

MITIGATING RESIDENTIAL EXPOSURE TO SECONDHAND TOBACCO SMOKE

NEIL E. KLEPEIS ^{a,*} AND WILLIAM W NAZAROFF ^b

^a*Environmental Health Sciences, School of Public Health, University of California, Berkeley, CA 94720*

^b*Department of Civil and Environmental Engineering, University of California, Berkeley, CA 94720*

April 20, 2006

ABSTRACT

In a companion paper, we used a simulation model to explore secondhand tobacco smoke (SHS) exposures for typical conditions in residences. In the current paper, we extend this analysis to evaluate the effectiveness of physical mitigation approaches in reducing nonsmokers' exposure to airborne SHS particulate matter in a hypothetical 6-zone house. Measures investigated included closing doors or opening windows in response to smoking activity, modifying location patterns to segregate the nonsmoker and the active smoker, and operating particle filtration devices. We first performed 24 scripted simulation trials using hypothetical patterns of occupant location. We then performed cohort simulation trials across 25 mitigation scenarios using over 1,000 pairs of nonsmoker and smoker time-location patterns that were selected from a survey of human activity patterns in US homes. We limited cohort pairs to cases where more than 10 cigarettes were smoked indoors at home each day and the nonsmoker was at home for more than two thirds of the day. We evaluated the effectiveness of each mitigation approach by examining its impact on the simulated frequency distribution of residential SHS particle exposure. The two most effective strategies were the isolation of the smoker in a closed room with an open window, and a ban on smoking whenever the nonsmoker was at home. The use of open windows to supply local or cross ventilation, or the operation of portable filtration devices in smoking rooms, provided moderate exposure reductions. Closed doors, by themselves, were not effective.

Key Words: exposure mitigation, doors, windows, filtration, smoker segregation

1 INTRODUCTION

Secondhand tobacco smoke (SHS) consists of particles and gases that have emanated from the burning tip of a tobacco product, or that have been drawn and exhaled by a smoker, and then undergone dilution and transformation in the surrounding environment. Many potentially harmful inorganic and organic species are present in SHS (Jenkins et al., 2000).

SHS inhalation exposure is formally defined as the confluence in space and time of a given air concentration of SHS and the breathing zone of a person (Zartarian et al., 1997). For the purposes of this paper we define exposure concentration to an SHS constituent to be the average attributable air pollutant concentration in the breathing zone of the exposed individual [$\mu\text{g m}^{-3}$] over a 24-h time period. We use the contemporaneous attributable concentration in the rooms occupied by the exposed individual as a proxy for the breathing zone concentration.

Indicators of SHS exposure have been associated with a variety of health problems (USEPA, 1992; OEHHA, 1997), and SHS has been identified by the US government as a known human carcinogen (USDHHS, 2005). The California Air Resources Board has recently declared SHS to be a toxic air contaminant (CARB, 2005). Many have advocated education and counseling to reduce SHS exposure (Gehrman and Hovell, 2003), especially in residences where much of the total exposure to SHS occurs (Klepeis et al., 2001). Children in the US have a potential residential SHS exposure prevalence near 40% (Pyle et al., 2005; McMillen et al., 2003; Schuster et al., 2002).

While total household smoking bans are the most protective, rules that restrict smoking to designated areas or times can lead to substantial reductions in the SHS exposure of young occupants (Wakefield et al., 2000; Bakoula et al., 1997; Biener et al., 1997). However, the relative effectiveness of specific measures, such as smoker-nonsmoker segregation, door closures, or ventilation from open windows, is unknown. Few SHS exposure studies

*Corresponding author. Please visit <http://klepeis.net>

have measured personal exposure concentration profiles concurrently with dynamic human behavior patterns and relevant housing characteristics. The lack of quantitative information on mitigation effectiveness impairs the specification of efficacious interventions.

The current study addresses gaps in SHS exposure knowledge by quantifying the potential effectiveness of several residential SHS exposure mitigation approaches through the use of computer simulation. The approach we take in analyzing SHS mitigation is based on the systematic study of five physical factors, most of which are controllable by household occupants and which may influence residential SHS exposure to varying degrees. These factors, which are illustrated in Figure 1, include the symmetry of air flow, operation of air cleaning devices, positions of doors during smoking, positions of windows during smoking, and in-home occupant location patterns. Three other factors, the type of home and the number of smoking and nonsmoking occupants, were held fixed to simplify the analysis. In exploring exposure levels, we constructed scenarios across combinations of the different factor levels (see Table 1) and chose appropriate model parameter values relevant to expected conditions in real homes.

2 METHODS

To quantify the effectiveness of various mitigation alternatives, we used a computerized multi-zone exposure simulation model based on established principles in the fields of indoor air quality and exposure (Klepeis and Nazaroff, 2006). A central assumption of the model is that SHS pollutants emitted from a cigarette are rapidly mixed in typical residential rooms, such that concentrations do not vary appreciably throughout the room when averaged over periods of several minutes or more. This assumption is supported by empirical evidence (Klepeis, 1999).

To isolate the effects of interior door position, window position, and local filtration on SHS exposure, we limited our analysis to airborne SHS particles and a hypothetical 6-zone house (Figure 2) in which the air handling system always remained inactive. We used constant values for many physical and environmental parameters that are representative of typical US homes (Tables 2 and 3).

In this paper, the term “exposure” refers to a nonsmoker’s 24-h integrated breathing zone concentration of simulated SHS particles. This parameter, in units of $\mu\text{g m}^{-3} \text{ min}$, is calculated by summing the minute-by-minute breathing zone concentration time series. The term “exposure concentration” refers to the time-average concentration in the breathing zone of the nonsmoker, \bar{E}_p , computed by dividing the integral exposure by the duration of the simulated period, 1440 min. For any particu-

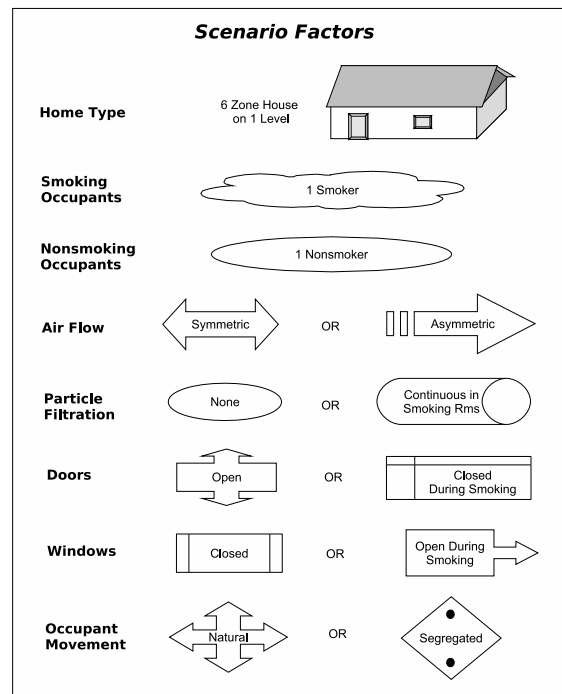


Figure 1: Illustration of eight exposure-related scenario factors and the corresponding factor levels considered in the present simulation-based analysis of residential SHS exposure mitigation. Table 1 summarizes all of the scenarios that were constructed based on these factors. Three factors, the home type and the number of smoking and nonsmoking home occupants, were fixed for all simulations. Five additional dichotomous factors – air flow symmetry, particle filtration in smoking rooms, door position during smoking episodes, window position during smoking episodes, and occupant movement patterns – were the main study variables that were expected to influence SHS exposure to varying degrees. We systematically studied these factors by constructing specific scenarios as described in the text. The model input parameters for physical and environmental quantities, which are associated with different factors, are presented in Tables 2 and 3.

