_{2.5}$. The results indicate that, on average, outdoor-source PM contributes about 20–25% of the total PM exposure to Los Angeles County individuals not exposed to environmental tobacco smoking (ETS), and about 15% for those who are exposed to ETS. The model computes both the fractional contribution of outdoor concentrations to total exposure and the effect of exposure uncertainties on the estimated slope of the (linear) concentration–response curve in time-series studies for PM health effects. The latter considers the effects of measurement and misclassification error on PM epidemiological time-series studies. The paper compares the predictions of a conventional PM epidemiological model, based solely on ambient concentration measurements at a central monitoring station, and an exposure simulation model, which considers the quantitative relationship between central-monitoring PM concentrations and total individual exposures to particulate matter. The results show that the effects of adjusting from outdoor concentrations to personal exposures and correcting dose–response bias are nearly equal, so that roughly the same premature mortalities associated with short-term exposure to both ambient $PM_{2.5}$ and PM_{10} in Los Angeles County are predicted with both models. The uncertainty in the slope of the concentration–response curve in the time-series studies is the single most important source of uncertainty in both the ambient- and the exposure-health model.
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Introduction

Ambient particulate matter originates both from natural processes and anthropogenic emissions such as fossil fuel combustion, industrial manufacturing, and construction. Over the past three decades, epidemiologists have identified various physical and chemical mass-based measures of airborne particulate matter, for example, total suspended particles (TSP), PM_{10} (particulate matter less than 10 μm in aerodynamic diameter), $PM_{2.5}$ (particulate matter less than 2.5 μm in aerodynamic diameter), and sulfate as important aerosol measures or components to be statistically significantly associated with observed acute or chronic health effects data. These include premature mortality, increased hospital admissions and emergency room visits (primarily among the elderly and individuals with cardiopulmonary disease), increased respiratory symptoms and disease (in children and individuals with cardiopulmonary disease such as asthma), decreased lung function (particularly in children and individuals with asthma) and alterations in lung tissue, structure and respiratory tract defense mechanisms (Logan, 1953; Lave and Seskin, 1973; Dockery et al., 1989; Schwartz and Dockery, 1992; Ito and Thurston, 1996; U.S. Environmental Protection Agency [U.S. EPA], 1996a,b; Lipsett et al., 1997). The evidence provided by these and other epidemiological studies became the basis for the policy assessment of the EPA Office of Air Quality Planning and Standards (OAQPS) while reviewing the National Ambient Air Quality Standard (NAAQS) for particulate matter (U.S. EPA, 1996b). Published in 1996, the OAQPS Staff Paper performed a systematic risk analysis to assess the expected reduction of possible adverse health effects under alternative ambient PM standards in Southeast Los Angeles County and Philadelphia. The Staff Paper also used sensitivity analysis to show the effect of several uncertain assumptions on the mortality risk associated with short-term exposure to PM. While this effort and the resulting PM NAAQS have been challenged (May 14, 1999

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U.S. Appeals Court Decision[1]), no other framework or methodology has yet to emerge as broadly accepted for estimating PM-associated health effects.

A key concern with the approach provided by PM epidemiological studies involves its sole reliance on ambient monitoring station measurements of PM, and relationships derived from these data. Since PM health effects should derive from total personal exposure to PM, including PM inhaled while indoors and outdoors, the plausibility of the empirical relationship between ambient PM and acute mortality (and morbidity) is called into question (Mage et al., 1999). This viewpoint is supported by recent personal exposure studies that suggest that ambient particles of outdoor origin contribute only a faction of the total personal daily exposure to PM, that personal particulate matter concentrations are substantially higher on average than outdoor concentrations, and that personal exposures exhibit a greater range of variability than ambient concentrations. Furthermore, simple regression analyses indicate that measured personal exposure is only modestly correlated with indoor concentration and even less correlated with outdoor concentration (Sexton et al., 1984; Spengler et al., 1985; Lioy et al., 1990; Clayton and Pellizzari, 1991; Clayton et al., 1993; Özkaynak et al., 1996a,b; Wallace, 1996).

As a result, many have expressed concern that error is introduced in the statistical analysis of health and pollution data when using ambient PM measurement as a surrogate for personal exposure, and that this measurement error limits the validity and biases the prediction of the magnitude of the health effects associated with PM (Özkaynak et al., 1986; Lebret, 1990; Thomas et al., 1993; Lipfert and Wyzga, 1995a,b; Navidi and Lurmann, 1995; Wacholder, 1995; Gamble and Lewis, 1996; National Research Council, 1998; Zeger et al., 1999; Wilson et al., 2000). This concern applies both to estimates for the overall population and to estimates for particular subgroups.

Individual characteristics affect both the exposure to, and the risk from inhaled, PM. Sensitive subgroups such as the elderly, chronic obstructive pulmonary disease (COPD) patients or children likely have a greater risk from a given PM exposure (e.g., a higher concentration–response slope and/or a lower threshold). However, some of those subgroups also tend to have restricted or modified activities relative to the general population and subsequently a different pattern of exposure. The report by the National Research Council on "Research Priorities for Airborne Particulate Matter" (National Research Council, 1998) states that "the investigation of relationships between actual personal exposures and outdoor air–particle concentrations is crucial for validating and interpreting the results of epidemiological studies." This report further notes "Understanding the relationship between particulate–matter mass concentrations measured at fixed outdoor sites and actual human exposure to particulate matter will help guide and improve decisions about ambient pollution control strategies."

The goal of this paper is to reexamine the existing relationship used by the EPA and to propose a new methodology that takes exposure into consideration in an integrated manner, and to quantitatively characterize various sources of uncertainties within such a framework. There are a number of other significant uncertainties involved in estimating health effects from PM that are not addressed in this study. These include the implications of PM chemical composition (Lipfert and Wyzga, 1999; Mage, 2002) and copollutant effects (Lipfert and Wyzga, 1997; Carrothers and Evans, 2000), both of which can confound estimated dose–response relationships and result in site-specific variations in apparent PM sensitivity. In this study, only the effects of ambient PM are considered and all PM of a given size class (i.e., PM_{10} or $PM_{2.5}$) is assumed to have equal potency, regardless of its origin (in particular, indoor-source and outdoor-source PM are assumed equal in their contribution to premature mortality, per unit exposure). Furthermore, the health endpoint utilized in this paper, premature mortality, does not distinguish between deaths advanced by only a small amount of time among those who would have otherwise died shortly after the exposure date, and deaths involving longer reductions in life span (Schimmel and Murawsky, 1978; Zeger et al., 1999). A number of health impact studies now attempt to differentiate between these by estimating the value of a statistical life *year* (Hammit, 2000). This paper is not intended to present a full characterization of all of the uncertainties affecting estimates of PM-associated mortality; considerable ongoing and future research will be needed to accomplish this. Rather, it is intended to demonstrate how current models can be better formulated and interpreted when one additional factor, the relationship between ambient concentrations and personal exposure, is included explicitly. The technique presented in this paper is based on the development of a time-weighted exposure model; while such an approach has only limited power for predicting individual exposure compared to direct measurement for each participant, it is useful for characterizing the general distribution of exposures in a population. The estimated population exposure distribution provides a basis for an estimation of the misclassification error in epidemiological studies and for subsequent application in predicting the impact of reductions in ambient concentrations on personal

[1]On February 27, 2001, the Supreme Court upheld authority delegated to the Environmental Protection Agency by the 1970 Clean Air Act to set strict clean air standards for PM and ground level ozone without regard to economic cost.

