MODELING RESIDENTIAL EXPOSURE TO SECONDHAND TOBACCO SMOKE

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ABSTRACT

We apply a simulation model to explore the effect of a house's multicompartment character on a nonsmoker's inhalation exposure to secondhand tobacco smoke (SHS). The model tracks the minute-by-minute movement of people and pollutants among multiple zones of a residence and generates SHS pollutant profiles for each room in response to room-specific smoking patterns. In applying the model, we consider SHS emissions of airborne particles, nicotine, and carbon monoxide in two hypothetical houses, one with a typical 4-room layout and one dominated by a single large space. We use scripted patterns of room-to-room occupant movement and a cohort of 5,000 activity patterns sampled from a US nationwide survey. The results for scripted and cohort simulation trials indicate that the multicompartment nature of homes, manifested as inter-room differences in pollutant levels and the movement of people among zones, can cause substantial variation in nonsmoker SHS exposure.

Key Words: indoor air quality, multiple compartments, human activity patterns, particles, nicotine

1 INTRODUCTION

Secondhand tobacco smoke (SHS) consists of contaminants present in the air owing to the combustion of tobacco products, most commonly cigarettes. It is a mixture of exhaled mainstream smoke and sidestream smoke from smoldering tobacco that has been diluted with ambient air, and consisting of thousands of organic and inorganic chemical species in both gaseous and particle phases (Jenkins et al., 2000). Nicotine is a major volatile organic constituent of SHS (Daisey et al., 1998; Singer et al., 2003), which has contributed to its extensive use as an SHS tracer. Inhalation exposure to SHS has been associated with many maladies, including sudden infant death syndrome (SIDS), lung cancer, and heart disease mortality (NCI, 1999; CARB, 2005).

Summed over populations, homes are recognized as the predominant locations where people are exposed to SHS (Klepeis et al., 2001). SHS contributes significantly to residential particulate air pollution, increasing concentrations by 10's of μg m^{-3} on average (Özkaynak et al., 1996; Neas et al., 1994; Spengler et al., 1985). It appears that 30−40% of children in the US are at risk of exposure to SHS in their home (McMillen et al., 2003; Schuster et al., 2002).

We hypothesize that the complex dynamics of SHS pollutants and the complex behavior of human beings in typical multiroom dwellings can result in a large range of nonsmoker exposures to SHS constituents. To the extent that this hypothesis is true, understanding the variation in exposure and the role of influencing factors could be important for epidemiological studies and risk assessments. Knowledge about this variability could also form a basis for developing interventions aimed at reducing SHS exposure.

Löfroth (1993) determined that moderate to substantial differences in concentrations of SHS pollutants can occur between rooms of smoking households when interior doors are left open. Field studies of combustion air pollutants in homes containing gas stoves or heaters have shown that air pollutant concentrations can vary significantly among rooms (Palmes et al., 1977).

Research on modeling human exposure to air pollution, including multizone exposure in residences, has been active for a few decades (Ott et al., 1988; Sparks et al., 1991; Koontz and Nagda, 1991; Wilkes et al., 1992; Burke et al., 2001). However, previous modeling efforts have not precisely characterized the influence of housing characteristics and human activity on SHS exposure. The current work builds on past proven multizone indoor air pollutant or exposure models and applications, particularly those by Nazaroff and Cass (1989), Sparks et al. (1991), Koontz and Nagda (1991), Wilkes et al. (1992), Miller and Nazaroff (2001), and Ott et al. (2003). Our broad goals are to iden-

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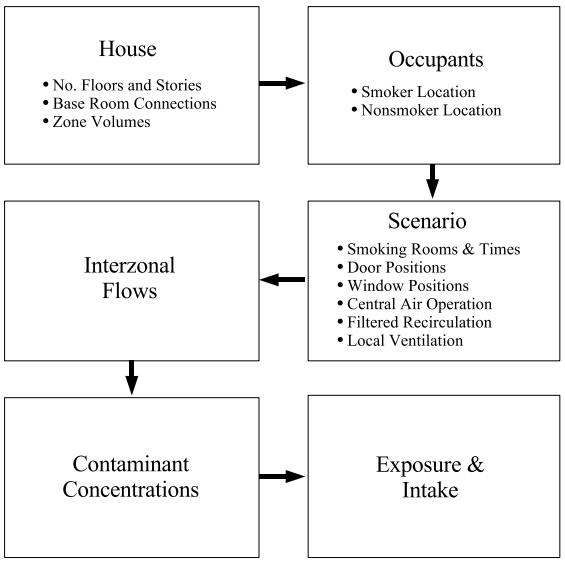

Figure 1: The logical flow between components of a simulation model used to predict residential exposure to SHS pollutants.

tify and quantify important determinants of residential SHS exposure. Specifically, in this paper we seek to elucidate the influences of contaminant transport and occupant location in residential environments in which room-to-room concentrations can vary.

2 METHODS

We have developed a computerized model that tracks the individual minute-by-minute location of a smoker and a nonsmoker as they move among rooms of a house during a single day. The model incorporates key aspects of the house configuration, including time-dependent door and window positions and the operation of a central air handling system. The model was developed using the R language (R Development Core Team, 2005).

Figure 1 depicts the conceptual flow of the model. First, the house and occupants are selected. Next, a scenario is selected, which includes specification of the behavior of the occupants with respect to doors, windows, central air operation, filtration, and smoking restriction by time or room. Dynamic interzonal air flow rates are determined for each particular scenario. Finally, the model calculates room-by-room contaminant concentrations and smoker and nonsmoker exposure and intake. Figure 2 presents an example of simulated time profiles for cigarette activity, occupant location, room concentrations, and smoker/nonsmoker exposures.

The model assumes pollutants are well mixed within each room. This assumption is supported by studies that show SHS concentrations rapidly achieve spatial uniformity under typical residential conditions (Klepeis, 1999). Although close proximity to a pollutant source may lead to elevated instantaneous exposure (McBride et al., 1999), time-averaged exposures are unlikely to be strongly affected by within-room proximity.

We define SHS exposure as the confluence of a person and SHS contaminant concentrations. We simulate a minute-by-minute SHS exposure time series for smoking and nonsmoking occupants by matching the minutes they spend in each room of the house over a 24-h period with the contaminant concentrations attributed to SHS simulated in each room. We assume SHS concentrations are well-mixed in each room so that a person's breathing zone SHS concentration is identical to the SHS concentration in the room that he or she occupies. The 24-h "integrated exposure" and "exposure concentration" refer to the integral or time average of the exposure time series in units of μg m^{-3}-min or μg m^{-3}, respectively. To estimate SHS pollutant intake [μg d^{-1}], we multiply integrated exposure by an inhalation rate of 7.8 m^3 d^{-1}, which is representative of inhalation rates for children or for an inactive female adult. This inhalation rate is consistent with oxygen needs for human metabolism in contrast to adult rates of about 20 m^3 d^{-1}, which are commonly used as reference values in risk assessments (Layton, 1993).

The following sections summarize the components of the simulation model, including data inputs, structure, and assumptions, as well as the design of simulation trials. We used fixed physical and environmental model parameters for most of the simulation experiments reported here. The parameter values represent central estimates based on the current literature.