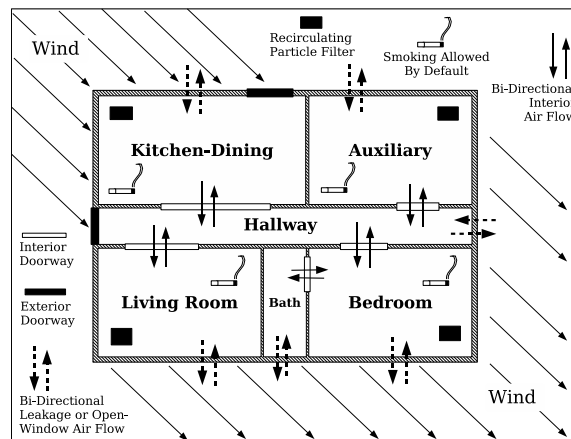


Figure 2: Floorplan for the hypothetical 6-zone house used to conduct all SHS exposure simulations.

Table 1: Summary of SHS Exposure Mitigation Scenarios Examined in the Present Work

Scenario ^a	Home Type ^b	No. Smkrs. ^c	No. Nonsmkrs. ^d	Air Flow ^e	Particle Filtration ^f	Door Position ^g	Window Position ^h	Occupant Movement ⁱ
<i>Scripted Scenarios</i>								
A	6Z	1	1	S	N	AO	AC	F, N, A
B	6Z	1	1	S	N	SC	AC	F, N, A
C	6Z	1	1	S	N	SC	SO	F, N, A
D	6Z	1	1	S	N	AO	AO	F, N, A
A'	6Z	1	1	A	N	AO	AC	F, N, A
B'	6Z	1	1	A	N	SC	AC	F, N, A
C'	6Z	1	1	A	N	SC	SO	F, N, A
D'	6Z	1	1	A	N	AO	AO	F, N, A
<i>Cohort Scenarios</i>								
a1	6Z	1	1	S	N	AO	AC	U
a2	6Z	1	1	S	N	AO	AC	B
b1	6Z	1	1	S	N	SC	AC	U
b2	6Z	1	1	S	N	NC	AC	U
b3	6Z	1	1	S	N	AO	SO	U
b4	6Z	1	1	S	N	AO	NO	U
c1	6Z	1	1	S	N	SC	SO	U
c2	6Z	1	1	S	N	SC	NO	U
c3	6Z	1	1	S	N	NC	SO	U
c4	6Z	1	1	S	N	NC	NO	U
d1	6Z	1	1	S	N	AC	AC	U
d2	6Z	1	1	S	N	AC	SO	U
d3	6Z	1	1	S	N	AC	NO	U
e1	6Z	1	1	S	N	AO	AO	U
e2	6Z	1	1	S	N	SC	AO	U
e3	6Z	1	1	S	N	NC	AO	U
e4	6Z	1	1	S	N	AC	AO	U
f1	6Z	1	1	A	N	AO	AO	U
f2	6Z	1	1	A	N	SC	AO	U
f3	6Z	1	1	A	N	NC	AO	U
f4	6Z	1	1	A	N	AC	AO	U
g1	6Z	1	1	S	N	AO	AC	A
g2	6Z	1	1	S	N	SC	AC	I
g3	6Z	1	1	S	N	SC	SO	I
h1	6Z	1	1	S	S	AO	AC	U

^a This table presents all of the scenarios treated in the present work in terms of eight different factors (home type, number of smoking or nonsmoking occupants, air flow symmetry, particle filtration, door and window positions during smoking episodes, and occupant movement patterns). A–D & A'–D' are scripted scenarios where simulated occupant movements followed one of three patterns (see note *i*). a–h are cohort scenarios involving 1,037 simulated smoker-nonsmoker pairs.

^b All scenarios involved a single-story 6-zone house (6Z). Figure 2 shows the floorplan of the house.

^c All scenarios had a single smoker occupant.

^d All scenarios had a single nonsmoker occupant.

^e Air flows were either symmetric (S) or asymmetric (A) across building boundaries.

^f Filtration was either continuously active in smoking rooms (S) or was not active at all (N).

^g Doors were left open for all times except when occupants were in the bathroom or sleeping (AO), they were closed by smokers during smoking episodes (SC), they were closed by nonsmokers during smoking episodes (NC), or they were closed by both occupants during smoking episodes (AC).

^h Windows were left closed for all times (AC), they were opened by smokers during smoking episodes (SO), they were opened by nonsmokers during smoking episodes (NO), or they were opened by both occupants during smoking episodes (AO).

ⁱ For scripted scenarios, the nonsmoker exhibited "follower" (F), "napper" (N), or "avoider" (A) behavior (see the text). For cohort scenarios, the occupants followed natural, unmodified movement patterns (U), the nonsmoker avoided the smoker during smoking episodes (A), the smoker was isolated in the living room during smoking episodes (I), or the smoker was banned from smoking in the house when the nonsmoker was at home (B).

Table 2: Model Input Parameter Values: Physical and Environmental Quantities

Parameter	Value	Reference(s)
Low-Activity Inhalation Rate	0.0054 m ³ min ⁻¹	Layton (1993)
Cigarettes Smoked	30 d ⁻¹	Nazaroff and Singer (2004); Kopstein (2001)
SHS Particle Mass Emissions	10 mg cig ⁻¹	Klepeis et al. (2003)
Cigarette Duration	10 min	Ott et al. (2003)
Particle Deposition Rate	0.1 h ⁻¹	Lai (2002); Xu et al. (1994)
Air Flow, Open Doorway	100 m ³ h ⁻¹	Miller et al. (1997); Ott et al. (2003)
Air Flow, Closed Doorway	1 m ³ h ⁻¹	Miller et al. (1997); Ott et al. (2003)
Open Window Flow Addition	150 m ³ h ⁻¹	Howard-Reed et al. (2002); Alevantis and Girman (1989)
Filtration Flow Rate	80 m ³ h ⁻¹	See note <i>a</i>
Filtration Removal Efficiency	100%	See note <i>b</i>
House Volume	287 m ³	USCB (2002)
Base Air-Exchange Rate	0.5 h ⁻¹	Murray and Burmaster (1995); Wilson et al. (1996)

^aThe air flow through the filtration device was set at a reasonable rate for typical bedrooms. At 80 m³ h⁻¹, the device will process two rooms worth of air every 0.5–1.5 hours for small- to medium-sized rooms (20–60 m³).

^bReadily-available HEPA filters have single-pass efficiencies exceeding 99.9% for all particle sizes.

Table 3: Characteristics of the Simulated House

^a Rooms	Volume [m ³]	^b Base Leakage Flow [m ³ h ⁻¹]
Kitchen-Dining (Inlet)	100	50
Living Room (Outlet)	50	25
Bedroom (Outlet)	50	25
Auxiliary Room (Outlet)	50	25
Hallway (Inlet)	30	15
Bathroom (Outlet)	7	3.5

^aDuring periods of “asymmetric air flow”, some rooms experienced a net inflow of air from the outdoors (“inlet” rooms) and other rooms had a net outflow to the outdoors (“outlet” rooms).