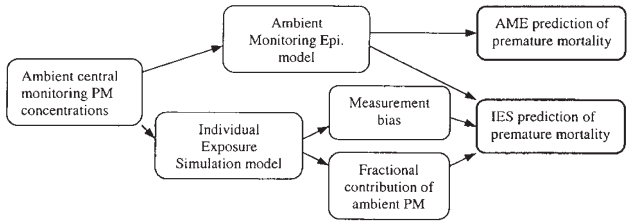


Figure 1. The framework for the predictions of premature mortalities due to ambient PM by the ambient monitoring epidemiology (AME) model and the individual exposure simulation (IES) model.

exposure and associated health outcomes (Spengler et al., 1985; Wilson et al., 2000).

The effects of including exposure are demonstrated by comparing the results of alternative modeling approaches, as summarized in Figure 1. The PM ambient monitoring epidemiology (AME) model uses ambient PM concentrations from a central monitoring station and epidemiological ambient concentration–response relationships to estimate the premature mortality associated with ambient PM. The dose–response relationships are based on daily mortality, time-series regression studies. The individual exposure simulation (IES) model simulates daily individual PM exposures from outdoor and indoor sources, uses the simulation results to estimate both the bias from measurement and misclassification error and the fractional contribution of outdoor PM to personal exposure, and estimates the premature mortality associated with ambient PM accordingly. We compare the predictions of the expected annual number of premature mortality cases in Los Angeles County associated with ambient PM by the AME model and the IES model, indicate the portion of the total exposure to PM in the IES model that is from outdoor versus indoor sources, and discuss the ranges and sources of uncertainties in the overall analysis. The PM AME/IES models can be evaluated for the general population and/or subgroups of the population to estimate health effects associated with PM. In this paper, only estimates of premature mortality for the general population are presented.

The AME model

Model Description

The PM AME model regression analysis that statistically links air pollution and health effects has the general form of:

$$E(Y) = \exp(\beta_{PM} C_{out} + confounders) \quad (1)$$

In this model, the expected value of the health endpoint (mortality count in this case) Y is modeled as an exponential function of the explanatory variables (the observed ambient particulate matter (PM) concentration, C_{out}, and other covariates). The relative risk (RR) associated with a change in the air pollution concentration is $RR = \exp[\beta_{PM} \times \Delta C_{out}]$, where the slope, β_{PM}, is the log-relative risk of death associated with a unit change in measured ambient concentration. The risk model for PM is given by (U.S. EPA, 1996a,b):

$$\Delta y = y[RR - 1] = y[e^{\beta_{PM} \times \Delta C_{out}} - 1] \quad (2)$$

where the predicted excess mortality (Δy) is a function of the baseline health incidence rate (y), the change in the ambient PM air concentration (ΔC_{out}) and the slope of the PM concentration–response curve (β_{PM}). Implicit in Eqs. 1 and 2 is the assumption that the ambient concentration serves as an unbiased surrogate for total personal exposure. It is believed that this assumption is true when indoor-generated PM concentrations are statistically independent of outdoor-generated PM concentrations; then variations in PM health endpoints associated with the ambient component of personal exposures can be separately estimated (Wilson et al., 2000).

Uncertainty Analysis for the AME Model

The uncertainty analysis considers both structural uncertainty and parameter uncertainty in the PM AME model. Uncertainties in three aspects of the model are considered: the dose–response slope, different threshold levels, and the influence of natural background concentrations. The values of the regression coefficients presented in epidemiological studies include confidence intervals to reflect statistical uncertainty associated with limited sample sizes. However, confounding factors, which affect the health endpoints, but are not considered in the model can also contribute uncertainty in a manner not captured by classical confidence intervals. Extrapolating the estimated RRs in epidemiological studies and applying them to other locations is thus difficult and uncertain. The structural form of the concentration–response function is also uncertain. There is a possibility that the range over which the estimated RR exceeds 1.0 extends down to zero concentration (no threshold), or the RR may only exceed 1.0 above some cutpoint (threshold). Finally, our models attempt to calculate the *excess mortality at ambient PM levels that is due to PM resulting from anthropogenic emissions*. As such, it is also important to characterize the background PM level that would occur in the absence of anthropogenic emissions. The overall assumptions of the uncertainty analysis of the AME model are summarized in Table 1, based primarily on the EPA Staff Paper (U.S. EPA, 1996b).

Regression Coefficient

The uncertainties in the estimates of slope factors in epidemiological studies are mainly caused by measurement errors and multicollinearity problems. Time-series epidemiological studies use measures of the daily variation of ambient PM concentration and daily mortality/morbidity

Table 1. Summary of uncertainties incorporated in the integrated uncertainty analysis of the AME model (modified from the EPA Staff Paper) (U.S. Environmental Protection Agency, 1996b).

Uncertainty	Distribution
Coefficient (β) in concentration–response function	Based on distribution of β's obtained from pooled results of mortality studies reviewed by the EPA Staff Paper. The pooled relative risk for a 25 $\mu g/m^3$ increase for $PM_{2.5}$ is 1.04 (1.00, 1.07) and 1.04 (0.99, 1.09) for a 50 $\mu g/m^3$ increase for PM_{10}, where the numbers in the parenthesis are the 95% confidence intervals for the estimated RRs. To match this, the coefficient (β) in the concentration–response function for $PM_{2.5}$ is modeled as normal (0.0015, 0.00088); with β normal (0.000784, 0.000728) for PM_{10}, truncated at zero. (i.e., a hormesis effect is not considered, only RRs ≥ 1 are simulated). Studies included in the pooled analysis for PM_{10} are the six cities study (Schwartz et al., 1996), Chicago, IL (Ito and Thurston, 1996), Utah Valley, UT (Pope et al., 1992), Birmingham, AL (Schwartz, 1993), and Los Angeles, CA (Kinney et al., 1995). Only the six cities study (Schwartz et al., 1996) is used for $PM_{2.5}$.
Cutpoints (threshold) in concentration–response function	Four cutpoints (background, 10, 18, 30 $\mu g/m^3$ for $PM_{2.5}$ and background, 20, 30, 40 $\mu g/m^3$ for PM_{10}) with equal probability for three discrete weighting schemes (Case I–Case III) in the Staff Paper (see cutpoint weighting schemes below).
Background concentration	Uniform distributions on the interval (1, 5 $\mu g/m^3$) and (5, 11 $\mu g/m^3$)for $PM_{2.5}$ and PM_{10}, respectively.

Cutpoint weighting schemes. Moving from Case I to Case III, more probability is assigned to higher cutpoint threshold values

$PM_{2.5}$ (PM_{10})	*Case I*	*Case II*	*Case III*
Background	0.5	0.2	0.05
10 (20) $\mu g/m^3$	0.3	0.3	0.15
18 (30) $\mu g/m^3$	0.15	0.3	0.5
30 (40) $\mu g/m^3$	0.05	0.2	0.3

data to obtain the regression coefficients of the concentration–response relationships along with a measure of their uncertainty (e.g., 95% confidence intervals). Inclusion of other pollutants in the regression model (e.g., sulfate, ozone and nitrate) causes collinearity problems, and the measurement errors of the pollutants also introduce uncertainties to the estimate exposure–risk relationship (Lipfert and Wyzga, 1995a). Other plausible confounders such as weather may lead to further uncertainties in the regression model (Lipfert and Wyzga, 1995b; U.S. EPA, 1996a,b). In addition to these site-specific uncertainties in the fitted model for a particular location, there are uncertainties in the application of the estimated risk from a given study location(s) to other sites of interest (Dominici et al., 2000a,b). The uncertainty estimate for the regression coefficients in the EPA Staff Paper are based on the studies listed in Table 1, and summarized in U.S. EPA (1996b). The uncertainty distributions for the values of the regression coefficients are summarized in Table 1.