2.1 RESIDENTIAL AIR FLOW CHARACTERISTICS

A house's size, its layout, and the air flow rates across its room boundaries determine how emitted pollutants are dispersed among rooms or removed from the house. A home's door positions, window positions, or the operation of its air handling unit may change over time, altering the flow rates between rooms and with the outdoors.

During simulations, the model translates the overall house configuration at any moment into interzone air flow rates. Broadly, we model the flow of air into and out of the rooms in each simulation by establishing a starting (base) state where all interior doors are open, all exterior windows are closed, the central air handling equipment is turned off, and leakage flows from outdoor air exchange are symmetric (balanced) into and out of each room. This base state is perturbed during particular time intervals as conditions change, such as when a door is closed, the heating and air-conditioning (HAC) system is activated, or there are asym-

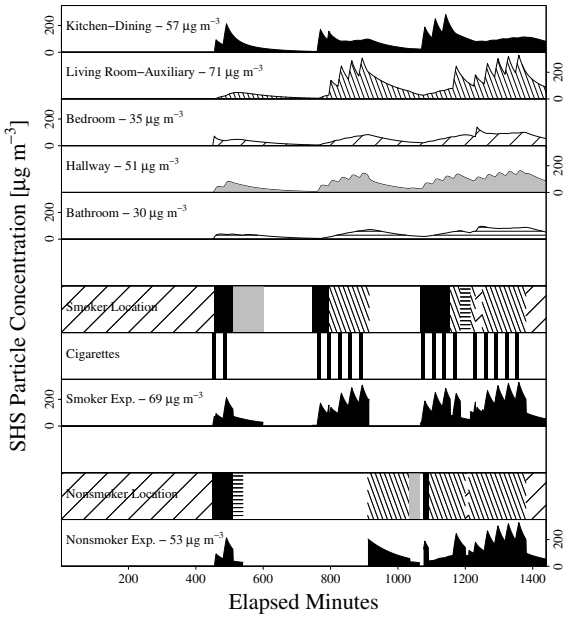

Figure 2: Example simulated 24-h time-profiles for the base flow state of House #2. Labels indicate 24-h average room and exposure concentrations. Fill patterns for room concentrations match those of the location profiles. Blank space in location patterns corresponds to time spent outside of the house.

metric leakage flows. For this paper, windows are always assumed to be closed.

We use a representative value of 0.5 h^{-1} for the whole-house leakage air-exchange rate (Murray and Burmaster, 1995). Under base conditions, symmetric indoor-outdoor leakage rates are assigned to each room in proportion to the room's volume. Asymmetric leakage air flows in houses are likely to occur when wind drives flow from one side of the house to another. For asymmetric cases, we use the same total whole-house ventilation rate as for the symmetric base case, but we assign incoming flows from the outdoors to a set of "inlet" rooms, and outgoing flows to the outdoors via separate "outlet" rooms.

When the HAC fan is operating, it is assumed that five house volumes per hour of air flow are distributed to rooms with supply registers (Bearg, 1993) in proportion to their volume. The HAC system induces recirculation of air so that it travels from supply vents to a single return vent located in the hallway. For simulations with intermittent HAC duty, the system is active for 10% of the total time any occupant is awake for individual "on" periods of 10-min. To be consistent with most modern US residences, we consider only central, forced-air heating and air conditioning with no ventilation component. We assign 10% of the flow to leaks from supply ducts to the outdoors and add it back as infiltration into each room from the outdoors (in response to depressurization). An HAC-induced infiltration effect of this magnitude has been described (Robison and Lambert, 1989).

When leakage flows are symmetric and the HAC fan is off, flows across interior doorways are immediately assigned equal values in either direction. Representative values of 100 m^3 h^{-1} and 1 m^3 h^{-1} were selected for air flow through open and closed doorways, respectively (Miller and Nazaroff, 2001; Ott et al., 2003). Under conditions with asymmetric leakage flow or HAC fan operation, the flows across doorways are unbalanced. For these cases, we devised a heuristic to assign directional air flows through rooms, balancing the air flow of each room, of the HAC system, and, thus, of the house as a whole. Generally, this balancing procedure "pushes" excess air flow from inlet to outlet rooms or from supply to return registers through paths of least resistance. Open doors are assigned zero resistance while closed doors impede air flow so that some of the excess contributes to enhanced building leakage. After the balancing procedure, symmetric flows are superimposed over asymmetric doorway flows so that the two cases have comparable flow magnitudes.

2.2 HUMAN ACTIVITY PATTERNS

We expect the location of the receptor and source individuals, and especially the relationship between their time-locations, to play a key role in determining exposure outcomes. To study this effect, we designed scripted location patterns with different degrees of smoker-nonsmoker spatial overlap for use in simulations. Subsequently, to provide realistic variation in location patterns during simulations, we sampled in-home location data for people who participated in the USEPA's National Human Activity Pattern Survey (NHAPS). These data provide minute-by-minute information about the daily duration and sequence of time spent by Americans in various locations, including the rooms of their home (Klepeis et al., 2001).

Figure 3 (right panel) illustrates the character of the NHAPS time-activity data using plots of stacked location timelines. Each individual in the figure is represented by a thin horizontal strip with different fill patterns designating the different rooms in their own home they were reported to visit. While the time-activities of NHAPS respondents are fairly specific in terms of what rooms are visited, the survey lacks information on the layout of respondent's homes including the number of rooms in their household, their uses, and their configuration. To partly overcome this data gap, we assigned adults to the master bedroom and children to an auxiliary space for sleeping. Adult smoker and nonsmoker pairs with ages within 10 years of each other were assumed to sleep in the same room.