^bThe baseline air flow between each room and the outdoors due to leakage through the building envelope.

lar simulation, exposure and exposure concentration differ numerically by a constant, so that the effectiveness of a mitigation measure is the same for each metric. The individual intake fraction is calculated by dividing the total mass of residential SHS particles inhaled by a person ($\bar{E}_p \times 1440\text{-min} \times$ the inhalation rate given in Table 2) by the total mass of SHS particles emitted into the residence for the same period (Bennett et al., 2002). The difference in exposure concentration for a base case and each mitigation scenario is calculated as $\Delta = \bar{E}_p^{base} - \bar{E}_p$, where \bar{E}_p^{base} is the 24-h average base exposure concentration in the absence of any attempted mitigation. A useful relative indicator of exposure reduction, the percent effectiveness, is defined as $\eta = 100\% \times \frac{\bar{E}_p^{base} - \bar{E}_p}{\bar{E}_p^{base}}$. The values of Δ and η are positive when exposure is reduced by a given mitigation measure.

All of the mitigation scenarios that we analyzed in our research are summarized in Table 1. In Phase I, we quantified the effects of door and window positions on exposure for different types of nonsmoker activity patterns by performing 24 simulation trials using the scripted nonsmoker location patterns introduced in Klepeis and Nazaroff (2006). The three scripted behaviors consist of

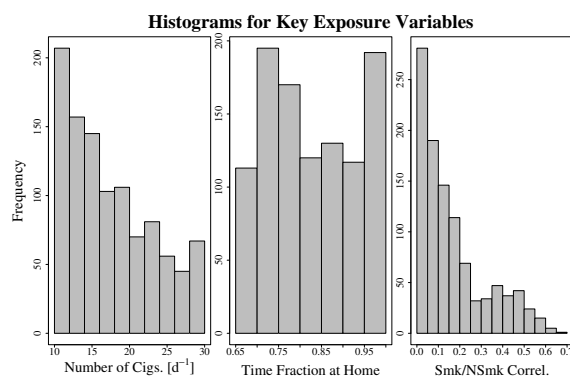


Figure 3: Frequency distributions for three key simulation variables for 1,037 simulated households. The variables are the number of cigarettes smoked in the house over the 24-h simulation period, the fraction of the day the nonsmoker person spent at home, and the fraction of the day the smoker and nonsmoker spent in the same room.

a “follower”, who was always in the same room as the smoker, an “avoider”, who was never in the same room as the smoker, and a “napper”, who spent some time with the smoker, but slept in a separate room. In Phase II, we sought to understand how mitigation measures could impact a population with realistic variation in their activity patterns by conducting simulation trials using the “high risk” pairs of smokers and nonsmoker from the original Klepeis and Nazaroff cohort ($n = 1,037$ out of 5,000 total), which was sampled from the National Human Activity Pattern Survey (NHAPS) (Klepeis et al., 2001). By construction, between 10 and 30 cigarettes were smoked in the house during each cohort simulation and all nonsmokers spent at least $\frac{2}{3}$ of their time at home (see Figure 3, first two panels).

The true correlation in location patterns between multiple persons in US residences, including smoking and nonsmoking cohabitants, is unknown. In the absence of multi-person data, we matched smokers, who were re-

quired to be over the age of 18, with a random nonsmoker from a pool that was interviewed on the same day of the week. This random pairing of house occupants may have resulted in the underrepresentation of smoker and nonsmoker pairs who spent much of the day in the same room, such as a parent and their small child. However, as shown in our previous work, the high exposure simulation cohort experiences realistic exposures and therefore should be useful for evaluating the potential effectiveness of measures to reduce exposure in the home environment.

For each simulation, an exposure mitigation technique was applied in which the house's ventilation, air flows, or air filtration rates are modified in response to the dynamic closing of doors, opening of windows, or operation of portable filtration devices. Ventilation and interior air flows are treated as symmetric or asymmetric (Klepeis and Nazaroff, 2006). In the symmetric case, equal air flows are assigned in each direction across interior or exterior house boundaries. Open windows act independently to increase pollutant removal rates. Asymmetric flows can result when prevailing winds impinge on one side of a house, creating interior crossflows that are modulated by door positions. For simulations with asymmetric flows, we assign incoming flows to "inlet" rooms (Kitchen-Dining Room and Hallway) and outgoing flows to "outlet" rooms (Living Room, Bedroom, Auxiliary Room, and Bathroom). A heuristic is used to determine directional air flows through interior doorways. When doors are closed, a small portion of the directional flow is assumed to flow to or from the outdoors, increasing the air-exchange rate. The last step in the heuristic is to add base symmetric air flows to the directional air flows.

During all simulations, doors and windows in smoking rooms were closed or opened, respectively, in response to simulated smoking activity during complete *smoking episodes*, rather than during each 10-min period of actual smoking. A *smoking episode* was defined as a continuous time period during which a smoker was awake, he or she occupied a particular room in the house, and he or she smoked one or more cigarettes. Conversely, a *nonsmoking episode* was defined as a continuous time period marked by one of the following conditions: (1) the smoker was in a particular room of the house and did not smoke while in that room; (2) the smoker was outside of the home; or (3) the smoker was asleep. Smoking rooms were limited to the main four rooms of the house, excluding the bathroom and hallway.

For Phase I *scripted simulations*, we defined a base case and three mitigation scenarios as summarized below. In the default state, interior doors were always open, except when subjects were in the bathroom or sleeping in the bedroom, windows were always closed, and filtration units were always off. For the three

mitigation scenarios, default conditions were altered during smoking episodes. All four scripted scenarios were performed once for each of the three scripted nonsmoker patterns under symmetric air flow conditions (A-D). The resulting 12 simulations were repeated for asymmetric flows (indicated in the discussion of results by a prime, e.g., A') for a total of 24 simulation trials.

- A. Base.** No mitigation strategies were attempted.
- B. Smk-Door.** The smoker closed the door between the hallway and a room in which a smoking episode occurred, unless the nonsmoker was also present, in which case the door was left open.
- C. Smk-Door Smk-Window.** In addition to the behavior described in B, the smoker opened a window in the room in which a smoking episode occurred.
- D. Smk-Window Nonsmk-Window.** The smoker and nonsmoker each opened a window in the room or rooms they occupied during smoking episodes so that two windows were opened when they were in separate rooms and a single window was opened otherwise. The window was closed in rooms the occupants exited during a given smoking episode, and the window was then opened in the room they subsequently entered.

For Phase II *cohort simulations*, we expanded the number of scenarios to 25, organizing them into eight groups (a-h) as summarized below. Default conditions of open interior doors (when not sleeping or in the bathroom), closed windows, and inactive filtration units applied during all simulations, except for specific conditions related to smoking episodes. All cohort scenarios involved symmetric air flow, except for those in the *Windows-Asymmetric* group.

- a. Bounding.** Either no conscious mitigation strategies were employed or there was a ban on smoking activity during times when the nonsmoker was at home (Base; Time Ban). These two reference scenarios were expected to approximately bound exposures for each simulated individual across all simulation trials.
- b. Door or Window.** During smoking episodes, the smoker or nonsmoker closed the door between the hallway and the room he or she occupied *or* opened a window in the room he or she occupied

(Smk-Door; NSmk-Door; Smk-Window; NSmk-Window).