Threshold Level

The threshold level is defined as the concentration below which no potential harmful effects are expected to occur. There is limited information to determine the adequacy and accuracy of threshold assumptions. Most ecological time-series studies provide little evidence for the existence of a threshold (Pope et al., 1992; Schwartz, 1994; Schwartz and Zanobetti, 2000; Schwartz et al., 2001). However, such concentration–response relationships should be cautiously interpreted since individual threshold levels may be obscured in the population analysis and misclassification errors and confounding variables can mask the existence of an overall threshold, especially when ambient concentration is used as a surrogate for population exposure in the regression analysis (U.S. EPA, 1996a,b; Cakmak et al., 1999). While several animal studies have used particles to expose normal or impaired animals by inhalation and found pathological mechanisms to explain some of the observed health effects in experimental animals (Chen et al., 1992; Saldiva et al., 1992; Godleski et al., 1996), those experiments have been conducted at high dose relative to the animal, and there is no evidence in the animal studies to support or reject a particular threshold theory.

Background Concentration

The background level is the concentration that would be present in the absence of anthropogenic emissions. It is important in terms of our risk calculation of the expected premature mortality associated with an increased PM concentration above the "background" concentration from natural emissions. Background levels of PM vary by geographic locations and seasons. Emissions of sea salts, organic particles, natural combustion or organic aerosols from vegetation are the natural sources for fine and coarse particles. The aerosol background values can be derived from field measurements at "clean" monitoring stations or mathematical modeling. In the EPA Criteria Document (U.S. EPA, 1996a), two studies were used to estimate the upper and lower bound of the background level of PM in North America. Trijonis et al. (1990) attempted to estimate

$PM_{2.5}$ and PM_{10} "natural" background concentrations from biogenic and geogenic sources which are at or below those possibly associated with preindustrial conditions over North America. The results obtained by Malm et al. (1994) from the IMPROVE study involved monitoring measurements from rural/remote sites in national parks, wilderness areas and national monuments from March 1988 to February 1991. Since this monitoring study did not exclude anthropogenic sources, it is therefore treated as the upper bound of the estimated background concentration. In general, the estimated background concentrations are higher in the eastern than those in the western United States, and are higher in the summer than in the winter season (U.S. EPA, 1996a).

Ambient Concentrations

The air quality data for Los Angeles County used in this study are summarized in Table 2. The PM_{10} and $PM_{2.5}$ monitoring information are provided by the California Air Resources Board, where data from seven monitoring stations are available for PM_{10} and two stations for $PM_{2.5}$ in 1997 (California Air Resources Board, 1999). Comparing the median concentrations in Table 2 to the background and threshold cutpoint assumptions in Table 1, it is seen that the median concentrations ($\sim$15 and 36 $\mu g/m^3$ for $PM_{2.5}$ and PM_{10}, respectively) are well above background, but between the first and second threshold values considered for $PM_{2.5}$ (10 and 18 $\mu g/m^3$) and between the second and third threshold values considered for PM_{10} (30 and 40 $\mu g/m^3$). As such a significant portion of the days are indicated to have PM concentrations below threshold for some of the threshold-cutpoint assumptions considered.

To characterize interindividual variability in ambient PM exposure concentration for a randomly chosen individual within Los Angeles County, the time-series data of the logarithm of PM for the monitoring stations were averaged as shown in Figure 2, and the residual computed for all data points. Let $X_{i,t}$ be the ambient PM concentration measured at air monitoring station i on day t, where t=1 to 365. PM

Table 2. Statistical summary of daily (24-h average) PM concentrations in Los Angeles County, 1997.

	$PM_{2.5}$[a]	PM_{10}[b]
Annual average ($\mu g/m^3$), μ_x	17.34	38.79
Median ($\mu g/m^3$)	14.95	35.57
$\mu_{\ln(X)}$	2.70	3.57
$\sigma_{\ln(X)}$	0.53	0.43
Max of 24-h avg. ($\mu g/m^3$)	68	116
98% ($\mu g/m^3$)	44.17	85.90

[a]Based on data from two monitoring stations: Azusa and North Long Beach (total N=103).

[b]Based on data from seven monitoring stations: Azusa, Burbank–W Palm Avenue, North Long Beach, Los Angeles–North Main Street, Santa Clarita–County Fire Station, Hawthorne, Lancaster–W Pondera Street (total N=406).

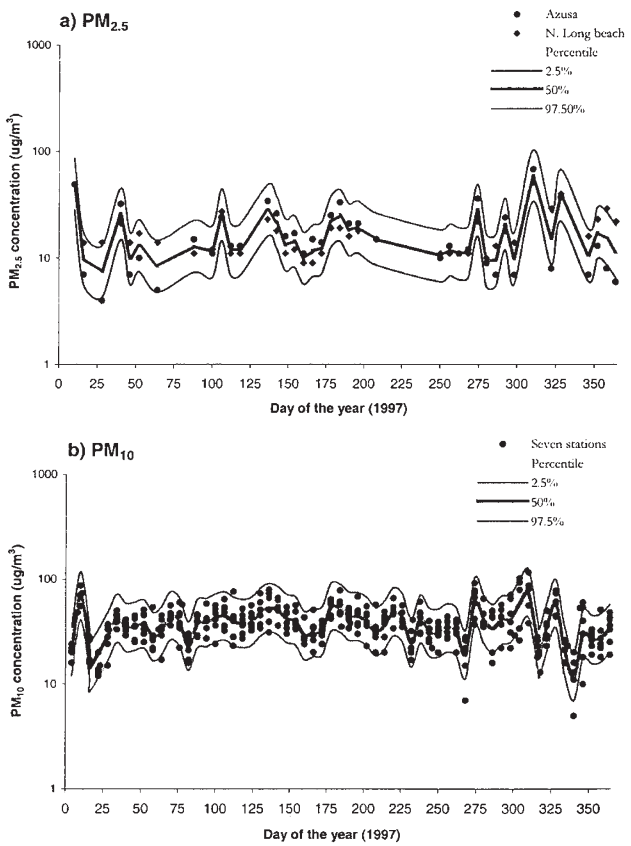


Figure 2. Variability distributions for ambient PM concentrations across Los Angeles County on each day throughout the year. These are used to simulate the ambient exposure PM concentrations for an individual located randomly in the County. The air quality data for Los Angeles County, 1997 are summarized in Table 2. The median values (center lines) and 95% probability intervals (surrounding lines) are shown for the lognormal model fitted to daily observations at the multiple stations.

concentrations are assumed to be lognormally distributed, both from station to station within a day (reflecting spatial variation on a given day), and from day to day (for a given station as well as for the county-wide geometric mean). Since X is LN($\mu_{\ln(X)}$, $\sigma^2_{\ln(X)}$), it follows that $Y=\ln(X)$ is N(μ_Y, σ^2_Y). On a given day t,μ_{Y_t} is the mean value of $\ln(X_{i,t})$, computed as $\mu_{Y_t}=(1/n_t)\sum_{i=1}^{n_t}\ln(X_{i,t})$, where n_t=number of stations with a valid measurement on day t. The standard deviation of the residual from the daily μ_{Y_t} is computed across all days as $\sigma_{\mu_Y}=(1/N-1)\sum_t\sum_{i=1}^{n_t}(\ln X_{i,t}-\mu_{Y_t})^2$ where N is the total number of measurement ($=\sum_t n_t$). This standard deviation reflects the location to location variability of ln(PM) on a given day within Los Angeles County. The resulting values,σ_{μ_Y}=0.28 for $PM_{2.5}$ and σ_{μ_Y}=0.27 for PM_{10}, correspond to coefficients of variation (i.e., standard deviations divided by the means, for the spatial variation of PM concentration on a single day) of 0.29 for $PM_{2.5}$ and 0.28 for PM_{10}. The observed PM data are plotted, along with the computed daily geometric means (exp[μ_{Y_t}]) and 95%

intervals ($\exp[\mu_{Y_t} - 1.96\sigma_{\mu_Y}]$,$\exp[\mu_{Y_t} + 1.96\sigma_{\mu_Y}]$) for $PM_{2.5}$ and PM_{10} in Figure 2a and b, respectively.