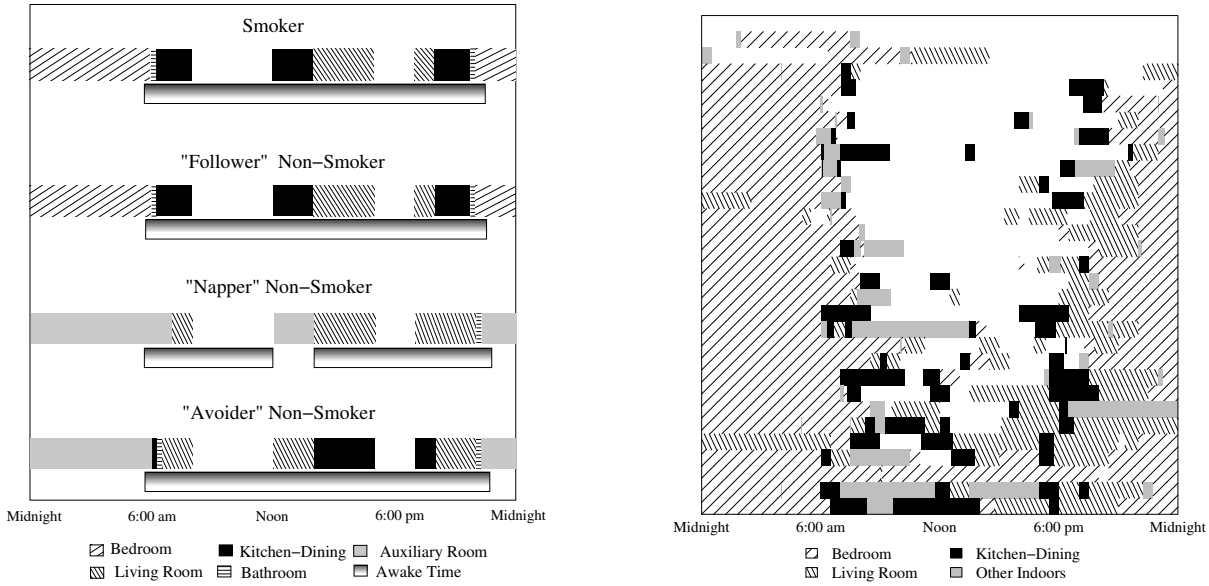

Figure 3: Scripted smoker and nonsmoker time-activity patterns used in the Phase I simulations of House #2 (left) and location patterns for 30 individuals sampled from NHAPS (right). The NHAPS sample is sorted from top to bottom by least to most total time spent at home. The vertically stacked time strips employ different fill patterns to indicate times when an individual was reported to occupy particular indoor locations. Blank spaces designate periods when the person was outside the residence.

2.3 EMISSIONS AND SMOKER CHARACTERISTICS

We selected $1\frac{1}{2}$ packs (30 cigarettes) to represent the daily consumption of a typical medium-level smoker (Nazaroff and Singer, 2004; Kopstein, 2001) with an average cigarette smoking duration of 10 min (Ott et al., 2003). Because NHAPS lacks information on the timing of smoking behavior, we superimposed smoking activity on the in-home movement of each designated smoker. We assumed that cigarettes were smoked in even intervals throughout the smoker's waking hours so that the timing and location of active cigarettes smoked in the home followed naturally from each smoker's location time series. Those cigarettes that fell during times when the smoker was not in the house, or during time spent in the bathroom or hallway, were not included in the analysis.

We simulated mass concentrations of SHS particles, nicotine vapor, and carbon monoxide (CO), which is a representative nonreactive gas. We did not differentiate by particle size or incorporate particle deposition in the lung. Gravimetrically determined values for total SHS particle emission factors reported in the literature for cigarettes are in the approximate range of $8-20$ mg cig^{-1} (see Klepeis et al., 2003). We chose 10 mg cig^{-1} to represent typical SHS particle emissions from a cigarette and 50 mg cig^{-1} to represent typical cigarette CO emissions (Martin et al., 1997). To represent typical SHS nicotine emissions, we selected 5 mg cig^{-1} based on reported values for total sidestream mass yield (Daisey et al., 1994; Eatough et al., 1989).

From reported deposition rates (Xu et al., 1994) and the mass-weighted size distribution of SHS particles (Klepeis et al., 2003), we selected a size-integrated value of 0.1 h^{-1} for the SHS particle deposition loss-rate coefficient. Particle deposition is an appreciable but not critical removal mechanism for SHS, since total particle removal is likely to be dominated by ventilation at typical rates on the order of $0.5-1$ h^{-1} for exchange with the outdoors and as much as 5 h^{-1} for exchange between rooms.

In contrast to SHS particles, which undergo irreversible deposition, nicotine and other semi-volatile SHS species sorb rapidly and substantially to indoor surfaces (Löfroth, 1993; Piadé et al., 1999) and may subsequently desorb from surfaces, slowly reentering indoor air. Van Loy et al. (2001) found that a linear kinetic model provided a good fit to their data for experiments involving carpet and wallboard. We used their model and their estimated coefficient values for wallboard, which were 1.4 m h^{-1} and 0.00042 h^{-1}, respectively. This model assumes that the sorption of nicotine onto surfaces is fully reversible, an aspect of nicotine dynamics that remains unresolved (Piadé et al., 1999; Singer et al., 2003)

2.4 MODELING INDOOR AIR QUALITY

In exploring residential SHS exposure, we consider two indoor air quality (IAQ) models, one for average concentrations in a single interior space and one for dynamic concentrations in a multizone indoor environment. Our use of the single zone and multi-compartment IAQ models fol-

lows prior formulations by Nazaroff and Cass (1989) and Nazaroff and Singer (2004).

The governing mass balance equation for nonsorbing SHS pollutants in a single zone can be written as follows:

$$\frac{dy(t)V}{dt} = -VAy(t) - VDy(t) + n(t)e \qquad (1)$$

where $y(t)$ is the airborne concentration of SHS in the zone at time t [μg m^{-3}], V is the volume of the zone [m^3], A is the outdoor air-exchange rate [h^{-1}], D is the rate coefficient for loss due to other first-order processes [h^{-1}] such as particle deposition, $n(t)$ is the number of active cigarettes at time t, and e is the emission rate for a single cigarette [μg cig^{-1} h^{-1}]. This equation assumes that air in the zone is well mixed and that air from outside of the zone is pollutant free. A solution to Equation 1 for estimating concentrations of SHS particles in a single-zone structure is:

$$\overline{y} = f\frac{\tilde{N}E}{V(A+D)} \qquad (2)$$

where $\overline{y}$ is the average airborne SHS pollutant exposure concentration [μg m^{-3}] in the house over averaging time T, which for convenience is set equal to 1 day, $\tilde{N}$ is the number of cigarettes smoked in the residence over the one-day period [d^{-1}], E is the SHS pollutant mass emission factor [μg cig^{-1}], and f is a correction factor. Equation 2 can be interpreted as an approximation to the daily average exposure concentration occurring in a well mixed house. In this case, the factor f can account for compartmental and proximity effects, so that a modeled occupant can experience lower- or higher-than-average concentrations. The factor can also account for time spent by the nonsmoker outside of the home, which may correlate in time with higher or lower indoor SHS species concentrations. Equation 2 is strictly valid under certain restrictions and approximately true in general.

The central component of our investigation is a multizone IAQ model, which describes pollutant dynamics and generates time series of pollutant concentrations in different rooms of the house. The rate of change in particle mass for a given compartment is equal to a flow rate weighted linear combination of concentrations in all compartments plus terms to account for emissions and deposition within the compartment. The system is expressed as n coupled linear differential equations, one for each well-mixed compartment, with time dependent coefficients, $k_{ij}(t)$:

$$\frac{dy_i(t)}{dt} = k_{i0}(t) + k_{i1}(t)y_1(t) + k_{i2}(t)y_2(t) + \cdots + k_{in}(t)y_n(t) \qquad (3)$$

where y_i, $i = 1,2,3,\ldots,n$, are the pollutant masses present in each air compartment, and $k_{ij}(t)$, $i = 1,2,3,\ldots, n$; $j = 0,1,2,3,\ldots, n$, are the time dependent coefficients corresponding to rates of gain (positive) or loss (negative) of pollutant mass. For brief intervals, the $k_{ij}(t)$ coefficients in Equation 3 can be considered constant and written in terms of fixed physical parameters, including per-room emission rates, and air flow rates between zones, to and from the outdoors, and to and from a forced-air system. The system is solved using a Runge-Kutta algorithm (Galassi et al., 2005), where the response variable values at the end of one time step are used as input into the next step at which time the parameter values might have changed. We used this approach to study the exposure of household residents to SHS when they visit various rooms in a house as environmental conditions changed arbitrarily across time.