- c. Door and Window.** During smoking episodes, the smoker and/or nonsmoker closed the door between the hallway and the room he or she occupied *and* opened a window in rooms he or she occupied with only a single door ever closed and a single window ever open (Smk-Door Smk-Window; Smk-Door NSmk-Window; Nonsmk-Door Smk-Window; NSmk-Door NSmk-Window).
- d. Doors.** Both the smoker and nonsmoker closed the doors between the rooms each occupied and the hallway during smoking episodes. In addition, in some trials, either the smoker or the nonsmoker opened the window of the room he or she occupied during smoking episodes (Smk-Door NSmk-Door; Smk-Door Nonsmk-Door Smk-Window; Smk-Door NSmk-Door NSmk-Window).
- e. Windows.** During smoking episodes, both the smoker and nonsmoker opened a window and, in some trials, one or both closed the door between the hallway and rooms each occupied (Smk-Window NSmk-Window; Smk-Door Smk-Window NSmk-Window; NSmk-Door Smk-Window NSmk-Window; Smk-Door NSmk-Door Smk-Window NSmk-Window).
- f. Windows-Asymmetric** During smoking episodes, both the smoker and nonsmoker opened a window in their room and, in some trials, one or both closed the door between the hallway and rooms each occupied (Smk-Window NSmk-Window; Smk-Door Smk-Window NSmk-Window; NSmk-Door Smk-Window NSmk-Window; Smk-Door NSmk-Door Smk-Window NSmk-Window).
- g. Avoid-Isolate.** The nonsmoker avoided being in the same room as the smoker during smoking episodes with the new location randomly selected from available main rooms, or the smoker was isolated in the living room during smoking episodes where they closed the door and, in one trial, also opened the window

(Avoid; Isolate Smk-Door; Isolate Smk-Door Smk-Window).

- h. Filtration.** Smoking rooms were equipped with a 100% efficient portable particle filtration device, which was operated continuously during awake times (Smk Filtration).

The only “location modifying” cohort scenarios were those in the *Avoid-Isolate* group. These scenarios are in contrast to all the other mitigation strategies considered, which use the unmodified NHAPS data. For location-modifying mitigation strategies using smoker isolation, the smoker consumed the same number of cigarettes in the house as before, only changing the location of smoking.

For the base case of both scripted simulations (Phase I) and cohort simulations (Phase II), all windows were left closed all day, no filtration devices were active, and all interior doors were left open, except when occupants were sleeping or using the bathroom. This case was expected to result in the highest simulated exposures for all members of the cohort. Other cases were viewed as perturbations of this base scenario.

When the simulated smoker and nonsmoker occupied the same room, there was the potential for conflict with respect to their door-closing and window-opening behavior. To resolve ambiguity, we made the door behavior of the nonsmoker take precedence during nonsmoking episodes and also for smoking episodes during which no door- or window-related mitigation strategies were in effect. However, for smoking episodes during which certain door- and/or window-related mitigation strategies were in effect, door and window positions reflected an attempt to maximize the immediate reduction of the nonsmoker’s SHS exposure. Hence, when any door-closing strategies were active, the door to a smoking room was always left open during a smoking episode whenever the nonsmoker and smoker were in the same room so that smoke could be cleared from that room into the rest of the house more rapidly. Similarly, when any window-opening strategies were active, the window to a smoking room was always left open whenever the nonsmoker and smoker were in the same room during a smoking episode.

3 RESULTS AND DISCUSSION

As summarized in Table 4, the scripted simulations provide a general understanding of the effects of avoidance behavior, and door and window positions, on nonsmoker exposure and individual intake fraction. As expected, increased levels of smoker avoidance by the nonsmoker resulted in correspondingly lower nonsmoker exposures. The “avoider” exposure was always 2–4 times lower than

the “follower” exposure. When the smoker closed his or her door during smoking episodes, there was a small additional decrease in scripted “napper” and “avoider” exposure. Even though prior research by Miller et al. (1997) and Ott et al. (2003) has indicated that closed doors are effective barriers against SHS, once they are opened after a smoking episode ends, occupants can still be significantly exposed to residual SHS. When a window was opened during smoking episodes in addition to closing the door, exposures were reduced by as much as two thirds. A similar or larger reduction occurred whenever the smoker and nonsmoker both opened windows during smoking episodes but kept doors open. For cases when air flows were asymmetric, the “napper” and the “avoider”, who spent time in “downwind” rooms from the smoker, experienced elevated exposures relative to the symmetric flow cases.

After establishing from the Phase I scripted simulations that physical measures can, in fact, have a substantial and coherent impact on exposure, we proceeded to examine group-level effects based on cohort simulations using a virtual population of 1,037 smoker-nonsmoker pairs. We evaluated each mitigation scenario for its ability to reduce SHS exposure in terms of its impact on the simulated frequency distribution of 24-h average exposure concentrations, \bar{E}_p (see Table 5 and Figures 4 & 5). The overall effectiveness of each mitigation strategy was indicated by median values of Δ , the absolute change in exposure concentration, and η , the percent reduction in exposure concentration (see Table 6). We also evaluated each strategy based on the proportion of people who received no benefit ($\Delta = 0$ and $\eta = 0$) or actually had increased exposures ($\Delta < 0$ and $\eta < 0$) relative to the base case.

While the members of our selected cohort are not strictly representative of any real population, their simulated exposures are expected to be indicative of real exposures. To confirm that different random selections of cohort members would result in similar exposures, we calculated the ratio of the 90th percentile confidence band half-length to the mean as a measure of error in the sample mean relative to the theoretical population mean. We found that the distribution of 1,037 nonsmoker simulated exposures was fairly stable with the confidence band half-length $\sim 10\%$ of the distribution mean.

A benefit of using a simulated population with high potential exposure to study the effects of mitigation strategies was that most of the 24-h average particle exposure concentrations, \bar{E}_p , and many of the Δ 's were approximately lognormally distributed. Consequently, their medians (or geometric means) could be used to compare the central tendency across different simulation trials. Although individual exposures for most scenarios were positive, 26% of simulated exposures for nonsmoking members of the 1,037-pair cohort for the time-ban

scenario ($n=265$) were zero. Thus, when the time ban was enforced, a quarter of nonsmokers in this cohort had their household exposure eliminated entirely. There were also many zero and negative values of Δ and η for some scenarios (Table 6). To represent the complete log-probability plots of distributions that include zero or negative values, we calculated probabilities using all the data. The plotted distributions (Figures 4 and 5) were truncated at the percentile corresponding to the lowest positive value. In calculating the GM and GSD in Table 5, only positive-valued exposures were used, but the other statistics in the table were calculated using all simulated values.

3.1 BASE DISTRIBUTION OF EXPOSURE CONCENTRATIONS

The variation in base exposure concentrations, which had a range of 1 to $165 \mu\text{g m}^{-3}$, was a direct result of complex patterns in the timelines of smoker and nonsmoker movement, combined with the multizone character of the simulated residence. The median base exposure concentration was $32 \mu\text{g m}^{-3}$ ($21 \mu\text{g m}^{-3}$ standard deviation). The middle 80% of exposures varied between 12 and $63 \mu\text{g m}^{-3}$, exhibiting a separation of approximately $50 \mu\text{g m}^{-3}$ or a factor of 5 (Table 5). Although the cohort was constructed to represent the highest fifth of exposures – with a maximum of 30 cigarettes smoked indoors during the day and point estimates for house volume and other physical parameters – the central tendency and variation reflected in our results is broadly comparable with that reported across several empirical studies of residential SHS concentrations or personal SHS exposure concentrations (Özkaynak et al., 1996; Spengler et al., 1985; Quackenboss et al., 1989; Heavner et al., 1996; Jenkins et al., 1996), suggesting that our findings are indicative of real circumstances.

3.2 COMPARATIVE EVALUATION OF MITIGATION STRATEGIES

When mitigation strategies were in effect, median exposure concentrations were 3 to $29 \mu\text{g m}^{-3}$ and standard deviations were 3 to $21 \mu\text{g m}^{-3}$ (Table 5). Median differences from the base exposures (Δ) were 1 to $29 \mu\text{g m}^{-3}$ and the median percent effectiveness (η) ranged from 3% to over 90% (Table 6).