IES model

Population-based daily average exposure models for PM_{10} and $PM_{2.5}$ are developed based on the microenvironment approach (Duan, 1982; Sexton et al., 1984; Spengler et al., 1985; Özkaynak et al., 1996a) and the indoor source model developed and modified by Koutrakis et al. (1992) and the Particle Total Exposure Assessment Methodology (PTEAM) study (Özkaynak et al., 1996b). The PTEAM study conducted the first probability-based survey of personal exposure to PM and the elements of the composition of PM in Riverside, CA. The microenvironment approach estimates an individual's total exposure in a specific time period by summing the products of pollutant concentrations in particular locations and the time spent in those locations, or microenvironments; assuming that the pollutant is homogeneously distributed in each microenvironment and in steady-state conditions (Duan, 1982). A mass balance model (Koutrakis et al., 1992) that incorporates penetration factors, decay rates, and source strengths for both PM_{10} and $PM_{2.5}$ size fractions is used to calculate particle concentrations in indoor environments. A probabilistic two-dimensional simulation approach (Frey, 1992) is used to propagate population variability and parameter uncertainty through the exposure model.

The IES model simulates a distribution of daily-averaged personal exposure that represents the distribution of population exposure in a city for a period of time. The sources of personal exposure include outdoor ambient particles, indoor cooking, environmental tobacco smoking (ETS), other indoor sources and unexplained particles that may be generated while engaging in various indoor activities. We assume that concentrations of PM of ambient origin and indoor-source PM emission rates are uncorrelated in time (Mage et al., 1999).

The AME model in Section 2 is replaced by using the simulated "true" population exposures and "true" exposure–response regression slopes corrected according to Cochran (1968) and Wacholder (1995), as described in the next section. As noted previously, the IES model assumes that all PM of a given size has equal potency on a per mass basis, regardless of its origin. The health risks and uncertainty distributions are estimated; then compared with the results from the AME model (Section 2) that is based solely on ambient concentrations and the unadjusted relationship of the time-series studies.

Model Description

The exposure components of the IES model include simulated ambient (outdoor) concentrations, an indoor penetration, source and concentration model; and a microenvironment model to calculate the daily total personal exposure to PM. The overall exposure model is explained in detail in Appendix A. With this model, the equation for the average total daily personal exposure can be expressed as:

$$C_{per} = \gamma + C_{out}T_{out} + C_{in}T_{in} = \gamma + C_{out}T_{out} + \frac{Pa}{a+k}C_{out}T_{in} + \left[\frac{N_{cig}S_{cig} + t_{cook}}{(a+k)Vt} + \frac{Q_{other}}{(a+k)V}\right]T_{in} \quad (3)$$

where

- γ = derived personal cloud concentration, calculated as the difference between the personal exposure measured by a monitor carried by a person and the time-weighted average indoor and outdoor concentrations ($\mu g/m^3$)
- C_{in} = indoor concentration ($\mu g/m^3$)
- C_{out} = ambient PM concentration ($\mu g/m^3$)
- T_{out} = fraction of time spent outdoors during the 24-h monitoring period
- T_{in} = fraction of time spent indoors during the 24-h monitoring period, $T_{in} = 1 - T_{out}$
- P = penetration coefficient
- a = air exchange rate (h^{-1})
- V = volume of room or house (m^3)
- k = decay rate due to diffusion or sedimentation (h^{-1})
- t = duration of the monitoring period (h)
- N_{cig} = number of cigarettes smoked during the 12-h monitoring period
- S_{cig} = mass of particles generated per cigarette smoked (μg/cigarette)
- t_{cook} = time spent cooking (h) during the 12-h monitoring period
- S_{cook} = mass of particles generated per hour of cooking ($\mu g/h$)
- Q_{other} = mass flux of particles from all other indoor sources ($\mu g/h$)

The intercept, γ, is commonly referred to as the "personal cloud" effect. Positive values of γ are determined when, as is commonly observed, total personal exposures exceed values attributable to all measured sources of indoor and outdoor particles. Methods for estimating the value of γ from existing studies are presented in Appendix A. Eq. (3) is the general formula for personal exposure in ETS exposure homes. We assume $N_{cig} \equiv 0$ for non-ETS exposure homes. The population average personal exposure is the weighted average of personal exposure concentrations in ETS and non-ETS exposure homes, weighted by the fraction of homes where there is ETS exposure (Center for Disease Control, 1991; Overpeck and Moss, 1991).

The total exposure model can be simplified by noting that the total PM personal exposure concentration, C_{per}, for an individual is related to their outdoor particle concentration, C_{out}, by the equation:

$$C_{per} = \text{alpha} \times C_{out} + R \quad (4)$$

where the slope alpha is the personal exposure factor that measures the change in personal exposure per unit change in measured ambient concentration and R is the factor that captures the residuals contributed from indoor sources and the personal cloud. From Eq. (3), alpha is the factor that describes the contributions of particles of outdoor origin to actual human exposures and it is a function of the time spent indoors and outdoors, the building air exchange rate, the particle penetration coefficient, and the decay rate:

$$\text{alpha} = T_{out} + T_{in}\frac{Pa}{a+k} \quad (5)$$

Outdoor concentrations have a greater influence on total personal exposure when the values of T_{out}, P and a are higher, and less influence when the values of T_{in} and k are higher.

To implement the IES model, we use Eqs. (3) Eqs. (4) Eqs. (5) to simulate daily individual personal exposure for a sample size of I random people (i=1, I) for each day of the year. (This simulation is implemented for I=1000 random people for 91 days in each season using the median Latin Hypercube method in the Analytica™ [Lumina Decision Systems, Los Gatos, CA] software package.) Each day, a natural log mean ambient PM concentration for Los Angeles County, μ_{Y_t}, is simulated by sampling from the lognormal distribution with $\mu_{\ln(X)}$=2.70 and $\sigma_{\ln(X)}$=0.53 for $PM_{2.5}$ and $\mu_{\ln(X)}$=3.57 and $\sigma_{\ln(X)}$=0.43 for PM_{10} (see Table 2). Then for each person sampled on that day, a value of $C_{out,t}$ is sampled from the lognormal distribution using the value of μ_{Y_t} simulated above, and with σ_{μ_t} equal to 0.28 for $PM_{2.5}$ and 0.27 for PM_{10} (see Section 2.2.4). The time activity, indoor source, air exchange rate and outdoor–indoor penetration factors for individual i are simulated independently. This leads to an estimate of total personal exposure for each of the i people sampled for each day of the year. Sources of information and estimated distributions for the IES model input variables are described in the next section and Table 3.