To treat gaseous species that sorb onto room surfaces and potentially desorb back into the air, we used $2n$ coupled compartment equations, with n equations for air compartments and n additional equations for *surface compartments*, one per room. We simulated sorption using the model of Van Loy et al. (1997, 2001). Mass is added to the surface compartment and simultaneously subtracted from air compartments through a linear sorption term, $v_i \frac{S_i}{V_i} y_i(t)$, where v_i is the sorption uptake coefficient [m h^{-1}] and $\frac{S_i}{V_i}$ is the sorbing surface-to-volume ratio for room i [m^{-1}]. Mass is removed from the surface compartment through a desorption term, $\xi_i z_i(t)$, where ξ_i is the desorption rate coefficient [h^{-1}], and $z_i(t)$ is the sorbed contaminant mass present in surface compartment i [mg m^{-2}].

We determined values of the correction factor f in Equation 2 by dividing 24-h average particle exposure predicted by the dynamic, multi-zone model by the single-zone model prediction of exposure when using a value of f=1. The calculated values of f provide a measure of the error in the single-zone model relative to the multizone model.

2.5 DESIGN OF SIMULATION TRIALS

In Phase I of our research, we performed 72 scripted simulation trials where we restricted the movement of house occupants to a small number of hypothetical location patterns. For these trials we studied SHS nicotine and particles, two types of houses (see Table 1 and Figure 4), three types of non-smoker location profiles (Figure 3, left panel), three levels of HAC activity (off, 10% intermittent, and 100% continuous), symmetric or asymmetric leakage flows, and fresh or nicotine-loaded surfaces (0 and 50 mg m^{-2}). We defined a base simulation trial generally corresponding to base air flow conditions (HAC off and symmetric leakage flows). Windows were always closed and interior doors were open, except for bathroom usage or sleeping periods. For time when the nonsmoker and smoker occupied the same room, the door behavior of the nonsmoker took precedence. We used the fixed input parameter values described above for all scripted trials. The first scripted nonsmoker

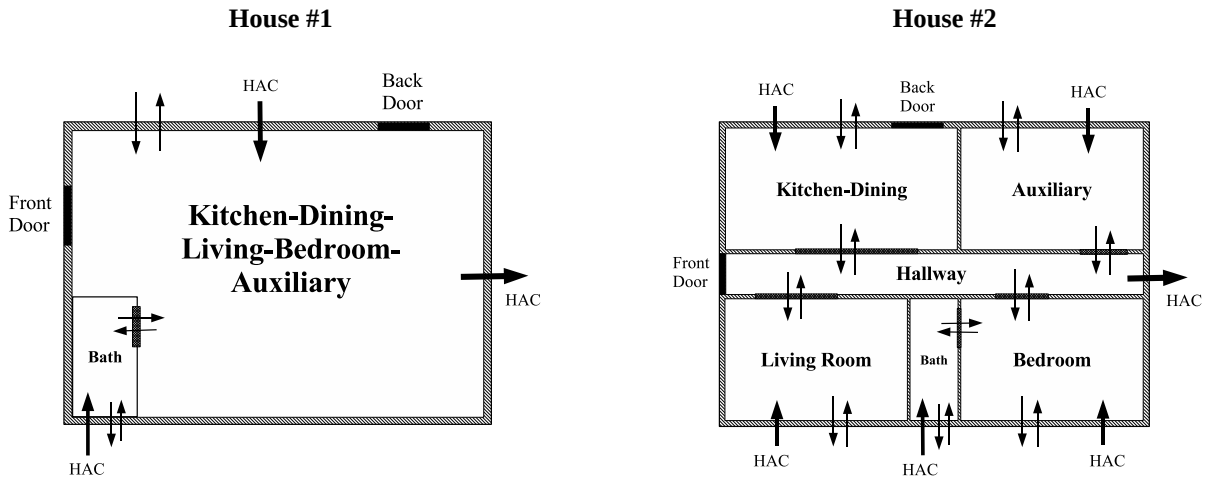

Figure 4: Floor plans showing indoor and outdoor connections and HAC supply and return air paths for two houses used in the simulation of residential SHS exposure (also see Table 1).

location profile, termed the "follower", corresponds to an infant, a small child, or perhaps a spouse, who spends all their at-home time in the same room as the smoker. The second profile, the "napper", corresponds to a child or adult who spends some time in the same room as the smoker, but takes a 2-hour nap in the middle of the day in a separate room and sleeps in a separate room at night. The third profile, the "avoider", never spends any time in the same room as the smoker.

For Phase II of our research, we randomly sampled a cohort of 5,000 location pattern pairs from NHAPS, which allowed us to explore the effect of realistic variation in movement patterns and to calculate frequency distributions of exposure for a hypothetical population. To avoid bias from differences between smoking and nonsmoking subjects or across housing types, we used the NHAPS time-location data for nonsmokers living in detached homes to provide simulation inputs for both smokers (over age 18) and nonsmokers. Because reliable information on the interdependence of activities between members of households in the US are unavailable, we matched a nonsmoker with each smoker by randomly selecting a person of any age from those who gave an interview for the same day of the week as the smoker. We did not match occupants based on housing-related variables, since the reported NHAPS activity pattern characteristics were found to be fairly uniform for reported home sizes.

Most commonly, members of the cohort smoked between 4 and 12 cigarettes in their home over the course of the day and nonsmokers spent between 50 and 80% of their day at home. Due to random sampling, the distribution of cohort pairs may underrepresent smoking and nonsmoking occupants that spend substantial amounts of time in the same room. Many of the smoker and nonsmoker matched pairs spent a small portion of their day in the same room of the house. For this reason, results taken across the complete cohort may be somewhat less relevant for certain demographic groups, such as a smoking parent who cares for a small child.

The same model input parameters and door-related behaviors were used in the cohort trials as for the scripted trials, including the number of cigarettes each smoker consumed (30 cig d^{-1}), a portion of which were smoked in the house. However, the cohort pairs were limited to occupying House #2.

As with the Phase I scripted trials, we exposed the Phase II cohort to SHS particles for both asymmetric and symmetric air flow conditions and for 10% intermittent and 100% continuous HAC operation, and we defined a base simulation trial corresponding to base air flow conditions in the house, except doors were closed for bathroom and sleeping times. We also exposed the cohort to SHS CO and nicotine under base air flow conditions and to nicotine with both fresh and loaded surfaces. We refined our specification of the initial nicotine surface concentrations in each room for cohort trials by conducting a residential simulation experiment of chronic smoking activity lasting 5,000 days. After approximately 2,000 days (5+ years) of habitual smoking, the results of this experiment showed that surface nicotine concentrations in each room had plateaued at values ranging from 40 to 80 mg m^{-2}. We used these values as input into the surface-loaded exposure simulations.