The two most effective strategies were the time ban (median $\Delta = 28 \mu\text{g m}^{-3}$; median $\eta = 90\%$) and the isolation of the smoker in combination with a closed door and open window (median $\Delta = 29 \mu\text{g m}^{-3}$; median $\eta = 93\%$) (Table 6). These two strategies resulted in more benefit for nearly all members of the cohort than other strategies. Opening the window in the closed room with an

Table 4: Scripted 24-h Average Personal SHS Particle Exposure Concentration and Individual Intake Fraction by Mitigation Scenario and Nonsmoker Activity

Mitigation Scenario	Nonsmoker Activity		
	Follower	Napper	Avoider
<i>24-h Average Particle Exposure Concentration, \bar{E}_p [$\mu\text{g m}^{-3}$]</i>			
A. Sym (Base)	61	41	24
B. Sym-Smk-Door	61	37	21
C. Sym-Smk-Door-Smk-Window	24	13	8
D. Sym-Smk-NSmk-Window	24	13	5
A'. Asym	59	45	25
B'. Asym-Smk-Doors	59	43	23
C'. Asym-Smk-Door-Smk-Window	26	19	11
D'. Asym-Smk-NSmk-Window	26	19	9
<i>Individual Particle Intake Fraction [ppm]</i>			
A. Sym (Base)	2600	1700	1000
B. Sym-Smk-Door	2600	1600	880
C. Sym-Smk-Door-Smk-Window	980	550	320
D. Sym-Smk-NSmk-Window	980	550	230
A'. Asym	2500	1900	1000
B'. Asym-Smk-Door	2500	1800	950
C'. Asym-Smk-Door-Smk-Window	1100	810	480
D'. Asym-Smk-NSmk-Window	1100	790	380

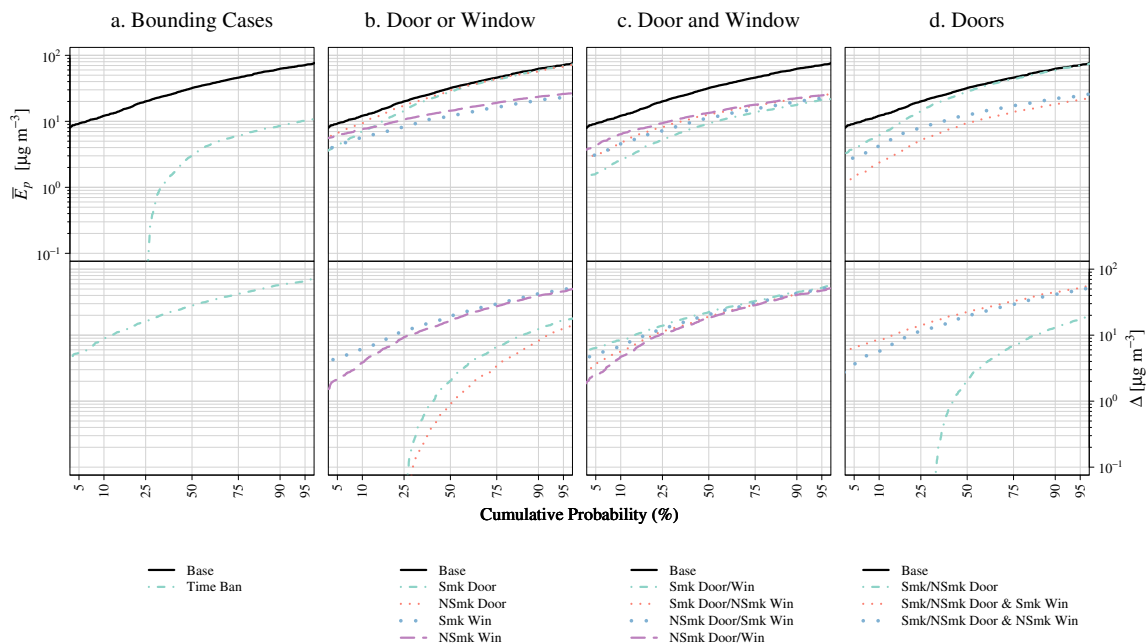
**Figure 4:** Log-probability plots of the 24-h average cohort SHS particle exposure, \bar{E}_p , for the base simulation trial and trials corresponding to mitigation strategies in groups **a.** through **d.** (top panels). The distribution of differences, Δ , in individual exposure from the base case is also presented for each mitigation strategy (bottom panels).

Table 5: Statistics from the Simulated Distribution of Cohort 24-h Average Nonsmoker SHS Particle Exposure Concentration (\bar{E}_p) [$\mu\text{g m}^{-3}$] for Each Exposure Mitigation Strategy

Group	Mitigation Scenario ^a	Std.					Percentiles	
		Mean	Dev.	Median	GM	^c GSD	10th	90th
a. Bounding	a1. Base	35	21	32	29	2.0	12	63
	a2. Time Ban ^b	3.7	3.5	3.1	3.6	2.7	0.0	8.5
b. Door or Win	b1. Smk Door	31	21	28	23	2.5	7.1	58
	b2. NSmk Door	32	20	29	26	2.1	9.3	58
	b3. Smk Win	13	5.9	12	11	1.7	5.7	21
	b4. Nsmk Win	15	6.2	15	14	1.6	7.6	24
c. Door and Win	c1. Smk Door/Win	9.9	6.0	9.3	7.7	2.3	2.6	18
	c2. Smk Door NSmk Win	13	6.7	13	11	2.0	4.7	22
	c3. NSmk Door Smk Win	12	6.0	11	10	1.9	4.6	20
	c4. NSmk Door NSmk Win	14	6.3	13	12	1.7	6.4	22
d. Doors	d1. Smk/NSmk Door	31	21	28	23	2.6	6.1	59
	d2. Smk/NSmk Door Smk Win	9.9	6.2	9.4	7.5	2.4	2.4	18
	d3. Smk/NSmk Door NSmk Win	13	6.8	13	11	2.1	4.3	22
e. Windows	e1. Smk/NSmk Win	11	5.6	9.9	9.2	1.8	4.1	18
	e2. Smk Door Smk/NSmk Win	9.0	5.8	8.1	6.7	2.5	1.9	17
	e3. NSmk Door Smk/NSmk Win	10.1	5.7	9.3	8.3	2.0	3.5	17
	e4. Smk/NSmk Door Smk/NSmk Win	8.9	5.9	8.0	6.5	2.6	1.8	16
f. Wins-Asym	f1. Smk/NSmk Win	12	5.9	11	11	1.7	5.1	20
	f2. Smk Door Smk/NSmk Win	11	6.2	10	8.7	2.2	3.3	19
	f3. NSmk Door Smk/NSmk Win	12	5.9	11	10	1.7	4.8	20
	f4. Smk/NSmk Door Smk/NSmk Win	11	6.2	10	8.6	2.2	3.2	19
g. Avoid-Isolate	g1. Avoid	24	14	21	20	1.8	9.8	39
	g2. Isolate Smk Door	12	10	9.3	7.5	2.9	1.6	25
	g3. Isolate Smk Door/Win	2.7	2.9	1.9	1.7	3.0	0.4	5.8
h. Filt	h1. Smk Filtration	14	9.3	12	11	2.4	3.4	26

^aThere were 1,037 exposure values generated for each scenario, one for each simulated cohort pair. All simulations were for the same house (Figure 2 and Table 3), and the model input parameters were held constant across all scenarios (Table 2).

^bAll exposure values were positive, except for the "Time Ban" scenario for which 265 values were zero. Values of zero were included in the calculation of all "Time Ban" statistics, except for GM and GSD.

^cGSD is dimensionless.