Input Variables

The probabilistic microenvironment exposure model simulates various microenvironment concentrations and time activity patterns in terms of probabilistic distributions of occurrence frequency, independent of their actual time of occurrence. The exposure scenarios are largely adopted from the PTEAM study (Özkaynak et al., 1996b). The input variables in the exposure model are collected from various studies (Table 3). Variables are indexed by season, and/or daytime versus nighttime and size of PM ($PM_{2.5}$ and PM_{10}). As indicated in Table 3, the variabilities of the model inputs across the population are described by normal or lognormal distributions from various studies. All primary input variables to the IES model represent only one dimension with either uncertainty or variability (Table 3). Note that the fraction of the population with ETS exposure is the only variable that represents uncertainty in the exposure model (portraying our lack of knowledge of the "true" value in the target population), while other variable distributions, such as for air exchange rates, characterize the heterogeneity over time or across individuals in the target population. From Eq. (5), we can see that the calculated alpha represents a one-dimensional variability distribution for the target population. Later in Section 4.3, to examine the importance and sensitivity of model predictions of premature mortality to uncertainty associated with the variability distributions utilized, we assume a 20% uncertainty in the mean values of alpha in the exposure model. The sensitivity analysis results are discussed later in Section 4.3.

Measurement Error and Misclassification Bias in the IES Model

Since total PM exposures are now to be used as the explanatory variable in the dose–response relationship, a correction must first be made to account for the fact that the slope in this relationship (Eq. (2)) has been estimated using data for the surrogate variable, ambient concentration. In a classical error model for exposure measurement, it is assumed that the observed variable Z (e.g., ambient PM concentrations) is related to the true value of exposure X, with:

$$Z = X + E \quad (6)$$

where E is the measurement error, assumed to have zero mean and to be independent of the true value (X). However, in our simulation model, we predict a strong correlation between the true value X and the measurement error E. Therefore, the classical error model is no longer valid. If Y is the dependent variable we are interested in, such as health effects that may be correlated with ambient air pollution; then:

$$Y = A + \beta_z Z + \text{error} \quad (7)$$

and the relationship between β_z and the true regression coefficient β_x (from the model Y=a+$\beta_x X$+error) is given by:

$$\beta_z = \text{BIAS} \times \beta_x \quad (8)$$

Cochran (1968) and Wacholder (1995) define the effect of nondifferential misclassification, when there is a

Table 3. Input distributions and their dimensions in the IES model.

Input	Statistical distribution[a]		Dimension	Reference
Air exchange rate, a (h^{-1})	Spring: LN(−0.479, 0.668) Summer: LN(0.053, 0.912) Fall: LN(−0.878, 0.71) Winter: LN(−0.680, 0.647)		Variability	Murray and Burmaster, 1995
House volume, V (m^3)	LN(5.612, 0.554)		Variability	Murray, 1997
Penetration coefficient, P	$PM_{2.5}$ Day: N(1, 0.097)[b] Night: N(0.89, 0.0697)[b]	PM_{10} Day: N(1, 0.15)[b] Night: N(0.88, 0.075)[b]	Variability	Özkaynak et al., 1996b
Decay rate, k (h^{-1})	$PM_{2.5}$ Day: N(0.27, 0.118) Night: N(0.39, 0.109)	PM_{10} Day: N(0.91, 0.336) Night: N(0.43, 0.136)	Variability	Özkaynak et al., 1996b
Number of cigarettes, N_{cig}	Day: LN(1.47, 1.06) Night: LN(1.27, 0.90)		Variability	Özkaynak et al., 1996b
Smoking source, S_{cig} (μg/cig)	$PM_{2.5}$ Day: N(10,900, 3360) Night: N(16,900, 10,770)	PM_{10} Day: N(23,200, 10,770) Night: N(19,000, 6144)	Variability	Özkaynak et al., 1996b
Cooking time, t_{cook} (min)	Day: TN(28.32, 42.12)[c] Night: TN(5.76, 15.42)[c]		Variability	Özkaynak et al., 1996b
Cooking source, S_{cook}, (μg/h)	$PM_{2.5}$ Day: N(1560, 499) Night: N(693, 531)	PM_{10} Day: N(5380, 1654) Night: N(1640, 916)	Variability	Özkaynak et al., 1996b
Other indoor source, Q_{other} (μg/h)	$PM_{2.5}$ Day: N(1460, 1009) Night: N(784, 626)	PM_{10} Day: N(14,300, 5275) Night: N(2120, 1150)	Variability	Özkaynak et al., 1996b
% of population with ETS exposure	N(0.42, 0.042)		Uncertainty	Center for Disease Control, 1991; Overpeck and Moss, 1991
Average time spent outdoors during the 24 h monitoring period (h), $T_{out}\times 24$	Spring: TN(3.36, 2.92)[c] Summer: TN(4.07, 2.92)[c] Fall: TN(3.69, 2.92)[c] Winter: TN(3.21, 2.92)[c]		Variability	Johnson, 1987; Wiley et al., 1991

[a]All normal distributions are truncated at zero, i.e., all variables have nonnegative values. Seasons are defined as spring: March, April, May; summer: June, July, August; fall: September, October, November; winter: December, January, February. Day is defined from 7 AM–7 PM and night from 7 PM–7 AM.
[b]The penetration coefficients are bounded to be ≤ 1; that is, the values are assigned to be 1 if they were greater than 1 from the simulation.
[c]TN denotes a truncated normal distribution with the indicated mean and standard deviation. The parameters of the parent normal distribution are selected to match these moments (see Johnson and Kotz, 1970).

correlation between the true values and measurement errors, as BIAS:

$$\text{BIAS} = \frac{\beta_z}{\beta_x} \equiv \frac{\sigma^2 + \phi}{\sigma^2 + 2\phi + \omega^2} \tag{9}$$

where $\sigma^2 \equiv$ variance (X), $\omega^2 \equiv$ variance (E), and $\phi \equiv$ covariance (X, E). The correlation coefficient between X and E is given by $\rho = \phi/(\sigma\omega)$.

From Eqs. (4) and 6), X is the true PM exposure and alpha$\times C_{out}$ is the true value of PM total personal exposure that is contributed from outdoor particles.[2] Since we intend to estimate the increased risk of health effects due to exposure to outdoor ambient PM, it is alpha$\times C_{out}$ that is relevant to regulatory policy. The exposure–response relationship in the IES model thus becomes:

$$\Delta y = y[\, e^{\frac{\beta_x}{\text{BIAS}} \times \text{alpha} \times C_{out}} - 1\,] = y[\text{RR} - 1] \tag{10}$$

The results of Eq. (10) are computed by the IES model, and then compared to those of the AME model computed using Eq. (2).

Implementation

The steps in the comparative assessment of the AME and IES models are summarized as follows:

1. Simulate ambient PM concentrations for randomly chosen individuals within Los Angeles County. A median concentration for Los Angeles County is simulated by sampling from the lognormal distribution summarized in Table 2. Then for each person sampled on that day, a value

[2]The average personal exposure to ambient particles is the time-weighted sum of the outdoor exposure to ambient particles and of the indoor exposure to ambient particles that have infiltrated indoors.