3 RESULTS AND DISCUSSION

Using the results of our Phase I scripted and Phase II cohort simulation trials, we calculated 24-h average exposure concentrations for each nonsmoker (time-integrated exposure [μg m^{-3}-min] divided by 1440 min). We also calculated the ratio of 24-h nonsmoker inhaled intake of particle

Table 1: Room Characteristics for Two Simulated Houses

House No.	[a]Rooms	[b]Volume [m^3]	[c]Surface-to-Volume Ratio [m^{-1}]	[d]HAC Flow [$m^3\ h^{-1}$]	[e]Base Leakage Flow [$m^3\ h^{-1}$]
1	Main*	280	1.2	+1260/−1435	140
	Bathroom	7	3.5	+32	3.5
2	Kitchen-Dining*	100	1.6	+502	50
	Living Room	50	1.9	+251	25
	Bedroom	50	1.9	+251	25
	Auxiliary Room	50	1.9	+251	25
	Hallway*	30	2.2	−1435	15
	Bathroom	7	3.5	+36	3.5

[a]Rooms with an asterisk (*) are inlet rooms for asymmetric flow cases.
[b]The total house volume of 287 m^3 for both houses was selected based on central estimates of house floor area derived from the results of the 2001 American Housing Survey (USCB, 2002).
[c]Minimum surface-area-to-volume ratios corresponding to rooms with listed volumes.
[d]Air flow for supply (+) or return (−) to/from the house HAC system for each room of the house occurring when the HAC is active. The supply rates have been reduced by 10% from the designed flow rate of 5 h^{-1} to account for supply duct leakage.
[e]Air flow between each room and the outdoors due to base leakage through building cracks and crevices when the HAC is inactive. During HAC operation, supply duct leakage leads to additional infiltration.

mass to the total mass of particles emitted by cigarettes in the home over the same 24-h period, i.e., the *intake fraction*, in units of parts per million ($\frac{mg\ inhaled}{mg\ emitted} \times 10^6$) (Bennett et al., 2002). For selected trials, we calculated the nonsmoker's particle intake relative to the emissions from a single cigarette, i.e., the *equivalent daily SHS cigarette particle intake*, in units of percent of a cigarette's SHS per day ($\frac{mg\ inhaled\ d^{-1}}{mg\ emitted\ cig^{-1}} \times 100$). Generally, the mean (median) of all exposure metrics for the full cohort was found to be stable, having a 90% confidence band half-length less than 10% of the mean (median).

About 200 of the original cohort sample size of 5,000 were not analyzed because zero cigarettes were smoked in the house or zero time was spent by the nonsmoker in the house. Those cohort pairs for which the nonsmoker spent more than $\frac{2}{3}$ of their time at home and the smoker consumed 10−30 cigarettes in the home during the day tended to have higher absolute exposures. (The maximum theoretical number of cigarettes that could be smoked in the house was 30, since each smoker in the cohort smoked at a fixed rate of 30 cig d^{-1}). We refer to this set as the "high risk" group (n = 1037). The exposure metrics for the high risk nonsmokers were approximately lognormally distributed so that different cohort simulation trials could be conveniently compared in terms of their geometric means (GM) and geometric standard deviations (GSD).

3.1 EFFECT OF HUMAN ACTIVITY

Broadly, our results from Phase I and II show that the multicompartment character of homes can lead to substantial variation in nonsmoker exposure for major components of SHS (total particle mass, CO, and nicotine). Throughout our analyses of scripted and cohort exposures, we found that the amount of time spent in the same room as a smoker had a large effect.

For example, in House #1, which has a single large living area, the base Phase I scripted 24-h SHS particle exposure concentration was 33 $\mu g\ m^{-3}$ and the individual intake fraction was 1400 ppm, regardless of the nonsmoker activity pattern. In contrast, the 24-h average exposure concentration of House #2 occupants varied from 24 to about 60 $\mu g\ m^{-3}$ and their intake fraction varied from 1000 to 2600 ppm, when going from "avoider" to "follower" behavior (Table 2). Notably, these *individual* intake fractions are much larger than estimates of *population* intake fraction for pollutant releases to outdoor air from motor vehicles in US urban areas (Marshall et al., 2005) or power plants (Levy et al., 2003), which average approximately 14 ppm and 1 ppm, respectively. The results obtained for SHS here compare favorably to the limited intake fraction information published for indoor releases. For example, Smith (1993) estimated a US population intake fraction of 2400 ppm for indoor exposure to SHS particles. By applying an indoor air quality model and assuming receptors occupy fixed locations in individual rooms, Lai et al. (2000) estimated individual intake fractions for residential exposure to a nonreactive gas of approximately 1500−2000 ppm.

Phase II cohort results also exhibited strong dependence on activity patterns. For example, the average 24-h SHS particle exposure concentration for high risk members of the cohort was 35 $\mu g\ m^{-3}$, which was at least double those for individuals in groups with fewer cigarettes smoked at home or less time spent at home by the nonsmoker (see Table 3). Futhermore, those nonsmokers in the high risk group who spent more than 20% of their time in the same room as the smoker had nearly double the median particle exposure concentration of those who spent under 20% of their time with the smoker. The interquartile range in

Table 2: Phase I. Average 24-h Nonsmoker SHS Particle Exposure Concentration [μg m^{-3}] and Individual Intake Fraction (iF) [ppm] for Scripted Simulation Trials in House #2 by Flow Scenario and Nonsmoker Activity

Exposure Metric	Flow Scenario[a]	Scripted Nonsmoker Activity		
		Follower	Napper	Avoider
24-h Ave.	HAC-0%—Symm (Base)	61	41	24
	HAC-10%—Symm	53	36	23
	HAC-100%—Symm	30	23	16
	HAC-0%—Asymm	59	45	25
	HAC-10%—Asymm	52	40	24
	HAC-100%—Asymm	31	25	17
iF	HAC-0%—Symm (Base)	2600	1700	1000
	HAC-10%—Symm	2200	1500	970
	HAC-100%—Symm	1200	970	660
	HAC-0%—Asymm	2500	1900	1000
	HAC-10%—Asymm	2200	1700	1000
	HAC-100%—Asymm	1300	1000	700

[a]Combinations of HAC activity and flow symmetry. The "base" flow scenario was defined as when the HAC was off and leakage flows were symmetric across house boundaries.

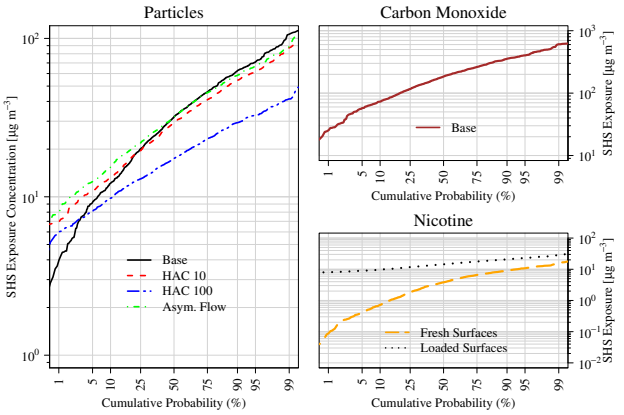

Figure 5: Phase II results. Log-probability plots of 24-h average particle, carbon monoxide, and nicotine SHS exposure of nonsmokers simulated for 1,037 "high risk" cohort households.

the simulated base frequency distribution of 24-h particle exposure concentration was more than 30 μg m^{-3}. Ninety-eight percent of simulated 24-h mean particle nonsmoker exposure concentrations for high risk individuals spanned a factor of roughly 30 from about 4 to more than 100 μg m^{-3} (Figure 5).