Table 6: Statistics from the Simulated Distribution of Individual Differences, Δ , Between the Base Cohort 24-h Average Nonsmoker SHS Particle Exposure Concentration [$\mu\text{g m}^{-3}$] and that for each Exposure Mitigation Strategy and the Mitigation Percent Effectiveness, η , Sorted from Least to Most Effective According to Median Values^a

Group	Mitigation Scenario	n_{neg}	n_{zero}	Δ [$\mu\text{g m}^{-3}$]			η [%]		
				Med.	10th	90th	Med.	10th	90th
b. Door or Win	b2. NSmk Door	185	70	0.9	-0.4	8.2	3.1	-1.1	30
d. Doors	d1. Smk/NSmk Door	326	0	2	-3.2	13	7.9	-8.8	53
b. Door or Win	b1. Smk Door	265	0	2	-2.4	12	8.2	-6.9	49
g. Avoid-Isolate	g1. Avoid	7	148	8.8	0	27	30	0	53
b. Door or Win	b4. NSmk Win	0	4	17	3.9	40	54	31	65
c. Door and Win	c4. NSmk Door NSmk Win	0	4	18	4.7	41	59	35	69
h. Filt	h1. Smk Filtration	0	0	20	8.4	36	61	54	75
b. Door or Win	b3. Smk Win	0	0	19	6.2	42	62	51	69
c. Door and Win	c2. Smk Door NSmk Win	4	0	19	5.6	41	62	43	73
d. Doors	d3. Smk/NSmk Door NSmk Win	4	0	19	5.7	41	62	44	75
c. Door and Win	c3. NSmk Door Smk Win	0	0	20	6.9	43	65	55	73
f. Wins-Asym	f1. Smk/NSmk Win	23	0	21	5.2	43	65	41	76
f. Wins-Asym	f3. NSmk Door Smk/NSmk Win	23	0	21	5.5	43	66	42	76
g. Avoid-Isolate	g2. Isolate Smk Door	40	0	20	3.6	47	67	25	94
e. Windows	e1. Smk/NSmk Win	0	0	22	7.5	44	68	60	75
f. Wins-Asym	f2. Smk Door Smk/NSmk Win	14	0	22	6.7	45	69	49	83
f. Wins-Asym	f4. Smk/NSmk Door Smk/NSmk Win	14	0	22	6.9	45	69	50	83
e. Windows	e3. NSmk Door Smk/NSmk Win	0	0	22	7.9	45	70	61	79
c. Door and Win	c1. Smk Door/Win	0	0	22	8.6	45	70	62	83
d. Doors	d2. Smk/NSmk Door Smk Win	0	0	22	8.6	45	70	62	84
e. Windows	e2. Smk Door Smk/NSmk Win	0	0	23	9	46	73	66	87
e. Windows	e4. Smk/NSmk Door Smk/NSmk Win	0	0	24	9	46	73	66	88
a. Bounding	a2. Time Ban	0	0	28	8.9	58	90	65	100
g. Avoid-Isolate	g3. Isolate Smk Door/Win	3	0	29	10	58	93	80	99

^a“Med.” designates the median values. The values of Δ and η were negative or zero (n_{neg} and n_{zero}) for some nonsmoking individuals, indicating that their exposure increased or was unchanged, respectively, due to the corresponding mitigation strategy. Non-positive values were included in the calculation of all statistics shown in the table. All simulations were for the same house (Figure 2 and Table 3), and the model input parameters were constant across all scenarios (Table 2).

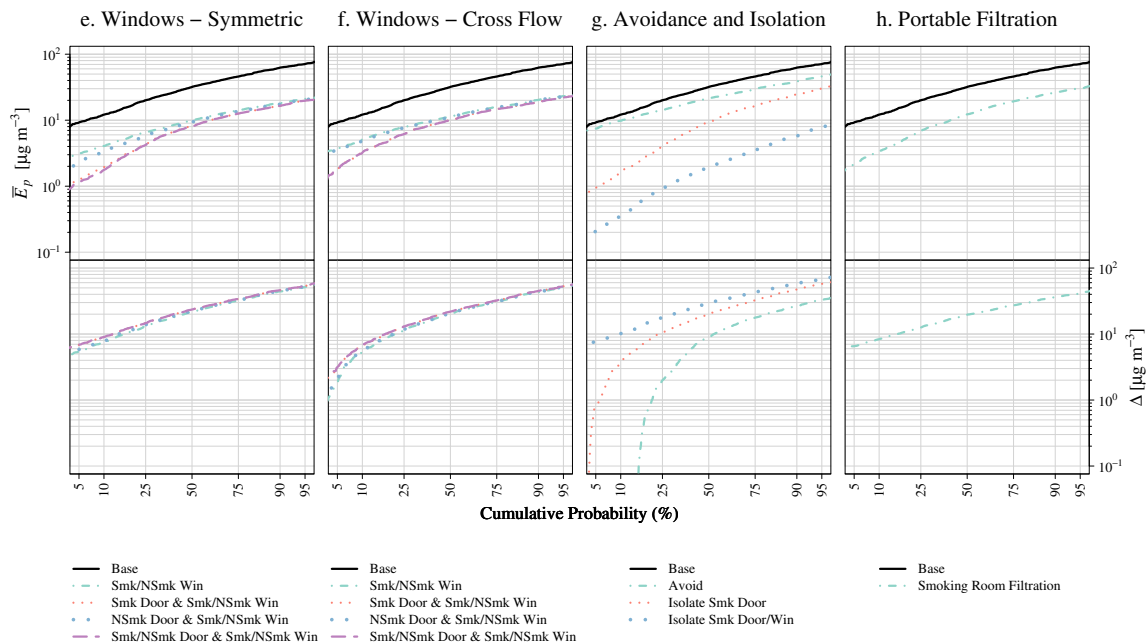


Figure 5: Log-probability plots of the 24-h average cohort SHS particle exposure, \bar{E}_p , for the base simulation trial and trials corresponding to mitigation strategies in groups e. through h. (top panels). The distribution of differences, Δ , in individual exposure from the base case is also presented for each mitigation strategy (bottom panels).

isolated smoker reduced median exposures by $9 \mu\text{g m}^{-3}$ more than did closing the door alone. The time ban was beneficial for more people, since 26% of exposures were eliminated completely, whereas no exposures were eliminated for smoker isolation (with door closed and window open).

The least effective mitigation scenarios involved the use of doors alone to impede smoke transport to nonsmokers during smoking episodes. The median Δ for each of the three door-only scenarios was only $1\text{--}2 \mu\text{g m}^{-3}$ with a median η of 3–8%. Controlling the smoker's door behavior was slightly more beneficial than controlling the nonsmoker's. The exposure drop for door scenarios occurred in the lower 50% of exposures, indicating that the people with higher exposures received little benefit (Figure 4b, d). For approximately 25–30% of cases, the door-only scenarios either had no beneficial effect or increased nonsmoker exposures.

Thus, it appears that while doors are effective in slowing the passage of air pollution between two compartments, they are not necessarily effective in reducing exposure for a population with realistic variation in residential movement patterns. Some persons following typical, unmodified location patterns in their homes can experience reduction in exposure by closing the door to rooms where smokers are active. However, when smoking and

nonsmoking household occupants spend an appreciable amount of time in the same room, the effectiveness of these door-related strategies is strongly diminished. Also the doors-closed case does not speed removal of SHS pollutants from household air and so delayed permeation into other rooms can lead to delayed inhalation exposures. Those persons who already spend time removed from the smoker experience small additional reductions owing to the mitigation measure, whereas others, who spend more time near the smoker, receive negligible benefit.