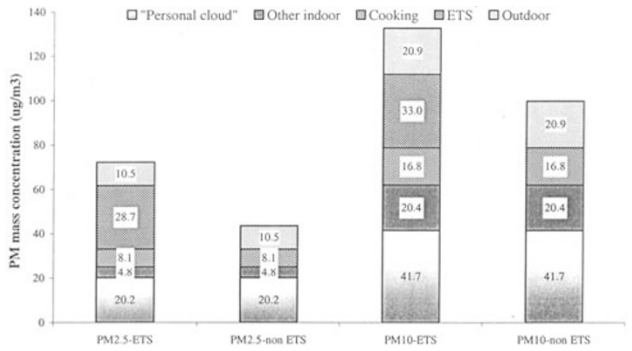


Figure 3. Sources of indoor- and outdoor-origin particles and their average contributions to simulated (IES model) daily average personal exposure of $PM_{2.5}$ and PM_{10}. Results shown for ETS- and non-ETS-exposed population.

of $C_{out,t}$ is sampled from the lognormal distribution with a $\mu_{\ln(X)}$ determined from the median concentration simulated for that day, and $\sigma_{\ln(X)}$ equal to 0.28 for $PM_{2.5}$ and 0.27 for PM_{10} (see Figure 2a and b and Section 2.2.4).

2. Estimate the uncertainty distributions of the epidemiological regression coefficient (β), and threshold level associated with daily exposure to $PM_{2.5}$ and PM_{10}. The integrated uncertainty distributions of the dose–response curve apply equally to each individual in the variability distribution that is simulated in Step 1.

3. Calculate the predicted annual number of premature mortalities associated with exposure to ambient PM by Eq. (2) and the uncertainty of this prediction.

Steps 1–3 determine the results for the AME model.

4. Construct the PM exposure model from Eq. (3). The assumed distributions and sources of information for the variables are described in Table 3.

5. Calculate alpha and the BIAS factor from Eqs. (5 and 9) in the IES model. Note that alpha and BIAS are simulated independently in order to represent their difference. To correct for the misclassification errors in epidemiological studies, BIAS should be calculated from the representative average variability and uncertainty distributions in cities where epidemiological studies were conducted. On the other hand, alpha is calculated separately from the simulation that characterizes the variability distributions for the population in *an application city* where we wish to estimate the impact of possible air pollution control strategies. In the example that follows, Los Angeles is used as both the epidemiology study site (for BIAS) and the application site (for alpha).

6. Calculate the new predicted values of the annual number of premature mortalities associated with total exposure to ambient PM by Eq. (10).

Steps 4–6 determine the results for the IES model.

7. Compare the results from the AME model and the IES model.

Results

The average exposure simulation results of the IES model are summarized in Figure 3. For non-ETS exposed individuals, outdoor-source PM comprises about 24% of the total personal exposure for $PM_{2.5}$, and 21% of the total exposure for PM_{10}. For ETS exposed individuals, outdoor-source PM comprises about 15% of the daily total personal PM exposure for both $PM_{2.5}$ and PM_{10}.

Alpha, BIAS and Net Correction Factor

Based on Eqs. (2) and (10), the IES model and the AME model are different from each other in two terms: alpha and the BIAS factor. The mean values of alpha, BIAS, and the net bias-correction factor (alpha/BIAS) are summarized in

Table 4. Summary statistics of mean value of simulated infiltration factor, alpha, BIAS, and alpha/BIAS for $PM_{2.5}$ and PM_{10} for Los Angeles indexed by season.

	Spring	Summer	Fall	Winter
$PM_{2.5}$				
pa/(a+k)	0.60	0.68	0.53	0.57
alpha	0.66	0.74	0.60	0.62
BIAS	0.62	0.81	0.61	0.70
alpha/BIAS	1.06	0.91	0.97	0.88
PM_{10}				
pa/(a+k)	0.46	0.56	0.39	0.42
alpha	0.54	0.64	0.48	0.50
BIAS	0.60	0.67	0.54	0.58
alpha/BIAS	0.91	0.95	0.88	0.86

The infiltration factor, pa/(a+k), is the equilibrium fraction of ambient PM that is found indoors. Alpha is the factor that describes the contributions of particles of outdoor origin to actual human exposures. BIAS is the estimated effect of measurement error bias caused by the sole use of ambient exposure data in the time-series studies. Alpha/BIAS is a correction factor in the absence of threshold effects; values above 1.0 indicate that the IES model prediction is higher than the AME model prediction (without thresholds). Values below 1.0 indicate the opposite.

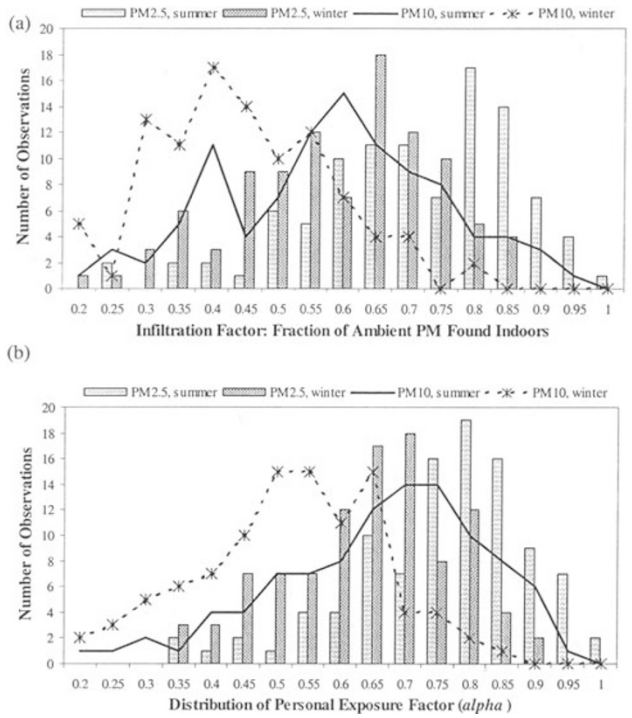


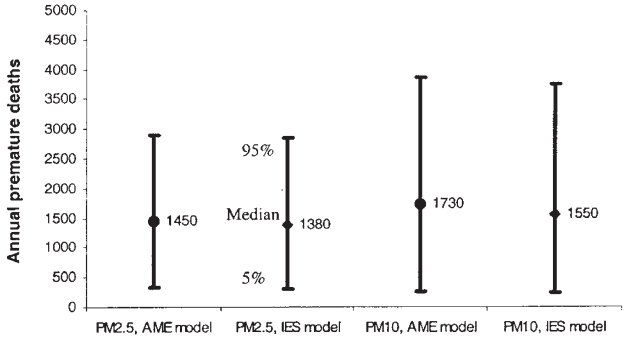
Figure 4. Simulated distributions of (a) infiltration factor: faction of ambient PM found indoors, and (b) personal exposure factor, alpha, by size of PM ($PM_{2.5}/PM_{10}$) and by season.

Table 4, indexed by PM_{10}, $PM_{2.5}$, and season. The infiltration factor is the equilibrium fraction of ambient PM that is found indoors; while alpha is the fraction of ambient PM found in personal exposure. The distributions of the infiltration factor, $pa/(a+k)$, and alpha are plotted in Figure 4.

As shown in Table 4 and Figure 4, the values of the infiltration factor and alpha for $PM_{2.5}$ are greater than those for PM_{10} across all seasons; that is, the percent contributions of particles of outdoor origin to total human exposure is greater for $PM_{2.5}$ than for PM_{10}. In general, mean values of $pa/(a+k)$ and alpha are highest in the summer due to higher air exchange rates and greater time spent outdoors in the summer. The average values of alpha throughout the year are equal to 0.65 for $PM_{2.5}$ and 0.54 for PM_{10}. These findings are generally consistent with previous results from the literature (Özkaynak et al., 1996a,b; Rojas-Bracho et al., 2000; Wilson et al., 2000). Simple regressions of personal PM_{10} exposures on outdoor concentrations found the regression coefficients (alpha) to be 0.54 in the PTEAM study (Özkaynak et al., 1996b) and 0.56 in the study by Lioy et al. (1990).