Although CO does not undergo surface deposition as particles do, the pattern of CO exposure metrics was similar to that for particles. For the high risk simulated cohort, the cohort mean 24-h average SHS CO exposure concentration was 200 μg m^{-3} (0.2 ppm). The central ninety-eight percent of simulated 24-h CO exposure concentration for high risk nonsmokers ranged from approximately 25 to 600 μg m^{-3} (Figure 5). These exposures are relatively low compared to health standards for CO. However, such exposures scale linearly with per-cigarette emissions. Therefore, these results can be used to estimate exposure to any nonreactive gaseous SHS component for which emission factors are known.

We obtained average SHS equivalent particle intakes of 5% and 2.5% SHS cigs d^{-1} for nonsmokers over a 24-h period for scripted follower and high-risk cohort simulation trials, respectively. Since the ratio of SHS to mainstream smoke (MS) yields for particles is of the order of 1 (Jenkins et al., 2000), over months or years of exposure, a person living with a smoker might inhale tobacco smoke particles equivalent to actively smoking many packs of cigarettes. Furthermore, for many constituents of tobacco smoke, sidestream-to-mainstream yields are of the order of 10 (Jenkins et al., 2000, Table 3.2). Nonsmoker inhalation intake of these species expressed in equivalents to the number of cigarettes smoked per day would be correspondingly higher than determined here for SHS particles.

Because nicotine sorbs rapidly and strongly onto surfaces, the simulated relative differences in room-to-room concentrations during smoking are greater for nicotine than for particles or CO. This effect was also observed during Löfroth's house monitoring studies (1993). The simulation results indicate that avoidance behavior had a larger proportional effect on exposure to nicotine than for either particles or CO. There were sharp decreases in 24-h average exposure concentrations across House #2 scripted nonsmoker activity under base air flow conditions and initially nicotine-free surfaces from "follower" (12 μg m^{-3}) to "napper" (7 μg m^{-3}) to "avoider" (2 μg m^{-3}).

3.2 EFFECT OF INTERZONAL AIR FLOW

The operation of a house's HAC system decreased most simulated exposures relative to the base scenario, because of increased infiltration caused by supply duct leaks. Turning on the HAC system in House #2 intermittently for 10% of daily waking times reduced 24-h average scripted exposure concentrations by 1−8 μg m^{-3} (4−13%), while continuous HAC operation during waking periods reduced average scripted exposure by 8−31 μg m^{-3} (30−50%; Table 2). With continuous operation of the HAC system during waking hours, the upper 95% of the cohort had significantly lower exposures (Figure 5). The GM of particle exposure for the high risk cohort group was 17 μg m^{-3} when the HAC was fully active and 28−31 μg m^{-3} for the other flow scenarios (Table 4). The GM of the high risk individual SHS particle intake fraction was 1200−1300 ppm for inactive or intermittent HAC conditions, dropping to 720 ppm when the HAC was continuously active.

Generally, when a home's HAC system is activated, air is expected to recirculate in the house so that air pollutants in rooms where smoking occurs are delivered more rapidly to other rooms in the house. We observed this effect for House #2; there were smaller differences between scripted exposures for different nonsmoker activities when the HAC was active (Table 2). The recirculation of pollutants by the

Table 3: Phase II. Average 24-h Nonsmoker SHS Particle Exposure Concentration [μg m^{-3}] for Cohort Simulation Trials by Fraction of Time Spent by the Nonsmoker and Smoker in the Same Room (Overlapping), Number of Cigarettes Smoked Indoors, and Fraction of Time Spent by the Nonsmoker at Home[a]

Fraction	No.	Fraction			Sample	Std.		Percentiles	
Overlap.	Cigs	at Home	n	n_{zero}	Mean	Dev.	Median	10th	90th
–	–	–	4798	48	17	17	12	1.0	39
–	< 10	< 2/3	1426	38	6.6	7.5	3.8	0.11	17
–	< 10	> 2/3	988	1	11	9.5	8.7	1.2	25
–	> 10	< 2/3	1347	9	18	14	14	3.0	36
–	> 10	> 2/3	1037	0	35	21	32	12	62
< 0.2	–	–	3728	42	14	14	10	0.83	34
< 0.2	> 10	> 2/3	731	0	29	15	27	11	50
> 0.2	–	–	1070	6	26	24	19	2.4	59
> 0.2	> 10	> 2/3	306	0	49	24	47	19	80

[a]Statistics are presented for households where more than zero time was spent at home by the nonsmoker and more than zero cigarettes were smoked. The listed results are for base air flow conditions where the HAC is off and leakage flows are symmetric. A dash (−) in a cell of a factor column signifies that for the corresponding statistics all cohort members were considered across that factor.
Abbreviations: Fraction Overlap. = fraction of the simulated 24-h period that the nonsmoking receptor and the smoker spent at home together in the same room; No. Cigs = number of cigarettes smoked in the home by the smoker during the simulated 24-h period, maximum of 30 smoked at home; Fraction at Home = fraction of the simulated 24-h period that the nonsmoking receptor spent at home; n = total simulated sample size; n_{zero} = sample size with zero exposure (included in statistics); Sample Mean = the sample arithmetic mean; Std. Dev. = the sample standard deviation; Median = the sample median; 10th and 90th = 10th and 90th percentiles of the distribution.

intermittent operation of the HAC system for 10% of waking hours caused the lower 25% of nonsmoker particle exposures in the high risk cohort group to increase slightly (Figure 5).

For both scripted and cohort simulations, the symmetry of flows across building boundaries had little effect on 24-h average nonsmoker particle exposure (Table 2 and Table 4). These effects are slight, because the directionality of flows was small compared to the base bidirectional flow across doorways.

3.3 EFFECT OF NICOTINE LOADING

When we simulated nicotine exposure for the case of substantial loading of nicotine on walls, the exposure of all household occupants increased, resulting in a decrease in the proportional range of exposures. When 50 mg m^{-2} of reversibly sorbed nicotine was initially sorbed, the scripted nicotine exposure rose for the "avoider" from 1−2 to 10−11 μg m^{-3} and for the "follower" from 7−12 to 16−21 μg m^{-3}. Long-term loading of reversibly sorbed nicotine on household surfaces in the high risk cohort resulted in more than a quadrupling in the GM of nonsmoker nicotine exposure (3 to 14 μg m^{-3}) and a large reduction in the GSD (Table 4). The distributions of 24-h average nonsmoker exposure concentration for the high risk cohort group with loaded surfaces were more nearly lognormal than for fresh surfaces and spanned less than an order of magnitude from about 8 to 30 μg m^{-3}. The fresh-surface distributions spanned three orders of magnitude from 0.1 to about 20 μg m^{-3} (Figure 5).