We were interested in how the distribution of exposure would change if nonsmokers never occupied the same room as a smoker for any smoking episodes. We found that avoidance of the smoker was much more effective (g1 median $\eta = 30\%$) than door-only scenarios (b1, b2, and d1 median $\eta = 3\text{--}8\%$) (Table 6). The avoidance scenario resulted in a somewhat narrower range of exposures with the middle 80–90% ranging from about 10 to $40 \mu\text{g m}^{-3}$ (Table 5 and Figure 5 g), but it had no beneficial effect for 14% ($n = 148$) of nonsmokers and it increased exposures in several cases ($n = 7$). Systematic avoidance was much more effective than doors alone at reducing the highest exposures, but, as with doors, residual SHS in the smoking room and elsewhere led to some delayed inhalation exposures that were not strongly diminished from the base case.

Strategies employing open windows, filtration, and isolation of the smoker in a single room behind a closed door were significantly more effective than door-only or systematic avoidance scenarios with median Δ 's of 17–24, 20, and 20 $\mu\text{g m}^{-3}$, respectively, and median η 's of 54–73% (Table 6, Figures 4b, c and 5e, f). The closing of the smoker's door during window-open scenarios improved the effectiveness by 4–8% relative to cases where the smoker's door was left open. The position of the nonsmoker's door had little influence (1–5%) on the effectiveness of window-open scenarios. Concurrent window opening by the smoker and nonsmoker was up to 19% more effective in reducing exposure than single-window strategies (based on median η values).

3.3 INSTITUTING HOUSEHOLD SMOKING RESTRICTIONS

Each household has particular social dynamics that may affect the success of attempts to change smoker behaviors. A smoker may be willing to adopt simple measures that happen to be not very effective, while refusing to adopt more effective measures that are more invasive. A residence may also have peculiar environmental characteristics, which may affect the implementation of specific mitigations or their effectiveness. For example, it may be difficult to control door and window positions or to change locations in a small apartment that has only one or two main rooms.

Some mitigation strategies may not be practical because they use too much energy or create too much discomfort or annoyance for household occupants. Controlling door position is likely to have the smallest energy requirement and smallest impact on occupant perception. In contrast, depending on climate and weather conditions, maintaining open windows could have a large impact on both energy use and occupant comfort. Finally, air filtration devices may consume substantial amounts of electricity, and the noise associated with their operation may disturb occupants.

In the near future, it is likely that most smoking restrictions will involve voluntary SHS exposure interventions stemming from education, media, or outreach, rather than originating from government regulation (Gehrman and Hovell, 2003). Pediatricians are well positioned to counsel families to reduce or eliminate children's SHS exposure in the home (Winickoff et al., 2005). One motivation for household smoking restrictions is that they may ultimately act to modify rates of smoking. By restricting where, when, or under what conditions smokers can light up, and by helping to engage smokers in dialogs about SHS exposure, restrictions may lead a smoker to consume fewer cigarettes or even to quit entirely (Gilpin et al., 1999; Farkas et al., 1999). As a policy goal, smok-

ing cessation or total smoking bans are desirable, because they would substantially reduce or completely eliminate the exposure of a smoker's family to SHS and also eliminate the smoker's own exposure to mainstream tobacco smoke.

4 CONCLUSION

We have used established principles in the fields of indoor air quality and exposure science to explore the effectiveness of specific residential secondhand tobacco smoke (SHS) exposure mitigation strategies. By using a mechanistic exposure model, one can describe pollutant and human spatio-temporal dynamics at high resolution. This approach permits a quantitative characterization of exposure and an improved understanding of mechanisms. We expect that the results presented here reasonably represent SHS particle exposures that occur in actual US residences. The base predicted 24-h average SHS particle exposure for a high-exposure cohort is $\sim 30 \mu\text{g m}^{-3}$, which is the same approximate value reported for SHS-related particle concentrations measured in several residential monitoring studies.

From the results of 24 scripted simulations and cohort simulations involving 1,037 pairs of smokers and nonsmokers, we have associated varying degrees of success in controlling SHS exposures with simple mitigation alternatives. These results can help inform public health researchers and practitioners in their efforts to reduce SHS exposure in homes.

Major findings in the current work are as follows:

- The most effective strategies for reducing residential exposure for nonsmokers, short of a complete ban on in-home smoking, are those that involve either banning smoking while nonsmokers are at home or isolating the smoker during smoking episodes in a separate room by themselves with that room's door closed and a window open.
- Moderate reductions in nonsmoker exposure can be attained by opening windows during smoking episodes, particularly in rooms occupied by the active smoker, by isolating the smoker in a separate room, or by operating a filtration device during waking hours in rooms where smoking is allowed.
- Approaches that involve only closing doors during smoking episodes when the smoker and nonsmoker are coincidentally in separate rooms, or simply having the nonsmoker avoid a room in which the smoker is active, are, by themselves, not very effective. Their effectiveness is limited because nonsmokers can enter rooms immediately after smoking occurred where sizable SHS concentrations are still present.

In the future, extensions of the model used here might be applied to a real population of people with specific demographic characteristics, including particular housing characteristics and human activity patterns. One could generate tabulations or provide interactive feedback regarding expected exposures for particular intervention or epidemiological target groups. In addition, one might use quantitative information from this and analogous studies to develop web-based tutorials, short courses, and brochures for direct use by students and the general public.

ACKNOWLEDGMENTS

This research was funded in part by a University Partnership Agreement (UPA) between the US Environmental Protection Agency (EPA) and Lawrence Berkeley National Laboratory (LBNL) via Interagency Agreement DW-988-38190-01-0 with the US Department of Energy (DOE) under Contract No. DE-AC03-76SF00098. Support for completing this manuscript was also supplied through a grant from the Flight Attendant Medical Research Institute (FAMRI) to the Department of Statistics at Stanford University. Paul Switzer and Wayne Ott of Stanford provided useful comments on early drafts of this manuscript. The computerized model used in this research was made possible by the R Core Team who developed the R language for statistical computing (R Development Core Team, 2005). The model is available to other researchers in source-code form (see <http://exposurescience.org>).