Values of BIAS from our simulation are on average higher (i.e., closer to 1.0, indicating less bias) for $PM_{2.5}$ than for PM_{10}, and higher in the summer (Table 4). The average values of BIAS across all seasons are 0.69 for $PM_{2.5}$ and 0.60 for PM_{10}. In the PTEAM study, in which real personal PM_{10} monitoring measurements were used to calculate the bias factor using the same equation, the values are reported to be always smaller than 0.6, i.e., the misclassification errors bias the regression coefficients for the exposure variable toward the null (Özkaynak and Spengler, 1996). Özkaynak and Spengler concluded that based on the evidence of the PTEAM study in which the bias was calculated, exaggeration or reversal of PM effects is not expected and therefore the true PM health risk coefficient is more likely to be underestimated by the epidemiological studies (Özkaynak and Spengler, 1996). Cakmak et al. (1999) in their simulation that considers the influence of measurement error in distinguishing between linear no-threshold and linear threshold models, also infer an attenuated effect for the slope coefficient in the presence of measurement error.

As indicated by Eq. (10), alpha/BIAS is the net correction factor for predicting changes in relative risk from changes in ambient concentrations using the IES model, rather than the AME model. The seasonal mean values of alpha/BIAS range from 0.88 to 1.06 for $PM_{2.5}$ and from 0.86 to 0.95 for PM_{10} (Table 4). The mean values of alpha/BIAS across all seasons are equal to 0.96 for $PM_{2.5}$ and 0.90 for PM_{10}. The IES model thus predicts a lower corrected exposure–response regression slope (and a lower sensitivity to changes in ambient PM) for both $PM_{2.5}$ and PM_{10} compared to the AME model. As such, we predict that previous predictions of changes in premature mortality resulting from changes in ambient concentration have generally slightly overestimated this change for both $PM_{2.5}$ and PM_{10}.



Annual premature deaths	$PM_{2.5}$	PM_{10}
AME Model	1450 (330 ~ 2,900)	1730 (260 ~ 3,850)
IES Model	1380 (320 ~ 2,860)	1550 (230 ~ 3,750)

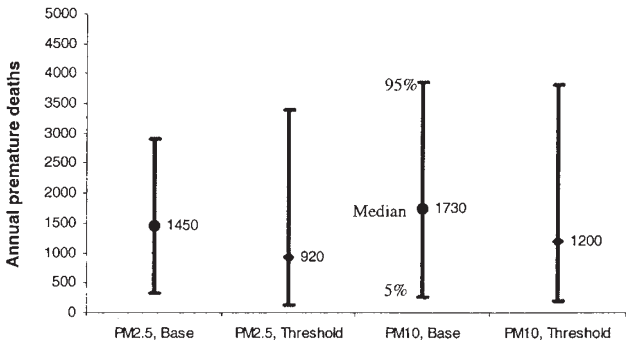
Figure 5. Integrated uncertainty assessment of predicted premature mortality associated with daily exposure to ambient PM in Los Angeles County, 1997. Results shown for the general total population, including the median, 5th, and 95th percentile predictions (population: 9.1 million).

Prediction of Premature Mortality and Associated Uncertainty Intervals

The results of the integrated uncertainty analysis described in Sections 2 and 3 are plotted in Figure 5. Figure 5 shows that the predicted median values of mortality risk associated with exposure to ambient PM are slightly higher for the AME model than for the IES model for both $PM_{2.5}$ (1440 vs. 1380 annual premature deaths in Los Angeles County) and PM_{10} (1730 vs. 1550 annual premature deaths). The 90% credible intervals are roughly the same in both models. In the case where threshold uncertainty exists in the AME model, as analyzed in the EPA Staff Paper and summarized in Table 1, the predictions are 30% lower with wider credible intervals for both $PM_{2.5}$ and PM_{10} (Figure 6).

Sensitivity Analysis

Tornado diagrams (Clemen, 1996) are used to compare the sensitivity of the model to the variables in the integrated uncertainty analysis. Figure 7 shows the results of the sensitivity analysis for $PM_{2.5}$ and PM_{10} in both the AME model and the IES model. The bars represent the 90% range for the estimated number of premature mortalities in the total population when only the specified variable is uncertain, keeping all other variables at their base-case values. The results show that the slope and threshold uncertainties are important in determining the expected number of premature mortalities at current air quality levels in Los Angeles County in the AME model (Figure 7a and b). The threshold uncertainty plays a more important role for $PM_{2.5}$ than for PM_{10} in the AME model. To test for the importance of the uncertainty about the population variability distributions in



Annual premature deaths	$PM_{2.5}$	PM_{10}
AME Model	1450 (330 ~ 2,900)	1730 (260 ~ 3,850)
AME Model with threshold assumption	920 (140 ~ 3,400)	1200 (200 ~ 3,810)

Figure 6. Comparison of predicted premature mortality associated with daily exposure to ambient PM in the AME model when threshold uncertainty exists. Median and 90% credible intervals are simulated for the general population in Los Angeles County, 1997, including the median, 5th, and 95th percentile predictions (population: 9.1 million).

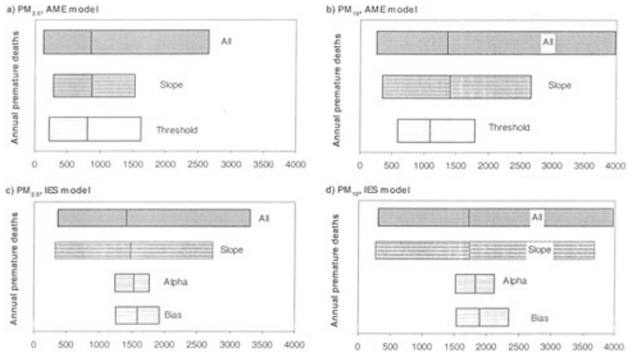


Figure 7. Sensitivity diagrams for total annual number of premature deaths associated with daily exposure to ambient PM in Los Angeles County, 1997. The bars represent the 90% range (5th–95th percentile) for the estimated premature mortality when only the specified variable is uncertain, keeping all other variables at their base values. (a, b) are the sensitivity analysis for the AME model; (c, d) are for the IES model. Slope, threshold, alpha and BIAS are the variables that vary in the sensitivity analysis.

the exposure model and the resulting estimates of alpha and BIAS, we assigned 20% uncertainties to the mean values of alpha and BIAS. The sensitivity analysis shows that the uncertainties for the values of alpha and BIAS in the exposure model contribute a moderate amount to the overall uncertainty in predicted annual premature mortality (Figure 7c and d). Decomposition of the uncertainty in alpha indicates that the air exchange rate contributes roughly half of this uncertainty while the rest of the uncertainty results from, in decreasing order, the uncertainty in the decay rate, time spent outdoors and penetration rates.