3.4 COMPARISON OF SINGLE-ZONE AND MULTI-ZONE EXPOSURE MODELS

The simplified model with f=1 predicted a 24-h average particle exposure concentration of 46 μg m^{-3} for all scripted scenarios (Phase I). Values of f in the single-zone exposure model ranged from 0.1 to 1.3 for scripted scenarios, indicating errors of -90 to +30%. For House #2, the unadjusted single-zone model tended to underestimate scripted "follower" nonsmoker exposure for base conditions (f = 1.3), overestimate them when the HAC was on continuously (f = 0.6), and strongly overestimate "avoider" exposure (f = 0.3−0.5). The simplified model best matched the multizone model for "napper" behavior under base conditions (f = 0.9) or when the HAC was active intermittently for either "follower" or "napper" behavior (f = 1.2 and 0.8, respectively).

For cohort simulation trials (Phase II), we calculated the geometric mean and standard deviation of the distribution of the single-zone correction factor across high risk households (GM_f and GSD_f). The unadjusted single-zone model generally overestimated multi-zone nonsmoker exposure and intake fractions for particles and CO (Table 4). For example, GM_f was 0.6−0.8 for base and asymmetric flow conditions. When the HAC was running continuously, GM_f for particles was about 0.4. In contrast, simulated multi-zone nicotine exposure was strongly underestimated by the single-zone model with GM_f equal to 5−6 when walls were preloaded with nicotine. For clean wall simulations, GM_f was only slightly above 1.0, but GSD_f was almost 3, revealing wide variation in the predictive power of the single-zone model.

3.5 LOCAL SENSITIVITY OF EXPOSURE TO ENVIRONMENTAL AND PHYSICAL PARAMETERS

We tested the sensitivity of exposure outcomes to parameter choices by conducting a new set of simulations in which we introduced small positive and negative perturbations (25%) in eight physical and environmental parameters. We calculated a normalized sensitivity coefficient, $\frac{\delta_{GM}}{\delta P}$, for each reference value and the two perturbations, where δ_{GM} is the change in the geometric mean of the 24-h average nonsmoker particle exposure concentration distribution divided by its reference value, and δP is the change in the parameter value divided by its reference value. The sensitivity analyses were only conducted using the high risk simulation cohort.

Nonsmoking exposures were exactly proportional to increases or decreases in per-cigarette emissions ($\frac{\delta_{GM}}{\delta P}$ = 1.0). For negative changes in house volume, the sensitivity coefficient was -1.22, meaning that a given decrease in volume yielded a 22% larger increase in exposure. A positive perturbation in house volume resulted in a 25% smaller reduction in exposure ($\frac{\delta_{GM}}{\delta P}$ = -0.75). A decrease in the house air-exchange rate resulted in a 87% proportionate increase in exposure and an increase resulted in a 61% proportionate decrease. In contrast, exposure was fairly insensitive to changes in particle deposition rate, only increasing by about 15% and decreasing by about 14% of the extent of negative and positive parameter perturbations, respectively. Exposure was insensitive to local perturbations in interroom air flow through open doorways and to the cigarette duration ($|\frac{\delta_{GM}}{\delta P}| < 0.1$).

4 SUMMARY AND CONCLUSIONS

We developed a mechanistic simulation tool to study the complex interplay among spatially and temporally varying factors affecting secondhand tobacco smoke (SHS) exposure in residential environments. This approach enables exploration of how small changes in individual behavior on time scales of minutes or hours can have large impacts on time-averaged exposure concentrations, owing to changes in SHS concentrations in rooms and in the proximity of the nonsmoker to SHS emissions.

During Phase I of our research, we conducted 72 scripted simulation trials of residential SHS exposure, systematically varying the time-location patterns of smoker and nonsmoker occupants in two types of hypothetical houses. In Phase II, we conducted simulation trials for a 5,000-person cohort with time-location patterns obtained from a nationally representative sample of US interviewees. We found generally that the presence of multiple compartments in residences can have a substantial effect on exposure to SHS. Key specific findings from our study are summarized below:

- Apart from pollutant-specific effects, the effect of time spent in the same room as the smoker was greater than any other study factor, leading to variation across orders of magnitude.
- Operation of a central air handling system reduced nonsmoker SHS exposure concentrations by up to ∼50% because of increased outdoor air exchange caused by supply duct leakage.
- Nonsmoker SHS exposure was sensitive to the number of cigarettes smoked indoors, SHS emission factors, house air exchange rate, and house volume.
- Nonsmoking members of a high risk cohort, for which between 10 and 30 cigarettes were smoked in the house per day and the nonsmoking receptor spent more than $\frac{2}{3}$ of the time at home, had exposure concentrations that were more than twice as large, on average, as nonsmokers in groups with fewer than 10 smoked cigarettes and less than $\frac{2}{3}$ time spent at home.
- The following results were obtained for the high risk group. The geometric means of baseline 24-h nonsmoker average SHS particle exposure concentrations were 29 μg m^{-3} for particles, similar to the reported contribution of smokers to $PM_{2.5}$ indoor air concentrations and personal respirable particle exposure (Özkaynak et al., 1996; Phillips et al., 1998). The corresponding geometric means for carbon monoxide and nicotine (initially clean surfaces) were 170 μg m^{-3} and 3 μg m^{-3}, respectively. The average SHS particle individual intake fraction was 1200 ppm and the equivalent SHS particle cigarette intake was 2.5% of a cigarette's worth of SHS emissions per day.

These investigations build on the proven success of prior work in indoor air quality modeling. The results are broadly consistent with those of large field studies, and, therefore, we expect the findings to be informative with respect to SHS concentrations in real homes. Future applications of the model could examine the effect of correlation in location patterns between smokers and nonsmokers in a given household for occupants belonging to specific demographic and role groups, such as a caregiver and a small child. We expect that this modeling framework and the simulation results could be useful to environmental health researchers and practitioners who wish to obtain a quantitative understanding of residential exposure to SHS, perhaps in connection with interventions, epidemiology, or health risk assessment.

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Table 4: Phase II. Geometric Means and Geometric Standard Deviations for Distributions of 24-h Average Nonsmoker SHS Particle, Carbon Monoxide, and Nicotine Exposure Concentration [μg m^{-3}] and Individual Intake Fraction (iF) [ppm] for Cohort Simulation Trials in High Risk Households[a]

Metric	Simulation Trial	GM	GSD	[b]GM_f	[b]GSD_f
24-h Avg	Particles–HAC-0%–Symm (Base)	29	2.0	0.72	1.8
	Particles–HAC-0%–Asym	31	1.7	0.77	1.5
	Particles—HAC 100%–Symm	17	1.5	0.42	1.4
	Particles–HAC 10%–Symm	28	1.7	0.69	1.5
	CO–HAC-0%–Symm (Base)	170	1.9	0.70	1.7
	Nicotine Fresh–HAC-0%–Symm (Base)	3.1	2.9	1.2	2.7
	Nicotine Loaded–HAC-0%–Symm	14	1.3	5.6	1.4
iF	Particles–HAC-0%–Symm (Base)	1200	1.8	0.65	1.8
	Particles–HAC-0%–Asym	1300	1.6	0.70	1.6
	Particles–HAC 100%–Symm	720	1.5	0.38	1.5
	Particles–HAC 10%–Symm	1200	1.6	0.63	1.6
	CO–HAC-0%-Symm (Base)	1400	1.8	0.63	1.8
	Nicotine Fresh–HAC-0%–Symm (Base)	260	2.8	1.1	2.8
	Nicotine Loaded–HAC-0%–Symm	1200	1.5	5.1	1.5

[a]"High risk" households are defined as those where the smoker consumes more than 10 cigarettes per day in the house and the nonsmoker spends more than two thirds of the day at home (n=1037).
[b]The values in these columns are the geometric means and standard deviations of the simplified time-averaged single-zone correction factor, f (see Equation 2).