REFERENCES

- Alevantis, L. E., Girman, J. R., 1989. Occupant-Controlled Residential Ventilation. In: *The Human Equation: Health and Comfort*, Proceedings of the ASHRAE/SOEH Conference IAQ '89. American Society for Heating, Refrigerating, and Air-Conditioning Engineers, San Diego, pp. 184-191.
- Bakoula, C. G., Kafritsa, Y. J., Kavadias, G. D., Haley, N. J., Matsaniotis, N. S., 1997. Factors modifying exposure to environmental tobacco smoke in children (Athens, Greece). *Cancer Causes & Control* 8 (1), 73-76.
- Bennett, D. H., McKone, T. E., Evans, J. S., Nazaroff, W. W., Margni, M. D., Jolliet, O., Smith, K. R., 2002. Defining intake fraction. *Environmental Science and Technology* 36 (9), 206A-211A.
- Biener, L., Cullen, D., Di, Z. X., Hammond, S. K., 1997. Household smoking restrictions and adolescents' exposure to environmental tobacco smoke. *Preventive Medicine* 26 (3), 358-363.
- CARB, Jun. 2005. Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant. Tech. rep., California Environmental Protection Agency, California Air Resources Board, Office of Environmental Health Hazard Assessment, Sacramento, CA.
URL <http://www.arb.ca.gov/regact/ets2006/ets2006.htm>
- Farkas, A. J., Gilpin, E. A., Distefano, J. M., Pierce, J. P., 1999. The effects of household and workplace smoking restrictions on quitting behaviours. *Tobacco Control* 8 (3), 261-265.
- Gehrman, C. A., Hovell, M. F., 2003. Protecting children from environmental tobacco smoke (ETS) exposure: A critical review. *Nicotine & Tobacco Research* 5 (3), 289-301.
- Gilpin, E. A., White, M. M., Farkas, A. J., Pierce, J. P., 1999. Home smoking restrictions: Which smokers have them and how they are associated with smoking behavior? *Nicotine & Tobacco Research* 1 (2), 153-162.
- Heavner, D. L., Morgan, W. T., Ogden, M. W., 1996. Determination of volatile organic compounds and respirable suspended particulate matter in New Jersey and Pennsylvania homes and workplaces. *Environment International* 22 (2), 159-183.
- Howard-Reed, C., Wallace, L. A., Ott, W. R., 2002. The effect of opening windows on air change rates in two homes. *Journal of the Air and Waste Management Association* 52 (2), 147-159.
- Jenkins, R. A., Guerin, M. R., Tomkins, B. A., 2000. *The Chemistry of Environmental Tobacco Smoke: Composition and Measurement*, 2nd Edition. Lewis Publishers, Boca Raton.
- Jenkins, R. A., Palauskas, A., Counts, R. W., Bayne, C. K., Dindal, A. B., Guerin, M. R., 1996. Exposure to environmental tobacco smoke in sixteen cities in the United States as determined by personal breathing zone air sampling. *Journal of Exposure Analysis and Environmental Epidemiology* 6 (4), 473-502.
- Klepeis, N. E., 1999. Validity of the uniform mixing assumption: Determining human exposure to environmental tobacco smoke. *Environmental Health Perspectives* 107 (SUPP2), 357-363.
- Klepeis, N. E., Apte, M. G., Gundel, L. A., Sextro, R. G., Nazaroff, W. W., 2003. Determining size-specific emission factors for environmental tobacco smoke particles. *Aerosol Science and Technology* 37 (10), 780-790.
- Klepeis, N. E., Nazaroff, W. W., 2006. Modeling residential exposure to second-hand tobacco smoke. *Atmospheric Environment* (In Press).
- Klepeis, N. E., Nelson, W. C., Ott, W. R., Robinson, J. P., Tsang, A. M., Switzer, P., Behar, J. V., Hern, S. C., Engelmann, W. H., 2001. The National Human Activity Pattern Survey (NHAPS): A resource for assessing exposure to environmental pollutants. *Journal of Exposure Analysis and Environmental Epidemiology* 11 (3), 231-252.
- Kopstein, A., 2001. Tobacco Use in America: Findings from the 1999 National Household Survey on Drug Abuse. Tech. Rep. Analytic Series A-15, DHHS Publication No. SMA 02-3622, Substance Abuse and Mental Health Services Administration, Office of Applied Studies, Rockville, MD.
- Lai, A. C. K., 2002. Particle deposition indoors: A review. *Indoor Air* 12 (4), 211-214.
- Layton, D. W., 1993. Metabolically consistent breathing rates for use in dose assessments. *Health Physics* 64 (1), 23-36.
- McMillen, R. C., Winickoff, J. P., Klein, J. D., Weitzman, M., 2003. US adult attitudes and practices regarding smoking restrictions and child exposure to environmental tobacco smoke: Changes in the social climate from 2000-2001. *Pediatrics* 112 (1), e55-e60.
- Miller, S. L., Leiserson, K., Nazaroff, W. W., 1997. Nonlinear least-squares minimization applied to tracer gas decay for determining airflow rates in a two-zone building. *Indoor Air* 7 (1), 64-75.
- Murray, D. M., Burmaster, D. E., 1995. Residential air exchange rates in the United States - Empirical and estimated parametric distributions by season and climatic region. *Risk Analysis* 15 (4), 459-465.
- Nazaroff, W. W., Singer, B. C., 2004. Inhalation of hazardous air pollutants from environmental tobacco smoke in US residences. *Journal of Exposure Analysis and Environmental Epidemiology* 14 (Suppl. 1), S71-S77.
- OEHHA, 1997. Health Effects of Exposure to Environmental Tobacco Smoke. Tech. rep., California Environmental Protection Agency, Office of Environmental Health Hazard Assessment (OEHHA), Sacramento, CA.
URL http://www.oehha.org/air/environmental_tobacco/finalalets.html
- Ott, W. R., Klepeis, N. E., Switzer, P., 2003. Analytical solutions to compartmental indoor air quality models with application to environmental tobacco smoke concentrations measured in a house. *Journal of the Air and Waste Management Association* 53 (8), 918-936.
- Özkaynak, H., Xue, J., Spengler, J., Wallace, L., Pellizzari, E., Jenkins, P., 1996. Personal exposure to airborne particles and metals - Results from the particle TEAM study in Riverside, California. *Journal of Exposure Analysis and Environmental Epidemiology* 6 (1), 57-78.
- Pyle, S. A., Haddock, G. K., Hymowitz, N., Schwab, J., Meshberg, S., 2005. Family rules about exposure to environmental tobacco smoke. *Families, Systems, & Health* 23 (1), 3-16.
- Quackenboss, J. J., Lebowitz, M. D., Crutchfield, C. D., 1989. Indoor-outdoor relationships for particulate matter - exposure classifications and health effects. *Environment International* 15 (1-6), 353-360.
- R Development Core Team, 2005. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria, ISBN 3-900051-07-0.
URL <http://www.R-project.org>
- Schuster, M. A., Franke, T., Pham, C. B., 2002. Smoking patterns of household members and visitors in homes with children in the United States. *Archives of Pediatrics & Adolescent Medicine* 156 (11), 1094-1100.
- Spengler, J. D., Treitman, R. D., Tosteson, T. D., Mage, D. T., Soczek, M. L., 1985. Personal exposures to respirable particulates and implications for air-pollution epidemiology. *Environmental Science and Technology* 19 (8), 700-707.
- USCB, 2002. American Housing Survey for the United States: 2001. Tech. Rep. Series H150/01, U.S. Census Bureau, Current Housing Reports, U.S. Government Printing Office, Washington, DC.
- USDHHS, 2005. Report on Carcinogens, Eleventh Edition. Tech. rep., U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, Washington, DC.
URL <http://ehp.niehs.nih.gov/roc>
- USEPA, 1992. Respiratory Health Effects of Passive Smoking: Lung Cancer

- and Other Disorders. Tech. Rep. EPA/600/6-90/006F, U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC.
URL <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=2835>
- Wakefield, M., Banham, D., Martin, J., Ruffin, R., McCaul, K., Badcock, N., 2000. Restrictions on smoking at home and urinary cotinine levels among children with asthma. *American Journal of Preventive Medicine* 19 (3), 188–192.
- Wilson, A. L., Colome, S. D., Tian, Y., Becker, E. W., Baker, P. E., Behrens, D. W., Billick, I. H., Garrison, C. A., 1996. California residential air exchange rates and residence volumes. *Journal of Exposure Analysis and Environmental Epidemiology* 6 (3), 311–326.
- Winickoff, J., Berkowitz, A., Brooks, K., Tanski, S., Geller, A., Thomson, C., Lando, H., Curry, S., Muramoto, M., Prokhorov, A., Best, D., Weitzman, M., Pbert, L., 2005. State-of-the-art interventions for office-based parental tobacco control. *Pediatrics* 115 (3), 750–760.
- Xu, M. D., Nematollahi, M., Sextro, R. G., Gadgil, A. J., Nazaroff, W. W., 1994. Deposition of tobacco smoke particles in a low ventilation room. *Aerosol Science and Technology* 20 (2), 194–206.
- Zartarian, V. G., Ott, W. R., Duan, N. H., 1997. A quantitative definition of exposure and related concepts. *Journal of Exposure Analysis and Environmental Epidemiology* 7 (4), 411–437.