Discussion

This paper presents a simulation method for estimating daily individual personal PM exposures and the risks of premature mortality associated with exposure levels contributed from ambient PM concentrations. This application considers the PM personal exposures from both indoor and outdoor sources and the measurement error bias caused by the sole use of ambient exposure data in the epidemiological studies. The predicted risks of premature mortality associated with ambient PM concentrations estimated from the IES model are roughly the same as those estimated from the AME model. The findings are very similar to a recent study conducted by Dominici et al. (2000) using a multistage Poisson regression model. That study evaluated the effects of exposure measurement error on estimates of the effects of particulate air pollution on mortality in time-series studies in Baltimore, Maryland for the period 1987–1994, using the exposure data from the PTEAM study and four other data sets on personal exposure to PM_{10}. Since no direct personal exposure data are available in the target city (Baltimore), Bayesian hierarchical modeling and data augmentation were used to estimate the effective relative rate of mortality

associated with average personal exposure, β_x. They found that the exposure measurement error results in an estimated 56% underestimate in the regression coefficient for PM_{10} (i.e., the log relative rate of mortality from total personal exposure indicates a mean 1.4% increase in mortality per 10 units of change in PM_{10} as opposed to the log relative rate of mortality from ambient exposures that associates a 0.9% increase per 10 $\mu g/m^3$). This translates into BIAS=0.64 in the unadjusted regression coefficient. Furthermore, their study found the posterior mean effect of personal exposure to ambient measurements on the risk of mortality (as represented as $\beta_x \times$alpha or β_z/BIAS$\times$alpha) is equal to 0.79, or equivalently a net correction factor of 0.89. Our simulation results find that BIAS=0.69 for $PM_{2.5}$ and 0.60 for PM_{10} for the regression coefficients in the AME model and mean net correction factors equal to 0.96 for $PM_{2.5}$ and 0.90 for PM_{10}.

The possible existence of a threshold is acknowledged in the AME model and the effect of the threshold uncertainty on predicted premature mortality is shown in Figures 6 and 7. Watt et al. (1995) suggest that the design of ecological time-series studies and their statistical method may not be adequate to detect a true threshold effect. In contrast, the simulation experiments by Cakmak et al. (1999) indicate that standard statistical methods are likely to detect and give nearly unbiased estimates of threshold concentrations even under conditions of extreme exposure measurement error, but that the uncertainty in the threshold estimates increases with the degree of exposure measurement error.

The threshold assumption based on ambient measurements cannot be transferred readily to the IES model where personal exposures include both indoor and ambient-originated particles. A clear procedure to adjust threshold assumptions when translating from the ambient measurement model to the total exposure model is not yet apparent. This is beyond the scope of this paper, but remains an area where future research, model development and testing are needed.

The adjustments in estimated premature mortality associated with the use of the IES model are most pronounced for the winter/fall period, when air exchange rates are lower and people spend less time outdoors. Uncertainties associated with the slope and the threshold assumptions are the most important factors in the sensitivity analysis for AME models. The sensitivity analysis of the IES model suggests that the slope remains the most important factor and the uncertainties in the values of alpha and BIAS contribute a moderate increase of uncertainty to the overall estimation. Thus, while the incorporation of an exposure model is important to account for indoor–outdoor particulate relationships and to correct biases in epidemiological PM slope factors, the most critical uncertainties remain in the dose–response relationships. The uncertainties addressed in this paper are, as noted earlier, limited to a model where PM_{10} and $PM_{2.5}$ are considered as single, homogeneous pollutants. Continuing study of exposure and dose–response relationships is necessary to characterize the implications of other key uncertainties in the PM health assessment, including the effects of PM composition, copollutants, and other covariate factors.

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Appendix A

Microenvironment model (Duan, 1982; Sexton et al., 1984; Spengler et al., 1985; Özkaynak et al., 1996b), indoor source model (Koutrakis et al., 1992) and the derivation of personal cloud effect

Microenvironment Approach

The average of an individual's total exposure in a specific time period is the sum of the products of air pollution concentrations associated with a specific location or activity and the time spent in that location, or microenvironment, divided by the duration of the monitoring period:

$$C_{\text{per}} = \sum_i C_i t_i / \sum_i t_i \qquad \text{(A1)}$$

where C_{per} is the average total personal exposure to PM in a specific time period, and C_i is the average PM concentration in a specific location or for a specific activity in the ith location over the time interval t_i.

Indoor Source Model

Particle concentrations in indoor environments are a function of the air exchange rate (a) for the building, the penetration factors (P), the decay (or deposition) rates (k), and the source strength (Q_{is}) for the given size particle (Koutrakis et al., 1992):

$$C_{\text{in}} = \frac{PaC_{\text{out}} + Q_{is}/V}{a + k} \qquad \text{(A2)}$$

The PTEAM study (Özkaynak et al., 1996b) identified the important indoor sources as smoking, cooking and "other" by use of multivariable analyses (ANOVA and regression analysis), with:

$$Q_{\text{is}} = (N_{\text{cig}} S_{\text{cig}} + T_{\text{cook}} S_{\text{cook}})/t + Q_{\text{other}} \qquad \text{(A3)}$$

Personal Cloud Derivation

The values of personal cloud exposure concentrations (γ) for the general population are not readily available in the literature; we therefore calculate the γ's from the following relationships. The value of γ for PM_{10} is calculated as:

$$\gamma_{(\text{PM10})} = C_{\text{per,PM10}} - C_{\text{out,PM10}} T_{\text{out}} - C_{\text{in,PM10}} T_{\text{in}}.$$

$C_{\text{per,PM10}}$, $C_{\text{in,PM10}}$ and $C_{\text{out,PM10}}$ are obtained from the PTEAM study (Özkaynak et al., 1996b) (Table A-1) and we use the Wiley et al. (1991) survey for information on T_{in} and T_{out}.

For $PM_{2.5}$, since direct measurements of $C_{\text{per,PM2.5}}$ are unavailable, our calculation is more approximate. We use the information from PTEAM to obtain the partitioning of the personal clouds into two size fractions. Regression of personal cloud on fine and coarse fractions in the PTEAM study gives the following relationship:

$$\gamma_{(\text{PM10})} = \text{intercept} + aC_{\text{in,PM2.5}} + bC_{\text{in,PM10}-\text{PM2.5}}$$

Table A-1. PM_{10} particle concentrations (Özkaynak et al., 1996b).

	$C_{\text{out,PM10}}$	$C_{\text{in,PM10}}$	$C_{\text{per,PM10}}$
Daytime	N(96.95, 59.15)	N(98.29, 64.91)	N(143.94, 79.59)
Nighttime	N(87.13, 48.82)	N(65.24, 38.80)	N(78.64, 42.26)

We estimate the value of $\gamma_{(PM2.5)}$ by assuming $\gamma_{(PM2.5)}$ is the fine fraction of $\gamma_{(PM10)}$. However, given the equation above, the partitioning of the intercept into two size fractions is unknown. Therefore, the following equation is used to estimate $\gamma_{(PM2.5)}$:

$$\gamma_{(PM2.5)} = \text{intercept} \times \frac{a \times C_{in,PM2.5}}{a \times C_{in,PM2.5} + b \times C_{in,PM10-PM2.5}} + aC_{in,PM2.5}$$

The values of intercept a and b are given in Table A-2.

The overall estimated $PM_{2.5}$ personal cloud averaged 20.1 $\mu g/m^3$. No existing studies that we know of have measurements on the $PM_{2.5}$ personal cloud for the general population. Studies conducted for sedentary (disabled or elderly) populations have fairly small sample sizes (N=10 and 21) and activities were restricted. These studies have found mean values of the estimated $PM_{2.5}$ personal cloud to be $\sim$3 $\mu g/m^3$ (Bahadori, 1998; Williams et al., 2000). However, both studies have indoor and outdoor $PM_{2.5}$ concentrations two to three times lower than what were measured at the PTEAM study.

Table A-2. Regression of personal cloud ($\gamma_{(PM10)}$) on fine and coarse fractions (Özkaynak et al., 1996b).

	Daytime	Nighttime
Intercept	15.93	48.93
a	0.15	0.11
b	0.07	0.19