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REFERENCES

Bearg, D. W., 1993. Indoor Air Quality and HVAC Systems. Lewis Publishers, Boca Raton.

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.

Burke, J. M., Zufall, M. J., Özkaynak, H., 2001. A population exposure model for particulate matter: Case study results for $PM_{2.5}$ in Philadelphia, PA. Journal of Exposure Analysis and Environmental Epidemiology 11 (6), 470–489.

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

Daisey, J. M., Mahanama, K. R. R., Hodgson, A. T., 1994. Toxic Volatile Organic Compounds in Environmental Tobacco Smoke: Emission Factors for Modeling Exposures of California Populations. Final Report Contract A133-186, California Air Resources Board, Sacramento, lBL No. 36379.

Daisey, J. M., Mahanama, K. R. R., Hodgson, A. T., 1998. Toxic volatile organic compounds in simulated environmental tobacco smoke: Emission factors for exposure assessment. Journal of Exposure Analysis and Environmental Epidemiology 8 (3), 313–334.

Eatough, D. J., Benner, C. L., Bayona, J. M., Richards, G., Lamb, J. D., Lee, M. L., Lewis, E. A., Hansen, L. D., 1989. Chemical composition of environmental tobacco smoke. 1. Gas-phase acids and bases. Environmental Science and Technology 23 (6), 679–687.

Galassi, M., Davies, J., Theiler, J., Gough, B., Jungman, G., Booth, M., Rossi, F., 2005. GNU Scientific Library Reference Manual - Revised Second Edition. Network Theory, Ltd., Bristol, UK.
URL http://www.network-theory.co.uk/

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.

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., 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.

Koontz, M. D., Nagda, N. L., 1991. A multichamber model for assessing consumer inhalation exposure. Indoor Air 1 (4), 593–605.

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., Thatcher, T. L., Nazaroff, W. W., 2000. Inhalation transfer factors for air pollution health risk assessment. Journal of the Air and Waste Management Association 50 (9), 1688 – 1699.

Layton, D. W., 1993. Metabolically consistent breathing rates for use in dose assessments. Health Physics 64 (1), 23–36.

Levy, J. I., Wilson, A. M., Evans, J. S., Spengler, J. D., 2003. Estimation of primary and secondary particulate matter intake fractions for power plants in Georgia. Environmental Science and Technology 37 (24), 5528–5536.

Löfroth, G., 1993. Environmental tobacco smoke: Multicomponent analysis and room-to-room distribution in homes. Tobacco Control 2, 222–225.

Marshall, J. D., Teoh, S. K., Nazaroff, W. W., 2005. Intake fraction of nonreactive vehicle emissions in US urban areas. Atmospheric Environment 39, 1363–1371.

Martin, P., Heavner, D. L., Nelson, P. R., Maiolo, K. C., Risner, C. H., Simmons, P. S., Morgan, W. T., Ogden, M. W., 1997. Environmental tobacco smoke (ETS): A market cigarette study. Environment International 23 (1), 75–90.

McBride, S. J., Ferro, A. R., Ott, W. R., Switzer, P., Hildemann, L. M., 1999. Investigations of the proximity effect for pollutants in the indoor environment. Journal of Exposure Analysis and Environmental Epidemiology 9 (6), 602–621.

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., Nazaroff, W. W., 2001. Environmental tobacco smoke particles in multizone indoor environments. Atmospheric Environment 35 (12), 2053–2067.

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., Cass, G. R., 1989. Mathematical modeling of indoor aerosol dynamics. Environmental Science & Technology 23 (2), 157–166.

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.

NCI, 1999. Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protection Agency, Smoking and Tobacco Control Monograph No. 10. Tech. Rep. NIH Pub. No. 99-4645, US Department of Health and Human Services, National Institutes of Health, National Cancer Institute, Bethesda, MD.

Neas, L. M., Dockery, D. W., Ware, J. H., Spengler, J. D., Ferris, B. G., Speizer, F. E., 1994. Concentration of indoor particulate matter as a determinant of respiratory health in children. American Journal of Epidemiology 139 (11), 1088–1099.

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.

Ott, W. R., Thomas, J., Mage, D. T., Wallace, L. A., 1988. Validation of the Simulation of Human Activity and Pollutant Exposure (SHAPE) model using paired days from the Denver, CO, carbon monoxide field study. Atmospheric Environment 22 (10), 2101–2113.

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.

Palmes, E. D., Tomczyk, C., Dimattio, J., 1977. Average NO_2 concentrations in dwellings with gas or electric stoves. Atmospheric Environment 11 (9), 869–872.

Phillips, K., Howard, D. A., Bentley, M. C., Alvan, G., 1998. Environmental tobacco smoke and respirable suspended particle exposures for nonsmokers in Beijing. Indoor + Built Environment 7 (5-6), 254–269.

Piadé, J. J., D'Andrés, S., Sanders, E. B., 1999. Sorption phenomena of nicotine and ethenylpyridine vapors on different materials in a test chamber. Environmental Science and Technology 33 (12), 2046–2052.

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`

Robison, D. H., Lambert, L. A., 1989. Field investigation of residential infiltration and heating duct leakage. ASHRAE Transactions 95 (3), 542–550.

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.

Singer, B. C., Hodgson, A. T., Nazaroff, W. W., 2003. Gas-phase organics in environmental tobacco smoke: 2. Exposure-relevant emission factors and indirect exposures from habitual smoking. Atmospheric Environment 37 (39-40), 5551–5561.

Smith, K. R., 1993. Fuel combustion, air pollution exposure, and health: The situation in developing countries. Annual Review of Energy and the Environment 18, 529–566.

Sparks, L. E., Tichenor, B. A., White, J. B., Jackson, M. D., 1991. Comparison of data from an IAQ test house with predictions of an IAQ computer model. Indoor Air 1 (4), 577–592.

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.

Van Loy, M. D., Lee, V. C., Gundel, L. A., Daisey, J. M., Sextro, R. G., Nazaroff, W. W., 1997. Dynamic behavior of semivolatile organic compounds in indoor air. 1. Nicotine in a stainless steel chamber. Environmental Science and Technology 31 (9), 2554–2561.

Van Loy, M. D., Riley, W. J., Daisey, J. M., Nazaroff, W. W., 2001. Dynamic behavior of semivolatile organic compounds in indoor air. 2. Nicotine and phenanthrene with carpet and wallboard. Environmental Science and Technology 35 (3), 560–567.

Wilkes, C. R., Small, M. J., Andelman, J. B., Giardino, N. J., Marshall, J., 1992. Inhalation exposure model for volatile chemicals from indoor uses of water. Atmospheric Environment 26A (12), 2227–2236.

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.